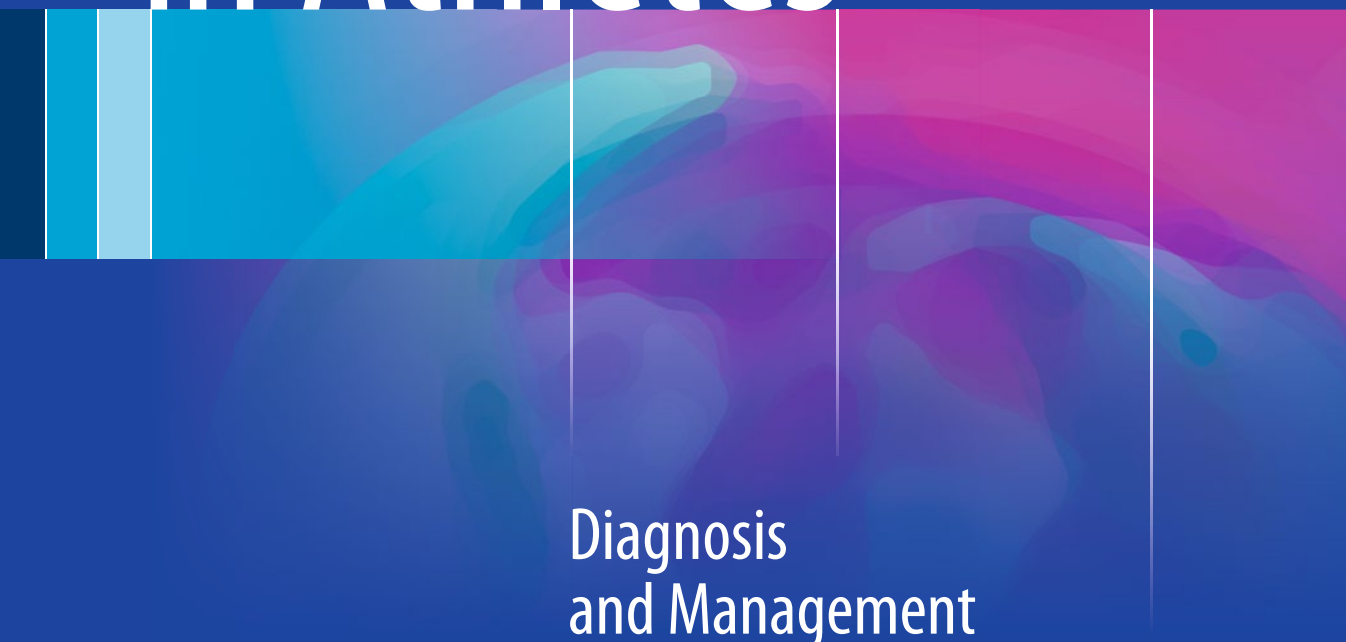
Timothy L. Miller
Christopher C. Kaeding
Editors

Stress Fractures in Athletes



Diagnosis and Management

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To my family, in particular my parents, Tom and Kathy,
and my wife, Nicole, for always believing in me and always
showing patience and understanding when I take on
"yet another project."
To the many athletes who have had their seasons
and careers cut short due to stress fractures and other overuse
injuries, know that your hard work and efforts have not gone
in vain and, in fact, were the inspiration for this book.
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and mentoring this journey and love affair with stress
fractures would have never begun.

Timothy L. Miller, MD

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advice, encouragement and "prodding." Without his influence
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Christopher C. Kaeding, MD

Preface

Stress Fractures in Athletes has been in many ways a labor of love for the editors. It is the culmination of many years of experience with stress fractures both as athletes and as team physicians. This textbook compiles the many concepts, experiences, and techniques required to approach and treat the complexities of stress-induced injuries to bone among the athletically active population. We truly appreciate the contributions of the authors—many of whom are considered pioneers and leaders in the field of Sports Medicine—who have provided their invaluable insights and pearls on the evaluation and treatment of stress fractures. As a developing field of Sports Medicine and Orthopaedics, Endurance Medicine continues to expand its understanding of overuse injuries as athletes continue to push the limits of running, jumping, biking, swimming, skiing, rowing, cross-fit sports, adventure sports, and many other demanding activities. Traditional treatment strategies for stress fractures such as simply stopping the causative activity or sport are no longer considered an acceptable option for many competitive athletes. Alternative training methods, including a holistic approach to the evaluation, treatment, and prevention of stress-induced injuries to bone, are now the standard of care as is evidenced throughout the 16 chapters of this book. This textbook details treatment options for bony injuries throughout the body from the spine and pelvis to the hands and feet. Even though it is too early to determine whether we can obviate the need to have athletes completely abstain from their sport of choice in response to a stress fracture, we can decrease the time lost from training and competition and allow for a more safe and predictable return to full activity. It is our hope that this textbook will be a valuable guide for sports medicine physicians, orthopaedists, athletic trainers, physical therapists, coaches, parents, and athletes in their evaluation and treatment of stress fractures.

Columbus, OH, USA

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Abbreviations

AIIS	Anterior inferior iliac spine
AP	Anteroposterior
ASIS	Anterior superior iliac spine
BMD	Bone mineral density
BMI	Increased body mass index
COX	Cyclooxygenases
CT	Computed tomography
DEXA	Dual-energy X-ray absorptiometry
ECSW	Extracorporeal shock wave
EMF	Electromagnetic fields
EMG	Electromyography
ESWT	External shockwave therapy
FABER	Flexion, abduction, and external rotation
MRI	Magnetic resonance imaging
NSAIDs	Non-steroidal anti-inflammatories
pQCT	Peripheral quantitative computed tomography
PSIS	Posterior superior iliac spine
PTH	Parathyroid hormone
SCFE	Slipped capital femoral epiphysis
SI	Sacroiliac
SPECT	Single-photon emission computed tomography
STIR	Short tau inversion recovery
Tc-99m-MDP	Technetium-99m-methylene diphosphonate

Part I

Presentation and Diagnosis of Stress Fractures

1 Pathophysiology and Epidemiology of Stress Fractures

David Wasserstein and Kurt P. Spindler

Stress Fracture Pathophysiology

To understand the pathophysiology of stress fractures in bone, a review of basic bone biology, including normal bone metabolism and turnover, is necessary. From this understanding, the pathophysiology of stress fracture development will be outlined. Finally, this section will identify individual clinical parameters that have been linked to the development of stress fractures, and summarize their implication and relevance.

Bone Biology

Bone has two forms at the microscopic level—woven and lamellar bone. Woven bone is immature with random orientation and collagen that is not stress oriented. Lamellar bone, in contrast, is mature and organized with stress-oriented collagen [1]. The mechanical properties of lamellar bone can change depending on the direction of the applied force. The macroscopic subtypes of lamellar bone include cortical and cancellous (trabecular) bone. The former is denser and has a low turnover rate. It is composed of packed osteons also called Haversian systems, which are connected by Haversian canals (Fig. 1.1). These canals contain the neurovascular supply of bone. Cancellous bone, however, has a higher turnover and is between 30 and 90 % porous, depending on the location. Cancellous bone is found more commonly in the metaphysis of long bones, compared to cortical bone, which is found in the diaphysis.

The matrix of bone is approximately 40 % organic and 60 % inorganic [1]. The organic portion of bone is primarily type-1 collagen—the component that provides tensile strength. The remaining organic portion (~10 %) consists of proteoglycans, which provide compressive strength, and matrix proteins. The function of these matrix proteins (e.g., osteocalcin) is to promote mineralization and bone formation. The inorganic component includes calcium hydroxyapatite, which is responsible for compressive strength, and osteocalcium phosphate. The inorganic component is also the mineral portion, which plays a role in calcium metabolic pathways [1].

Normal bone metabolism is a balanced sequence of bone turnover that includes bone breakdown, known as osteoclastogenesis, and bone formation, known as osteoblastogenesis. Osteoclasts are the cells primarily responsible for osteoclastogenesis, and osteoblasts for

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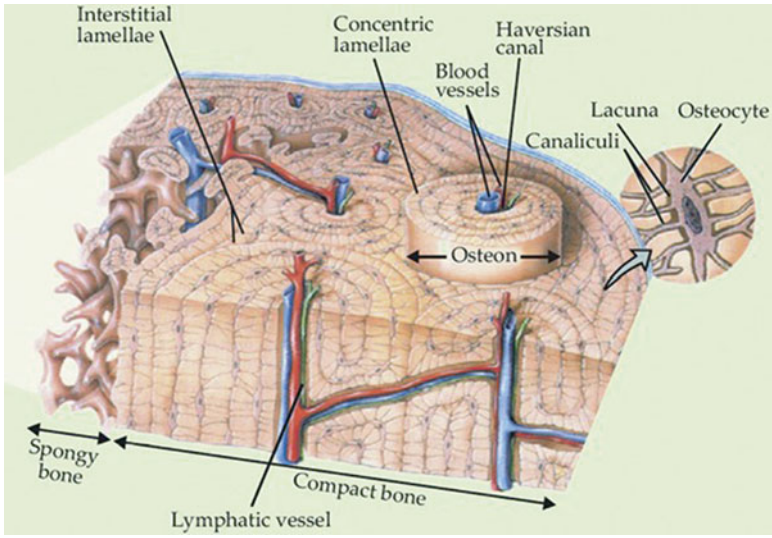

Fig. 1.1 Illustration of the Haversian system and vascular supply in cortical bone. With permission from Springer Science + Business Media: Initiation Fracture Toughness of Human Cortical Bone as a Function of Loading Rate, 2013, C. Allan Gunnarsson

osteoblastogenesis. Many endogenous hormones regulate metabolism, including parathyroid hormone (PTH), calcitonin, growth hormone, thyroid hormone, estrogen, and testosterone. Endogenous and exogenous steroids, including vitamin D and glucocorticoids, also regulate both calcium and bone metabolism [1]. Factors that promote bone formation do so by either promoting osteoblastogenesis (e.g., PTH, vitamin D) or suppressing osteoclastogenesis (e.g., calcitonin, estrogen). Factors that promote bone breakdown typically suppress osteoblastogenesis (e.g., glucocorticoids).

When stress is applied to bone, Wolff's law dictates that bone will remodel in response to mechanical stress. The exact method by which bone remodels is not truly understood, but two theories predominate. In the piezoelectric charge theory, tensile-sided strain is said to create electropositive forces that stimulate osteoclastogenesis, while the compression side is subject to electronegative forces that stimulate osteoblastogenesis [1]. The result is the formation or remodeling of bone to increase bone mass on the compressive side in response to mechanical stress. A second theory, the Hueter–Volkmann law, states that bone remodels in small packets of cells in a process called osteoclastic tunneling. Here, there is bone resorption followed by capillaries to introduce blood supply and osteoid-producing cells to lay down new osteoid [1].

Bone Pathophysiology in Stress Fractures

"Stress fracture" constitutes a spectrum of injury that includes bone strain, stress reaction, and stress fracture. The etiology is repetitive loading in the setting of inadequate bone remodeling. The spectrum of injury reflects to some degree the quantity of strain, although exact thresholds are not known and likely mediated by numerous individual host factors in addition to the inciting activity. In general, repetitive injury is more likely to occur in the lower extremity, which sees greater loads than the upper extremity in ambulatory athletes, and with activities that are high volume and offer repetitive loading. Running, for example, produces ground reaction forces approximately five times greater than walking. The result of excess strain is an accumulation of microdamage leading to fatigue reaction or fatigue failure. When the area of

fatigue failure is inadequately repaired, it can result in crack initiation in the bone [2] (Fig. 1.2). A simple model is illustrated in Fig. 1.3.

Stress injury may also occur with normal strain, but this is typically in the setting of depressed bone remodeling. These injuries are known as insufficiency reactions or fractures. They are more common in the setting of metabolic diseases, hormonal imbalances, and osteoporosis. In the setting of older persons with osteoporosis, both reduced remodeling and structural changes in the trabecular and cortical bone leading to reduced biomechanical strength, and contribute to the susceptibility to insufficiency fracture at physiologic loads [3]. The dichotomy of fatigue failure and insufficiency is certainly more of a continuum with respect to athletes. These individuals experience greater than physiologic strain through activity but also exhibit risk factors for insufficiency failure, putting some subpopulations of athletes at greater risk.

Another special consideration in the pathophysiology of stress fractures in athletes is the influence of skeletal muscle. Muscles may protect the tibia during running by producing shear forces that counteract the joint reaction forces and result in reduced net shear stresses in the tibia. It has been hypothesized that reduced lower leg muscle strength increases the risk of stress fracture through this mechanism [4, 5], and the concept may extend to other common areas of stress fracture. This theory has only been tested in one clinical study, where a significantly lower knee extension power was observed in a case–control study of female runners with and without stress fractures [6]. Others have hypothesized that this potential protective effect of muscle may be diminished with the fatigue associated with excessive training, or be reduced in new exercisers and military recruits [7].

Finally, there is an oxidation deprivation theory of stress fracture development, which deserves some attention. In this theory, the repeated load of an activity such as running is thought to cause decreased oxygen delivery [8] and brief ischemia [9, 10] in weight-bearing bones. This ischemic environment is thought to stimulate the bone-remodeling process, specifically by increasing osteoclastogenesis [11]. The result is weakened bone that is less able to withstand subsequent loads, thereby increasing susceptibility to further stress-related injury. This theory may explain some observations that those new to activity are more at risk [12, 13].

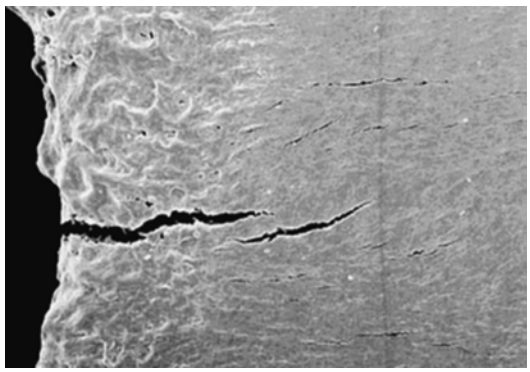

Fig. 1.2 Crack initiation in bone. Reprinted by permission from Macmillan Publishers Ltd: Nature Materials, Nalla RK, Kiney JH, Ritchie O. Mechanistic fracture criteria for the failure of human cortical bone, 2(3). Copyright 2003

Host Risk Factors for the Development of Bone Stress Injury

Bone Mineral Density and Bone Thickness

Although lower bone mineral density (BMD) is likely a stronger etiological factor in insufficiency fracture development, there is evidence that BMD

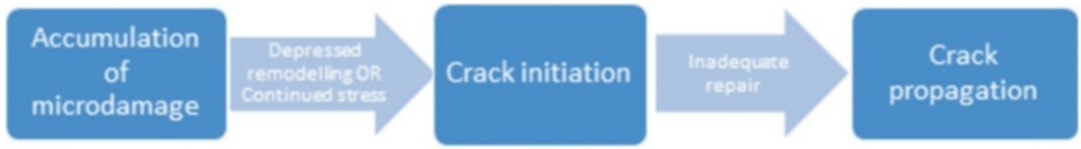

Fig. 1.3 A simple model for the propagation of stress injury in bone

also plays a role in athletes experiencing fatigue failure-related stress fractures. Loud et al. [14] performed a case–control study of female athletes aged 13–22 years who were diagnosed with their first stress fracture. These patients were matched by age and ethnicity to two controls. The authors demonstrated that cases had lower spine BMD for their age, despite no differences in menstrual irregularity or physical activity participation. Similarly, the odds of a stress fracture were three times that for persons with a family member diagnosed with osteoporosis.

Another case–control study [6] of female athletes aged 18–45 years with and without stress fractures noted that after adjusting for body weight, those with stress fractures had thinner tibial cross-sectional area, lower trabecular BMD, and less cortical area of the posterior tibia.

These associations have been confirmed by prospective studies. The first [15] was a 12-month study of both female and male track and field athletes aged 17–26 years. At baseline, females with lower BMD in the spine were at significantly greater risk of developing a stress fracture. A study of military cadets [16] since has demonstrated that smaller tibial cortical area, lower tibial bone mineral content and smaller femoral neck diameter increased the risk of developing a stress fracture in males, and smaller femoral neck diameter was a risk factor in females.

Genetics

There appears to be some genetic susceptibility to stress fracture. Early investigation concluded that ethnicity was a risk factor for the development of stress fracture, with lower rates seen in African-American compared to Caucasian and Asian women. Much of this difference, however, may be related to inherited differences in bone metabolism through bone mineralization. One study has demonstrated an inherited difference in calcium excretion [17].

The association between a family history of osteoporosis in first degree relatives and increased risk of developing a stress fracture among athletes [6] also suggests there is a genetic role in bone turnover as a risk factor.

Nutritional Factors

Dietary and nutritional factors may play a role in the pathophysiology of stress fracture. Calcium and vitamin D are important components of normal bone metabolism and contribute to BMD, with the former being a mineral building block and the latter playing a role in both calcium homeostasis and bone turnover. One randomized trial of female military recruits found a 20 % reduction in fracture injuries with supplementation of 2,000 mg elemental calcium and 800 IU vitamin D compared to no supplementation [18]. Other research has been inconclusive as to whether dietary intake of calcium is important in the development of stress fractures [19, 20].

Other macronutrients may play a role in susceptibility to stress fractures, although the potential pathophysiologic mechanisms are unclear. Merkel et al. [21] demonstrated that among asymptomatic female military recruits, only those females with low iron anemia developed a stress fracture.

Menstrual Irregularity

Late-onset menarche appears to be a risk factor for stress fracture development [15, 16]. It is unclear whether this is due to low peak bone mass attainment, or whether it is a marker of another influence such as excessive training, or low body weight/body fat. The association is further confounded by the fact that under normal circumstances female athletes appear to reach menarche later than their non-athlete counterparts [22].

Disordered menstruation has also been linked to stress fracture risk. Estrogen functions to increase bone mass by inhibiting osteoclastogenesis. It may also function by reducing the adaptation to stress [23]. As such, numerous studies have demonstrated that female athletes who are amenorrheic [19, 24, 25] or oligomenorrheic [19, 20, 26] are at increased risk of stress fracture. Authors have hypothesized about the combined role of menstrual irregularities and low BMD in some female athletes with the so-called “female athlete triad” (disordered eating, amenorrhea, and decreased BMD).

Summary

Bone stress injury occurs via an imbalance of repetitive stress and normal bone remodeling/recovery in response to that stress. Although the paradigms of fatigue failure (high stress overwhelming normal turnover) and insufficiency failure (normal stress overwhelming disordered turnover) are a simple means of conceptualizing this disorder. In reality components of both will contribute to stress injury in any one individual. This is further complicated when one considers that many of the host factors that influence the pathophysiology of bone stress injury are also interrelated. The findings from a study such as that performed by Cosman et al. [16] illustrate that even with the current state of knowledge, we can explain only a small proportion of the risk for stress fracture development. More research is warranted.

From a practical standpoint, the clinician who will diagnose and treat patients with bone stress injuries must understand the basics of bone biology, including stress remodeling. Once a diagnosis has been made, further probing into the potential role of etiologic factors is recommended. This may include diet and nutritional deficiencies, menstrual irregularity, family history, and training volume. Some of these factors may be modifiable and useful in both the treatment of the current stress injury, as well as the prevention of future injury.

Stress Fracture Epidemiology

The epidemiology of stress fractures is described as the occurrence of stress fractures in athletic populations, and is typically expressed on the basis of exposure (e.g., number of stress fractures per athlete-years or per athlete-exposures). One of the challenges in defining the incidence of stress fractures lies in accurately determining the exposure component. Stress fracture cases are comparatively easy to identify, typically through chart records or physician visits. The challenge of a retrospectively designed study is that while it may identify most or all stress fractures over a given time period, accurate information regarding athletic exposure is comparatively lacking. Consistent and accurate injury reporting data is important to identify risk factors, at-risk subpopulations, and monitor the effectiveness of interventions.

A second complicating factor in deciphering the literature defining the occurrence of stress fractures in athletes is the method of diagnosis. Older studies used modalities such as X-ray, which can have poor sensitivity in identifying changes [27]. Many newer studies utilized bone scan or MRI techniques, which offer greater sensitivity and will identify stress fractures at an earlier stage. The MRI is so sensitive that it can detect stress reaction, a precursor to stress fractures, and thus studies utilizing this method of detection will report a greater incidence/occurrence but for a broader spectrum of clinical disorder. Many of these topics are explored in further detail in the remaining chapters of this text.

This heterogeneity in diagnosis, study design, and accuracy of exposure precludes the pooling of data to formulate incidence rates by sport or activity, at the current time. Therefore, this chapter will focus on a descriptive review of the literature, the most robust of which originates from military populations. Studies from various sports will also be reviewed and interpreted. A preference towards higher level of evidence studies published in the last 10 years is given.

Stress Fracture Epidemiology: Military

Military populations are a unique group that facilitates epidemiological research on stress fractures. Patient follow-up and activity exposure can be well controlled and documented, which allows for more homogeneous comparisons and higher level of evidence designs such as prospective cohorts. Additionally, large numbers of patients can be recruited for study, which is helpful when investigating a condition that typically occurs infrequently or when performing multivariate analyses to identify risk factors.

Most important, however, is that military personnel appear to have a higher incidence of stress fractures than the general population due to the suddenly increased and extensive exercise associated with training. Accordingly, military studies on stress fractures have been performed all over the world, including the USA [16, 24, 27–29], Finland [30], and Israel [31].

A common theme in this population is a higher reported occurrence or incidence of stress fractures among females compared to males. In one study of cadets, 19.1 % of females and 5.7 % of males reported at least one stress fracture [16]. Similarly, in the largest studies of US Army recruits [28], the incidence of stress fractures was 79.9/1,000 female and 19.3/1,000 male recruits. This pattern holds true internationally. An Israeli military study [31] identified a similar discrepancy (ratio 2.13) of bone scan positive stress fractures in females (23.9 %) to males (11.2 %). A similar pattern was seen among a prospective cohort of 152,095 Finnish conscripts [30], where the ratio of female to male bone stress injury on MRI was 9:2. The overall incidence rate of stress fractures in this population was 311/100,000 person-years (95 % confidence interval: 277–345).

There also appears to be a difference in the distribution of stress fracture location between male and female military personnel. Compared to males, females have higher reported rates of stress fracture for the pelvis [30, 31], sacrum [30], and tibia [30, 31].

These sex differences have prompted many researchers to specifically study female recruits. Shaffer et al. [24] identified a stress fracture rate of 5.1 % in a cohort (N=2,962) of female US marine recruits. All stress fractures occurred in the lower extremity, most commonly in the tibia, followed by the metatarsal bones, pelvis and femur. In regression analysis the odds of developing a stress fracture were more than five times higher among recruits who were amenorrheic during the prior year (odds ratio 5.64, 95 % confidence interval 2.8–25.8). Lower aerobic performance on a timed run also increased the odds of developing stress fractures in the pelvis and femur.

In a separate study of female US Marine Corps recruits [25], the same authors reported on all overuse injuries of the lower extremity [24]. They determined an incidence rate of lower extremity stress fractures of 1.0/1,000 days of training exposure. Having multiple overuse injuries was common, and in multivariate regression analysis, again lower aerobic fitness and amenorrhea predicted increased odds of stress fracture.

Among lower extremity stress fractures in military populations, the tibia and metatarsals appear most common [16, 29]. A rarer occurrence is the calcaneal stress fracture. One study identified calcaneal stress fractures from MRI in recruits who had undergone ankle MRI for exercise induced heel or ankle pain [27]. The incidence rate of stress fractures among all recruits during the study period was 2.6/10,000 person-years (95 % confidence interval 1.6–3.4). Most calcaneal stress fractures were found in the posterior aspect of the bone, and 22/34 (65 %) were associated with stress fractures in other tarsal bones. A comparison to plain radiographs in the same patients revealed only 15 % had abnormal films, attesting to the higher sensitivity and ability to detect stress changes at an earlier stage by MRI.

Stress Fracture Epidemiology: Running

Runners are at higher risk of developing stress fractures. In many cases, however, athletes may compete in multiple sports, and attributing stress fractures solely to running can be challenging. A survey study of 1,505 runners performed in 1990 [32] identified female long-distance runners at highest risk for stress fracture.

Since that survey, two prospective cohort studies have attempted to better define the epidemiology of stress fractures in runners. One study of 748 competitive high school cross-country and track and field runners identified a 5.4 % and 4.0 % rate of stress fractures in girls and boys, respectively [33]. The tibia and metatarsal bones were among the most commonly affected. Multivariate models identified late menarche, low BMI and a prior history of stress fracture as significant contributors to increased risk of new onset stress fracture. In a second, smaller cohort study [34] of competitive high school runners followed for 3 years, stress fractures were identified in 21/230

Table 1.1 Stress fracture epidemiology by miscellaneous sports

Reference	Sport	Study design	*N*	Incidence	Notes
Pearce et al. [40]	Rugby	Prospective cohort	12/899 (8 %)	–	Navicular SF associated with longest time away
Ekstrand et al. [41]	Football	Prospective cohort	51/2379	0.04/1,000 h	78 % fifth metatarsal; 29 % re-injury; 3–5 months absence
McCarthy et al. [42]	Women's basketball	Case series	506 (7.3 %)	–	WNBA player injury reports at draft
Frost et al. [43]	Cricket	Prospective cohort	248	51.6/10,000 player-h	Professional; SF to low back had longest return to play
Ekegren et al. [44]	Ballet	Prospective cohort	266	Not stated	SF had longest return to participation

(9.1 %) athletes, representing an incidence of 0.06 stress fractures per athlete exposure.

Stress Fracture Epidemiology: Tennis

The nature of tennis lends the potential for stress fracture development in both the racket hand and lower extremity from running and sudden stops. Abrams et al. [35] reviewed the literature for case reports on uncommon stress fracture locations in tennis players, and identified them in the ischium, first rib, humerus, sacrum, patella, hook of hamate, ulna, and distal radius. Another study [36] examined a case series of high level junior tennis players, noting seven cases of second metatarsal stress fractures postulated to be related to racket grip.

The largest tennis study followed 139 elite tennis players of a median age 20 years, 65 % male and 57 % professional status over the course of 2 years [37]. In total, 15 players had 18 stress fractures for a rate of 12.9 %. The most common location was the navicular (5/18), pars interarticularis (3/18), metatarsals (2/18), tibia (2/18) and lunate (2/18). There were also more stress fractures among juniors (20.3 %) compared to professionals (7.5 %). Unfortunately, none of these studies provided a metric for exposure to calculate an incidence rate.

Stress Fracture Epidemiology: Pediatric/Adolescent Athletes

Particular attention has been directed towards pediatric/adolescents with respect to describing stress fractures. This is an important subpopulation due to potentially open physes and associated metabolic changes that accompany menarche. A national survey study of adolescent girls [38] has followed 6,831 girls aged 9–15 years for 7 years. Among them, 267 (3.9 %) developed a stress fracture. Multivariate modeling demonstrated that running, basketball, cheerleading, and gymnastics were all significant predictors of developing a stress fracture.

In a retrospective case series of pediatric athletes with open physes, Niemeyer et al. [39] followed 19 children with 21 stress fractures over a mean 4.8 years. The mean age at diagnosis was 14 years, and most fractures were found in the lower extremity. They noted tibial stress fractures were more likely to accompany sports with sudden stops, and were also associated with a longer course of treatment.

Stress Fracture Epidemiology: Other Sports

Individual case reports and series have been published, documenting the occurrence and incidence of stress fractures in various sports. These are reviewed in Table 1.1.

Summary

The reported incidence and occurrence of stress fractures in the literature is variable. The most robust data from the military suggests that new activity (i.e., recruits) and females have the highest

incidence of stress injury. Among athletes, the pattern of injury and incidence/occurrence varies by sport and level of competition.

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General Treatment Concepts for Stress Fractures

2

Tianyi Wang, Gordon Matheson, and Marc R. Safran

Basic Principles

Stress fractures result when the rate of osteoblastic new bone formation is unable to keep pace with the rate of osteoclastic bone resorption after microdamages from repetitive stresses to the musculoskeletal system. These injuries typically occur in the setting of athletic overuse—either absolute or relative to the individual's baseline conditioning. Stress fractures fall along a spectrum of bone injury, ranging from normal bone remodeling in response to physiologic forces on bone to acute fracture when the demands outweigh the mechanical strength of bone. Stress fractures lie between these two extremes, where the body's reparative abilities are less than the accumulation of loads placed on the bone. General treatment goals in the management of stress fractures manipulate this balance of bone homeostasis, either by decreasing activity levels while the bone heals and/or augmenting the body's fracture-healing capacities (Fig. 2.1). The cornerstone of treatment relies on activity modification and rest to halt progressive damage while the body works to heal the bone.

Timing to allow an athlete to return to play is among the most challenging decisions for practitioners. Factors to take into account include duration and progression of symptoms, location of injury and its propensity for fracture completion, risks of nonunion or consequences of complete fracture, timing in season of sport, as well as proposed treatment plan and rehabilitation. Overtreatment of low-risk stress fractures may lead to athlete deconditioning and need for increased duration of rehabilitation. Conversely, some high-risk fracture patterns have significant risk of nonunion or progression to complete fracture and may be more amenable to surgical fixation in order to allow faster union and return to activity [1]. Nonoperative treatment protocols for lower extremity stress fractures may range from non-weight bearing with crutches, allowing aerobic cross-training without impact activities, or avoidance of only pain-provoking activities. With careful attention, a significant proportion of stress fractures that arise in athletes may be successfully treated with aggressive nonoperative management and the patient and practitioner may be able to avoid unnecessary surgical intervention and risk.

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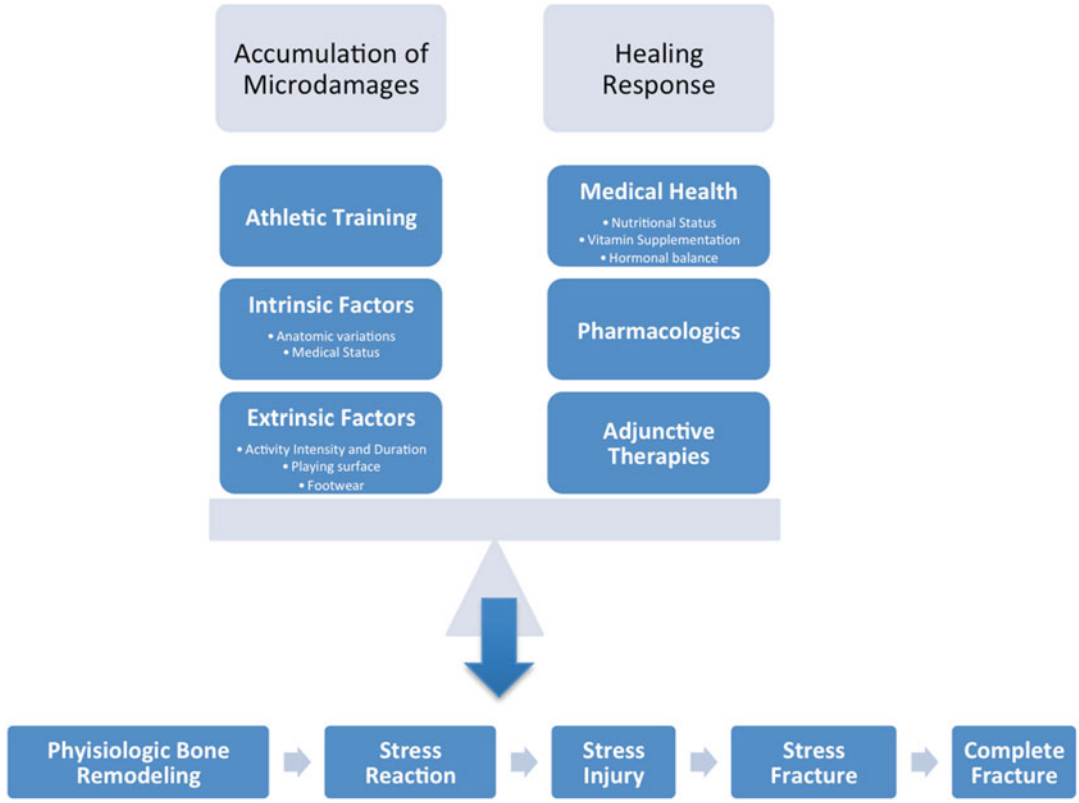

Fig. 2.1 Diagram of bone metabolism remodeling in response to stress. *Top*: Balance between factors that contribute to microdamage accumulation and variables that affect the body's natural healing response. *Bottom*: Spectrum of bone remodeling. As rate of demands outweight the body's healing response, further damages increase risk of stress injury and complete fracture

Initial Evaluation

Once a stress fracture has been identified in an athlete, it is prudent for the practitioner to identify possible contributing factors for why the injury occurred, both to allow the fracture to heal as well as to prevent recurrence. These risk factors may be divided into extrinsic or intrinsic variables. Extrinsic factors are defined by the training environment and include training duration and intensity, exercise surface and footwear, and nutritional status. Intrinsic factors are specific to the patient and include anatomic variations (femoral version, leg length discrepancies, genu valgum/varum, pes planus/cavus), gender, smoking status, medical and endocrinologic history, and medications [2].

It is well known and understood that stress fractures are associated with large increases in intensity and duration of athletic participation or practice over a short period of time. Thus, a history of training and play is critical in the evaluation of the athlete with a stress fracture. The majority of studies evaluating stress fractures stem from individuals participating in military boot camp—likely due to the marked increase in duration and intensity of training during the initiation of military duty. Additionally, an informal study performed in 1999 on college athletes at an NCAA Division 1 program found that 50 % of stress fractures occurred in freshman athletes. This is also likely due to the significant increase in the duration and intensity of training at the collegiate level, as compared to the high school level.

In conjunction with treatment, a diagnostic evaluation is recommended to rule out any metabolic pathology that would increase an athlete's risk for developing a fracture or delay healing [3]. A careful history and physical examination is necessary to evaluate for eating disorders, smoking and alcohol abuse, hormonal imbalance, sexual dysfunction, thyroid disorder, and menstrual irregularities. Laboratory work-up includes urinalysis, complete metabolic panel with analysis of serum calcium, albumin, alkaline phosphatase, and serum vitamin D levels [3]. DEXA (dual-energy X-ray absorptiometry) scan may be considered in patients with multiple or recurrent stress injuries.

Of special consideration in female patients is the "Female Athletic Triad," traditionally defined as the inter-related combination of eating disorders, amenorrhea, and osteoporosis [4]. However, a new description in 2014 by the Female Athlete Triad Coalition describes the constellation of "Low Energy Availability" with or without disordered eating, menstrual dysfunction, and low bone mineral density as another definition that places female athletes at risk for amenorrhea, osteoporosis, and stress fractures [4, 5]. Circulating estrogens have been shown to increase bone marrow density through decreased calcium resorption by osteoclasts. Patients with Female Athletic Triad and amenorrhea are thought to subsist in a lower estrogen state with resultant osteopenia and increased risk for stress fractures. Treatment focuses on adjusting caloric intake and reducing activity to support an overall anabolic metabolic rate [4]. If the Female Athlete Triad with amenorrhea is suspected, treatment should be initiated consisting of evaluation by a multidisciplinary team including physicians, sports dietitians, and mental health professionals.

Lloyd et al. retrospectively reviewed the relationship of menstrual status and stress fractures in collegiate women athletes. The authors found the frequency of stress fractures in women with irregular menses (15 %) was nearly four times that of female athletes who had regular menses (4 %) [6]. Similarly, Barrow et al. stratified the prevalence of stress fractures in competitive collegiate long distance runners according to regularity of menstrual cycles. There was an inverse relationship with menstrual consistency and injury; stress fractures occurred in 29 % of athletes who had 10–13 menses/per year, 39 % of athletes who had 6–9 menses/year, and 49 % of athletes who had 0–5 menses/year [7]. Additionally, 47 % of those who had less than 5 menses per year admitted to an eating behavior disorder.

In order to further evaluate the contributions of hormonal therapy, Cobb et al. randomized 150 female runners between 18 and 26 years of age to treatment with oral contraceptive (30 mcg of ethinyl estradiol and 0.3 mg of norgestrel) or no treatment [8]. After 2 years, the authors found a nonsignificant trend towards decreased stress fracture incidence with oral contraceptive use; the authors also did report a high noncompliance rate in the treatment group. Further studies are needed to elucidate the interaction between hormonal status and stress fractures incidence. Certainly in the female athlete, identifying those at risk for Female Athletic Triad/Low Energy Availability is crucial and may prevent further progression of serious health consequences.

In addition to hormonal factors, lifestyle choices may place athletes at risk for further injury. A study of female military recruits entering basic training found those who reported alcohol intake of more than ten drinks per week, smoking history, and exercising fewer than three times per week prior to start of basic training had increased risks of stress fractures [9].

Extrinsic Factors

One population that has been studied extensively in evaluation of both risk factors and treatment efficacy of stress fractures are military personnel. These typically are relatively young patients subjected to standardized rigorous activity requirements in multiple intervals per year, often with sudden increase in activity level from baseline. This demographic of patients is at high risk for stress fractures, with some reports of up to 31 % incidence and the majority occurring in either the femoral or tibial shaft [10]. Milgrom et al., in a study of Israeli military recruits, randomized 390

recruits to 14 weeks of basic training in either standard infantry boots or high-top basketball shoes to evaluate the role of shoe design and cushioning. The authors found lower incidence of metatarsal stress fractures and foot overuse injuries (metatarsalgia and arch or heel pain) in those using basketball shoes but no difference in overall overuse injuries. Additionally, no differences in the incidence of tibial or femoral stress fractures were identified [11]. The same group also evaluated the effect of a cushioning shoe insert in a prospective study of military recruits treated with or without shock-absorbing orthotic worn in military boots [12]. There was a significant decrease in femoral stress fractures with the use of orthotics but this was not seen in metatarsal or tibial stress fractures. No differences were found in time to onset or location of stress fractures. With this data, Milgrom et al. randomized 404 recruits to three groups: group 1 was given custom semirigid orthotics with army boot, group 2 was given soft orthotics with army boot, and group 3 received only army boots [13]. All military boots used in this study had soles designed like basketball shoes. After 14 weeks of basic training, the femoral and tibial stress fracture rate was 15.7 % for those in semirigid group, 10.7 % for soft orthotic group, and 27 % for the control group. When comparing the patients who received any orthotic (semirigid or soft) to those without, the authors found a significant decrease in total stress fracture rate with orthotic use. No differences in stress fracture rate were identified between the two types of orthotics. However, the analysis was limited by dropout rate of nearly 50 %, as many recruits preferred their own custom orthotics that they were using before the military. The authors recommended the use of orthotic inserts in the military population for prophylaxis of stress fractures. These findings have been corroborated in other clinical series of military recruits, with either orthotic use or modified shoe type reducing stress fracture incidence [14–17].

In addition to footwear characteristics, training surface has also been thought to play a role in the development of stress fractures. Current studies, though, have not demonstrated an association between type of training surface and incidence of stress fractures. Only limited data exists to associate running on concrete surfaces with lower extremity injuries [18]. Evidence is poor due to methodology and sampling bias, but some investigators recommend considering minimizing time on hard, uneven surfaces based upon intuitive reasoning [18–22].

Swissa et al. evaluated the role of pretraining in reducing the risk of stress fractures in military recruits. Their hypothesis was that recruits with increased levels of impact activity prior to basic training had decreased stress fractures. The authors prospectively evaluated 295 recruits and found 78 % were involved in some sort of sport activity prior to basic activity. No correlation was found between those who participated in pretraining sport activity and incidence of stress fractures, averaging 31 % for trained and untrained groups [23].

Intrinsic Variables

Individual anatomic variables have also been investigated as possible risk factors for stress fractures and may serve as potential targets for prevention. Milgrom et al. prospectively evaluated the role of midfoot position and stress fractures and found increased prevalence of femoral and tibial stress fractures in military recruits with pes cavus. However, metatarsal stress fractures were more frequently seen in those with pes planus [14]. The authors suggest that a high arch is a more rigid position and has decreased ability to absorb shock, as opposed to a more flexible low-arched foot [19]. Other studies have confirmed the theory that rigid pes cavus is more common in patients with femoral stress fractures. Additionally, excessive foot pronation position was more commonly seen in patients with stress fractures of tibia and fibula, thought to be related to increased tibial torsion during the support phase of running [24].

Leg length discrepancy has also been associated with increased risk of stress fractures [22]. Surveys of distance runners revealed leg length discrepancies are a major contributing factor to running injuries [22]. The longer leg was associated more

frequently with tibial, metatarsal, and femur fractures. Incidence of stress fractures has been shown to increase with increasing magnitude of leg length discrepancy [19, 25]. Bennell et al. prospectively evaluated 53 female athletes and found leg length discrepancy of more than 0.5 cm was seen more frequently in those who sustained stress fractures (70 % vs. 36 %). Fractures occurred with similar frequency in the longer and shorter limb [26]. The authors also noted athletes who had decreased calf muscle mass had higher rates of stress fractures, suggesting calf muscles strength may protect bone from repetitive excessive force.

Treatment Modalities

Activity Modification

Often the first step in the management of stress fractures includes adjusting an athlete's activity below a threshold that the body's reparative process can keep up with. Patient reported pain is useful as a proxy for "too much" activity. However, the maintenance of cardiovascular fitness is crucial for an athlete and may be preserved through aerobic activities that limit weight bearing and impact, including cycling (with or with an immobilizing device), upper extremity cycling (ergometry) in the presence of a lower extremity stress fracture, pool exercises, or weight unloading training in harnesses or decreased gravity treadmills (Fig. 2.2). Some reports found distance runners have improved stamina after cross-training in a pool as opposed to other low-impact modalities [27]. It may take up to 6–8 weeks for healing of stress fractures to occur [28].

Matheson et al. reported on a series of 320 patients with stress fractures identified by radiographs and bone scan [24]. The athletes were initially treated with nonsteroidal anti-inflammatories (NSAIDs), symptom control, and activity modification. Athletes were allowed normal weight bearing during day-to-day activities with the avoidance of pain-provoking maneuvers. Cycling, swimming, and running in water were allowed as low-impact alternatives. After athletes were pain free for 10–14 days, gradual reintroduction of sport was allowed. No patients treated in the series progressed to complete fracture and the majority of athletes were able to return to activity with excellent results. The authors concluded the effectiveness of conservative treatment for the treatment of most stress fractures.

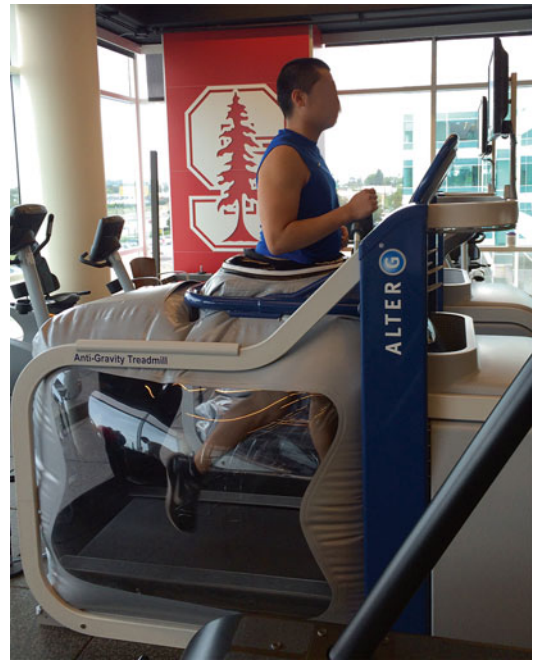

Fig. 2.2 Gravity-reduction treadmill

External bracing of the lower extremity has also been evaluated as a treatment adjunct for the management of stress fractures. Dale et al. described the contribution of pneumatic braces on fractures in a canine model [29]. Animals were treated with tibial osteotomy and external fixator placement and allowed unrestricted weight bearing for 48 days to simulate partial healing. External fixators were then removed and the subjects were divided into groups treated with either standard cast or pneumatic brace. There was significantly greater torque to failure, energy absorbed, and angle of rotation for those treated with pressurized brace. Histologic studies also demonstrated increased periosteal bone formation and greater bone density at fracture site in treatment group as compared to standard group. Human studies have similarly found a positive benefit to the use of such braces. Swensen et al.

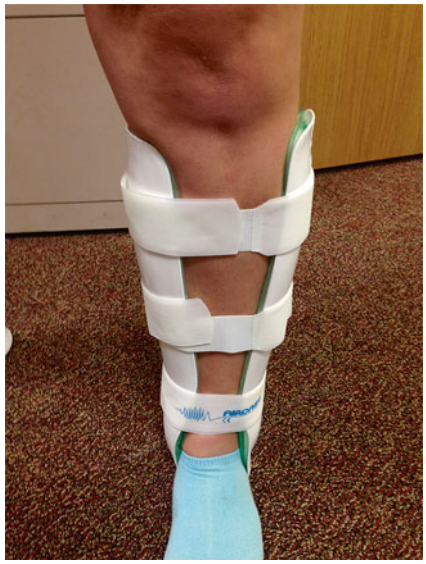

Fig. 2.3 Pneumatic compression ankle brace

randomized 18 athletes with tibial shaft stress fracture to examine recovery after use of pneumatic leg brace. The control group was treated with rest and allowed return to activity once had they had three consecutive pain-free days. The treatment group had pneumatic leg brace applied and followed the same return to play guidelines. Athletes receiving the brace demonstrated earlier return to light activity and accelerated return to full activity (21 vs. 77 days) [30]. The authors proposed that compressing the soft tissue may increase intravascular hydrostatic pressure and produce downstream effects that stimulate osteoblast bone formation.

A recent Cochrane review evaluated the effect of pneumatic braces for stress fractures and showed a significant reduction in time before returning to full activity (Fig. 2.3) [31]. When evaluating trials in aggregate, the mean acceleration of healing measured approximately 33 days when the braces where used.

Pharmacologic Therapies

A variety of pharmacologics have been used in order to both treat and prevent stress fractures [2]. Much of the literature stems originally from the treatment of acute fractures with further studies that have been focused on stress fractures and its applicability to the athletic population [32].

Vitamins

Calcium and vitamin D have long been understood as essential vitamins in the maintenance of bone health. Calcium is a significant component in the inorganic substrate of bone. Vitamin D is obtained from diet, sunlight, or supplements and facilitates intestinal absorption of calcium [33]. Vitamin D is metabolized into 1,25-dihydroxyvitamin D, which serves as an active metabolite to enhance intestinal mucosal calcium absorption via active calcium transporters in the mucosal epithelium [34]. In the elderly, oral vitamin D has been shown to reduce risks of insufficiency fractures in randomized trials [35]. In the setting of stress fractures, the addition of oral calcium and vitamin D are advocated if laboratory values are low [3]. Recommended daily allowances are 1,000 mg of calcium and 800–1,000 IU of vitamin D. However, some authors recommended higher doses for athletes with stress fractures, up to 1,500 mg calcium and 3,000 IU of vitamin D [3, 34].

Nieves et al. followed 125 female distance runners between 18 and 26 years of age and found stress fracture incidence was reduced with increased baseline intake of dietary calcium and dairy products. The effective fracture risk decreased by 62 % with each additional cup of skim milk per day. Women who consumed less than 800 mg of calcium per day had six times the stress fracture rate than those who consumed more than 1,500 mg per day [36, 37]. Lappe et al. randomized 5,201 female Navy recruits to either treatment with 2,000 mg calcium and 800 IU vitamin D or placebo. A total of 309 stress fractures were diagnosed (5.9 %) over an 8-week period. Recruits who were treated with supplementation had a 20 % lower incidence of stress fractures (6.6 % vs. 5.3 % in intent-to-treat analysis, 8.6 % vs. 6.8 % in as-treated analysis). The authors stated that if the entire population of 14,416 women who entered basic training in

the 2-year recruitment period were treated with vitamin supplementation, 187 stress fractures would have been prevented [37, 38].

NSAIDs

NSAIDs are commonly used medications to treat musculoskeletal disease. Their therapeutic effects are achieved via inhibition of cyclooxygenases (COX), enzymes that participate in the pro-inflammatory response. NSAIDs ultimately reduce the production of prostaglandins, whose downstream effects include pain, swelling, and other inflammatory symptoms manifested through histamine and bradykinin release [39, 40]. During the cascade of fracture healing, COX enzymes are active at injury sites to produce prostaglandins. These chemicals are released by both local osteoblasts and osteoclasts to serve as potent stimulators of bone turnover. Bone formation and resorption, as well as local angiogenesis, support cell metabolic activity along the pathways of endochondral and intramembranous ossification [41]. There is concern, though, that use of NSAIDs may inhibit fracture healing and increase nonunion rates.

Wheeler et al. reviewed the interactions of NSAIDs on bone healing. Multiple animal studies have shown NSAIDs (including ibuprofen and indomethacin) may retard the rate of fracture healing, decrease fracture callus strength, and may lead to increased nonunion rates [42]. Other theories surmise NSAIDs may inhibit differentiation of mesenchymal stem cells into chondrocytes and osteoblasts [42]. Kidd et al. evaluated the effect of NSAIDs on stress fractures in an animal model. Stress fractures were created via cyclic loading of rat ulnas. The animals were then treated with ibuprofen 30 mg/kg/day or control and examined at 2, 4, and 6 weeks. Use of ibuprofen decreased both resorption and bone formation after 6 weeks when measured via histomorphometry [43].

Despite the detrimental consequences of NSAIDs on fracture healing in animal studies, human studies have not clearly demonstrated the same effects. The majority of clinical studies are retrospective with assorted results, finding either small association or no difference with NSAID use [42]. Only two randomized studies exist evaluating the role of NSAIDs in fracture healing. Adolphson et al. randomized 42 patients with distal radius fractures to either treatment with piroxicam 20 mg/day for 8 weeks or placebo. All patients were treated nonoperatively with dorsal plaster splints for 4 weeks. At final 8-week follow-up, no difference in bone-mineral density, rate of fracture healing, radiographic findings, or functional recovery was found between the two groups. Patients receiving piroxicam did demonstrate decreased pain scores in the early follow-up paper. However, six patients, all in the piroxicam group, had loss of reduction and required external fixation for stability and were excluded from final analysis [44]. Burd et al. evaluated the role of NSAIDs in 282 patients who had open reduction and internal fixation of an acetabular fracture [45]. Patients at risk of heterotopic ossification were randomized to receive either radiation or indomethacin (25 mg three times a day for 6 weeks) for prophylaxis. The authors found that patients who received indomethacin had increased rates of nonunion for associated long bone fracture (26 % vs. 7 %). However, the two groups were randomized according to their acetabular surgery and differed in injury severity scores and rates of open fracture. Concerns with randomization protocol, data collection outcomes, and methodological deficiencies exist in current clinical studies and limit the ability to draw conclusions regarding therapeutic effect of NSAIDs on fracture healing. Currently, there are no randomized studies of NSAIDs in the healing of stress fractures, and extrapolation of results from acute traumatic fractures may not be fully applicable given differing clinical scenarios. Further large, prospective studies with careful attention to methodology are required to better define the role of NSAIDs and eventually guide physician decision making.

Bisphosphonates

Bisphosphonates have been used to treat many different bone diseases caused by excessive

osteoclast activity. Its mechanism of action is through suppression of bone resorption through suppression of osteoclast activity, accomplished via inhibiting farnesyl pyrophosphate synthase. Isoprenylation of GTP-ases is interrupted to interfere with the attachment of osteoclasts to the bone surface and promotion of osteoclast cell apoptosis [46]. Significant evidence for bisphosphonates has been published in the literature regarding its ability to prevent of postmenopausal fragility fractures. Large randomized trials demonstrate decreased incidence of new vertebral compression fractures and hip fractures in elderly females prescribed bisphosphonate therapy [46–49].

The role of bisphosphonates in bony healing in the setting of acute fractures has been investigated as well. Adolphson et al. performed a randomized, double-blinded study in postmenopausal females with distal radius fractures. Patients either received 500 mg of clodronate twice a day for 8 weeks or placebo. At 2-month follow-up, those treated with clodronate had a greater ratio of bone-mineral density at the fracture site when compared to contralateral side (53 % vs. 33 %). At 12-month follow-up, effective increased bone density at the fracture site was still maintained. There were no differences in pain or clinical function scores at 3-month follow-up [50].

Despite these findings, convincing clinical improvements for the use of bisphosphonates in stress fractures have not yet been clearly borne out in the literature. Stewart et al. treated five collegiate female athletes 18–24 years old with tibial stress fractures with pamidronate, a second generation bisphosphonate [51]. Patients received intravenous infusions of pamidronate, comprised of five doses of 60–90 mg administered on a weekly basis. Four of five patients were able to achieve primary outcomes of ability to continue training without pain. All five athletes reported improvement in pain after the first dose. No improvement in outcome was found between the 60- and 90-mg dosing, with 90 mg having a higher rate of nausea as an unintended side effect. Though results are encouraging, further prospective studies with comparison groups are needed.

Milgrom et al. randomized 324 military recruits to treatment with risedronate or placebo in order to evaluate stress fracture prophylaxis. Patients in the treatment arm were given 30-mg oral dose of risedronate for 10 days as a loading dose during the first 2 weeks of basic training. They then continued 30 mg by mouth every week for the next 12 weeks. The authors found no differences in stress fracture incidence between groups using either intention-to-treat analysis or an as-treated comparison. Total incidence of stress fractures was 14.5 % in the risedronate group and 13.2 % in the placebo group. No significant differences in side effects were found between the two groups [52].

Use of bisphosphonate treatment is not without risk. Recent evidence associates long-term bisphosphonate use with atypical femur fractures. These typically occur in the subtrochanteric or diaphyseal femur after low-energy trauma, thought to be due abnormal bone remodeling due to osteoclast inhibition, resulting in accumulation of microfractures [53]. Bisphosphonates have been shown to decrease bone toughness in animal studies, resulting in increased brittleness and decreased ability to absorb energy prior to fracture [54, 55]. Canine studies have also shown that bisphosphonates, by suppressing the body's natural bone remodeling process, leads to the accumulation of microdamages in bone [55–58]. Caution should especially be noted with use of bisphosphonates in young women of childbearing age. Animal studies have shown bisphosphonates to cross the placenta and decrease fetal weight, affect bone growth, and may be associated with increased neonatal deaths. As bisphosphonates are stored in bone and slowly released, they may have circulating half-life of over ten years [59]. Risks for nonunion must be carefully weighed against future teratogenic complications; many authors suggest withholding bisphosphonate treatment in young females.

Forteo (Teriparatide)

Teriparatide is a synthetic parathyroid hormone (PTH) approved in 2002 for treatment of osteoporosis. PTH functions to maintain calcium and phosphate homeostasis via increasing tubular

resorption in the kidneys, increasing renal production 1,25-dihydroxyvitamin D, and by mobilizing calcium from bone stores [60]. Though originally understood to conserve calcium in the body, PTH has more recently been discovered to have anabolic effects for bone and have receptors located on osteoblasts. Animal studies demonstrated administration of PTH in a continuous infusion resulted in decreased body weight, hypercalcemia, and peritrabecular marrow fibrosis and resorption. However, administration in a pulsatile manner increased anabolic effects and ultimately results in osteoblast proliferation and bone formation [61]. Other animal studies demonstrate PTH role in the activation of resting osteoblasts and stimulating osteoprogenitor cells [32].

Teriparatide was initially approved after randomized trials in osteoporotic women revealed it increased bone marrow density by up to 13 % and decreased risk of vertebral and nonvertebral fractures by at least 50 % to 72 % [62]. Neer et al. published a randomized, blinded trial treating 1,637 postmenopausal women with low-dose PTH (20 mcg administered subcutaneously daily), high-dose PTH (40 mcg administered subcutaneously daily), or placebo. Patients in both treatment groups had decreased rates of new vertebral fractures, nonvertebral fragility fractures, and increased bone marrow density [62]. The medication was well tolerated by patients with only minor side effects of nausea and headache.

The use of synthetic PTH has been evaluated for the treatment of acute fractures as well. Komatsubara et al. evaluated femoral osteotomies performed in a rat model and treated subjects with various dosing regimens of PTH or control [63]. Treatment was administered via subcutaneous injections three times a week for 3 weeks. The authors found treatment at 30 mcg/kg administered both before and after osteotomy increased the mechanical strength of callus through faster remodeling of woven bone to lamellar bone, increased cortical shell formation, and stronger ultimate load to failure at 12-week follow-up. Accelerated appearance of the cortical shell was likely the main contributor of increased mechanical strength. The same group also investigated PTH in a monkey model treated with femoral osteotomy and stabilized with plate fixation [64]. Subjects were randomized to control, low-dose or high-dose PTH via twice a week subcutaneous injection for 3 weeks. At 6-month follow-up, the authors found acceleration of the body's natural healing process in a dose-dependent manner. Increased ultimate stress and elastic modulus was seen for the high-dose treatment group. The authors also noted decreased callus area and increased callus mineralization, resulting in stronger intrinsic material properties such as ultimate stress, elastic modulus, and toughness. No differences were found in ultimate load, stiffness, load to failure. Andreassen et al. found PTH increased callus formation, strength, mineralization, and mechanical strength in rat tibia fracture model [65, 66]. Other animal studies have demonstrated similar findings of accelerated rate of normal fracture healing, resulting in increased callus strength at comparable time points [67].

Peichl et al. performed one of the first human clinical trials evaluating the effect of PTH in acute fractures and demonstrated accelerated fracture healing [68]. Sixty-five postmenopausal female patients who sustained unilateral fractures of the pubic bone or ischial rami with T-score less than −2.5 were included. Patients were randomized to treatment with either placebo or daily injections of 100 mcg of PTH. All patients received calcium and vitamin D supplementation. Patients were evaluated with CT scans every fourth week for evaluation of bony bridging. The authors found significantly accelerated healing with earlier bony bridging in the treatment group (7.8 vs. 12.6 weeks). After 8 weeks from injury, 100 % of fractures in the treatment groups had healed, as opposed to 9.1 % in control group. Patients allocated to the treatment group also demonstrated improved clinical scores as assessed by VAS pain scores and timed "up and go" test (22.9 vs. 54.3 s). No laboratory abnormalities were noted with treatment. Aspenberg et al. randomized postmenopausal women with dorsally angulated distal radius fractures treated with closed reduction and immobilization to either 8 weeks of daily injections of placebo or

teriparatide at 20 or 40 mcg. The authors found shorter time to radiographic healing in the low-dose group but no differences in the high-dose group. There were no differences in fracture displacement or clinical scoring outcomes [69]. The majority of clinical studies evaluating the effect of teriparatide are performed in elderly, osteoporotic populations. No studies are yet published describing the use of PTH in the treatment of stress fractures; it is not yet known whether these effects can be extrapolated to a younger, more active patient population. However, the senior author has been using teriparatide in the management of stress fractures in high-level athletes with anecdotal results of earlier return to play.

Adjunctive Therapies

Low-Intensity Pulsed Ultrasound

Ultrasound functions as propagating pressure waves arising via vibration from a source material, which transfers mechanical pressure to recipient tissue. Though the exact mechanism of action has not been fully elucidated, many studies suggest these waves accelerate the endochondral ossification process through afferent mechanoreceptors and alter cell signaling. Potential changes in blood flow patterns may occur as well [70]. The clinically effective ultrasound signal approved by the United States Food and Drug Administration consists of a 1.5-MHz ultrasound wave pulsed at 20 % duty cycle at an intensity of 30 mW/cm^2 [71].

Wolff's law dictates that bone remodels according to mechanical forces within its environment. Animal studies have demonstrated the effect of ultrasound waves as an external, mechanical stimulus to bone growth. Yang et al. used a rat femur-fracture model and found greater maximum torque and torsional stiffness at 3 weeks after injury in subjects treated with pulsed ultrasound [72]. Biochemical analysis demonstrated increased expression of genes associated with cartilage formation, but no differences in cell number or mineral content. The authors suggested this may due to accelerated chondrogenesis and endochondral ossification. Similar findings have been seen in other animal studies, where groups receiving ultrasound therapy demonstrated increased torque to failure and stiffness [73].

Kristiansen et al. performed a prospective multicenter trial that randomized 60 patients with dorsally angulated distal radius fractures treated nonoperatively with either an ultrasound device or placebo for 20 min per day for 10 weeks [70]. The authors found faster time to union by 37 days and accelerated radiographic signs of healing, as well as decreased loss of reduction in patients receiving ultrasound device. Though no adverse reactions or side effects were reported with use of the device, no relevant clinical outcomes were reported. Heckman et al. reported accelerated healing in tibial diaphyseal fracture with the use of a pulsed ultrasound device in a prospective blinded multicenter trial [74]. Sixty-seven patients with tibial shaft fractures were randomized to cast treatment with or without external ultrasound stimulator. Treatment was started within 1 week of injury and used for 20 min per day for 20 weeks or until the fracture healed; placebo groups were given a non-functioning machine. The authors found significant decrease in time to clinical healing (86 vs. 114 days), decreased time in cast (94 vs. 120 days) and decrease in time to overall (clinical plus radiographic) healing (86 vs. 154 days). Patients reported very high compliance with the device. Low-intensity ultrasound also accelerated healing response in smokers and reduced the incidence of delayed unions [75, 76].

Rue et al. randomized 26 midshipmen with tibial stress fractures in a double-blind study to pulsed ultrasound or placebo device. Patients received 20-min daily treatments until they were asymptomatic or signs of healing were noted on plain radiographs. The authors found no differences in total number of treatments needed, days needed to return to duty, or total days of symptoms [77]. The average treatment duration was 24–26 sessions.

A systematic review analyzed 13 randomized trials studying the use of low-intensity pulsed ultrasound in fractures. Only five studies assessed

clinically relevant outcomes, with one trial demonstrating a positive effect. Two studies with the best quality evidence demonstrated no difference in functional outcome. Overall there was low-to-moderate quality evidence with conflicting results and most trials only reporting proxy outcomes for clinical benefit [78, 79]. A recent Cochrane review demonstrated no significant difference in time to union as assessed by clinical outcomes. Subgroup analysis reported possible acceleration of nonoperatively treated fractures but overall insufficient data to recommend routine use [80]. The apparent consensus from most meta-analyses concludes that pulsed ultrasound may speed union rates as assessed by radiographs. However, improvements in patient-reported scores or clinical effect have not yet been convincingly reported in the literature [78, 80]. Pulsed ultrasound may have a role in accelerating healing in acute fractures and may be considered decreasing nonunion rate in at risk population, such as smokers and the elderly. Few risks or complications have been associated with its use [76]. The results are promising but need larger trials with more relevant outcomes are needed. At this point no clinically significant effect for low-intensity pulsed ultrasound has been established for stress fractures.

Extracorporeal Shock-Wave Therapy

Extracorporeal shock-wave (ECSW) therapy is commonly used in the treatment of renal nephrolithiasis but has more recently been introduced in musculoskeletal applications. The theoretic mechanism of action is that shock waves emitted from the device can produce microfractures at target tissue, thus stimulating an inflammatory response for bone healing and neovascularization [81]. These waves are induced through fluctuations of acoustic energy, pass through tissues and center at a given focal point. Mechanoreceptors are thought to receive the signals and regulate gene expression within the nucleus. In bone tissues, this is thought to promote bone morphogenetic protein production and osteoblast differentiation [82]. Johannes et al. evaluated nonunion healing in a canine model after resection osteotomy of radius and treated at 12 weeks after osteotomy. Subjects received either ECSW therapy or control. The authors found increased radiographic union rate in the group treated with ECSW therapy at 12-week examination [83].

A cohort series by Schaden et al. followed 115 patients with delayed or nonunion treated with single shock-wave treatment performed under anesthesia with postoperative immobilization. Seventy-six percent of patients had eventual bony consolidation though there was no report of time needed, poor detail of inclusion criteria, and lack of a comparison group [84]. Another clinical series of 43 patients with tibial or femoral nonunions treated with ECSW and found 72 % of patients had bony bridging at mean 4-month follow-up [85]. Wang et al. published a prospective series of patients who received ECSW therapy for treatment of long bone nonunions and reported 61 % achieving bony union rate at 6 months and 80 % at 12 months [81]. Similar rates of healing have been reported in other clinical series for treatment of tibia nonunions [86]. Furia et al. retrospectively compared treatment of fifth metatarsal nonunions at the metaphyseal-diaphyseal junction with screw fixation or ECSW. Although the authors found no differences in union rates, there were fewer complications in the group treated with ECSW [87].

Few randomized, prospective studies exist evaluating the efficacy of ECSW therapy in acute fractures. Wang et al. randomized 56 patients with acute, high-energy long bone fractures to treatment with surgical reduction and fixation with or without ESWT, performed directly after surgical fixation. The authors demonstrated a decreased nonunion rate (11 % vs. 20 %) for the treatment group at 1-year follow-up, with increased radiographic signs of fracture healing, improved VAS scores and greater proportion of patient's weight-bearing status at intermediate time points and similar infection rates [88]. Complications with use of ECSW therapy are often local reactions and include hematoma, swelling, and petechia [81, 84]. No studies have yet been performed to examine its role in the treatment of stress fractures.

Electrical Stimulation

Use of electromagnetic stimulation has also garnered interest in the treatment of nonunions. Basic science research suggests that pulsed electromagnetic fields may encourage mineralization, increase angiogenesis, and modify DNA synthesis [89]. Other reports postulate that the electric field stimulation of bone promotes flow of electrolytic bone fluid, simulating the effects of mechanically derived fluid flow to produce downstream biochemical changes in protein synthesis and osteogenesis [90]. Case series demonstrate electrical stimulation improves healing in long bone nonunions [91, 92]. Simonis et al. randomized 34 patients with tibial nonunions to treatment with external fixator with or without electrical stimulation and found association between use of electrical stimulation and radiographic union. However different rates of smokers and prior operations between groups may have confounded the results [93].

Beck et al. randomized 44 patients with posteromedial tibial stress fractures to either capacitatively coupled electric field stimulation or placebo treatment. All patients were instructed to wear the device for 15 h per day, given supplemental calcium (500 mg/day) and instructed to rest from provocative training. When evaluated on aggregate, no difference could be found in time to healing with use of the device. However, more severe stress fractures healed more quickly with the device than with placebo by 24.5 days. Increased hours of compliance was associated with greater reduction in time to healing in the treatment group than in placebo [90]. A meta-analysis by Mollon et al. reviewed use of electromagnetic stimulation in 11 randomized trials. Four studies demonstrated nonsignificant trends toward accelerated union in the setting of delayed or nonunion. Overall pooled analysis of the published literature demonstrated no significant therapeutic effect [94]. A systematic analysis in 2007 supported it as effective adjunct in the treatment of nonunions of long bones [89]. A Cochrane review investigating electromagnetic field stimulation applied towards nonunions or delayed unions found limited outcome measures and overall no significant difference in clinical scores, with inconclusive evidence to guide clinical practice [95]. Thus, there is conflicting evidence supporting the use of electrical stimulation in the treatment of stress fractures, with a potential benefit being noted in the treatment of more severe stress fractures.

Hyperbaric Oxygen

Some surgeons have proposed the use of hyperbaric oxygen therapy in order to augment fracture healing and decrease nonunion rates. Treatment comprises of placing a patient in an airtight vessel and increasing pressure within the vessel while simultaneous administering 100 % oxygen, intending to increase the partial pressure of oxygen in peripheral tissues [96]. Cultured osteoblasts in vitro exposed to hyperbaric oxygen had increased bone formation and demonstrated greater osteogenic activity [97]. Animal studies have shown evidence of accelerated healing, increased bone marrow density, and increased torsional strength and of fractures treated with intermittent hyperbaric oxygen therapy [98, 99]. However, a recent Cochrane meta-analysis identified only few animal studies that supported its use and no clinical trials of sufficient quality [96]. Three randomized clinical trials are currently ongoing to evaluate the role of hyperbaric oxygen treatment in acute fracture care. However, we have identified no studies at the time evaluating the role of hyperbaric oxygen therapy in the treatment of stress fractures.

Surgical Decision Making

To assist with physician decision-making, Diehl et al. divided stress fractures into low-risk and high-risk categories, defined by the location and grade of the injury [1]. Low-risk stress fractures heal predictably without the need for aggressive

Table 2.1 Treatment guide for low-risk stress fractures[a]

Symptoms	Goal	Treatment suggestions
Any level of pain	Heal injury	Titrate activity to a pain-free level for 4–8 weeks depending on the grade of injury
		Braces/crutches
		Modify risk factors
Pain with no functional limitations	Continue participation	Titrate activity to a stable or decreasing level of pain
		Closely follow
		Modify risk factors
Pain with functional limitation	Continue participation	Decrease activity level to point at which pain level is decreasing and until a functional level of pain has been achieved, then titrate activity to stable or continued decrease level of pain
		Modify risk factors
Limiting pain intensifies despite functional activity modification (i.e., unable to continue to perform at any reasonable functional level despite activity modification)	Heal injury	Complete rest
		Immobilization
		Surgery
		Modify risk factors

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intervention, often after 4 to 8 weeks of activity modification (Table 2.1). Some surgeons recommend a maximum of 10 % increase in activity per week once the athlete is pain free and fracture site is non-tender. For high-risk stress fractures, the authors recommend complete fracture healing prior to returning to activity (Table 2.2). This may be achieved either via absolute rest and non-weight bearing, or consideration of surgical intervention with possible internal fixation. Advantages of surgical fixation include accelerated healing and faster return to play, avoidance of recurrent fracture, and prevention of catastrophic fracture progression. Insufficient treatment of high-risk fractures may place the athlete at risk for significant complications depending on the location of the fracture (i.e., femoral neck, talus, navicular, fifth metatarsal, among others) [1].

A common rationale for the consideration of surgical fixation occurs in stress fractures with greater risk of nonunion or progression to complete fracture. Stress fractures of the anterior tibial diaphysis (the "dreaded black line" seen on radiographs) are subjected to constant tensile stress that may predispose it to delayed healing [3]. Reports of nonoperative treatment of these injuries describe delay in return to sport even at 1 year post injury [100] and significant chance of not being able to return to full activity [101]. Tension-sided stress fractures of the femoral neck, which occur on the superior femoral neck, have high propensity for progression as well as catastrophic consequences if they progresses to complete fracture; thus, fixation is often recommended for these injuries to prevent sequelae of delayed union, avascular necrosis, and termination of athletic careers [102–104]. A report of 42 patients with displaced femoral neck stress fractures demonstrated worse prognosis with delayed treatment and improved results with anatomic reduction and fixation [105]. The central zone of the tarsal navicular bone is subjected to significant shear loads and has limited vascular supply. These factors place it at greater risk for nonunion and surgical fixation in athletes may allow for more predictable healing with earlier return to sport [106]. Specific decisions of when and how surgical intervention would best be performed are dependent on site and severity of fracture, and addressed individually in later chapters.

Table 2.2 Management of and return-to-play strategies for high-risk stress fractures[a]

Anatomic site	Complications	Suggested treatment	Level of data
Femoral neck	Displacement	Tension: Strict NWB or bed rest	Level C (expert opinion)
	Nonunion	Surgical fixation	Level D (case series)
	Avascular necrosis	RTP when healed	
		Compression: NWB until pain-free with radiographic evidence of healing, then slow activity progression	
		RTP after no pain on examination or with any activities	
		Surgical fixation (optional)	
Anterior tibia	Nonunion	Nonoperative: NWB until pain-free with ADL; pneumatic leg splints	Level A (RCT)
	Delayed union	RTP with slow progression after nontender and pain-free with ADL (9 months)	Level B (nonrandomized)
	Fracture progression	Operative: intramedullary nailing	Levels C and D
		RTP is usually faster (2–4 months)	
Medial malleolus	Fracture progression	Nonoperative: (No fracture line)	Levels C and D
	Nonunion	4–6 weeks pneumatic casting	
		Avoid impact; rehabilitation	
		RTP when nontender, no pain with ADL	
		Operative: (fracture line, nonunion, or progression)	
		ORIF with bone graft	
Tarsal navicular	Nonunion	Nonoperative: NWB cast 6–8 weeks, then WB cast 6–8 weeks	Levels C and D
	Delayed union	RTP is gradual after pain-free with ADL	
	Displacement	Orthotics and rehabilitation suggested	
		Operative: (complete, nonunion)	
		RTP only when healed	
Talus	Nonunion	Nonoperative: NWB cast 6–8 weeks	Level C
	Delayed union	RTP is gradual after pain-free with ADL	
		Orthotics and rehabilitation suggested	
		Operative: reserved for nonunion	
Patella	Displacement	Nonoperative: (nondisplaced)	Level C
	Fracture completion	Long-leg NWB cast 4–6 weeks	
		Rehabilitation following	
		RTP is gradual after pain-free with ADL	
		Operative: horizontal—ORIF	
		Vertical—lateral fragment excision	
		RTP when healed	
Seasmoids	Nonunion	Nonoperative: NWB 6–8 weeks	Level C
	Delayed union	RTP is gradual after pain-free with ADL	
	Refracture	Operative: excision if fail nonoperative	
Fifth metatarsal	Nonunion	Nonoperative: (no fracture line)	Levels C and D
	Delayed union	NWB cast 4–6 weeks followed by WB cast until healed	
	Refracture	RTP after nontender and pain-free	
		Operative: (fracture line, nonunion, or individual at high risk for refracture)	
		Intramedullary screw fixation	
		RTP 6–8 weeks, early ROM/rehabilitation	

ADL activities of daily living, *NWB* non-weight bearing, *ORIF* open reduction with internal fixation, *RCT* randomized controlled trial, *ROM* range of motion, *RTP* return to play, *WB* weight bearing

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Summary

The management of stress fractures may be a challenging task for physicians involved in the care of athletes. Once diagnosed, often a multidisciplinary approach is important in order to identify metabolic risk factors, training regimens, and anatomic variables that may place an athlete at greater risks. Modification of training intensity, training surface, and footwear may decrease forces seen by bone. Supplementation of calcium and vitamin D are low-risk and simple methods to decrease risk of occurrence. New evidence suggests pharmacologics such as bisphosphonates and recombinant PTH may accelerate fracture healing, though should be used after careful discussion of risk and benefits with the patient. Adjunctive therapies are also evolving in order to augment the body's natural reparative abilities; many studies are ongoing in order to evaluate their role in the management of fractures. However, with careful attention to pathogenesis, activity modification, and medical treatment, many of these injuries may be successfully treated nonoperatively in order to allow athletes both safe and expeditious return to play.

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Biomechanics and Gait Analysis for Stress Fractures

3

Eric T. Greenberg, Scott Greenberg, and Kari Brown-Budde

Introduction

Stress fractures and other overuse injuries are often a result of successive events of micro trauma, and are associated with repetitive cyclical loading activities such as running, jumping, and marching [1, 2]. In the athletic population, stress fractures have been found to represent approximately 20 % of all injuries [3], with 33–56 % affecting the tibia [3–5]. The incidence in runners alone has been documented to range from 1.5 to 31 %, with females being more susceptible than males [5–9]. There are many reasons for the development of stress injuries in the lower extremity in the running population including inefficient running biomechanics, bone structure, anthropometric characteristics, running experience, improper training, inadequate overall fitness levels, muscle imbalances, running surface, and diet [9–14]. This high prevalence of overuse type injuries in running sports warrants the inspection of running biomechanics and identification of specific characteristics that may be linked with stress fracture injuries.

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Overview of Running Biomechanics

In order to understand the negative effects of abnormal movement on stress related injuries, a brief overview of normal running biomechanics will be discussed. Though walking and running have some commonalities, there are significant differences between the two that are important to appreciate (Table 3.1). Though both are cyclical in nature, walking is comprised of phases of both single and double limb support. Running, though similarly incorporates a single limb support phase, additionally contains a period of "float" where neither foot is in contact the ground. Running is also associated with higher movement velocities, greater joint excursions, increased balance demands, differences in the displacement of center of gravity, and most importantly elevated forces acting upon the body. In fact, vertical ground reaction forces have been found to be 2–3 times greater in running when compared to walking [15]. Figure 3.1 illustrates the normal kinematics of the lower extremity and pelvis during running.

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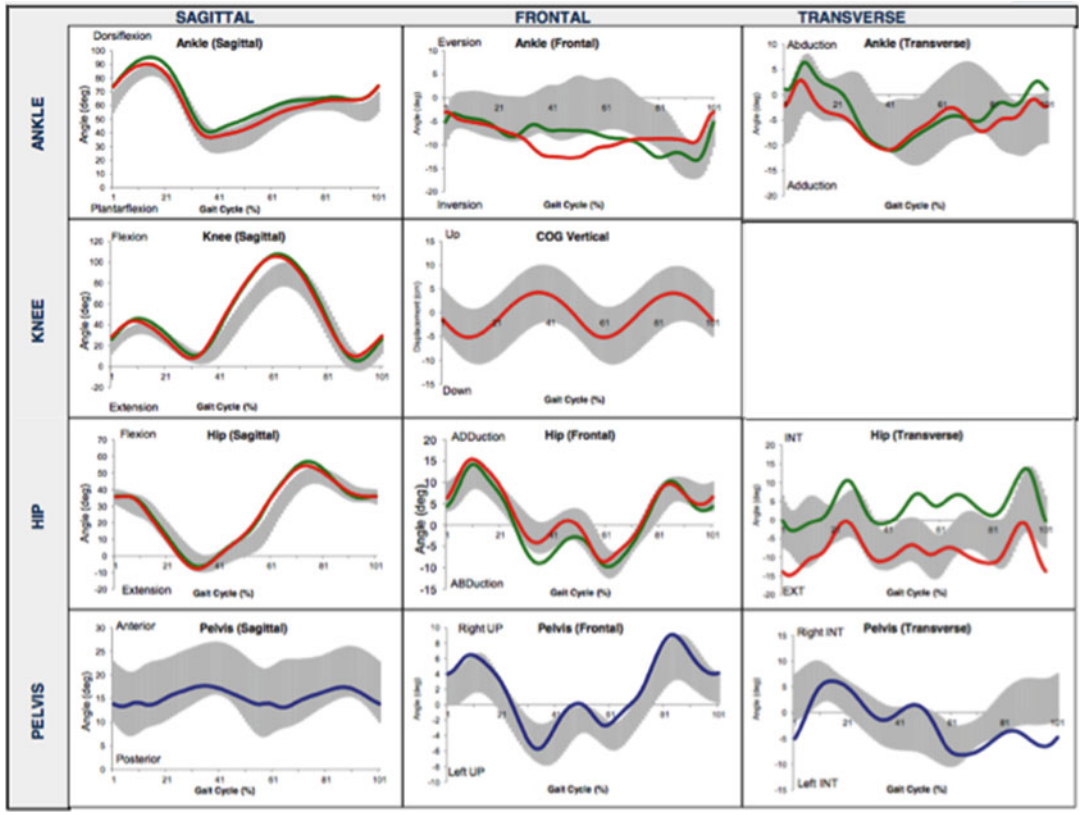

Fig. 3.1 Normal kinematic curves of the lower extremity during running. Courtesy of University of Florida Health Sports Performance Center, Running Analysis

Table 3.1 Biomechanical differences between walking and running

	Walking	Running
Ground reaction forces	1.5 × body weight	2–3 × body weight
Velocity	Lesser	Greater
Ranges of motion	Lesser	Greater
Support phase	Single and double limb support	Single limb support only
	Greater time spent in stance	Lesser time spent in stance
Swing phase	One limb at a time	Overlap between limbs (period of "float")
Center of gravity	Higher	Lower
Base of support	Wider	Narrower
Muscular demands	Lesser	Greater

There are three main phases of running: stance, swing, and float. As running speed increases, such as when a runner transitions from jogging to sprinting, the time spent in the stance phase decreases. The first half of the support phase is accountable for the proper force absorption and attenuation, with the second half more responsible for preparing the limb for forward horizontal propulsion. The support phase begins as the foot contacts the ground. A synchronous coordination of movement, starting from the interaction between the ground and the foot and continuation up the kinetic chain, is vital to healthy running biomechanics (Table 3.2). At this time, the process of force dissipation, or "pronation," is required. Pronation throughout the lower leg is comprised of subtalar joint eversion, forefoot abduction, talocrural joint dorsiflexion, and tibia

Table 3.2 Lower kinetic chain biomechanics

	Pronation	Supination
Lumbopelvic	Ipsilateral side bending	Contralateral side bending
	Contralateral pelvis drops	Contralateral pelvis raises
	Anterior innominate rotation	Anterior innominate rotation
Hip	Flexion	Extension
	Adduction	Abduction
	Internal rotation	External rotation
Knee	Flexion	Extension
	Abduction	Adduction
	Internal rotation	External rotation
Ankle	Dorsiflexion	Plantarflexion
	Internal rotation	External rotation
STJ	Talus adduction	Talus abduction
	Talus plantarflexion	Talus dorsiflexion
	Calcaneal eversion	Calcaneal inversion
MTJ	Dorsiflexion	Plantarflexion
	Abduction	Adduction
	Inversion	Eversion

internal rotation. Continuing up the kinetic chain, the knee then progresses into a more flexed, internally rotated, and valgus position. The hip will subsequently flex, adduct, and internally rotate and precede pelvic elevation and anterior rotation. Ultimately, the lumbosacral spine will extend and laterally flex towards the stance limb. During the second part of the stance phase, the body will prepare for eventual limb unloading and swing phase, requiring the foot to re-supinate and become more rigid [16]. Due to the complex nature of the running motion, it is imperative that an individual working with the running athlete have a keen understanding of normal running biomechanics.

Static Biomechanical Assessment

A thorough biomechanical assessment is an important aspect of the management and prevention of stress fractures. Intrinsic biomechanical properties have been implicated as possible contributing factors not only in stress fractures but also in other overuse injuries of the lower extremities. Though results are conflicting, it has been hypothesized that the structure and alignment of the lower extremity may influence the body's ability to control the forces associated with high impact activities [17]. Though there are many possible structural irregularities within the kinetic chain, specific structural characteristics have been linked with stress fractures and should be addressed. It is recommended that a static biomechanical assessment be performed in conjunction with a kinematic assessment to allow for a complete picture of the mechanics contributing to injury.

Foot Type Classification

Various methods of classifying foot structure have been presented in the literature, including, but not limited to, the Foot Posture Index [18, 19] and longitudinal arch angle [20, 21]. Classification of foot type attempts to distinguish between the static postures of a hyper-pronated, supinated, or neutrally aligned foot (Fig. 3.2). Though static foot structure has been postulated to contribute to pathology, results in the literature are conflicting [17, 22–24]. A hyper-pronated, low-arched foot is thought to contribute to excessive torsional forces about the tibia and strain on lower extremity musculature, as increased eccentric demands are required to control excessive motion.

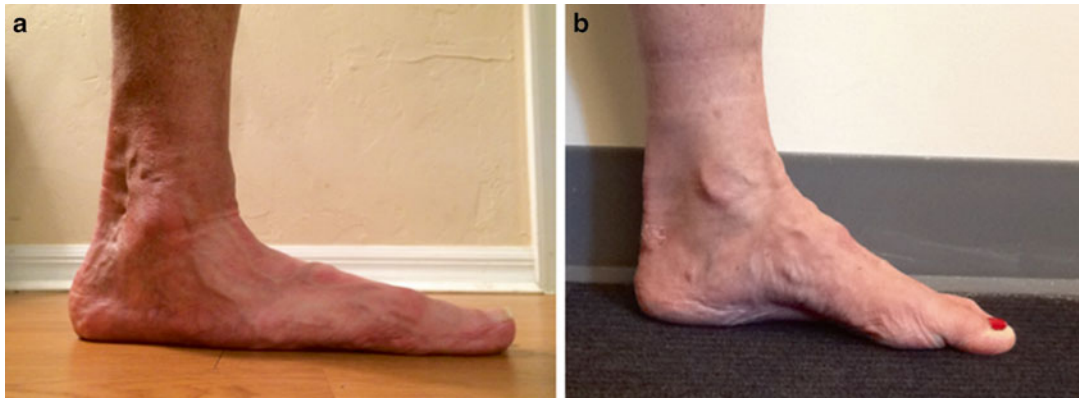

Fig. 3.2 (**a**) Low-arched, pronated foot type; (**b**) high-arched, supinated foot type

Conversely, a supinated or high-arched foot is thought to contribute to excessive stress on the bony architecture due the decreased ability to adequately absorb and dampen ground reaction forces [25].

Other Structural Links to Stress Fracture

Aside from foot structure, other lower extremity biomechanical characteristics should also be assessed. Structural leg length inequality seems to have the most evidence regarding its relationship to lower extremity stress injuries. It is believed that differences in side to side leg length may lead to altered lower extremity loading patterns, however preference as to the risk of injury on either the longer or shorter limb has not been consistently established [17, 24, 26–28]. Excessive forefoot, rear foot or tibial varus, q-angle, tibial torsion, and genu varus, valgus, or recurvatum are all thought to be factors contributing to tibial stress fractures, though the evidence about the true extent is conflicting [5, 16, 29]. Until research is able to more clearly and consistently demonstrate the contribution of static biomechanical measures to overuse injury, attention should be placed at any identified abnormalities.

Running Gait Analysis

The role of formal gait analysis is to determine whether a relationship exists between an individual's abilities and limitations and their individual gait pattern, to either enhance performance or assist with injury management [30]. Similarly to the evaluation of walking, running gait can be analyzed along a continuum ranging from real-time observational inspection, video motion analysis, or with the use of more extensive measurement devices and instrumentation. Analyses can be further performed by observing the runner while running on a treadmill or over level ground. A treadmill is often utilized as a means of gathering information from various angles while easily capturing successive foot strikes. However, there is conflicting evidence regarding whether running on a treadmill mimics the conditions of over ground running [31–37]. Provided the treadmill has limited intra-stride length variability [38], a sufficiently stiff surface [34, 38], and a belt speed that is adequately regulated [34], running on a treadmill can be generalized to over ground environments [37]. Conversely, it has also been concluded that increasing running speed on a treadmill is mechanically different from that while running over ground [35, 36]. Despite these claims, instrumentation may be setting dependent, though it is advisable to perform the analysis in the conditions in which the runner routinely trains.

Observational Gait Analysis

Observational gait analysis is the most widely utilized and least expensive form of running analysis. It is a gross analysis where the examiner will

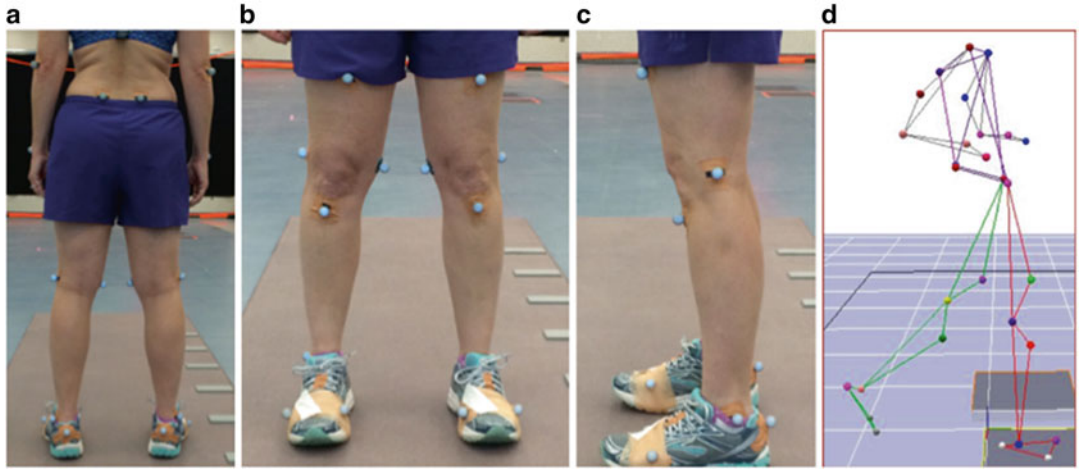

Fig. 3.3 Example of marker placement during running gait assessment; (**a**) posterior view, (**b**) anterior view, (**c**) lateral view, and (**d**) representation of an associated computer model

directly observe a runner with the naked eye in real-time. Attention is placed on the movements associated within each phase of the running cycle and is monitored in each of the three cardinal planes of motion. Anatomical markers may be placed on specific bony landmarks to assist with observation of body segments, and is especially beneficial when looking at more subtle movements, such as rotation in the transverse plane.

Due to the high limb velocities associated with running, it is difficult to examine specific amounts of motion or the simultaneous coordination of multiple joint segments of the lower kinetic chain [39]. Therefore, each joint or anatomical region is individually observed and examined in accordance with period of the running phase. Usually, observation of the loading phase of stance follows a "bottom up" approach, identifying the lower extremity's ability to pronate first at the foot and ankle, followed by progression up the lower kinetic chain. Conversely, when evaluating the later phase of stance, which focuses on the ability of the lower extremity to supinate, an observer can utilize a "top down" methodology [15].

Motion Analysis

The use of a video capture device will help enhance the accuracy of observational analysis. It will further allow a more quantitative description of gait parameters in relation to joint kinematics, however lacks the ability to measure associated forces acting upon the body. Motion capture is helpful to observe the movements and positions of multiple joint segments simultaneously, and assist with identifying other characteristics of gait including cadence, step length, stride length, and running speed [15]. To maintain a level of consistency and accuracy with measuring joint angles and positions within and between assessments, the marking of certain bilateral anatomical landmarks is advised (Fig. 3.3). A typical marker setup may include the anterior superior iliac spine (ASIS), posterior superior iliac spine (PSIS), greater trochanter, lateral femoral condyle, tibial tubercle, knee joint line, bisection of the posterior calcaneus, and second ray. Tight fitting clothing with increased exposure of the skin is also helpful when assessing true human movement.

The simplest form of motion capture is using a standard video camera (30–60 frames/s) that can view the runner from anterior, posterior, or lateral views. Though there has been evidence for the effectiveness of two-dimension motion analysis [40], especially in regards to observing frontal plane joint projections [41], there are inherent limitations of its use with gait analysis. Transverse plane and combined tri-planer motions are harder to interpret and often less accurate. Due to the subtlety of transverse plane motion, which is out of plane in relation to the view of the motion

capture device, true representations of rotational movements are more difficult to appreciate. However, the information gathered can still be useful and may assist a medical professional in directing future management of the healthy or injured runner.

More advanced forms of motion analysis systems have been developed that enable a clinician to readily capture three-dimensional measurements [42]. Though often more expensive, they are feasible for clinical use and provide more accurate representations of movement. With the use of reflective markers, computers assist in the capturing and processing of information regarding motion of limb segments, joint velocities, and joint angles [15].

Multiple high-speed motion capture systems and intricate computer software are also utilized for analyzing running in three-dimensions. Three-dimensional analysis is ideal, and has been found to be accurate in assessing running kinematics [43]. Kinematic data can further be utilized in conjunction with force plate data and electromyography (EMG). However, due to the costliness and longer setup times of such instrumentation, these measures are often utilized for laboratory, experimental gait analysis purposes. The use of force plates will provide information pertaining to how ground reaction forces in the vertical, horizontal, and transverse planes are imposed onto the body during the stance phase of running. This can be done with force plates mounted in the floor. Often, a laboratory setting makes the simulation of a normal running stride difficult and potentially unattainable due to limited space and the location of the force plate relative to the runner's normal stride [44, 45]. Due to the high stride to stride variance in ground reaction force measurements of both over ground and treadmill running, sufficient steps are preferred and best gathered via an instrumented treadmill [46]. Whether with level ground observation or via instrumented treadmill, measures of vertical loads, horizontal shear, vector patterns, joint torques, and center of pressure are helpful in the prevention and management of bony stress injuries [15]. EMG data is captured either by surface or fine wire electrodes, and can help in the analysis of a runner's neuromuscular control and patterns of muscle timing.

Running Biomechanical Assessment

For organization and clarity purposes, characteristics of running gait associated with stress fractures will be discussed separately in relation to the cardinal planes of motion (Table 3.3). This will assist a clinician who is performing a running analysis without the resources of extensive instrumentation to identify characteristics reported in the literature to be associated with stress fractures in running athletes.

Sagittal Plane Mechanics

Sagittal plane joint position and motion at the trunk, hip, knee, and ankle must be closely examined to identify possible risk factors that

Table 3.3 Biomechanical considerations for stress fractures during running

Force characteristics during limb loading	• Impact peak amplitude • Instantaneous vertical ground reaction force loading rate • Average vertical ground reaction force loading rate • Free moment
Sagittal plane	• Foot strike pattern • Peak tibial acceleration • Stride and step lengths • Cadence • Peak knee flexion during stance • Peak hip flexion during stance
Frontal plane	• Hip adduction • Knee abduction • Tibial outward tilting (excessive crossover) • Rear foot eversion • In-toe or out-toe
Transverse plane	• Hip internal rotation • Knee internal rotation • Tibial internal rotation • Foot pronation/supination

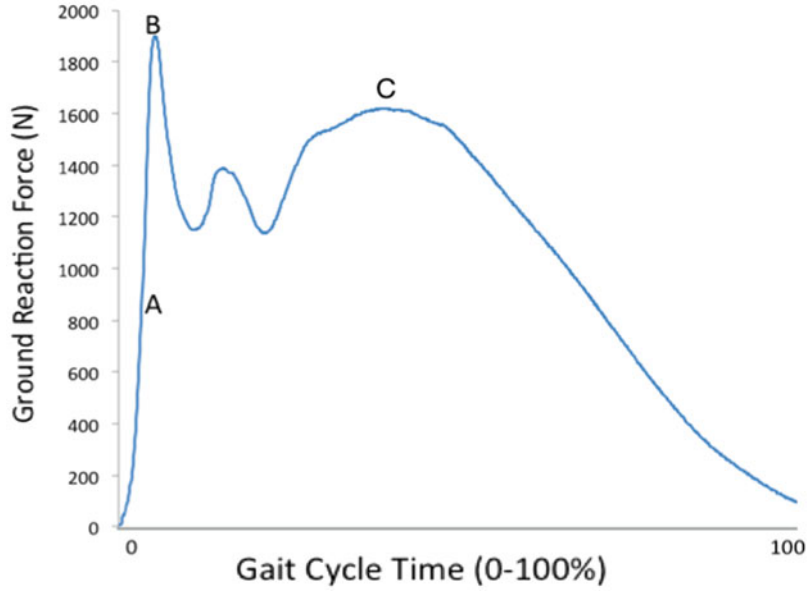

Fig. 3.4 Running ground reaction curve. "A" depicts the slope of the line used for determination of vertical loading rate; "B" depicts the impact peak; "C" depicts the active peak. Courtesy of University of Florida Health Sports Performance Center, Running Analysis

may predispose a running athlete to injuries. This can best be accomplished through inspecting an athlete from the lateral view. Joint positions during loading are highly related to kinetic and kinematic data that may place a runner at risk for injuries such as stress fractures. Reports show that 600,000–1,920,000 runners per year will sustain a stress fracture [1]. Due to this high volume of injuries, attempts to understand the mechanisms of these injuries have been the goal of recent research, with repeated impact loading being deemed the primary root cause [6]. Ground reaction forces, the forces exerted by the ground on a body [47], have been commonly measured as a way to approximate the external load acting on body structures during running.

To fully understand the effects of forces acting on the body, certain terminology should be defined. Loading rate is defined as the speed at which forces are applied to the body and can be identified using accelerometers or by the initial slope of the ground reaction force curve as the foot makes contact with the support surface. The steeper this slope of early stance on the ground reaction force curve, the more rapid the force application onto the body (Fig. 3.4). Vertical average loading rate, vertical impact peak, and vertical instantaneous loading rate seems to be higher in those who sustained overuse running related injuries [48].

Foot Position and Strike Pattern

The foot is typically divided into three sections, the rear foot, forefoot, and mid foot. The rear foot is made up of the talus and calcaneus, while the forefoot contains the metatarsals and phalangeal bones. The section of the foot between the rear and forefoot is termed the mid foot and houses the remaining tarsal bones, including the navicular, cuboid, and three cuneiforms. The manner and location at which a runner contacts the ground and loads the foot can differ and will alter how the body accepts ground reaction forces (Table 3.4). A rear foot strike pattern is characterized by loading the posterior-lateral heel and the foot dorsiflexed. Conversely, with a forefoot strike pattern the foot in slight plantar flexion and inversion, first loads the anterior aspect of the foot just proximal to fourth and fifth metatarsal heads (Fig. 3.5).

Fig. 3.5 Examples of strike patterns: (**a**) rear foot strike; (**b**) forefoot strike

Aside from location of contact, other biomechanical differences exist between strike patterns. As a foot is loaded at the rear foot, the center of pressure is transferred from a posterior lateral to anterior medial aspect of the foot, allowing pronation to occur first at the rear foot then by the forefoot. The medial longitudinal arch and plantar fascia, which assists in shock attenuation, is loaded at mid stance of the running cycle. When a foot contacts the ground more on the forefoot, the center of pressure goes in a posterior direction as the heel lowers to the ground and then changes direction to move anteriorly to prepare for unloading. Pronation occurs in the reverse direction, first working to assist with force dissipation at the forefoot, then at the rear foot. The medial longitudinal arch and associated plantar fascia also works to attenuate forces at impact [49]. Location of strike pattern can be best assessed from viewing the runner from a lateral perspective and identifying both ankle position and location of loading at initial contact.

Table 3.4 Differences between strike patterns

	Rear foot	Forefoot
Location of foot contact	Postero-lateral heel	Lateral forefoot proximal to fourth and fifth metatarsal heads
Ankle position at contact	Dorsiflexion	Slight plantar flexion and inversion
Center of pressure	Posterior lateral to anterior medial	Anterior to posterior (as heel lowers) followed by anterior
Loading of arch	Mid stance	Impact
Direction of foot pronation	Rear foot to forefoot	Forefoot to rear foot
Effect at knee	More work	Less work
Effect at ankle	Less work	More work

Various strike patterns have been a topic of continuing debate and highly studied in the running literature [50–53]. Both rear foot and forefoot strike patterns present with inherent benefits and pitfalls in relation to biomechanical links to injury. One study revealed runners who habitually rear foot strike have higher rates of repetitive stress injuries when compared to forefoot strikers, due to an inability of the body to effectively attenuate ground reaction forces [54]. Others, on the contrary, have hypothesized that forefoot striking places the foot and ankle at greater risk of injury, in part due to an increase in eccentric work and strain at the foot and ankle [55, 56].

Stress injuries are often due the repetitive overload due to an inability to properly dissipate associated forces, resulting in micro trauma of bodily tissues. The manner at which the body accepts loading forces varies between the strike patterns and is illustrated in the different shape of their representative ground reaction force curves (Fig. 3.6). A significant correlation exists between tibial shock and vertical ground reaction force loading rates, especially at the early parts of

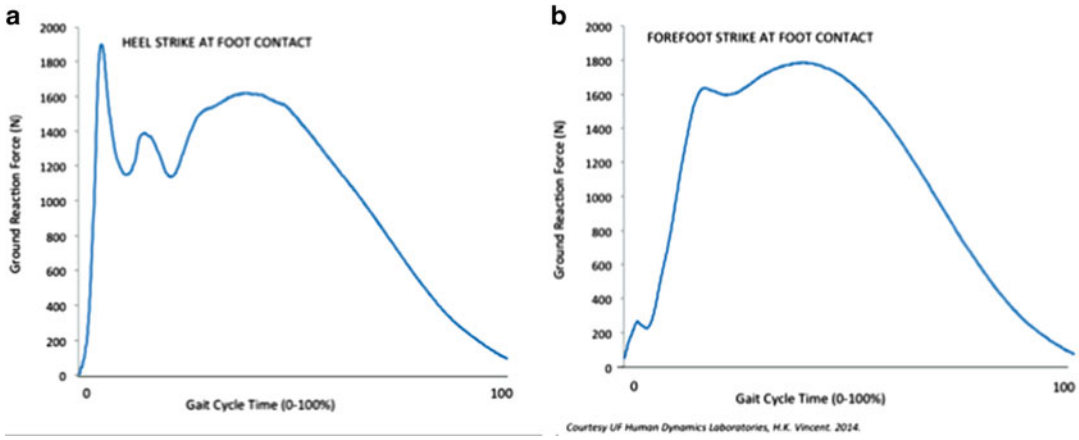

Fig. 3.6 Examples of ground reaction curves demonstrating the (**a**) rear foot strike significant impact peak and rapid vertical loading rate of a rear foot strike pattern and (**b**) smoother curvature, lack of impact transient, and slower loading rate of a forefoot strike pattern. Courtesy of University of Florida Health Sports Performance Center, Running Analysis

stance phase, and lower extremity stress fractures. Within the first 10 % of the stance and lasting about 10–30 ms, the first peak in force, or "impact transient," occurs when a heel initially contacts the support surface. The presence of an impact transient is a sudden rise in force following initial ground contact and exemplifies forces acting rapidly on the body [57]. A second peak, the "active peak," ensues during mid stance and can last up to 200 ms [58]. A rear foot strike contains double impact peaks, while a forefoot striker has an overall flatter curve, lack of an impact transient, and decreased rate of loading [49, 51]. The disappearance of impact transient in forefoot strikers may be attributed to the pre-activation of the triceps surae complex that will assist in the dampening of impact forces [3].

Different strike patterns will affect how loads are placed on the body along with the runner's strategies at attenuating these forces. Specifically, the biomechanical demands at the ankle and knee are altered. Compared to a rear foot strike pattern, by landing on one's forefoot, a runner with a forefoot strike will have decreased overall work at the knee with an increased work at the ankle. This increased demand at the foot and ankle over successive foot strikes may predispose an athlete to stress injuries about the metatarsals and bones of the forefoot [55]. However, the decrease in work at the knee may allow an athlete to resist fatigue and shift the strain away from the knee, which is the most common site for running related injuries.

Hip and Knee

Gaining an appreciation of how impact loads are absorbed by the body is critical when quantifying the risk factors associated with stress fractures. Peak joint flexion ranges at the hip and knee can easily be observed in the sagittal plane. Alterations in the hip and knee joint have been linked to the amount of impact shock during loading at initial contact of the running cycle [59]. It has been determined that an increase in knee flexion during loading enhances dampening of ground reaction forces, thus decreasing injury risk [60, 61]. However, controversy arises as to the negative effects of knee flexion at contact [62]. Though a certain amount of knee flexion is desired to help attenuate shock, excessive knee flexion throughout stance may result in an increase in tibial shock, and a greater risk for stress injury [63]. A more flexed knee at contact will decrease overall knee range of motion excursion and possibly contribute to greater amounts of tibial shock. In conjunction, more knee flexion at loading will result in a more vertical tibial shaft and closely align the long axis of the tibia with the vertical component of the ground reaction force and subsequent tibial shock [62].

Stride Characteristics

Step length is the distance measured from the contact of one foot from that of the other, while stride length is a measure of the distance covered between successive ground contacts of the same foot. Stride length can be readily measured by marking designated increments on the side of the treadmill or along the running surface and calculating the distance between successive foot strikes. Running stride has been found to me much more smooth in competitive runners when compared to their recreational counterparts [64]. Generally, runners also tend to utilize a preferred stride rate and length, however they may not be the most optimal in regards to running economy and force attenuation [65]. Overstriding and an increased braking force has been linked to a history of stress fractures [66, 67], while shortening stride length decreases the risk of stress related injuries [68]. This may be due to the influence of the ground reaction force [69], positive effects on the amount of work required of the hip and knee [70], shorter times spent in the support phase, and pre-activation of leg musculature [65]. Magnitude and direction of ground reaction force vectors have been associated with tibial stress fractures by increasing the bending load acting on the tibia. It has been determined that a more posteriorly directed sagittal plane ground reaction force vector will increase the bending load acting on the tibia [69]. This posteriorly directed force is seen in runners who contact the ground in front of their base of support, regardless of strike pattern [67].

Cadence, also known as step rate, is the number of foot contacts made in a minute. The relationship between cadence and stride lengths has been clearly identified [70]. At a given speed, as cadence increases, stride length will decrease. Some authors recommend that 180 steps per minute is the desired cadence for healthy running [67], while others have found that increasing cadence by 5–10 % of a runner's preferred step rate is helpful in decreasing impact forces and subsequent injury [70]. It has been determined that stress fracture development is more dependent on loading magnitude than loading exposures. Though an increase in step rate will result in more overall contacts and loading cycles over the course of a given distance, the factor of decreased loads over each contact is more crucial [68]. In other words, it is less stressful for the body to deal with an increase in foot contacts than the effects of spending excessive time in contact with the ground.

Frontal and Transverse Plane Mechanics

The evaluation of a runner from the anterior or posterior view in the clinical setting will allow for biomechanical assessment of frontal plane, and to a lesser extent transverse plane joint projections. Attenuation of forces has been found not only to occur by the effects of sagittal plane kinematics, but also with the ability to absorb impact with respect to frontal plane movements [5, 71]. In fact, it has been found that the presence of three frontal plane movement characteristics, maximum hip adduction, maximum rear foot eversion, and absolute value of free moment, was able to successfully predict a history of stress fractures with 83 % accuracy [4].

The mere presence of heightened vertical forces will place bony structures at risk for breakdown and injury. However, not all athletes presenting with a lower extremity stress fracture present with high levels of shock [71, 72]. Dynamic lower extremity alignment is thought to play a role in the body's ability to properly attenuate ground reaction forces. During normal running, forces of up to two to three times body weight are imparted on the body [73]. Improper biomechanics and dynamic alignment may potentially alter the normal distribution of normal loads, and contribute to excessive strain on body tissues. Thus, due to the repetitive nature of running, long bones may be seven times less resistant to fatigue and subsequent micro trauma [71, 74].

Both local and distal biomechanical factors have been shown to be associated with various lower extremity overuse injuries including stress fractures [75–79]. Due to the interdependence of the kinetic chain, distal factors may directly influence neighboring structures and contribute to excessive strain to a specific injury site.

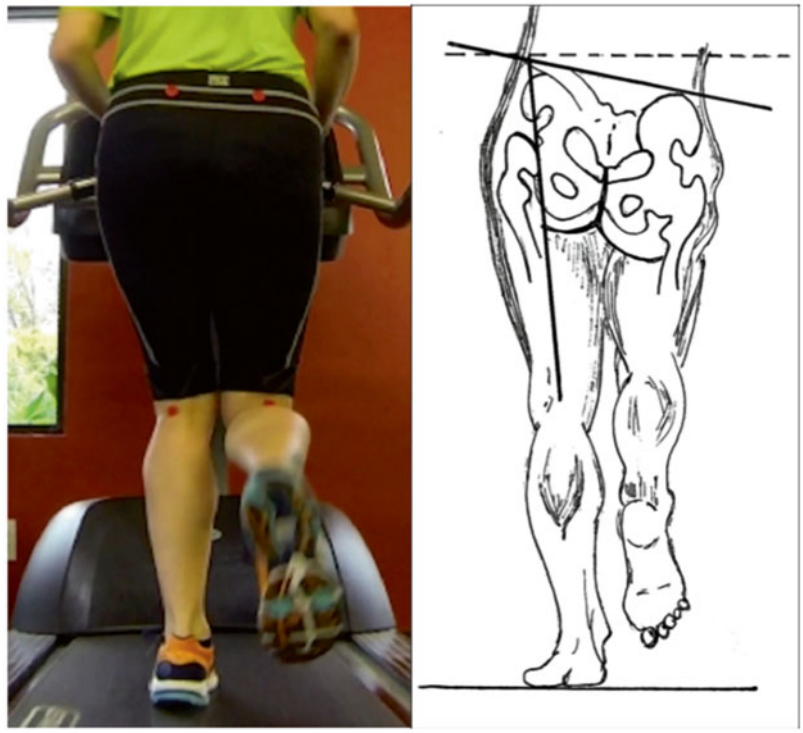

Fig. 3.7 Examples of unlevel PSIS (*circles* on posterior aspect of pelvis) and excessive contralateral right pelvic drop during left mid stance

As discussed earlier, tri-planar motions of pronation and supination have a direct effect on the entire lower extremity kinetic chain. Therefore, careful inspection of the trunk, pelvis, hip, knee, lower leg, ankle, and foot during the entire stance phase through swing preparation is warranted to identify specific biomechanical limitations and/or abnormalities.

Trunk, Pelvis, and Hip

Characteristics of frontal and transverse plane hip kinematics during functional activities, such as running, have been implicated to contribute to various lower extremity injuries and stress fractures [71, 72, 76]. Normally, the body attempts to attenuate ground reaction forces throughout the entire lower kinetic chain, including the lumbopelvic complex. At mid stance, the swing leg is parallel with the contralateral stance limb and a runner's center of mass is directly over their base of support. There is a subtle yet normal drop of the unsupported pelvis and relative hip adduction of the stance hip. This assists in efficient attenuation and adequate transfer of ground reaction forces. However, a significant increase in peak hip adduction angles throughout the stance phase of their running gait cycle has been found in those with a history of stress fracture and in those with a risk of future development [4, 48, 71, 80]. As a result of the excessive hip adduction, despite normal forces, extensive bending of the tibia, with compression medially and tension laterally, increases the susceptibility to injury [4], especially on the tension side. Though a relationship exists between the presence of hip adduction and tibial strain, it is not yet known as to which, if any, is the precipitating factor. However, observing the ability of a runner to maintain a relatively level pelvis and normal degree of hip adduction (angle formed between the line from bilateral PSIS and the line from PSIS to lateral femoral condyle) during the initial contact and mid stance is recommended to assist in both the prevention and management of those with lower extremity stress injuries (Fig. 3.7).

As previously mentioned, when loading the limb during initial contact and mid stance, the hip will flex in the sagittal plane and adduct in the frontal plane. Due to the body moving concurrently in three planes, transverse plane hip internal rotation motion is also expected. Excessive transverse plane motion has been associated with many overuse injuries to the lower extremities, however its presence as a risk factor and significance in stress fractures are inconclusive [5, 71, 75].

Identification and determination of excessive hip internal rotation on two-dimensional observation are difficult. However, any relevant information regarding degree of hip internal rotation motion will be helpful in the recognition of faulty movement characteristics. Normal hip rotation is characterized by a posteriorly facing popliteal fossa, where a lateral orientation supports the presence of excessive femoral internal rotation. When viewing a runner from the anterior perspective, viewing patella position can further support this finding. A femur in a misaligned, internally rotated position will correspond to a medially facing patella, whereas a femur in neutral rotation, regardless of any degree of frontal plane involvement, will face directly anterior.

Knee and Lower Leg

Stress fractures have been associated with pathological biomechanics about the knee and lower leg, including the tibia and fibula. As previously discussed, the lower extremity works as a unit and to infer that abnormal mechanics occur in isolation at a particular region is not advised. However, there have been certain findings at the knee that have been found to be common and consistent in those with tibial stress fractures. These include increased peak knee internal rotation [5, 71], decreased knee adduction and shift towards a more valgus positioning [5, 71], and excessive frontal plane tibia outward tilting [81].

Human long bones are most susceptible to torsional loads and, consequentially, repetitive stress injuries [81]. A reduction of knee adduction, often associated with lower extremity pronation, is a biomechanical term to categorize a frontal plane dynamic misalignment, where the knee is in a more valgus position. Due to the knee joint being comprised of the femur and the tibia, excessive femoral or hip adduction will directly affect frontal plane knee position. The inability for the runner to maintain some degree of separation between both knees throughout the stance phase of the running cycle can represent the presence of excessive knee abduction or reduced knee adduction (Fig. 3.8). In the presence of such a finding, specific interventions have been successful to reduce frontal plane loading to the knee including gait retraining [77, 78], hip abductor and external rotator muscle strengthening [82], and biomechanical orthotic device prescription [83].

The degree of tibial outward tilt has been found to correspond to magnitude of torsional moments. There is some evidence to suggest that this frontal plane characteristic has more effect on torsional loading to the lower limb when compared to the sagittal and transverse planes, accounting for 76 % of the variability during running [81]. Outward tilt is defined as the proximal aspect of the tibia moving away from midline of the body with the distal aspect moving towards the midline. A foot contact that is closer to the midline of the body will directly result in increased outward tilting of the tibia and subsequent increase in maximum torsional loads across the bony shaft Therefore, identification of the foot location at initial contact and position throughout the stance phase in relation to the midline of the body should be assessed due to its direct relationship with amount of torsional loads. This is best viewed and analyzed from a posterior and/or anterior view during the running gait assessment.

Foot and Ankle

The foot is the first part of the runner's body to make contact with the support surface. This interface is the first defense the body has for ensuring proper limb positioning and attenuation of vertical and torsional forces. Foot architecture directly supports its function as a mobile adaptor, having many joints and articulations, musculotendinous structures and degrees of freedom to adapt to the high demands of running. To dampen the loads and aid in shock attenuation, the foot and ankle

Fig. 3.8 (**a**, **b**) Significant knee abduction, femoral adduction, and decreased knee separation during right mid stance

has the ability to "pronate," characterized by closed kinetic chain subtalar joint eversion, plantar flexion and adduction, forefoot abduction, and tibial internal rotation [16]. Without sufficient pronation, the body will be unable to attenuate the high loads associated with impact activities such as running, jumping, and marching, and ultimately lead to repetitive stress injury. However, excessive pronation is potentially problematic, as it will place an excessive amount of strain on soft tissue and osseous structures [14]. Many frontal and transverse planes characteristics about the foot and ankle complex have been linked to stress injuries of bone and are often related to excessive, premature, prolonged, or limited amounts of pronation.

Pronation is a tri-planer motion that is not easily detected or studied in two-dimensional analyses and usually requires more extensive instrumentation. However, an increased maximum rear foot eversion angle, a component of pronation, has been associated with tibial stress fractures in runners [4, 14, 17, 71, 84]. There is evidence of joint coupling between rear foot eversion, tibial internal rotation, and knee internal rotation [85, 86]. For every 2° of rear foot eversion, the tibia and knee will internally rotate 1°, allowing inferences as to rotation about the tibia with calcaneus frontal plane projections [85]. It has also been postulated that excessive calcaneal eversion will contribute to premature muscle fatigue and subject the tibia to abnormal torsional loads. One of the functions of the tibialis posterior is to decrease tensile loads of the medial aspect of the tibia, due to the location of its bony attachment. As the tibialis posterior works to eccentrically control foot eversion, increased loading demands may contribute to fatigue and diminish the tibia's ability to tolerate tensile loads [4, 84].

Rear foot calcaneal eversion angles can be appreciated by observing a runner posteriorly during a gait analysis. A line bisecting the calcaneus and a line bisecting the posterior tibia form the angle of representative of calcaneal eversion (Fig. 3.9). The ability to detect the presence of excessive eversion in the clinical setting is not without obstacles. The runner's shoe can hinder

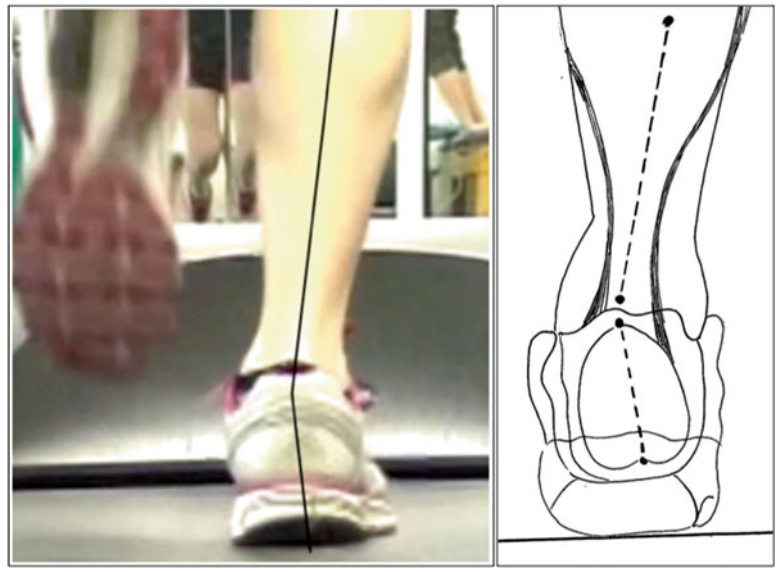

Fig. 3.9 Measures of calcaneal eversion angle formed by a line bisecting the posterior calcaneus and another line bisecting the posterior tibia

an accurate measurement by not permitting adequate viewing of the bony anatomy. Secondly, the amount of total available eversion is relatively small and to determine normal from abnormal may also be difficult. However, this still may assist a clinician in determining the amount of calcaneal motion in the frontal plane.

High values of free moment have been linked to excessive torsional loads through the foot. Free moment is a measure of torque that acts between the foot and the ground while running [4]. It can also be described as the resistance to in-toeing and out-toeing and has been shown to share a direct relationship with amount of rear foot eversion [48]. These forces are transmitted up the lower kinetic chain, placing the tibia and lower extremity at risk of overuse injury [71]. Heightened loads will contribute to a rapid and premature foot over-pronation in attempts to attenuate forces. Attention to the position of the foot in regards to toe-in and toe-out can be appreciated by inspection of a runner from the posterior view. Treadmills that allow an examiner to view the feet of the athlete during stance from the anterior view can also assist in the assessment of absolute free moment by identifying the presence of abnormal foot abduction or adduction.

Though excessive or prolonged pronation in the stance phase has been associated with stress fractures of the lower extremity, the lack of a runner's ability to attenuate shock can also contribute to excess strain on the skeletal system [87]. The high-arched, pes cavus foot type may be more rigid and thus less adaptable to the support surface. The lack of mobility contributes to inadequate shock attenuation and increased forces passing through the bones of the foot, tibia, femur, and pelvis [17, 24]. Assessment should not be limited to static assessment, but instead be performed in conjunction with a dynamic running evaluation. In the case where an athlete presents with a flexible pes cavus foot type, where the foot has the ability to move through a range of motion into pronation, the ability to adapt to the support surface may not be inhibited. However, in the instance where a foot lacks a sufficient amount of mobility, increased rigidity upon ground contact may contribute to excessive loads placed about the skeletal system. This is often represented by a decrease in rear foot

eversion mobility and a decrease in the lowering of the medial longitudinal arch of the foot. In this case, means of increasing foot mobility and other interventions to decrease impact may be warranted to allow for better load attenuation [22].

General Treatment and Gait Retraining

The management of stress fractures usually consists of an approximate 6-week period of relative rest to allow for bony repair and remodeling in which the athlete is permitted to perform cross-training activities such as swimming and light cycling. Full weight bearing is usually acceptable, as bone will lay down bone trabeculae in response to the loads that are applied to the system. In cases where the athlete cannot tolerate full weight acceptance, a period of immobilization and/or limitation of weight bearing may be needed. Patient education regarding proper training habits is also paramount to ensure successful rehabilitation and decrease the likelihood of recidivism.

The restoration of muscle strength, flexibility, and neuromuscular control of the running athlete are integral parts of a complete rehabilitation program. Interventions aimed at strengthening the gluteal musculature have been found to be successful in the management of lower extremity overuse injuries. Other strengthening activities that target the muscles responsible for controlling pronation about the foot, lower leg, knee, hip, and pelvis are also recommended. Increasing the flexibility and range of motion to acceptable levels will also assist the runner to work more at their midranges of motion throughout the running cycle and prevent excessive strain on static stabilizers. Movement retraining can also be integrated to promote proper coordination between body segments to allow for maintenance of more efficient skeletal alignment with functional activities.

It is unclear whether an alteration in running mechanics will result, despite a reduction of symptoms, following strength, flexibility training, and nonspecific movement training [77]. Gait retraining, utilizing principles of motor learning, has been shown to be successful in the management of stress fractures and other lower extremity overuse injuries. Retraining the athlete's running gait has focused on decreasing impact loading by decreasing tibial acceleration [1, 10], increasing step rate [70], and decreasing dynamic knee valgus [88]. Augmented feedback, in the form of mirrors, amplitude of tibial acceleration, and metronomes, may be administered in specific schedules to enhance the intrinsic learning and retention of a new movement pattern. Interventions aimed directly at influencing abnormal running biomechanics that have been associated with stress injuries allow runners to limit excessive loading forces in the early part of the stance phase [1, 10, 70] as well as promote more optimal dynamic alignment [88] to limit excessive strain imparted on the lower extremity.

Summary

The evaluation, management, and prevention of bony stress injuries in athletes are multifactorial. The importance of a thorough medical workup, keen understanding of the possible etiologies of injury, identification of associated training errors, adequate physical examination, and knowledge of running biomechanics are imperative when managing these types of injuries in the running athlete. When performing a physical biomechanical examination and formal running analysis, it may be difficult to determine specific factors that are contributing to musculoskeletal breakdown due to the abundance of information. A practitioner working with this patient population should have a good sense of normal biomechanics, movement patterns, and running specific factors that influence an athlete's risk of developing stress fractures.

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Classification of Stress Fractures

4

Christopher C. Kaeding and Timothy L. Miller

Defining a Stress Fracture

Stress fractures of bone, also known as fatigue fractures or fatigue failure of bone, are common and troublesome injuries in athletes and non-athletes alike. Typically occurring in individuals who perform repetitive tasks, these fractures result from an overuse mechanism in bone [1–6]. With every strain episode of bone, regardless of its magnitude, microdamage occurs in the bone in the form of microcracks. These microcracks occur in areas of stress concentration. The initiation sites of these microcracks tend to occur at areas of discontinuity in the bone such as haversion canals and lacunae. In healthy homeostatic bone, the microdamage elicits a reparative response and the bone is repaired restoring it to its initial structural state. In hypertrophying bone, the microcracks result in a positive adaptive response that results in the overall bone being stronger than its original state. In both of these conditions the microdamage does not accumulate. When the creation or propagation of microcracks occurs more quickly than the bone can repair them, fatigue failure of the bone occurs.

Stress fractures begin as an increased number or size of microcracks that are not repaired. These microcracks can coalesce or propagate to create a frank fracture line. This fracture line can progress from an incomplete crack to a complete fracture, to a displaced fracture, and possibly to nonunion (Fig. 4.1). This progression of microdamage in the bone is dependent on the biologic healing potential of the bone. The more capable the bone is to heal the microcracks, the less likely the microdamage is to progress. As is apparent from this review of the pathophysiology of fatigue failure of bone, stress fractures represent a continuum of structural damage and are not a single consistent entity. They have a spectrum of severity with variations in treatment and prognosis.

A distinction should be drawn between a stress fracture and an insufficiency fracture as these are not the same injury and occur via different mechanisms. Though related, a stress fracture occurs when an essentially normal bone breaks after being subjected to repetitive tensile, compressive, or torsional stresses, none of which, individually, would be large enough to cause a bone to fail in a person without underlying bone disease. Insufficiency fractures occur when the mechanical strength of a bone is reduced to the point that a stress, which would not be sufficient

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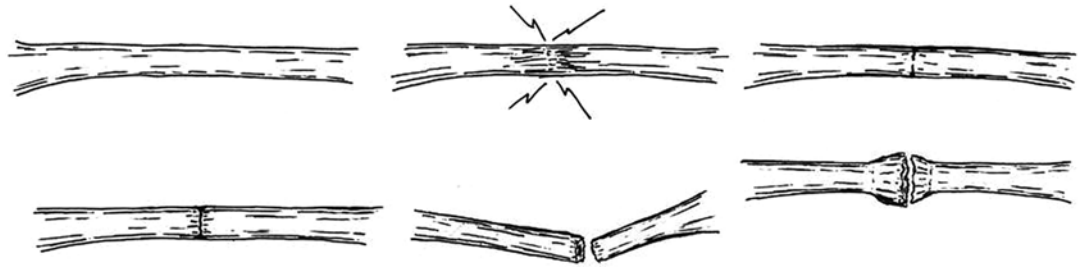

Fig. 4.1 Diagram illustrating the spectrum of severity of bony stress injury including normal bone, stress reaction with no fracture, incomplete fracture, complete fracture without displacement, displaced fracture, and nonunion

to fracture a healthy bone, breaks the weakened bone. Insufficiency fractures, their causes, and treatment strategies will be covered in a separate chapter of this textbook.

Features of a Quality Classification System

The reliability of a classification system requires comparison to the gold standard [7]. Furthermore, the validity of a classification system is dependent upon the accuracy with which the system describes the true pathologic process. Audige's quality criteria from 2004 reflected the importance of clearly described categories and inclusion/exclusion criteria for determining inter- and intra-observer reliability [7].

According to Garbuz et al., a classification system should help orthopaedic surgeons characterize a problem, suggest a potential prognosis, offer guidance in determining optimal treatment, characterize the nature of a problem, and influence treatment decision-making, ultimately improving outcomes [8]. The same authors further asserted that a classification system should form a basis for uniform reporting of treatments [8].

Stratifying patients with stress fractures into prognostic and treatment groups has historically been difficult given the lack of a single widely applicable standard classification system. Textbooks and review articles have cited techniques for describing stress fractures at a particular location, but have rarely been validated as a method for determination of stress fracture severity, risk, and prognosis [9–16]. An understanding of the basic science of fatigue failure of bone correlates well with our clinical experience that structural failure occurs along a spectrum from micro-fractures to complete structural failure.

Because stress fractures have various degrees of structural failure and healing potential, it is important that we develop standardized categorization and descriptive instruments. Descriptive systems should identify the clinically relevant attributes of the injury in a reproducible fashion and should do so in a simple, inexpensive, safe, and widely applicable manner. For a comprehensive description of stress fractures, these characteristics should be incorporated into a system that describes not only the extent of the structural damage but also the healing potential.

High-Risk vs. Low-Risk Stress Fractures

Unlike most traumatic fractures, in the case of stress injuries of bone the size and extent of the fracture line vary greatly, and the healing potential varies by location. Some locations typically heal very readily. Other locations, such as the junction of the metaphysis and the diaphysis of the proximal fifth metatarsal, tend to have an increased risk of delayed union, nonunion, and refracture. Boden et al. described high-risk and low-risk stress fractures by their location [17, 18]. Those locations that have a tendency toward delayed union, nonunion, or refracture are classified as high-risk stress fractures. The varied healing potentials may be related to biologic and/or biomechanical factors of the different anatomic sites.

Table 4.1 Anatomic sites for high-risk stress fractures[a]

• Femoral neck (tension side)
• Patella (tension side)
• Anterior tibial cortex
• Medial malleolus
• Talar neck
• Dorsal tarsal navicular cortex
• Fifth metatarsal proximal metaphysis
• Sesamoids of the great toe

[a]Adapted from [17]

An important distinction regarding stress fractures is whether they are high- or low-risk fractures (Table 4.1). This classification system has been proposed many times in the literature [17–22]. Such a system provides a reproducible way for medical personnel to determine the course of treatment and the timeframe of recovery before the athlete can return to play. Stress fractures are considered to be high-risk fractures if they have any of the following characteristics. First, these fractures have a predilection to progress to complete fracture (fifth metatarsal), delayed union (anterior cortex tibia), or nonunion (tarsal navicular). Second, a delay in diagnosis and treatment can either prolong an athlete's non-weightbearing status and his or her restriction from sport, or change a nonsurgical treatment to one requiring operative fixation with or without bone graft.

These high-risk sites possess a common biomechanical characteristic [3–5, 17, 18]. The initiation of their associated fracture lines typically occurs on the tension side of the bone or in a watershed (relatively avascular) area of the vascular supply (e.g., superior side of the femoral neck, anterior cortex of the tibial shaft, lateral aspect of the proximal fifth metatarsal, and the dorsal side of the tarsal navicular) [5]. Because bone is less resistant to tensile than compressive forces, this likely puts the bone at these locations at increased risk for microcrack initiation. Why these "high-risk" locations have an increased risk of impaired healing is likely a result of additional influences beyond the biomechanical factors.

The biomechanical factors of being on the tensile side of the bone explain the increased requirement for a healing response, but biologic factors may come into play as well. For example, the proximal junction of the fifth metatarsal diaphysis/metaphysis is a vascular watershed area with suboptimal blood supply to support fracture healing. Locations of high-risk stress fractures may be the combination of increased micro-failure due to biomechanical conditions coupled with impaired biologic healing capacity. Table 4.1 lists the locations of commonly described high-risk stress fractures.

A common example of a poor natural history of a high-risk stress fracture is neglect of an early proximal fifth metatarsal stress failure that results in either an acute fracture or, should it heal, a subsequent refracture. Recognition of this fracture as a high-risk location and early intramedullary screw fixation will often lead to timely healing, and the athlete can resume his or her career with a markedly decreased risk of re-injury.

When compared with high-risk fractures, low-risk fractures have an overall favorable natural history. In contrast to high-risk fractures, which tend to be on the tension side of bone, low-risk fractures tend to occur on the compression side of bone and typically heal readily. Low-risk fractures are less likely to develop a delayed or nonunion, recur, or have a significant complication should it progress to complete fracture. Low-risk stress fractures can typically be treated with activity modification and rarely require surgical intervention. Low-risk fractures include the femoral shaft, medial tibia, ribs, ulna shaft, and first through fourth metatarsals. Anatomic location of the fatigue bone failure is the distinguishing characteristic between high- and low-risk fractures. Determining whether the stress fracture is in a high-risk vs. a low-risk location is key to optimal care as it impacts both treatment and prognosis discussions. This characteristic makes judging a stress fracture to be either high-risk or low-risk an important element in the "classification" of the injury. Table 4.2 describes the key elements of high-risk vs. low-risk stress fractures.

The goal in treating athletes is to make an expeditious diagnosis of a stress fracture because those with stress fractures classified as low-risk fractures can participate in modified sports activity, whereas athletes with stress fractures classified as high-risk should be aggressively managed with non-weightbearing activity or surgery [3–5, 19, 20].

Table 4.2 Key elements of high-risk vs. low-risk stress fractures[a]

High-risk fractures
Occur where tensile forces are concentrated
Natural history is concerning for delayed union or nonunion
Often require aggressive treatment including surgery or strict non-weightbearing
Low-risk fractures
Occur on the compression side of bone
Natural history favorable for healing
Usually respond to nonsurgical treatment with rest and gradual return to causative activity

[a]Adapted from [5]

This obviously important clinical implication of the fracture being identified as either high- or low-risk makes it one of the most important classifications of fatigue failure of bone the clinician can make.

Current and Historical Classification Systems

A recent literature review by Miller et al. revealed 26 stress fracture classification systems [21]. Table 4.3 lists the classification systems reviewed [17, 18, 23–44]. The goal of this review was to determine what classification and grading systems have been referenced in the literature for stress fractures. At the outset of this review, the authors of this study asked two questions: (1) "What classification systems are used in the evaluation and treatment of stress fractures?" and (2) "What are the features of each classification system?" It is clear from their review that many classification systems have been developed and applied to stress fractures since Breithaupt first categorized the injury in 1855 [1]. In 42 articles and citations, 27 classification systems were described or referenced.

These systems were reviewed and analyzed for features such as being generalizable, having been evaluated for intra-observer and inter-observer reliability, whether the biologic healing potential was incorporated, and what type of evaluation was required to determine the classification. For example, some systems used a biopsy, some used only a bone scan, and others required multiple imaging techniques or mandated a specific study with computed tomography (CT) or magnetic resonance imaging (MRI). Mandating an imaging modality is fraught with issues affecting safety, expense, ease of use, and availability of the classification system for certain locations. This greatly impairs the generalizability of the classification system. If the biologic healing potential component is not included, the description of the stress fracture is incomplete. As previously discussed, not all locations in the skeleton have equal capacity to heal bony injuries. If the classification has not been statistically analyzed for intra-observer or inter-observer reliability, the validity of the evaluation is open to question.

The four most commonly referenced classification systems are those of Zwas et al., Blickenstaff-Morris et al., Devas et al., and Arendt et al. [23, 24, 27, 44]. However, since the early 1990s when MRI became commonly available, the classification system of Arendt et al. has been the most commonly referenced system. The reason for this change in frequency of reference is likely due to the increased specificity of MRI over X-ray and bone scintigraphy for diagnosing stress fractures. Arendt's system includes both MRI and bone scan and is generally considered an academic, radiological method for classification. It is commonly applied for research purposes.

With regard to validation of classification systems, 18 of the 27 referenced systems were correlated with patients' clinical outcomes. Only one of the systems, that of Arendt et al., was analyzed for inter- and intra-observer reliability [23]. The reason for this low number is likely due in large part to the time and scientific environment during which most of the classification systems were first published. Given that the majority of classification systems referenced were originally described before 1989 and, therefore, prior to the age of evidence-based medicine, it is likely that validation of systems was not a major consideration for many of the original authors.

Table 4.3 Stress fracture classification systems[a]

Systems cited	Generalizable	Site	Imaging	Clinical parameters	Other	Clinical correlation	Publication
Arendt	+		XR, BS, MRI			+	1997
Blickenstaff-Morris	–	Fem neck	XR			+	1966
Boden	+		XR	Location, Natural Hx		+	2001
Brukner	+		XR			–	1999
Chisin	+		BS			+	1987
Devas	–	Fem neck	XR			+	1965
Edwards	–	Tibia	XR, BS, MRI	Pain and duration		+	2008
Elton	+		XR			–	1968
Ernst	–	Fem neck	XR			+	1964
Floyd	+		BS, XR	Pain		–	1987
Fredericson	–	Tibia	MRI			+	1995
Fullerton-Snowdy	–	Fem neck	XR and BS			+	1988
Gaeta	–	Tibia	CT			+	2005
Griffiths	+		MRI			+	1995
Johnson	–	Fem neck	XR		Path	–	1969
Jones	+		BS			–	1988
Kiuru	+		MRI			–	2001
McBryde	+		XR			–	1975
Naval Med Ctr-SD	–	Fem neck	XR and MRI			+	1996
Romani	+		U/S	Pain		+	2000
Roub	+		BS, XR		Path	–	1979
Savoca	+		XR	Location		–	1971
Saxena	–	Navicular	CT			+	2000
Torg	–	Fifth met	XR			+	1984
Wilson	+		XR			+	1969
Yao	+		MRI			+	1998
Zwas	+		BS			+	1987

[a]Adapted from [21]

In conclusion, many classification systems currently exist for stress fractures employing various imaging modalities, but few include clinical parameters. Though many are generalizable, no general classification system that includes both radiographic and clinical parameters has been validated with inter- and intra-observer reliability analysis and clinical correlation. A gold standard classification system for grading stress fractures has not historically been available.

Kaeding–Miller Classification System [22]

As discussed previously, fatigue failure of bone occurs across a spectrum of structural failure and in areas with variable healing potential. A generalizable system to describe these injuries for clinicians has not historically been available. To be clinically relevant, any comprehensive description

Table 4.4 Kaeding–Miller stress fracture classification system[a]

Grade	Pain	Radiographic findings (CT, MRI, bone scan or X-ray)
I	–	Imaging evidence of stress fracture
		No fracture line
II	+	Imaging evidence of stress fracture
		No fracture line
III	+	Non-displaced fracture line
IV	+	Displaced Fracture (>2 mm)
V	+	Nonunion

[a]Adapted from [22]

of stress fractures must correlate with the prognosis and must affect treatment decision-making. In order to do this incorporating a description of both the extent of the fracture and its healing potential is required. Only describing the extent of the fracture is inadequate, as not knowing if the "incomplete" fracture is in a low- or high-risk location precludes prognosis and treatment recommendations. If we only mention the location, with its unique healing potential, we are limited by not knowing if the "fracture" is simply an increased number of microcracks or a complete structural failure. We refer to the extent of the fracture as the fracture grade. For an accurate discussion or study of stress fractures, the location and fracture grade diagnostic study employed must be described.

Kaeding and Miller undertook a study to develop a new classification system to determine fracture grade, which, when coupled with location, would provide a comprehensive description of a specific stress fracture [22]. The authors sought to design a system possessing the characteristics of being reproducible, generalizable, easily applied, and clinically relevant. This classification system (Table 4.4) uses three descriptors: (1) fracture grade, (2) fracture location, and (3) imaging modality used. With this information, we hypothesized that the clinically relevant characteristics of a stress fracture could be described in a reproducible, easily applied, and generalizable manner.

A key step in the development of this system was to develop a simple reproducible manner to describe fracture grade that, when coupled with location (reflecting healing potential/risk), would provide a user-friendly, clinically relevant description of a stress fracture. We believe that the proposed stress fracture classification system achieves these goals. In describing stress fractures, the size and extent of the actual fracture vary greatly, and the biologic healing potential varies among fracture locations. The variation in healing potential may be related to the specific fracture having developed characteristics of a nonunion or because the natural history of stress fracture location is favorable or unfavorable for healing. These two concepts, fracture grade and location, have important implications on treatment options and prognosis. Garbuz et al. stated that a classification system should be compared with the gold standard classification system [8]. Unfortunately, no gold standard classification system exists for describing stress fractures.

A classification system that is complex, difficult to remember, or difficult to apply is not likely to be of considerable benefit to the clinician. The Kaeding–Miller classification system is simple and easy to use, but still captures key clinical features while being widely applicable and reproducible [22]. Questions regarding prognosis and optimal treatment of a stress fracture cannot be answered without knowing its location, the extent of the structural damage, and the presence or absence of nonunion. The authors feel that each of these parameters is clinically relevant and necessary to accurately describe a stress fracture. Boden et al.'s description of high-risk and low-risk stress fractures was a great contribution to the understanding and care of stress fractures, but adding fracture grade to this concept advances our description of a stress fracture [17, 18]. Knowing that a fracture is at a high-risk location is important, but knowing whether it is a Grade II or Grade IV fracture at a high-risk location is of even greater benefit for treatment and prognosis. A low-grade stress fracture at a low-risk site has a better prognosis for time to recovery than a higher grade injury at the same low-risk site. Therefore, the management of bony stress injuries should be based on the location and grade of the injury. These two criteria provide the physician with important information when evaluating a patient with a stress fracture,

communicating with colleagues and patients, and formulating a treatment plan.

Very early fatigue failure of bone may be asymptomatic. Several authors have reported that athletes can have asymptomatic fatigue fractures. Matheson et al. studied 320 athletes with positive bone scans for stress fractures and concluded that 37 % of the lesions were asymptomatic [45]. Nussbaum et al. studied bone scans in ballet dancers and found that three of ten stress fractures and 13 of 19 stress reactions were asymptomatic [46]. Bergman et al. reported MRI evidence of tibial stress reaction in 43 % of asymptomatic distance runners [47]. Groshar et al. studied military recruits during active training with bone scintigraphy and found that 26 % of stress fractures were asymptomatic [48]. Gaeta et al. found that 17 % of distance runners had painless tibial stress reactions on high-resolution CT imaging [32].

As the microcracks propagate and coalesce, these fatigue failures of bone can progress from being asymptomatic to clinically painful. One large study by Arendt and Griffiths demonstrated that stress fractures with greater structural damage took longer to heal than lower grade injuries [19, 23]. This study demonstrated that the grade of injury has prognostic implications.

Kaeding and Miller have proposed a comprehensive descriptive system for stress fractures [22]. This includes a grading scale for classifying the extent of structural failure. Examples of each grade are shown in Figs. 4.2, 4.3, 4.4, 4.5, and 4.6. Grade I injuries are asymptomatic, usually incidental findings on imaging studies (Fig. 4.2). Grade II injuries have imaging evidence of fatigue failure of bone, but no fracture line (Fig. 4.3). Grade III injuries have a fracture line with no displacement (Fig. 4.4). Grade IV fractures are displaced (Fig. 4.5), and Grade V stress fractures are chronic having gone onto nonunion (Fig. 4.6). This system is summarized in Table 4.4. This classification system has been shown to have high inter- and intra-observer reliability [22]. Coupling this fracture grade with the location of the fracture provides a more comprehensive description of the injury that takes into account both the extent of structural failure and healing potential of the injury.

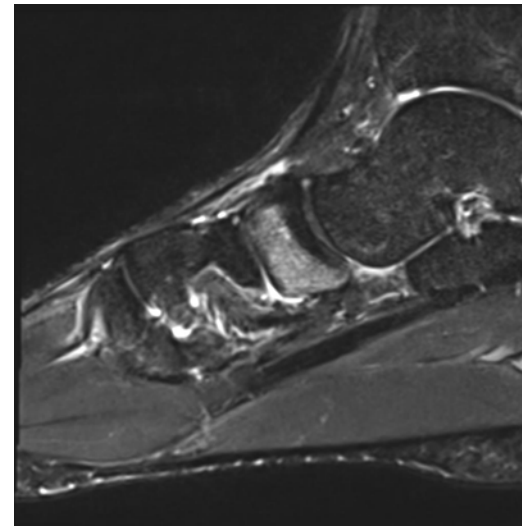

Fig. 4.2 T2 axial cut MRI demonstrating a Grade I stress fracture of the navicular in a 22-year-old male collegiate distance runner. The patient presented with pain over the third metatarsal shaft with no pain or tenderness at the navicular. A Grade II stress fracture was evident at the third metatarsal

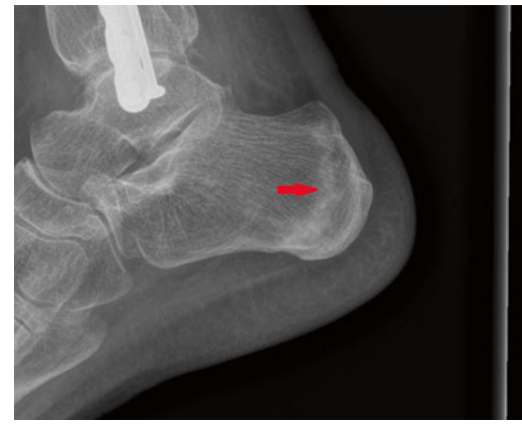

Fig. 4.3 Lateral plain radiograph of the foot in a 20-year-old female lacrosse player with progressive heel pain demonstrating a Grade II stress fracture of the posterior calcaneal body (*arrow*). The patient has undergone previous open reduction internal fixation of the fibula and medial malleolus

When reporting the stress fracture grade in this system, the imaging modality used should be reported. For example, a CT scan revealing a nondisplaced fracture line in a tarsal navicular in a healthy collegiate basketball player would be reported as a Grade III tarsal navicular stress frac-

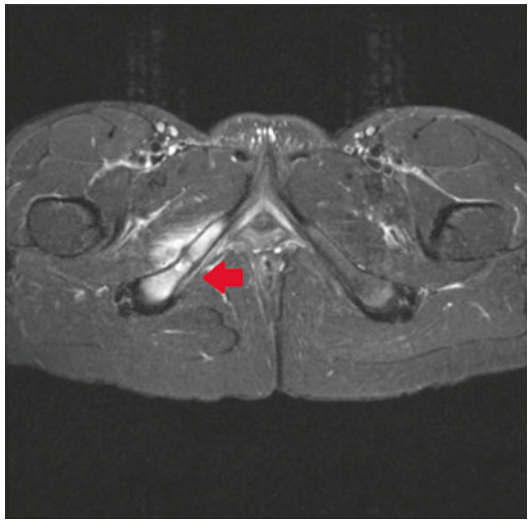

Fig. 4.4 Axial cut T2 MRI of the pelvis demonstrating a Grade III (complete non-displaced) stress fracture of the right inferior pubic ramus in a 19-year-old female collegiate distance runner with worsening right anterior pelvic pain over a 4-week period and absent menses for 5 months

ture on CT scan. The requirement to provide imaging modality gives a comprehensive description of the injury, which is both clinically relevant and useful in research to ensure comparable groups. We did not mandate a specific imaging modality for the system. To mandate that a bone scan, CT scan, or MRI be performed would result in the system being less easily applied. For example, if a fifth metatarsal proximal metaphyseal–diaphyseal fracture shows evidence of a nonunion on radiographs, mandating a bone scan or MRI adds little to the clinical description. If the clinician does not pursue imaging beyond the radiograph, the system is not, in a practical sense, generalizable to all sites. A downside to not requiring single or multiple imaging modalities is that the fracture grade in the new classification system may change depending on which modality is used. Because of this aspect of the grading system, it is required that the imaging modality used is mentioned when grading the stress fracture.

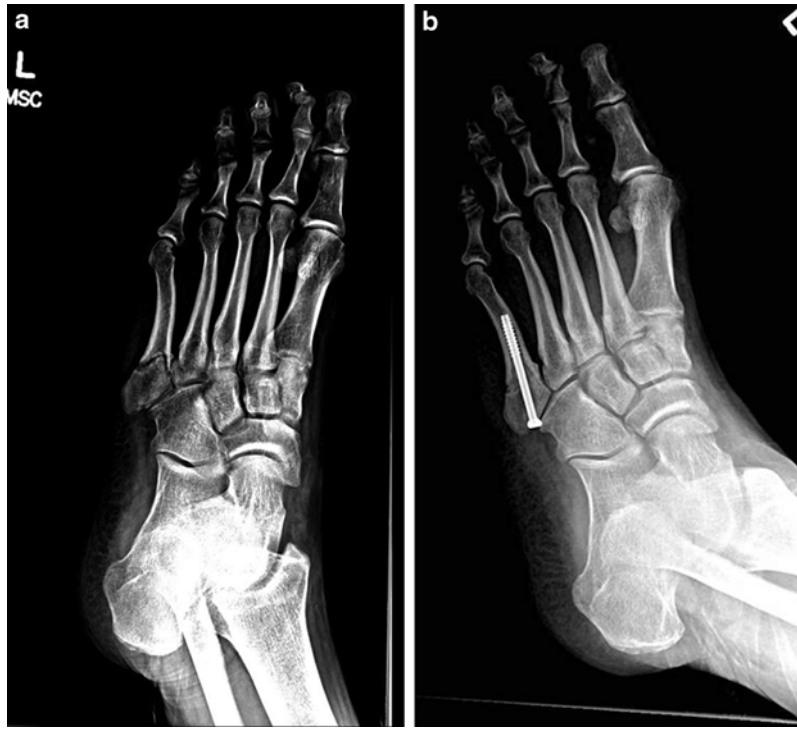

Fig. 4.5 (**a**) Oblique foot radiograph demonstrating Grade IV (displaced) fifth metatarsal stress fracture in a 21-year-old male college basketball player with worsening lateral foot pain for 5 weeks, acutely worsened with an ankle inversion injury. (**b**) Intramedullary screw fixation was required for fracture stabilization and healing of the fracture

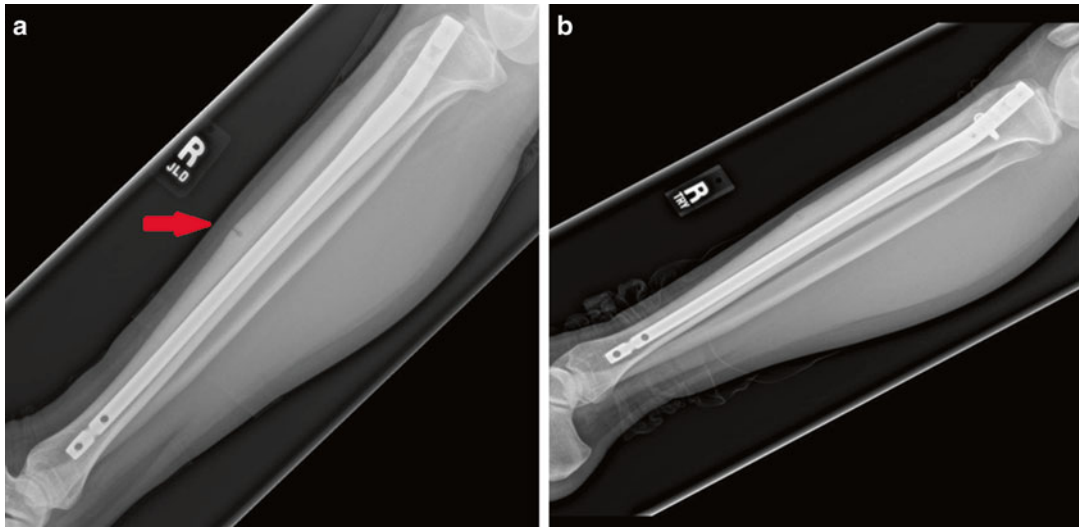

Fig. 4.6 Plain radiographs of a 19-year-old female professional ballet dancer with chronic anterior tibial pain and Grade V (nonunion/"dreaded black line" stress fracture of the anterior tibial cortex (*arrow*) (**a**). Final treatment required operative fixation with an intramedullary rod (**b**). Cortical thickening with fracture healing is evident 4 months post-surgery

Two key features of the Kaeding–Miller system are that it is generalizable and has been validated with intra- and inter-observer reliability. It is of great benefit to have a single, reproducible, and easily used grading system that describes the clinically relevant parameters throughout the body. The more concise and reproducible the classification system, the more accurate the communication between clinicians and patients who are being counseled will be. The responses of the clinicians who evaluated the system and their ability to reproduce the classification system were evidence that evaluators found the system easy to apply and understand. Almost perfect intra-observer agreement was found among 15 evaluators of the classification system which included orthopaedists, primary care sports medicine specialists, and physician assistants. Substantial-to-almost perfect inter-observer reliability was observed for the classification grades among the same evaluators. Fourteen of the 15 evaluators (93.3 %) reported that the system was easy to remember, would facilitate communication regarding stress fractures among medical colleagues, and would be used in their practice in the future. Additionally, the same group of evaluators was able to reproduce the system from memory with 97.3 % accuracy [22].

Summary

Stress fractures of the axial and appendicular skeleton are troublesome overuse injuries for athletes and non-athletes alike. This type of fractures represents a fatigue failure of bone, occurring with a spectrum of severity of structural injury with healing potential varying by location. Though many stress fracture classification systems exist in the literature, there is only one comprehensive classification system for stress fractures incorporating both clinical and radiographic characteristics of the injury that is applicable to all bones. Though many are generalizable, only the Kaeding–Miller classification system has been validated for inter- and intra-observer reliability.

We have described an easy-to-use and readily applied classification system that incorporates clinically relevant parameters and is generalizable to all stress fractures. It has been validated for intra-observer and inter-observer reliability.

This classification system describes the clinically relevant characteristics of a stress fracture in a reproducible manner, enhancing the description, communication, and research of stress fractures. A gold standard classification system for grading stress fractures is yet to be determined. As with any system an ideal technique of classifying stress fractures is reproducible, generalizable, easy to use, and clinically relevant, with four key descriptors: fracture location, risk assessment, fracture grade, and imaging modality used to make the diagnosis.

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Imaging of Stress Fractures

5

Jason E. Payne and Joseph S. Yu

Introduction

Athletes are particularly prone to injuries that are related to overuse. In the general athletic population, the incidence of stress fractures is about 1 % but may vary according to activity, for instance, up to 20 % in runners [1, 2]. The location where a stress fracture develops also is specific to a particular sporting activity [3, 4]. It is reported that 60 % of athletes presenting with a stress fracture have experienced a prior stress fracture [5]. In the osseous tissues, overuse injuries produce stress-induced changes that may alter the architecture of the bone. Stress is defined as any force or absolute load that is applied to a bone. These forces arise from having to bear unusual weight or repetitive load, or are created when there is an imbalance of muscles [6–8]. Wolff's Law dictates that a change in the mechanical environment of a bone from new or intermittent stress elicits the remodeling of the osseous architecture of that bone to adjust to its new environment [9]. Increases in muscular strength often precede strengthening of the bone, and this can create an imbalance between the relative strength of these tissues. Furthermore, when muscles fatigue during exercise, the protective effect of muscle tension diminishes reducing the ability of bone to resist stress.

A stress fracture represents unsuccessful adaptation by a bone under duress [10, 11]. Stress fractures are generally divided into two categories. *Fatigue* stress fractures occur when normal bone is subjected to repetitive stresses that lead to mechanical failure as a consequence of inadequate remodeling of microfractures. An example of this occurs when an athlete abruptly changes a training regimen, not allowing sufficient time for bone to remodel in response to the added stress. *Insufficiency* fractures occur when normal stresses are applied to an abnormal or pathologic bone that is incapable of adaptation. Fatigue stress fractures related to overuse are relatively common in certain groups, particularly athletes and military personnel [12, 13]. The incidence of stress fractures among females in the military tends to be higher than in men, but this difference has not been consistently observed in athletes [14–17]. The most common pathologic bone abnormality in older athletes that increases the risk for stress fractures is osteoporosis, with the highest reported prevalence occurring in postmenopausal women [18, 19]. A variety of other conditions associated with abnormal underlying bone also predispose an athlete to insufficiency

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fractures including rheumatoid arthritis, corticosteroid use, and diabetes mellitus [20, 21].

Forces resulting in osseous injury can be classified as compression, tension, and/or shear. It is useful to consider these forces when assessing the morphologic properties of a stress fracture. For instance, distant runners tend to develop stress fractures in the posteromedial aspect of the tibia owing to repetitive compressive forces whereas dancers and jumping athletes tend to develop tibial stress fractures in the anterior tibial shaft due to tensile forces.

Evolution of Imaging

The imaging appearances of stress-induced injuries change over time and the rate of change is affected by factors such as the bone involved, location of injury, inciting activity, and age [22]. The sensitivity of radiography for early diagnosis of stress fractures is low because forces tend to distribute along long segments of the cortex producing subtle changes at the surface of the bone and the periosteum [13, 18]. This early phase is referred to collectively as a stress response or stress reaction. If the cyclic loading continues, progressive deformation of the bony architecture localizes to a focal weakened area of the bone resulting in a uni-cortical break in the cortex, or a true stress fracture. Athletes who develop fatigue fractures often exhibit the following triad: a new or different activity has been introduced in their training, the activity is strenuous, and the activity is repeated cyclically. In a stress reaction, there is still active healing of the microfractures but in a stress fracture, the progressive forces ultimately exceed the elastic range of the bone leading to structural failure.

Stress fractures account for at least 10 % of patients encountered in a typical sports medicine practice [1]. Imaging has traditionally provided diagnostic support for evaluation of these patients with modalities depicting variable sensitivity and specificity according to the stage along the continuum of a stress injury [23]. Radiography continues to be a low-cost frontline technique but is limited by a lack of sensitivity especially early in the process. The first effective modality to have an impact on the diagnosis of osseous stress injuries was whole body bone scintigraphy utilizing technetium-99m-methylene diphosphonate (Tc-99m-MDP). Stress fractures are visible on bone scans days to weeks earlier than radiographs. For many years, it served as the gold standard for early confirmation of stress-induced changes related to increased bone metabolism and osteoclastic activity. The limitation of bone scintigraphy was that it lacked specificity in areas that ordinarily resulted in an increase in radiopharmaceutical uptake, however, the advent of triple-phase scanning with additional angiographic and blood pool phases contributed to improved specificity [24].

Although computed tomography (CT) has shown superior spatial resolution in comparison to other imaging modalities, its role in evaluating patients with stress fractures continues to be limited. Recently, however, utilization of multidetector CT has increased due to the ability to depict the stress fracture line in coronal and sagittal high-resolution multiplanar-reconstructed CT images as well as 3D volume rendered images [25]. This has increased the utilization of CT for differentiation of stress fractures from other entities such as osteoid osteoma which may have similar radiographic appearances. Ultrasound also has a limited role in the diagnosis of stress-related injuries although it has the ability to assess the superficial cortical surface in bones close to the skin as well as fracture lines, periosteal reactive changes including callus formation, edema in periosseous tissues, and increased perfusion [26, 27].

Most recently, magnetic resonance imaging (MRI) has been shown to be extremely sensitive to the pathophysiologic changes that are associated with stress-induced conditions and provides greater specificity than radionuclide imaging owing to its superior spatial resolution [28, 29]. MRI has been efficacious in characterizing early changes of stress injuries with high sensitivity and specificity to local hyperemia and edema, periostitis, bone marrow changes, and cortical failure and is considered the current gold standard [30]. It also has been useful in estimating clinical severity, guiding therapy, and estimating the duration of disability [31].

Imaging Techniques

Radiography

The initial workup of an athlete with pain should begin with a radiographic evaluation of the area involved. Because the initial imaging features of a stress fracture are subtle, radiographs should be done with precision (optimal positioning) and in a comprehensive manner, i.e., with all required projections, and accompanied with a proper history. Accuracy is increased when the radiographs are optimized and a reliable search strategy is employed [32]. A common approach is to critically evaluate the integrity of the cortex for changes in density (Fig. 5.1), as well as for periosteal reactive and endosteal reactive changes (Fig. 5.2). The medullary cavity should be assessed for the presence of impacted trabeculation and linear uni-cortically based sclerotic bands. Other findings include transverse or longitudinal breaks in the cortex (Fig. 5.3) as well as trabecular angulation and distraction which may be a manifestation of progression (Fig. 5.4). Altered cortical morphology which may be either focal thickening or thinning is usually an indication of a chronic condition.

It is important to realize that the location and orientation of developing stress fractures influence the radiographic appearance so that fractures at the ends of tubular bones tend to depict linear areas of sclerosis whereas fractures in the shaft of a tubular bone may be simply a lucent cortical break or focal periostitis [33]. Longitudinal stress fractures have the appearance of a thickened cortex with a vertically oriented lucency in the cortex (Fig. 5.6). In bones composed largely of cancellous bones such as the tarsus and femoral neck, the first sign of a stress fracture may be simply focal linear sclerosis (Fig. 5.7). In these cases, initial findings are subtle blurring of the trabecula secondary to microfractures. As healing of the microfractures progresses, linear sclerosis

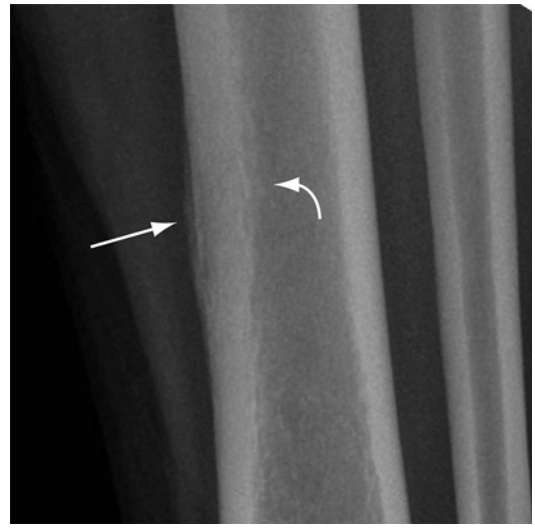

Fig. 5.2 Chronic radiographic findings of stress response. Close up of radiograph of the mid-tibia shows mature periosteal (*arrow*) and endosteal reactive changes (*curved arrow*) associated with focal areas of osteopenia in the cortex

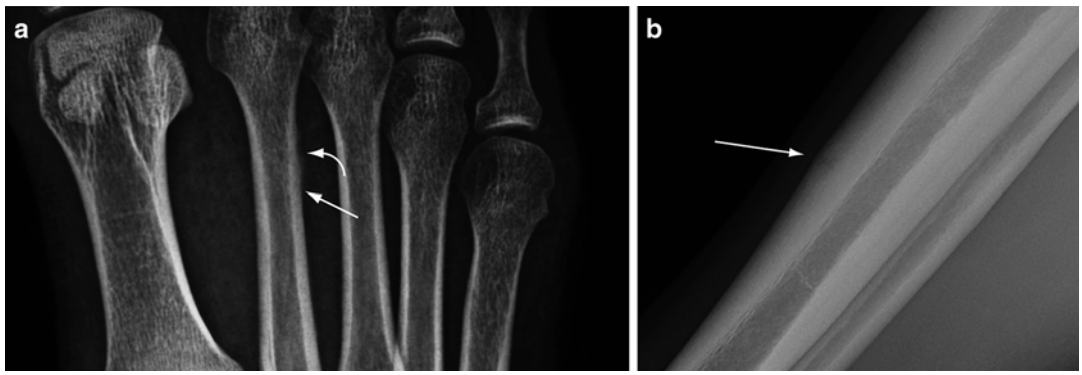

Fig. 5.1 Early stress response on radiography. (**a**) Frontal radiograph of the forefoot shows focal osteopenia of the lateral cortex of the distal second metatarsal shaft (*white arrow*) and periostitis (*curved arrow*). (**b**) Lateral radiograph of the tibia shows focal cortical osteopenia (*arrow*)

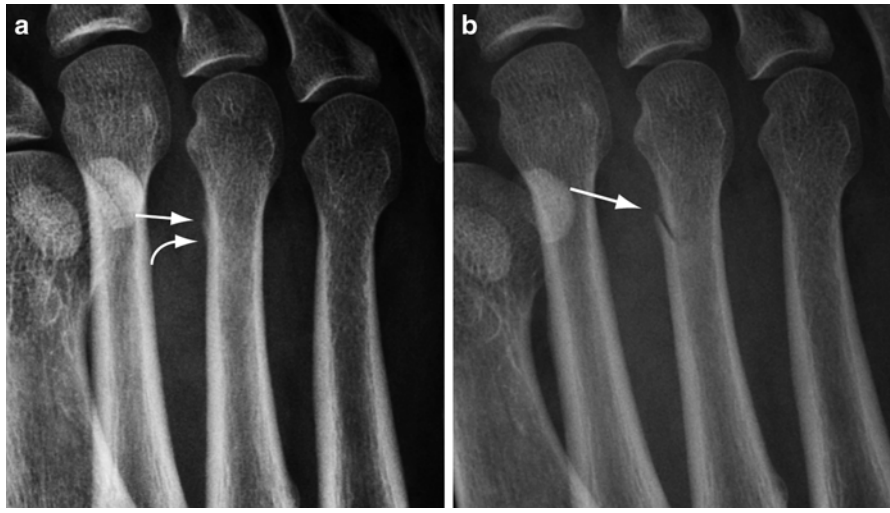

Fig. 5.3 Radiography of early stress fracture. (**a**) Oblique radiograph of the forefoot shows periostitis of the medial cortex of the third metatarsal shaft (*arrow*) and a subtle lucency (*curved arrow*) representing the start of a break in the cortex. (**b**) Follow-up image in 3 weeks shows completion of the cortical fracture with oblique lucency (*arrow*)

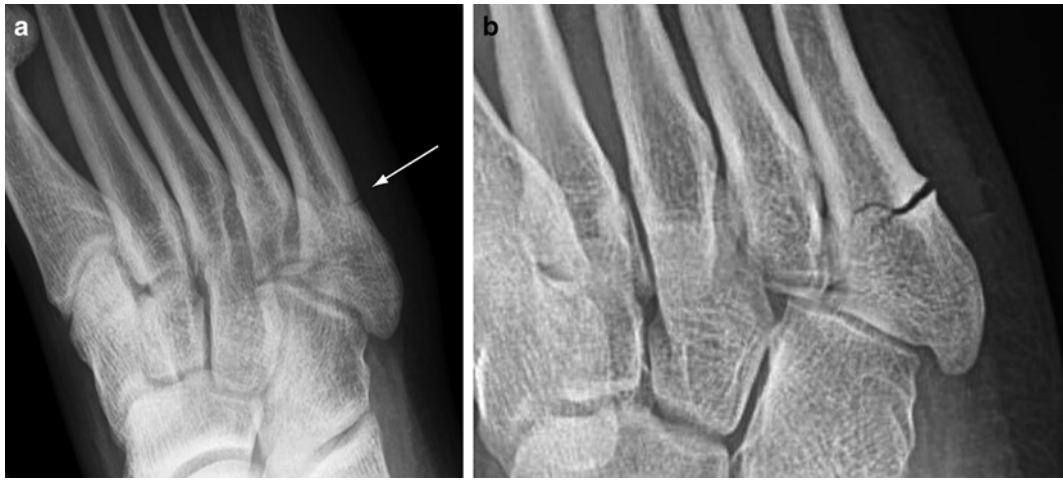

Fig. 5.4 Stress fracture progression. (**a**) Oblique forefoot radiograph in one patient shows a classic Jones stress fracture involving the lateral cortex of the proximal fifth metatarsal shaft (*arrow*). The fracture was isolated to the cortex. (**b**) Another patient with a Jones stress fracture shows extension into the medullary cavity after the athlete felt a "pop" while running

appears oriented perpendicular to the course of the trabecular with extension to one cortical surface. Radiographic detectable changes usually become conspicuous weeks to months after the onset of symptoms and the timing and nature of the changes varies with the level of activity. However, it is noteworthy that imaging findings may not be necessarily sequential.

The sensitivity of radiographs for early stress fractures is as low as 15 % and follow-up radiographs may demonstrate findings in only 50–54 % of cases [7, 34]. Development of subsequent radiographic findings is often determined by whether there is cessation of the inciting stress that is affecting the bone. Prior studies comparing radiography to bone scintigraphy have reported a

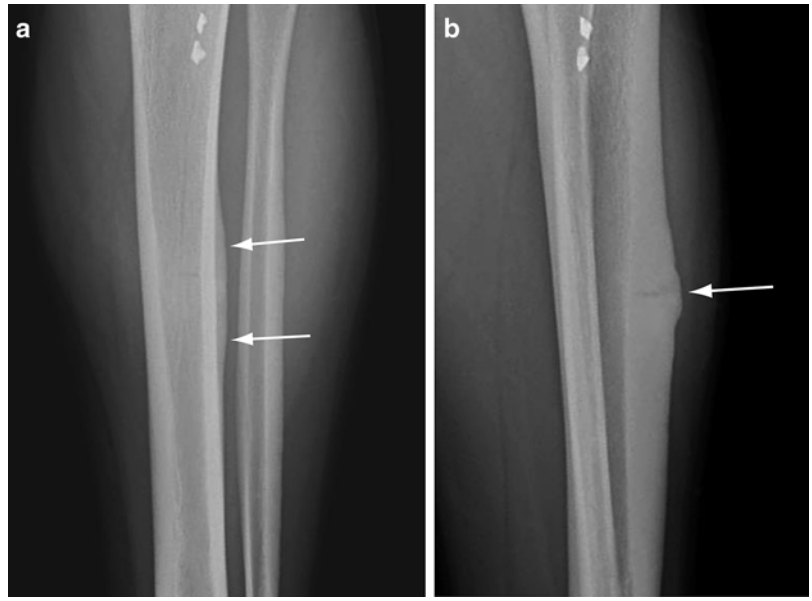

Fig. 5.5 Striated stress fracture. (**a**) Frontal radiograph of the tibia shows periosteal elevation along the anterolateral cortex of the mid-tibia (*arrows*). (**b**) Lateral view shows a transverse lucency through the cortex with more pronounced periosteal reaction directly adjacent to the fracture (*arrow*)

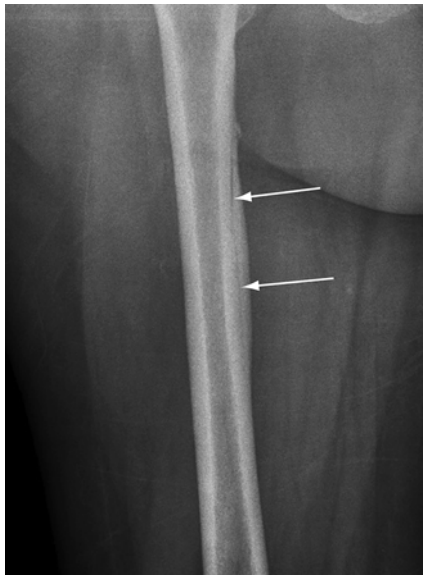

Fig. 5.6 Radiography of longitudinal stress fracture. Frontal radiograph of the femur shows a linear lucency (*arrows*) within the medial femoral cortex oriented along the longitudinal axis of the bone

sensitivity of 56 %, a specificity of 94 %, an accuracy of 67 %, positive predictive value of 95 %, and a negative predictive value of 48 % [28]. There are many classifications available for grading the radiographic features of stress fractures but currently none have been ubiquitously utilized [35, 36].

Tomosynthesis, or digital radiography, has recently been shown to be superior to conventional radiography in detection of occult fractures and it may have an application in the evaluation of stress fractures [37]. This new imaging technique can depict both cortical as well as trabecular changes, so its performance is considered only slightly lower to that of CT but at lower radiation exposure [38].

The differential diagnosis for stress fracture on radiography is limited particularly as specificity of the study increases in the chronic phase of the fracture. Chronic osteomyelitis may present with periosteal and endosteal reactive changes resulting in cortical thickening but clinically, these two entities are not at all similar. Occasionally, a stress fracture may mimic a tumor [39]. Osteoid osteoma may result in cortical thickening and reactive bone formation and is often encountered in a similar patient population as stress fracture. The presence of a central lucent nidus as well as a less linear pattern of sclerosis and clinical history can aid in differentiation.

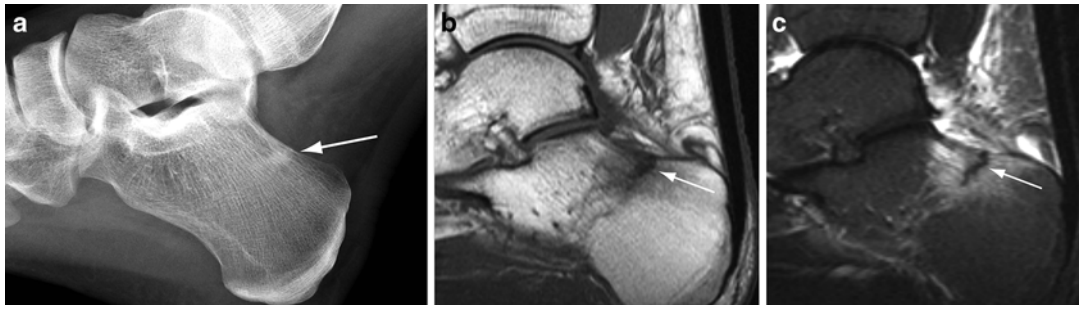

Fig. 5.7 Stress fracture in cancellous bone. (**a**) Lateral radiograph of the calcaneus demonstrates a linear area of sclerosis perpendicular to the trabeculation in the superior calcaneus (*arrow*). Sagittal T1-weighted (**b**) and STIR (**c**) MR images show a uni-cortical, low-signal fracture line (*arrows*) surrounded by intense bone marrow edema

Radionuclide Scintigraphy

Bone scintigraphy had for many years been regarded as the gold standard for evaluating stress-induced injuries and although recently supplanted by MRI, it continues to be widely utilized in many situations. It measures bone response to injury by depicting areas of increased osseous metabolism through the localization of radionuclide tracers, particularly Tc-99m-MDP. The degree of uptake depends on the rate of bone turnover and local blood flow, and abnormal uptake may be seen within 6–72 h of injury [7, 34, 40]. Whole body bone scans can be performed with relatively low cost and have the advantage of being able to image the entire skeletal system at once, which is useful in cases when more than one area is symptomatic. The sensitivity of bone scintigraphy is nearly 100 % [7].

The specificity of bone scintigraphy, however, is limited by any process that increases blood flow and has osteogenic activity such as arthritis, infection, malignancy, infarctions, and metabolic conditions. The specificity can be improved by performing a three-phase study [24]. The first phase includes a dynamic flow study with images obtained at 1 s intervals for 60 s after the injection of radiopharmaceutical and is followed by a static "blood pool" image (second phase) obtained a few minutes later. These phases depict vascularity and soft tissue involvement, respectively. The third phase is the standard 2- to 4-h delayed images depicting the osteoblastic response. An acute stress fracture will be positive in all three phases while a chronic stress fracture tends to show activity only on the delayed images [7]. Another limitation of scintigraphy in patients with stress fractures is that the scintigraphic abnormality may take 4–6 months to resolve rendering the modality inadequate for sequential follow-up studies [41]. Several grading schemes are available to characterize the severity of a stress fracture according to its scintigraphic features.

The characteristic scintigraphic appearance of a stress fracture in delayed static images is intense, fusiform cortical uptake along the long axis of the bone at the level of the fracture (Fig. 5.8) [42]. However, there can be a wide spectrum of findings representative of the pathophysiologic continuum of the process and the variations in the orientation of the fracture such as in a longitudinal fracture (Fig. 5.9). A stress reaction is manifested by an area of less intense radionuclide uptake along the cortex corresponding to areas of remodeling bone during the period that radiographs are typically normal.

Athletes who are involved in rigorous training regimens may present with multiple symptomatic regions of bone that show abnormal radionuclide uptake, and these findings have been shown to represent both stress reaction and frank stress fractures. However, some patients also depict abnormal uptake in regions of bone that are not symptomatic. This likely represents the earliest manifestation of bone remodeling

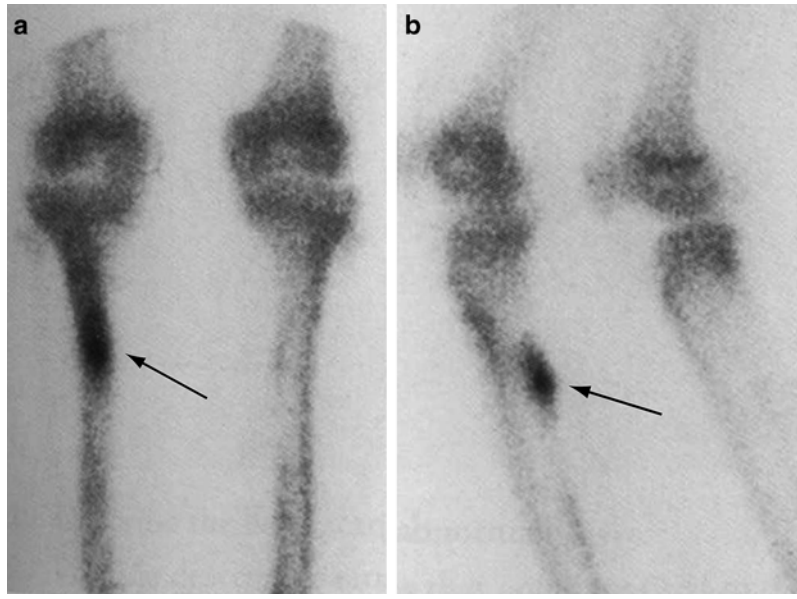

Fig. 5.8 Typical scintigraphic findings of a stress fracture. Delayed static images of the tibia from a whole body bone scan utilizing Tc-99m-MDP in the frontal (**a**) and oblique (**b**) projections show a characteristic appearance of a stress fracture in the tibia depicted as a fusiform region of radionuclide uptake oriented along the long axis of the bone (*arrows*)

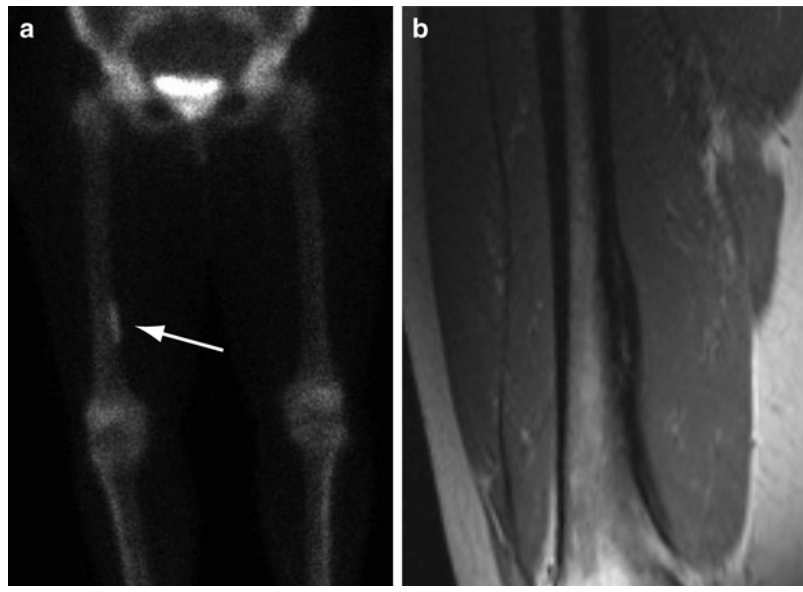

Fig. 5.9 Longitudinal stress fracture on scintigraphy. (**a**) Delayed frontal static bone scan image utilizing Tc-99m-MDP shows a thin, linear area of increased activity in the medial cortex of the distal right femoral shaft (*arrow*). (**b**) Coronal T1-weighted MR image shows a longitudinal stress fracture depicted as a linear area of intermediate signal intensity within the thickened cortex aligned to the axis of the bone

[43]. The asymptomatic foci have been reported in as high as 46 % of subjects in one series [44]. With continued activity, these may progress to symptomatic stress injuries.

The application of planar scintigraphy in combination with single-photon emission computed tomography (SPECT) has been recently advocated for increasing the accuracy of grading stress fractures. In a recent study evaluating patients with known femoral neck stress fractures diagnosed with MR imaging, the sensitivity of planar scintigraphy alone was reported to be 50 % while the sensitivity for planar scintigraphy in combination with SPECT increased to 92 % [45]. Similarly, the accuracy for scintigraphy alone was 12.5 % but increased to 70 % when SPECT was added. SPECT has also been shown to improve the diagnostic accuracy of stress fractures at the pars interarticularis region of the spine, a process that is commonly observed in adolescent athletes with back pain. SPECT has been shown to provide more detailed anatomic depiction of the region in comparison to MRI and higher sensitivity in comparison to planar scintigraphy alone [46–48]. However, SPECT is limited in the spine owing to a high rate of false positives and false negatives [49].

Ultrasound

Sonography has a very limited role in the evaluation of stress fractures and is not recommended as a stand-alone study [50]. However, studies have shown that this modality may occasionally be used to assess the superficial surface of the cortex in bones that are located close to the skin such as in the ankle/feet and tibia [51]. Cortical irregularities such as periostitis and callus formation can be depicted as well as muscular edema around the bone, and compression of the probe is useful in confirming pain. Color Doppler imaging can demonstrate areas of hyperperfusion at and near the stress fracture.

Recent studies have demonstrated a sensitivity of 82 % and a specificity of 67–76 %, but predictive values offer a wide range with studies reporting a 59–99 % positive predictive value and a 14–92 % negative predictive value [26, 52].

Computed Tomography

The role of CT in the assessment of stress-related injuries continues to be relatively limited despite advances in technology. CT is less sensitive than both MRI and nuclear scintigraphy in depicting the early changes of bone remodeling from repetitive stress [7, 29, 31]. However, the ability to produce thin-section, multiplanar-reconstructed images in order to provide high resolution and detailed depiction of cortical bone does relegate CT to an important adjunctive role when the imaging features in other modalities are equivocal [53]. CT is clearly superior to both sonography and conventional radiography. The earliest finding of a stress injury on CT is focal cortical osteopenia, but this is not a common observation because CT is typically not a first-line study (Fig. 5.10). CT manifestations that are distinctive of stress injuries, however, include thickening of the cortex, periosteal reactive changes, intramedullary sclerosis, and longitudinal and transverse lucent fracture lines. The main limitation of CT is that these findings may not develop until the patient has been symptomatic for several weeks. However, high-resolution CT is currently the most sensitive modality for detecting subtle cystic

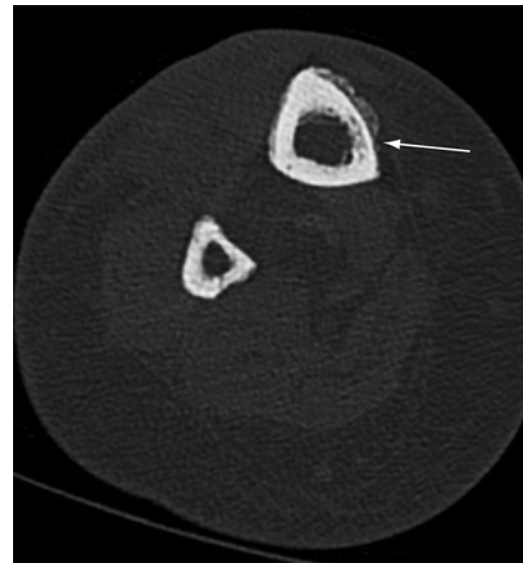

Fig. 5.10 Computed tomography, early stress response. Axial CT image of the tibia shows focal osteopenia in the cortex of the bone (*arrow*) where it is undergoing stress-induced changes

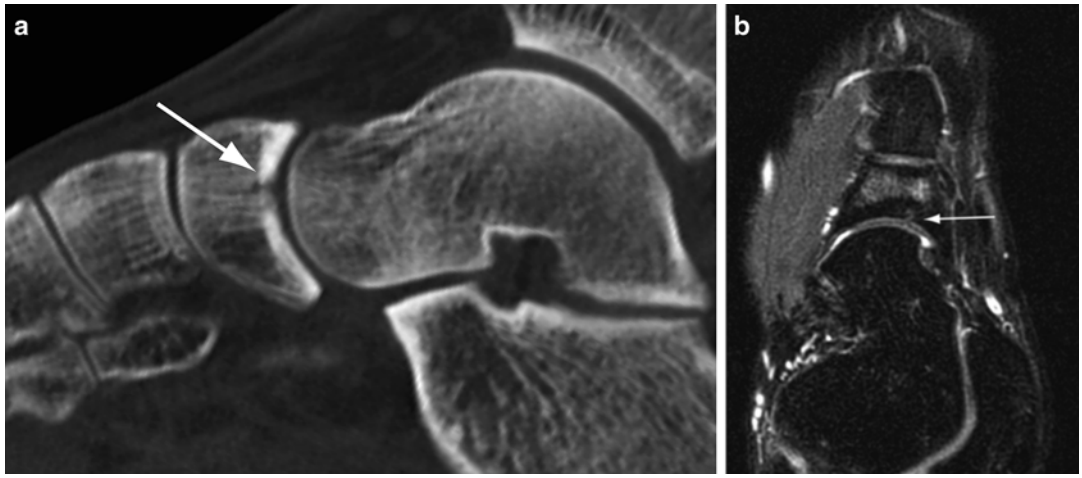

Fig. 5.11 Cortical resorption cavity. (**a**) Sagittal multiplanar CT image of the foot shows focal osteopenia in the cortex at the point of the fracture (*arrow*) indicating a developing cortical resorption cavity. (**b**) Axial STIR MR image shows the cystic defect in the cortex (*arrow*) and bone marrow edema in the medullary space

changes in the cortex that characterize cortical resorption cavities (Fig. 5.11). Once a fracture line in the cortex develops, the defect is easily demonstrated by conventional axial images as well as by multiplanar reformatted or 3D volume rendered images (Fig. 5.12) [54].

CT is advantageous in certain situations over other imaging techniques. It is useful in differentiating healing from progression (Figs. 5.13 and 5.14). Certain location-specific conditions are better suited for CT. Stress fractures that affect the tarsal navicular are often difficult to diagnose because the symptoms associated with this condition are often vague and there may be a paucity of specific physical findings [55]. Additionally, the overall density of the navicular can, in part, obscure the linear focus of sclerosis that accompanies a stress fracture on radiography. In these cases, CT is useful in elucidating the imaging characteristics of the stress fracture such as the extent of abnormality, orientation, and if there are indicators of avascular necrosis. A similar challenge may occur in patients with pars interarticularis fractures. Fracture lines are often difficult to visualize utilizing other modalities such as MRI but are clearly illustrated on CT [49, 56]. Occasionally, cortical thickening may be a nonspecific finding. For instance, the radiographic manifestation of an osteoid osteoma may mimic those of a stress fracture because both conditions thicken the cortex and are associated with variable periosteal reactive changes. By utilizing thin-section CT images, these entities can be reliably differentiated by the identification of the lucent nidus that is the classic feature of osteoid osteoma within the region of cortical thickening and sclerosis [57]. The power of CT over MRI is in its ability to penetrate the high-attenuation cortical bone. Although MRI remains the single best method for evaluating early stress injuries, it is relatively insensitive to changes that occur only within the cortex. Therefore, the subset of cortical stress injuries that are characterized by osteopenia, resorption cavities, and striations are better suited for evaluation by CT [58]. Longitudinal stress fractures of the tibia caused by repetitive torsional loading in runners are another subset of fractures that are best evaluated with CT. The longitudinal orientation and extension of the fracture negates the effectiveness of radiographs and though MRI is capable of depicting the abnormality, CT has been reported to be more sensitive in identifying the fracture line itself [59].

Peripheral quantitative computed tomography (pQCT) is a CT technique that has demonstrated potential in the evaluation of stress fractures by the acquisition of high-resolution images of the extremities at lower radiation doses than with conventional CT. The pQCT

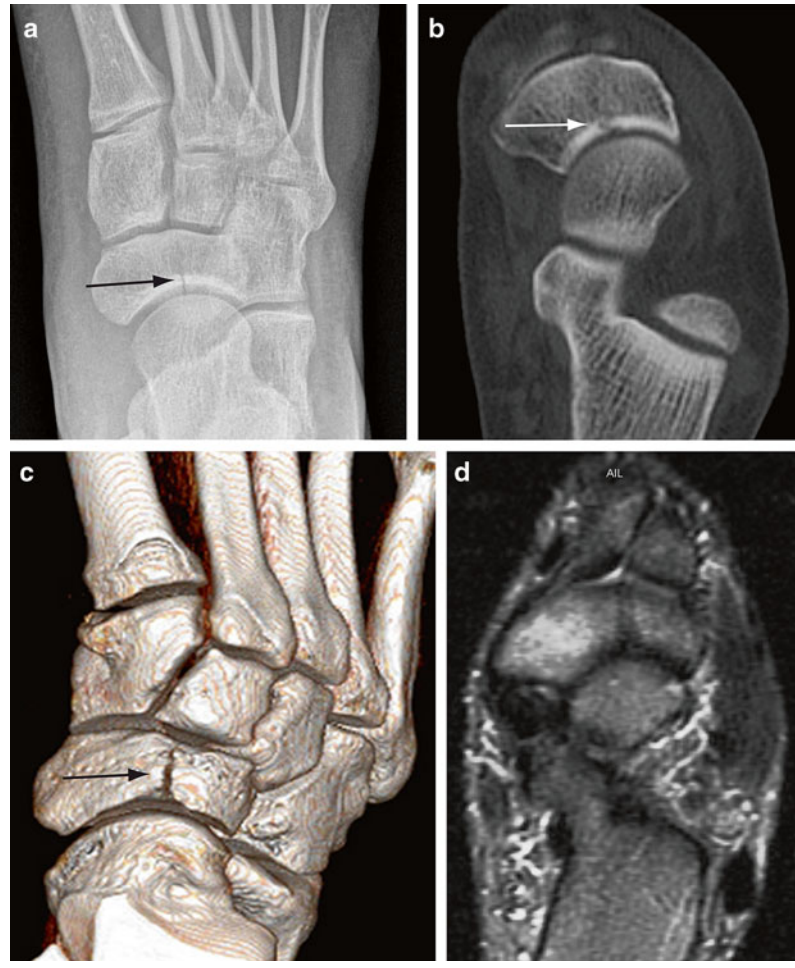

Fig. 5.12 Subacute navicular stress fracture. (**a**) Frontal foot radiograph of a college basketball player shows a vertical lucency (*arrow*) in the lateral aspect of the navicular. (**b**) Axial multiplanar CT image confirms the stress fracture (*arrow*) as well as normal bone density throughout the tarsal bone. (**c**) 3D volume rendered CT image depicts the entire stress fracture (*arrow*) in one image. (**d**) T2-weighted MR image demonstrates bone marrow edema in the medial and lateral bone fragments

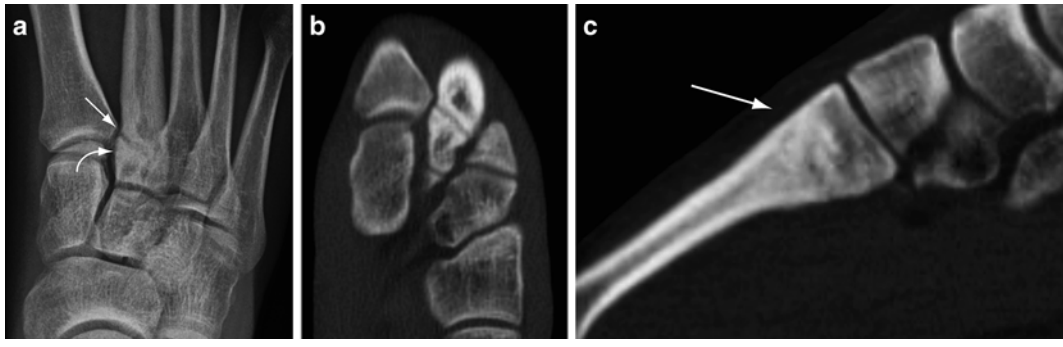

Fig. 5.13 Chronic stress fracture. (**a**) Frontal radiograph of the foot demonstrates a transverse lucency near the base of the second metatarsal bone (*arrow*). Sclerosis adjacent to the fracture is evident (*curved arrow*). Axial (**b**) and sagittal (**c**) multiplanar CT images more optimally characterize the stress fracture and also shows that the dorsal cortex is intact (*arrow*)

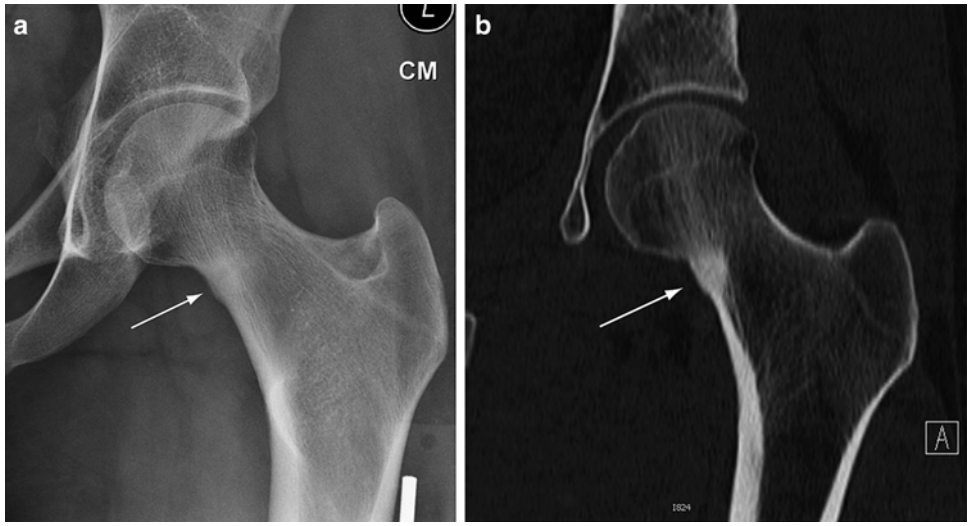

Fig. 5.14 Computed tomography of healing stress fracture. (**a**) Frontal radiograph of the hip shows a region of sclerosis on the compressive side of the femoral neck with focal periosteal reactive changes (*arrow*). (**b**) Coronal multiplanar CT image shows that the fracture line has nearly filled in and is no longer evident (*arrow*)

images afford detailed portrayal of the structure and mineralization of bone at the location of the stress fracture. As such, it may have application in monitoring the stress fracture throughout the healing phase [60, 61].

Magnetic Resonance Imaging

MRI is currently the gold standard for diagnosing and classifying stress-induced injuries. Several important features of this imaging modality have contributed to its emergence as a superior tool for assessing these conditions including unparalleled contrast, outstanding spatial resolution particularly with higher strength magnets, and the capability to image in an infinite number of geometric planes [62]. Additionally, it does not utilize ionizing radiation which is ideal in the athletic population who tend to be younger [63]. MR images, in general, can be obtained in a shorter period of time than with a scintigraphic examination, and provides images that are exquisitively sensitivity to the subtle changes seen in patients with early stress fractures. Numerous studies have shown that MRI outperforms radiography, CT, and radionuclide scanning [28, 29, 31, 64, 65].

MRI examinations are optimized by utilizing dedicated coils which serve to increase the signal-to-noise ratio and decrease artifacts. Higher strength magnets, such as 3-T systems which are becoming more commonplace, offer higher spatial and contrast resolution, shorter scanning times, and improved conspicuity of bone marrow edema than conventional 1.5-T systems [66]. The sensitivity is comparable for both MR systems and routinely, 1.5 T MR images are typically adequate for diagnosis and characterization of stress fractures [67, 68]. Typical sequences applied include short tau inversion recovery (STIR), which is commonly used in screening since it has the highest sensitivity to edema, and fast spin-echo sequences with fat-saturation which are excellent in preserving high spatial resolution. A T1-weighted sequence is generally prescribed to further characterize the inherent signal intensity of lipoid marrow. Intravenous gadolinium is not frequently administered in the evaluation of stress fracture. However, dynamic enhancement has been reported in patients with higher grade stress reactions and stress fractures caused by increased tissue perfusion. This may be useful in cases where the pre-contrast MR images show a callus, fracture, or muscle edema,

and in situations where there is a concomitant malignancy or infection [69].

MRI is an effective diagnostic technique in patients who show strong clinical manifestations of a stress fracture but have normal initial radiographs [70, 71]. Like scintigraphy, MRI depicts changes in the bone and periosteum weeks before any radiographic abnormality develops. The early stages of a stress fracture are characterized by focal hyperemia and bone marrow edema that correlates with the development of microfractures and osseous resorption. Endosteal reactive changes, periostitis, and periosseous edema are important early observations on STIR or T2-weighted spin-echo images, and are characteristic of stress reactions (Fig. 5.15) [65, 72]. Edema appears bright in signal intensity on these sequences. Focal periosteal elevation develops as the process becomes more severe (Fig. 5.16). As the injury progresses and becomes more severe, marrow edema appears on T1-weighted images as areas of low-signal intensity (Fig. 5.17). As breakdown of the cortical bone progresses, a frank stress fracture forms either transversely or longitudinally (Figs. 5.18 and 5.19) [64]. The most common patterns of a fatigue stress fracture on MRI are a linear, uni-cortically-based abnormality of low-signal intensity surrounded by a larger, ill-defined region of marrow edema, or a linear cortical abnormality with adjacent muscular or soft tissue edema [73–75]. Callus formation indicates a more chronic stress fracture. The MRI features in the continuum of a developing stress fracture parallel to those that are observed on bone scintigraphy.

Reportedly, MRI has comparable sensitivity to nuclear scintigraphy. Specificity, accuracy, positive predictive value, and negative predictive value are all superior at 100 %, 90 %, 100 %, and 62 %, respectively [29]. Additionally, MRI has a distinct advantage by depicting the surrounding soft tissue structures thus permitting concomitant evaluation of muscular, tendinous, or ligamentous structures. In the athletic population,

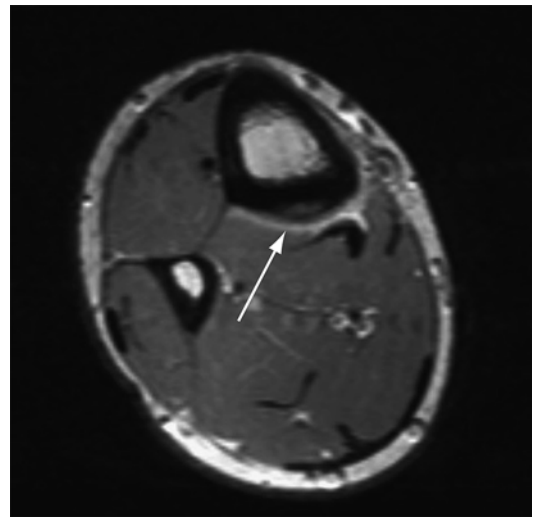

Fig. 5.16 MR of chronic stress reaction. Axial proton-density MR image shows periosteal elevation in the posterior cortex of the tibia (*arrow*) and adjacent inflammation

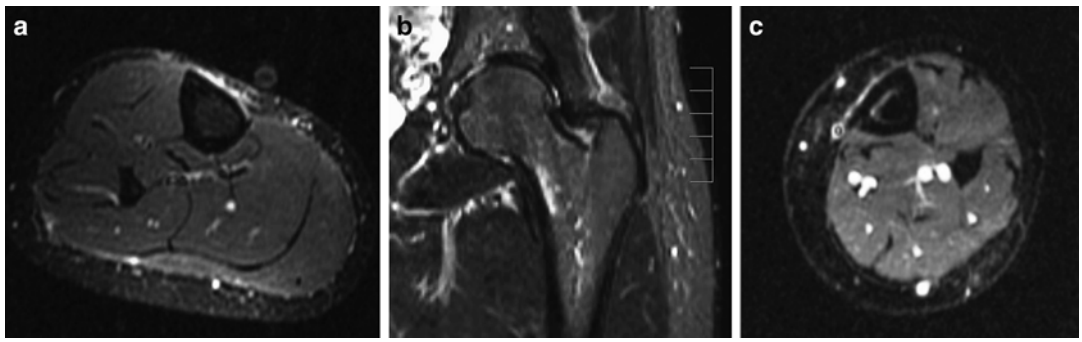

Fig. 5.15 Different stress responses on MR imaging. Fluid-sensitive MR images in three different athletes. (**a**) Periostitis along the medial cortex of the tibia manifests as linear high-signal intensity along the outer cortex. (**b**) Endosteal reaction with marrow edema along the endosteal surface of the femoral neck. (**c**) A patient with a more severe stress response shows both periosteal and endosteal reactive changes

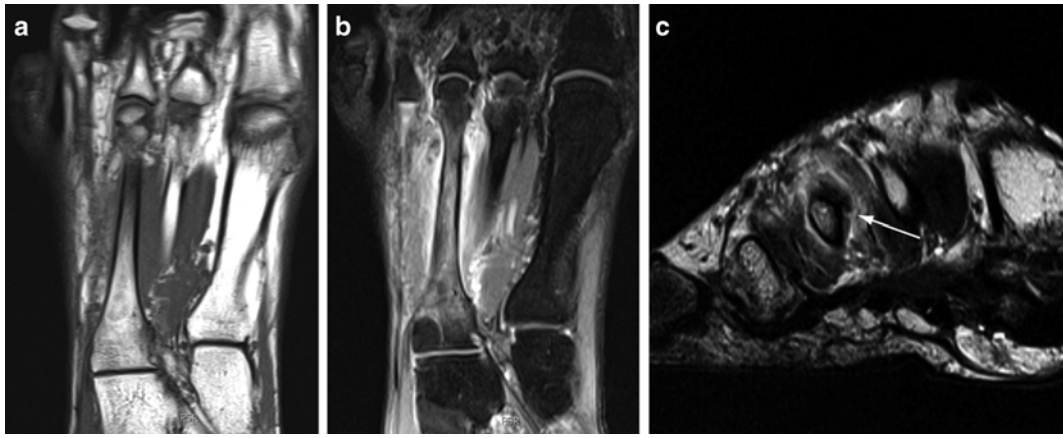

Fig. 5.17 MR features of developing stress fracture. (**a**) Axial T1-weighted MR image shows low-signal intensity bone marrow in the third metatarsal bone from edema. (**b**) Corresponding T2-weighted image shows adjacent periosteal inflammation shown as linear high-signal intensity fluid along the cortex on both sides of the bone. (**c**) There is rupture of the medial periosteum (*arrow*) and edema in the surrounding interosseous muscle

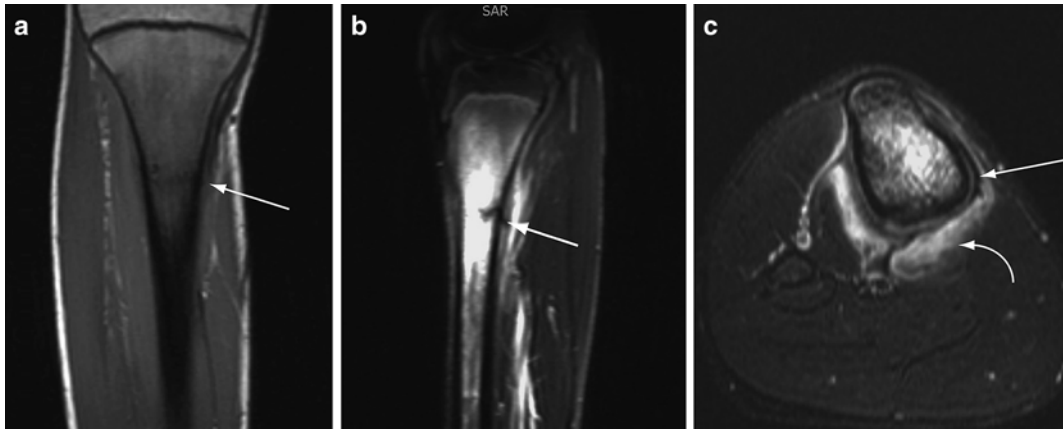

Fig. 5.18 Typical stress fracture on MR imaging. Coronal T1-weighted (**a**) and sagittal STIR (**b**) images of the tibia show marrow edema and periostitis as well as edema in the adjacent posterior soft tissues. The transverse stress fracture is low in signal on both sequences (*arrows*) and surrounded by a larger region of marrow edema. (**c**) Axial fluid-sensitive image demonstrates extensive periosteal elevation (*white arrow*) and periosseous soft tissue edema (*curved arrow*)

injuries to any of these structures may mimic the symptoms of a stress fracture, which are sources that reduce the specificity of nuclear scintigraphic studies. Another feature of MRI that should be underscored is its ability to assess regions of the skeleton that are challenging with other imaging modalities. For instance, insufficiency fractures of the pelvis, proximal femur, and superior acetabulum in elderly patients are often difficult to visualize on CT studies but unequivocally demonstrated on MR images (Fig. 5.20) [76, 77]. Femoral neck stress fractures that are optimally shown on MRI may be occult by radiography or scintigraphy. Any delay in diagnosis of these stress fractures increases the potential for completion of the fracture. Lastly, the anatomic detail of stress fractures afforded by MRI allows distinction between different types of stress fractures, such as compressive and tensile type stress fractures of the femoral neck, the latter requiring operative fixation [70, 78].

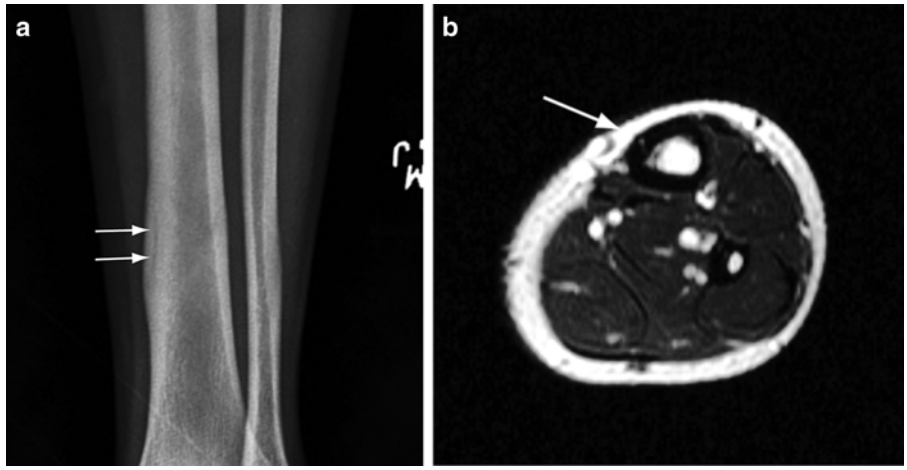

Fig. 5.19 Longitudinal stress fracture. (**a**) Frontal radiograph of the tibia shows a linear lucency within the thickened medial cortex (*arrows*) in the distal shaft. (**b**) Axial T2-weighted MR imaging shows the vertically oriented break in the cortex (*arrow*)

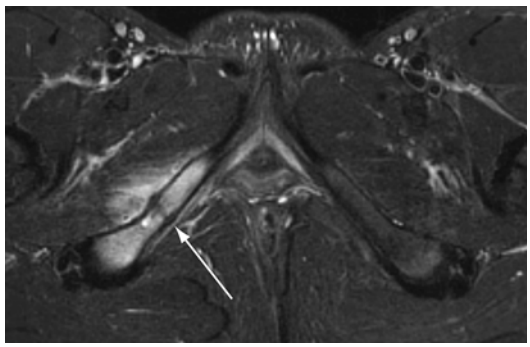

Fig. 5.20 Pubic ramus stress fracture. Axial STIR image demonstrates a stress fracture of the right inferior pubic ramus (*arrow*) with intense surrounding marrow edema and periosseous edema. The abnormality was radiographically occult due to the oblique orientation of the pubic bone

The majority of proposed grading systems have been for stress injuries of the tibia [41, 79]. Many of the classifications attempt to correlate clinical and imaging findings to those on nuclear scintigraphy but an exact correlation has not been reported to date. Owing to superior spatial and contrast resolution, grading systems that are based on MR findings have shown superior accuracy over other classifications, thus improving the prescription of appropriate clinical management. Also because the clinical impact of varying MR or scintigraphic grades often has no influence on an athlete's ability to return to active participation, some investigators have suggested simplifying the grading systems to reflect findings that have a strong clinical correlation such as the presence of a cortical fracture [29, 58, 80]. For instance, unless a fracture line is present, patients with MR grades ranging from grade 2 to 4a who show variable severity of periostitis and bone marrow edema may be theoretically combined into one grade since the time that the athlete is not permitted to play is similar among these grades, while the development of a fracture, a grade 4b abnormality, requires a prolonged period away from athletic participation and constitutes a more severe grade [80].

The appearances of stress fractures on MRI can occasionally overlap with those of benign and malignant processes [75]. The linear orientation of a stress fracture when it is present helps to differentiate it from the more fusiform cortical thickening that may be observed in a patient with a neoplastic process, or the serpiginous intramedullary appearance that is characteristic of osseous infarctions. In-phase and out-of-phase images utilize the differences in the interaction of water and lipid protons in the magnetic field to assess for the presence of fat and water in areas of bone marrow. Stress fractures and other nonneoplastic processes preserve the fat content of normal

marrow whereas neoplastic processes tend to result in replacement of the fat [81]. Other advanced imaging techniques such as chemical shift imaging, diffusion-weighted imaging, and MR spectroscopy are also available for further tissue characterization when it is required.

The primary limitation of MRI is the cost as it is one of the most expensive imaging techniques available. Utilization must be performed precisely and accurately. False-negative examinations may occur in the setting of technical error such as heterogeneous fat-saturation and partial volume effects, interpretive error, or protocol error by inappropriately selecting the wrong MR sequences. The sensitivity of MRI to edema may result in an errant positive finding if a patient is asymptomatic, so it is important to interpret an examination with proper history and with available correlation to pertinent physical findings [82–84].

Summary

Radiography remains the initial imaging examination in a patient suspected of having a stress fracture. A number of options are available for further evaluation depending on the phase of the injury but most experts agree that MRI is now the gold standard owing to its superior spatial and contrast resolution, high sensitivity, and specificity to both early and late findings, and the lack of ionizing radiation. When available, MRI should be the next modality employed.

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Part II

Management of Stress Fractures

Stress Fractures of the Lumbar Spine

6

Matthew J. Grierson, Rebecca A. Speckman, Mark A. Harrast, and Stanley A. Herring

Introduction

Spondylolysis is a condition involving the pars interarticularis (pars), located in the posterior elements of lumbar vertebrae. Early clinical manifestations of pathology at the pars may involve only a stress response, but this can later progress to bony discontinuity and, eventually, spondylolisthesis, the translation of one vertebra upon another. In 1976, Wiltse, Newman, and Macnab proposed a classification system for spondylolysis and spondylolisthesis based on etiology: (I) dysplastic, (II) isthmic, (III) degenerative, (IV) traumatic, or (V) pathologic [1]. An isthmic spondylolisthesis (type II) is the term used for vertebral slippage specifically due to a pars defect.

Clinicians should consider spondylolysis high on the differential any time an athlete presents for evaluation of low back pain. Approaches to diagnosis and treatment vary throughout the literature. The dissonance underscores the importance in understanding the epidemiology, pathophysiology, and natural history of spondylolysis as well as the logic behind individual decision pathways. Given the fact that spondylolysis is encountered frequently in asymptomatic populations, multiple radiologic studies may be necessary to assist the clinician in developing a rational strategy to correlate diagnostic findings with a patient's symptoms, severity of injury, and goals of care [2]. This chapter reviews the science behind available diagnostic and treatment options for isthmic spondylolysis and discusses reasonable clinical algorithms that sports medicine providers may consider when helping an athlete maximize return-to-play goals with long-term functional outcomes.

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Epidemiology

Early epidemiologic studies relied on plain radiographs to establish a diagnosis of spondylolysis, and likely underestimated its population prevalence compared to work utilizing more sensitive imaging techniques (see section "Diagnostic Imaging"). Among Caucasians, the prevalence of a spondylolysis has been estimated to be about 3–6 % [3–5]. Males are affected about 2–3 times more often than females, and defects occur most often at L5 compared to other levels (79–95 %) [2–14]. Lesions are frequently bilateral, and there appears to be a strong association with spina bifida occulta [2, 4, 5, 7, 12, 15–20].

Population-based studies suggest that spondylolysis is an acquired condition that occurs early in life, associated only with bipedal ambulation. It is not found in infants [4, 21–25] or non-ambulatory populations [26, 27]. In the 1950s, Daniel Baker, M.D., conducted a landmark population-based study on the prevalence of spondylolysis in 500 first-graders from a single geographic cachement [4]. The point prevalence of spondylolysis in children aged 6 years old was 4.4 % using 2-view lumbar plain radiographs for detection. By early adulthood, the cumulative incidence of spondylolysis had only reached 6 %.

Several other cohorts have reported similar findings. In a study of 4,200 cadaveric adult spines, the overall prevalence of spondylolysis was 4.2 % and this value did not change significantly between age groups from 20 to 80 years old [5]. Another study evaluating a Finnish cohort of 554 subjects found MRI evidence of spondylolysis in 6 % of young adults aged 18–21 [28]. There was no correlation between findings of spondylolysis and patient-reported low back pain or functional limitations, underscoring the notion that although lesions are common, many are not symptomatic.

Athletes

Investigations in elite or adolescent athletes with low back pain have shown a higher prevalence. In one cohort, clinicians found evidence for active spondylolysis in 55 % of athletes aged 10–30 who presented for evaluation of low back pain [29]. In another group, 47 % of adolescent athletes presenting with low back pain were found to have a spondylolysis [30, 31], compared to only 5 % of adults presenting with similar findings.

Soler and Calderon studied the occurrence of spondylolysis in 3,152 elite Spanish athletes (mean age: 20.6 ± 6.7 years) using posteroanterior and lateral radiographs [11]. The study population included all athletes who underwent medical checkups at the National Center of Sports Medicine in Madrid, Spain, between April 1988 and May 1997, irrespective of low back pain symptoms. Among this cohort, the investigators found an overall point prevalence of 8.0 %, but noted considerable variability by sport. Disproportionate prevalence was found among track and field throwing sports (26.7 %), heptathlon and decathlon (12.9 %), bobsledding (20 %), boxing (14.3 %), judo and wrestling (11.2 %), fencing (10.7 %), gymnastics (14.0 % overall; 17.0 % for artistic gymnastics, 9.8 % for rhythmic gymnastics, and 16.1 % for trampoline), rowing (16.9 %), swimming (10.2 %), volleyball (10.0 %), and weightlifting (12.9 %). In this study, 46.4 % of athletes with spondylolysis had at some time mentioned low back pain, compared to 23.5 % of the athletes without spondylolysis ($P < 0.01$). Other studies have confirmed high rates of radiographic evidence of spondylolysis in gymnasts [32–34], swimmers [35], football players [36, 37], and other athletes [38–40]. Fortunately, although radiographic evidence of isthmic defects are common, asymptomatic athletes who present with these findings are able to maintain intensive athletic training programs for many years without developing symptoms (see section "Outcomes") [13].

Anatomy, Biomechanics, and Pathogenesis

Spondylolysis is a defect in the pars interarticularis, or the isthmus between the superior and inferior articular facets (Fig. 6.1). It lies directly between the lamina and the pedicle, corresponding

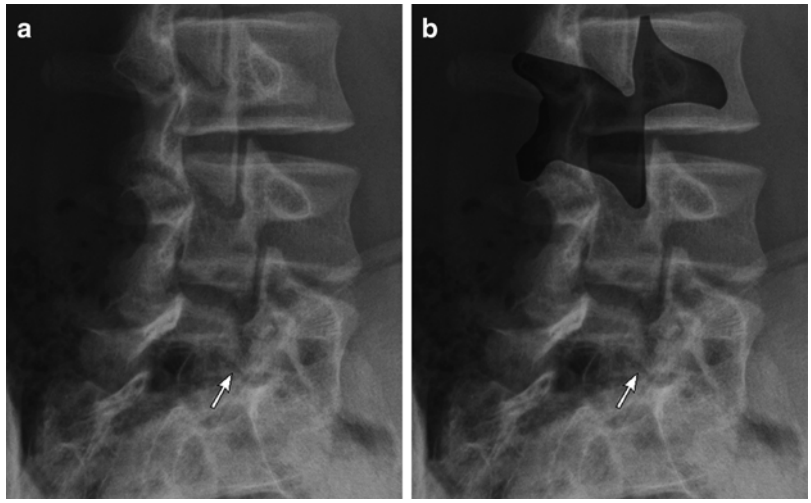

Fig. 6.1 Oblique lumbar radiographs demonstrating spondylolysis (*arrows*). (**a**) The pars interarticularis is the "isthmus" located between the superior and inferior articular facets and between the lamina and pedicle. (**b**) This corresponds with the neck of the "Scotty dog" seen on oblique radiographs

with the neck of the traditional "Scotty dog" outlined on oblique lumbar radiographs (see Fig. 6.1). The most accepted theory of pathogenesis reflects the hypothesis that repetitive physical stresses to an area weakened by a variable combination of congenital, genetic, and biomechanical anatomic conditions will eventually lead to structural failure [41, 42]. Bilateral pars defects effectively divide the vertebra into anterior and posterior components, which then predisposes to translation of one vertebra on another (i.e., spondylolisthesis).

The mechanism for a pars injury involves repetitive extension or rotation of the lumbar spine, which causes shearing forces to develop between the inferior articular process of the level above and the superior articular process of the level below. Capener first described this "bony pincer" mechanism in 1931 [41, 43], which is consistent with the higher prevalence of spondylolysis seen in athletes whose discipline includes repeated lumbar extension [11]. Investigators have subsequently studied specific lumbosacral factors that predispose to an injury in this region, finding that greater anterior inclination angles of the lumbosacral endplates and increased degrees of lumbar lordosis correlate both with increased rates of pars fractures at L5 compared to age-matched controls and decreased rates of union following conservative management [17, 44]. Chung et al. also found that a smaller inter-facet distance from L4 to S1 and a greater lordosis at L5 to S1 are associated with L5 spondylolysis in young patients [45].

Congeni has described three subpopulations that account for the variable biomechanical factors that can predispose to injury [46, 47]. Type I is a hyperlordotic female athlete with increased range of motion and flexibility, such as a dancer or gymnast. Type II is a muscular male athlete with decreased hamstring and erector spinae flexibility who is undergoing a rapid growth spurt, such as football players and weightlifters. Type III is an athlete new to a sport or activity and undergoing a newly increased amount of training, often in conjunction with poor core strength and trunk flexibility. Understanding the factors that predispose to injury is important and can potentially influence the success of a rehabilitation treatment program.

History and Physical Exam

There have been few studies that objectively assess the clinical presentation of symptomatic spondylolysis, and the vast majority of patients

Fig. 6.2 One-legged hyperextension test. The examiner asks the athlete to raise one leg and lean backward. It is positive if it reproduces the patient's concordant symptoms of low back pain [29]

who have radiographic evidence of a pars defect developed the condition without symptoms [4, 41]. Many authors describe the typical presentation as focal low back pain, with occasional radiation into the buttock or proximal lower limb [31, 38, 48–51]. Onset may be insidious or after an acute injury, and athletes with low-grade symptoms may experience more severe symptoms after a period of heavy exertional stress [52, 53].

Studies to determine the diagnostic accuracy of physical examination maneuvers are also scarce. Findings frequently described as characteristic of spondylolysis include exaggerated lumbar hyperlordosis, ipsilateral paraspinal muscle spasm, tight hamstrings, pain with extension, and tenderness to palpation over the site of the pars lesion [11, 47, 49, 51, 53, 54]. Neurologic examination in isolated spondylolysis should be normal, and other diagnoses should be considered when patients present with radicular symptoms.

Many clinicians have cited the one-legged hyperextension test as pathognomonic for spondylolysis [49, 50, 53, 54]. This maneuver is positive when the athlete leans backward while standing on one leg if the motion reproduces his or her concordant pain (Fig. 6.2). Masci et al. studied the diagnostic accuracy of this test in young, active subjects with low back pain, but found no association of a positive one-legged hyperextension test with the presence of active spondylolysis diagnosed by single-photon-emission computed tomography (SPECT) (followed by CT, if positive) and MRI [29]. Thirty-nine of the 71 subjects (54.9 %) were found to have spondylolysis. Sensitivity and specificity for pars lesions ranged from 50 to 55 % and 46 to 68 %, respectively, depending on side. Moreover, the positive likelihood ratios ranged from 1.01 to 1.54 and the negative likelihood ratios ranged from 0.98 to 1.01, suggesting that

the test has limited ability to alter pretest probability. Given the high prevalence of spondylolysis in adolescent athletes presenting with low back pain, it is imperative for clinicians to maintain a high index of suspicion for this condition any time a young athlete presents with low back pain.

Diagnostic Imaging

Establishing evidence of a symptomatic spondylolysis poses several diagnostic challenges. Many radiologic findings are common in asymptomatic individuals, underscoring the importance of correlating an athlete's symptoms with positive imaging results. The clinician must consider not only the sensitivity and specificity of any test ordered but also the risk of cumulative radiation exposure. There have been no direct comparisons of clinical outcomes and treatment based on diagnostic imaging choice [2]; thus, a thoughtful approach is necessary to optimize treatment and return-to-play decisions based on diagnosis and stage of injury.

Radiographs

The oblique course of the pars interarticularis relative to the vertebral column limits the sensitivity of routine radiographs in visualizing an abnormality of this region (Fig. 6.3). A defect is described as a lucency or discontinuity involving the collar of the "Scotty dog" seen on lateral oblique radiographs (see Fig. 6.1). Diagnostic accuracy depends on both the orientation of the fracture line and the degree of cortical disruption [55, 56]. Early studies found that clinicians were not able to view 12–20 % of fractures when using anteroposterior (AP) and lateral views alone. This led many to suggest that oblique views were necessary to adequately visualize an isthmic defect [3, 57, 58].

However, more recent work has challenged the diagnostic value of oblique radiographs, and in 2013, Beck et al. demonstrated no statistical difference between the sensitivity of 2-view and 4-view studies in detecting spondylolysis (Table 6.1) [59]. Considering the fact that only one-third of pars lesions are aligned within 15° of

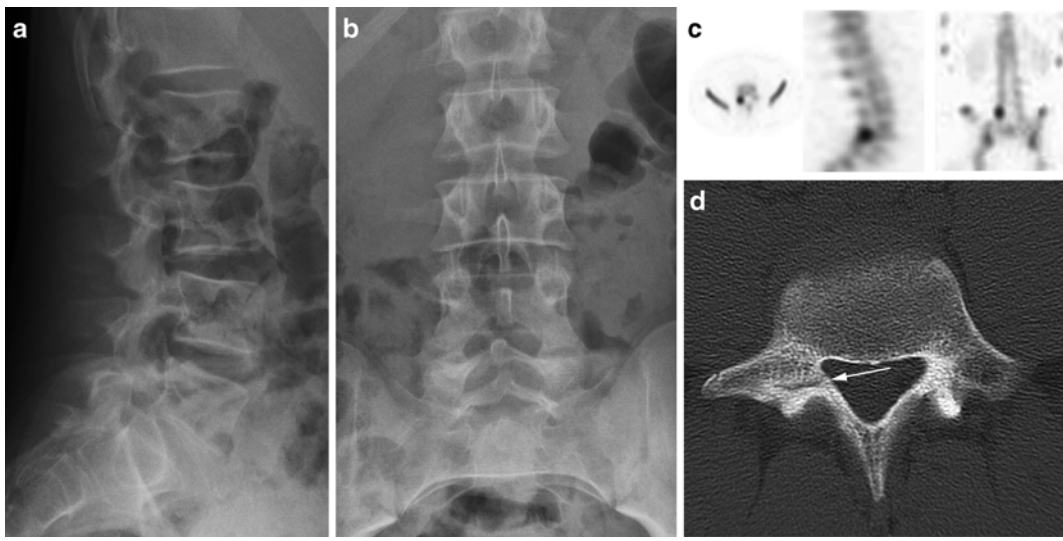

Fig. 6.3 Images taken from a 14-year-old young male soccer player diagnosed with active spondylolysis after 3 months of progressive low back pain. Although initial imaging demonstrated normal (**a**) AP and (**b**) lateral radiographs, the diagnosis was later established by (**c**) SPECT (axial, sagittal, and coronal views shown) and (**d**) CT (the *arrow* points to an early pars defect)

Table 6.1 Radiation dose and diagnostic value of 2-view or 4-view lumbar radiographs for spondylolysis [59]

Modality	Radiation dose (mSv)	Sensitivity	Specificity
2 view: AP/lateral	0.72	0.59	0.96
4 view: AP/lateral, bilateral oblique	1.26	0.53	0.94

Table 6.2 Radiation dose of lumbar imaging modalities [59, 64]

Modality	Dose
CT lumbar spine	~6 mSv
Fine-cut 2-level lumbar CT	1.2–1.4 mSv
Bone scan with SPECT	5–7 mSv
MRI	None

the lateral oblique plane [56], this newer data is not surprising. The additional cost and risk of ionizing radiation exposure involved in obtaining oblique views (increased by 75 %, see Table 6.1) likely overshadow any routine diagnostic benefit [59].

Bone Scintigraphy

As noted before, pars defects are common. They have an estimated prevalence of 4–6 % within the general population by age 18 and 8–26 % of elite adolescent athletes [4, 11, 60]. Thus, the sports physician must discern not only whether a lesion is present but also whether it correlates with an athlete's symptoms. Treating an inactive non-union could lead to delays in definitive treatment and eventual return to sports. Nuclear medicine studies, such as bone scans and SPECT, are sensitive in detecting the metabolic bone activity associated with a symptomatic lesion. They can detect an early stress reaction and offer prognostic information about whether or not a defect is likely to respond to treatment [61].

The three-dimensional nature of SPECT favors its use over simple bone scans by helping to localize an abnormality to the posterior elements of the spine. Unfortunately, although SPECT can detect early lesions prior to evidence of an abnormality on CT or MRI [29, 62], it exposes the athlete to ionizing radiation and lacks the specificity to justify its use in isolation. Thus, although SPECT represents the most sensitive diagnostic standard in detecting a symptomatic spondylolysis, other imaging modalities are needed to grade the injury and differentiate it from other less common diagnoses, such as tumor or infection [63].

Computed Tomography (CT) and Staging

For symptomatic lesions identified on SPECT, fine-cut CT (1 mm slices) scans provide the necessary osseous and cortical detail to accurately stage spondylolysis [63]. Generally, because of added radiation (see Table 6.2) [64], attention is turned to only 1–2 levels based on metabolic activity seen on prior scintographic analysis. Unfortunately, CT scans are not as sensitive as SPECT, and up to 20 % of pars abnormalities seen on SPECT are not demonstrated on thin slice CT [62]. These lesions likely represent early stress reactions [63], which are important to detect as they warrant special treatment considerations to facilitate bony healing and limit progression to a true fracture.

Morita et al. first described a classification system of three categories for pars lesions based on CT characteristics: early, progressive, and terminal [65] (Fig. 6.4). These categories accounted for the severity of injury, degree of cortical discontinuity, as well as the chronicity of the lesion. "Early" lesions involve a small fissure or a hairline defect through the pars. "Progressive" pars defects are wider and associated with occasional small fragments. Sclerotic cortical changes characterize "terminal" lesions, which are also known as pseudoarthrosis.

Grading the severity of a pars stress injury becomes particularly important when discussing prognosis or return to play, as early stress injuries heal more readily than progressive fractures and certainly more than terminal ones which rarely, if ever, heal [17, 66]. This was demonstrated by Fujii et al., who examined 239 pars defects and found evidence for bony union after treatment of 62 % of early stage lesions, compared to 8.7 % of

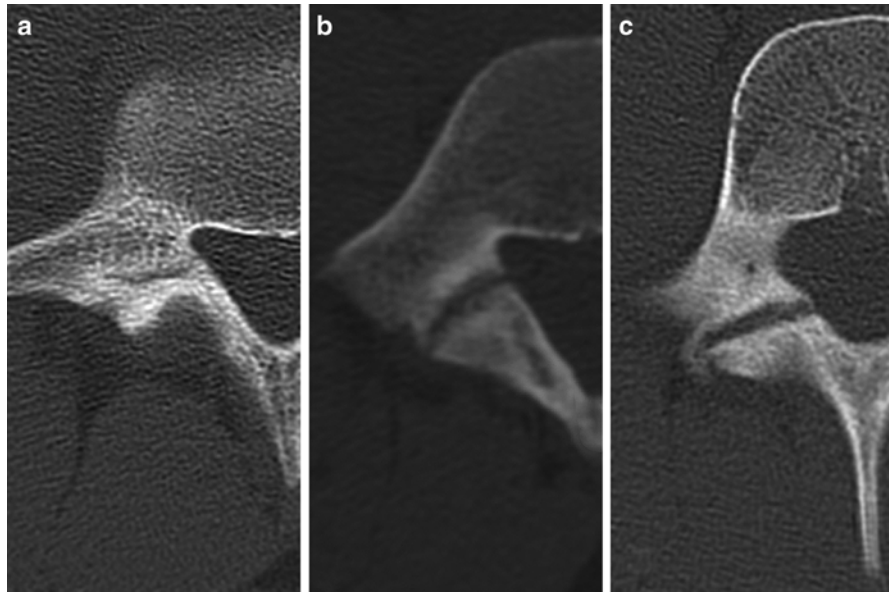

Fig. 6.4 CT demonstrating early, terminal, and progressive right-sided spondylolysis. (**a**) Early lesions involve a small fissure or hairline defect through the pars. (**b**) Progressive pars defects are wider and associated with occasional small fragments. (**c**) Sclerotic cortical changes characterize terminal lesions or pseudoarthrosis [65]

progressive lesions and 0 % of terminal lesions [17]. Sairyo et al. later attempted to correlate both CT and MRI findings with clinical outcomes and found that in a study of 68 pars defects, early lesions (86 %) or progressive lesions with high T2 MRI signal changes involving the adjacent pedicle (60 %) were the most likely to heal [66]. Similar to previous findings, none of the terminal lesions developed evidence of bony union.

Magnetic Resonance Imaging (MRI)

Given concerns over radiation exposure from SPECT and CT (see Table 6.2), there will be increasing pressure to optimize and incorporate MRI protocols and techniques. Although cortical detail is sacrificed compared to CT, MRI is excellent at visualizing soft tissues when considering alternate diagnoses. MRI may play a role in staging spondylolysis, particularly when T1 and T2 changes suggest edema in the area of the pars or pedicle [66]. But not all bony defects are visible, and the sensitivity of MRI greatly depends on image slice thickness, spacing, and orientation.

One recent case series of 74 athletes found MRI missed the diagnosis in 64 % of patients who had been diagnosed by CT [67], but Campbell et al. were the first to directly compare MRI, CT, and SPECT [68]. Using SPECT with CT as the diagnostic standard, they established that although MRI was able to detect 39/40 (98 %) of the pars defects seen, it correctly graded only 29 (72 %) of the lesions. The greatest discrepancy involved earlier stage injuries, or those with the greatest potential for healing [17].

Unfortunately, the study by Campbell et al. utilized nonstandard image sequences (oblique sagittal images), limiting the ability to generalize the results. Studies of more standard sequences reveal that MRI likely only detects around 80 % of pars lesions seen on SPECT [29]. As work emerges examining the utility of stronger magnets (3 T) and improved imaging protocols, perhaps MRI will one day emerge as the new "gold standard" in diagnosing early stage lesions. But until these protocols are adopted as standard practice, the discerning clinician should exercise caution when interpreting data or basing treatment decisions solely on MRI findings.

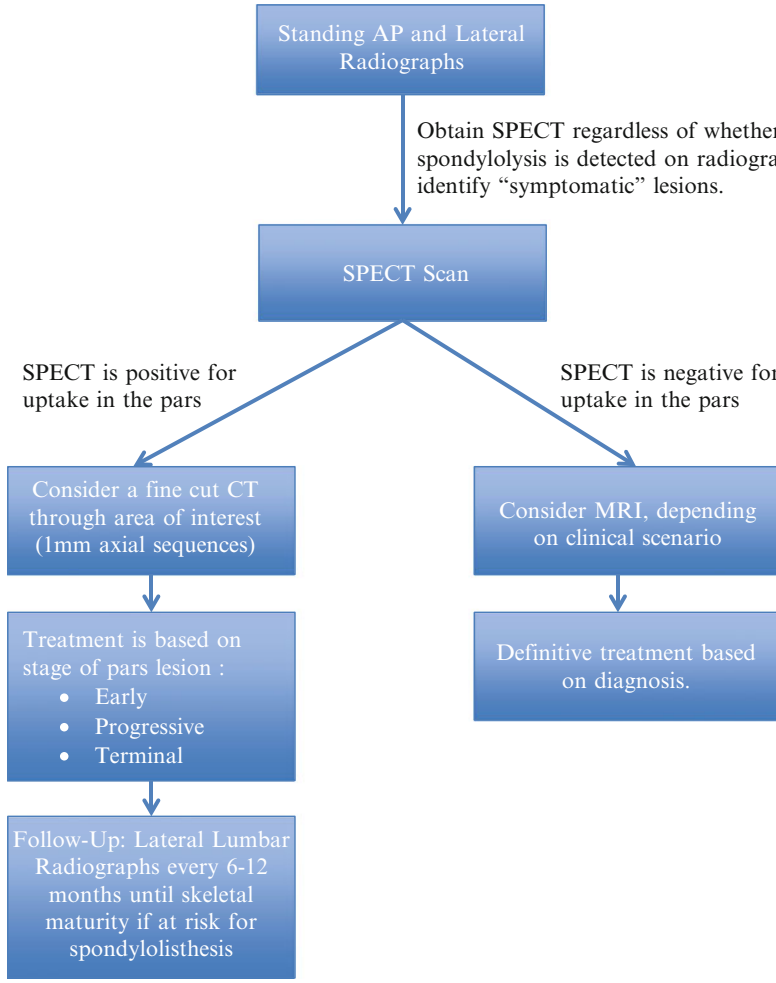

Fig. 6.5 Authors' recommended diagnostic work-up for spondylolysis

Reasonable Imaging Algorithm

Standaert and Herring outlined a reasonable imaging algorithm based on a thorough review of the available literature (Fig. 6.5) [63]. This involves first obtaining limited plain radiographs (i.e., standing AP and lateral) to identify a spondylolisthesis or other gross bony abnormality. Assuming these findings do not change diagnostic considerations, a SPECT scan of the lumbar spine should be performed. If this identifies an area of increased metabolic activity in the pars interarticularis, then a thin-cut CT (1-mm axial sequences) can be performed through only the identified level(s) to confirm the diagnosis and to stage the lesion, thus helping to gauge prognosis. If the SPECT is negative, then other diagnoses should be considered. If symptoms are present for greater than 6 weeks, further evaluation with MRI is reasonable. At the end of treatment for a spondylolysis, follow-up standing lateral radiographs should be considered every 6–12 months in athletes with bilateral defects or if a listhesis is already present to evaluate for progressive slip until the patient reaches skeletal maturity.

Treatment

After diagnosing spondylolysis, the clinician must prepare to educate the patient and family regarding the etiology, pathophysiology, natural history, and available treatment options. A "one-sized-fits-all" approach risks alienating the athlete, who faces considerable internal and external pressures to return early to sport. Again, a thoughtful approach is necessary to support each athlete and his or her family in developing realistic treatment goals that maximize long-term functional outcomes (see Fig. 6.6 for a reasonable treatment algorithm). Any plan should account for the athlete's age, noted biomechanical risk factors, training goals, sports participation, and severity of injury.

Fortunately, most athletes do well with nonsurgical care (see section "Outcomes"). Despite variable treatment protocols cited throughout the literature, limiting physical activity beyond routine daily tasks is the mainstay of any treatment plan. However, some physicians also advocate for the universal use of a lumbosacral orthosis to facilitate bony healing. Unfortunately, there is little science to guide the prescription of one brace type over another (e.g., soft corset or rigid antilordotic orthosis) and limited consensus on optimal duration of use, which can range from a few weeks to an entire year [63].

Bracing

Bracing advocates emphasize its theoretical role in limiting mechanical stresses across the pars [69]. However, biomechanical studies have challenged this hypothesis, demonstrating that lumbosacral orthoses paradoxically facilitate, rather than restrict, lumbosacral segmental motion [70]. This is obviously problematic for treating spondylolysis, given most injuries occur at L5. The fact that lumbar orthoses are effective in limiting whole body motion [70, 71] may better account for the benefits seen in research and clinical practice [63].

Interestingly, studies have consistently demonstrated similarly favorable outcomes regarding pain control and bony healing regardless of the bracing type or protocol used [17, 30, 54, 72, 73]. One of the hallmark bracing studies performed by Steiner and Micheli retrospectively examined a cohort of 67 adolescents diagnosed with spondylolysis [54]. Each athlete was prescribed an antilordotic-modified Boston back brace to be worn 23 h per day. Sports participation was allowed in the brace if there was no pain. After an average 2.5-year follow-up, 78 % of the athletes had achieved good or excellent clinical results, while only 18 % had evidence of bony healing of the pars fracture.

Unfortunately, the study lacked a control group and did not stratify patients based on radiographic stage of injury, limiting the authors' ability to comment on the concrete benefits of bracing compared with physical activity restriction alone [63]. This study and others demonstrate the disconnect between bony healing and clinical outcome [4, 17, 19, 30]. There has been no evidence to date through either comparative or cohort studies to demonstrate that bracing more safely or effectively facilitates earlier return to sports and, in certain patients, it may inadvertently reinforce a somatic focus on the injury. Coupled with the possible unnecessary financial costs involved with bracing, there is insufficient data to support uniform use in all athletes who develop spondylolysis.

Attempts to stratify which athletes experience the greatest rates of bony healing suggest that pars injuries are more likely to heal when an athlete presents with early stage, unilateral lesions found at levels L4 or higher [17, 66]. Fujii et al. demonstrated this well in their study of 239 pars defects treated with a soft corset, where they noted that only 3.5 % of the bilateral L5 "progressive" lesions achieved union compared to 100 % of the unilateral L4 "early" lesions [17]. But no study has definitively demonstrated a greater benefit of bracing over simple activity restriction, and studies investigating different bracing protocols have shown similar long-term outcomes even when athletes admit to not consistently using their brace [17, 54, 63, 72, 73]. Furthermore, a recent meta-analysis of 471 patients found that 89.0 % of subjects treated with a brace, compared with 85.8 % of subjects

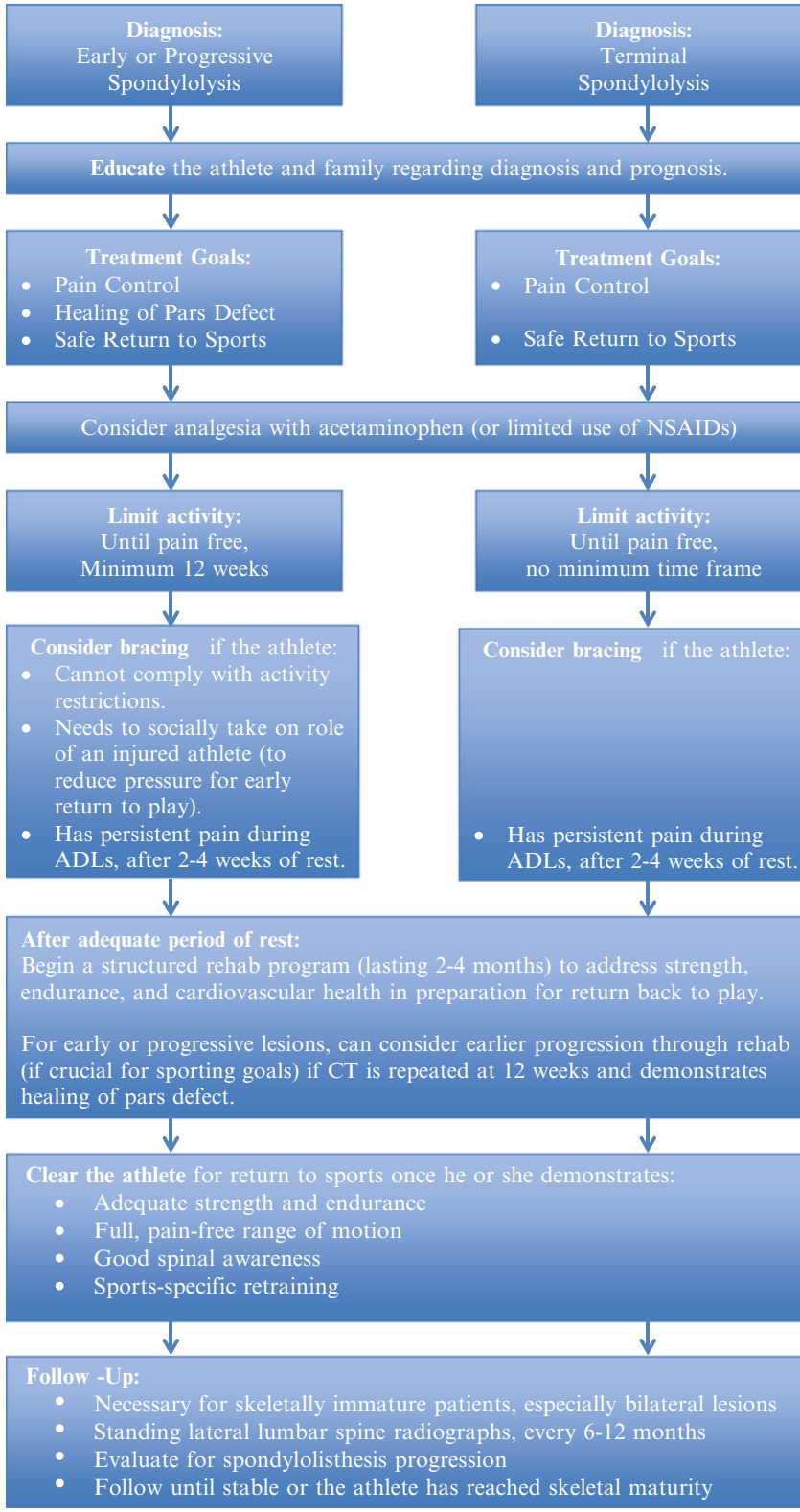

Fig. 6.6 Authors' recommended treatment protocol for spondylolysis

treated without a brace, achieved successful clinical outcomes ($P=0.75$) [74].

Thus, many clinicians now emphasize the primary goal of helping an athlete to abstain from exertional activity when working to heal an early or, in some cases, a progressive symptomatic pars lesion or to decrease symptoms from a terminal pars defect. Bracing is then reserved for cases where an athlete experiences insurmountable pressure to maintain an active schedule, or if severe pain persists during activities of daily living despite several weeks of rest [63, 72], as there is no evidence that bracing improves bony healing. A recent study of 34 soccer players found this type of protocol to lead to good or excellent outcomes 94 % of the time [75]. Hopefully, future study in this area will help delineate whether there is any clear benefit of bracing over activity restriction alone and help clarify treatment algorithms based on stage or severity of injury.

Activity Modification and Rest from Sports

Although many questions remain in designing an optimal treatment protocol, there is little doubt that activity modification (e.g., not participating in sports), while difficult, is paramount for an athlete to achieve a favorable clinical outcome. In one hospital-based clinic, although athletes reported less than 50 % compliance with the treatment protocol, 86 % stayed away from sports for at least 3 months. Those athletes who rested from sports for at least 12 weeks were 16.4 times more likely to have the most favorable clinical outcome compared to athletes who returned early [72]. This is a simple and convincing statistic that can be used when counseling young patients and their families.

For athletes who have the potential to heal a pars defect, it is important to recognize that no studies have yet demonstrated healing of early or progressive lesions prior to 12 weeks [63, 76]. Thus, it is recommended that athletes with early or, in some cases, progressive lesions rest for at least 3 months. Symptomatic terminal lesions are unlikely to achieve bony union with nonsurgical treatment [17], and thus these athletes may begin return-to-play conditioning protocols once they achieve adequate pain control [63]. When earlier stage lesions are detected (e.g., when a CT is negative for pars lesions after a positive bone scan) a shorter period of rest could be considered, but there are no studies to guide precise management for this population [63].

Rehabilitation Stages

After an adequate period of rest, if the athlete is pain-free with full range of motion, a rehabilitation protocol ideally should be instituted prior to clearing the athlete to return back to sports. Generally, repeat imaging to verify healing provides little clinical benefit, except when an athlete has an urgent need to progress more quickly through a rehabilitative protocol [77]. Rehabilitation after spondylolysis should typically progress through three separate stages that correlate with specific goals in functional activity progression [78]. However, even during the pre-rehabilitation phase, the clinician should screen for psychosocial factors that may predispose to persistent pain or disability (e.g., anxiety, depression, catastrophizing behavior, passive coping styles) and work to implement relevant changes early in treatment.

The acute stage of rehabilitation should focus on low-impact cardiovascular activity and core strength training, with attention to biomechanical features that could predispose the athlete to future injury. Preserving range of motion, strength, and aerobic conditioning while maintaining spinal stability is the primary goal for the initial acute stage, which often lasts around 4 weeks [77, 79]. During this stage, athletes should focus on neutral spine stabilization techniques and learn to engage the deep stabilizing muscles along the spine, including the transversus abdominis and multifidi, without activating more global musculature or creating excess intersegmental motion [80]. Progressive loads are added through use of the limbs and eventually the athlete is taught to engage the core throughout the day with special attention to tasks that had previously caused pain. Initially, the athlete can be provided tactile,

auditory, and visual feedback in a quiet environment with little consequence or distraction [79]. This can later translate to healthy movement patterns that become second nature or habit for the athlete during routine daily tasks.

The next stage, recovery, begins about 16 weeks after initiation of treatment for early or progressive lesions [77]. If there is a need to advance the athlete more quickly, this stage can be started as early as 12 weeks after initiation of treatment for an early pars fracture, as long as repeat CT imaging demonstrates a healed osseous union [77]. During the acute phase of rehabilitation, the athlete should develop comfort engaging and controlling his or her segmental spinal muscles, considered integral factors of core strength and stability. Thus, during recovery, physiotherapy shifts its focus to incorporate more advanced spinal stabilization techniques outside of the neutral plane. Work during this phase should include a thorough kinetic chain analysis while aerobic conditioning, strength training, and range of motion exercises are adapted and tailored to the athlete's sport [77].

The final stage of rehabilitation incorporates more functional tasks, including dynamic multiplanar spinal stabilization techniques followed by progressive general athletic and sports-specific re-training [77]. At this step, it can be helpful to partner with a physical therapist, athletic trainer, or strength and conditioning coach who has experience in the athlete's chosen sport in order to help refine the athlete's skill and technique. The entire rehabilitation protocol should take approximately 2–4 months, depending on the athlete's motivation, skill level, severity of injury, and progress. At any time, if the pain returns, then the physician should consider alternate diagnoses and prescribe additional rest until the athlete is symptom-free.

Once the athlete demonstrates appropriate strength and conditioning with full, pain-free range of motion, he or she may safely return to sports. An athlete that progresses forward as expected will reasonably return within 5–7 months after initial diagnosis [63, 77]. If pain persists after 6 months of treatment, or if the athlete develops neurologic symptoms, then surgical referral can be considered after investigating alternate diagnoses or psychosocial barriers that may impact recovery.

Outcomes

When defining outcomes after treatment for spondylolysis, clinicians consider both measures of function (including return to sport) and anatomic correlates from imaging. Resolution of a symptomatic spondylolysis can involve complete osseous union, healing with fibrous tissue, or nonunion [47]. Some have attributed difficulties in achieving osseous unions secondary to a physical barrier created by a communicating synovial pseudoarthrosis at the site of injury [81]. Others have implicated biomechanical factors in determining specific anatomic outcomes, such as whether or not a lesion is bilateral or involves L5 [17, 61]. Regardless, nonunion is a problem only when symptomatic, and the overwhelming majority of patients do well with early diagnosis and treatment even in the absence of bony healing [4, 17, 19, 30, 61].

Miller et al. followed patients for 9 years and found that no athletes with bilateral pars defects achieved osseous union, compared to 100 % healing rates from unilateral lesions. However, greater than 90 % of the athletes achieved good or excellent functional outcomes (e.g., participation in full activities, including sports) regardless of whether or not there was evidence for healing [61]. Similar findings have been described in other cohorts followed for up to 45 years [19, 30].

Spondylolisthesis is relatively common after spondylolysis, and patients present with an average slip of 9–14 % [13, 15, 19]. Spondylolisthesis is graded I–IV from lateral radiographs based on the percentage slip of one vertebral body over another. Grade I corresponds to a translation of 0–25 %, grade II corresponds with a slip of 25–50 %, and so forth [82]. Fortunately, the risk for progression to a clinically significant slip is low [2, 17, 83], and participation in competitive sports has been shown to not affect the progression of a spondylolytic spondylolisthesis [13].

When examining radiologic and clinical factors that could influence progression, Frennard et al. followed 47 adolescents for an average of 7 years and noted that only 4 % of patients achieved a slip progression greater than 20 % [84]. Danielson et al. followed a similar cohort of 311 patients followed for an average of 3.8 years [15]. Neither group was able to find any clear predictive variables for slip progression. These and other studies have confirmed that clinically significant slip progression is uncommon if there has been less than 30 % anterolisthesis at the time of diagnosis, and those that do progress tend to do so during a patient's early adolescent growth spurt [4, 13, 83–85]. Therefore, most authors recommend follow-up with standing lateral lumbar radiographs every 6–12 months until a patient reaches skeletal maturity, as no other factors have been found to predict progression [63].

Finally, early access to appropriate treatment is certainly important to achieving a positive functional outcome, and delayed treatment may lead to higher rates of surgical intervention [9, 50]. Surgery may be required in 9–15 % of cases of spondylolysis or low-grade spondylolisthesis, but is not generally required to control pain [9, 54, 86]. Either a direct pars repair or posterior fusion can be considered, but no head-to-head trials have proved superiority [87, 88].

Recurrent Pain After Returning to Sport

Occasionally, an athlete may present with recurrent symptoms of low back pain after an initial successful return to sports. In this setting, the clinician must maintain a broad differential diagnosis, and understand that the athlete may be presenting with either a new stress injury or an irritation of previous problem. Non-modifiable risk factors may predispose the athlete to recurrent injury at the same level. Understanding whether new symptoms stem from a novel (or even unrelated) injury, reinjury of previously healed defect, or irritation of a pseudoarthrosis is important for counseling the patient on treatment and prognosis.

The clinician must exercise clinical judgment when deciding on the need for additional imaging, particularly when the young athlete has undergone multiple recent studies involving ionizing radiation. Clinical research provides little guidance in this area, but there may be situations where the physician and athlete are content treating a new injury conservatively with an additional period of rest, even without having an exact anatomic diagnosis.

Associated Injury and Other Lumbar Stress Fractures

Although fatigue injuries of the pars interarticularis have been the focus of this chapter, any zone of the neural arch can fail in the right setting. Pediculolysis, a stress fracture involving the pedicle, is unusual, but this is the second weakest area of the vertebral arch after the pars [89–91]. Most reported cases have involved young, athletic individuals with clinical presentations similar to that of spondylolysis [92, 93].

Pedicle stress injuries likely result from abnormal forces on the neural arch after a contralateral spondylolysis or spinal surgery (either laminectomy or fusion), although independent lesions can also occur [89, 90, 94–97]. Some authors describe associated reactive sclerosis in the region of the pedicle, although it is not clear whether this is always a precursor to injury [90, 94]. Presumably, diagnostic work-up including SPECT and CT should adequately visualize this area, and pediculolysis should be considered when radiologic attention is turned to this area. Osteoid osteoma and osteoblastoma are the major differential diagnostic considerations; misdiagnosis could lead to unnecessary excision of tissue and exacerbation of instability that had initially created the lesion. Published cases have described successes with both conservative and surgical management for pediculolysis [94, 95].

Stress fracture of the lamina (i.e., laminolysis) is another fatigue injury involving the neural arch (Fig. 6.7), potentially as a complication from contralateral spondylolysis [98–100]. Diagnosis and treatment parallel that of spondylolysis,

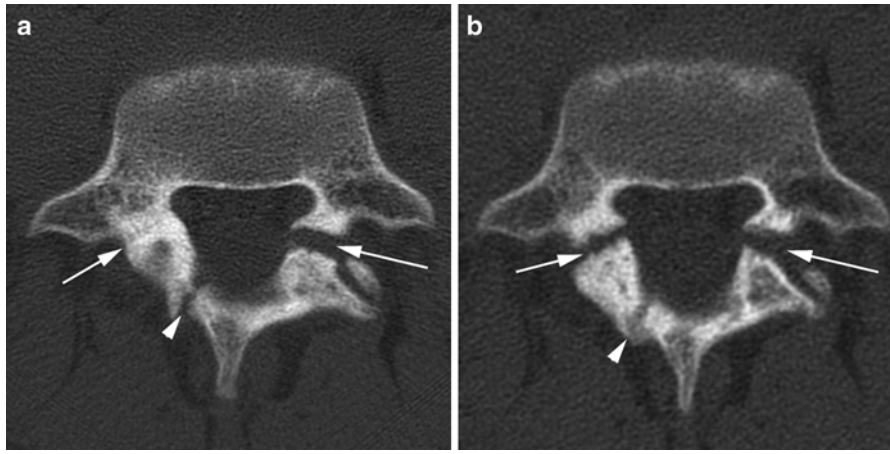

Fig. 6.7 Bilateral spondylolysis with laminolysis. (**a**) Example of a 15-year-old young male athlete with several months of worsening low back pain. His CT at initial presentation demonstrated evidence of early right and terminal left spondylolysis (*arrows*) as well as right-sided laminolysis (*arrowhead*). (**b**) After treatment, a follow-up CT demonstrated bilateral terminal pars defects (*arrows*) as well as evidence of fibrous healing of the lamina (*arrowhead*)

based on several published cases that reflect positive outcome with conservative care [98–100].

Summary

Lumbar isthmic spondylolysis is common among adolescent athletes with low back pain. Establishing an accurate diagnosis and developing an appropriate initial treatment plan are key to helping the athlete return safely back to his or her sport. As emerging research clarifies optimal diagnostic and treatment approaches, the sports medicine physician should understand that a rational treatment approach should include goals of minimizing time away from sporting activity, facilitating osseous healing where appropriate, reducing iatrogenic disability, and limiting exposure to ionizing radiation.

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Stress Fractures of the Pelvis

7

T. Sean Lynch, Ronak M. Patel, Nirav H. Amin, and Richard D. Parker

Introduction

Stress fractures are a very common overuse injury in the athletic population. Overall, these injuries consist of approximately 15–20 % of all visits to sports medicine clinics [1, 2]. Pelvic stress fractures make up 1.3–5.6 % of all stress fractures seen in athletes [3, 4]. These injuries are considered "low-risk" stress fractures that can predominately be treated with rest and gradual resumption of activity. Although these injuries do not get the same notoriety as stress fractures of the tibia or metatarsals, these injuries are still prevalent in the active running and female military populations. Pelvic stress fractures are generally not at the top of the differential diagnosis when an athlete presents with low back and/or groin pain so a strong level of suspicion is required to ensure proper diagnosis.

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Early stress injuries have been found to be asymptomatic in 17–43 % of athletes [5–9]. In general, these injuries occur when normal bone is exposed to excessive, repetitive force. In the setting of pelvic stress fractures, this can be caused by the inability of nearby muscles to absorb the shear forces acting on the bone or due to traction from the muscle on the bone itself [10]. As a result, the bone homeostasis is altered with the predominance of osteoclastic function with osseous breakdown in the form of microcracks in the bone. This failure can then propagate to create a frank fracture line, which can progress to a complete and displaced fracture with potential for nonunion [11, 12].

Sacral Stress Fractures

Sacral stress fractures are uncommon athletic injuries that present as an insidious onset of low back and buttock pain. This is an overuse injury in which the athlete will complain of discomfort towards the end of or at the end of a workout. This injury was first described by Volpin et al. in an athletic population of three military recruits all with a sacral wing stress injuries [13]. Overall, the injury prevalence is unknown [14], but these individuals are usually athletes who participate in repetitive, load-bearing activities such as long distance running, weight-lifting, and military marching. Additionally, the senior author (RDP) has treated this injury in a professional women's

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basketball player. It is important to note that the sacrum is the keystone arch of the pelvis and it is believed that these injuries are the result of repetitive loading of the vertebral column. These stresses are then dissipated from the spine and concentrated in the sacrum and sacral ala [15].

Presentation and Physical Examination

In addition to low back pain, these patients will present with paramedian tenderness on either side of the sacrum or directly on the sacroiliac (SI) joint [16–19]. Unilaterality of sacral stress injuries has been attributed to leg length discrepancy with the longer limb more commonly affected [18]. These patients will otherwise have a benign physical examination with full range of motion of their lumbar spine and a normal neurologic exam of their lower extremities; however, sciatica may also be a presenting symptom that mimics intervertebral disc disease [20]. The examiner should ask the patient to perform a single-leg hop test in order to simulate load-bearing activities to recreate the patient's discomfort. There also might be a positive FABER test (flexion, abduction, and external rotation) with discomfort to the SI joint on the side of the affected extremity. These sacral stress fractures can also be associated with pubic-related stress injuries as a result of abnormal shear stresses that are transmitted through the pelvic ring due to instability at the symphysis [20].

Diagnostic Imaging

Radiographic evaluation should begin with plain radiographs of the lumbosacral spine and pelvis to rule out more common causes of pain; however, the utility for diagnosing these injuries with plain radiographs is very poor [21]. The sensitivity of plain radiographs is typically not high enough to show sacral stress fractures. It has been reported that 85 % of sacral fractures are missed on X-ray [22]. These images are compromised by overlying soft-tissue, bowel gas, and the geometry of the sacrum [23]. As a result, the most common imaging modalities to diagnose these injuries are magnetic resonance imaging (MRI) and bone scintigraphy (scans). MRIs can detect the earliest signs of a stress injury such as bone marrow and cortical bone edema with high sensitivity (86–100 %) and specificity (100 %) [24, 25]. Additionally, MRI is useful in the staging of a stress fracture. Bone scans are highly sensitive in detecting stress injuries not seen on radiographs as they can display increased radiotracer uptake in areas of high bone turnover 1–2 weeks before plain radiographs [26]. However, unlike MRI, bone scans come with the risk associated with ionizing radiation. Finally, computed tomography (CT scans) can be helpful in the detection as well as the assessment of healing of these injuries (Fig. 7.1a–d).

Pubic Rami Stress Fractures

Wachsmuth first described stress fractures of the pubic ramus in three male military recruits in 1937 [27]. Although these are rare injuries with few described in the literature, they have been found to be common in female military personnel and athletes [28–31]. In the female military population, it has been hypothesized that an increased force is placed on the pubic rami in order for these recruits to maintain the same stride length as their male colleagues [32]. Another explanation is a greater concentration of cancellous bone in this pelvic region in the female gender [33].

Pubic ramus stress fractures usually occur at the medial portion of the inferior pubic ramus adjacent to the pubic symphysis. This injury is caused by excessive contraction of the adductor magnus muscle at its origin between the ischial tuberosity and the inferior pubic ramus [29]. The adductor magnus pulls the lateral aspect of the pubic ramus when the hip is extended thus initiating the development of these fractures [30]. These injuries have been postulated to occur more frequently in females because females have a greater reliance on hip external rotation in their gait cycle [34, 35] (Fig. 7.2).

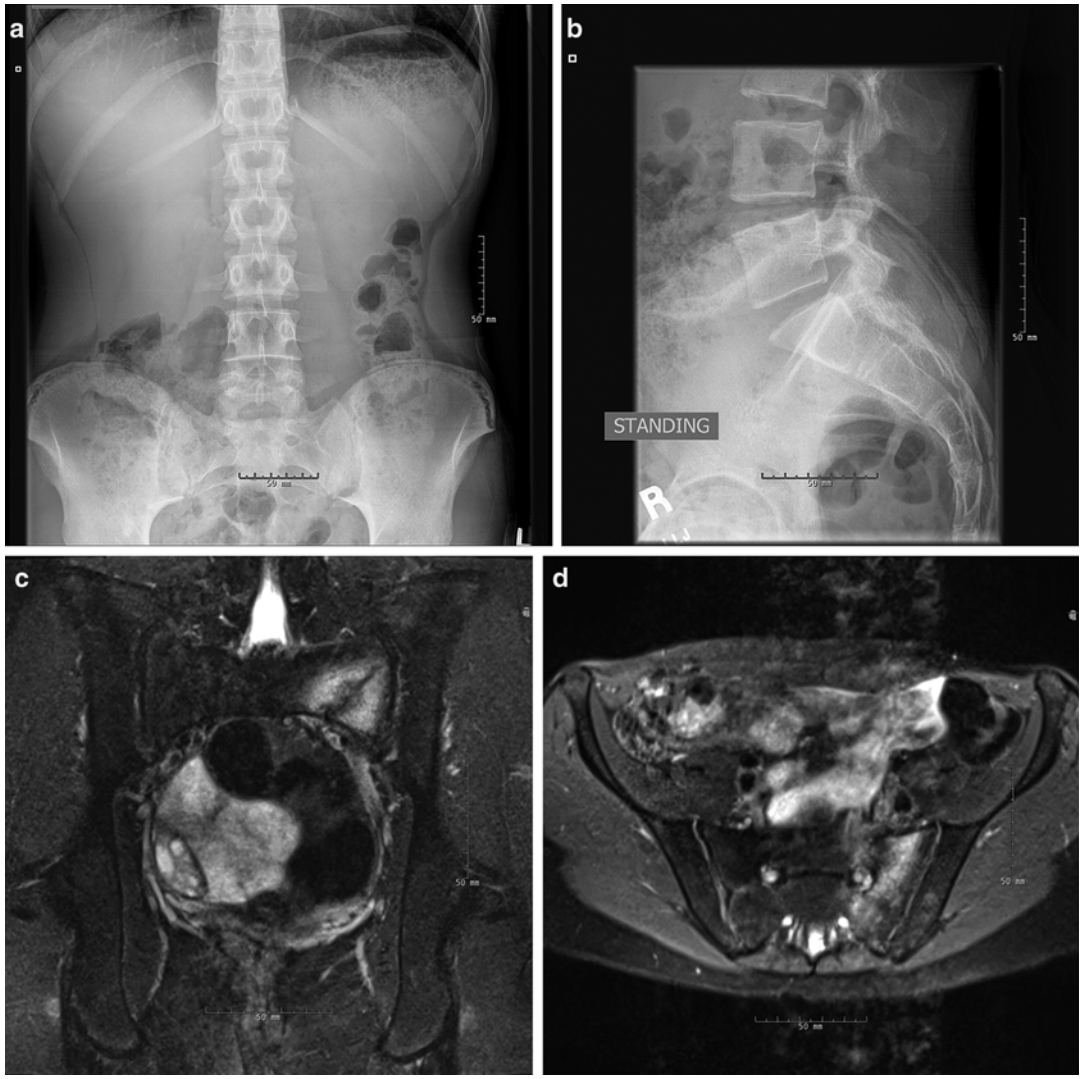

Fig. 7.1 An 18-year-old collegiate track and field athlete with a left sacral stress fracture. (**a**, **b**) The anteroposterior (AP) and lateral radiographs of the lumbosacral spine do not show any osseous abnormalities indicative of a sacral stress fracture. (**c**, **d**) Coronal and axial T2 MRI, respectively, demonstrate a linear fracture line within the left sacral ala involving the left first and second sacral neural foramina with associated adjacent marrow edema compatible with sacral stress fracture

Presentation and Physical Examination

Typically, patients present with groin pain that is exacerbated by activity. On physical examination, the athlete may have an antalgic gait as well as demonstrate exquisite tenderness to palpation at the pubic ramus. The patient will display full range of motion of the hip; however, there may be discomfort with hip external rotation and pain with resisted adduction. Additionally, the patient will demonstrate a positive "standing test," the inability to stand unsupported on the affected lower extremity [30].

Diagnostic Imaging

Diagnostic evaluation begins with an anteroposterior pelvis radiograph that can show a nondisplaced fracture at the inferior pubic ramus

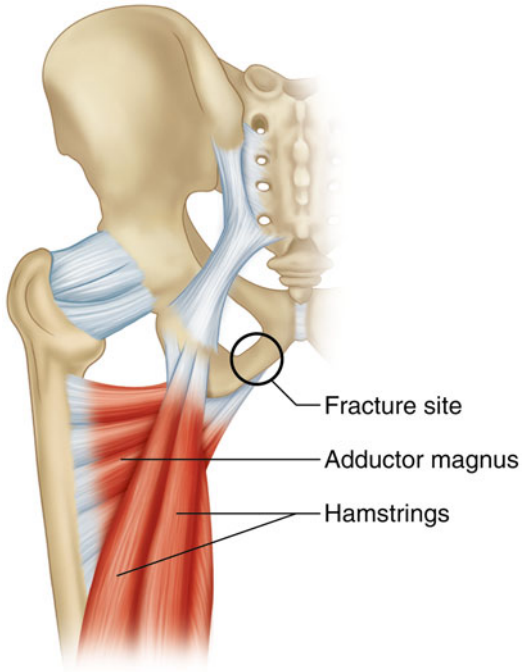

Fig. 7.2 The origin of the adductor magnus and hamstring muscles, shown with the most common site of pelvic stress fracture

near the pubic symphysis. Unfortunately, in the early stages of this injury, plain radiographs will likely not pick up this stress fracture; therefore, bone scintigraphy can be considered since it is a more sensitive indicator of early stress fractures when compared to plain radiographs [36]. Additionally, MRI has been found to provide more diagnostic information such as the presence of fracture lines and bone edema.

Iliac Wing Stress Fractures

Iliac wing stress fractures are extremely rare, consisting of 4 % of all pelvic stress fractures [37, 38]. There are only a total of four case reports in the literature of these injuries: two case reports of iliac wing injuries that extend into the SI joint, and two other reports that do not involve the SI joint. Patients with this condition are usually long distance runners complaining of lateral-based hip pain without a history of trauma. The mechanism of injury is believed to be the result of osseous failure secondary to two opposing repetitive loads: cephalad traction from the musculature of the abdominal wall and a caudal force from the abductors inserting onto the iliac crest (Fig. 7.3a–f).

Apophyseal Avulsion Fractures

Apophyseal avulsion fractures are stress injures about the pelvis that are present in adolescent athletes and, occasionally, patients in their mid-20s [39]. These stress injuries are the result of indirect trauma caused by a forceful concentric or eccentric contraction of the muscle that is attached to the apophysis. This causes failure through the growth plate and is the result of the inherent weakness across the unfused apophysis of a skeletally immature athlete. In a more skeletally mature athlete with the same mechanism of injury, these injuries would result in a musculotendinous tear of the affected muscle (Fig. 7.4).

In this adolescent population, there are two types of epiphyses: pressure epiphyses and traction epiphyses. A pressure epiphysis is at the end of long bones and is subjected to the pressures across the joint. Meanwhile, a traction epiphysis is an apophyseal growth plate on which a major muscle or muscle group originates or inserts and is most commonly involved in these avulsion injuries (Table 7.1). Within the traction epiphysis, the epiphyseal plate is the weakest point because the tendon's Sharpey's fibers that attach to the growth plate are stronger than the junction of cells between the calcified and uncalcified epiphysis. As a result, osseous failure occurs within the zone of hypertrophy of the growth plate [40].

In a radiologic epidemiologic study, Rossi et al. studied more than 1,000 radiographs and found 203 pelvic apophyseal avulsion injuries [41]. In this series, the most common injuries were at the ischial tuberosity (54 %) followed by the anterior inferior iliac spine (22 %), the anterior superior iliac spine (19 %), the pubic symphysis (3 %), and the iliac crest (1 %). Soccer players were at greatest risk (74 injuries) followed by gymnasts (55 injuries) [37].

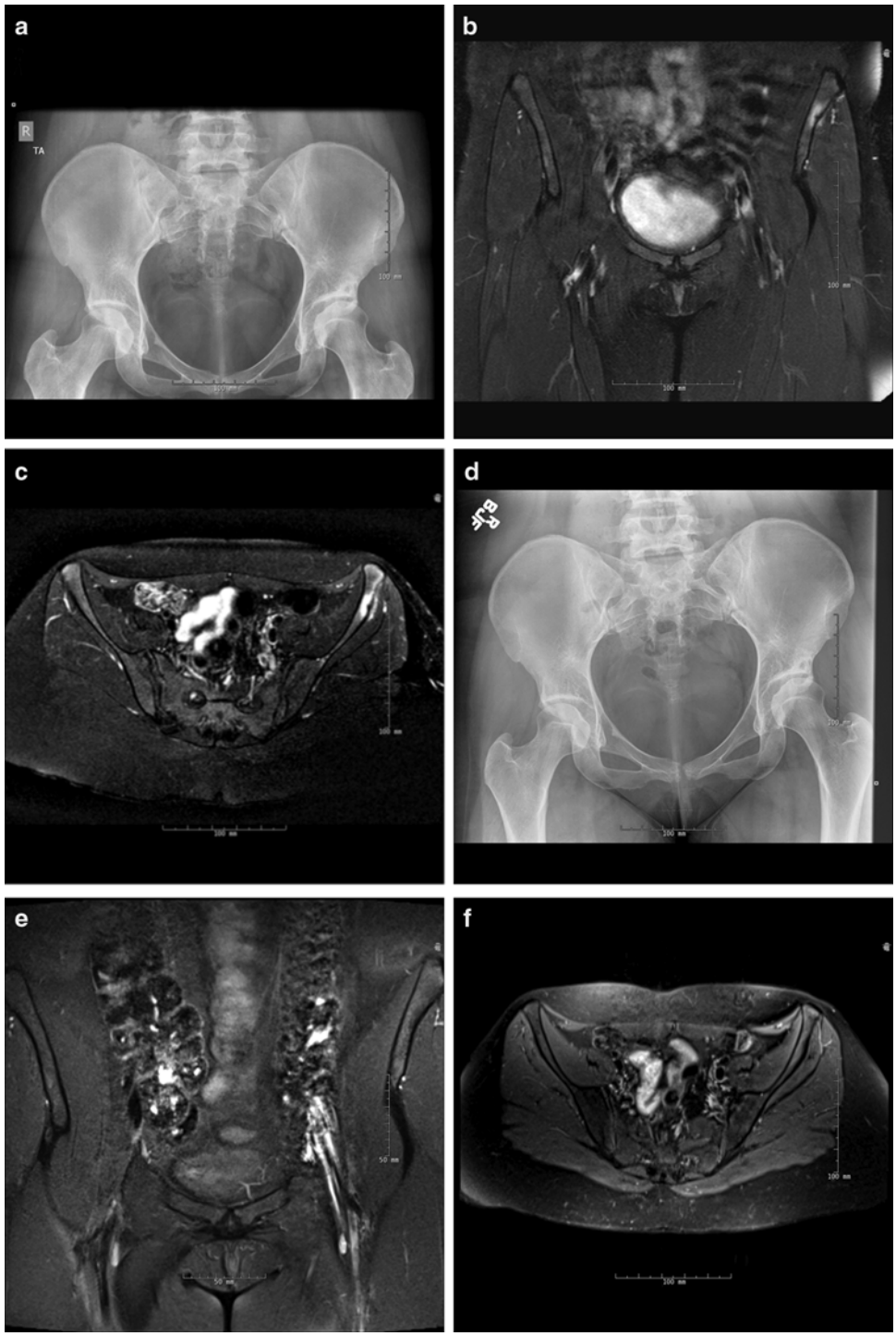

Fig. 7.3 A 20-year-old marathon runner with a left iliac wing stress fracture. (**a**) The anteroposterior (AP) pelvis radiograph shows some sclerosis on the left iliac brim with no obvious fracture line. Part (**b**) is a coronal T2 MRI image demonstrating a small area of ill-defined marrow edema involving the left iliac wing just inferior and posterior to the ASIS. Part (**c**) is an axial T2 MRI image demonstrating a small linear area of decreased signal along the lateral cortex with mild periosteal edema. Both findings are consistent with stress fracture. Part (**d**) is an AP pelvis radiograph 3 months after presentation after cessation of activity with no interval change; however, parts (**e**) and (**f**) are coronal and axial T2 MRI images demonstrating resolution of the bony edema

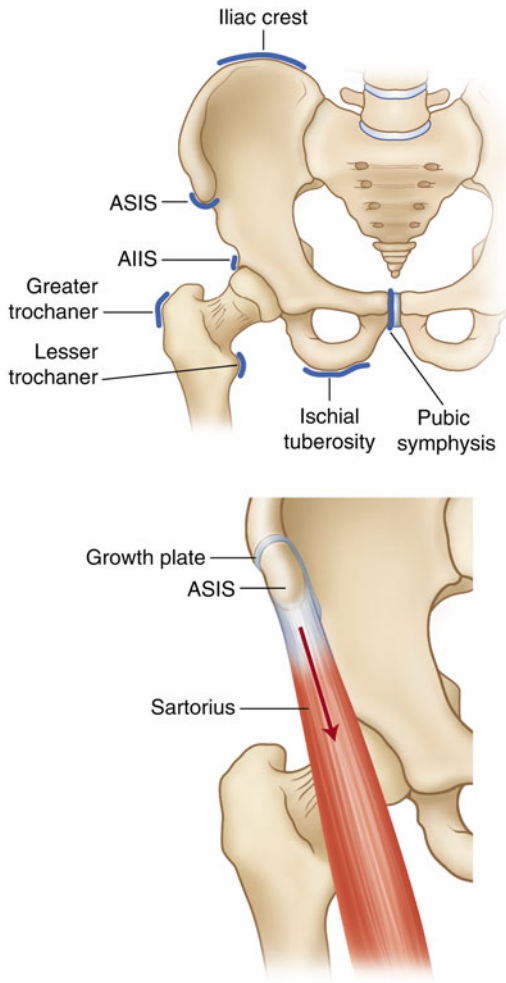

Fig. 7.4 The most commonly affected apophyses. Inset: The ASIS is the attachment site for the sartorius and some fibers of the tensor fascia lata. Avulsion of the ASIS occurs from a strong sudden pull of the sartorius with the hip in extension and the knee in flexion, most commonly in sprinters, hurdlers, and other running athletes

Table 7.1 Common sites of apophyseal avulsion fractures about the pelvis and their muscular attachments

Common site of avulsion in pelvis	Muscle attachment
Anterior inferior iliac spine (AIIS)	Direct head of the rectus femoris
Anterior superior iliac spine (ASIS)	Sartorius and tensor fascia lata
Iliac crest	External and abdominal obliques
Ischial tuberosity	Proximal hamstring
Pubic symphysis	Hip adductors

Presentation and Physical Examination

The affected athlete presents with an acute onset of pain that occurs during a sporting maneuver. Generally, there is swelling and localized tenderness to palpation about the site of injury with discomfort exacerbated by the use of the attached muscle. The affected extremity will maintain a position that places the least amount of tension on the involved muscle and the patient will guard against active contraction of this muscle.

Diagnostic Imaging

The clinical evaluation is usually diagnostic; however, plain radiographs are helpful to determine the degree of bony displacement and the size of the avulsed fragment. These injuries are usually evident on plain anteroposterior pelvic radiographs, but an additional oblique projection might be needed to appreciate the fracture (Figs. 7.5a, b and 7.6a, b). MRI can be useful to evaluate for avulsion fractures in children with ossification centers that have not ossified yet as well as to assess for apophysitis. Additionally, ultrasound can be considered as a cost-effective modality that has been found to be accurate in diagnosing these avulsion injuries [42].

Management

The majority of the aforementioned pelvic stress fractures can be treated through nonsurgical means consisting of rest, activity modification, and then a gradual return to athletics. Additionally, any patient with a suspected pelvic stress fracture should undergo a metabolic evaluation consisting of blood and urine work as well as the intake of calcium and vitamin D. A three-phase program has been developed to get athletes back to sport with the presence of pain as the guiding factor in progression through the program [25, 43].

Stage 1 consists of avoiding painful activities. Some sacral fractures may require the athlete to

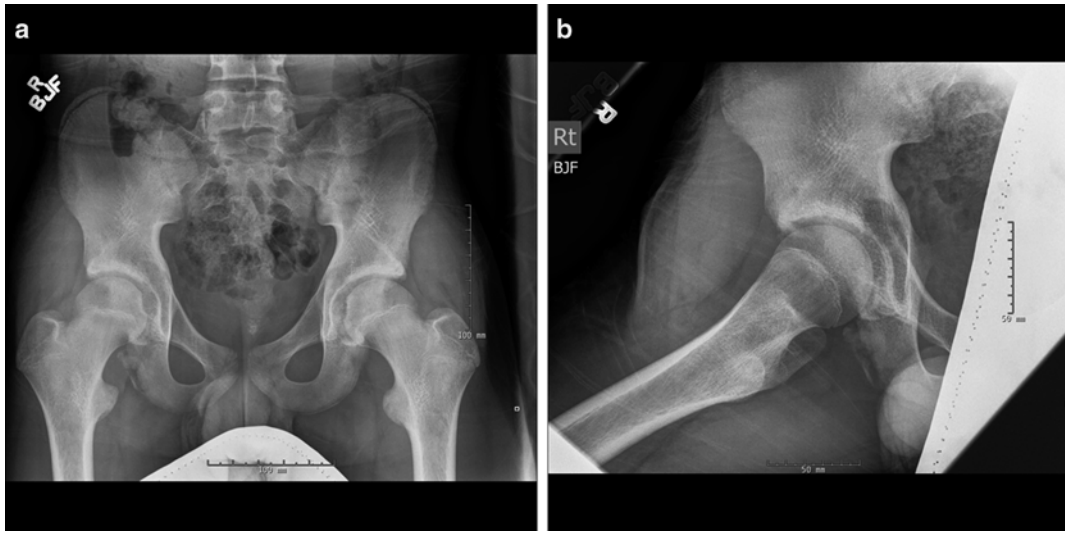

Fig. 7.5 A 15-year-old football player with subacute avulsion of the right ischial tuberosity. (**a**, **b**) The anteroposterior (AP) and right frog-leg lateral radiographs demonstrating a healing ischial tuberosity avulsion fracture

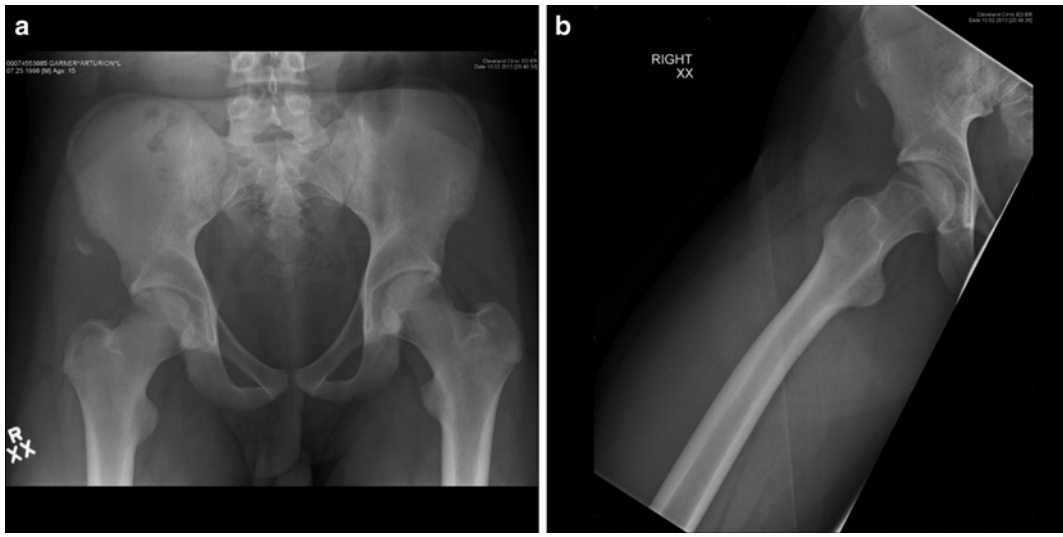

Fig. 7.6 A 15-year-old basketball player with antecedent pain at the anterior superior iliac spine (ASIS) with an acute avulsion of the right ASIS that occurred while attempting to dunk the basketball. (**a**, **b**) The anteroposterior (AP) and right frog-leg lateral radiographs demonstrate the healing avulsion fracture

be non-weightbearing with the assistance of crutches. Once pain with ambulation has been eliminated, these crutches can be discontinued. *Stage 2* is then initiated once the patient has been pain-free with activities of daily living for 3–5 days. This second phase focuses on strength training and correcting any pelvic muscular imbalance through light-weight and nonimpact exercises. Additionally, sports-specific muscular rehabilitation is initiated at this time. *Stage 3* allows the athlete to gradually return to play with progression to normal athletic load for the athlete's specific sport. Overall, this process can range from 3 to 18 weeks [25]. It is important to

note that premature return to sport prior to complete union can increase the risk of complications such as delayed union or nonunion.

Apophyseal avulsion fractures are also treated conservatively similar to the previously described program. These athletes generally return to athletics no earlier than 2 months after the initial injury [44]. Unlike the other pelvic stress fractures, apophyseal injuries can be amenable to surgical intervention with open reduction internal fixation in order to prevent disability in competitive athletes. Some advocate operative intervention if the avulsed fracture fragment is more than 2 cm in size; however, these indications and the optimal timing of surgery still remain debated [45].

The Role of Nutrition

In athletes with stress fractures, nutritional and hormonal imbalances are frequently prevalent. Any patient with a suspected pelvic stress fracture should undergo a metabolic evaluation consisting of blood and urine work as well as the dietary intake of calcium and vitamin D. These two minerals have been found to be integral components of nutrition required to achieve and maintain bone homeostasis. Calcium provides strength to the bone through a mineralized matrix and serves as the primary storage of calcium in the body. Vitamin D assists with the absorption of calcium from the digestive tract and renal systems and promotes bone growth and remodeling. Additionally, these minerals have been shown to improve bone density and prevent fractures at all ages. These injuries are most commonly seen in the female gender as this group of athletes experiences the loss of the bone-maintaining effects of estrogen. Additionally, these athletes have a reduction in calcium and vitamin D all of which place them at an increased risk of stress fractures.

Calcium supplementation has been shown to be beneficial in stress fracture prevention [46, 47]. In female military recruits and athletes, Tenforde et al. suggested that a daily intake of 1,500 mg of calcium may reduce the incidence of these stress injuries [48]. Meanwhile, for individuals who are at risk for low bone mineral density (BMD) or have sustained a stress fracture, they should also be screened for serum 25(OH)D levels. Previous studies have found a relationship between vitamin D insufficiency and the occurrence of stress fractures [49]. McCabe et al. recommend that patients should receive 800–1,000 IU of vitamin D3 daily with the therapeutic goal of at least 50 nmol/L (20 ng/mL) to as high as 90–100 nmol/L (36–40 ng/mL) [50]. A higher dietary intake of vitamin D has been found to be protective against fracture, but the exact role of vitamin D in fracture prevention is still unclear and requires further investigation [51]. Despite this, it should be stressed that any hormonal and nutritional deficiency should be addressed to optimized normal development and repair of bone.

Summary

Although pelvic stress injuries are rare, they can be a frustrating problem for both the athlete and the physician. Athletes with this injury will often present with vague complaints that can contribute to delay in diagnosis and treatment; therefore, a thorough history and physical examination with judicious use of imaging is vital to successfully diagnosing these injuries. The majorities of these injuries do not require operative intervention and can be treated with rest, cessation from the causative activity, and a gradual return to sport. In these athletes, it is important to comprehensively evaluate them with a metabolic and nutritional assessment. Early recognition and prompt care are paramount to successful treatment of these injuries.

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Stress Fractures of the Femur

8

Marc S. Haro, Julia R. Bruene, Kathleen Weber, and Bernard R. Bach Jr

Introduction

Stress fractures are relatively common injuries in persons engaging in athletic activity. Stress fractures account for approximately 10 % of all injuries evaluated by sports medicine health care providers, and those occurring in the femur are the fourth most commonly diagnosed [1]. Femoral stress fractures are generally seen in athletes and military recruits who engage in repetitive impact loading of the lower extremity. Long distance runners, jumpers, dancers, female athletes, and older athletes appear to be at higher risk for developing femoral stress fractures [2]. Based on etiology, femoral stress fractures can be globally categorized into two types: insufficiency fractures and fatigue fractures. Insufficiency fractures occur as a result of normal physiologic stresses on bone that is deficient in structure. Conversely, fatigue fractures are a product of repetitive impact stress of structurally normal bone [3]. A third much less common type includes atypical femur fractures, which have been correlated with bisphosphonate use in the treatment of osteoporosis. Femoral stress fractures are further categorized based on anatomic location. Diagnosis of femoral stress fractures in a prompt and efficient manner is important as delayed diagnosis may lead to further complications. If not detected early, femoral stress fractures may progress to complete or displaced fractures, which may require surgical management. This chapter focuses on the epidemiology, classification, pathophysiology, diagnosis, and management of femoral stress fractures in athletes and active individuals.

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Epidemiology and Risk Factors

Femoral stress fractures are thought to be an underdiagnosed condition; therefore, the true incidence is difficult to determine [4]. When categorizing femoral stress fractures based on

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Table 8.1 Risk factors for femoral stress fractures

• Training errors
• Sudden increase in exercise intensity and/or frequency
• Poor footwear
• Leg length discrepancy
• Coxa vara
• Pes cavus
• Insufficient energy intake
• Low baseline bone density/mass
• Menstrual dysfunction
• Low dietary calcium intake
• Low lean muscle mass in lower extremity
• Diagnosis of eating disorder
• History of prior stress fracture or reaction

anatomic location, the incidence varies. The most common locations for stress fractures are at the femoral neck and femoral shaft. Fullerton and colleagues estimate femoral neck stress fracture incidence of 11 % [5]. Johnson et al. postulated that the occurrence of femoral shaft stress fractures is actually much higher than previously thought, and estimated an incidence of approximately 20 % of all stress fractures [6]. A meta-analysis of stress fractures performed by Snyder et al. demonstrated a significant variation in the occurrence of femoral stress fractures as a percentage of total stress fractures in all anatomic locations; studies ranged from 2.8 to 33 % [7].

While all athletes are at risk for either fatigue or insufficiency fractures, certain populations show an increased incidence of femoral stress fractures (Table 8.1). Long distance runners, jumping athletes, and dancers (particularly ballet dancers) appear to have a higher risk of developing stress fractures of the femur [8]. Typically stress fractures arise during a time of increased exercise intensity and frequency. Military recruits also manifest a higher risk, particularly for stress fractures involving the distal shaft of the femur [9]. A variety of risk factors for femoral stress fractures have been identified. While maladaptive training techniques appear to be the most prevalent type of risk factor, many other intrinsic and extrinsic factors may contribute [10–12].

Female athletes are at a higher risk for developing femoral stress fractures than their male counterparts [13]. Irregularity of menstrual cycles and bone mineral density are closely related. Female athletes who experience oligomenorrhea or amenorrhea appear to have an increased risk of femoral stress fractures [14]. Other contributing factors include inadequate calcium and vitamin D intake, low baseline bone density, insufficient energy intake, low lean muscle mass in the lower extremities, and long-term high intensity training [15]. Female athletes who manifest one or all components of the female athlete triad are at particularly high risk for developing femoral stress fractures. The female athlete triad consists of low energy availability (with or without concomitant eating disorder), menstrual disturbances, and altered bone mineral density [16]. Similarly, female athletes who participate in high intensity physical activity before the onset of puberty are at increased risk of primary or secondary amenorrhea, stress fractures, and non-healing fractures [14].

Recently, increased attention has been paid to the risk of developing femoral stress fractures with long-term use of bisphosphonates. Neviaser and colleagues described case reports of low energy atypical subtrochanteric femur fractures with prolonged use (typically greater than 5 years) of alendronate [17]. Several other case reports and series further raised concerns that atypical femur fractures were occurring in patients taking bisphosphonate medications as well as other medications such as glucocorticoids and proton pump inhibitors (Fig. 8.1) [18–25]. However, results from subsequent large registry-based studies and meta-analyses and systematic reviews have been somewhat controversial and inconclusive. Some studies have found no difference in the ratio of typical intertrochanteric to atypical subtrochanteric fractures in untreated and alendronate-treated groups, suggesting that both fractures should be considered osteoporotic in nature [21, 26]. However, a systematic review and meta-analysis by Gedmintas et al. reported an increased risk of atypical femur fractures, subtrochanteric fractures, and femoral shaft fractures in those taking bisphosphonate medications [27]. A report by Shane et al. and the Task Force of the American Society for Bone and Mineral Research

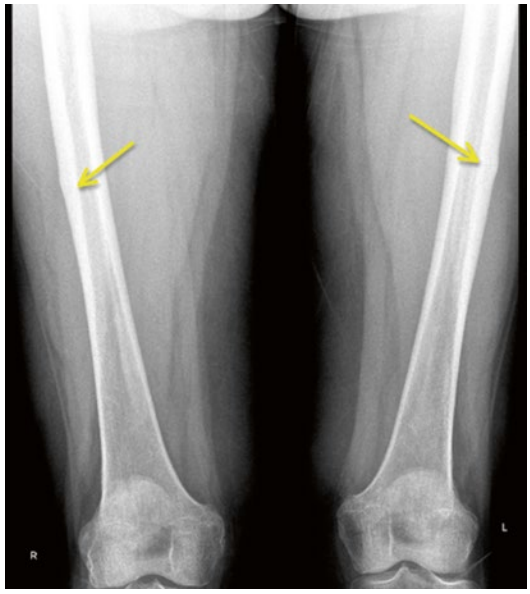

Fig. 8.1 Bilateral tension side femoral shaft fractures in an individual taking prolonged bisphosphonate therapy for osteoporosis

indicated that while atypical femur fractures are very rare, there is increasingly compelling data that a causal link between bisphosphonates and atypical femur fractures exists [28].

Pathophysiology and Classification

The femur is the largest bone in the body and transmits significant force with any loading activity. In general, bone is strongest in compression and weakest in tension. The femur is typically well suited to handle the compressive forces sustained with physiological activity. Unfortunately, in certain situations bone can fail, resulting in fracture. Running can impose ground reaction forces on the femur 3–8 times that of walking [29]. An insufficiency fracture occurs when the bone quality or structure is not able to withstand normal forces that are placed upon it [3, 30]. In these cases disrupted or abnormal bone remodeling weakens the bony structure and allows injury at otherwise normal physiological strain levels. This can occur when bone is weakened most commonly from osteoporosis but can also be attributed to various medications or other abnormal metabolic processes [22, 23, 27, 31, 32] (Table 8.2). A fatigue fracture occurs when repetitive or excessive strain is placed upon normal bone and the subsequent frequency or intensity of the strain exceeds the bone's ability to repair itself. Initially microscopic fractures occur and the bony remodeling process, whether from too frequent or too intense loading, cannot keep up with the excessive strain. Instead of the microfractures healing, the process propagates to a macroscopic crack and may eventually lead to a fracture.

Table 8.2 Conditions that predispose to insufficiency fractures

• Osteoporosis
• Osteomalacia/Ricketts
• Fibrous dysplasia
• Paget disease
• Osteogenesis imperfecta
• Osteopetrosis
• Hyperparathyroidism
• Irradiation
• Diabetes mellitus

It is believed that muscle fatigue and conditioning may play a role in the development of stress fractures. The muscles about the femur have broad attachments and have the ability to help transmit and distribute forces [33, 34]. With muscle fatigue or improper conditioning, this ability is lost and forces may be concentrated at certain points along the bone. The geometry of the femur and the distribution of bone mass play a large role in where the femur is loaded and where fractures occur [35]. The femur can be divided into three basic zones: the proximal femur, the femoral shaft, and the distal femur. The proximal femur can be further broken down into three regions: the femoral head, neck, and the peritrochanteric region.

The femoral head experiences the majority of forces on its superior surface where it articulates with the acetabulum. This can be demonstrated anatomically as the trabecular pattern forms a principle compressive group that extends from the medial calcar to the superior aspect of the femoral head. The principle compressive group is a bony strut that helps transmit forces through the femoral head to the shaft (Fig. 8.2).

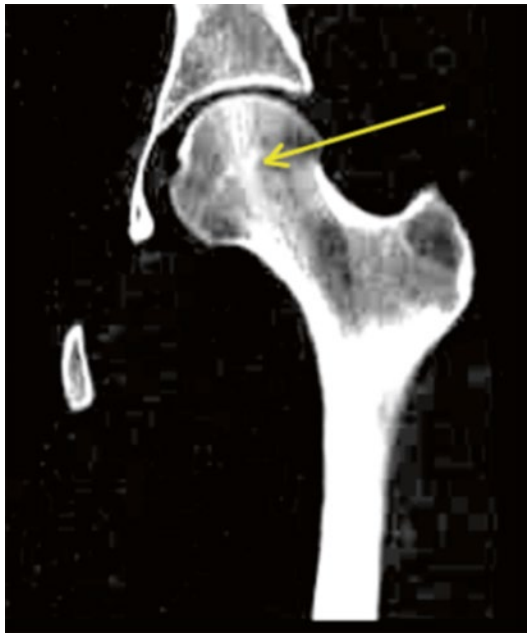

Fig. 8.2 CT scan of femur with arrow demonstrating the principle compressive group of the bony trabeculae within the femoral head and neck

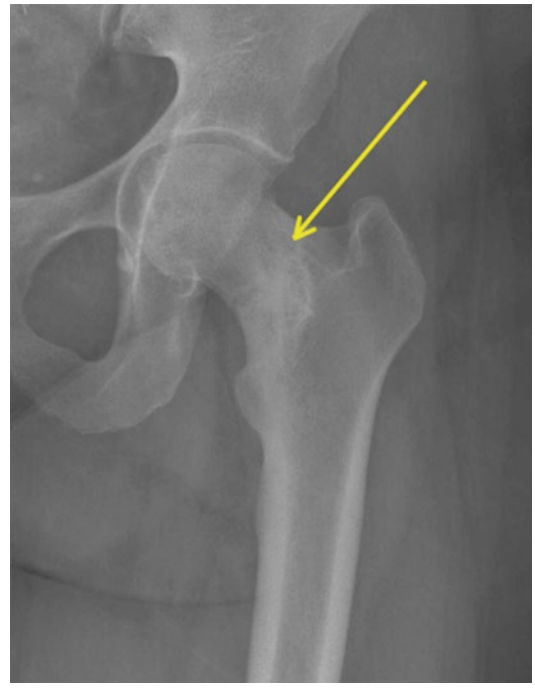

Fig. 8.3 Plain radiograph of femur demonstrating compression side stress fracture of the femoral neck (*arrow*)

When the bony trabecular structure is weakened or repetitively overloaded, compression fractures typically occur in this location [36].

Given that the mechanical axis of the lower extremity falls medial to the majority of the femur, the majority of the medial aspect of the femur is under compression and the lateral aspect is under tension. As a result, repetitive strains to the neck will typically cause compression fractures on the medial aspect of the femoral neck or tension type fractures on the lateral aspect. There have been several classification schemes that have been used to describe and guide treatment of femoral neck stress fractures. Early attempts at classifying femoral neck stress fractures simply classified them into two groups: complete and incomplete fractures [37]. This classification was aimed at guiding treatment, as incomplete fractures were believed to heal with conservative measures. However, complete fractures typically did not have satisfactory outcomes. Devas subsequently attempted to radiographically classify femoral neck stress fractures based upon their biomechanics [38]. Femoral neck stress fractures were classified into compression type fractures and transverse fractures. Compression fractures were seen on the medial or inferior femoral neck where callous or sclerosis was noted (Fig. 8.3). These fractures were felt to be stable injuries that would typically heal with conservative measures alone. Transverse fractures, which were later renamed "distraction" type fractures, typically began as a crack on the tension side, or the superior surface, of the femoral neck. These fractures have a high risk of propagating from the lateral side and may eventually become displaced. The first study to prospectively evaluate the diagnosis and treatment of femoral neck stress fractures was done by Fullerton [39]. Using radiographs and radionuclide studies he followed 54 femoral neck stress fractures. As a result, he modified the classification scheme for femoral neck stress fractures to three major groups: compression side, tension side, and displaced. He found that compression-sided injuries may be radiographically negative and only show increased signal on the compression side of the femoral neck on radionuclide scan, or as noted by

Table 8.3 Naval Medical Center femoral neck stress fracture classification

Type	MRI/radiograph feature	Treatment
Compression	No visible fracture	Nonsurgical
	<50 %	Nonsurgical
	>50 %	Surgical
Tension	Any	Individually based decision
Displaced	Any	Emergent surgical fixation
Atypical tensile	Any	Nonsurgical

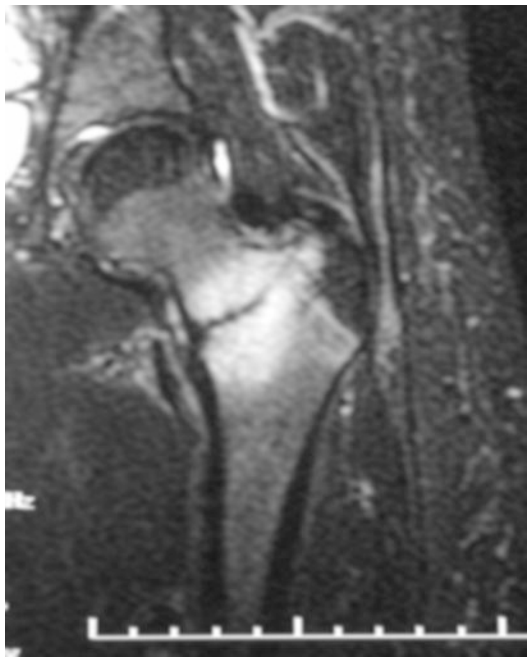

Fig. 8.4 Coronal T2 MRI demonstrating a nearly complete femoral neck stress fracture

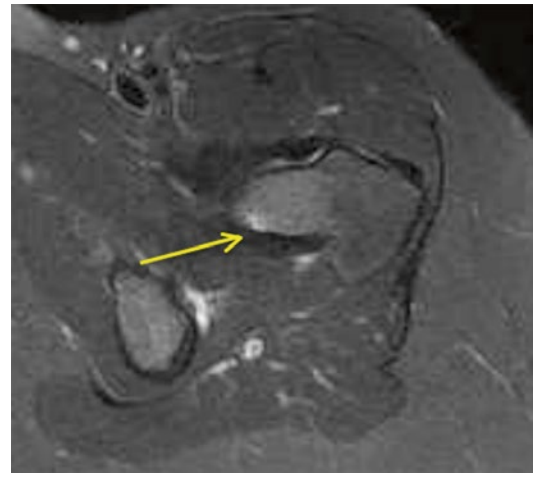

Fig. 8.5 Axial STIR MRI of the proximal femur demonstrating a stress fracture of femoral neck less than 50 % of the total width of the femoral neck (*arrow*)

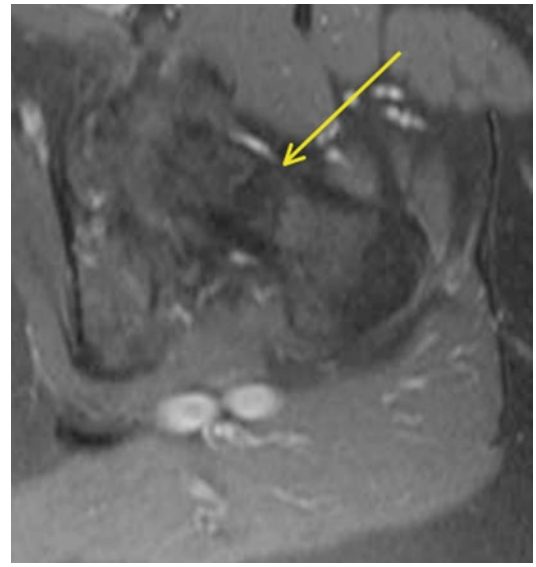

Fig. 8.6 Axial STIR MRI of the proximal femur demonstrating a femoral neck fracture greater than 50 % of the total width (*arrow*)

Devas, it could show callous or sclerosis in this region. In tension-sided stress fractures, radiographs may also be negative, have significant callous formation, or even an overt fracture line on the superior surface of the femoral neck. With the advent of magnetic resonance imaging (MRI) and its increased use in detecting stress fractures in general, Shin et al. at the Naval Medical Center in San Diego further modified the classification scheme to include the use of both MR and plain radiographic features (Table 8.3) [40] (Fig. 8.4). Their classification further subdivided compression type fractures into those with a fatigue line that was less than 50 % of the femoral neck (Fig. 8.5) and those with more than 50 % (Fig. 8.6) of the femoral neck. Tension-sided fractures typically, along with the previously noted radiographic findings, may demonstrate increased signal on T2 or STIR along the superior aspect of the femoral neck.

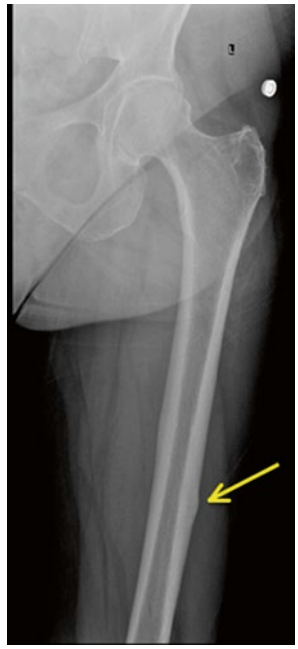

Fig. 8.7 An atypical femur fracture with cortical beaking (*arrow*) on the tension side of the femoral shaft

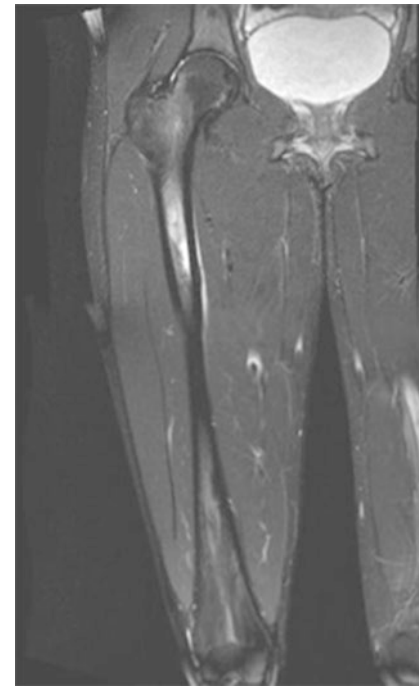

Fig. 8.8 Coronal T2 MRI of the femur demonstrating the common location of femoral shaft stress fractures at the posteromedial junction of the proximal and middle third of the femur

Stress fractures in the subtrochanteric region and shaft are relatively rare. They typically occur in athletes or military recruits, but they may also occur in individuals with osteoporosis and have been correlated with prolonged bisphosphonate use as previously noted (Fig. 8.7). Femoral shaft fractures are typically classified anatomically based upon their location in the femur. They may be divided into proximal third, middle third, and distal third, as well as based on whether they occur on the medial or lateral cortex. The majority of femoral shaft stress fractures, especially in runners, appear to occur in the posteromedial aspect of the femur at the junction of the middle and proximal third of the femoral shaft [6, 41–43] (Fig. 8.8). This is the area of the femur that experiences the greatest compressive forces [44]. Stress fractures in the distal shaft are more commonly seen in military recruits [45]. Distal third of the femur may be further classified into supracondylar, condylar, or subchondral insufficiency type stress fractures, and all of these are quite rare [9, 46].

Diagnosis

The diagnosis of femoral stress fractures includes a thorough history, comprehensive physical examination, and imaging studies. Laboratory studies, although not typically necessary for diagnosis of stress fracture, may help to explain the etiology of an insufficiency or pathologic fracture [47]. Prompt diagnosis of femoral stress fractures requires a high index of suspicion, as many stress fractures can present with vague, nonspecific symptoms. Up to 75 % of femoral stress fractures can be missed or misdiagnosed on initial evaluation [48]. Femoral stress fractures can be mistaken for muscle strains, synovitis, bursitis, infections, or neoplasms [49]. The differential diagnosis for femoral stress fractures can also include slipped capital femoral epiphysis (SCFE), Legg–Calve–Perthes syndrome, or hip dysplasia [10, 49] (Table 8.4). Obtaining a

Table 8.4 Differential diagnosis of femoral stress fractures

Proximal femur	Femoral shaft	Distal femur
• Bursitis • Tendinitis • Avascular necrosis • Muscle strain/injury • Infection • Neoplasm • Snapping hip syndrome • Slipped capital femoral epiphysis (SCFE) • Osteitis pubis • Piriformis syndrome • Sacroiliac injury • Acetabular fracture • Pelvic fracture • Legg–Calve–Perthes syndrome • Hip impingement • Arthritis (degenerative or inflammatory)	• Muscle strain • Muscle or bone contusion • Compartment syndrome • Infection • Neoplasm	• Femoral condyle avascular necrosis • Infection • Muscle strain • Knee arthritis (degenerative or inflammatory) • Knee cartilaginous or ligamentous injury • Patellofemoral pain syndrome • Plica syndrome • Neoplasm

thorough history is key. It is important to gather information regarding the athlete's exercise regimen, dietary intake, history of prior stress fractures, past medical history, and menstrual cycle. Typically athletes with femoral stress fractures will report an insidious onset of gradually worsening leg, thigh, or hip pain that may worsen with activity. Athletes with femoral neck stress fractures may report symptoms as early as 2 weeks after increasing exercise intensity, and typically report pain in the groin [39]. With femoral shaft fractures, athletes often present with activity-related pain in the thigh or ipsilateral knee [45]. Pain may limit athletes from participation in athletic activities, and sometimes may be associated with night pain.

The general location of the athlete's pain may guide physical examination, although the clinical presentation can be variable. A comprehensive physical examination, including the affected limb, contralateral leg, hip, and knee, as well as the pelvis and lumbosacral spine, is required. Athletes with femoral stress fractures may ambulate with an antalgic gait. Typically muscle tone and bulk are normal, and swelling is absent. Overlying skin changes such as erythema and ecchymosis should not be seen. Given the deep nature of the femur, the point of maximal tenderness may be difficult to elicit. Femoral shaft stress fractures may exhibit no tenderness to palpation at all, while more superficial subcondylar fractures may be tender to touch. Deep palpation of the groin may elicit pain in femoral neck stress fractures [39]. Athletes with femoral neck stress fractures may experience pain with logrolling, passive extremes of motion, and straight leg raise. Tuning fork testing, heel striking, and percussion along the femur do not reliably correlate with femoral stress fractures [5].

Athletes with distal supracondylar stress fractures or femoral condyle fractures may exhibit joint line tenderness of the knee and pain with knee range of motion.

Hop testing, where the athlete hops on the affected leg and is asked to localize his or her pain, can be an effective physical examination strategy. An estimated 70 % of athletes with a positive hop test are found to have femoral stress fractures [50]. Fulcrum testing of the femoral shaft can be a useful physical exam tool as well. In fulcrum testing, the athlete sits at the edge of the exam table, with the examiner's hands placing gentle downward pressure on the knee and anterior pressure on the posterior proximal femur [6]. A positive test will induce pain along the femoral shaft (Fig. 8.9).

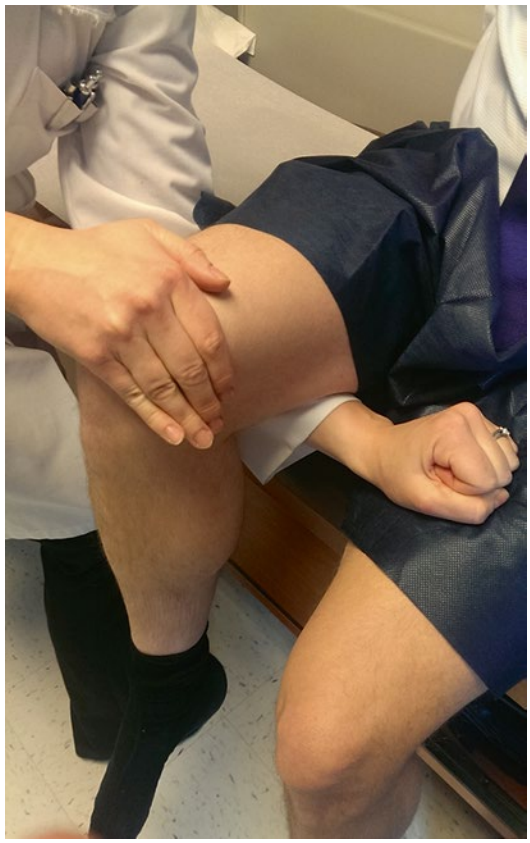

Fig. 8.9 The fulcrum test is used to help detect the presence of femoral shaft stress fracture. The examiner's arm is placed under the proximal thigh of the affected leg and then the other hand applies a downward force to the distal femur. The test is positive if pain is elicited

Imaging

Imaging studies are readily used in the diagnosis of femoral stress fractures. It is the changing structure of bone in response to stress loading that allows imaging studies to aid as diagnostic tools [51]. The most commonly used imaging modalities include plain radiographs, MRI, and nuclear scintigraphy. Conventional radiographs are typically the first imaging studies obtained in the evaluation of suspected femoral stress fractures. Radiographic evidence of stress fracture may be subtle, and in the early stages may include a faint radiolucency in the cortical bone of the femur [31]. With resultant bone modeling, the endosteum may become irregular, thickened, and show signs of sclerotic change (Fig. 8.10). Periosteal reaction involving both the cortex and endosteum follows as the fracture continues to heal. Generally, new periosteal bone formation can be seen about 10 days after the injury process begins, and peak formation occurs at 6 weeks [31, 52]. Often radiographs may appear negative in early stages of the disease process.

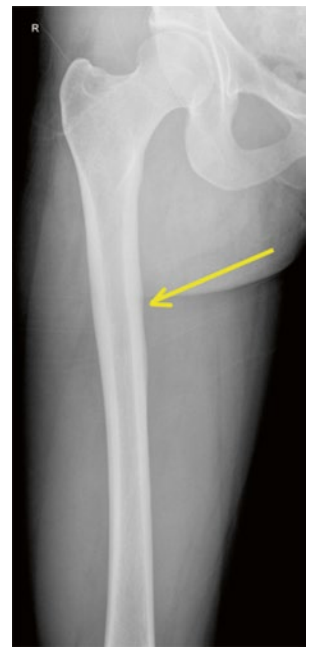

Fig. 8.10 Cortex thickening due to stress fracture of proximal medial femoral shaft (*arrow*)

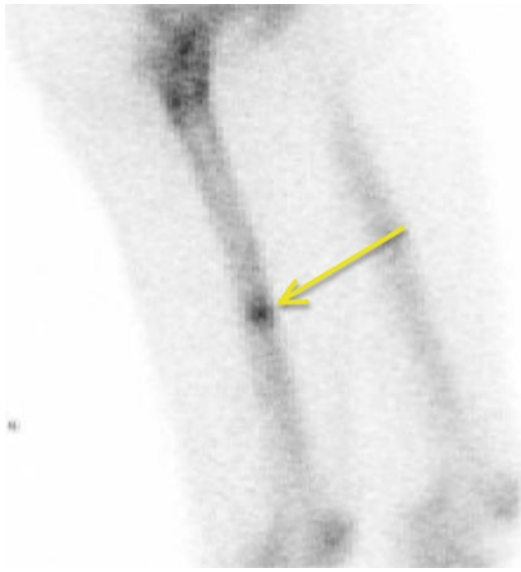

Fig. 8.11 Nuclear scintigraphy (bone scan) showing a stress reaction in the femoral shaft (*arrow*)

A mere 10 % of femoral stress fractures show discrete radiographic evidence within the first week [40]. Obtaining serial radiographs at week 2 or 3 still may not manifest characteristic evidence of fracture healing or new bony callus formation [53]. Therefore, any athlete with persistent femoral pain greater than 2 weeks should be considered highly suspicious for stress fracture despite negative conventional radiographs. In these cases, more advanced imaging techniques are often necessary to confirm the diagnosis.

Nuclear medicine scintigraphy, also called bone scans, may aid in the early diagnosis of femoral stress fractures (Fig. 8.11). Nuclear scintigraphy is sensitive to early bony remodeling changes, and can detect stress fractures or stress reactions within 72 h after the initial injury [54]. In nuclear bone scans, radioactive tracer labeled for bone, typically technetium 99m diphosphonate, is injected intravenously. The patient then undergoes imaging after allowing time for localization of the tracer to areas of bony remodeling. A diagnostic bone scan will show radiotracer uptake in an area of bony remodeling, increased osteoblastic activity, trabecular microfracture, periosteal reaction, or callus formation [55]. Bone scans involve radiation exposure, approximately 44 times that of a standard chest radiograph [3]. This exposure should be considered when choosing an imaging study in the evaluation of a suspected femoral stress fracture. Bone scans, while quite sensitive for bony turnover and remodeling, are not always specific for stress fractures. Any condition producing increased bone turnover, including osteogenic tumors, infection, trauma, or inflammation will result in increased radiotracer uptake [5]. Conversely, negative bone scans can reliably exclude the diagnosis of femoral stress fractures. Nonetheless, bone scans can be a helpful tool in the diagnosis of femoral stress fractures when findings are correlated with plain radiography and a detailed patient history. Roub et al. showed that 20–40 % of athletes undergoing imaging for suspected femoral stress fractures had initial negative conventional radiographs, followed by diagnostic nuclear bone scans showing radiotracer uptake consistent with stress fractures [56]. Therefore, nuclear bone scans are a good second-line imaging modality in the diagnosis of femoral stress fractures, and should be considered in athletes with symptoms or physical examination findings suspicious for stress fracture and non-diagnostic conventional radiographs.

MRI has become an increasingly popular tool in the diagnosis of femoral stress fractures. Like nuclear scintigraphy, MRI can detect early bone marrow changes and bony remodeling related to stress reactions and fractures, thus aiding in prompt diagnosis. Additionally, MRI offers more intricate detail of surrounding soft tissue, and provides information about bone structure and function. Telltale characteristics of femoral stress fractures, including bone turnover and remodeling, periosteal reaction, and fracture lines, are well visualized with MRI. Use of a water-sensitive pulse sequence (e.g., fat suppression) allows for the detection of endosteal bone marrow edema, which is one of the earliest changes seen in stress fractures [57]. Its ability to detect early bony remodeling affords MRI an advantage as compared to conventional radiographs, as it can improve outcomes by facilitating quicker diagnosis and treatment (Fig. 8.12). Advantages of MRI over nuclear scintigraphy include precise

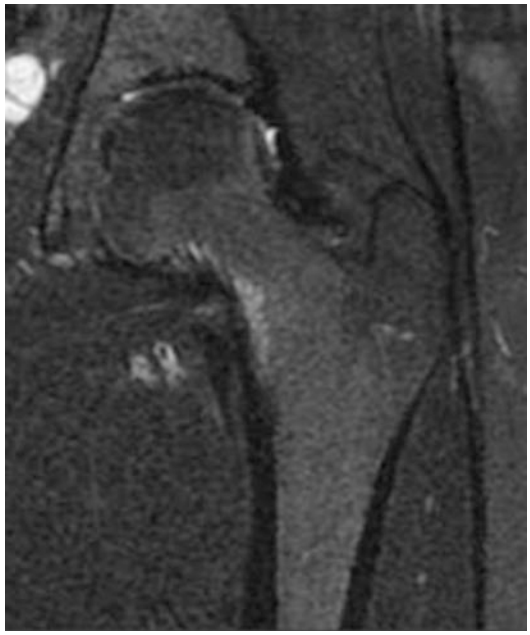

Fig. 8.12 Early detection of medial femoral neck stress reaction with the use of coronal STIR MRI with increased signal over the compression side of the femoral neck

localization of fractures, comprehensive evaluation of surrounding structures to rule out other bone or soft tissue pathology, and the absence of radiation exposure. Given that MRI is sensitive for bone marrow edema, it is important to correlate MRI findings with the athlete's clinical situation. Bone marrow edema may persist after diagnosis and treatment of stress fractures, even as cortical healing continues. Femoral neck stress fractures, even when managed appropriately, may show persistent bone marrow edema on MRI for up to 6 months after initial injury [58]. Also, bone marrow edema may be detected in asymptomatic athletes, such as long distance runners. The clinical relevance of this finding in an asymptomatic athlete is controversial, and may represent pathology or simply physiologic response to bony stresses of exercise. In summary, MRI can provide helpful information in the diagnosis of stress fractures of the femur, particularly when conventional radiographs are equivocal.

Computed tomography (CT) imaging is not as commonly used to diagnose femoral stress fractures. The ability to visualize cross-sectional views of long bones allows CT to demonstrate some characteristics of stress fractures, such as periosteal elevation or fracture line [51]. However, where CT lacks precision is in the determination of the acute or chronic nature of the lesion. Unlike MRI, CT does not have the ability to show bone marrow edema. Therefore, old or quiescent stress fractures may appear more acute or active on CT imaging as compared to MRI or bone scan [40]. High-resolution, thin cut CT may be useful to differentiate osteoid osteoma from stress fracture. Also, CT may be a more helpful tool in the diagnosis of stress fractures of the axial skeleton rather than the femur, and may be useful if MRI is contraindicated [51].

Ultrasonography is another emerging diagnostic tool in the evaluation of stress fractures. In areas such as the distal tibia or metatarsals, the superficial cortex can be visualized using ultrasound, showing cortical buckling and hypoechogenic callous formation [51]. However, the relatively deep anatomical location of the femur often precludes the ability to obtain a clear diagnostic image.

Management

Effective management of femoral stress fractures can be thought of as a continuum, with increasing intensity of intervention. The basis of management of femoral stress fractures lies in prevention. Education of athletes, coaches, athletic trainers, and parents is key to identifying risk factors and preventing the development of stress fractures. Educating athletes as well as those who care for and support them can also lead to efficient and prompt diagnosis of femoral stress fractures once they occur.

Nonoperative management of athletes with femoral stress fractures very often yields excellent outcomes [1–3, 59]. Nonoperative management, while not requiring surgery, still involves active treatment. The mainstay of nonoperative management is rest [43] and activity modification. Depending on the location and severity of the femoral stress fracture, the intensity of rest and off-loading varies from weight bearing as tolerated

to complete bed rest [2, 3]. Crutches are often utilized to aid in off-loading the extremity. Many athletes benefit from a structured program with gradually increasing activity levels. It is often beneficial to have this program monitored and guided by an appropriately trained athletic trainer or physical therapist. Generally, the healing process occurs over a period of 6–8 weeks but may take up to 12 weeks [3, 6]. As the athlete transitions to higher activity levels, careful attention should be paid to pain symptoms. If an athlete experiences increased pain when, for example, transitioning from a non-weight bearing to partial weight bearing status, he or she should not be allowed to progress to this next phase to allow time for additional healing. Provided that the athlete is pain-free, conditioning exercises such as swimming and stationary biking are permitted. It is important to provide structured follow-up clinical examinations and imaging studies to ensure appropriate healing of the fracture.

Correction of training errors or other factors contributing to the development of femoral stress fractures is crucial to effective management. This can be accomplished via subjective means, such as obtaining a thorough history of the athlete's training regimen, or objective means, as with a gait analysis [13]. Female athletes diagnosed with femoral stress fractures should also be evaluated for female athlete triad, including a thorough menstrual history, dietary log, and consideration of bone density testing [14, 16]. Ensuring proper nutrition to provide a positive energy balance is key [16]. Additionally, supplementation of dietary calcium and vitamin D may be helpful in these cases.

Unfortunately there are some cases in which the patient fails conservative treatment. If despite adequate rest and correction of medical and training issues, the patient continues to have pain, or if there is radiographic progression of the fracture, the fracture displaces, or is at high risk for future displacement, operative intervention is typically recommended. The type of intervention typically depends on the type and location of the fracture.

Stress fractures in the proximal femur experience the greatest amount of strain and are at the highest risk for eventual displacement, nonunion, or avascular necrosis. As noted previously, subchondral stress fractures of the femoral head can typically be managed non-operatively with protected weight bearing and rest. However, if there is collapse of the head, these patients will often continue to have pain and disability and may eventually require total hip arthroplasty.

In the femoral neck, due to the high risk for nonunion or osteonecrosis of the femoral head, any displacement of a stress fracture warrants urgent operative treatment. An open anatomic reduction of the femoral neck with placement of either cannulated screws or a dynamic hip screw is recommended [59, 60]. Despite this, there is still a concern that these fractures may lead to osteonecrosis of the femoral head [39]. Other fractures of the femoral neck vary in their need for internal fixation. In most cases, compression type fractures seen on the medial surface of the femoral neck typically can often be managed non-operatively. These fractures typically do not have an identifiable fatigue fracture line on MRI, or if one is present, it involves less than 50 % of the femoral neck. If there is a fatigue fracture line that is greater than 50 % the width of the femoral neck, operative intervention is recommended. In these cases it is recommended that the fracture be stabilized with three percutaneously placed 6.5 or 7.0 mm cannulated screws (Fig. 8.13). The treatment of tension-sided stress fractures of the femoral neck remains somewhat controversial. There have been several studies that have shown

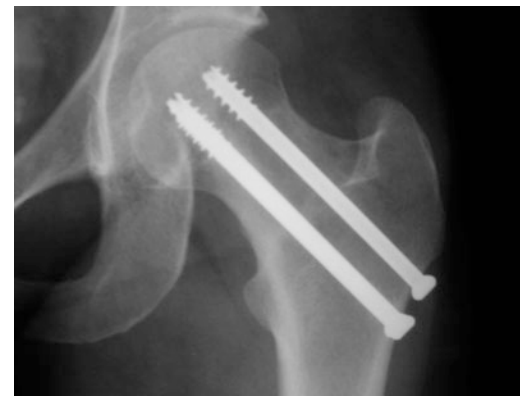

Fig. 8.13 Percutaneous fixation of a non-displaced femoral neck stress fracture with three 6.5 mm cannulated screws

successful conservative management of these high-risk stress fractures; however, due to the potential grave complications with fracture displacement, most recommend early operative intervention [38]. The studies that have shown success with conservative management typically recommend up to 3 weeks of bed rest and up to 14 weeks of protected weight bearing [61]. However while this may be successful in some patients, compliance with strict bed rest and prolonged weight bearing restrictions is very difficult. Unfortunately, if patients are not compliant, or the fracture progresses or displaces, the consequences can be disastrous [11]. We currently recommend early percutaneous fixation of non-displaced tension-sided stress fractures with three percutaneously placed cannulated screws. The procedure has low morbidity and allows early-protected weight bearing with crutches as the fracture heals.

Most subtrochanteric or femoral diaphyseal stress fractures occur in the medial or posteromedial cortex. If caught early, conservative management including rest, protected weight bearing, and time away from the causative activity can be successful [42]. However, due to the difficulty in diagnosis, not infrequently these fractures present as displaced fractures. In these cases, or in cases where progression of a fracture is seen radiographically or the individual continues to have pain despite conservative measures, operative fixation is recommended. In most cases an intramedullary nail fixation is recommended. With atypical femur fractures, as with other fractures of the femoral shaft, if they are caught early, conservative management with protected or non-weight bearing can be attempted. However, if pain persists greater than 3 months or there is radiographic progression or lack of improvement, operative intervention is typically recommended. Intramedullary fixation is often recommended for the surgical treatment of atypical femur fractures (Fig. 8.14). Bisphos-phonates impair osteoclast function and direct bone healing with rigid internal fixation depends on functional osteoclasts. Intramedullary devices are load-sharing devices that allow healing through indirect means and allow early-protected weight bearing.

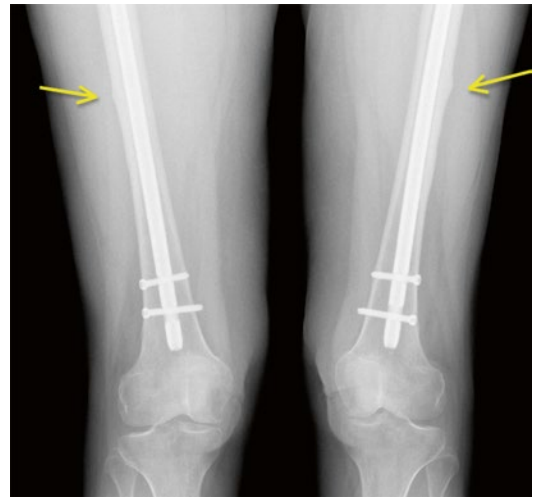

Fig. 8.14 Anteroposterior radiograph demonstrating intramedullary nailing of symptomatic bilateral atypical femur fractures with lateral cortical beaking noted on tension side of femoral shaft (*arrows*)

Return to Sports

After a period of relative rest and conservative management, typically around 8–12 weeks in duration, individuals may consider returning to activity. Before returning to activities they should have no tenderness to palpation and be asymptomatic with full weight bearing [42]. Radiographic evidence of bony healing should also be seen. Once athletes are able to bear weight and progress with daily activities without pain, they can gradually progress back into activity. Early on, if individuals are pain-free, cross-training with activities such as aquatic therapy may be considered to maintain a basic level of fitness. Athletes should be extensively counseled that the return to activity must be very gradual and begin at a much lower intensity than their typical regimen. As mentioned previously, it is vital to correct any errors in training to reduce risk of injury recurrence. If during the return to

training and competition athletes experience any recurrence of pain or discomfort, activity must be curtailed and the causes of the pain investigated.

Summary

Femoral stress fractures are relatively common in individuals that participate in repetitive loading activities such as athletes or military recruits. They can occur anywhere along the length of the femur but occur most commonly in the femoral neck or the proximal shaft. Often stress fractures of the femur can be difficult to diagnose and are attributed to muscle strains or fatigue. Advanced imaging techniques can be very useful to diagnose and help guide treatment, as depending on the location and nature of the fracture, different interventions may be warranted. Non-displaced fractures and low-risk compression type fractures often respond favorably to conservative treatment. All displaced fractures or fractures that are at a high risk for displacement should be treated operatively to help minimize potentially devastating complications. With proper recognition and management, femoral stress fractures can have successful outcomes in the majority of cases.

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Stress Fractures of the Patella

9

Gregory A. Brown, Mark R. Stringer,
and Elizabeth A. Arendt

Introduction

Patella stress fractures were first described by Muller in 1943 [1]. Patella stress fractures are rare and the majority of information regarding these uncommon stress fractures comes from case reports (Table 9.1). Because the focus of this book is stress fractures in athletes, this chapter will focus on the incidence, biomechanics of injury, diagnosis, and management of patella stress fractures in athletes.

Definition

Stress fractures occur because of repetitive loading of a bone. A stress fracture occurs when the repetitive load (stress) is less than the ultimate strength of the bone, as opposed to a traumatic fracture which occurs when a single loading episode exceeds the ultimate bone strength and the bone fractures. In engineering terms, a stress fracture is a fatigue failure that occurs when the repetitive load and cycles exceed the endurance strength of a material. Non-biologic materials, such as metals and polymers, cannot auto-repair so a fatigue failure occurs when the microscopic damage accumulates and results in a macroscopic failure. However, biologic tissues, such as bone, have the potential to heal and repair themselves. Therefore, microscopic damage (microfractures) must accumulate faster than the bone can repair and remodel itself in order for a catastrophic macroscopic failure (displaced fracture) to result.

Mason et al. [2] summarize Daffner and Pavlov's work [3] to describe two types of stress fractures. "There are two types of stress fractures: fatigue fractures and insufficiency fractures. Fatigue fractures occur when increased stresses are applied to normal bone. Insufficiency fractures occur when normal stresses are applied to bone that has been weakened by a generalized disorder such as osteoporosis or osteomalacia." With the possible exceptions of females with the female athlete triad or older athletes with osteoporosis, patellar stress fractures in athletes should be classified as fatigue fractures.

Patellar stress fractures have been described in children with cerebral palsy [4–8]. The presumed mechanism of injury is the increased quadriceps muscle forces required for ambulation with a "crouched" gait. Additionally, many children with cerebral palsy have seizure disorders and certain antiseizure medications can cause osteoporosis. Because this subgroup of

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Table 9.1 Case reports of uncommon patella stress fractures

Author	Year	Sex	Age	Unilateral or bilateral	Fracture type	Fracture location	Sport	Treatment
Devas	1960	M	23	U	Longitudinal	Lateral facet	Running	Fragment excision
Devas	1960	M	28	U	Transverse	Middle 1/3	Running/ hockey	Suture tension band
Sugiura	1977	M	12	U	Transverse	Distal 1/3	Soccer	–
Sugiura	1977	M	17	U	Transverse	Distal 1/3	High jump	–
Hanel	1981	M	15	B	Transverse	Distal 1/3	Basketball	ORIF
Tibone	1981	M	34	B	Transverse	Distal 1/3	Basketball	Fragment excision, patellar tendon repair
Dickason	1982	M	12	U	Transverse	Distal 1/3	Soccer	Cast
Hensal	1983	M	17	B	Transverse	Middle 1/3	Basketball	ORIF K-wire tension band
Iwaya	1985	M	12	U	Longitudinal	Lateral facet	Running	Activity modification
Iwaya	1985	M	11	U	Longitudinal	Lateral facet	Japanese fencing	Activity modification
Iwaya	1985	F	10	U	Longitudinal	Lateral facet	Gymnastics	Activity modification
Dickoff	1987	M	25	U	Longitudinal	–	Running	Activity modification
Schranz	1988	M	27	U	Longitudinal	Lateral facet	Running	Fragment excision
Jerosch	1989	M	20	U	Transverse	Distal 1/3	Soccer	ORIF K-wire tension band
Rockett	1990	M	20	U	Transverse	Distal 1/3	Basketball	Knee immobilizer
Teitz	1992	M	23	U	Transverse	Distal 1/3	Skiing/ sailboarding	ORIF K-wire tension band[a]
Teitz	1992	F	36	U	Transverse	Distal 1/3	Belly dancing	Cast
Oginni	1993	M	29	U	Transverse	Distal 1/3	Palmwine tapper	Fragment excision, patellar tendon repair
Pietu	1995	M	16	U	Transverse	Distal 1/3	Basketball/ skiing	Splint
Garcia Mata	1996	M	23	U	Transverse	Junction middle/ distal 1/3 s	Soccer	Splint/cast
Mason	1996	F	48	U	Transverse	Middle 1/3	Running	ORIF
Mason	1996	F	15	U	Transverse	Distal 1/3	Gymnastics	Knee immobilizer
Mason	1996	M	23	U	Transverse	Middle 1/3	Basketball	Activity modification
Orava	1996	M	25	U	Transverse	Distal 1/3	Volleyball	Cast
Orava	1996	F	19	U	Longitudinal	Lateral facet	Running	Fragment excision
Orava	1996	F	19	U	Transverse	Distal 1/3	High jump	ORIF tension band
Orava	1996	M	22	U	Transverse	Distal 1/3	Soccer	ORIF K-wire tension band
Orava	1996	F	21	U	Transverse	Distal 1/3	Orienteering	ORIF tension band
Brogle	1997	M	22	U	Transverse	Distal 1/3	Basketball	ORIF cannulated screws
Garcia Mata	1999	M	12	U	Transverse	Junction middle/ distal 1/3 s	Soccer	ORIF screw fixation
Mayers	2001	M	21	U	Transverse	Distal 1/3	Weight lifting	ORIF tension band
Crowther	2005	M	35	U	Transverse	Distal 1/3	Tennis	ORIF K-wire tension band
Carneiro	2006	M	64	B	Transverse	Middle 1/3	Running	ORIF K-wire tension band
Keeley	2009	M	20	U	Transverse	Distal 1/3	Basketball	ORIF single cortical screw
Sillanpaa	2010	F	18	U	Longitudinal	Lateral facet	Floorball/ floor hockey	Absorbable cross pins

[a]Patient required second ORIF procedure with screw and K-wire tension band

patients are not usually competitive athletes, the diagnosis and management of this patient subgroup will not be included in this chapter.

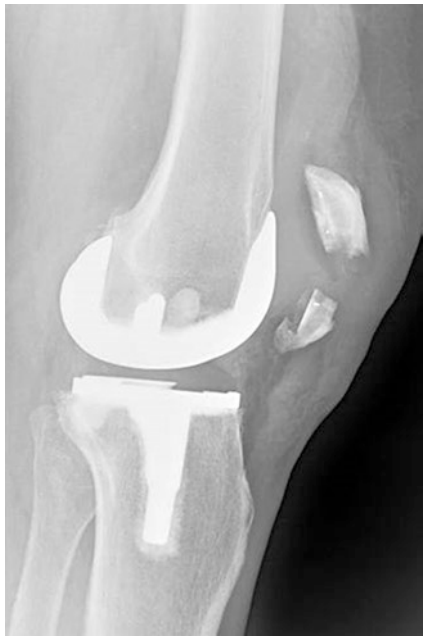

Fig. 9.1 Lateral radiograph showing an atraumatic transverse patella fracture after a total knee arthroplasty. Published with kind permission of © Gregory A. Brown 2014

Patella stress fractures after total knee arthroplasty have been described [7, 9]. It is likely that such fractures are insufficiency fractures due to osteonecrosis of the patella (Fig. 9.1). This patient fell after he felt a "pop" and his knee buckled. The fracture was treated with a locked reconstruction plate (Fig. 9.2).

Patellar stress fractures have been described after previous surgical procedures involving the patella. Boden and Osbahr [7] describe patellar stress fractures after harvesting bone-patellar tendon-bone grafts for anterior cruciate ligament reconstruction. The stress concentration effect of the bony defect increases the effective stress at the site of the bony defect resulting in a stress fracture. Gregory et al. [10] reported three cases of longitudinal patella stress fractures after transosseous extensor mechanism repair. The stress concentration effect of the longitudinal drill holes increased the effective stress in the patella causing a stress fracture. MRI images of one of the cases demonstrated tunnel enlargement or cystic changes that would further increase the stress concentration effect. Thaunat and Erasmus [11] reported on three cases of avulsion fractures of the medial patella after previous medial patellofemoral ligament reconstruction. However, all

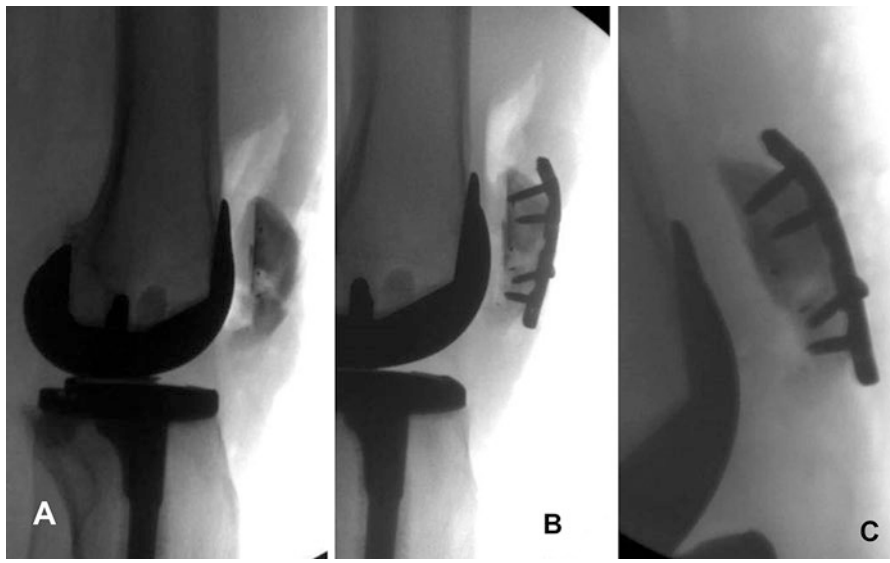

Fig. 9.2 Lateral radiographs depicting (**a**) provisional reduction with cerclage suture; (**b**) patella fracture short segment fixation with a locking reconstruction plate; and (**c**) healed fracture 3 months postoperatively. Published with kind permission of © Gregory A. Brown 2014

three events were associated with trauma, so the designation of this being a stress avulsion fracture could be debated.

A bipartite patella arises when a secondary patella ossification center fails to unite with the primary ossification center. According to the Saupe classification as cited by Gaheer et al. [12], there are three types of bipartite patella: (1) inferior pole (Type I, 5 %), (2) lateral margin (Type II, 20 %), and (3) superolateral pole (Type III, 75 %). The junction of the bipartite patella fragment may become painful due to repetitive stress [13] and may be considered a "stress" response. If the superolateral Type III bipartite patella becomes persistently painful and does not respond to non-operative management, it can be excised and the quadriceps tendon repaired to the patella. Okuno et al. [14] reported four cases of traumatic separation of a Type I bipartite patella. Two cases were treated with open reduction and internal fixation and two cases were treated with cast immobilization.

Incidence

The incidence of patella stress fractures is exceedingly rare. Two retrospective series [15, 16] and one prospective series [17] of stress fractures in athletes do not report patella stress fracture incidence (presumably due to the rarity of the diagnosis). The largest series [16] notes the anatomic distribution of stress fractures is tibia (49.1 %), tarsals (25.3 %), metatarsals (8.8 %), femur (7.2 %), fibula (6.6 %), pelvis (1.6 %), sesamoids (0.9 %), and spine (0.6 %). Wall and Feller [18] report a German series by Csizy, Babst, and Fridrich with an anatomic distribution of tibia (33 %), navicular (20 %), metatarsals (20 %), femur (11 %), fibula (7 %), and pelvis (7 %). The incidence of patella stress fractures is so rare; neither series delineates patella stress fractures. However, Iwamoto and Takeda [15] include a review of four series that delineate patella stress fractures: Sugiura (2/162 = 1.2 %), Tajima (2/111 = 1.8 %), Sakai (1/251 = 0.4 %), and Iwamoto (1/196 = 0.5 %). The pooled incidence is 0.83 % (6/720). Therefore, the incidence of patella stress fractures appears to be less than 1 %. Given the percentage of sports medicine patients with stress fractures presenting to a sports medicine clinic is approximately 2 % (196/10,276 = 1.91 %) [15] and patellar stress fractures comprise less than 1 % of stress fractures, the probability of seeing a patient with a patella stress fracture is less than 0.02 % (2/10,000).

Because the incidence of patellar stress fractures is so low, all case reports published in English are tabulated in Table 9.1. Twenty-three authors reported on 35 cases (Devas—2 cases [19], Suguira as reported by Mason [2]—2 cases [20], Hanel—1 case [21], Tibone—1 case [22], Dickason—1 case [23], Hensal—1 case [24], Iwaya—3 cases [25], Dickoff—1 case [26], Schranz—1 case [27], Jerosch—1 case [28], Rockett—1 case [29], Teitz—2 cases [30], Oginni—1 case [31], Pietu—1 case [32], Garcia Mata—2 cases [33, 34], Mason—3 cases [2], Orava—5 cases [35], Brogle—1 case [36], Mayers—1 case [37], Crowther—1 case [38], Carneiro—1 case [39], Keeley—1 case [40], and Sillanpaa—1 case [41]). Table 9.1 includes the case report author, year of publication, patient sex, patient age, unilaterality or bilaterality of fracture, fracture type (transverse or longitudinal), fracture location, sport, and treatment.

Patellar stress fractures appear to be more common in males (relative risk = 3.4). The incidence of patella stress fractures in males is 77.1 % (27/35) and in females is 22.9 % (8/35). Iwamoto and Takeda [15] report nearly equal incidence of male and female stress fractures in sports-related injury clinic visits in their 10-year retrospective series: male 1.95 % (125/6,415) and female 1.84 % (71/3,861). Similarly, Bennell et al. [17] report nearly equal incidence of male and female stress fractures in their prospective series of track and field athletes: male 20.4 % (10/49) and female 21.7 % (10/46).

Patella stress fractures are usually unilateral (89 %—31/35), as opposed to bilateral (11 %—4/35). The mean age is 22.7 years with a standard deviation of 10.8 years. Transverse patellar stress fractures (77 %—27/35) are more common than longitudinal stress fractures

(23 %—8/35). The most common sports played by athletes developing patella stress fractures are running (23 %—8/35), basketball (23 %—8/35), and soccer (17 %—6/35). Of note, longitudinal stress fractures are associated with running. Five of the eight longitudinal patella stress fractures were in runners as opposed to three of the transverse stress fractures being in runners. This was statistically significant ($p = 0.0074$).

Twenty of the 27 transverse fractures were in the distal 1/3 of the patella. Two were at the junction of the middle and distal thirds. Five were in the middle 1/3 of the patella. No patella stress fractures occurred in the proximal 1/3 of the patella. All of the longitudinal stress fractures were in the lateral facet.

Patella stress fractures were treated with multiple methods. In general, non-displaced or minimally displaced fractures were treated with activity modification and/or splinting or casting. Displaced small fragments that were too small for internal fixation were excised and the patellar tendon or lateral retinaculum was repaired. Displaced fractures with larger fragments were internally fixed with a variety of techniques including suture tension band, wire tension band, K-wire tension band, cannulated screw tension band, and screws only. All authors reported return to activities after healing. Patients typically return to sports at near pre-fracture levels between 3 and 6 months [40]. One case required a second operation for internal fixation [30].

Biomechanics of Injury

Matheson et al. [16] and Keeley et al. [40] reviewed the two theories explaining the etiology of stress fractures. The first theory is that muscle fatigue reduces the shock absorption of the muscles allowing the bones to be subjected to higher stresses. Because the patella is a sesamoid bone and the patellar forces are directly related to the quadriceps force, a reduction in the quadriceps force would reduce the stresses on the patella (see section "Biomechanical Analysis"). Thus, it is unlikely that this first theory applies to patella stress fractures.

The second theory asserts that direct repetitive muscle forces cause patella stress fractures [42]. Since the patellar tendon force and patellofemoral joint reaction force are both related to the quadriceps muscle force, patellar stress fractures are the result of direct repetitive tensile and bending forces generated by the quadriceps musculature. Pietu and Hauet [32] described two types of activities that cause patellar stress fractures. Repetitive sudden forceful contractions as used in jumping (basketball) or kicking (soccer) are one type of activity that results in stress fractures, and this is consistent with the case report analysis noted above. A lower force with greater frequency, such as running, could also cause a stress fracture.

The biomechanics of the patellofemoral joint were described by Terry [43]. The patellofemoral joint reaction force is:

$$F_{pf} = \sqrt{F_q^{\,2} + F_{pt}^{\,2} - 2F_qF_{pt}\cos\theta}$$

where F_{pf} is the patellofemoral joint reaction force, F_q is the quadriceps muscle force, F_{pt} is the patellar tendon force, and θ is the knee flexion angle. If one assumes that the patellar tendon force is equal to the quadriceps muscle force, the equation simplifies to:

$$F_{pf} = 2F_q \sin\theta/2$$

This assumption is reasonable because the ratio of the quadriceps muscle force and patellar tendon force ranges from 0.7 to 1.3 [44].

The patellar tendon inclination angle (PaTIA) provides apparent flexion to the knee joint when the knee is fully extended. Thus, a more accurate estimate of the patellofemoral joint reaction force is (Fig. 9.3):

$$F_{pf} = 2F_q \sin\left(\text{PaTIA}/2 + \theta/2\right)$$

where PaTIA is calculated as follows:

$$\text{PaTIA} = \sin^{-1}[(\text{Patellar thickness} - \text{Tibial tubercle height})/\text{Patellar tendon length}].$$

The sagittal plane force balance of the patella is as shown in Fig. 9.4. The patella is subjected to a bending stress by the posterior components of the quadriceps muscle force and patellar tendon

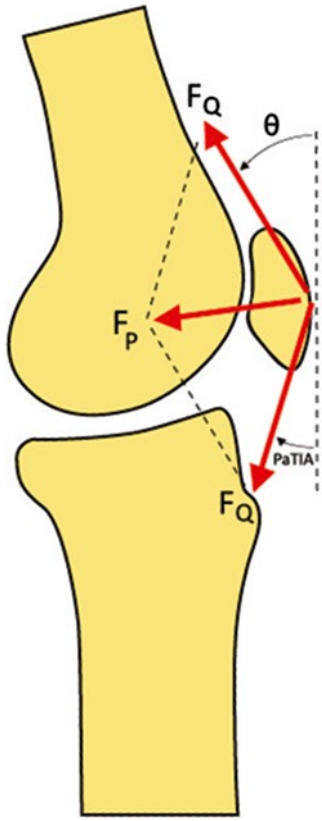

Fig. 9.3 Schematic demonstrating the patellofemoral joint reaction force resulting from quadriceps muscle contraction with knee flexion (θ) and patellar tendon inclination angle (PaTIA). Published with kind permission of © Gregory A. Brown 2014

force with the patellofemoral joint reaction force as the fulcrum in the center of the patella. The moment at the midpoint of the patella is:

$$M = \left(F_{pf}/2\right)\left(l_{pat}/2\right) = F_{pf}l_{pat}/4$$

where l_{pat} is the patella length. The cross-sectional area of the patella is $A_{pat} = 3t_{pat}w_{pat}/4$, the area centroid is $c = 7t_{pat}/18$ from the anterior surface of the patella, and the area moment of inertia is $I_{pat} = 37t_{pat}{}^3w_{pat}/864$. The patella thickness is t_{pat} and the patella width is w_{pat}. Then the maximum bending tensile stress is on the anterior surface of the patella.

$$\sigma = Mc/I = \left(84F_{pf}L_{pat}\right)/\left(37t_{pat}^2w_{pat}\right)$$

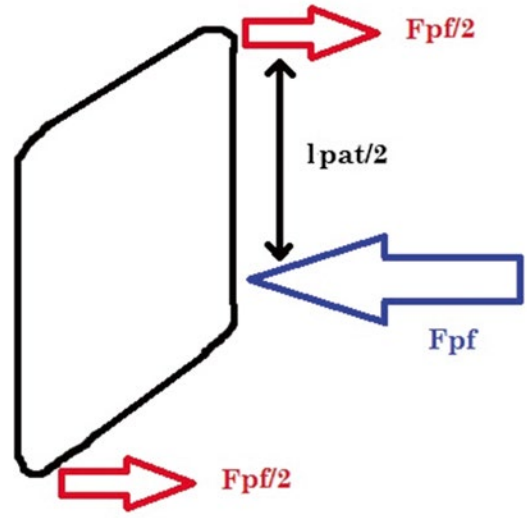

Fig. 9.4 Schematic showing patella bending moment resulting from the patellofemoral joint reaction force (F_{pf}) and posteriorly directed quadriceps muscle force component ($F_{pf}/2$) and posteriorly directed patellar tendon force ($F_{pf}/2$). Published with kind permission of © Gregory A. Brown 2014

Since the patella width is approximately equal to the patella length, the stress calculation can be further simplified to

$$\sigma = \left(84F_{pf}\right)/\left(37t_{pat}^2\right)$$

Additionally, there is a tensile stress to the patella by the vertical components of the quadriceps muscle force and the patellar tendon force (Fig. 9.5).

$$F_{vert} = F_q\cos\left(\text{PaTIA}/2\theta/2\right)$$

The tensile stress is $\sigma = F_{vert}/A_{pat}$ and is added to the bending stresses to obtain the total tensile stress on the anterior surface of the patella. Boden and Osbahr [7] report that it has been postulated that transverse patellar stress fractures are initiated on the anterior surface of the patella and these biomechanical calculations confirm that theory.

As mentioned previously, the majority of transverse patella fractures occur in the distal 1/3 of the patella. Since the maximum bending stress occurs at the fulcrum of the patellofemoral reaction force, this force must be located in the distal

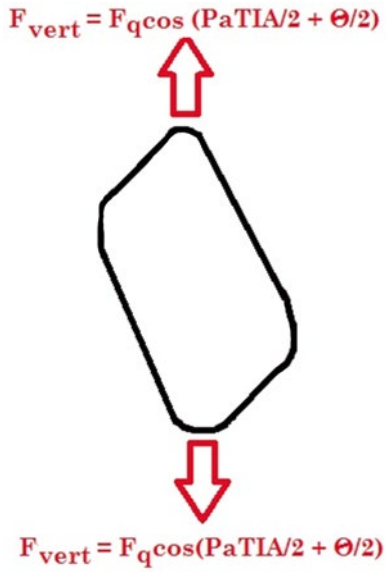

Fig. 9.5 Schematic showing patella tensile forces resulting from the proximally directed quadriceps force (F_{vert}) and the inferiorly directed patellar tendon force (F_{vert}). Published with kind permission of © Gregory A. Brown 2014

1/3 of the patella. Lengsfeld et al. [45] note that the articulating point of the patella moves from the distal end to the proximal end of the patella during flexion. Therefore, most patellar stress fractures must develop from excess loading/stress in early flexion.

Longitudinal patellar stress fractures have a different loading pattern. If the lateral retinaculum and iliotibial band is tight, the patellofemoral joint reaction force is shifted into the lateral trochlea and lateral patellar facet. This creates a bending moment in the sagittal plane of the patellar lateral facet (Fig. 9.6). The maximum tensile stress would be in a longitudinal line on the anterior surface of the patella. Anecdotal evidence may support this theory. Devas [46] reported needing to excise a longitudinal stress fracture fragment because the fragment could not be reduced. Presumably the fragment could not be reduced because the lateral retinaculum was too tight to allow reduction. Keeley et al. [40] reported performing an iliotibial band release as an adjunct to open reduction and internal fixation of a patella stress fracture, but the stress fracture was a transverse stress fracture.

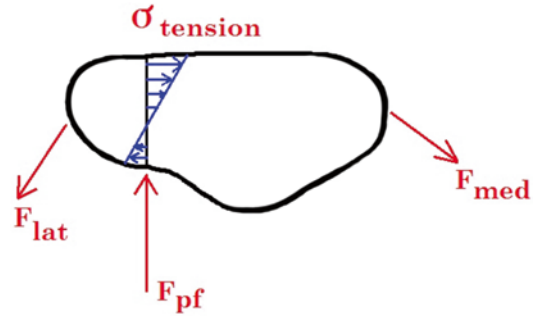

Fig. 9.6 Schematic demonstrating theoretical bending and tensile stresses that give rise to longitudinal patella stress fractures. Published with kind permission of © Gregory A. Brown 2014

The high incidence of longitudinal stress fractures in runners may be correlated with a tight iliotibial band.

Diagnosis

Because of the high risk for patella stress fracture displacement and/or delayed union [7, 47], the diagnosis of patella stress fracture must be considered in the differential diagnosis for anterior knee pain in athletes. In order to make this rare diagnosis, a high index of suspicion is needed to confirm the diagnosis. In particular, Iwamoto et al. [48] noted the difficulty of distinguishing diagnoses of Sinding-Larsen-Johansson syndrome from osteochondritis, stress fracture, or tendonitis.

The typical history for a patella stress fracture is gradually worsening anterior knee pain over weeks to months. Athletes may report a sensation of a "pop" or "crack" if the stress fracture displaced acutely. Their training or exercise regimen may be increasing in intensity and usually includes repetitive jumping (basketball), kicking (soccer), or running activities [40] (Fig. 9.7a, b). Physical examination demonstrates tenderness to palpation in the distal 1/3 of the patella (transverse fracture) or along the lateral border of the patella (longitudinal fracture). There may be minimal or no swelling. There may be an effusion (hemarthrosis) if the stress fracture has acutely displaced.

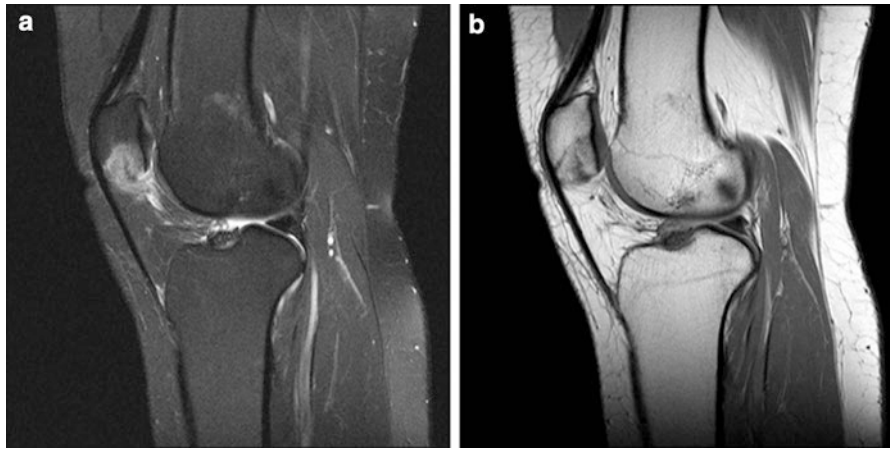

Fig. 9.7 T2 (**a**) and T1 (**b**) sagittal MRI scans of the knee of a 35-year-old female runner with chronic anterior knee while training for a marathon. Images demonstrate a non-displaced midbody to inferior patella stress fracture. Published with kind permission of © Elizabeth A. Arendt 2014

Radiographic evaluation of the painful knee begins with three views (anteroposterior, lateral, and sunrise). A transverse fracture is best seen on the lateral view. A longitudinal fracture is best seen on the sunrise view. The anteroposterior view is best for differentiating a bipartite patella. Because patellar stress fractures are so rare, the sensitivity and specificity of radiographs is low for stress fractures in general. Initial radiographs are sensitive in one-third to one-half of the cases [49]. If there is a fracture line present, the sclerotic edges are likely to be present.

Historically, bone scintigraphy was used to diagnose stress fractures. Bone scans are very sensitive for stress fractures but not very specific [50]. As MRI has become readily available, MRI has become the imaging modality of choice, particularly for periarticular pain when the soft tissues can be imaged as well as the bone. Arendt and Griffiths [51] describe a radiographic grading system for stress fractures for radiographs, bone scans, and MRI (Table 9.2).

As noted in section "Definition" of this chapter, a bipartite patella is not a patella stress fracture though stress reaction may be present. Typically, the location of the secondary ossification center and the radiographic characteristics of the unfused zone allow for differentiating a patellar stress fracture from a bipartite patella. This distinction is important because the secondary ossification center may require excision. Fixation may not result in fusion of the secondary ossification center to the patella and therefore be unsuccessful in resolving anterior knee pain.

Management

Management of a patellar stress fracture is relatively straightforward once the diagnosis has been confirmed. Treatment depends on the displacement of the fracture and type of fracture. If the stress fracture is non-displaced (transverse or longitudinal), the recommended treatment is rest, activity modification with possible immobilization if compliance is a concern. The timing for return to activities is noted in Table 9.2.

For displaced transverse fractures, a K-wire tension band or cannulated screws tension band is the preferred technique. If using a K-wire tension band, consider using a "lazy" or horizontal figure of eight pattern for the 18 gauge wire. The horizontal figure of eight configuration provides 2–3 times more compression than the vertical figure of eight configuration depending on the angle of the oblique crossing wire.

Table 9.2 Radiologic grading of stress fractures [51]

	X-ray	Bone scan	MR image	Treatment
Normal	Normal	Normal	Normal	None
Grade 1	Normal	Poorly defined area of increased activity	Positive STIR image	3 weeks rest
Grade 2	Normal	More intense but still poorly defined	Positive STIR plus positive T2	3–6 weeks rest
Grade 3	Discrete line (?); discrete periosteal reaction (?)	Sharply marginated area of increased activity focal or fusiform	Positive T1 and T2, but without definite cortical break	12–16 weeks rest
Grade 4	Fracture or periosteal reaction	More intense transcortical localized uptake	Positive T1 and T2 fracture line	16+ weeks rest

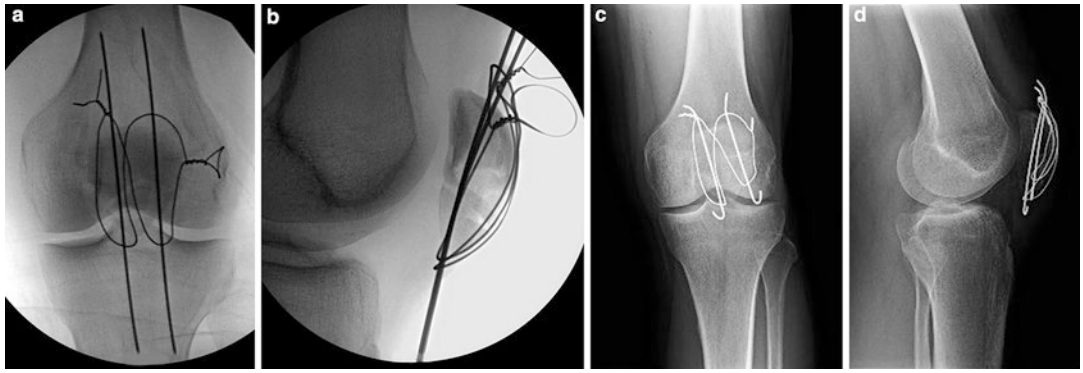

Fig. 9.8 Intra-operative C-arm images of a horizontal figure of eight tension band technique: (**a**) anteroposterior and (**b**) lateral. Postoperative radiographs of a healed patella fracture treated with a horizontal figure of eight tension band technique: (**c**) anteroposterior and (**d**) lateral. Published with kind permission of © Gregory A. Brown 2014

$$F_{\text{horizontal}} / F_{\text{vertical}} = \left(1+\cos\theta\right) / \left(\cos\theta\right)$$

An example of the horizontal figure of eight configuration for a traumatic patella fracture is provided in Fig. 9.8.

It is the author's experience (GAB) that K-wire tension bands irritate the surrounding soft tissues and usually require removal. The author's preferred technique is to use a cannulated screw tension band technique. For distal 1/3 transverse fractures, the cannulated screws are inserted from distal to proximal (retrograde). Partially threaded screws that are 4–6 mm short of the full length of the patella are selected (Fig. 9.9). Shorter screws have two important benefits: (1) if the screw is too long, the 18-gauge wire is bent over the self-cutting/self-tapping edge of the screw and the 18-gauge wire will fatigue and break prematurely; and (2) if the screw is long, the vertical limb of the tension band will compress the screw axially, but provide no additional compression across the fracture site.

For transverse fractures with a fragment too small for fixation, the fragment should be excised, and a patellar tendon repair with transosseous tunnels should be performed.

For small displaced longitudinal fractures, excise the fragment. After evaluating the lateral soft tissues, consider a lateral retinacular release or lengthening. An important surgical point is to assure no continued tightness of the lateral peri-patella soft tissue structures.

For large displaced longitudinal fractures, internal fixation of the fragment with cannulated screws from lateral to medial is recommended. Given the relatively small medial to lateral soft tissue tension, a tension band wire is not required. As with small longitudinal fractures, after evalu-

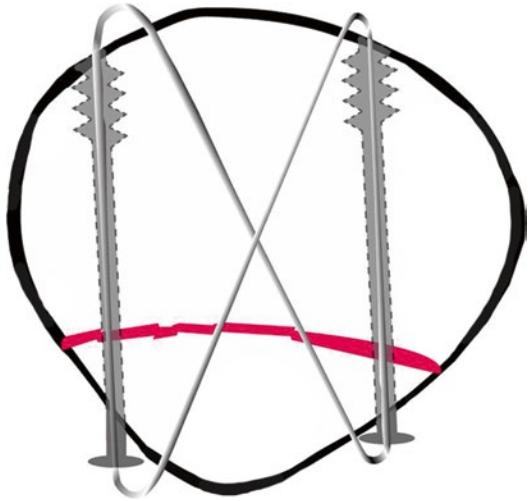

Fig. 9.9 Schematic demonstrating cannulated screws and tension band technique for distal 1/3 patella stress fracture. Screws should be inserted retrograde (distal to proximal) so the threads do not cross the fracture. Screws should be 5–10 mm short so the tension band wire can add additional compression. Published with kind permission of © Gregory A. Brown 2014

ating the lateral soft tissues, a lateral retinacular release or lengthening should be considered as discussed above.

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Stress Fractures of the Tibia

10

Joshua D. Harris and Kevin E. Varner

Introduction

Stress fractures are common overuse injuries. Repetitive high-intensity or extended duration of training places athletes at risk for stress fractures [1]. Stress fractures are most commonly observed in the tibia (24 %), tarsal navicular (18 %), metatarsal (16 %), fibula (16 %), and femur (7 %) [2]. Athletes in cross country, track and field, recreational and competitive running, triathlon, soccer, basketball, and dance are at risk for tibial stress fractures [2]. Military recruits are also at risk for overuse injury, including stress fracture of the tibia [3]. History, physical examination, and imaging studies are essential to quickly and efficiently make the diagnosis of tibial stress fracture. The main differential diagnosis includes tibial stress fracture, medial tibial stress syndrome ("shin splints"), posterior tibial tendinopathy, exercise-induced compartment syndrome, and popliteal artery entrapment.

Diagnosis of tibial stress fractures may be challenging. However, after the diagnosis is made, often times the appropriate treatment may be even more difficult. For high-risk stress fractures, the physician–patient or physician–athlete discussion frequently extends beyond that of the history, physical examination, and imaging studies. A discussion is warranted with the athlete and sometimes the parents, spouse, coach, trainer, agent, and team. The conversation often entails specific events, tournaments, scholarships, salaries, prize money, and endorsements. Further, dialogue regarding in-season/out-of-season timing, athletic career timing, and length of non-operative versus operative management is posed. Fully informed consent allows for proper treatment decision-making. Successful treatment may only be obtained with a proper non-operative or surgical plan and a compliant patient. It is the clinician's responsibility to be fully informed and disclose to the patient the length of time for each possible arm of the treatment algorithm. Although the decision is clearly multifactorial, it is of paramount importance that the clinician, at all times, keep the patient's best interests in mind.

Relevant Anatomy and Biomechanics

The tibia is the second longest long bone in the human body. It is the primary weight-bearing bone in the leg (up to 93 % load transmission; 7 % via the fibula) [4]. Its strong diaphysis is composed of thick cortical walls and is triangular in cross-sectional area, with proximal and distal metaphyseal and epiphyseal flared expansions. The proximal tibial plateau is covered with hyaline

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articular cartilage and by the medial and lateral menisci, which are separated by the medial and lateral tibial spines. The medial plateau is more concave and congruent with the femoral condyle than the more convex lateral plateau, which may influence articular cartilage defect mechanics and subsequent surgical decision-making. The posterolateral aspect of the proximal tibia articulates with the anteromedial aspect of the proximal fibular head at the proximal tibiofibular joint. The interosseous membrane is a thick band of fibrous connective tissue bridging the tibia and fibula distally to the ankle syndesmosis (inferior tibiofibular) complex. The distal tibial plafond is also covered with hyaline articular cartilage, articulating with both the talar dome and fibular (incisura) articular surfaces. From the distal medial tibia projects the medial malleolus, a rigid bony stabilizer of the ankle mortise and the origin for the stout deltoid ligamentous complex. The soft tissue coverage of the tissue is asymmetric, with abundant muscular coverage laterally and posteriorly. However, the anteromedial aspects are quite deficient and exposed with only skin, minimal subcutaneous tissue, and periosteum coverage.

The biomechanics of the tibia are primarily dependent upon the knee and ankle articulations and bridging musculotendinous units. Proximally, the "screw-home mechanism" describes the relative tibiofemoral rotation that occurs during the final 20–30° of knee extension, as the tibia rotates externally relative to the femur [5]. Upon initial knee flexion, the mechanism reverses and the tibia rotates internally. Distally, the ankle mortise is highly congruous with intimate articulation between the tibia, fibula, and talus in all positions of ankle dorsiflexion and plantarflexion. Ankle dorsiflexion is coupled with talar external rotation and fibular posterolateral translation and external rotation. Ankle plantarflexion is coupled with internal talar rotation. Even small amounts of ankle mortise disruption can lead to dramatic increases in articular contact pressures (one millimeter of lateral talar displacement reduces the articular contact area by 42 %, with subsequent increase in contact pressure and supraphysiologic articular cartilage load and wear) [6]. In addition to articular biomechanics, the powerful gastrocnemius-soleus complex, with explosive ankle plantarflexion seen in jumping sports, places a large tensile stress on the anterior tibia, and a subsequent anterior bend and convexity. This, coupled with relative anterior tibial hypovascularity, is a clear example of the potential risk for anterior tibia stress fractures in which abnormal stress can weaken normal bone. Similarly, a pronated foot in endurance athletes, including runners, with weak subtalar inversion may permit excessive proximal rotational torque at the tibia, with a consequent increase in risk of tibia stress injury and medial tibial stress syndrome.

Medial tibial stress syndrome is a commonly encountered diagnosis in the overuse injury evaluation of the endurance athlete. Although the evidence suggests that medial tibial stress syndrome may exist along a continual spectrum with that of tibial stress fracture, this is debatable and definitively unproven [7]. Nonetheless, medial tibial stress syndrome is essentially a periostitis of the tibia due to the tensile pull of the posterior calf musculature, with coexistent tendinopathy, periosteal remodeling, and stress reaction of the tibia [8].

Definition and Classification

Stress fractures represent a wide spectrum of bone injury. They are characterized by an imbalance between bone breakdown and repair. The continuum of injury begins with the inability of the bone to repair microcracks that occur with overuse. As the microcracks increase in both absolute number and size, a distinct "macrocrack," or fracture, may occur and be visualized with imaging studies. Further, nondisplaced fractures may eventually displace and some fractures may even progress to nonunion. This variable spectrum of stress responses in bone is best illustrated in the Kaeding–Miller classification of stress fractures (Table 10.1).

Grades I and II injury lack a distinct fracture line. However, they do have imaging evidence of stress reaction (hyperintensity on magnetic resonance imaging T2-weighted and STIR [short-tau inversion recovery], increased uptake on technetium-99m-labeled methylene diphosphonate

Table 10.1 Kaeding–Miller stress fracture classification system[a]

Grade	Pain	Imaging findings	Description
I	No	Imaging evidence of stress fracture, no fracture line	Asymptomatic stress reaction
II	Yes	Imaging evidence of stress fracture, no fracture line	Symptomatic stress reaction
III	Yes	Non-displaced fracture line	Non-displaced fracture
IV	Yes	Displaced fracture	Displaced fracture
V	Yes	Nonunion	Nonunion

Radiographic findings may be from plain radiographs, magnetic resonance imaging (MRI), computed tomography (CT), and bone scan

[a]Reproduced with permission from Journal of Bone and Joint Surgery American. July 3, 2013;95(13):1214–20. The Comprehensive Description of Stress Fractures: A New Classification System. Kaeding C, Miller T

bone scan [triple phase bone scintigraphy], or cortical thickening and sclerosis on plain radiographs and computed tomography [CT]). The difference between Grade I and II is the absence (Grade I) or presence (Grade II) of symptoms. Visualization of a fracture line on imaging makes the fracture a Grade III or higher. The difference between Grade III and IV is the absence (Grade III) or presence (Grade IV) of fracture displacement. A Grade V injury represents fracture nonunion.

Stress fractures may additionally be dichotomized into "low-risk" and "high-risk" [9]. High-risk stress fractures are those prone to displacement, nonunion, delayed union, and re-fracture. High-risk fractures present a challenging situation, often requiring either surgery or a lengthy duration of nonsurgical management, which, for some competitive athletes, may be potentially career-ending. High-risk fractures include anterior tibial diaphysis, femoral neck tension side, patella tension side, talar neck, proximal fifth metatarsal, central dorsal tarsal navicular, medial malleolus, and hallucal sesamoids [9]. Not all stress fractures of the tibia, however, are high risk. Posteromedial tibial stress fractures, frequently seen in runners and other endurance athletes, as opposed to the tension-sided anterior cortical fracture, are on the compression side of the tibial shaft and respond more favorably to nonsurgical treatment.

Table 10.2 Risk factors for fatigue tibial stress fractures in runners

- Rapid increase in training intensity
- Rapid increase in training mileage
- Leg-length discrepancy
- Knee stiffness
- Increased hip adduction
- Subtalar eversion and foot pronation
- Pes cavus
- Female athlete triad
- Hard running surfaces
- Old, worn running shoes

Risk Factors

Stress fractures may occur in two distinct scenarios: abnormal stresses applied to normal bone (fatigue) or normal stresses applied to abnormal bone (insufficiency). Thus, risk factors are based upon these two inciting causes. Insufficiency fractures may commonly be due to metabolic bone disease, endocrinopathy, chronic renal disease, post-radiation therapy, smoking, infection, and benign or malignant bone tumors (pathologic fracture). These fractures are frequently observed in the sacrum, vertebral bodies, pubic rami, calcaneus, and proximal femoral diaphysis [10]. Fatigue fractures are overuse injuries. Thus, training errors, competition, nutrition, equipment, extremity biomechanics, and bony alignment all play significant roles in the development of fatigue stress fractures (Table 10.2). Girls and women with the "female athlete triad" are especially at risk for bone stress injuries, including both stress reaction and stress fracture [11]. They often exhibit characteristics of both fatigue and insufficiency fracture, with amenorrhea or oligomenorrhea, overall energy deficiency imbalance, and low bone mineral density osteoporosis [11].

History

The patient's history is a key component of establishing a correct diagnosis in the patient assessment for a suspected stress injury of bone (Table 10.3). It requires not only asking specific

Table 10.3 Clinical history pearls for evaluation of tibial stress fracture

• History of prior stress fracture
• Recent increase in training intensity, duration, or equipment
• Focal pain localization—with activity, including weight bearing, running, jumping
• Pain progression from after activity, to with activity, to activities of daily living, to rest
• Female athlete triad

questions pertinent to a chief complaint, but also *actively* listening and responding with successive adaptive questions related to the patient's responses. It is advantageous to obtain the history and examine the patient prior to viewing images.

The history of present illness can often all but definitively make a diagnosis prior to even examining the patient or reviewing images in assessment of the possible tibial stress fracture. Characterization of the principal symptoms attributable to the chief complaint should describe seven entities [12]: Location, quality, severity, timing (onset, duration, frequency), setting, exacerbating and relieving factors, and associated manifestations. Pertinent positive and negative findings from the past medical history, family history, social history, review of systems, and medication list are all also very relevant. Patient demographics and epidemiology must also not be overlooked, especially age and gender, as these factors do play a significant role in evaluating overuse injuries.

Pain Localization

Pain location is an important component in clinical diagnosis. In the diagnosis of tibial stress fracture, it is vital to localize exactly where the patient feels the most severe pain, either at rest or with activity. If necessary, it may be beneficial to have the patient run or jump for 5–10 min to reproduce the symptoms and better pinpoint the location of pain. The main additional differential diagnosis in the evaluation of shin pain in possible overuse injury is medial tibial stress syndrome, or "shin splints." In diaphyseal tibial stress fractures, the pain is frequently insidious in onset, over a prodromal 2- to 4-week course, and often coincides with a change in training volume and/or intensity. Initially, the pain is after activity, then progresses to during activity, especially weight-bearing during running or jumping on the affected leg, and may progress to activities of daily living and/or at rest. Anterior or anterolateral, tension-sided, tibial stress fractures often present in jumpers, ballet, or dancers at the central one-third of the diaphysis and the athlete typically points with one finger "this is where it hurts." Compression-sided fractures often present in runners at the posteromedial tibia (similar to medial tibial stress syndrome). However, these fractures may present proximally, distally, or in the mid-shaft. Patients may point to a focal area of pain or may more broadly or vaguely state that "it hurts around this area." Patients with medial tibial stress syndrome similarly complain of vague diffuse exertional pain along the posteromedial border of the tibia at the mid-distal portion of the shaft.

Patients with stress fractures in other locations of the tibia often complain of focal tenderness to touch and weight-bearing pain. Similar to diaphyseal fractures, these fractures may initially be only after activity, progress to pain with activity, and eventually be present with activities of daily living and/or rest. In medial malleolar stress fractures, patients will complain of weight-bearing pain at the medial malleolus, especially with running, and pain with ankle motion. In patients with proximal tibia stress fractures, activity-related weight-bearing pain is common at the location of the fracture.

Exacerbating and Relieving Factors

Circumstances that aggravate a painful sensation often clue the clinician into the diagnosis. Factors that relieve pain include rest, medications, and procedures. In patients with tibial stress fracture, exacerbating factors include running (e.g., compression side posteromedial tibia), jumping or dancing (e.g., tension-side anterior tibia due to force of posterior calf musculature), and weight bearing (e.g., ambulation). Relieving factors usually include rest, reduction in weight bearing and

loading of the leg, anti-inflammatory medications, and ice cryotherapy.

A helpful history pearl that may distinguish medial tibial stress syndrome and tibial stress fracture is the pain response to a training session. Patients with stress fractures tend to have worsening of pain in the location of the fracture with a single training session as the session progresses to the point that the athlete sometimes has to stop due to pain. Athletes with medial tibial stress syndrome, in the early stages, may actually have pain at the beginning of a training session that gradually subsides during that training session.

Other Findings

The clinician must be cognizant of other possible contributing coexistent pathology that may predispose the athlete to stress fracture. A thorough foot exam may reveal rigid pes cavus, subtalar pronation, tarsal coalition, muscle imbalance, weakness, or stiffness. Pes planus has also been shown to predispose to medial tibial stress syndrome [8]. However, other studies have shown, rather than pes planus, it is the ratio of subtalar everter to inverter muscles (in favor of eversion) that is predictive of increased risk of medial tibial stress syndrome [13]. Evaluation of leg-length is necessary as a significant leg-length discrepancy is associated with tibial stress fracture [14].

Physical Examination

The physical examination for the lower limb with an overuse injury should be comprehensive and systematic. This allows for consistency and reproducibility during examination of patients with not only tibial stress fractures (Table 10.4), but all potential causes. It should, just as questioning during a proper history, be adaptive as well. Physical assessment of any limb or joint requires visual inspection, palpation, motion, strength, and special (e.g., vibration tuning fork [15], tuning fork with stethoscope [16], and hop [17]) testing. Further, in order to understand if pathology is present in the involved tibia, the clinician must also thoroughly examine the contralateral tibia as well, with the knowledge that bilateral stress fractures may coexist. Extensions of the tibial stress fracture physical examination require evaluation from as far proximal as the lumbar spine and down the entire lower extremity as needed. This requires an assessment of coronal plane alignment, femoral version, tibial torsion, and pedal arch. Further, assessment of core strength, hip impingement, knee and ankle stability, and musculotendinous unit tightness (e.g., hamstring, hip adductors, iliotibial band, gastrocnemius-soleus-Achilles, plantar fascia) is warranted. Although the physical examination of the possible tibial stress fracture should focus on the presenting chief complaint, a comprehensive physical examination should also identify other abnormalities that may predispose the patient to other overuse injuries (i.e., injury prevention).

Table 10.4 Physical examination pearls in the evaluation of tibial stress fractures

• Focal point tenderness at the site of the fracture
• Edema, palpable periosteal thickening
• Positive single-leg “hop” test, although nonspecific
• Positive “tuning fork” test
• Absent compartment swelling, nerve symptoms
• Evaluate for possible contributing coexistent pathology (e.g., muscular tightness, contracture)
• Running gait observation and analysis

Tibia-Specific Physical Examination

The key physical examination finding to distinguish a tibial stress fracture from other causes of leg pain is primarily focal point tenderness at the location of the fracture, usually the anterior or medial tibia. As opposed to the latter, patients with medial tibial stress syndrome frequently have more diffuse, nonfocal tenderness along the posteromedial middle to distal one-third of the tibia and not the anterior tibia. If the patient reports that the pain only occurs after an activity, such as running, then the clinician should have the patient go outside of the clinic and run, or in the training room or physical therapy arena, run on a treadmill. The tenderness may be exacerbated

Fig. 10.1 (**a**) Posterior still-frame photograph from a treadmill running gait analysis of an elite-level distance runner demonstrating 7° of pelvic tilt. (**b**) Lateral still-frame photograph from the same runner demonstrating stride length with neutral foot-strike. (**c**) Still-frame photograph of gait analysis performed on an elite-level long jumper via aquatic treadmill as the athlete recovered from a tibial stress fracture

by a tuning fork test. The tuning fork test has a sensitivity, specificity, positive predictive value, and negative predictive value of 75 %, 67 %, 77 %, and 63 %, respectively. The single-leg hop test is also frequently utilized in the evaluation of all lower extremity stress fractures, not just the tibia [18]. However, this test is nonspecific, as it is also positive in up to 46 % of patients with medial tibial stress syndrome [19]. Edema and palpable periosteal thickening are also observed in patients with tibial stress fractures. In patients with medial tibial stress syndrome, edema is usually absent.

Inspection

Thorough inspection of the tibial stress fracture requires observation of the core and entire lower extremity. In addition to the patient himself/herself, shoe wear patterns should also be observed. One should also note prior surgical incisions and observe any deformity or asymmetry in alignment, swelling, calluses, and blisters. The clinician should observe for any swelling, edema, ecchymosis, or erythema. Gait evaluation by either observation in clinic or outside of clinic, on a treadmill or via digital video analysis gives a real-time evaluation of biomechanical factors that may predispose to stress injuries of the tibia (Fig. 10.1a–c).

Palpation

This is a key component in the tibial stress fracture evaluation. All osseous and soft tissue structures warrant palpation. In the leg, this includes, among others, the subcutaneous tibia, the knee joint, tibial tubercle, fibular head, medial

and lateral malleoli, the popliteal fossa, calf musculature and Achilles tendon, and plantar fascia. The patient with a tibial stress fracture may exhibit focal tenderness with percussion testing, a positive tuning fork and single-leg hop test, and edema. Patients with medial tibial stress syndrome usually exhibit more diffuse, nonfocal tenderness along the posteromedial mid-distal tibia without edema. Patients with medial malleolar stress fractures have tenderness over the medial malleolus and pain with forced passive ankle dorsiflexion and rotation.

Motion, Strength, and Special Testing

Assessment of motion and strength in the evaluation of tibial stress fractures mandates analysis of both limbs for comparison. Although different clinicians may have their own specific routine for assuring completeness and minimizing patient movement between sitting, standing, supine, and prone, it is important to ensure that a complete exam is performed and documented for every patient. It is also often helpful to examine the "normal" uninvolved limb before examination of the involved limb. It is important to assess for tightness or contracture in certain muscle groups especially the iliopsoas, iliotibial band, common adductors, hamstring complex, gastrocnemius-soleus-Achilles complex, and plantar fascia. The Thomas test may be utilized to assess for hip flexor tightness [20]. The Ober test may elicit iliotibial band tightness [21]. The Silfverskiöld test may be used to determine gastrocnemius tightness (improved ankle dorsiflexion while the knee is flexed) versus Achilles tightness (no difference in ankle dorsiflexion with knee flexion or extension) [22]. The clinician must also rule out chronic exertional compartment syndrome in patients with exertional leg pain and overuse injury. Progressive leg pain, swelling, clumsy foot, and numbness or tingling with activity warrants an evaluation for the latter. Pre- (>15 mmHg) and post- (one [>30 mmHg] and five [>20 mmHg] minutes) exercise compartment pressure measurements may yield this diagnosis. Nerve entrapment is infrequent in the athlete's lower extremity, but must be ruled out in patients with neurological symptoms such as numbness, tingling, "pins and needles," or burning pain. Possible affected nerves include the saphenous, common peroneal, deep peroneal, superficial peroneal, and tibial. Reproduction of the nerve symptoms with compression and a positive Tinel's sign are suggestive of nerve entrapment syndrome.

Imaging

Imaging for tibial stress fractures includes plain orthogonal radiographs, non-contrast magnetic resonance images in three planes (axial, sagittal, coronal), computed tomography, and technetium-99m-labeled methylene diphosphonate bone scan (Triple Phase Bone Scintigraphy). In the early course of a tibial stress fractures, plain X-rays are usually negative (10 % sensitivity) [2]. After 3 weeks, radiographs may illustrate direct or indirect signs of fracture (periosteal or cortical thickening or sclerosis, endosteal thickening or sclerosis, a discrete fracture, or callus [30–70 % sensitivity]) [23]. When the "dreaded black line" (Fig. 10.2) is present, the fracture takes on more characteristics of a nonunion and rarely responds to conservative treatment, often requiring intramedullary nail placement (Fig. 10.3) [24, 25]. On plain X-rays, medial malleolar stress fractures are often vertically oriented. Magnetic resonance imaging provides the best anatomic detail, with higher specificity than bone scan, and either equal or superior sensitivity versus bone scintigraphy [23, 26]. If the diagnosis is needed earlier than the appearance of plain radiographic findings, magnetic resonance imaging may demonstrate the presence of a stress fracture (Fig. 10.4). Sensitivity, specificity, accuracy, and positive and negative predictive values for magnetic resonance imaging in tibial stress fractures was 88 %, 100 %, 90 %, 100 %, and 62 %, respectively [26]. The latter for computed tomography scan was 42 %, 100 %, 52 %, 100 %, and 26 %, respectively [26]. The sensitivity of bone scan was 74 %. Although magnetic resonance imaging is advantageous based on its diagnostic performance and lack of ionizing radiation, it is the most expensive of the imaging modalities available to diagnose tibial stress fracture.

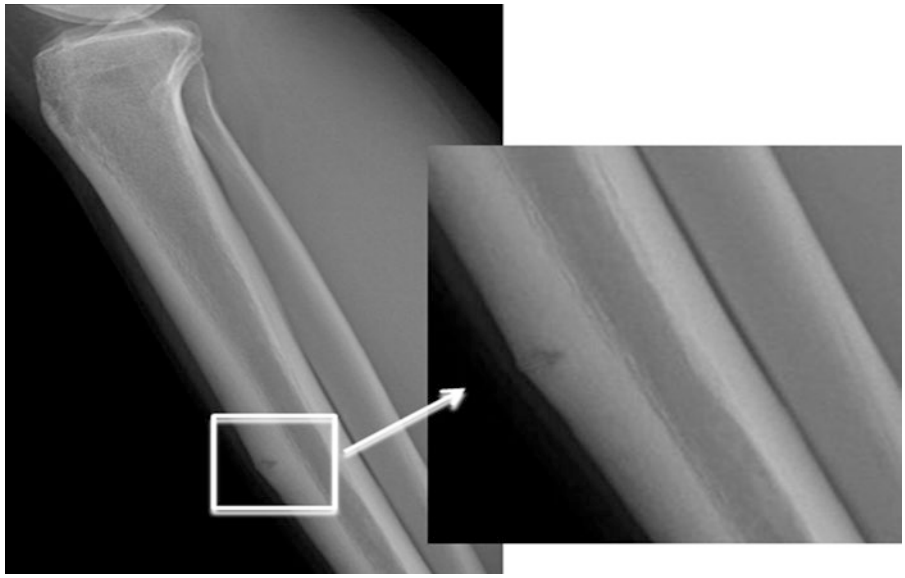

Fig. 10.2 Lateral radiograph of the tibia and fibula in a patient with an anterior tibial stress fracture, illustrating the "dreaded black line." The coned-in view zooms in on the fracture line and also illustrates anterior cortical thickening

Fig. 10.3 Lateral X-ray of the tibia and fibula following intramedullary nail placement in a patient with a "dreaded black line."

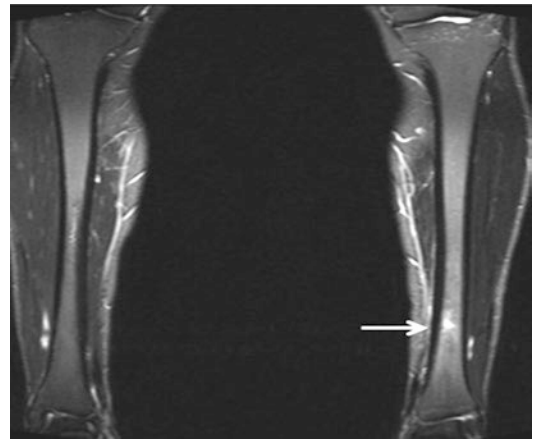

Fig. 10.4 Coronal plane T2-weighted bilateral leg magnetic resonance image of a patient with leg pain illustrating a tibial stress reaction (Kaeding–Miller Classification Grade II) in the middle—distal tibial diaphysis, without a discrete fracture line visible

Treatment

Initial management of tibial stress fractures should include a period of rest, activity modification, immobilization, and reduced weight bearing. For both low- and high-risk stress fractures, a metabolic bone disease evaluation should be performed and further laboratory work-up instituted based on risk factors including a history multiple stress fractures (Table 10.5). Metabolic deficiencies, such as calcium and/or vitamin D, may be easily identified and corrected, as long as the underlying cause is identified and addressed.

Table 10.5 Metabolic bone disease laboratory evaluation

• Comprehensive metabolic panel (especially calcium, phosphorus, magnesium)
• Albumin
• Alkaline phosphatase
• Vitamin-D
• Endocrine and sex hormones (e.g., thyroid, parathyroid, estrogen, progesterone, GnRH, FSH, LH)

Currently, athletes with calcium and vitamin D deficiencies should intake 1,000–1,500 mg and 1,000–3,000 International Units of calcium and vitamin D daily, respectively. Although bisphosphonates are successful pharmacologic agents in the treatment of osteoporosis and osteopenia, their use in patients with tibial stress fractures is not currently recommended [27]. Despite the mechanism of action of bisphosphonate medications (inhibition of osteoclast-mediated bone resorption seen in early stress fracture), their clinical use has no high-level evidence in either the prevention or treatment of tibial stress fractures [28]. Although parathyroid hormone derivatives have been shown to improve stress fracture repair in animal models, their clinical use has no high-level evidence in either the prevention or treatment of tibial stress fractures [29]. Low-intensity pulsed ultrasound may be beneficial in the treatment of tibial stress fractures, with possible reduction in time to osseous union [30]. The mechanism of action of ultrasound is via a cascade of effects with initial integrin activation, leading to upregulation of COX-2 (cyclooxygenase), VEGF (vascular endothelial growth factor), and BMP-2, 4, 6, and 7 (bone morphogenic protein) [31–33]. This may translate to increased extracellular matrix formation in soft callus, increased enchondral ossification, osteoblast differentiation, and mineralization in hard callus, and remodeling of mineralized callus [32, 34–36]. Despite the latter basic science evidence, clinical evidence for treatment of tibial stress fractures is currently limited and should be utilized on a case-by-case basis [37]. In the setting of delayed union, nonunion, or stress fractures of the tibia, low-intensity pulsed ultrasound bone stimulators are indicated. Thus, in patients with or without delayed union of a tibial stress fracture, bone stimulation is indicated. Pneumatic leg braces have demonstrated efficacy in rehabilitation of tibial stress fractures, with faster healing and return to sport than control [38–40]. However, other studies have failed to show any difference between pneumatic braces and controls [17]. There is limited evidence illustrating that extracorporeal shock-wave therapy has benefit in the treatment of recalcitrant tibial stress fractures [41].

Low-risk, posteromedial tibia stress fractures are initially managed non-operatively. Complete cessation of sports, running, jumping, and any significant weight-bearing load is instituted until the patient is asymptomatic with walking. If this is unsuccessful after approximately 3–4 weeks, then a trial of complete non-weight bearing and immobilization may be used prior to return to activities. High-risk, anterior tibial cortex stress fractures are at significantly greater risk of nonunion, delayed union, and fracture completion and displacement than low-risk posteromedial tibia stress fractures [42, 43]. Thus, they are more frequently treated surgically with reamed intramedullary nailing [25]. An alternative to intramedullary nailing is open reduction and internal fixation with anterior tension-band plating with or without bone grafting [44]. In the in-season athlete, a difficult management situation exists. The length of non-operative treatment, up to 8–12 months, of anterior tibial stress fractures is intolerable for most competitive athletes. However, a surgical procedure is not without significant risk in this patient population, especially anterior knee pain after intramedullary nailing [45]. Medial malleolar stress fractures are prone to nonunion due to the high shear forces at the fracture site [46]. Thus, in patients with a discrete fracture line (≥Kaeding–Miller Grade III) or nonunion, surgical treatment is often recommended with two 4.0 mm partially threaded cancellous screws or a low-profile antiglide plate and screw construct.

Critical Points

- The anatomy and biomechanics of the tibia play a critical role in the development and management of tibial stress fractures.
- The differential diagnosis of patients with overuse injuries of the leg includes stress fracture,

medial tibial stress syndrome, exertional compartment syndrome, popliteal artery entrapment syndrome, and multiple nerve entrapment syndromes, among others.

- Risk factors for tibial stress fracture include prior stress fracture, recent increases in training intensity and/or duration, improper training technique or equipment, and the female athlete triad, among others.
- The history and physical examination of a patient with a tibial stress fracture generally indicates focal point tenderness at the site of the fracture, unremitting pain with weight-bearing activity, and pain with single-leg hop and tuning fork testing.
- Magnetic resonance imaging is the imaging modality with the highest diagnostic performance.
- Non-operative treatment is generally successful in low-risk posteromedial tibial stress fractures.
- Surgical treatment, including reamed intramedullary nailing and anterior tension-band plating, is a successful treatment for chronic anterior mid-diaphyseal tibial fractures that have failed nonsurgical treatment with high union rates, low complication rates, and early return to sport.

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K.E.V.: None.

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Stress Fractures of the Fifth Metatarsal

11

Amanda Weller, Christopher D. Harner, and MaCalus V. Hogan

Introduction

Fifth metatarsal fractures are common midfoot injuries among the athletic population including elite-level athletes. Fractures in this location first gained recognition after being described by Sir Robert Jones in 1902 [1]. At the time, his description was based on a case series including his own fracture and other fifth metatarsal fractures in variable locations. Since then, the orthopaedic community has continued to pay consistent attention to these fractures, developing more detailed descriptions of the various types of fifth metatarsal fractures. Perhaps more importantly, the various treatment options and outcomes associated with each type of fifth metatarsal fracture have been studied extensively. Although fractures of the fifth metatarsal can be acute and result from direct trauma, the proximal fifth metatarsal has also been identified as a location at which stress fractures commonly occur.

Stress fractures of the fifth metatarsal usually occur in competitive athletes who participate in running or jumping sports [2]. Stress fractures in this location are considered high risk and carry a tendency toward delayed union, nonunion, and refracture after incomplete healing [3–6]. DeLee et al. [7] have previously described the criteria for diagnosing a proximal fifth metatarsal fracture, which includes a history of prodromal symptoms over the lateral aspect of the foot, radiographic evidence of a stress reaction in the bone, and no history of treatment for a previous acute fracture of the fifth metatarsal. Lee et al. [8] further specified radiographic findings in detail: intramedullary sclerosis, periosteal reaction, and cortical hypertrophy. Much effort has been made to appropriately identify and treat these injuries as well as potentially prevent them from occurring. These investigations are understandable considering that a delay in diagnosis or inadequate treatment of a fifth metatarsal stress fracture can result in extended loss of playing time and possibly the end of an elite athlete's career [5].

Anatomy

The anatomy of the fifth metatarsal has been extensively studied and described. The proximal fifth metatarsal has traditionally been divided

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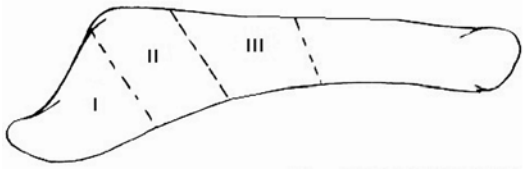

Fig. 11.1 An illustration of the three different zones of the proximal fifth metatarsal [2]. Reprinted with permission from Dameron TB Jr. Fractures of the proximal fifth metatarsal: Selecting the best treatment option. J Am Acad Orthop Surg 1995; 3(2)

into three zones (Fig. 11.1). Zone 1 is the most proximal and includes the cancellous tuberosity. Zone 2 is just distal to the tuberosity and extends to the level of the articulation of the fourth and fifth metatarsal. There are secure ligamentous attachments both plantarly and dorsally at this level. Zone 3 begins distal to the ligamentous attachments of zone 2. It extends distally into the tubular portion of the metatarsal diaphysis approximately 1.5 cm, and this zone is a common location for stress fracture. The peroneus tertius tendon inserts onto the dorsal surface of the metatarsal diaphysis distal to the tuberosity. The lateral band of the plantar aponeurosis inserts on the plantar aspect of the metatarsal styloid (Fig. 11.2).

Smith et al. [9] have described the blood supply to the fifth metatarsal. It arises from three sources: the nutrient artery, metaphyseal perforators, and periosteal arteries. The nutrient artery enters medially into the middle third of the bone and then divides into a distal and proximal branch (Fig. 11.3). The proximal branch is shorter than the distal branch and does not reach to the most proximal portion of the metatarsal. An abundance of small metaphyseal vessels feed the metatarsal at each end of the bone.

Pathophysiology

A stress fracture is a result of an accumulation of microdamage from repetitive loading that results in fatigue failure of the bone. The fifth metatarsal is considered a high-risk site stress fracture due to the stresses that it experiences combined with its discontinuous blood supply [8, 10–12].

In regard to blood supply, the area of the fifth metatarsal just distal to the metaphyseal–diaphyseal junction is considered a watershed area. At this location, the metaphyseal perforators and nutrient artery meet but are unable to provide diffuse equal blood flow to the entire area. The bone proximal to this is supplied well by the perforators, and the bone distal to this is supplied well by the nutrient artery. This poor blood supply leads to an impaired reparative biologic response to stresses at the junction [5].

Not only does this area possess a poor blood supply, but it also experiences a high level of stress, especially in athletes. Stress on the fifth metatarsal occurs when the heel is off the ground and body weight is primarily carried through the lateral column. This causes adduction of the fifth metatarsal, which is resisted proximally by ligamentous attachments, effectively resulting in a repetitive varus stress at the metaphyseal–diaphyseal junction [1, 13]. As a consequence, stress typically starts laterally and progresses medially. This results in the worrisome plantar lateral gap due to the tensile forces in this region accompanied by compression at the dorsomedial cortex [8, 14]. Additionally, the metatarsal head is more mobile than the proximal base, which has strong ligamentous and capsular attachments, causing a fulcrum effect to occur at the watershed metaphyseal–diaphyseal junction [15]. Biomechanical studies by Arangio et al. [16] have demonstrated that the principal stress across the bone peaks when the load is directed 30–60° off the horizontal plane. The location of this maximum stress was found to be concentrated in the proximal diaphysis of the bone in the region 3.38–4.05 cm distal to the tuberosity (Fig. 11.4). Further studies in athletes have determined that the greatest pressure differential between the base of the fifth metatarsal and the head occurs during the acceleration phase of running [17]. Based on this study, some prevention programs emphasize longer recovery time between accelerations to prevent build up of stresses in this high-risk location.

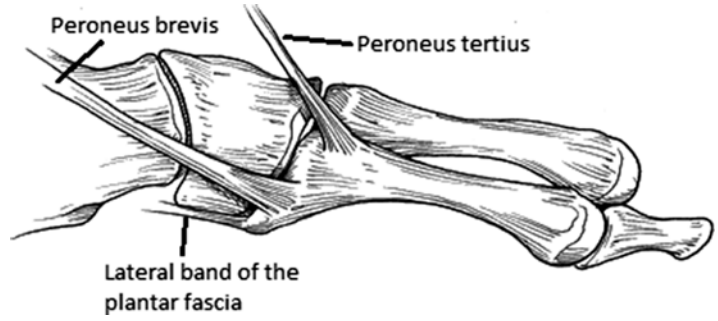

Fig. 11.2 The tendinous and fascial attachments at the proximal fifth metatarsal make this portion of the metatarsal more rigid than the distal portion [25]. Reprinted from Lawrence ST, Botte MJ. Jones' fractures and related fractures of the proximal fifth metatarsal. Foot Ankle. 1993;14: 358–365

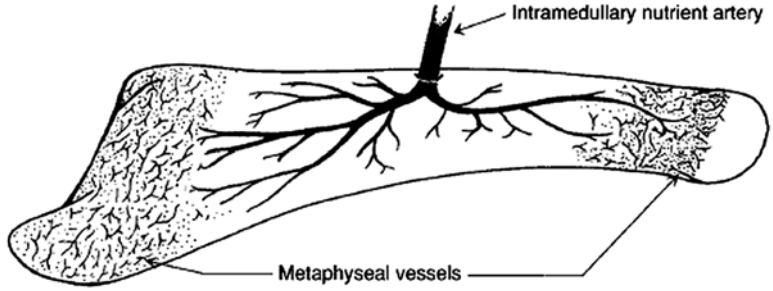

Fig. 11.3 Diagram illustrating the main blood supply to the fifth metatarsal. The watershed area lies at the transition between the proximal branch of the intramedullary nutrient artery and the proximal metaphyseal vessels [2]. Reprinted with permission from Dameron TB Jr. Fractures of the proximal fifth metatarsal: Selecting the best treatment option. J Am Acad Orthop Surg 1995; 3(2)

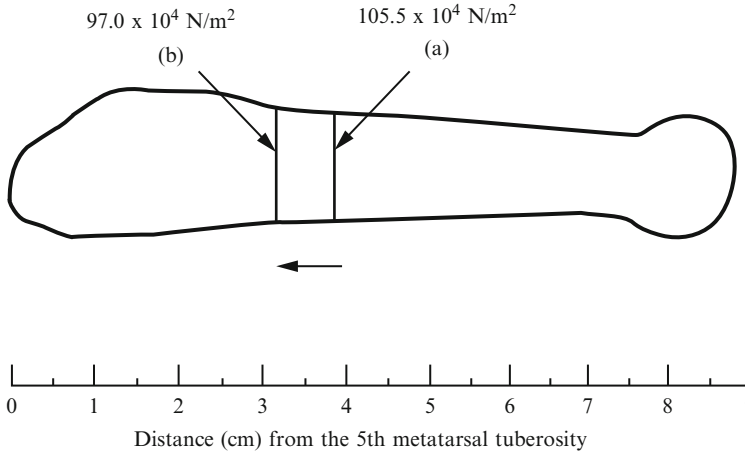

Fig. 11.4 This diagram delineates the location in the proximal fifth metatarsal that withstands the maximum principal stress under a 1 N vertical (**a**) and horizontal (**b**) load [16]. Both occur in the area of the metaphyseal–diaphyseal junction. Reprinted from Arangio GA, Xiao D, Salathe EP. Biomechanical study of stress in the fifth metatarsal. Clin Biomech. 1997;12(3):160–164

Classification

A classification system originally described by Torg [4] is commonly used for proximal fifth metatarsal fractures (Fig. 11.5). Torg type I indicates an acute fracture occurring with a chronic process. Radiographs will typically show prior periosteal reaction, a plantar-based fracture lucency, and no medullary sclerosis. Torg type II fractures occur in conjunction with medullary sclerosis and narrowing and are associated with delayed union. Radiographs of a Torg type III fracture reveal obliteration of the medullary canal, and this type frequently represents a nonunion.

A second classification system was more recently described by Lee et al. [8, 14]. This system is based on the chronicity of the fracture as well as the plantar gap of the fracture, which is measured from the standard oblique radiographic view (Fig. 11.6). Type A1 represents an acute, complete fracture of the proximal fifth metatarsal at the metaphyseal–diaphyseal junction. Type A2 represents an acute on chronic complete fracture at this location. A type B1 fracture is an incomplete fracture with a plantar gap measuring less than 1 mm. Finally, a type B2 fracture is an incomplete fracture with a plantar gap measuring 1 mm or greater.

Diagnosis

Identifying a fifth metatarsal stress fracture begins with a thorough history and physical exam. This should include a review of systems

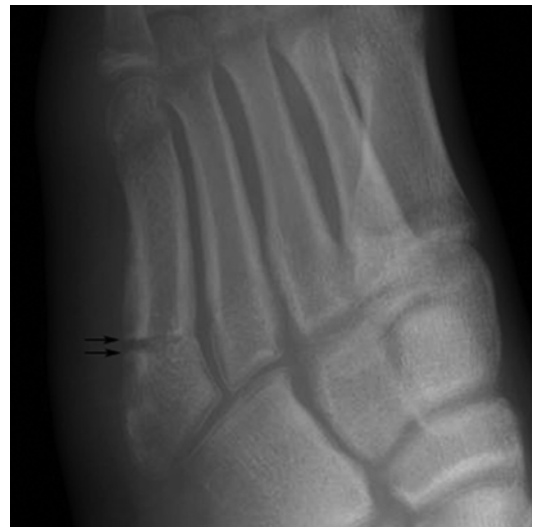

Fig. 11.6 An example of an oblique radiograph of the foot displaying a plantar gap in a fifth metatarsal stress fracture [14]. Reprinted from Lee KT, Young UP, Young KW, Kim JS, Kim JB. The plantar gap: another prognostic factor for fifth metatarsal stress fracture. Am J Sports Med. 2011 Oct;39(10):2206–2211

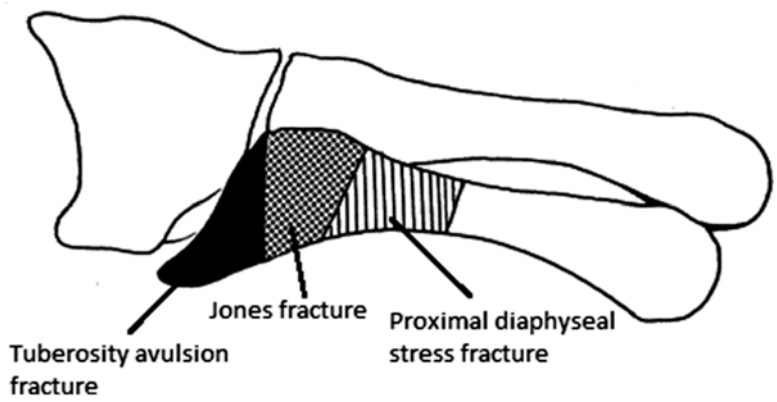

Fig. 11.5 An illustration of the different types of fractures occurring at different areas of the proximal fifth metatarsal [25]. Reprinted from Lawrence ST, Botte MJ. Jones' fractures and related fractures of the proximal fifth metatarsal. Foot Ankle. 1993;14:358–365

including any hormonal or nutritional irregularities, past medical history, and current medications. Additionally, the clinician should initiate a detailed assessment of training techniques. An injured athlete may present with an acute fracture or an insidious onset of pain with tenderness over the proximal fifth metatarsal. Swelling may be present and inversion of the foot typically exacerbates pain symptoms. An athlete presenting with an acute onset of symptoms may or may not have experienced prodromal symptoms leading up to this acute event [5]. The history and physical exam should focus on identifying both extrinsic and intrinsic risk factors for fifth metatarsal stress fracture [11]. Extrinsic factors include an overly intense training regimen or incorrect footwear and other equipment. Intrinsic factors include a high longitudinal arch, leg-length discrepancy, and excessive forefoot varus.

Imaging

Radiographs

Imaging studies should include an AP, lateral, and oblique radiograph of the affected foot with weightbearing films included if the patient can tolerate. Although early radiographs may be normal, most will be positive for a metatarsal stress fracture at the time of presentation [11]. In the setting of normal radiographs with persistent symptoms, follow-up films will often show callus or a lucent line indicating a stress process. Depending on the clinical scenario, radiographs may show an acute fracture with evidence of a chronic stress reaction at the metaphyseal–diaphyseal junction.

CT Scan

Computed tomography (CT) scan may be necessary for diagnosis or preoperative planning. In the setting of negative radiographs, a fracture may be seen with the use of CT scan. Chronicity may be difficult to determine from plain radiographs; in these cases, CT scan is useful to identify and quantify sclerosis and intramedullary canal obliteration. This can be helpful for preoperative planning because in fractures with canal obliteration, open reduction with bone grafting may be necessary.

MRI and Bone Scintigraphy

Magnetic resonance imaging (MRI) and Technetium-99m-labeled diphosphonate bone scan are most helpful in the case of a compelling clinical scenario with negative radiographs. They are both highly sensitive in diagnosing a stress fracture or stress reaction [10]. An MRI can reveal endosteal marrow edema and periosteal edema of the proximal fifth metatarsal indicating a stress reaction or impending fracture. It can also show a nondisplaced fracture line. Similarly, a bone scan will show increased uptake in the region of stress reaction which will be present prior to complete fracture. Both of these imaging techniques can make an early diagnosis of a stress process in the fifth metatarsal [2, 5, 10].

Treatment

Fifth metatarsal stress fractures have both nonoperative and operative treatment options. Deciding the best option requires careful consideration of the type of fracture as well as the activity level of the patient.

Nonoperative

There has been some success with nonoperative treatment, but the indications are relatively few. Dameron et al. [2] describe successful treatment of these fractures with activity modification and a metatarsal brace if the diagnosis is made early. Torg type I fractures have been successfully treated with non-weightbearing and cast immobilization for 6–8 weeks as long as intramedullary

sclerosis is not present [4, 5, 10, 18]. Clapper et al. [19] described a high nonunion rate and prolonged time to complete union when treating proximal fifth metatarsal fractures in 8 weeks of cast immobilization followed by transition to walking boot, but noted 100 % union rate with operative fixation and faster healing time. Chuckpaiwong et al. [20] recommend non-weightbearing cast immobilization for 4–6 weeks followed by a functional splint for another 4–8 weeks to achieve union in a Torg type 1 fracture.

Other nonoperative types of treatment for fifth metatarsal stress fractures have been explored. Research has shown that there may be a place for extracorporeal shock-wave therapy (ECSWT) in treating these fractures in athletes. The exact mechanism of action is unknown, but animal models have suggested that the high-energy acoustic waves induce neovascularization and therefore stimulate healing. Thevendran et al. [15] reported several studies exploring positive results after ECSWT, but they include different types and locations of stress fractures and small subject numbers. Consequently, strong evidence to support the routine use of this modality is lacking [10].

Electromagnetic bone stimulation has also been used in treating proximal fifth metatarsal fractures and has shown some promising results in the setting of stress fractures, delayed union, and nonunion; however, these are largely level IV studies [15]. This modality may be useful as an adjunct to surgery but high-level evidence is still lacking [15].

Operative

There has been agreement that operative fixation for fifth metatarsal stress fractures is an effective option and in many circumstances is the best choice for the patient given this high-risk anatomic location [4, 10, 11, 18, 20]. Due to the high incidence of delayed union or nonunion, aggressive management has been recommended especially in elite athletes, those with persistent pain, and those patients who develop a pseudoarthrosis [10]. Additionally, imaging that demonstrates even partial obliteration of the medullary canal by sclerosis, especially in a high-level athlete, should be treated operatively to avoid delayed healing [4, 10, 18, 20]. This includes both Torg type II and type III fractures.

The most common operative fixation includes an intramedullary screw with possible bone grafting [4, 7, 10, 18, 20, 21]. Additionally, a tension band technique with bone grafting has been shown to be successful [8, 14]. Porter et al. [21] reported 100 % clinical healing and nearly 100 % radiologic healing when using a 4.5-mm stainless steel cannulated screw in Torg type II and III fractures. The size of the screw diameter can be increased to fill larger intramedullary canals. In fractures with a large plantar lateral gap (greater than 1 mm) and sclerosis about the fracture, autogenous bone graft is recommended [8, 14, 15]. Despite overall reliable outcomes reported with use of a cannulated screw, many surgeons today utilize intramedullary fixation with a noncannulated screw due to the superior fatigue strength of a solid screw versus a cannulated screw [22, 23].

Internal fixation of a proximal fifth metatarsal stress fracture is generally a safe and fast procedure, but some complications have been reported following the operation. Some patients require screw removal after fracture healing due to persistent discomfort and prominence over the screw head [7, 18]. Some patients also report shoe-wear irritation at the lateral foot surgical site [20]. Screw fracture and malpositioning of intramedullary screws have also been reported [24].

Postoperative management includes early range of motion, restricted weight bearing, and return to sports only after clinical and radiologic healing has been achieved. Thevendran et al. [15] outlined a postoperative recovery course consisting of gradually increasing to weightbearing as tolerated in a walking boot over the first 6 weeks after surgery. Radiographs are taken at 6 weeks to ensure healing and the boot can then be removed and activity increased as symptoms allow. Expected return to sport is estimated to take approximately 10–12 weeks. Given the risk of refracture, computed tomography scan can be used to verify complete healing. Nonsteroidal anti-inflammatories are generally avoided during

the postoperative period until healing has been confirmed. Some advocate for metatarsal bracing during return to competitive athletics [2, 21].

Clinical Cases

Case 1

The patient is a 22-year-old female collegiate basketball player with left lateral foot pain after a recent game when another player fell on her left foot. She reported antecedent pain in this area of her foot prior to the acute injury. She has continued to play despite the pain. The patient had no pertinent medical problems and was a nonsmoker.

Radiographs are shown in Fig. 11.7a, b and reveal an incomplete fracture through the lateral cortex of the proximal fifth metatarsal at the metaphyseal–diaphyseal junction most consistent with a stress fracture. A CT scan obtained of the foot also revealed evidence of partial healing of the fracture as well as some sclerotic bone consistent with a stress fracture attempting to heal.

The fracture was identified during the basketball season and a discussion was conducted with the patient regarding immediate surgical treatment versus surgical treatment at the end of the season. The patient elected to continue to play through the season using a lateral column support in her shoe to help offload the lateral column as well as padding under her fifth metatarsal. She was able to complete the season with minimal pain while playing and underwent intramedullary screw fixation with autogenous bone graft for treatment to prepare her for a professional career after graduation. Postoperative radiographs at 10 days after surgery are shown in Fig. 11.7c.

Case 2

The patient is a 21-year-old male collegiate cross-country runner in his senior year who presented for evaluation after onset of pain in the left foot while running a race 10 days prior to his clinic visit. He continued to bear weight on the foot after the injury, but he was placed into a walking boot 2 days after the injury by his athletic trainer due to persistent symptoms and has been non-weightbearing while in the boot. He reported pain over the proximal fifth metatarsal and he recalls having some swelling over this area initially, which has now resolved. He did not recall having pain in the foot prior to this event.

Radiographs of the left foot are shown in Fig. 11.8. There is a fracture demonstrated to pass through zone II of the proximal fifth metatarsal with acute on chronic features. Notably there is mild sclerosis present within the medullary canal at the level of the fracture.

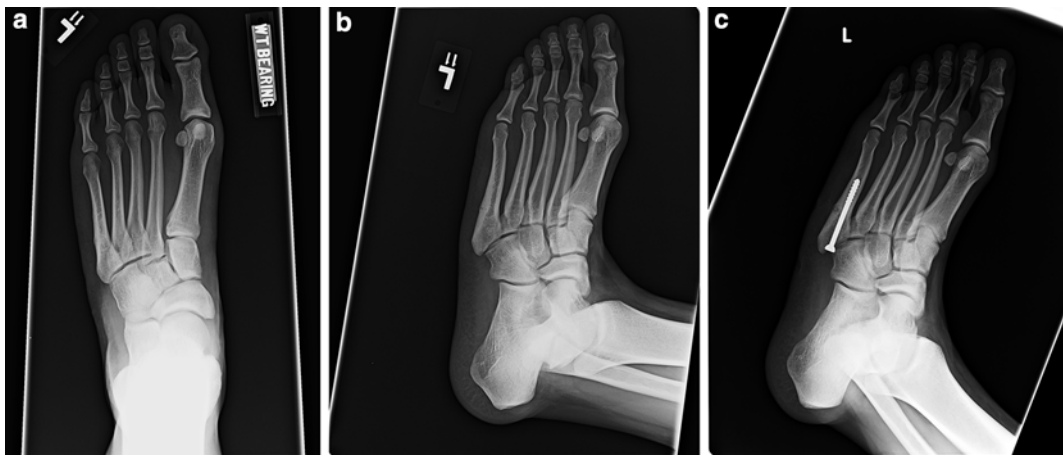

Fig. 11.7 AP (**a**) and oblique (**b**) radiographs of the left foot demonstrating a stress fracture of the proximal fifth metatarsal. Oblique radiograph of the left foot after intramedullary screw fixation (**c**)

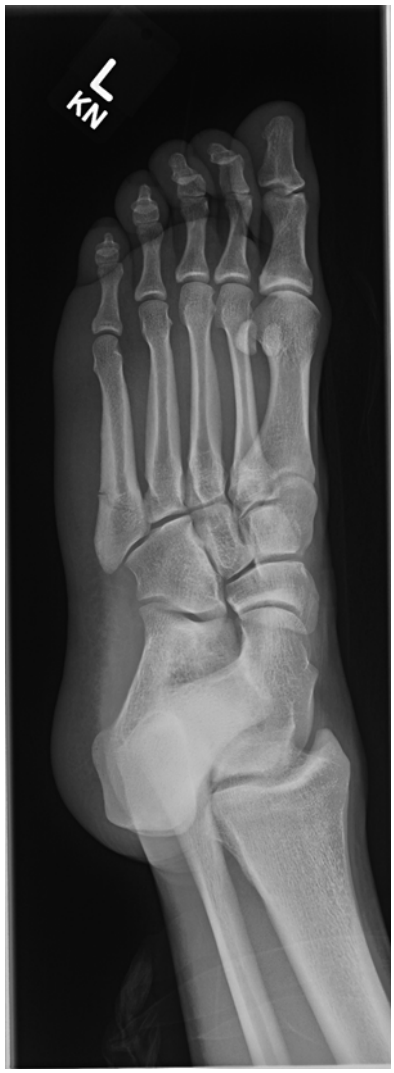

Fig. 11.8 Oblique radiograph of the left foot showing an acute-on-chronic proximal fifth metatarsal fracture

Due to his status as a high-level athlete and his injury pattern most closely representing a Torg type II fracture, operative intervention was indicated. The athlete underwent fixation with a 4.5-mm solid stainless steel screw with iliac crest autograft and bone marrow aspirate concentrate placed at the fracture site. Radiographs from just over 5 weeks after surgery are shown in Fig. 11.9. Postoperatively the patient remained in splint/cast immobilization for the first 4 weeks while the incisions healed. He was kept non-weightbearing

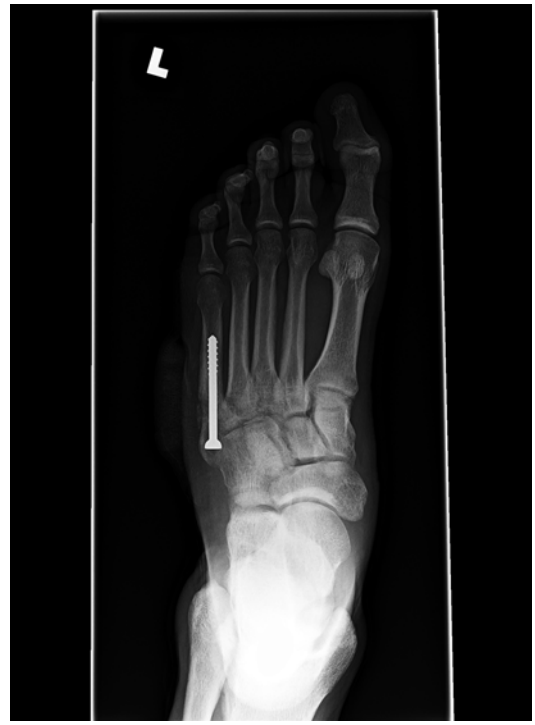

Fig. 11.9 AP radiograph of the left foot 4 weeks after intramedullary screw fixation and bone grafting for a proximal fifth metatarsal stress fracture

for 6 weeks until radiographic healing was evident and pain had completely resolved. He returned to competition at 3 months post-surgery. No refracture occurred.

Case 3

The patient is a 22-year-old collegiate-level football player who complained of lateral sided pain in his right foot for 6 months. He was able to play through the recent fall football season as a defensive back with the use of insoles, which initially relieved some of the pain. The pain returned, and he was having difficulty completing practices due to the pain. He had no past medical history and is a nonsmoker. On physical examination, tenderness to palpation was present over the base of the fifth metatarsal. Otherwise, he was nontender throughout the remainder of the forefoot and midfoot.

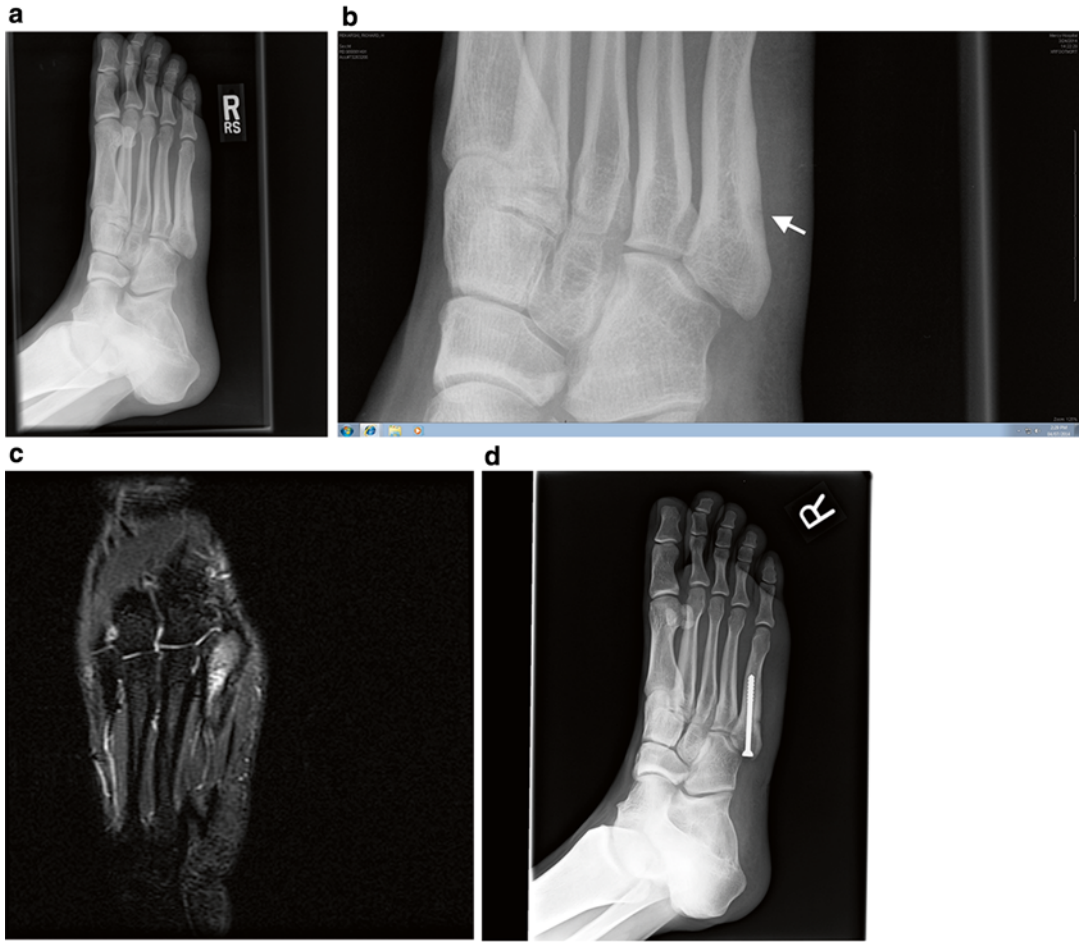

Fig. 11.10 Oblique radiograph of the right foot (**a**). Magnified oblique radiograph of the right foot showing subtle lucency in the lateral cortex of the proximal fifth metatarsal (**b**). Representative axial STIR MRI image of proximal fifth metatarsal stress reaction (**c**). Oblique radiograph on postoperative day 10 after intramedullary screw fixation (**d**)

Three views of the right foot were obtained including an oblique film shown in Fig. 11.10a. The radiologist reviewing the radiographs initially documented normal findings with no evidence of fracture. However, given the patient's history, on closer inspection there did appear to be slight lucency in the lateral cortex at the metaphyseal–diaphyseal junction as seen in Fig. 11.10b. Based on this finding and the clinical history, MRI of the right foot was obtained which clearly showed bony edema throughout the proximal fifth metatarsal and a subtle cortical defect consistent with a stress reaction and pending fracture at this location (Fig. 11.10c).

A discussion was had with the patient and his family regarding the stress reaction in the fifth metatarsal and concern for progression to a complete fracture. The patient desired to play football in the fall for his senior year. Given the duration of his pain and the MRI findings, the patient elected to move forward with intramedullary screw fixation using a 5.5 mm solid stainless steel screw with iliac crest bone autograft and bone marrow aspirate concentrate placed at the fracture site. Radiographs from 10 days after surgery are shown in Fig. 11.10d.

Outcomes

Despite advances in treatment techniques since the first descriptions of fifth metatarsal stress fractures, these injuries remain difficult to treat and the risk for reinjury even after proper treatment is not trivial. This is likely due to a combination of anatomical and biomechanical factors. A significant rate of refracture has been observed with nonoperative treatment of these fractures [7]; however, refracture rates after operative intervention and presumed healing have been reported to be up to 10.7 % [3]. In 2011, Lee et al. [14] determined that patients with a wider plantar gap preoperatively on radiographs have worse outcomes and require longer time to union with a higher nonunion rate and refracture rate than those patients with a small preoperative plantar gap. In 2013, Lee et al. [3] went on to observe that the development of refracture after surgery is associated with a higher body mass index. This group also found that patients whom had radiologic parameters on oblique radiographs associated with protrusion of the fifth metatarsal head also had higher refracture rates than those with normal parameters. Consequently, a longer period of protection before initiating rehabilitation and weightbearing is recommended for these patients.

Return to Sports Decision-Making

The timing of return to play for recreational or elite athletes is determined using clinical and radiographic factors. Porter et al. [21] described a return to play algorithm for athletes who underwent screw fixation for proximal fifth metatarsal fractures that allowed the athlete to wean out of a walking boot at 3 weeks postoperatively if pain free with ambulation and biking. The athlete could then begin stair-stepper exercises with use of a semirigid orthosis for protection. Running is allowed at 4–5 weeks postoperatively if pain free on the stair-stepper. Once an athlete was pain free while running for 30 min 3–4 days per week then he/she could begin a functional progression program and ultimately return to sport once the program could be completed without pain or apprehension and radiographs showed progression to healing. Kaeding et al. [5] agree that athletes must have a normal clinical exam and painless functional activity prior to being cleared for return to full play participation and typically occurs 6–8 weeks postoperatively, which is consistent with other reported timelines [10, 21].

Summary

Despite over a century having passed since Sir Robert Jones first described them, fifth metatarsal stress fractures remain a difficult injury to treat. The elite athlete that sustains this type of injury should be treated operatively with careful postoperative rehabilitation protocols and guided return to play criteria to avoid extended loss of playing time. A careful review of intrinsic and extrinsic risk factors for fifth metatarsal stress fractures must be conducted to avoid reinjury and possible career ending disability. Despite the difficulty of this injury, early diagnosis and appropriate treatment can produce acceptable outcomes for the elite or recreational athlete.

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Stress Fractures of the Ankle

12

Ljiljana Bogunovic, Jonathon D. Backus, and Rick W. Wright

Introduction

Stress fractures of the ankle are rare injuries, but possess the potential for prolonged disability and significant loss of time from sport if not appropriately diagnosed and treated. Similar to other stress fractures of the lower extremity, those about the ankle result from repetitive microtrauma that exceeds the capacity of bone remodeling. These injuries occur almost exclusively in highly active athletic or military populations [1]. Sports with significant running and jumping such as basketball, long distance running, and track and field/cross country are the most frequent causative activities [1–4]. Both nonoperative and operative treatment options exist, but the ideal method is dependent on the location of the fracture, the chronicity of the injury, and the demands of the athlete. Low-risk fractures involving the lateral malleolus can most often be successfully managed with a period of rest and/or activity modification, while fractures involving the medial malleolus have more limited healing potential, an increased risk for displacement and may require operative intervention [4–11].

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Stress Fractures of the Lateral Malleolus

Incidence, Pathophysiology, and Risk Factors

Approximately 6–11 % of lower extremity stress fractures involve the fibula [3, 12, 13]. The exact mechanism of lateral malleolar stress fractures is not well understood, as the fibula only experiences 2.3–10.4 % of weight-bearing load depending on ankle alignment and foot position [14]. Burrows described two different types of fractures occurring in the distal fibula: A proximal area of the distal fibula 5–6 cm from the tip of the lateral malleolus that occurred in young active males and a distal area of the distal fibula 3–4 cm from the tip of the lateral malleolus that occurred in middle-aged females [4]. The proximal stress fracture variant, termed "Runner's Fracture," is the more common of the two and is believed to be caused by repetitive eccentric contractions of the plantar and long toe flexors in combination with axial loading [4, 6, 13, 15, 16]. Devas and Sweetnam performed ankle radiographs on patients at rest and then with forceful plantarflexion while the leg was held in a wooden frame to control for rotation [6]. They found that the fibula approximated more closely with the tibia during forceful

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plantarflexion. From this experiment, they postulated that this approximation also occurs during the toe strike phase of running and may concentrate stress in the fibula at an area 5–6 cm from the tip of the lateral malleolus [6]. Other risk factors include malalignment of the foot and ankle. Excessive pronation of the foot with hindfoot valgus has been associated with an increased incidence of fibular stress fracture [12]. Valgus of the ankle joint is also believed to put the lateral malleolus at risk [15].

Presentation

Patients with stress fractures of the distal fibula often report the insidious onset of stiffness or pain in the lateral ankle [4, 6]. These symptoms worsen with activity and generally improve with rest. The course is typically progressive in nature if the offending sport or activity is continued. Patients will often have no pain with walking, but running or stair climbing may reproduce symptoms [6]. As with many overuse injuries, a detailed account of the patient's training regimen should be elicited. A sudden increase in mileage, recent addition of high-impact plyometric activity, change in the training surface, and change in footwear are all risk factors for stress fracture development [3, 6, 17].

Physical Examination

Care must be taken to distinguish a lateral malleolar stress fracture from lateral ankle impingement, lateral ankle ligament tears, syndesmotic injury, or peroneal tendon pathology. Physical examination reveals point tenderness 4–7 cm proximal to the tip of the lateral malleolus directly over the bone. Ankle range of motion is typically normal and no effusion is present [4]. Localized soft tissue swelling, however, may be appreciated early with this injury. In more chronic conditions, the swelling becomes firm and callus may be palpable at the site of the fracture [12]. As with all examinations of the foot and ankle, a thorough examination of standing alignment and gait must be performed. Comparison to the contralateral side will also aid in diagnosing lateral ankle pathology. Bilateral distal fibular stress fractures have been reported in the literature; however, this is very rarely encountered [4, 18].

Imaging

Standard AP, lateral and mortise radiographs should be obtained in all patients with persistent ankle pain. Some have also suggested additional oblique views in stress fracture diagnosis [19]. Radiographs taken with the ankle positioned in 45° of internal or 45° of external rotation can be helpful in detecting subtle injuries to the lateral malleolus and medial malleolus, respectively [20]. Initial radiographs of fibular stress fractures are often negative and the sensitivity of this imaging may be as low as 15 % [12, 21]. Typically, 4–6 weeks are required before osseous changes become evident on plain radiographs (Fig. 12.1a, b) [6]. Early signs may include a periosteal reaction adjacent to the lateral cortex and new bone formation 4–7 cm proximal to the distal lateral malleolus tip [6, 13, 15]. With time, callus formation or a frank fracture line may become visible [6]. These fracture lines are typically oblique, running from distal lateral to proximal medial; however, in cases of an incomplete fracture, the medial cortex is typically spared [6, 13].

In the absence of radiographic findings, advanced imaging with bone scan or MRI is frequently utilized to confirm the diagnosis. MRI has become the advanced imaging modality of choice given improved specificity and ability for concurrent evaluation of the surrounding soft tissues. Periosteal edema, identified on T2 sequence, is the earliest abnormality evident on MRI. With progression of injury, abnormal signal can be detected in the bone marrow on both T1 and T2 sequences, and ultimately progresses to involve the cortical bone (see Fig. 12.1b) [13]. In comparison to patients presenting with normal radiographs, those with evidence of stress fracture on plain films have higher grades of injury as determined with subsequent MRI [13].

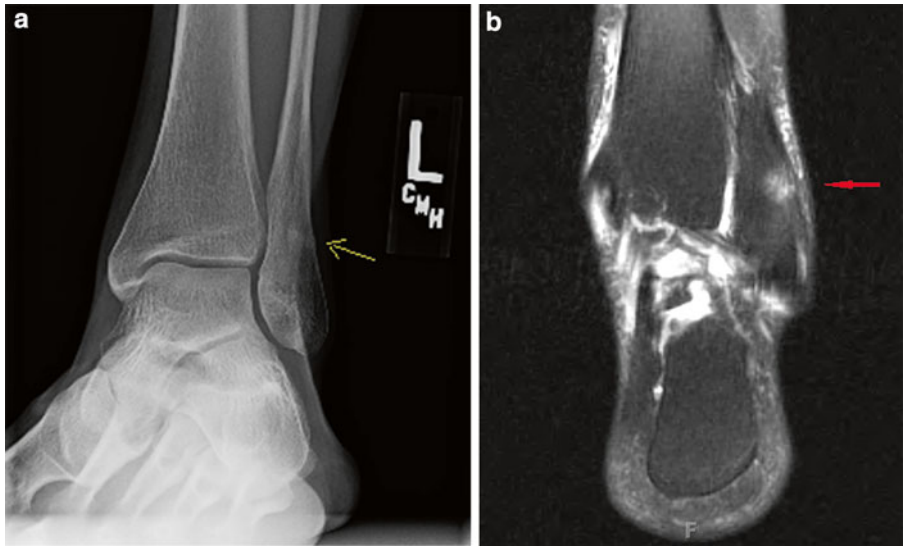

Fig. 12.1 (**a**) Mortise ankle radiograph in a 35-year-old female recreational runner with 3 months of left ankle pain. The patient was diagnosed with a lateral malleolar stress fracture. Notice a cortical reaction is visible approximately 4 cm proximal to the tip of the lateral malleolus (denoted by *arrow*). (**b**) T2-weighted MRI of the same patient. A periosteal reaction in addition to bone marrow edema can be appreciated in the lateral malleolus (denoted by *arrow*)

Treatment

Nonoperative Treatment

No consensus exists in the literature for nonoperative treatment of fibular stress fractures, but the mainstay of treatment includes rest and activity modification. Early diagnosis is essential in speeding recovery and return to sport. Some patients may benefit from a short period of immobilization with a walking cast or boot to minimize pain and stress at the fracture site; however, the use of an ankle brace, taping, or elastic bandage is preferred over prolonged immobilization [4, 15] (Fig. 12.2) Custom orthotics to correct flexible pes planovalgus or other malalignments may also be beneficial. Full weight bearing is allowed, provided that it does not cause pain, but all high-impact running and jumping activities should be discontinued. During this time, the patient is encouraged to maintain conditioning with low impact activities such as the stationary bike, elliptical trainer, swimming, and pool running. Treatment also incorporates exercises focusing on ankle range of motion, strengthening, and proprioception.

Fig. 12.2 Example of a walking boot that can be utilized in the nonoperative treatment of stress fractures of the ankle

Several authors anecdotally suggest that impact activities should be reintroduced gradually when there is no longer tenderness to palpation at the stress fracture site and when compression of the

fibula to the tibia does not elicit pain [6, 15]. Most athletes can be expected to return to full activity between 6 and 8 weeks [4, 6, 15].

Operative Treatment

The indications for operative treatment of lateral malleolar stress fractures are limited; however, cases of operatively managed fibular stress fractures do exist in the literature. Kottmeier et al. reported a case of a 19-year-old football player with a non-healing lateral malleolar stress fracture. The patient's history was notable for recurrent ankle sprains and progressive ossification of the syndesmosis. Resection of the synostosis led to subsequent healing of the stress fracture and return to full activity [22]. Another case study by Guille et al. reported on a patient with an external rotation malunion from a lateral malleolar stress fracture that ultimately required a nonunion take down with bone grafting and open reduction and internal fixation [23].

Stress Fractures of the Medial Malleolus

Incidence, Pathophysiology, and Risk Factors

The tibia is the most common site of stress fracture [1, 12, 24]. While 20–60 % of all stress fractures involve the tibial shaft, the incidence of medial malleolar stress fracture is far lower, estimated between 0.6 and 4.1 % [1, 7, 12, 24]. Unlike the low-risk stress fractures of the lateral malleolus, stress fractures of the medial malleolus have limited healing ability, a propensity for displacement, and the potential for prolonged disability. The development of these fractures appears to be highly dependent on repetitive high-impact activity [8, 9, 24–26]. Multiple series report fracture occurrence exclusively in high-level athletes participating in activities requiring significant running and jumping, such as basketball, football, and running [10, 24]. Shelbourne

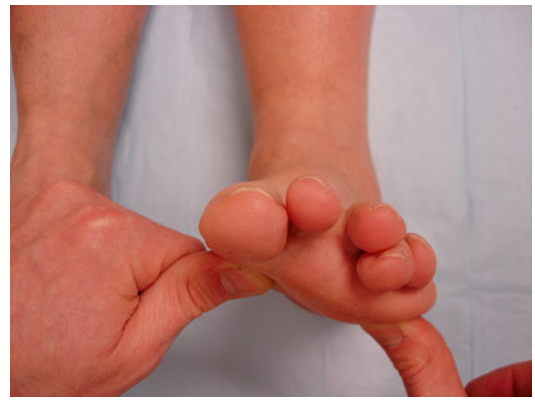

Fig. 12.3 An example of forefoot varus, a risk factor for stress fracture of the lateral malleolus. Image reprinted with permission of Jeffrey Johnson, M.D.

described a cascade of events leading to injury. During the heel strike phase of running, the ankle dorisflexes as the forefoot pronates. With forefoot pronation, the navicular assumes an abducted position relative to the talar head and an internal rotation force is transmitted across the talus. Talar forces are subsequently transferred to the medial malleolus [10]. Repeated high force contact between the talus and medial plafond leads to microtrauma and ultimately stress fracture. Intrinsic factors believed to increase this contact between the talus and medial malleolus include tibia varum and forefoot varus [3, 11, 27] (Fig. 12.3). The presence of an anteromedial talar osteophyte has also been suggested to exacerbate tibiotalar impingement and contribute to the development of fracture [28]. While the majority of these fractures occur in young and middle-aged adults, medial malleolar stress fractures have also been reported in skeletally immature athletes [8, 10, 25, 29].

Presentation

The diagnosis of a medial malleolus stress fracture can be challenging. As such, it is not uncommon for the diagnosis to be delayed weeks to months following the onset of symptoms [9]. Patients often present with vague, ill-defined

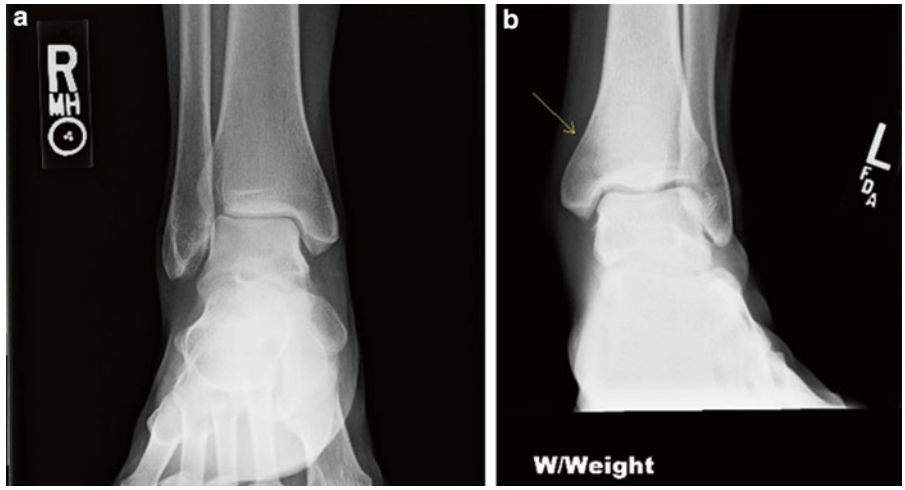

Fig. 12.4 (**a**) AP ankle radiograph of a 47-year-old active female with 2 weeks of medial ankle pain. These radiographs are normal but the patient went on to be diagnosed with a medial malleolar stress fracture. (**b**) AP radiograph of a 32-year-old male professional athlete with a clearly visible stress fracture line in the medial malleolus

medial ankle pain in the absence of trauma [8, 10]. As with the fibula, the pain is insidious in nature. It presents initially with activity and then may progress to pain at rest. Some patients may report a rapid exacerbation in pain after a period of mild symptoms. This finding should alert the physician to possible acute displacement of an underlying stress fracture [27]. As mentioned previously, nearly all patients with this injury will report a history of high-impact sports such as endurance running or basketball. They may also report a rapid increase or change in their sporting activities.

Physical Examination

Care must be taken to distinguish this condition from posterior tibial tendonitis and deltoid ligament injury. Physical examination findings include tenderness directly over the medial malleolus at the junction with the tibial plafond [8, 10]. Localized swelling, erythema, and warmth can be present at the site of fracture. Given the intra-articular nature of the fracture, an effusion of the ankle joint may be present and limit ankle range of motion [7, 10]. Running and/or jumping activity will often reproduce the patient's pain [10]. As mentioned previously, a thorough examination of standing alignment and gait must be performed and compared to the contralateral side.

Imaging

The high proportion of cancellous bone in the medial malleolus makes detection of a stress fracture difficult, even after the early phase of healing (4–6 weeks) when most other stress fractures become visible on plain radiographs (Fig. 12.4a). Early osseous changes, when present, include blurring of the trabecular margins and subtle sclerosis from peritrabecular callus formation. Over time a sclerotic line may be apparent within the cancellous bone. The characteristic medial malleolar stress fracture line appears on the posteromedial, concave side of the tibia and extends in a vertical or oblique direction from the junction of the medial malleolus and the tibial plafond into the tibial metaphysis (see Fig. 12.4b) [8, 10, 25]. Small lytic lesions surrounding the fracture line have also been described [25].

In some cases, changes on plain radiographs may never become visible [9]. Advanced imaging is recommended in all patients with negative radiographs, but with clinical signs and symptoms

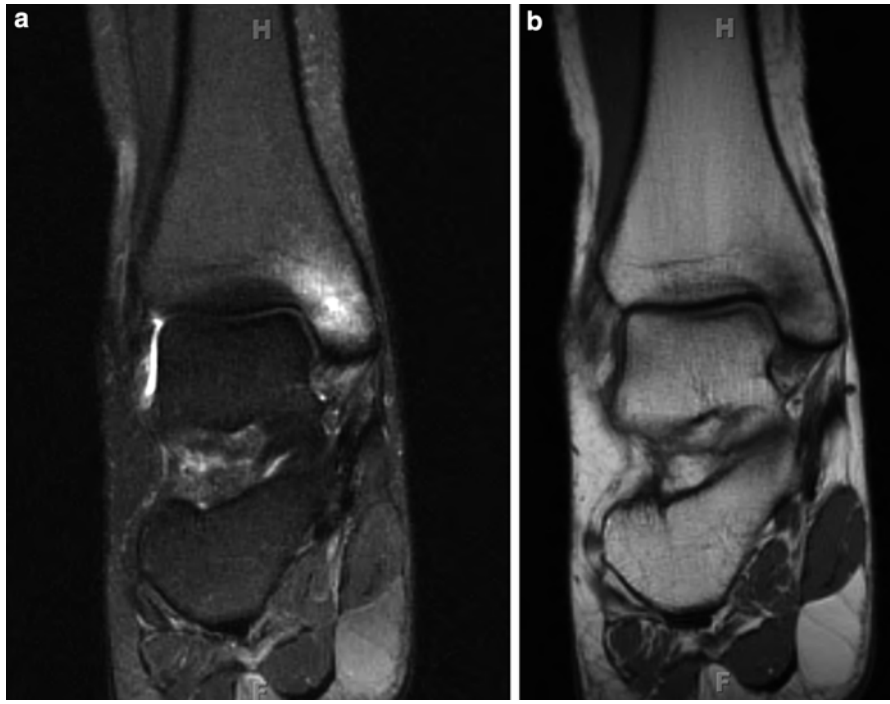

Fig. 12.5 (**a**) T2-weighted MRI of the patient introduced in Fig. 12.4a. Notice the significant bone edema noted in the medial malleolus. (**b**) T1-weighted image in the same patient. This sequence reveals a linear low-signal area that extends to the medial cortex consistent with the diagnosis of medial malleolar stress fracture

concerning for a stress fracture a high index of suspicion should be employed [9]. As with the fibula, MRI is preferred for the early detection of medial malleolar stress fractures [9]. The earliest osseous changes can be depicted on fat suppressed T2-weighted images or STIR sequences where soft tissue and bone edema result in high signal changes within the cancellous bone (Fig. 12.5a). As the injury and associated edema progress, abnormalities become visible on T1-weighted images as a linear area of low signal that runs perpendicular to the trabeculae and ultimately extends into the cortex (see Fig. 12.5b) [19]. Recently, ultrasound has also been suggested as a possible imaging modality for the diagnosis of medial malleolar stress fractures [21]. In a small series of patients, periosteal thickening, cortical irregularities, edema and pain with transducer compression were appreciated in nearly all patients. Furthermore, another recent study proved that ultrasound was 83 % sensitive and 76 % specific for metatarsal stress fractures when compared with MRI [30]. This low-cost modality could become more popular and beneficial in diagnosing malleolar stress fracture in the future; however, as with all ultrasounds imaging, the results are highly dependent on the skill of the ultrasound technician performing the exam. Bone scan may also be used in diagnosing medial malleolar stress fractures, but this imaging technique has been largely supplanted by MRI.

Treatment

Nonoperative

The treatment of medial malleolar stress fractures is controversial [1, 10, 11, 27]. While some authors recommend conservative treatment for all nondisplaced fractures, others recommend internal fixation through either an open or percutaneous approach if evidence of fracture is visible on plain radiographs [8, 10, 11]. Conservative treatment generally entails a period of immobilization in a cast, boot, or ankle stirrup air cast [10, 11, 27].

Careful follow-up with repeat imaging is required to monitor for displacement and delayed healing. Similar to conservative management of lateral malleolar stress fractures, patients are encouraged to maintain conditioning with low or non-impact activities and continue range of motion, strengthening, and proprioceptive therapy. Custom orthoses may also be beneficial if hindfoot and forefoot alignment corrections are warranted.

Several studies have reported successful conservative treatment of medial malleolar stress fractures in athletes. Orava et al. reported good outcomes and full return to sports in 5/8 patients. These fractures were diagnosed early (within 2–3 weeks of the onset of symptoms), were visible on plain radiographs, and were nondisplaced. Patients were allowed to weight bear as tolerated but were instructed to refrain from all running and jumping activities. Patients who attempted return to running and/or jumping after 2–3 months developed reoccurrence of pain and were treated with an additional period of activity limitation. The average time to return to sport was 4 months. Six months were required before the three top-level athletes were able to return to full activity [8]. Shelbourne et al. reported on the conservative treatment of three patients with stress fractures of the medial malleolus. In his series, patients had negative radiographs but had clinical signs and bone scan findings consistent with stress fracture. These patients were treated with a period of immobilization in an air cast brace followed by a gradual return to activity. All three returned to full athletic activity between 6 and 8 weeks [10]. In summary, nonoperative treatment of medial malleolar stress fractures can result in successful healing, but patients, especially those with evidence of fracture on plain film, must be willing to commit to a prolonged period of rest, activity modification, and potential immobilization.

Operative

The operative treatment of medial malleolar stress fractures is dependent on several factors, including the characteristics of the fracture, the duration of symptoms, and the physical demands of the patient. Most authors agree that operative fixation is required for all displaced fractures of the medial malleolus; however, the ideal treatment of nondisplaced fractures is more controversial. Shelbourne et al. recommended operative fixation of all nondisplaced fractures that are visible on plain radiographs. Those authors argued that operative treatment provides a more predictable rate of healing. Stress fractures of the medial malleolus have been reported to result in a delayed union or nonunion rate approaching 10 % [16]. Lempainen et al. suggested an increased risk of displacement in fractures with evidence of a "cortical crack" on MRI, and they recommended operative fixation in these patients even without positive findings on plain radiographs [9]. A delay in diagnosis and prolonged duration of symptoms (greater than 2–3 weeks) were also considered a poor prognostic indicator for nonoperative treatment in this study. In five patients who presented 6 weeks to 4 months after the onset of symptoms, 3–4 months of conservative treatment failed to improve their symptoms. All patients subsequently required operative treatment with screw fixation and eventually returned to pre-injury level of activity [9].

Many authors recommend operative fixation in all "in season" or high-level athletes with evidence of a stress fractures of the medial malleolus [8–10, 28]. Proponents of operative treatment argue a more rapid recovery and eventual return to activity [9, 26, 29, 31]. Operative treatment allows for faster mobilization and rehabilitation with return to full sports participation at approximately 6–8 weeks. Operative treatment also provides a more predictable outcome in highly active individuals. Complications of conservative treatment of athletes with medial malleolar stress fractures have been described in the literature. Reider reported a case of nonunion in a 21-year-old football player [32]. Shabat et al. described a 15-year-old gymnast who developed recurrence of pain and radiographic progression of her stress fracture 2 months following a course of nonoperative treatment [29]. Both athletes lost significant time from sport and ultimately required open reduction and internal fixation to achieve healing and return to play.

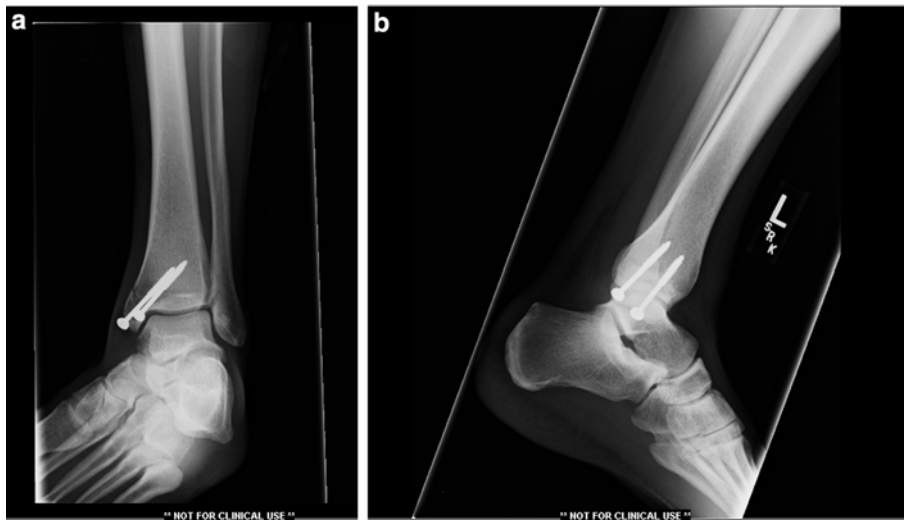

Fig. 12.6 (**a**) Mortise view and (**b**) lateral view of an ankle following fixation of a medial malleolar stress fracture with two partially threaded screws

No standardized technique has been established for operative fixation of malleolar stress fractures. Fixation can be obtained with both cannulated and solid partially or fully threaded screws either through direct visualization or percutaneously (Fig. 12.6). Many surgeons prefer to use screw fixation while visualizing the fracture directly through a curvilinear incision over the medial malleolus. The fracture site is compressed using one to two partially or fully threaded cancellous screws, directed under fluoroscopic guidance [8, 10, 27, 28]. Jowett et al. reported concomitant ankle arthroscopy and debridement of anterior impingement spur at the time of fracture fixation [28]. For patients with a delayed union following a trial of nonoperative management, Orava et al. described oblique drilling of the medial malleolus with a 2.2-mm drill bit [8]. Postoperatively, most patients are treated with a brief period (2 weeks) of partial weight bearing to allow for healing of the soft tissues. Range of motion exercises are encouraged immediately and immobilization is infrequently utilized. Running is reintroduced at 4–6 weeks post-surgery, and most patients are released to full activity between 6 and 8 weeks [8, 10]. No studies report the need for bone graft at the time of fixation and all report successful fracture union without mention of complications [3, 9, 10, 28, 32].

Summary

Stress fractures of the ankle are rare injuries, but should be considered in highly active patients with persistent pain about the lateral or medial malleoli. Delays in diagnosis are common and can result in prolonged disability and loss of time from sport. The treatment of these injuries is dependent on the location of the fracture and the demands of the patient. Nearly all stress fractures of the lateral malleolus and the majority of stress fractures of the medial malleolus can be successfully managed with rest and activity modification. Internal fixation is recommended for selected stress fractures of the medial malleolus. Operative indications include evidence of fracture on plain radiograph, presence of cortical disruption on MRI, and delayed diagnosis with prolonged duration of symptoms. Additionally, stress fractures occurring in the "in season" or high-level athlete who demands a fast and predictable return to sport is an indication for surgical stabilization. In assessing outcomes of ankle stress fractures, one must consider that the available literature is based on small cohorts and case series of patients, with no comparative studies available for review.

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Stress Fractures of the Calcaneus, Sesamoids, and Metatarsals

13

James D. Heckman, Marcus P. Coe, and Jason T. Laurita

Calcaneus

Stress fractures of the calcaneus were first described commonly among military recruits in the 1940s [1] and 1950s [2]. Hullinger [1] in 1944 reported the first large series of 53 calcaneal stress fractures in army recruits as the result of the "…repeated and rhythmic occurrence of minimal or subthreshold traumata…" with jumping being an important contributing factor. In 1959 Leabhart [2] reported on a very similar series of 134 marine recruits seen over an 18-month period of time. Ninety-eight of these recruits had bilateral calcaneal stress fractures, and he concluded that recruits who were older and less physically fit prior to enlistment were more likely to sustain a calcaneal stress fracture during basic training. Similarly, Greaney et al. [3] found a lower rate of stress fractures, including calcaneal fractures, among marine recruits who had a prior history of long distance running than among nonrunners. It is from these and other similar reports from the military experience [4–6] that the clinical course and treatment of these fractures have been defined.

In three large and more recent series of stress fractures in athletes [7], college athletes [8], or track and field collegiate athletes [9], among a total of 169 stress injuries to bone, only one calcaneal stress fracture was reported. Thus, in comparison to the reports in military recruits (where the proportion of calcaneal stress fractures ranged as high as 21 % of all bony stress injuries [3]), these fractures are relatively rare in athletes. When they do occur in athletes, distance runners are often affected [10, 11]. Among a group of nine runners using minimalist running shoes [12] one developed a calcaneal stress fracture which the authors attributed to the use of poor running technique; with minimalist running, heel strike is avoided and typically the impact stress is applied to the forefoot, which theoretically decreases the impact on the calcaneus.

Signs and Symptoms

As with stress fractures elsewhere in the lower extremity, calcaneal fracture symptoms usually develop 1–3 weeks after the onset of a new athletic

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activity [2], after the alteration of intensity, duration, or frequency of a training program or a change in footwear or training surface [13]. Typically, the runner will complain of plantar or diffuse heel pain that is aggravated by weight bearing and relieved by rest. Physical examination will show swelling and tenderness of the heel with pain elicited by medial–lateral compression of the calcaneus between the examiner's palms.

Imaging

While stress reactions and stress fractures have been described in all parts of the calcaneus [1, 6], Darby [5] has been credited for first describing the characteristic location and pattern of calcaneal stress fractures on plain radiographs. While initial radiographs may be normal in appearance, after 2 or more weeks of persistent symptoms a linear sclerotic line develops in the posterior body between the posterior articular facet and the tuberosity, perpendicular to the long axis of the calcaneus (Fig. 13.1) [14]. As these fractures are believed to result from repetitive compression, and not from tension or torsion on the calcaneus [15], little or no periosteal new bone formation will be seen. Both technetium (Tc) 99 bone scanning and magnetic resonance imaging (MRI) are more sensitive than plain radiography and can detect a calcaneal stress injury much earlier in its clinical course. Currently, MRI is the imaging method of choice when a calcaneal stress injury is suspected but not seen on plain radiographs and there is some uncertainty about the diagnosis clinically [6]. T1-weighted sequences (Fig. 13.2) and edema-specific sequences such as T2 fat-suppressed or short T1 inversion recovery (STIR) images (Fig. 13.3) have been recommended by Dodson et al. [14] as the best magnetic resonance images, as both display characteristic findings of stress injury to bone. T1 images will depict a dark fracture line traversing the posterior aspect of the body of the calcaneus, and the edema-specific images will demonstrate bright white high signal intensity marrow edema. Some caution must be used when interpreting MRI findings. While these imaging methods will detect most, if not all stress fractures, they are so sensitive that they may detect marrow edema in runners when no stress injury exists. Lazzarini et al. [16] found edema in the bones of the feet in 16 of 20 cross-country runners on STIR images. They also found edema in at least 2 of 12 nonrunner radiology residents who served as controls. Thus, the diagnosis of a stress injury of the calcaneus must be based on a correlation of clinical findings with the results of this very sensitive imaging technique.

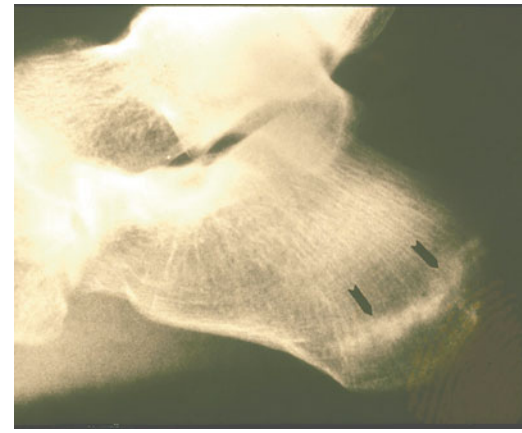

Fig. 13.1 Lateral radiograph of a classic stress fracture in the posterior body of the calcaneus approximately 3 weeks after the onset of symptoms in a long distance runner

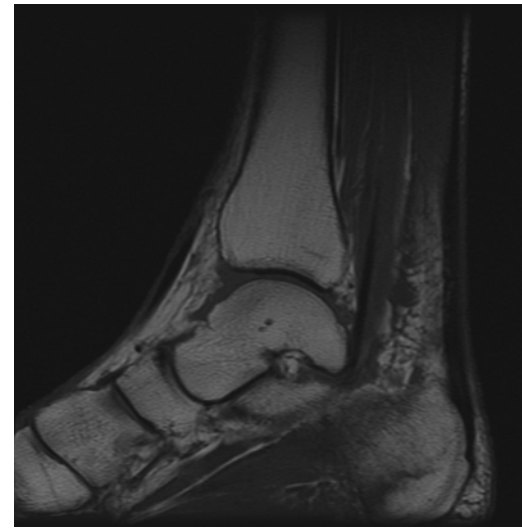

Fig. 13.2 T1-weighted MRI scan demonstrating a stress fracture of the calcaneus, as indicated by the dark, jagged line running perpendicular to the axis of the posterior tuberosity

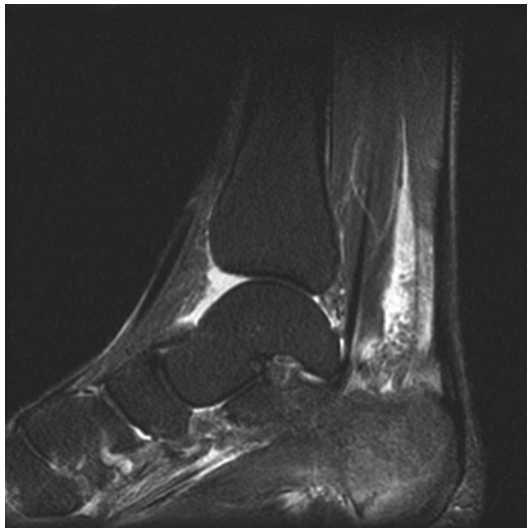

Fig. 13.3 Short tau inversion recovery (STIR) MRI image of the calcaneal stress fracture in the same patient as Fig. 13.2. Note the surrounding edema in the posterior tuberosity

Treatment

There is a strong consensus for nonoperative treatment of calcaneal stress fractures, and indeed the only report in the literature of surgical repair is that of a 67-year-old woman who sustained displaced fractures of both calcaneal tuberosities during a 3-week hiking trek [17]. Both fracture fragments were displaced substantially superiorly, compromising Achilles tendon function. The fragments were reduced and fixed with cancellous screws. In the several large series of calcaneal stress fractures reported from the military [1–3, 5, 6], and for the few calcaneal stress fractures reported in athletes [8, 11, 12], nonoperative management invariably has led to full resolution of symptoms and a return to the previous level of activity. Nonoperative treatment consists of 2–6 weeks of restricted weight-bearing activity, until the pain with weight-bearing resolves, followed by a gradual resumption of vigorous activities such as running and jumping. There are no reports on the need for cast immobilization during the acutely painful period, and long-term shoe-wear modification to treat recurrent or residual symptoms does not seem to be necessary.

The experience with military recruits has demonstrated that premature return to vigorous activity, prior to full resolution of weight-bearing symptoms, can lead to a recurrence of symptoms [2]. To avoid a recurrence of symptoms in athletes, Haverstock [13] recommended a two-phase plan to restore function. Phase I includes rest with non-weight bearing and activity restriction until the patient is pain free with weight bearing. This phase may take as long as 8 weeks. Then, in Phase II, activities are gradually reintroduced over a 6- to 12-week period. During this time, if pain returns activity should be decreased.

With the exception of the one case report noted above [17], no displacement of a stress fracture of the body of the calcaneus has been reported, and healing consistently occurs in this cancellous bone. To our knowledge, no long-term sequelae have been reported with this fracture.

Special Circumstances

Two cases of stress fracture of the anterior process of the calcaneus in association with a fibrous calcaneonavicular coalition have been reported in athletes [18, 19], and there is one report in the literature of a stress fracture of the body of the calcaneus in association with a talocalcaneal coalition [20]. In all three cases, the authors postulated that there was increased stress on the calcaneus due to restriction of rear foot motion caused by the coalition. Two of these fractures were treated nonoperatively with full resolution of symptoms. One of the anterior process fractures [19] was treated surgically with excision of the calcaneonavicular coalition and compression screw fixation of the stress fracture. This professional rugby player had returned to full activity without pain within 6 months.

A stress fracture of the anterior process of the calcaneus without a calcaneonavicular coalition was reported by Taketomi et al. [21] in a 14-year-old female basketball player. Because of persistent pain at 6 months after the onset of symptoms, the ununited fracture was treated surgically with multiple drilling across the fracture site with a 1.5-mm Kirschner wire. Following 4

weeks of postoperative non-weight bearing cast immobilization, the patient gradually resumed activity. The fracture was healed radiographically at 6 months, and she resumed playing basketball without pain.

In 2004, Ogden et al. [22] reviewed a group of 14 skeletally immature athletes with persistent heel pain for at least 9 months. All had the clinical diagnosis of Sever's disease, or calcaneal apophysitis. An MRI scan was obtained, and, in each case, it demonstrated edema and hemorrhage in the metaphysis of the calcaneus, consistent with the diagnosis of stress fracture. All 14 patients were treated with a cast (3) or a removable ankle-foot orthosis (11) and non-weight bearing for 3–4 weeks. In all cases the symptoms subsided following immobilization, and everyone was able to gradually resume sports participation without pain. The authors concluded that Sever's disease is a "… chronic (repetitive) injury to the actively remodeling trabecular metaphyseal bone that results in a variably sized stress injury…" and that it is not an apophysitis. This observation has not been confirmed by others.

In summary, stress fractures of the calcaneus result from impact loading and occur occasionally in runners. The typical fracture traverses the posterior body of the calcaneus and many can be diagnosed clinically. Following 2–3 weeks of symptoms, most can be confirmed radiographically as a condensation of new bone. In the absence of plain radiographic findings, magnetic resonance imaging can be used to identify the fracture line and associated marrow edema; however, caution must be used to not "over read" these MRI findings in athletes. Virtually all of these fractures heal completely with rest and avoidance of impact loading, allowing the athlete to resume his or her sport at previous levels of performance.

Sesamoids

Stress fractures of the sesamoids occur rarely in athletes, and the literature contains only a few sparse retrospective reports of small case series, usually focused on a common treatment scheme. As a result, there is little good evidence upon which to base treatment decisions.

The rarity of these fractures has been documented in three reports on stress injuries in athletes [7], college athletes [8], and collegiate track and field athletes [9]. A total of 169 stress injuries to bone were confirmed by scintigraphy [7] or MRI [8, 9] in these three studies; 87 occurred in the foot, but only three involved a sesamoid bone, two medial, and one lateral.

The medial sesamoid, which is larger and longer, lies within the substance of the tendon of the medial head of the flexor hallucis brevis, and the smaller and rounder lateral sesamoid lies within the substance of the tendon of the lateral head of the flexor hallucis brevis [23]. Portions of the abductor hallucis (medially) and the adductor hallucis (laterally) insert onto the sesamoids which are held together securely by the intersesamoid ligament [24]. Dietzen [25] pointed out that the sesamoids have no appreciable periosteum, as their plantar surfaces are covered by the plantar aponeurosis and dorsally they are covered by articular cartilage where they articulate with the metatarsal head.

Both sesamoids arise from multiple ossification centers between 7 and 10 years of age [24], and, not infrequently, the ossification centers fail to fuse together resulting in partition (bipartite or multipartite), particularly of the medial sesamoid [26]. While the reported rates of partition range widely from 0.1 to 33.5 %, the most frequently reported rates are 10 % for the medial and 1 % for the lateral sesamoid [24, 26]. Partition occurs with equal frequency in males and females, and, when present, it is bilaterally symmetrical in about 25 % of cases [27]. Partition of a sesamoid can be misinterpreted as a fracture on plain radiographs, and one study in military recruits has shown that mild to moderately increased scintigraphic activity over a bipartite sesamoid does not necessarily reflect clinically significant pathology [28]. These issues can cloud the assessment of the athlete with pain on the plantar aspect of the first metatarsal head.

Clinical Presentation

An athlete with a stress fracture of either sesamoid will develop pain that is usually well localized to the plantar aspect of the foot under the first metatarsophalangeal joint. The pain is usually insidious in onset, aggravated by athletic activity, and relieved by rest. These fractures are thought to result from repetitive forced dorsiflexion and seem to occur more frequently in running and jumping sports in which the great toe is loaded in dorsiflexion [27, 29]. In one series of 10 athletes [30], 7 were noted to have a plantarflexed first ray. Both sexes seem to be equally vulnerable, with certain sports showing a predilection (football in males [30] and gymnastics and dancing in females [30, 31]). In coed sports such as long distance running fracture rates are similar between males and females. Most of these fractures have been reported in young college age adults, with the youngest reported case occurring in a 7-year-old ballerina [31]. In addition to having activity-related pain on the ball of the foot, on physical examination most patients will have tenderness well localized to the affected sesamoid, some local swelling, and often increased pain with passive dorsiflexion of the great toe [32].

In many of the reported cases the diagnosis has been delayed, and in one series of 15 cases [33], the time between onset of symptoms and definitive diagnosis averaged 43 weeks. Three factors appear to be responsible for this delay. First, because these fractures occur in competitive athletes, many will try to "run through" or "play through" the pain and not report it to their medical team. Many athletes apparently do not seek medical attention immediately, and some do not do so for many weeks or months [34]. Secondly, in contrast, if the athlete is evaluated soon after the onset of symptoms, as with other stress injuries to bone, the initial radiographs may not demonstrate any abnormality, and the diagnosis of stress injury is not made. Finally, a slowly healing, or ununited, stress fracture may be difficult to distinguish on plain radiographs from a bipartite or tripartite sesamoid which has become symptomatic due to overuse.

The differentiation of a symptomatic partitioned sesamoid ("sesamoiditis") from a slowly healing stress fracture can be suggested by plain radiographs [35] and then facilitated by assessment with bone scanning [36, 37] and/or magnetic resonance imaging [35, 38]. On plain radiographs, the typical stress fracture is transverse and only minimally displaced with a fairly distinct fracture surface (Fig. 13.4). Mittlmeier and Haar [39] have pointed out that instability of a stress fracture may be demonstrated by progressive separation of the fragments over time. In contrast, the components of a partitioned sesamoid are usually rounded with smooth surfaces and, when measured in combination are larger than the size of a non-partitioned sesamoid [35] (Fig. 13.5). Technetium-99 bone scanning will demonstrate increased uptake at a stress fracture site, often before the fracture is visible on plain radiographs [36, 37], and magnetic resonance imaging can demonstrate both stress reactions and stress fractures with a higher degree of sensitivity and greater specificity than bone scanning [38]. It should be remembered that inflammation

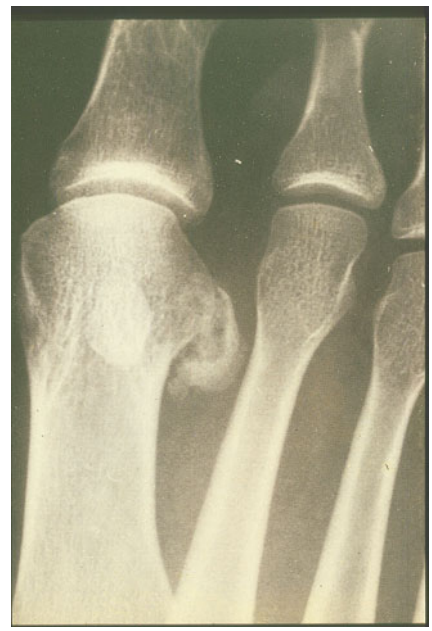

Fig. 13.4 Typical radiographic appearance of a fractured lateral sesamoid

due to overuse involving a partitioned sesamoid may result in a positive bone scan or magnetic resonance finding [35].

Magnetic resonance imaging is the diagnostic modality of choice currently when clinical signs and symptoms point to a stress reaction or fracture as the cause and plain radiographs are normal [35]. The best planes for imaging are coronal (perpendicular to the long axis of the first metatarsal) and sagittal. T1- as well as T2-weighted images (including fat suppression or STIR sequences) should be obtained (Fig. 13.6). Contrast enhancement does not appear to be necessary. These images will demonstrate slightly decreased intensity on T1-weighted images and marrow edema on the T2-weighted images. It should be remembered, however, that similar MRI findings can be seen with sesamoiditis and osteonecrosis of the sesamoid bones [35].

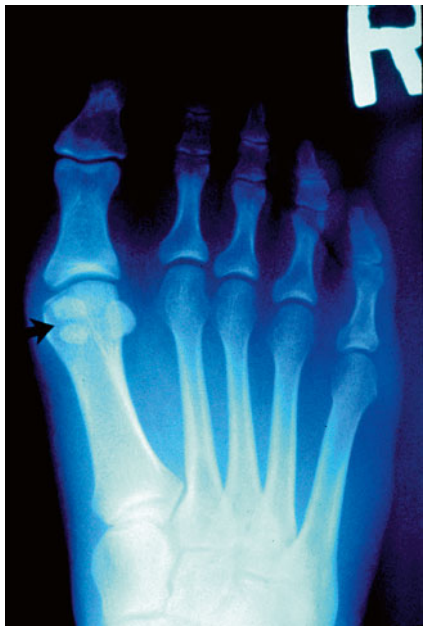

Fig. 13.5 Typical appearance of a bipartite medial sesamoid. Note the smooth rounded surfaces and also note that the two parts in total are much larger than the lateral sesamoid

In summary, stress fracture of the sesamoids is primarily a clinical diagnosis confirmed by imaging studies. When plain radiographs clearly demonstrate a fracture, further imaging is not necessary [35]. When the clinical findings strongly suggest a stress fracture, but X-rays are negative, MRI will usually demonstrate one when it is present. On the other hand, no evidence of increased uptake on bone scan, or no evidence of marrow edema on MRI in the presence of normal radiographic findings (or partition of the sesamoid) should lead to further investigation.

Treatment

Treatment of a stress fracture of the sesamoid is based in part upon the duration of symptoms prior to presentation. As noted above, many athletes will

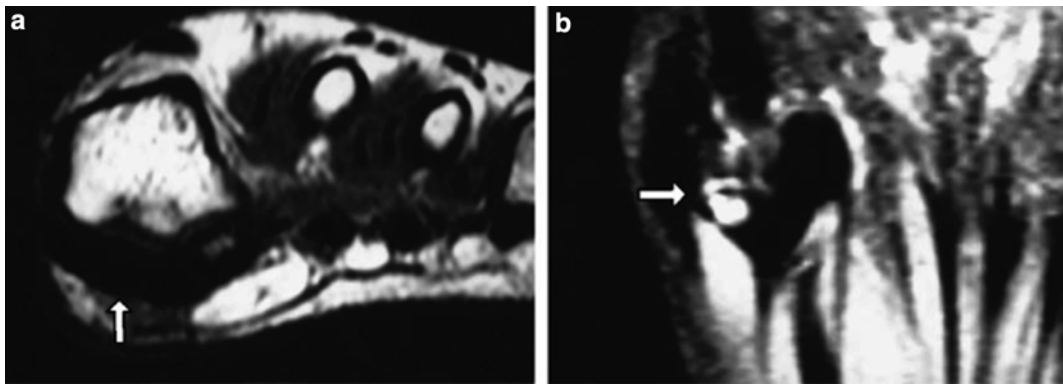

Fig. 13.6 (**a**) Axial T1 demonstrating a medial sesamoid stress fracture with marrow signal replacement by edema. (**b**) Coronal T2 fat-suppressed image of the same patient showing a transverse dark line consistent with fracture. Reprinted from Sports Medicine Clinics, 25(4), Joseph Wall, John F. Feller, Imaging of stress fractures in runners, 781–802. Copyright 2006, with permission from Elsevier

try to ignore the injury pain soon after its onset and may present only after several weeks or months of persistent pain that limits performance. Most of the clinical treatment series reported in the literature focus on surgical treatment in patients with persistent symptoms who have failed nonoperative treatment [37, 39–41]. In these cases, nonoperative treatment typically consisted of cessation of the athletic activity and use of a short leg cast for 6–8 weeks followed by the use of a custom molded orthotic device with gradual resumption of sports as symptoms allowed. Richardson [27] noted that stress fractures of the sesamoid "…usually heal adequately with rest and nonoperative treatment." He recommended non-weight bearing cast treatment for 6–8 weeks followed by the use of a molded orthotic device with relief under the first metatarsal head. Dietzen [25] described the use of a custom made orthotic device consisting of a rubber lined plastic arch support with a J-shaped pad to relieve pressure on the affected sesamoid. This orthotic device was further described by Axe and Ray [30]. Unfortunately, we could find no clinical series in the literature reporting on the effectiveness of early nonoperative treatment of sesamoid fractures.

Three techniques have been described for the surgical treatment of persistently symptomatic nonunion of stress fractures of the sesamoids: excision, bone grafting, and screw fixation.

Excision

In 1982 van Hal et al. [32] were the first to report successful treatment of four patients with ununited and persistently symptomatic stress fractures by complete excision of the sesamoid (two medial and two lateral). All four resumed athletic activities with no residual symptoms. In their series of 26 feet in 10 athletes and 13 "active patients," Saxena and Krisdakumtorn [42] excised the tibial sesamoid in 16 and the lateral sesamoid in 10 with generally good results. However, two patients developed hallux valgus, one developed hallux varus and one developed great toe stiffness requiring a subsequent arthrodesis. In a biomechanical study in cadavers, Aper et al. [43] concluded that complete resection of the medial, lateral, or both sesamoids resulted in a decrease in the effective moment arm of the flexor hallucis longus, but partial resection of either sesamoid alone did not. In light of these findings, more recent authors have recommended partial, rather than total, sesamoid excision, when possible [27, 29, 39]. Biedert and Hintermann [29] described the use of a medial incision to excise the proximal fragment of a tibial sesamoid nonunion with repair of the flexor tendon after excision of the fragment. Richardson [27] describes excision of the fibular sesamoid through both a dorsal and a plantar approach.

Bone Grafting

In the single largest reported series of patients with nonunion of the sesamoids, Anderson and McBryde [44] described a technique of autogenous bone grafting without internal fixation. Among a group of 24 patients, five had sustained an acute sesamoid fracture and the remaining 19 did not recall an acute injury but developed chronic pain as the result of daily athletic activities and were assumed to have stress fractures. Following failure of conservative treatment, all 24 underwent autogenous cancellous bone grafting of a minimally displaced (3 mm or less) tibial sesamoid nonunion. Twenty-one of the 24 were evaluated at long-term follow-up; in 19 cases the nonunion was healed radiographically and 17 patients had returned to their previous level of athletic activity. While there are no other reports in the literature documenting the effectiveness of this bone grafting technique, Anderson reports that he continues to use it in competitive athletes with good success as long as there is minimal diastasis between the fracture fragments, no gross motion between the fragments, and viability of both fragments [45].

Percutaneous Screw Fixation

In 2002 Blundell et al. [40] described a technique of percutaneous screw fixation of both medial (5) and lateral (4) transverse sesamoid fractures after they failed a course of nonoperative care. Eight of the fractures occurred gradually over time and appear to have been stress fractures; seven were in high-performance athletes. Through a stab incision, the distal pole of the sesamoid was exposed and using image intensification a guide wire was

placed across the fracture, which was then compressed with a self-tapping Barouk screw (DePuy International, Leeds, UK). At 6 months after surgery, all nine patients had returned to their previous level of athletic activity with no reported complications. Similar good results of compression screw fixation have been reported by Mittlmeier and Haar [39] and Pagenstert et al. [41]. This technique, however, should be used with caution and only in experienced hands, as the small sesamoid can be shattered easily by overaggressive screw compression.

In summary, in athletes stress fractures can occur in either sesamoid bone, probably as the result of excessive repetitive tension on the plantar plate structures of the first metatarsophalangeal joint. Pain underlying the first metatarsal head combined with local swelling and tenderness should make the examiner suspect a stress reaction or fracture even with normal appearing radiographs. Magnetic resonance imaging may facilitate the diagnosis. When diagnosed early, avoidance of pain inducing activity should lead to resolution of symptoms and fracture healing. Continued stress despite pain (as is often the norm with many athletes) may lead to delayed union or frank nonunion of the fracture. At this point it may be difficult to distinguish partition of the sesamoid from stress fracture, but this is likely only an issue of semantics, as persistent severe symptoms, unrelieved by activity and shoe-wear modifications, will require surgical intervention. Partial excision of an ununited fracture can lead to satisfactory results, as can autologous cancellous bone grafting. Alternatively, in skilled and experienced hands, when there is minimal displacement and two reasonably sized fracture fragments, percutaneous screw fixation can provide a simple means of securing healing and restoring the athlete to a high level of performance.

First Through Fourth Metatarsals

First Metatarsal

In large surveys of foot fractures, the first metatarsal has only rarely been implicated in stress fractures [46, 47]. These fractures typically occur at the proximal metaphyseal/diaphyseal junction [48]. There are case reports in non-athletes noting that increased medial stresses on the foot may be an inciting factor [49, 50]. Case reports of these fractures in athletes suggest that they can usually be treated nonoperatively. One such case report involved the apparent gradual evolution of a Salter-Harris III stress fracture at the base of the first metatarsal in a 14-year-old male athlete. While he could recall no specific trauma to the affected foot, the patient experienced pain dorsally over the first ray with extended walking or when jumping/landing while playing basketball. Although the specific etiology of this fracture was not identified, symptomatic treatment with rocker sole shoe modification and activity limitation yielded complete resolution of his symptoms [51]. In a similar case report, a 14-year-old female field hockey player was found to have bilateral first metatarsal stress fractures. The same treatment was initiated (rigid-soled shoes and relative rest), yielding similarly successful results with radiographic healing of the fracture and clinical resolution of symptoms [52].

Second, Third, and Fourth Metatarsals

Background

The middle metatarsals (second, third, and fourth) are cross-sectionally weaker than the first and fifth metatarsals [53]. Thus, it is no surprise that stress fractures of these metatarsals are common, representing a majority of the reported metatarsal stress fractures in athletes [48, 54–57]. Metatarsal stress fractures were initially recognized over 150 years ago in members of the military and called "march fractures." While they continue to be a frequent cause of injury in members of the military, especially new recruits, middle metatarsal stress fractures are also commonly seen in dancers and other athletes [54, 58–60].

Pathophysiology

Activities or conditions that increase chronic stress on the feet can predispose patients to lesser metatarsal stress fractures. Studies have linked several nonathletic factors to metatarsal stress fractures as well, including varus foot

deformities, diabetic neuropathy, osteoporosis, rheumatoid arthritis, and surgeries such as metatarsal osteotomy, resection of adjacent metatarsals, endoscopic plantar fasciotomy, and Keller bunionectomy [61–65]. Less common causes of lesser metatarsal fracture include reports of soft tissue osteochondromas and plantar ganglion cysts that alter the forces on the plantar aspect of the foot [66–68].

Military personnel represent a unique type of "athlete," and are often subject to unusual foot stresses, such as those that result from marching or operating military machinery. One study found that among Royal Marine military recruits in the UK, third metatarsal stress fractures were the most commonly experienced stress fracture, accounting for 38 % of all stress fractures recorded in the study [69]. Stress fractures in military recruits have been found to result from repetitive, low-impact plantar stresses caused by marching [15, 70], and from the mechanics of running particular to soldiers wearing boots. Biomechanical analyses have shown that forefoot loading pressures (secondary to significantly earlier peak rear foot eversion when running in military boots) were significantly higher in military recruits who had a history of metatarsal stress fractures [71].

While the precise microscopic pathogenesis of stress fractures in the middle metatarsals is not known, studies have shown that the material strength of the metatarsal bones, rather than the geometry, is of central importance in determining their susceptibility to stress fractures. In vitro tests of human second metatarsals demonstrated that a high bone mineral density and high volumetric cortical density were most strongly correlated with an ability to resist cantilever bending [72]. There has also been speculation that repeated stress leads to the formation of microcracks which then progress to overt stress fracture [73, 74]; however, in vitro studies of microcracks in metatarsals have yet to be causally linked to stress fractures [75].

Clinical Presentation

Although commonly associated with runners and ballet dancers, second through fourth metatarsal fractures can occur in any sport that involves repetitive loading of the forefoot [48]. These fractures often present with pain that is poorly localized, ill-defined, and only triggered by specific foot positions; swelling over the dorsum of the foot; and midfoot pain exacerbated by weight bearing [76–79].

Second metatarsal stress fractures in athletes tend to occur in one of two areas: (1) the base and (2) the distal diaphysis. Fractures in these two areas are associated with differing clinical patterns: proximal fractures have been found to be associated with lower bone mass, lower training volumes, and chronic/multiple metatarsal fractures, whereas distal fractures are found in higher-stress, higher-impact training, such as running [80]. Proximal fractures of the middle metatarsals have a worse prognosis than distal fractures, with increased recovery time, and higher rates of delayed union and nonunion [81, 82]. Stress fractures can also be seen in less common areas such as the metatarsal head.

Ballet dancers are at risk for foot injuries, and particularly for stress fractures of the second metatarsal base [77, 83–89]. This predilection for the base of the second metatarsal is relatively unique to dancers, and is thought to be caused by the foot and body movements that put unusual stresses on the feet, including standing "en-pointe" with the entire body weight supported on the toes and the fully plantarflexed foot. Similar stresses and fractures have also been seen in classical Irish dancers [90]. While often seen in dancers, this injury is not unique to dancing and also can be seen in sports such as basketball, soccer, and running [91]. It was traditionally thought that ballet dancers with a relatively short first ray experienced second metatarsal stress fractures more commonly than dancers with a relatively long hallux. However, studies in both dancers and non-dancers have found no correlation between hallux length and middle metatarsal stress fractures [92, 93].

Runners are especially prone to second through fourth metatarsal stress fractures [94], with up to 20 % of 211 collegiate runners sustaining stress reactions to these metatarsals over their careers [8, 9]. Running was the most common sport to cause a stress fracture in a database review of 320 athletes treated for stress fracture,

and those patients who sustained metatarsal stress fractures averaged 38 miles a week for men and 26 miles a week for women [57]. Among experienced runners with no history of metatarsal fracture, a change in footwear from regular running sneakers to barefoot-simulating shoes has been associated with the occurrence of second metatarsal stress fracture, supporting the idea that alterations in the direction and magnitude of repetitive foot stresses can play a role in the pathogenesis of middle metatarsal stress fractures in athletes [95].

Imaging

Imaging should begin with plain radiographs, which may show a progression of periosteal reaction over time, as in the second metatarsal shaft fracture seen in Fig. 13.7. More sensitive studies such as MRI can be used to identify stress reactions before they become evident on X-ray (Fig. 13.8). Recent studies have also shown the effectiveness of ultrasonography in early detection of metatarsal fractures that may not be visible on plain radiographs [78, 96–98].

Treatment

Treatment of second through fourth metatarsal stress fractures is generally nonsurgical. Nondisplaced fractures of the metatarsal shaft have a good prognosis, generally requiring only a soft dressing, a firm, supportive shoe or cast boot, and progressive weight bearing; though immobilization and non-weight bearing is sometimes indicated if less pain persists. An attempt at closed reduction may be indicated if there is dorsal or plantar displacement of the metatarsal shaft of more than 3–4 mm, or if angulation exceeds 10°, though the intermetatarsal ligament typically holds the metatarsal heads well aligned and prevents plantar prominence [99].

Supplemental bone stimulation of fracture healing can be considered in athletes. In one study of 19 young ballet dancers with fractures of the lesser metatarsals, early treatment was initiated with external shock wave therapy (ESWT) or pulsed electromagnetic fields (EMF), with good results in all of the dancers [100].

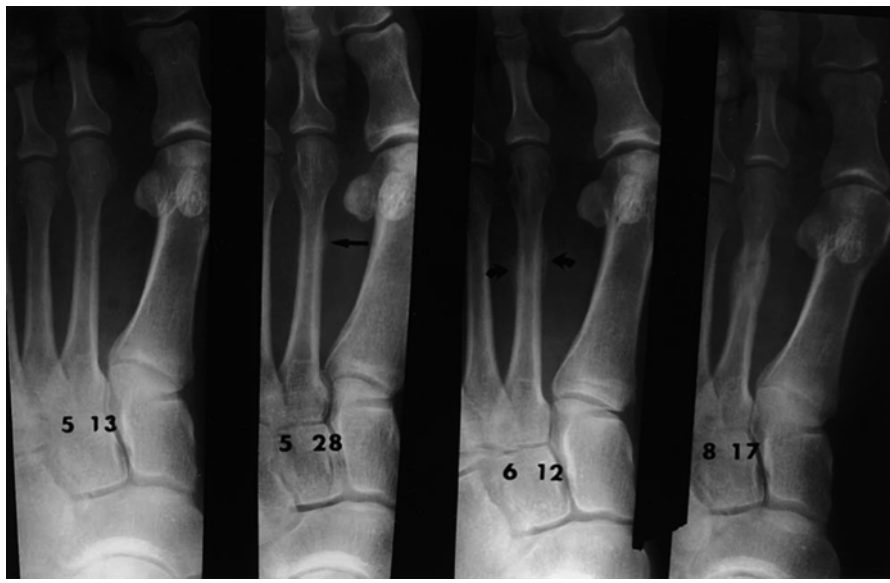

Fig. 13.7 Progression of a second metatarsal stress fracture over 3 months. Note the progression of periosteal reaction. Reprinted from Radiologic Clinics of North America, 40(2), Imaging of stress fractures in the athlete, 313–31. Copyright 2002, with permission from Elsevier

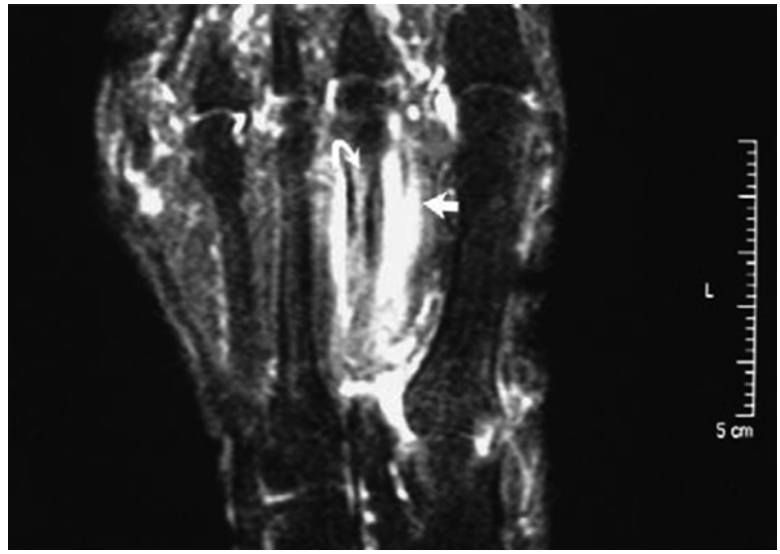

Fig. 13.8 Short tau inversion recovery (STIR) image of a second metatarsal stress fracture. The curved arrow shows bone marrow edema and the straight arrow surrounding soft tissue swelling. Reprinted from Sports Medicine Clinics, 25(4), Joseph Wall, John F. Feller, Imaging of stress fractures in runners, 781–802. Copyright 2006, with permission from Elsevier

Prevention

Prudent increase in training, footwear and early imaging with sensitive modalities may help prevent second through fourth metatarsal stress fractures. In several studies of stress fractures in military recruits, alteration of the stresses on the feet using orthotics was found to reduce the pathological stresses and the incidence of middle metatarsal stress fractures [79, 101]. A study of collegiate basketball players showed that prophylactic MRI imaging of the feet can reveal pathologic changes secondary to athletic stresses in the metatarsals, including bone marrow edema that may precede stress fracture [102]. Recognition of this pre-fracture state may allow prevention and earlier treatment.

Fifth Metatarsal

Classification/Grading

Historically, there has been confusion and inconsistency in the classification of fractures of the fifth metatarsal. Fractures of the proximal fifth metatarsal are often associated with the eponym Jones fractures. In 1902 Sir Robert Jones reported a small series of fractures, including his own, at the metaphyseal/diaphyseal junction of the fifth metatarsal [103]. These fractures were further clarified as extending into the articular facet between the fourth and fifth metatarsal [104]. Jones fractures have typically been considered acute injuries without prodromal symptoms, and therefore are not considered stress fractures.

The terms "pseudo-Jones fractures" and "dancer's fracture" also confuse the issue of classification. Pseudo-Jones fractures are avulsion injuries of the proximal fifth metatarsal base which exit proximal to the articulation of the fourth and fifth metatarsal, and are acute injuries [105]. The term dancer's fracture typically refers to an acute spiral oblique fracture of the fifth metatarsal shaft [106]. When describing fractures, it is more useful to avoid eponyms and to instead specify the location and chronicity of the fracture.

The fifth metatarsal has classically been divided into three zones with zone I representing avulsion injuries, zone II representing "Jones" fractures, and zone III representing stress fractures in the proximal 1.5 cm of the diaphysis (Fig. 13.9a) [55, 107–109]. It has been suggested by Polzer et al.

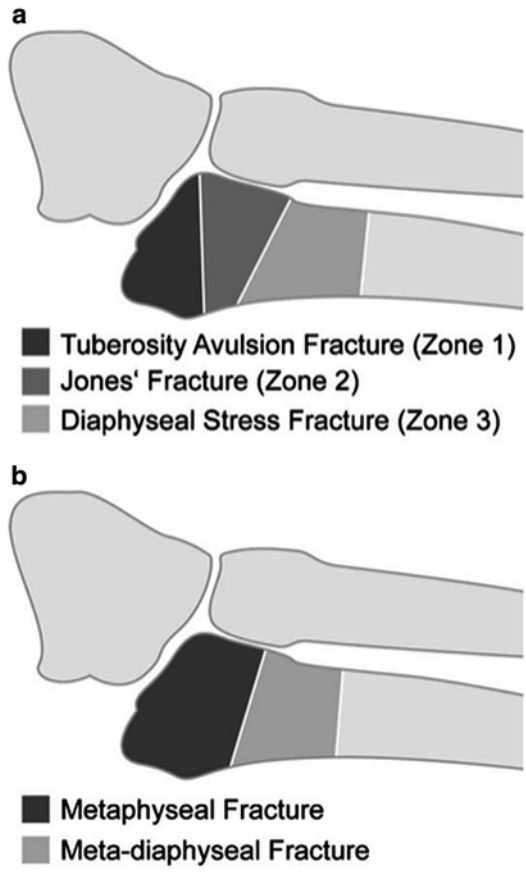

Fig. 13.9 (**a**) The classification of Lawrence et al., indicating the proximal diaphysis as the site of fifth metatarsal stress fractures [108]. (**b**) The classification of Plozer et al., which combines the region of traditional diaphyseal stress fractures with fractures that extend into the distal 4–5 metatarsal articulation, as both behave similarly. Reprinted from Injury, 43(10), Polzer H, Polzer S, Mutschler W, Prall WC, Acute fractures to the proximal fifth metatarsal bone: development of classification and treatment recommendations based on the current evidence, 21626–32. Copyright 2012, with permission from Elsevier

that, given the amorphous differentiation between zones II and III, fractures in both areas be considered as a single group, as their etiology, healing potential and treatment preferences are similar (see Fig. 13.9b) [110, 111]. Indeed, it appears that consistently differentiating between these zones is difficult, and any fracture distal to the tuberosity of the fifth metatarsal may have a component of stress and overuse, even if the fracture occurs after an acute incident.

One of the most frequently cited classification systems focusing on chronicity is that of Torg, which was designed to aid in clinical management. Torg originally described 46 fractures distal to the tuberosity of the fifth metatarsal, all of which occurred in athletes [112]. Type I fractures represent acute or acute-on-chronic injuries and have evidence of mild periosteal reaction and a sharp fracture line. Type II fractures are delayed unions and demonstrate a fracture line with associated periosteal reaction, with or without radiolucency from bone resorption around the fracture line, and with medullary sclerosis. Type III fractures are nonunions and demonstrate obliteration of the medullary canal along with periosteal hypertrophy and a widened fracture line. Among surgically treated fifth metatarsal stress fractures, there is evidence of increasing time to union with Type II and III fractures, supporting the clinical relevance of this classification [113, 114].

More recently, a classification system has been proposed that highlights the importance of fracture gap. Lee et al. divided fifth metatarsal stress fractures into complete (A) and incomplete (B) categories [113, 114]. Type A fractures are further subdivided into A1 fractures, which appear acute without signs of periosteal reaction, and A2 fractures, which are acute-on-chronic injuries and have evidence of periosteal reaction. Type B fractures are subdivided into B1 fractures, which have a fracture gap <1 mm, and B2 fractures, which have a fracture gap >1 mm. Among athletes treated with tension band wiring, the B2 fractures with a gap >1 mm demonstrated a significantly increased time to union, indicating that the presence of a fracture gap may well influence outcome in athletes [113, 114].

In summary, stress fractures of the fifth metatarsal occur distal to the tuberosity. They are classified based on signs of bony reaction to stress (periosteal reaction), the extent and size of the fracture line, and the degree of medullary sclerosis.

Pathophysiology

Stress fractures are a result of repetitive stress to bone beyond its capacity to remodel or repair itself, and stress fractures of the fifth metatarsal

are no different. There have been attempts to clarify the forces on the fifth metatarsal, the athletic maneuvers that cause stress fractures, the foot shape that may predispose athletes to them, and the reason that they have historically demonstrated difficulty in healing.

Biomechanical modeling has identified the area of maximal stress on the fifth metatarsal as 3–4 cm distal to the tip of the tuberosity, and occurring when an oblique force is exerted on the metatarsal at 30–60° below the horizontal plane [115]. This could occur in a variety of different maneuvers and actions during sporting endeavors. In contrast to a proximal fifth metatarsal avulsion injury, there is some evidence that more distal stress injuries are not inversion injuries, but occur with atraumatic repetitive loading common to many sports [116]. In a study of college athletes designed to determine the athletic maneuvers that caused the largest bending moment to the fifth metatarsal, the highest stress was seen during acceleration maneuvers, followed by straight line running, as opposed to cutting or jumping maneuvers [117].

There has been speculation that a high arch can give rise to excessive overloading of the lateral border of the foot and predisposes to fifth metatarsal stress fracture. In a group of 50 soccer players with stress fractures of the fifth metatarsal, radiographic parameters were compared against sport and age-matched controls. Those with fractures were found to have a more cavus foot (higher talocalcaneal angle, higher calcaneal pitch) [118]. Among a group of 68 professional football players with fifth metatarsal fractures of various types, including stress fractures, there was a statistically significant increase in the varus alignment of the foot [119]. In another study of 20 top-level athletes who developed fifth metatarsal stress fractures, 90 % had a varus midfoot structure [117].

In contrast, a case-control study of 10 professional soccer players with fifth metatarsal stress fractures revealed no increased static varus alignment among the injured players, and while they did dynamically unload the lateral boarder of the foot, it is unclear whether this was a reactive response or a potential cause of stress overload [120]. Beyond simple varus alignment of the foot, there is some evidence that the intrinsic shape of the fifth metatarsal may contribute to the development of stress fractures in athletes; a greater prominence and curvature to the fifth metatarsal (a larger 4–5 intermetatarsal angle and lateral deviation of the axis of the fifth metatarsal) was associated with fracture in a group of soccer players [118]. Additionally, when a group of 168 athletes treated with tension band wiring for fifth metatarsal stress fractures was reviewed, greater prominence and curvature of the fifth metatarsal was associated with a higher risk of refracture [121]. This study also identified an increased body mass index (BMI) in those patients with fifth metatarsal stress fractures [121].

The proximal diaphysis of the fifth metatarsal has been described as a watershed area of decreased vascularity, an area where the medially entering nutrient artery and the metaphyseal perforators coalesce [55, 122, 123]. This may have implications for healing, whether from an acute injury or repetitive injuries, and may also be a contributor to the formation of stress fractures in this area.

Clinical Presentation

As stress fractures are the result of repetitive load beyond the bone's capacity to handle that load, they are by definition chronic injuries, and they are usually preceded by prodromal symptoms. Lateral border foot pain can be present from 3 to 18 months prior to presentation [124]. As the fifth metatarsal is a relatively subcutaneous bone, pain is relatively easy to localize over the lateral border of the foot, and may be reproducible with palpation. However, chronic changes may not manifest themselves until an acute injury, and the so-called acute-on-chronic injuries may be the culmination of repetitive stress reactions in the bone that are subclinical and do not cause prodromal pain [112].

Fifth metatarsal stress fractures occur in a variety of sports and are less common than other stress fractures in the lower extremity. A database review of 320 athletes treated for bone-scan-confirmed stress fractures over 4 years found that metatarsal fractures were the third most common

stress fracture (8.8 %) behind tibial and tarsal fractures [57]. Running was the most common sport to cause a metatarsal stress fracture [57], however, second through fourth metatarsal stress fractures are more common in runners than fifth metatarsal stress fractures [94].

Large-scale studies have identified the relatively rare occurrence of fifth metatarsal stress fractures among basketball and football players. Among a group of 5,654 basketball players prospectively followed over two seasons, only 9 fifth metatarsal stress fractures were identified [125]. Among a group of 1,987 professional American football players from 2004 to 2009, the incidence of fifth metatarsal stress fracture was 3.42 % [119].

While fifth metatarsal stress fractures are often associated with football and basketball, they have also been described in a variety of other sports, including soccer, handball, baseball, gymnastics, lacrosse, field hockey, volleyball, and other sports that involve repetitive loading of the lower extremity [112, 116, 120]. Dancers experience a large number of overuse injuries of the foot and ankle, and, while second metatarsal stress fractures are typical in dancers in general, especially modern dancers may be at higher risk for fifth metatarsal stress fractures [84, 86]. Stress fractures in professional dancers, including fifth metatarsal fractures, may be related to prolonged periods of amenorrhea; a dietary and menstrual history should be taken on any female athlete with signs or symptoms of stress fracture [76].

It is also important to realize that not all fifth metatarsal stress fractures occur in the classic location; there are reports of stress fractures in the fifth metatarsal head and obliquely in the mid-diaphysis [126, 127].

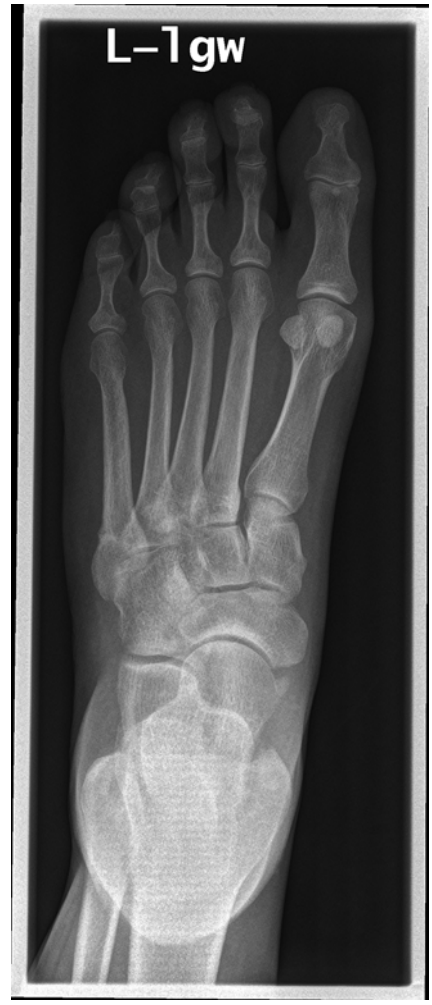

Fig. 13.10 Radiograph demonstrating lateral cortical thickening of the proximal fifth metatarsal diaphysis

Imaging

Imaging of suspected fifth metatarsal stress fractures should begin with plain radiographs of the foot, where lateral cortical thickening of the proximal diaphysis can be seen (Fig. 13.10) [116]. The cortical thickening may progress to include a radiolucent line in the same area (Fig. 13.11).

If radiographs are negative but clinical suspicion is high, more sensitive modalities should be pursued. X-rays may not show early stress reactions prior to periosteal reaction or fracture, but MRI will show focal marrow edema at this stage [128]. T2 and short tau inversion recovery (STIR) are the most sensitive sequences for picking up this early marrow edema [129]. Bone scan will show increased uptake in the presence of a stress reaction and should be positive before plain X-rays as well [129].

Treatment

The difficulty of treating fifth metatarsal stress fractures in athletes has been recognized for quite some time. In one of the earliest case series of 20 athletes (and one non-athlete) treated for

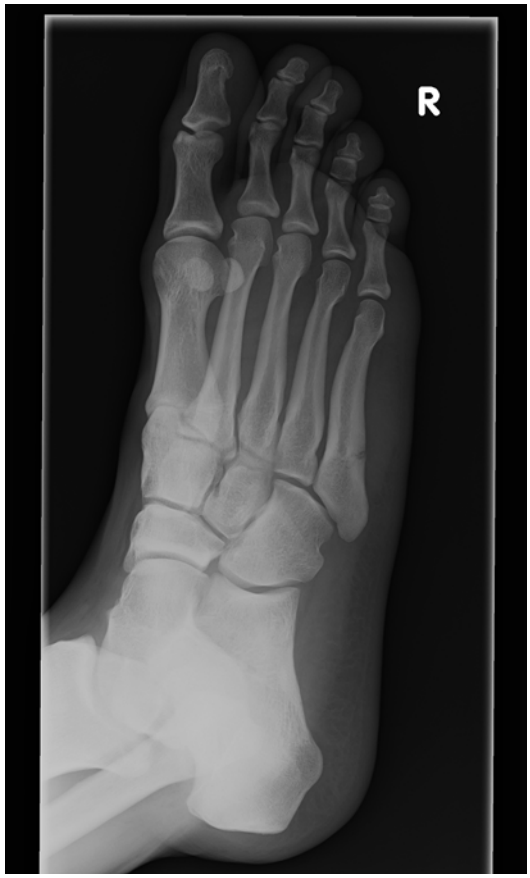

Fig. 13.11 Radiograph demonstrating a lucent fracture gap in the proximal fifth metatarsal shaft

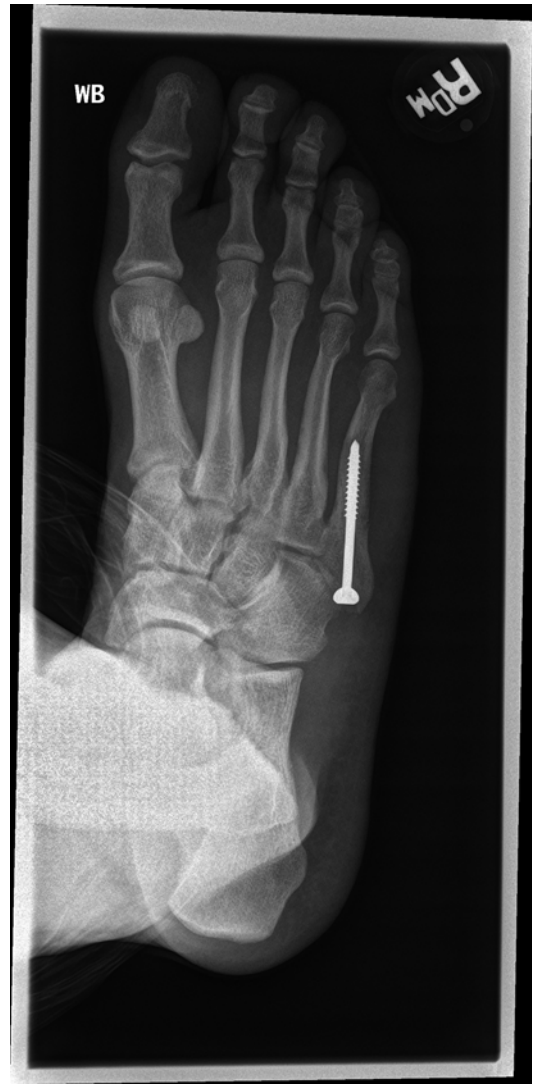

Fig. 13.12 Intramedullary screw fixation of a fifth metatarsal stress fracture

fifth metatarsal fractures operatively and nonoperatively, all but two patients had delayed healing of up to 20 months or symptomatic nonunions requiring bone grafting [130]. In another series collected over a period of 14 years, 6 of 12 fifth metatarsal stress fractures seen in athletes resulted in nonunion that needed further treatment [34].

Nonoperative treatment for fifth metatarsal stress fractures typically involves non-weight bearing or partial weight bearing in a cast or cast boot until symptoms resolve and X-rays show healing. Operative fixation typically focuses on stabilizing the fracture with hardware and bone grafting for recalcitrant fractures, with numerous techniques described, but many focusing on intramedullary screw fixation (Fig. 13.12). In general, with both operative and nonoperative treatment, return to play should be delayed until pain has resolved and radiographic signs of healing are observed.

Because of the propensity to delayed healing with nonoperative treatment, athletes may be best served with operative fixation of these fractures. In a study of 22 athletes with proximal fifth metatarsal stress fractures who were followed over an average of 3.5 years, 12 of 18 patients treated nonoperatively had incomplete healing at 6 months [116]. Several studies indicate that operative fixation may decrease healing time and the

time until return to sport. A systematic review of the literature from 1994 to 2010 revealed patients with operatively treated proximal fifth metatarsal fractures return to sport at an average of 12 weeks and nonoperatively treated patients return to sport at an average of 24 weeks [107]. Of the 21 studies identified, the level of evidence was relatively low: one level I study, 6 level III studies, and 14 level IV studies [107]. While their study group did exclude those with prodromal symptoms of greater than 2 weeks, the only level I study looking at treatment of proximal fifth metatarsal fractures (excluding avulsion fractures) showed a benefit in time to union and return to sport in the operatively treated group [131].

Numerous level III and IV studies have looked specifically at the treatment of fifth metatarsal stress fractures in athletes, most focusing on intramedullary screw fixation. In a group of nine basketball players with fifth metatarsal stress fractures treated with intramedullary screw fixation, union was achieved at an average of 9.5 weeks with return to play at an average of 9 weeks [125]. In another group of 10 athletes treated with intramedullary screw fixation, all fractures healed at an average of 7.5 weeks, with return to sport at 8.5 weeks, though 70 % of the patients had continued pain which was relieved by shoe-wear modification [124]. A case series of 24 athletes treated with 4.5 mm cannulated screws for fifth metatarsal fractures, some of which were stress fractures, showed 100 % functional union and return to sport at an average of 7.5 weeks [132]. In a series of 20 top-level athletes treated with intramedullary screws for fifth metatarsal stress fractures, there was one refracture and an average return-to-sport time of 9 weeks; all but one patient returned to their prior level of sport [133].

The size and length of screw used for intramedullary fixation does not appear to be of clinical relevance. A comparative case series of athletes showed no difference between results with 4.5- vs. 5.5-mm cannulated screws, though two 4.5-mm screws bent without breaking [134]. Similarly, no statistically significant difference in outcomes was seen with differences in screw length, screw fill, or screw diameter in a group of professional football players treated with intramedullary screw fixation [119].

Bone block grafting is another viable technique in athletes. Torg stressed the importance of reconstituting the obliterated medullary canal in delayed unions or nonunions by placing a block of bone graft across the fracture site [112]. In 10 soccer players, a reverse inlay bone block with no additional fixation resulted in return to play after 3 months [135].

The other most commonly used technique in athletes is tension band wiring. A case series of 42 elite athletes treated for a fifth metatarsal stress fracture with tension banding on the lateral aspect of the metatarsal over two bicortical screws showed all patients returned to their previous level of sport with an average time to union (as determined by CT scan) of 75 days [136]. Tension band wiring has also been shown to be a viable treatment option around intramedullary Kirschner wires, with all 27 athletes treated in one study returning to full activity by 14.5 weeks [137]. A cadaver study indicated that the stress placed on the proximal fifth metatarsal is rotatory and tensile, bringing into questions whether rotatory control of the proximal portion of a fifth metatarsal fracture is necessary [138].

Not all studies demonstrate the superiority of operative treatment. One study of professional football players treated for fifth metatarsal stress fractures showed a 12 % nonunion rate among operatively treated patients which did not differ statistically from a nonoperatively treated group [119]. There has also been some interest in alternate nonoperative modalities of treatment as well. In a small series of 10 soccer players with fifth metatarsal stress fractures, extracorporeal shock wave therapy allowed return to sport at an average of 8 weeks [139].

Prevention

Certain nonoperative measures may help prevent fifth metatarsal stress fractures. Among 11 collegiate basketball players, a medial arch support reduced stress on the fifth metatarsal during common maneuvers, and may reduce forces on the foot that can lead to stress fractures [140]. While not specific to fifth metatarsal stress fractures,

evidence from over 20 years of observation in Israeli army recruits underlines the importance of sleep, gradual increase in load bearing exercise, and rest at the first indication of prodromal symptoms to prevent progression to a complete stress fracture [141].

In summary, fifth metatarsal stress fractures are difficult to classify because of historical classification systems that focus on location and eponyms. Stress fractures typically occur distal to the tuberosity and are distinct from avulsion fractures. Intrinsic and extrinsic factors that contribute to overloading of the lateral border of the foot may increase the risk of stress fractures in athletes. These fractures are relatively rare and occur in a large number of sports, where they are typically, but not always, preceded by prodromal pain. Periosteal reaction and fracture lines are typically seen on plain radiographs, but higher-level imaging may show stress reactions prior to frank fracture. These fractures are particularly prone to delayed union or nonunion in athletes. Several studies, but few high-level studies, show that operative treatment decreases the time to return to full sporting activity.

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Stress Fractures of the Ribs and Girdle

14

Timothy L. Miller

Introduction

Stress fractures have been historically regarded as predominantly occurring in the lower extremities secondary to the repetitive impact loading of walking, running, or jumping [1–3]. Stress injuries of the ribs and shoulder girdle are much less commonly reported and subsequently these injuries are often omitted from the differential diagnosis of rib or upper extremity pain [4, 5]. Few case series have described the precipitating activities and common locations of these injuries given their infrequency. The two largest series of rib and upper extremity stress fractures include 44 cases described by Sinha and associates [6] and Miller and Kaeding's case series of 70 patients [7]. Sinha et al. found patterns of injury sites that were able to be grouped into five categories: (1) weight lifters, (2) weight bearers, (3) throwers, and (4) swingers. Miller and Kaeding further categorized the causative activities of rib and upper extremity stress fractures into (1) axial rotators, (2) rowers, (3) overhand throwers, (4) weightbearers, and (5) weight lifters.

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Rib and upper extremity stress fractures account for fewer than 10 % of all stress fractures but can be troublesome injuries for athletes and manual laborers [1–4]. As awareness of overuse injuries of the thorax and shoulder girdle has increased, so has the rate of diagnosis of stress fractures of the ribs and upper extremities [7]. Appropriate evaluation for these injuries requires a thorough history and physical examination. Radiographs may be negative early, requiring bone scintigraphy or MRI to confirm the diagnosis. Nonoperative and operative treatment recommendations are made based on location, injury classification, and causative activity. To prevent a delay in the diagnosis and treatment of these injuries, clinicians should be aware of the common precipitating mechanisms and locations of these injuries as well as the indications for operative and nonoperative treatment.

Rib and shoulder stress fractures are diverse in their presentation, appearance, and healing potential. Stress injuries to bone represent a continuum of mechanical failure ranging from simple bone marrow edema (stress reaction) to a small microcrack with minor cortical disruption to a complete fracture with or without displacement to nonunion. Most reported stress fractures of the ribs and upper extremities have been described in case reports and small case series. However, given the relative rarity and the failure to report upper extremity stress fractures, the exact percentage or likelihood of their development is difficult to determine with certainty.

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Risk Factors

In the shoulder girdle and ribs strain is generated by the rotational torque of swinging or throwing and by the tensile and compressive forces produced from muscle contraction [8]. Additionally, repetitive axial loading of upper extremity may generate forces sufficient to produce microtrauma to bone. Muscle function influences the amount of energy directly absorbed by the bones and joints therefore affecting their susceptibility to stress injury. As muscles fatigue, they are less able to dissipate externally applied forces. Two key modifiable risk factors for these injuries are pre-participation conditioning and the volume (frequency, duration, and intensity) of the causative activity [9]. Neuromuscular conditioning plays a significant role in enhancing the shock absorbing and energy-dissipating function of muscles and soft tissues [10]. Other predisposing factors include abnormal bony alignment, muscular imbalance, improper technique/biomechanics, and poor blood supply to specific bones [10].

Clinical Presentation

Athletes with atraumatic shoulder or chest wall discomfort associated with repetitive activity should be evaluated with concern for possible stress fracture [4, 10, 11]. Causative mechanisms may involve repetitive resisted scapular retraction, humeral torsion (e.g., pitcher, javelin) or weightbearing (e.g., cheerleading, gymnastics). Muscle contraction may generate both compressive and tensile loads on the skeletal structures of the thorax and upper extremity leading to stress injuries of bone [10]. Typically, patients cannot recall a specific injury or trauma to the injury site, and onset is most often insidious as in the lower extremities. Given the relative infrequency of rib and shoulder girdle stress fractures, the potential for concomitant soft tissue overuse injuries is high and must be diagnosed and treated if present.

Athletes with stress fractures of the ribs and shoulder girdle present initially with pain that is present only during the inciting activity [4]. If the activity level is not decreased or modified, symptoms usually persist or worsen. Those individuals who continue to train and compete without modification of activities may develop pain with activities of daily living and potentially progress to complete fracture with or without displacement [10].

Physical Examination

The physical examination performed for a suspected rib or shoulder girdle stress fracture should include evaluation of the neck, chest, heart, lungs, and abdomen to rule out non-musculoskeletal causes of shoulder, rib, and thoracic pain. Examination should begin with a thorough inspection of the skin and soft tissues. Palpation for tenderness, active and passive range of motion, and strength testing should be performed for all affected bones and joints of the cervical spine, scapulothoracic joint, sternoclavicular joint, acromioclavicular joint, glenohumeral joint, and the elbow. Unlike non-musculoskeletal sources of pain, stress fractures often produce reproducible point tenderness at the affected site. Soft tissue or bony swelling also may be present.

In the early stages of the injury, it may be necessary to have the patient perform or recreate the causative activity in order to reproduce the symptoms [10]. Any biomechanical causes of injury, including muscle imbalance or abnormal mechanics of the throwing or rowing motion, should be noted at this time. Tuning fork testing may help identify occult fractures. A thorough neurovascular exam is essential because vague exertional upper extremity pain may also be due to peripheral nerve entrapment and/or peripheral vascular disease, or other vascular etiologies such as deep vein thrombosis and thoracic outlet syndrome [10].

Differential Diagnosis

The most common differential diagnoses for rib stress fractures include the following [10]:

Costochondritis

Intercostal neuralgia

Intervertebral disk pathology
Skin infection (Methicillin-resistant *Staphylococcus aureus*, etc.)
Herpes zoster
Cardiac-related chest pain
Pneumothorax
Peripheral vascular disease
Pleuritis
Tumors

Causative Activities

Muscle contraction in the upper extremity and thorax produces tensile, compressive, and rotational stress on bone. Throwing and/or swinging motions are the two most common inciting activities to generate these forces [7]. Less common mechanisms of creating bone stress in the shoulder include repetitive axial loading, resisted retraction of the scapula, and weight lifting [7].

Miller and Kaeding reported on seventy cases of rib and upper extremity stress fractures in skeletally mature patients collected over a 10-year period [7]. The same authors continue to record a growing series of 80 rib and upper extremity stress fractures with 10 of the cases currently unpublished. Analysis of the causative activities of these cases showed notable patterns for causative activities. As mentioned earlier injury patterns have allowed division of the majority of patients with rib and upper extremity stress fractures into one of five categories. The categories include (1) upper extremity weight bearers (gymnastics, cheerleading), (2) rowers, (3) axial rotators (golf, tennis, discus), (4) overhead throwers, and (5) weightlifters. The distribution of these injuries is detailed in Table 14.1 [10].

A clear connection has been observed in rowers. All 11 rowers were diagnosed with stress fractures of the ribs. Ten of the 11 developed their stress fractures in the lower ribs. Five of 11 rowers developed stress fractures in multiple lower ribs. Like rowers, the axial rotator group showed a strong predilection for fractures of the ribs (7/10). Of the seven athletes with rib fractures, six occurred in the lower ribs with two athletes presenting with injury to multiple lower ribs. Among overhead throwers, patients showed a tendency for injuries around the elbow (9/16).

Weightlifters showed the greatest variability in anatomical location of injury, with injuries occurring as far proximal as the sternum and as far distal as the scaphoid. This group also showed a significantly disproportionate number of rib and shoulder girdle stress fractures (13/24). Notably, this group of patients sustained more injuries to the first rib (7/24) than any other group. A clear explanation for these injury patterns cannot be determined other than the variety of repetitive bending, torsional and axial loading forces applied to the thorax and upper extremity during weight training [10].

Table 14.1 Anatomic distribution of rib and upper extremity stress fractures by causative activity[a]

Weight bearer ($n=12$)	Rower and axial rotator ($n=21$)	Overhead thrower ($n=16$)	Weight lifter ($n=24$)	Miscellaneous ($n=7$)
Olecranon (1)	First rib (2)	Clavicle (2)	Acromion (4)	Phalanx (1)
		Scapula (1)		
Ulnar shaft (3)	Lower ribs (16)	First rib (3)	First rib (7)	Ulnar shaft (3)
Distal radius (2)	Ulnar shaft (1)	Lower ribs (1)	Proximal humerus (1)	Distal humerus (2)
Scaphoid (3)	Radial shaft (1)	Distal humerus (5)	Ulnar shaft (2)	Metacarpal (1)
First rib (1)	Metacarpal (1)	Olecranon (4)		
Sternum (1)		Scaphoid (4)		
Distal humerus (1)		Sternum (2)		
		Proximal radius (1)		
		Coracoid (1)		

[a]Adapted from [7]

Stress Fracture of the Ribs

Rib stress fractures have been reported in several sports, including discus, rowing, rugby, golf, weightlifting, volleyball, gymnastics, judo, tennis, table tennis, baseball, basketball, soccer, javelin, backpacking, and wind surfing [4, 7]. Tensile muscular forces (rather than axial compressive forces) are predominantly responsible for rib stress fractures, as this is a non-weightbearing location [8]. The most common sites of fracture include the first rib anterolaterally, the fourth through ninth ribs posterolaterally, and the upper ribs posteromedially [10, 11].

First Rib

The sports most commonly associated with first rib stress fractures (Fig. 14.1a, b) such as baseball pitching, basketball, lacrosse, weightlifting, ballet, javelin, and tennis involve repetitive overhead positioning of the arm [7, 12, 13]. Patients with first rib stress fractures present with insidious onset of dull, vague pain in the anterior cervical triangle and mid-clavicular region, with occasional radiation to the sternum and pectoral region [11]. Repetitive scalene muscle contractions elevate the first rib, while serratus anterior and intercostal muscles depress it [14]. These opposing forces generate bending and torsional forces leading to microtrauma. Prisk and Hamilton [15] proposed the "trapezius squeeze test." This test involves applying pressure to the anterior trapezius muscle, causing involuntary contraction of the muscle and eliciting rib pain. This test was found to be reliable for diagnosing first rib stress fractures on physical examination in five cases of stress fractures in ballet dancers [10, 15].

Second Through 12th Ribs

Repetitive strain on the torso is a cause of middle- and lower rib stress fractures (Fig. 14.2a, b), and these are most commonly described in athletes involved in rowing, discus, and golf [6, 7, 14, 16, 17]. Patients present with increasing lateral chest pain and are diagnosed most commonly by radionuclide scans [16]. Other athletic activities associated with these fractures include tennis, gymnastics, and throwing sports [4, 7]. Among rowers, fractures are found most commonly between the fifth and ninth ribs, and pain generally is greatest at the finish of a stroke and may be exacerbated by coughing, sneezing, or deep inhalation [7, 10, 14]. Among golfers, Lord et al. [18] described 19 cases of rib stress fractures. Sixteen of the 19 golfers sustained injury

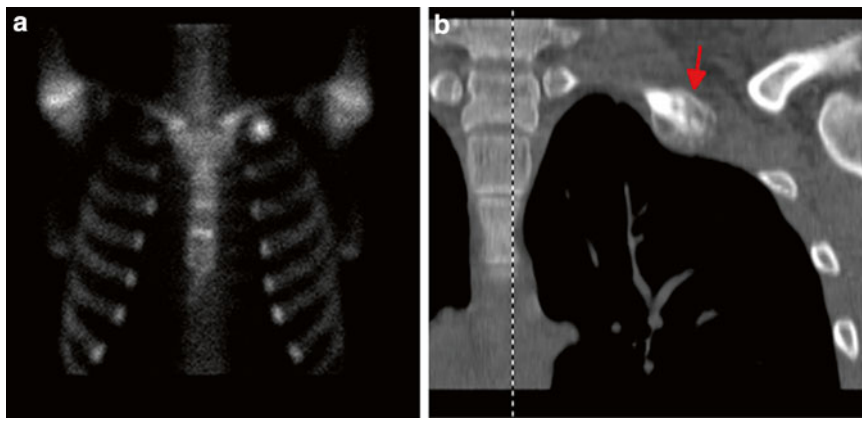

Fig. 14.1 Bone scan (**a**) and coronal CT scan (**b**) images demonstrating left first rib stress fracture in a male collegiate gymnast. CT scan demonstrates healing with abundant callus formation

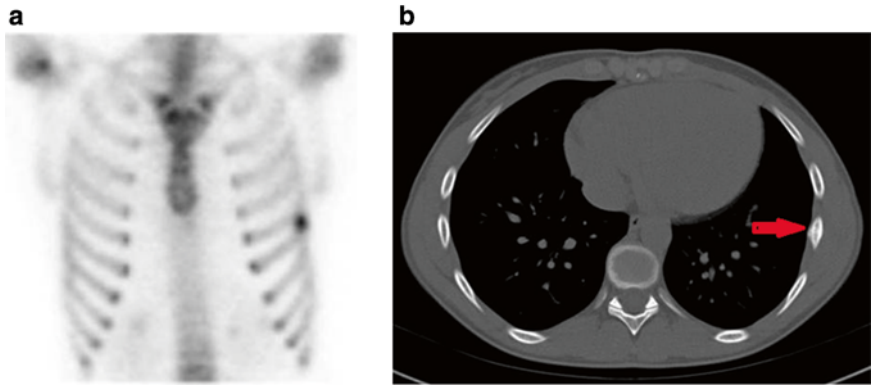

Fig. 14.2 Bone scan (**a**) and axial CT scan (**b**) images demonstrating stress fracture of the left mid seventh rib. CT scan demonstrates fracture callus present

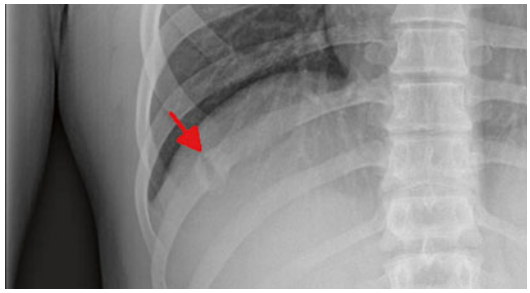

Fig. 14.3 Anteroposterior chest radiographs demonstrating nonunion (Grade 5) of the right tenth rib stress fracture in a male collegiate rower

on the leading arm side of the trunk. The posterolateral aspects of the fourth through sixth ribs were the most commonly injured sites [18]. The authors suggested that the ribs on the leading arm side are most commonly involved because of repetitive contraction of the serratus muscle through all phases of the golf swing on the leading side compared with the trailing side [18].

The treatment of rib stress fractures is nearly always nonoperative, with the initial goal being to provide symptomatic relief. In general, rib stress fractures rarely fail to heal with modification or complete discontinuation of the causative activity for 4–6 weeks [4]. Treatment includes relative rest by avoiding overhead lifting, throwing, or rowing sports. Nonunion of the ribs (Fig. 14.3) has been described, but this is very rare and may be asymptomatic [4, 15].

Sternum

Figure 14.4 shows the coronal and sagittal MRI images of a 28-year-old male competitive weight lifter with a midsternal stress fracture. Stress fractures of the sternum may be diagnosed on radiographs, CT scan, technetium bone scan, or MRI. Athletes with this injury typically present with dull to progressively sharp anterior chest pain. In addition to weightlifting, stress fractures of the sternum have also been described in athletes participating in golf [19] and wrestling [20]. In one case described in the literature, the athlete described an audible "pop" [21] while performing core exercises for the abdominal muscles during training. In all cases of sternal stress fractures described in the literature, the athletes were performing intensified repetitive activities of the pectoralis muscles or rectus abdominis in preparation for competitions. Relative rest from the causative activity led to resolution of symptoms within 6–10 weeks.

Scapula

Stress fractures of the scapula in athletes are uncommon [22, 23]. Cases reported in the literature include a gymnast, a baseball pitcher, a jogger carrying weights, and a professional football player [4, 10, 22, 24]. Additional cases have included stress fractures of the dominant shoulder

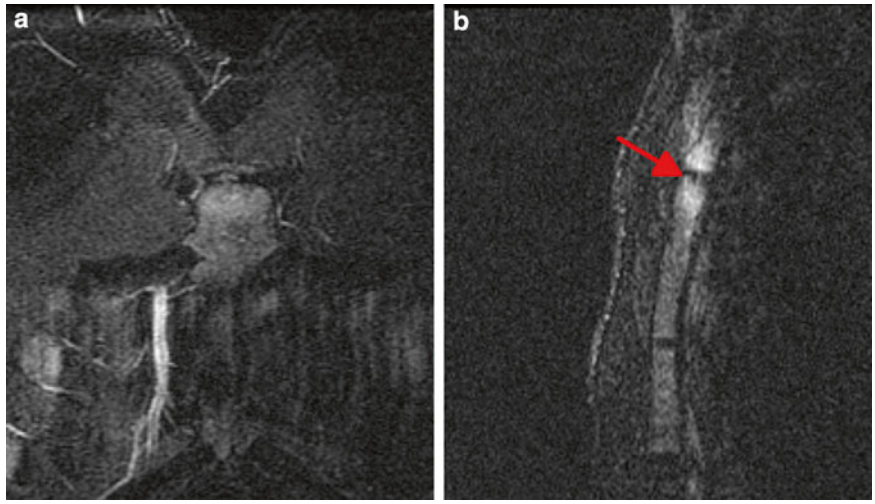

Fig. 14.4 T2 coronal (**a**) and sagittal (**b**) MRI series demonstrating stress fracture of the mid sternum in a competitive weight lifter

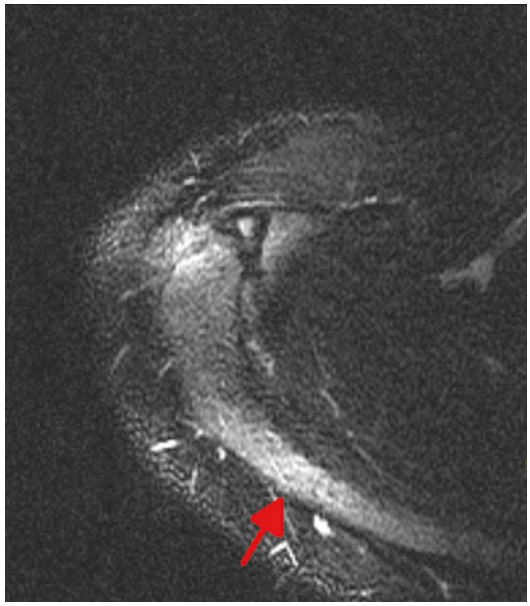

Fig. 14.5 T2 axial MRI demonstrating Grade 2 stress fracture of the medial scapular spine in the dominant right shoulder of a high school quarterback. An unstable os acromiale is also evident

in the scapular spine of a high school football quarterback (Fig. 14.5) and a trap shooter with a coracoid stress fracture [25] and an athlete undergoing shoulder rehabilitation following shoulder surgery. Scapular sites where stress fractures have been diagnosed include the coracoid, acromion

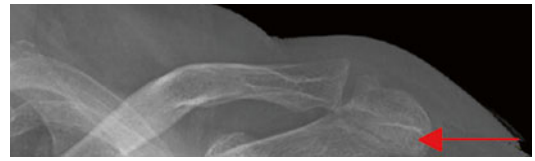

Fig. 14.6 Anteroposterior radiographs of the left shoulder demonstrating Grade 3 stress fracture of the acromion process

(Fig. 14.6), scapular spine, and scapular body [10, 22, 26]. Given that the scapula has a complex array of muscle attachments and corresponding bone stress patterns, these injuries represent a diagnostic challenge to clinicians. Depending on the motion, stress concentration occurs at a variety of locations in the scapula. Authors have theorized that the likely cause of these injuries is overuse or fatigue of one or more of the 17 muscles that control the scapula, leading to stress-related injury [4, 7, 10].

Clavicle

Reports of clavicular stress fractures have involved athletic activities such as rowing, diving, javelin, weightlifting, gymnastics, and baseball [4, 6, 23]. Abnormal bending, shear, and rotational forces can develop across the clavicle if there is any imbalance in muscular contraction between the

pectoralis major, deltoid, and sternocleidomastoid muscles [4]. Repetitive bone strain by these forces may exceed the reparative capacity of the bone and lead to a stress fracture. Seyahi et al. [23] described a patient with a clavicular stress fracture presenting as atypical severe arm pain radiating throughout the upper extremity and hemithorax. In the case of clavicular stress fractures, activity modification until pain is resolved, postural training, and scapulothoracic stabilization exercises have yielded symptom resolution [4, 23].

Proximal Humerus

Stress fractures of the proximal humerus have been described most commonly in throwing and overhead athletes and weight lifters [27–30]. In throwers and overhead athletes such as tennis players, poor conditioning and fatigue of the shoulder girdle musculature allows for increased rotational strain at the cortical surface predisposing to stress fracture. Bending forces generated by opposition of the deltoid and pectoralis major muscles is the suspected mechanism for the transversely oriented stress fractures in weight lifters [31]. Athletes either present with increasing arm pain of an insidious onset or acute on chronic pain or a "pop" following antecedent activity related pain of the shoulder or arm [4].

If incomplete or non-displaced, proximal humeral stress fractures may be treated nonoperatively in a sling or fracture brace until the athlete is pain-free with activities of daily living or radiographic healing is evident. Treatment for incomplete or non-displaced proximal humeral stress fractures should also include rest and cessation of the offending activity. However, 12 months may be required for the patient to become asymptomatic [10]. If there is displacement, open reduction and internal fixation may be necessary to ensure timely healing.

Little League Shoulder

Little League shoulder is epiphysiolysis of the proximal humerus secondary to repetitive microtrauma from overhead activity [32]. The proximal humeral physis fuses approximately between the ages of 14 and 17 years in females and between 16 and 18 years in males [33, 34]. Factors that contribute to the development of Little League shoulder include excessive throwing, poor technique, and muscular imbalance. Athletes typically describe diffuse shoulder pain that is worse with throwing often following an increase in the throwing frequency or intensity [35–37]. On physical examination patients demonstrate weakness with resisted abduction and internal rotation along with tenderness and swelling over the anterolateral shoulder.

Anteroposterior X-rays may reveal widening of the proximal humeral physis (Fig. 14.7a).

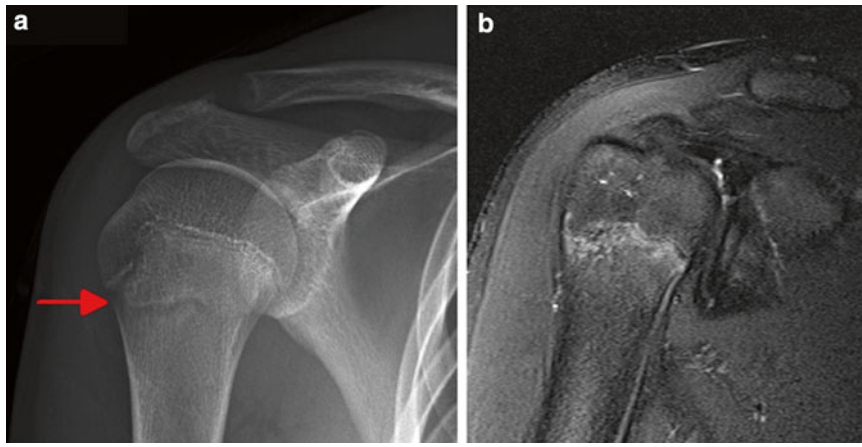

Fig. 14.7 AP radiograph (**a**) showing lateral widening at the proximal humeral physis and coronal T2 MRI (**b**) images demonstrating periphyseal stress fracture of the proximal humerus of the dominant right shoulder in a 14-year-old male baseball pitcher

Plain radiographs may also display fragmentation or demineralization of the metaphysis and periosteal reaction.[35–37] MRI of the shoulder may be required if the diagnosis is unclear (Fig. 14.7b). Treatment requires rest from throwing for 6–12 weeks followed by a progressive throwing program with alterations of throwing techniques and biomechanics as needed. The return to throwing progression begins with light tossing and gradual progression with increasing distance and velocity [35]. Potential complications of this injury include premature physeal closure with resultant humeral length discrepancy or angular deformity although with the great remodeling potential of the proximal humerus, these complications are rare. Proper throwing mechanics and close monitoring of the number of pitches thrown by a skeletally immature athlete are crucial for recovery from little league shoulder and prevention of further injury.

Diagnostic Imaging

X-Ray

Plain radiographs are usually negative early in the course of rib and upper extremity stress fractures. Although two-thirds of initial X-rays are negative, one-half will be positive once healing has begun 3 or more weeks after symptom onset (see Figs. 14.3, 14.6, 14.7a) [38]. Even after healing has taken place, radiographic findings such as cortical thickening and bone edema can be subtle and easily overlooked if the images are not thoroughly scrutinized [38, 39]. Depending on the severity and chronicity of the injury, radiographs may be inconclusive and require bone scan or MRI for definitive diagnosis. In the case of a proximal humeral periphyseal stress fractures (Little Leaguer's Shoulder), X-rays may initially be mistakenly read as a normal incompletely closed physis in a skeletally immature patient. MRI or bone scan is often necessary to make a diagnosis, with MRI being the authors' preferred modality due to the superior specificity (>85 %).

CT

Computed tomography (CT) is useful when the diagnosis of a stress fracture is indeterminate based on plain X-rays. CT scanning is useful for defining bony union and demonstrating evidence of healing by clearly showing the periosteal reaction and callus formation (see Figs. 14.1b and 14.2b). This imaging modality can also delineate a complete fracture from an incomplete fracture. CT is not as commonly used as MRI, however, due to the increased amount of radiation exposure and poor ability to evaluate surrounding soft tissue structures. CT scanning is useful for demonstrating evidence of healing by clearly showing the periosteal reaction and the absence of a discrete lucency or sclerotic fracture line [10].

Bone Scan

Bone scintigraphy has been shown to be 100 % sensitive for stress injuries of bone [39]. The greatest value of bone scintigraphy is that it allows early diagnosis of stress injuries and diagnosing bony stress injuries at multiple sites simultaneously (see Figs. 14.1a and 14.2a). This is often the case with rib stress fractures. Bone scans will often demonstrate increased uptake and a focused area of increased osteoblastic activity in the affected bone 1–2 weeks before radiographic changes occur [39]. Uptake on bone scan requires 12–18 months to normalize, often lagging behind the resolution of clinical symptoms [39]. Thus, bone scans are less helpful for guiding return to activity and/or sports participation. In the case of first rib injuries, bone scintigraphy has demonstrated 100 % sensitivity for early detection and diagnosis, but with a lower specificity than MRI [40].

MRI

Magnetic resonance imaging (MRI) is the most sensitive and specific imaging study available to evaluate stress injuries of bone [40, 41].

This imaging modality has demonstrated superior sensitivity and specificity over bone scan and CT for associated soft tissue abnormalities and edema and may delineate injury earlier than bone scan [41]. MRI has been used more frequently recently as the primary diagnostic tool for stress fractures. Its sensitivity is similar to that of a bone scan; however, it is much more precise in delineating the anatomic location and extent of injury [41].

Typical MRI findings on T2 sequences include a band of low signal corresponding to the fracture line, surrounded by diffuse high-signal intensity representing marrow edema (see Figs. 14.4, 14.5, 14.7b). Though expensive, it has the additional benefit of identifying soft tissue injuries. In summary, MRI is highly useful clinically for the diagnosis of many stress fractures, especially if used by musculoskeletal radiologists familiar with specific imaging protocols [42].

Classification

Stress fractures occur along a spectrum of severity. Not only does the severity of these injuries vary, but the clinical behavior of these injuries varies by location and causative activity. In addition to risk stratification of stress fractures which is based largely upon anatomic site, the "grade" or amount of cortical failure at a specific site is also used to describe the injury, determine prognosis, and develop the appropriate treatment plans [43]. The continuum throughout which stress fractures occur in the ribs and shoulder ranges from simple bone marrow edema (stress reaction) to a small unicortical disruption to a complete fracture with or without displacement and possible nonunion. The management of bony stress injuries should be based on the location, grade of the injury, and healing potential of the injury site. A combined clinical and radiographic classification system developed by Kaeding and Miller is shown in Table 14.2 [44]. This system has shown high inter- and intraobserver reliability among sports medicine and orthopedic clinicians [44]. A more indepth discussion of stress fracture classification and grading is presented in a different chapter of this textbook.

Table 14.2 Kaeding–Miller stress fracture classification system [44]

Grade	Pain	Radiographic findings (CT, MRI, bone scan, or X-ray)
I	–	Imaging evidence of stress FX *No* fracture line
II	+	Imaging evidence of stress FX *No* fracture line
III	+	Non-displaced fracture line
IV	+	Displaced fracture (>2 mm)
V	+	Nonunion

A combined clinical and radiographic classification system for stress fractures that has shown high intra- and inter-observer reliability

General Treatment Principles

The treatment for stress fractures of the ribs and shoulder girdle should be individualized to the patient's functional needs, symptom severity, causative activity, anatomic site, nutritional status, and fracture grade. Rehabilitation and training programs focused on proper mechanics and technique should be included in the treatment protocol after the fracture has been given sufficient time to heal [16, 18, 26]. If the fracture does not heal or symptoms persist beyond 4–6 weeks, the options for treatment are immobilization and restrictive bracing or potentially surgical fixation depending on site and injury severity [10]. Athletes with stress fractures at low-risk sites (those with adequate blood supply and low shear and tensile forces) and who are without functional limitations may continue their activities as tolerated using symptoms as a guide.

The decision to continue but decrease the causative activity in the presence of a stress fracture must be made in conjunction with the athlete only after thorough understanding of possible progression is conveyed. The activity may be continued if the athlete's pain level tolerates [10], however, close follow-up of these patients is necessary to ensure compliance with activity restrictions and prevent fracture progression to a higher-grade injury. This approach is acceptable if the risk and consequence of fracture completion are acceptable to the patient due to the importance of continuing their activity.

Unless contraindicated, patients may be permitted to cross-train during this time to maintain fitness and supplement training as the fracture heals.

Low-grade stress injuries or those without a clear fracture line at a low-risk site have a shorter time to recovery than a higher-grade injury at the same low-risk site [10]. The differences in treatment options between these two levels of severity of injury are duration of treatment, degree of activity modification, and need for immobilization (usually in a sling). The goal of treatment is symptomatic relief and to decrease the repetitive stress at the fracture site, thereby restoring the dynamic balance between damage and repair [9, 10]. This potentially involves a decrease in the volume of the offending activity, equipment changes, technique alterations, or cross-training. If pain persists or intensifies despite activity modification alone, treatment must be advanced to include complete rest, immobilization, or possibly surgical stabilization [10].

Return to Sports Participation

Return to sport decision making following a stress fracture is multifactorial. Despite advances in the imaging and understanding of stress fracture behavior, the decision to return to activity continues to challenge sports medicine practitioners. Critical to any return-to-play consideration is a thorough understanding by all parties (e.g., the physician, the athlete, the coaches, etc.) of the risk of possible injury progression. All patients, particularly those with stress fractures at sites with poor healing potential, must understand the risk of noncompliance with the treatment plan. A treatment plan should be tailored to athletic and personal goals, and the risks and benefits of continued participation thoroughly discussed.

As with most low-risk stress fractures, the point in the competitive season at which a rib or shoulder girdle stress fracture is diagnosed is often a major consideration for return to play. Athletes near the end of a competitive season or in the "off-season" may desire to be healed from their injury prior to returning training or competition. For these individuals, the treatment plan should include strict rest and activity modification to a pain-free level. In contrast, athletes at mid-season with low-risk stress fractures may desire to finish the season and pursue treatment for a cure at a later time. A gradual increase in activity can begin once the athlete is pain-free with activities of daily living and when the site is nontender [10].

Prevention of Rib and Shoulder Girdle Stress Fractures

Prevention of rib and shoulder girdle stress fractures is the preferred method of treatment. At the pre-participation physical examination an evaluation of risk should be made. This is especially important for individuals with a history of previous stress fractures. A history of prior stress fracture should alert the clinician to review that individual's risk factors. In females, correction of menstrual irregularities and poor nutritional status are critical. Team physicians involved with female athletes must also be vigilant for signs of the classic female triad of osteopenia, disordered eating, and amenorrhea. Calcium and vitamin D supplementation is often recommended in addition to general nutritional optimization. If biomechanical abnormalities are encountered, video analysis with appropriate muscular strengthening, proper equipment, and technique alterations is indicated for prevention of future injuries.

Summary

Stress fractures of the ribs, thorax, and shoulder girdle can be a source of pain and missed time from training and competition for athletes participating in a variety of sports. Stress fractures, along with bony tumors, insufficiency fractures, and neuralgias should be included in the differential diagnosis of patients with pain of the ribs and upper extremities who perform repetitive tasks. They are common injuries in rowing and throwing athletes and in individuals performing repetitive activities through their upper extremity. The diagnosis may be made if a high index

of suspicion is maintained and proper imaging studies are obtained. Treatment of these injuries, whether surgical or nonsurgical, should be individualized to the athlete's goals, sport, biomechanics, physiology, nutritional status, and injury site and severity. Factors influencing treatment decisions include: location (low vs. high risk), fracture grade, activity level, the timing of the competitive season, and the athlete's risk tolerance.

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Upper Extremity Stress Fractures 15

Jared A. Crasto, Sameer Jain, and Grant L. Jones

Introduction

Upper extremity stress fractures are much less common than lower extremity stress fractures but do occur nonetheless. These fractures are frequently the result of fatigue or overuse of the surrounding musculature. The major motions found to contribute to stress fractures in the upper extremity include weight lifting, weight bearing, throwing, and swinging [1]. Proper mechanics, training, and conditioning can aid in reducing the risk of these fractures. The early recognition and treatment of these fractures is important in treating and getting athletes back to competition quickly. A high clinical index of suspicion should be maintained in athletes complaining of activity-related pain. The majority of these fractures are able to be treated conservatively with a period of rest, with only a small fraction requiring operative intervention. This chapter seeks to describe the mechanism, presentation, diagnosis, treatment, and prognosis of stress fractures in the upper extremity that commonly affect athletes. It will begin with the humeral shaft and continue to the phalanges of the hand.

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Humerus

Two major patterns of stress fracture exist in the humerus and will be discussed separately below. These include spiral stress fractures and transverse stress fractures.

Spiral Stress Fractures

Mechanism: Throwing Athletes

Spiral stress fractures of the humerus have been reported in throwing athletes such as baseball pitchers [2–6], a cricketer [7], and a softball player [7]. In order to understand the mechanism of stress injury during throwing sports, a brief discussion of the mechanics of the throw is warranted. During the action of throwing a ball, the arm is abducted, extended, and externally rotated with the hand thrown as far back as possible.

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When the limit of this action is reached, the arm is then forcefully and rapidly flexed, partially adducted, and internally rotated; while the elbow is flexed, forearm partially pronated, and wrist flexed—finishing with the release of the ball, guided by the index and third fingers. When poor mechanics are employed, this motion is modified and places excess stress on the bone. Many throwers with desire to gain maximum power compromise form by extending the arm too far back, flexing the elbow too quickly, and snapping the action short. In this mechanism, the adductors and external rotators do not have time to relax before the rapidly contracting flexors and internal rotators—aided by the leverage of the flexed forearm traveling through the arc of internal rotation at the lower end of the shaft of the humerus against the upper end held in external rotation—twist the lower portion of the shaft of the humerus and cause a great deal of torsional stress [4]. Contributions from placing the elbow in extreme valgus position during this motion also add to the torsional stress on the humerus [8].

The same poor mechanics have been found in those throwers who extend the arm too far from the side of the body, using a "round arm" approach, where again internal rotation begins before external rotation is complete [9]. The bone becomes weakened through each of the aforementioned stresses, enough that a spiral fracture occurs with the violent phase of release and deceleration during the throwing motion [4, 10, 11]. Of note, the compressive forces of the biceps and triceps muscles on the humeral shaft are protective against the rotational torque created during the throwing motion, and fatigue of these muscles in the throwing athlete can also lead to increased risk of stress injury to the humerus [5, 12].

These fractures also have a propensity for affecting the younger population, with a large number of cases being reported in little league pitchers [2, 6]. These fractures are common in baseball pitchers with the injury having been described in a division I collegiate pitcher [5], recreational adults [4], and in members of a men's >30-year-old baseball league [3]. Authors have identified four common factors associated with these fractures in the older population: age over 30 years, prolonged period of layoff from throwing sport, lack of a regular exercise program during layoff period, and prodromal throwing arm pain [3].

Mechanism: Racquet Athletes

The mechanism of stress injury in racquet athletes is similar to throwing athletes, as the arc of motion during a service is similar to that of an overhand throw. The acceleration phase of the service motion begins when the arm moves into internal rotation, with a concomitant valgus stress at the elbow, which generates medial and posterior stresses contributing to an overall torsional stress on the humerus [13]. Torsional stress is also encountered during forehand shots, as the eccentric stretch and pre-tensing of the anterior shoulder musculature—particularly the internal rotators—is maximized by vigorous leg drive that positions the racquet down, behind, and away from the lower back in preparation for the drive to the ball [14]. This is a very important mechanism of power generation in racquet sports, and if the leg drive is ineffective, an increased stress is placed on the upper limb to maintain the same power. Spiral stress fractures in the humerus of racquet athletes share the same propensity for the adolescent population [14–16]. It is hypothesized that this is due to the contributions of a high degree of stress placed on immature bone, aggravated by growth spurts and inadequate muscular development [16].

Presentation

The most common presentation of a spiral stress fracture involves the insidious onset of increasing arm pain, worsened by aggravating motion (i.e., throwing or serving). Pain is poorly localized to the region of the middle and distal portion of the humerus, and follows a progressive course. The athlete usually notes the pain only after the aggravating motion initially, then progresses to feel the pain during the aggravating motion, then can progress to pain at rest. The duration of prodromal pain can last anywhere from 1 week [15] to 1 year [14]. The pain classically responds well to periods of relative rest from the aggravating activity, with flares of

pain on return, and progressive worsening that correlates with continuation and/or intensification of activity. In some cases athletes—during the aggravating action—encounter a loud "snap" or "pop" with accompanying extreme pain in the mid-humerus and loss of control over the action [2–4, 6, 7].

Diagnosis

Physical examination reveals point tenderness, pain, swelling, and deformity in the mid-to-distal humerus over the area of the stress fracture. While some cases showed no pain during full active and passive range of motion of the shoulder and elbow and uncompromised strength [5, 16, 17], most have pain with range of motion or with strength testing. Plain radiographs are the first study used in all cases, and can sometimes reveal some of the fractures, especially if complete or displaced. In cases where plain radiographs are equivocal, further testing is needed. In such cases, bone scan and MRI can be used. Bone scan will show an abnormal increased uptake in the mid-to-distal shaft of the humerus in the region of the origin of the brachialis muscle. While MRI will typically show a linear zone of diminished signal intensity within the distal humeral diaphysis on T1-weighted images, and increased signal in this zone on T2-weighted images. Medullary edema can be seen if MRI is used earlier in the disease process (Fig. 15.1a–c).

Treatment

The treatment for spiral stress fracture of the humerus is primarily nonsurgical, with very few reports of open reduction and internal fixation performed. There have been two cases reported of ORIF, one due to displacement of the fracture [7] and the other due to failure to maintain closed reduction [4]. Conservative treatment consists of a minimum 4 weeks' absence from aggravating activity with [2, 4, 6] or without immobilization [5, 14–16], and gradual resumption of activity over the next 4 weeks [18]. If immobilization is chosen, this can be achieved with a cuff and collar for 1 week with subsequent transition into a humeral fracture brace for 3 weeks [6, 19].

Prognosis

Patients treated for spiral stress fracture of the humerus have returned to play in as little as 3 weeks, but more typically take 8–12 weeks [5, 6, 14]. Even after delayed union, athletes are eventually able to return to sport though it can take up to 9 months [16]. Prevention of recurrence is important, and mitigating poor mechanics and enhanced strength training are both important factors. For throwing athletes returning from stress fractures, a good throwing program should be initiated beginning 3 months before the start of preseason training. For tennis players, athletes should work closely with coaches and analyze their mechanics diligently. They should specifically look at knee flexion and rotation of the

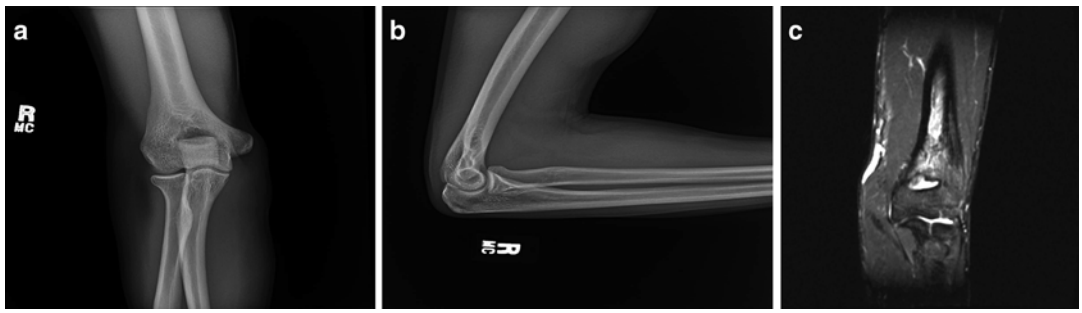

Fig. 15.1 A 19-year-old collegiate volleyball player with right elbow pain for 5 months. He attempted two courses of 4 weeks of rest with return of symptoms before diagnosis of distal humeral stress fracture. (**a**) Unremarkable anteroposterior (AP) radiograph of the right elbow. (**b**) Unremarkable lateral radiograph of the right elbow. (**c**) Coronal STIR image demonstrating increased edema within the distal humeral diaphysis indicative a stress reaction

trunk and upper limbs during the preparation phase of service and forehand strokes. The most common finding is poor leg drive motion during the preparation phase of each stroke [14]. Recognition of this flaw and adequate coaching to change the mechanics can lead to a presumed mitigation of the risk for recurrence by lessening the stress on the upper body while still maintaining adequate power [13, 14].

Transverse Stress Fractures

Mechanism

Transverse stress fractures of the humerus occur in the proximal and middle portions of the humerus and are rare, with very few case reports existing in the literature. Transverse stress fractures of the humerus are common in weight lifting with the injury being reported in a body builder [20] and in a competitive weight lifter [21]. Flat bench press seems to exacerbate the symptoms with the most pain being felt during this exercise. During the bench press exercise, the humerus effectively proceeds through an arc of shoulder extension, followed by shoulder flexion—with virtually no rotation of the humerus. Thus the rotational actions of the pectoralis major muscle must be countered by the other muscles of the shoulder girdle, including the deltoid and rotator cuff muscles [22]. The muscular tissues are able to dynamically redistribute the forces across the supporting skeleton, allowing the bone to effectively endure more load or stress, a phenomenon known as stress shielding [21]. A transverse stress fracture can occur with increased bending load across the bone, which is often the sequela of muscle fatigue that leads to the compromise of stress shielding [21, 22].

Presentation

In the reported cases, presentation involved significant pain localized to the proximal, anterior aspect of the affected arm, occurring during bench press and persisting for hours after the exercise. These symptoms were present for at least 4–6 weeks prior to diagnosis [20, 22]. Each patient tried a period of rest from bench press and other aggravating activities such as incline press, pectoral fly, biceps, and overhead exercises [20, 21]. Inevitably after the period of rest in which the pain subsided, ranging 1–6 weeks, a flare of the same pain occurred on their return to flat bench or other previously mentioned activities. One weight lifter progressed to a complete fracture and experienced an audible snap, with his arm giving way, on the second set of his normal weight lifting routine [22]. All of the patients denied the use of anabolic steroids.

Diagnosis

In most cases, the physical examination revealed tenderness to palpation in the region between the bony insertions of pectoralis major and anterior deltoid [20, 21]. There can be mild pain on manual resistance of shoulder movements, especially internal rotation and abduction [20]. Strength and range of motion of the shoulder and elbow usually does not show any deficits. Plain radiographs can show a transversely oriented radiolucency with surrounding periosteal reaction and sclerosis. Bone scans can confirm the diagnosis of stress fracture by showing abnormal focal increased uptake in the cortex of the affected mid-humerus.

Treatment

A period of 6–8 weeks free from all weight lifting activities involving the upper extremities, followed by gradual resumption of training is usually the first course of treatment for a transverse humeral stress fracture [20, 21]. If displacement or complete fracture has occurred, ORIF can be performed. Usually no bracing or immobilization is necessary.

Prognosis

These fractures typically respond quite favorably to a period of rest and a return to previous levels of competition can be expected once the fracture is completely healed, usually by 4 months. Progressing to full activities too quickly can lead to minor relapses in symptoms [20, 21]. If surgery is performed, return to sport can be unpredictable.

Olecranon

Four types of olecranon stress fractures have been reported in the literature. Two types tend to occur in skeletally mature athletes, and include fractures of the olecranon tip and oblique fractures through the midportion of the olecranon [23, 24]. The other two types tend to occur in skeletally immature athletes, and include transverse fractures, and osteochondroses [25, 26].

Presentation

These fractures present with dull aching and localized tenderness around the olecranon, worsened with activity, usually without recall of an acute injury [27, 28]. Symptoms evolve over a variable time frame which can be anywhere from a few weeks up to a year. These injuries are common in throwing athletes who will describe posteromedial elbow pain during the acceleration and follow-through phases of throwing [23, 24]. Very few athletes report pain at rest or while performing daily activities [29]. Clinically, tip fractures are more likely to present with a painful elbow after a particularly strong throw, whereas other stress fractures in the olecranon are more likely to present with a longer history of pain that lessens with rest and recurs when they resume throwing [28].

Diagnosis

Physical examination often reveals decreased arc of motion at the elbow, with flexion contractures that are common in the throwing population often present [30–33]. There is marked tenderness to palpation along the posteromedial aspect of the olecranon. In certain cases a milking maneuver may be positive, which could indicate a concomitant MCL injury [30]. The diagnostic modalities for each are similar, with plain radiographs often missing the diagnosis of stress fracture early in the process. Thus for the most accurate diagnosis to be made, bone scan or MRI must be considered [24, 27, 29]. The mechanisms, specific imaging findings, treatment, and prognosis will be discussed separately for each stress fracture in the remainder of this section.

Proximal-Third (Tip) Fractures

Mechanism

Olecranon tip fractures involve the proximal third of the olecranon and are classically described in javelin throwers [24], but have also been described in baseball pitchers [28, 30, 34] and gymnasts [26, 27, 35]. There is a component of repetitive abutment of the olecranon into the olecranon fossa causing osseous hypertrophy that is combined with traction from triceps activity during the deceleration phase of throwing [23, 24, 32, 33]. Olecranon tip fractures can occur more acutely than other olecranon stress fractures and as such are often diagnosed in the acute phase of injury [28].

Diagnosis

Plain radiographs can routinely confirm the diagnosis of an olecranon tip fracture [28]. Plain radiographs may show either the fracture itself, or the conditions that predispose to it, those being an increase in cortical thickness of the humerus (which produces a corresponding decrease in the available space within the olecranon fossa) and cubitus valgus (which exacerbates the potential impingement between the olecranon and the narrowed fossa) [33]. If plain radiographs are negative, they should be followed by a bone scan or MRI to make the most accurate diagnosis [24, 29].

Treatment

Fractures of the olecranon tip are prone to delayed unions, nonunions, or the formation of loose bodies. As such these fractures are frequently treated by surgical methods, including open reduction and internal fixation or tip excision [23, 34], though satisfactory results have been attained with rest and immobilization as well [23, 28]. Excision of loose bodies can lead to faster return to play for athletes [34].

Prognosis

Operative intervention can often lead to a faster return to play. Hulkko et al. compared return to play after both conservative and operative treatment. Both athletes returned to play with optimal healing; however the conservatively treated athlete returned after 18 months, and the operatively treated athlete returned after 2 months [23]. Six months is a reasonable expectation for return to play with conservative treatment. Earlier surgical intervention offers the possibility of earlier return to play, as well as a decreased risk of delayed unions, nonunions, and formation of loose bodies [35].

Middle-Third (Oblique) Fractures

Mechanism

Oblique stress fractures of the olecranon usually result from impaction of the medial olecranon on the medial wall of the olecranon fossa during valgus extension forces, which happens commonly during the acceleration phase of throwing [24, 33]. At the same time, the lateral aspect of the coronoid impinges against the intercondylar notch [30]. These two points of impingement provide the necessary stress to produce an oblique stress fracture. Oblique fractures of the olecranon have been classically described in baseball pitchers [24, 29, 30, 34] and javelin throwers [23].

Diagnosis

Physical examination reveals tenderness to palpation over the posteromedial elbow, and pain is reproduced on valgus stress testing, or forced hyperextension of the elbow. Extension lag in the affected extremity is also common [24, 30]. Plain radiographs are often negative or show subtle findings of periosteal reaction over the medial olecranon. In the setting of an acute stress reaction, MRI shows poorly defined, patchy areas of low signal intensity in the proximal posteromedial olecranon on T1-weighted images and areas of high signal intensity in the posteromedial olecranon on T2-weighted images, consistent with bone edema and hyperemia. In the setting of a more discrete, incomplete stress fracture, MRI shows more focal linear areas of intermediate signal throughout the cortex and subjacent cancellous bone of the articular surface [29] (Fig. 15.2).

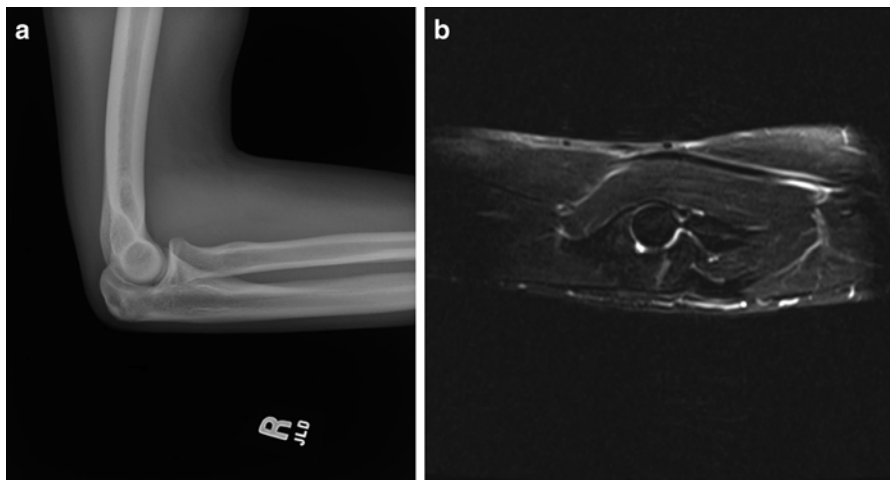

Fig. 15.2 A 15-year-old baseball pitcher with 2-month history of elbow pain. (**a**) Lateral radiograph of the right elbow demonstrating linear bone sclerosis at the olecranon. (**b**) T2 sagittal MRI image demonstrating transverse stress fracture of the olecranon

Treatment

Treatment involves a period of rest and avoidance of valgus stress (especially throwing) for at least 6 weeks, with some authors recommending restriction from sports for up to 6 months [24]. Some authors also advocate for an orthosis to limit full extension in the initial 4 weeks, as well as an interval-throwing program at 8 weeks before allowing return to play at about 12–14 weeks [29]. Though most respond to conservative management, occasionally operative management is required if there is no clinical or radiographic improvement [29, 30]. The recommendation for operative treatment of the oblique stress fracture pattern of the olecranon is using a tension-band type construct though other constructs have been shown to be effective [44].

Prognosis

Conservative treatment usually offers satisfactory results with a short period of rest and partial immobilization followed by gradual increased range of motion and rehabilitation. Schickendantz et al. showed professional baseball players managed conservatively returned to play at an average of 12–14 weeks and remained active in their sport in follow-up ranging from 2 to 7 years [29]. There have been reports of delayed surgical intervention in patients who showed a lack of healing after a 6 week course of conservative therapy. These patients were treated with a single screw and iliac bone pegs and were able to return to full activities within 4–6 months [30].

In summary, conservative therapy is the first-line treatment option and usually allows athletes return to play by 3 months [29]. Surgical intervention is reserved for the setting of failed conservative management.

Physeal (Transverse) Fractures and Osteochondroses

Mechanism: Transverse Fractures

Transverse fractures occur in skeletally immature patients and are caused by an overload of extension forces at the physis which is the weakest area of the olecranon [24, 32]. This typically results from traction and shearing forces on the olecranon acting at two sites: the insertion of the triceps tendon into the olecranon and the olecranon physis itself [26].

Mechanism: Osteochondroses

When the olecranon epiphysis is not fully ossified, traction forces may cause disturbance of blood flow and result in localized areas of avascular necrosis with disturbed ossification and fragmentation—better known as traction apophysitis or olecranon osteochondritis [32]. When the epiphysis is more mature (but not yet fused), these same traction forces can produce a Salter Harris type I stress fracture through the growth plate [26, 36, 37].

Presentation and Diagnosis

These injuries have been reported in young gymnasts [25–27, 38], adolescent baseball pitchers [25, 39, 40], wrestlers [41], and competitive adolescent divers [42, 43]. The stress fracture usually manifests as either a traction apophysitis with a more gradual onset or an avulsion of the physis which presents more suddenly. Physical examination can reveal an extension lag at the elbow that may worsen with continuation of the offending activity [41]. Plain radiographs can be normal in the setting of early disease, and a high degree of suspicion should be maintained in the adolescent athlete. Comparison radiographs of the contralateral elbow can be helpful. Radiographs later in the process reveal a widened physis, often with fragmentation of the olecranon apophysis [25, 38, 41]. A confirmatory sign is the presence of an irregular band of ossification within the growth plate, which results from disordered mineralization [38]. In the setting of negative radiographs, the use of bone scan or MRI can confirm the diagnosis [24, 27] (Fig. 15.3).

Treatment

Once the diagnosis is made, nonoperative treatment consisting of rest and avoidance of aggravating activities should be initiated immediately. In patients not responding to this therapy or who wish to return to competitive activities more quickly, surgical intervention can be discussed [40]. If a

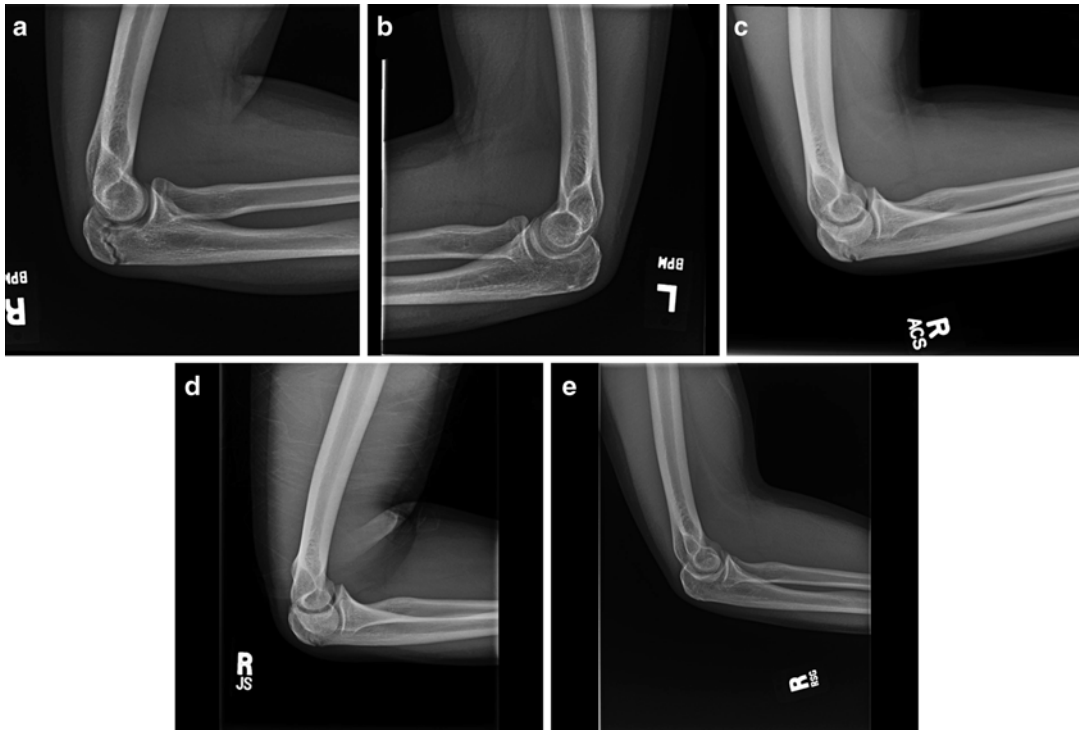

Fig. 15.3 A 14-year-old baseball pitcher with 2 months of right elbow pain. (**a**) Lateral radiograph of the right elbow showing widened, irregular olecranon apophysis. Notice the difference when compared to the contralateral elbow (**b**). A period of complete rest was initiated. (**b**) Lateral radiograph of the left elbow obtained at the same time as (**a**) showing fused olecranon physis. (**c**) Lateral radiograph of the right elbow obtained after a 2-month period of complete rest. Notice the progressive closure of the apophysis. (**d**) Lateral radiograph of the right elbow taken after 3 months of complete rest demonstrating continued closure of the olecranon apophysis. (**e**) Lateral radiograph of the right elbow at final follow-up 8 months after presentation showing complete closure of the apophysis. The athlete began a throwing program after a 3-month period of rest from initial presentation

nonunion develops, the treatment involves open reduction and internal fixation with bone grafting of the growth plate to promote fusion [36]. The options for fixation technique for the transverse stress fracture pattern of the olecranon include screw compression [23, 36, 39, 40] or tension band fixation with Kirschner wires [27, 40, 41]. The use of screw fixation is preferable, and has been shown to be more stable than tension band-only constructs [44]. Screw fixation also minimizes the potential need for hardware removal which can be common with tension band constructs [39, 45, 46].

Prognosis

Though conservative treatment of transverse fractures to the olecranon is recommended, it carries the inherent risk of nonunion [26, 27, 41]. Conservative management consists of rest with or without immobilization and usually returns the athlete to sport by 6 months. Surgical intervention is necessary in the setting of a nonunion, or can be considered if the athlete desires a speedier return to activity. Though no studies to date have confirmed a more rapid return to play with early surgical intervention, convention dictates it to be true [40].

Ulna

Mechanism

Stress fractures of the ulna have been reported in a variety of athletes, including tennis players [47–51], baseball and softball pitchers [52–55],

weight lifters [56–58], a body builder [59], riflemen [60], a volleyball player [54], a baton twirler [61], a cheerleader [62], an honor guard participant [63], a bowler [64], a golfer [65], a polo player [66], and a kendo player [67]. Though there are reports of stress fractures occurring proximally [52, 54, 64], distally [47, 50, 65], and even in the ulnar styloid [67], the middle one-third of the ulna has the smallest cross-sectional area as well as the most triangular (least circular) shape, making it the least resistant to torsional stress. The majority of cases described involve the middle one-third of the shaft [48, 49, 51, 53, 55–63, 66]. The torsional stress in the diaphysis of the ulna usually occurs between the origin of the flexor digitorum profundus and the outcropping muscles [49].

In throwing athletes, an ulnar shaft stress fracture may occur more proximally in the diaphysis as the mechanism is similar to that described for humeral stress fractures. During the acceleration phase there is internal humeral rotation and rapid extension of the elbow, which generates tremendous valgus forces about the medial aspect of the elbow joint and valgus extension forces posteriorly [4]. These stresses are transmitted primarily to the medial collateral ligament complex of the elbow, the flexor carpi ulnaris, and the medial flexor-pronator muscles, and thus load the proximal ulnar diaphysis [52, 54].

Presentation

Across each sport, presentation was very similar. Athletes presented with an insidious onset of pain in the ulnar shaft exacerbated by activity. The time before presentation ranged from weeks to months. In many cases, a recent increase in the length, intensity, or demands of training was noted [48–50, 54, 57, 58, 60–62, 66, 68]. Some athletes tried symptomatic treatment, which included anti-inflammatories, physiotherapy, and ice, none of which was significantly helpful. Rest from the offending activity tended to lessen the pain, with a classic flare on return to sport noted [51, 56–59, 62]. The location of the pain was reported anywhere from the proximal ulna into the elbow, down to the distal ulna into the wrist, and corresponded well with the location of the stress fracture.

Diagnosis

There is tenderness to palpation in the area of the stress fracture. Often resisted wrist flexion and extension or resisted pronation and supination of the forearm can reproduce the pain [48, 50, 54, 55, 57, 64, 66, 67]. Plain radiographs are often negative if performed within the first few weeks of injury. MRI or bone scan should be performed if radiographs are negative or equivocal.

Treatment and Prognosis

Ulnar shaft stress fractures should be managed non-operatively. Immobilization can be used for comfort, with immediate and total cessation from the offending activity. Often a 4–8 week period of rest from sport and/or immobilization is needed. Upon radiographic evidence of healing, or subsidence of symptoms, therapy can be begun if needed [47, 58, 65]. Most athletes are able to return to a pre-injury level of activity by 6–8 weeks after the initiation of conservative therapy.

Radius

Mechanism

Stress fractures of the radius in athletes have been described primarily in gymnasts [69–73], though case reports also exist in racquet sport athletes [74, 75], a basketball player [76], a pool player [77], an adolescent cyclist performing "wheelies" [78], and a field gun runner [79]. The epiphysis of the radius is the least resistant portion of the bone to shear and torsional forces [80], and this portion of the bone is particularly vulnerable when at its thickest, as seen during the growth spurt [81, 82]—placing adolescent athletes at higher risk. In all cases, repetitive torsional or axial loading forces on the radius

eventually lead to the development of a stress fracture. The vast majority of these fractures occur in the distal one-third of the radius.

Tumbling and vaulting in gymnastics tends to produce an impact angle of 60–90° to the wrist which places increased stress on the radius [71]. In twisting (Tsukahara) vaults, one wrist is removed from the ground first, leaving the remaining (fulcral) wrist to suffer much of the twisting. This explains why in many gymnasts with radius stress fractures the injury usually occurs in their fulcral wrist [71]. The use of very soft mats also exaggerates the amount of dorsiflexion of the wrist again placing increased stress on the radius [73]. In beam and tumbling workouts, the hands may be fixed in a particular position while the forearms are undergoing torsion in the direction of pronation and supination. These torsional forces may, with time and repetition, affect the epiphysis of the radius [73].

Stress fractures of the radius in a tennis [75] and badminton player [74] have similarly been reported in the adolescent population. In the case of the basketball player, the stress associated with "dunking" of a basketball repeatedly (with striking of the forearms on the metal rim of the basket) was believed to have contributed to the injury [76]. In the case of the pool player, putting excessive "English" (side spin) on the ball generated repetitive torsional forces to the radius that eventually lead to a stress fracture [77]. In the case of the adolescent cyclist performing "wheelies," the terminal motion of coming back down to the ground with a hard, jarring force to the forearm seemed to be the offending motion [78]. This patient's stress fracture was closer to the mid-distal one-third diaphyseal junction of the radius. Lastly, in the case of the field gun runner, repeated unaccustomed heavy loading of the 900 lbs. muzzle of the gun barrel to his forearms was believed to have incited the stress fracture [79].

Presentation

Regardless of the etiology of stress injury to the radius, presentation is similar. Patients present with an insidious onset of pain in the affected portion of the radius, usually the distal one-third or over the radial styloid process. Like most stress fractures, pain typically progresses over a course of a few weeks to a few months, is worse when performing the offending activity, and can usually be traced to a brisk increase in training demands. Pain is typically relieved with rest, whereas icing or use of nonsteroidal anti-inflammatories yields variable results. Pain can often be felt during active or passive forced dorsiflexion of the wrist, and can be worse immediately after exercises involving weight bearing on the upper extremities [70, 73].

Diagnosis

Physical examination will reveal tenderness to palpation over the affected area of the radius, with range of motion remaining largely uncompromised. A provocative maneuver such as active or passive forced dorsiflexion of the wrist will often reproduce the pain characteristic to the injury [70, 73–75]. Plain radiographs can be negative in the early stages of disease, but radial stress fractures are more likely to be visible on plain films versus other types of stress fractures [69, 74, 75]. One must look closely at the physis for characteristic stress response changes including widening of distal radial epiphysis, cystic changes of the epiphyseal plate, irregularity of the metaphyseal margin, and occasional haziness within the usually radiolucent area of the epiphyseal plate [70, 72, 73, 76–79]. Comparison radiographs of the contralateral extremity can be helpful. Most fractures are identified using plain radiographs so further imaging studies are rarely needed. If they are, bone scan or MRI can be used (Fig. 15.4).

Treatment

The mainstay of treatment in radial stress fractures is rest from the offending activity, with a short course (4–6 weeks) of immobilization for comfort in most cases [73, 74, 78]. Activities that do not cause the patient pain can be allowed to be

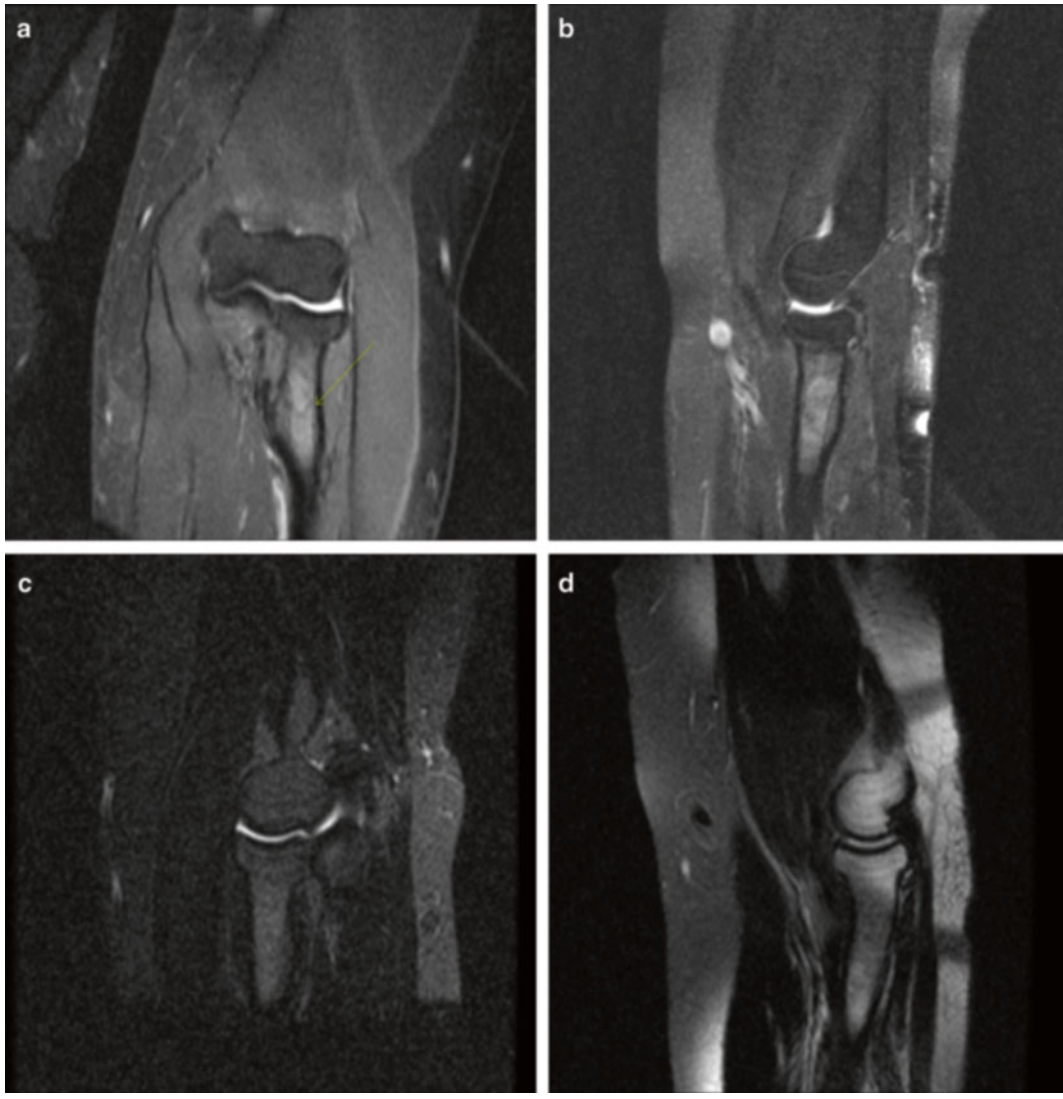

Fig. 15.4 A 40-year-old nurse who presented with a 3-month history of elbow pain with weight bearing in terminal extension. Plain radiographs were normal. (**a**) Coronal STIR image of the left elbow demonstrating proximal radius stress fracture. (**b**) Sagittal T2-weighted image demonstrating the same lesion. (**c**) Follow-up coronal STIR image demonstrating complete healing of the stress fracture after a 3-month period of rest. (**d**) Three-month follow-up sagittal T2-weighted image demonstrating the same healed lesion

continued depending how symptomatic they are. Gymnasts can often continue bar exercises to tolerance [71]. Nonsteroidal anti-inflammatory medications and physiotherapy can be used as necessary [70]. In order for the rest period to be lifted, the patient's wrist must be completely asymptomatic, the wrist should be non-tender to the touch, and most importantly there should be no pain during forced dorsiflexion of the wrist [73]. Taping of the wrist to prevent maximal/excessive dorsiflexion can often be helpful as the patient returns to activity [73].

Prognosis

At least a minimum of 8 weeks of rest are needed before a patient can return to sport, though it can take up to 3–6 months to make a full return. Patients who show radiographic evidence of radial stress fracture take longer to recover versus those in whom the diagnosis is made clinically [73]. All patients will make a full recovery but they need to be counseled that there is a propensity for re-injury, and proper precautions (taping, alteration of mechanics, etc.) need to be employed [71].

Scaphoid

Mechanism

Scaphoid stress fractures are rare and only case reports exist in the current literature. These included gymnasts [83–85], a shotputter [84], a cricketer [86], a diver [87], and a badminton player [74]. During the aforementioned activities, there is repetitive weight bearing or resistance that stresses the radiocarpal articulation, particularly when the wrist is dorsiflexed [87]. In gymnastics, the wrist is brought into acute dorsiflexion in handstands and other maneuvers. As the palm and metacarpal heads are progressively loaded with the body weight of the athlete, compressive forces are applied across the radioscaphoid articulation [85]. Since the proximal articular surface of the scaphoid is wedge shaped, forces drive the scaphoid palmarly, "like a watermelon seed is shot from the fingers" [88]. The volar capsule, radioscapholunate ligament and radiocapitate ligaments resist the tendency of the proximal scaphoid to subluxate toward the palm of the hand [85]. The distal scaphoid, however, is not as restricted, as the major support of the distal pole is the radiocollateral ligament complex, which becomes lax with slight radial deviation of the wrist. The scaphoid fails at the point receiving the greatest bending moment, at the level of the waist, as it extends beyond the floor of the taut palmar capsular structures [85, 88]. Though handstands and other maneuvers requiring acute dorsiflexion of the wrist exert considerable forces across the dorsiflexed wrist, they are still less than those required for an acute fracture. It is the cyclic application of these subthreshold forces that eventually leads to a stress fracture of the scaphoid at the waist [85].

Presentation

Patients present with an insidious-onset, worsening, and unilateral wrist pain without an antecedent trauma. The time from onset of symptoms to presentation can often be several weeks to months [86]. Pain is frequently located in the anatomical snuffbox or on the radial side of the wrist but can be poorly localized diffusely in the wrist [74]. Performing the sporting activity, where inevitably the wrist is dorsiflexed with or without weight bearing, usually exacerbates symptoms. Often times an athlete will self-abstain from the sporting activity for a short period of time, which will usually afford the patient a transient period of relief. Once the patient returns to the sport, a classic recurrence of pain can greet their return [84].

Diagnosis

Physical exam reveals tenderness to palpation in the snuff box or over the scaphoid tubercle in most cases. Range of motion is often decreased by 10–30° in both wrist flexion and wrist extension when compared to the unaffected wrist, and strength in the same planes can be decreased. Pronation, supination, and digital motion are unaffected. Pain is reproduced with radial deviation, wrist extension, and palpation over the scaphoid. There are reports of patients presenting with a positive Finkelstein test, although this is not common [87]. Plain radiographs are often negative [83, 85] but can reveal an increased bone density in the waist of the scaphoid depending on the chronicity of the injury [74, 84, 87]. MRI offers superior visualization, especially early on in the disease process in the setting of negative plain films with a high suspicion of stress fracture. MRI can show subtle fracture through the scaphoid waist that is typically accompanied with edema throughout the entire scaphoid. Bone scan can also be used though MRI is the preferred imaging modality.

Treatment and Prognosis

Unless a clear fracture line is seen, conservative treatment consisting of a short-arm thumb spica cast can be initiated. A 6-week course of cast immobilization is usually followed by transition to a removable orthosis for an additional 2–6 weeks. Removable orthosis may be used earlier in more trustworthy patients [74, 83–85]. Immobilization is discontinued when repeat imaging shows a healed fracture, and the patient is asymptomatic. Full return to activity as tolerated is allowed at this point.

Athletes requiring a faster return to sport can consider undergoing a percutaneous pinning of the scaphoid using cannulated scaphoid screw. Surgical risks are minimal and return to sport is significantly faster versus conservative treatment. Patients can be allowed to return to sport within 6 weeks if not sooner depending on symptoms. No specific studies looking at return to play after percutaneous fixation scaphoid stress fractures have been conducted, but these can be treated similarly to an acute, non-displaced, scaphoid fracture [89, 90]. All patients should be able to make a full return to sport regardless of which treatment option is chosen.

Metacarpals

Mechanism

Stress fractures of the metacarpals are rare, but have been reported to affect athletes involved in tennis [91–95], rowing [96], and softball [97]. Features common to many of these stress fractures include increased training load and changes in technique. A study that evaluated metacarpal stress fractures in seven teenaged tennis players [95] described a close association with increased training intensity, changes in stroke biomechanics, and Western-style racket grip with stress fractures occurring at the base of the second metacarpal. The Western grip uses the second metacarpal as a virtual arm on the handle of the racket, channeling all the tension on the base of the second metacarpal [93]. The second carpometacarpal joint is limited in the flexion and extension plane relative to the other metacarpals, which may result in more stress placed on its base and predispose it for stress injury.

In the case of the rower—who sustained a stress fracture to the fourth metacarpal bone—the authors noted the design of newer oars with larger blades requiring more musculoskeletal stress per rowing stroke [98], in combination with inexperienced rowers maintaining a firmer grip on the oar [96] as the mechanism of stress fracture for these athletes. In the case of the softball player—who sustained a stress fracture to the fifth metacarpal bone—the authors postulated that the fracture was caused by a new grip for a curveball pitch whereby abduction forces in conjunction with the muscle pull from the extensor carpi ulnaris was responsible for the fracture [97].

Presentation

Athletes will present with rapidly progressive pain in the dorsal aspect of the hand without antecedent injury or trauma. Pain is localized to the dorsal aspect of the affected metacarpal with a corresponding area of bony tenderness. The pain was maximal when performing the stressing action—serving and hitting forehands for the tennis players [91–95], when performing a rowing stroke for the rower [96], and when pitching a curveball for the softball pitcher [97]. A recent change in grip of the racket was noted in most of the tennis players [91–95], and an increase in training intensity seemed to be a common feature to all.

Diagnosis

On physical examination, there will be tenderness to palpation over the base of the affected metacarpal, without any compromise of range of motion anywhere in the hand or wrist. Initial plain radiographs can be negative with periosteal reaction as the only finding, though nondisplaced fracture line was visible in almost half of the reported cases. MRI or bone scan be used to confirm the diagnosis (Fig. 15.5).

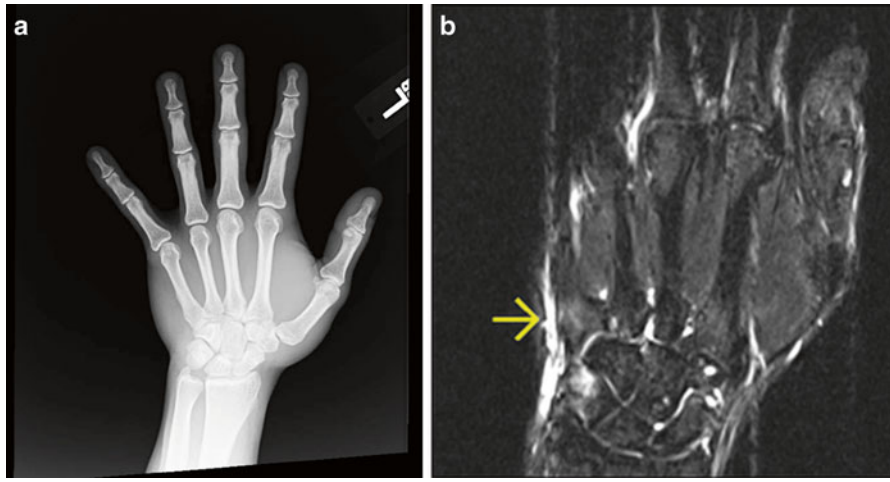

Fig. 15.5 A 28-year-old recreational softball player who complained of pain near the base of the fifth metacarpal and loss of throwing power. No prior history of trauma. (**a**) AP radiograph of the left hand demonstrating callus formation present at the base of the fifth metacarpal. (**b**) Coronal STIR MRI demonstrating stress fracture of the L fifth metacarpal base. *Yellow arrow* highlights the affected region. Patient's symptoms resolved after a short period of rest

Treatment and Prognosis

Athletes can be treated with a period of relative rest without immobilization. This can range from 4 to 8 weeks in duration depending on the patients symptoms. Short-term immobilization can be used for 1–2 weeks for comfort if needed. Surgery is rarely if ever needed for these fractures.

Patients can expect full return to pretreatment levels of activity and competition in their respective sports upon completion of the rest period. Athletes should be counseled on changing their grip and/or mechanics to help ensure prevention of symptoms from recurring.

Phalanges

Mechanism

There has been one reported case of phalangeal stress fractures, which occurred in an adolescent rock climber [99] and involved bilateral stress fractures of the middle phalanx of the middle fingers. The authors proposed mechanism of injury involves the fact that during prolonged gripping action required in rock climbing, the metacarpophalangeal joint remains extended while the interphalangeal joints are flexed to grip the rocks [100]. In a skeletally immature athlete, the joint capsule and ligaments of the finger are stronger than the physis, and thus the physis gives way during repetitive gripping episodes [99].

Presentation

The presentation of the reported case was described as a 1-week history of insidious-onset bilateral painful swelling of the proximal interphalangeal (PIP) joints of the middle fingers without history of other joint problems or direct trauma.

Diagnosis

On physical examination the climber exhibited swelling with tenderness to palpation over bilateral PIP joints with flexion decreased to 10° at the joint. Standard radiographs (anteroposterior and lateral) revealed bilateral Salter-Harris type III fractures of the base of the middle phalanx of the middle finger, one with minimal displacement and one non-displaced. No rotational deformity was present.

Treatment and Prognosis

Treatment was conservative and consisted of relative rest without immobilization. At 6-week follow-up the climber had improved to full range of motion but still had swelling of the joints. Radiographs showed reduction of both fractures. At 3-month follow-up the climber's symptoms remained and an MRI scan showed bilateral healing fractures. At 4-month follow-up one finger remained symptomatic, especially during rock climbing. The patient continued to be treated conservatively for the next 4 months with abstinence from rock climbing. At 12-month follow-up the patient was without symptoms, without residual deformity, and returned to full activity. Radiographs at 12-month follow-up showed healed fractures.

Conclusion

While rare, upper extremity stress fractures must be considered in the differential for any athlete complaining of activity-related pain. History and physical findings can be vague and plain radiographs are often negative. Bone scans or MRI can be used to help aid in the diagnosis of these injuries. The early recognition and treatment of these fractures is important in treating and getting athletes back to competition quickly. The majority of these fractures are able to be treated conservatively with a period of rest, with only a small fraction requiring operative intervention. Proper training and mechanics are crucial to helping prevent these injuries, as these fractures are frequently the result of fatigue or overuse of the surrounding musculature.

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Insufficiency Fractures

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Michael J. Tranovich and Vonda J. Wright

Stress fractures occur when bone is repetitively loaded over time without the presence of violent trauma. One type of stress fracture, the insufficiency fracture, is common in older individuals but much less common among younger athletes. In order to develop an understanding of insufficiency fractures it is important to distinguish insufficiency fractures from the pathological fracture and the more common fatigue fracture. Insufficiency fractures occur when normal or physiologic forces are applied on bone with abnormal elastic resistance. In contrast, fatigue fractures occur when repetitive forces are applied to bone of normal elastic resistance. Pathological fractures occur when bone is weakened by infection or tumor [1]. While insufficiency fractures are more commonly experienced in the elderly population, it is important to distinguish between causes of stress fractures in athletes because each type requires its own unique steps in diagnosis and management.

Causes of Insufficiency Fractures

Understanding the causes of insufficiency fracture is of great importance as the prevention and treatment of these fractures must involve correction or prevention of the underlying pathophysiology. Weakened bone leading to insufficiency fracture is commonly associated with osteopenia, osteoporosis, osteomalacia, Paget's disease of bone, or a history of treatment with radiation therapy.

Osteopenia and Osteoporosis

Insufficiency fractures are most commonly associated with bone of decreased density, also known as osteopenic bone. While aging athletes are at risk for osteopenia and consequently insufficiency fracture, it is possible for younger athletes to experience an insufficiency fracture as well.

Causes of osteopenia and osteoporosis can be primary or secondary. Primary causes of decreased bone density include age-related, juvenile, postmenopausal, and osteogenesis imperfecta while secondary causes include several endocrine, hematologic, hereditary, and nutritional disorders. Rheumatoid arthritis, sex hormone deficiency, steroid therapy, hyperparathyroidism, and renal osteodystrophy are a few of the secondary causes of osteopenia associated with insufficiency fracture [2]. Any individual that participates in athletics and possesses one or more of these risk factors for osteopenia also presents with an increased risk of developing an insufficiency fracture.

With the continuous advancement of joint preservation techniques, significantly more individuals are participating in sports well beyond

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40 years of age. With increasing age comes an unavoidable increasing amount of oxidative stress and accumulation of free radicals in bone. In addition to physiologic changes, genetics and dietary factors play a role in the development of aging bone. While sports participation may help in maintaining a healthy bone mass, exercise alone will not prevent the development of osteopenia and frank osteoporosis. Therefore, care must be taken to recognize all aging athletes at risk for developing osteopenia and take appropriate dietary and pharmacological steps to prevent osteopenia and decrease the risk of insufficiency fracture.

Diet also plays a role in bone health as protein, calcium, and vitamin D intake are associated with higher bone mineral densities and therefore protection from osteopenia [3]. In a study of female navy recruits it was shown that consumption of 2,000 mg of calcium and 800 IU of vitamin D per day was associated with a 20 % lower incidence of stress fracture [4].

Screening for osteoporosis plays a major role in preventing insufficiency fractures as early diagnosis and treatment can help those at risk to maintain a more normal bone density. In 2009 the American College of Preventative Medicine recommended that all adult patients ≥50 years of age be screened for risk factors for osteoporosis. It was recommended that all women ≥65 years of age and all men ≥70 years of age obtain dual energy X-ray absorptiometry (DEXA) testing to screen for osteoporosis [5]. Younger postmenopausal women and men ages 50–65 with one major or two minor risk factors for osteoporosis (Table 16.1) should also undergo DXA testing [5, 7].

In adults, DEXA is interpreted in terms of *T*-score. By the World Health Organization's reference values, a *T*-score refers to how many standard deviations a patients' bone density lies from the average bone density of a 20- to 29-year-old female. A *T*-score of ≤–2.5 at the femoral neck, total hip, or lumbar spine generally allows for the diagnosis of osteoporosis [8]. In contrast to the *T*-score, the *Z*-score is based on standard deviations from an age, gender, and race matched reference value. *Z*-scores are used for children and adolescents undergoing DXA testing [9].

Table 16.1 Risk factors for osteoporosis [6][a]

Major risk factors	Minor risk factors
Vertebral compression fracture	Rheumatoid arthritis
Fragility fracture after age 40	Past history of hyperthyroidism
Family history of osteoporotic fracture	Chronic anticonvulsant therapy
Systemic glucocorticoid therapy >3 months	Low dietary calcium intake
Malabsorption syndrome	Smoking
Primary hyperparathyroidism	Excessive alcohol intake
Propensity to fall	Excessive caffeine intake
Osteopenia apparent on X-ray film	Weight <57 kg
Hypogonadism	Weight loss >10 % of weight at age 25
Early menopause (before age 45)	Chronic heparin therapy

[a]Reprinted from American Journal of Preventative Medicine, 36/4, Lim LS, Hoeksema LJ, Sherin K, ACPM prevention practice committee, Screening for osteoporosis in the adult US population: ACPM position statement on preventative practice, 366–75, Copyright (2009), with permission from Elsevier

In premenopausal women, treatment for early osteopenia/osteoporosis includes investigation for underlying causes as well as ensuring adequate intake of calories, calcium (1,000 mg daily from diet), and vitamin D (600 IU D3 supplement) [10]. For premenopausal women, evidence is lacking for common pharmacologic treatments used in postmenopausal women such as bisphosphonates, selective estrogen receptor modulators, teriparatide, and denosumab. Smoking cessation [11], normalization of body weight, avoiding excessive dieting and weight swings [12], and limiting alcohol consumption [13] have been shown to be of benefit in preventing or treating decreased bone mineral density.

In postmenopausal women initial osteoporosis treatment begins with ensuring adequate vitamin D and calcium intake. Along with dietary calcium, supplemental calcium should be taken in doses of 500–1,000 mg/day so that total calcium intake equals around 1,200 mg/day. Intake of vitamin D should equal around 800 IU daily. Exercise and cessation of smoking are also important in this population. The National

Osteoporosis Foundation recommends pharmacologic intervention in postmenopausal women with a history of hip fracture or vertebral compression fracture or in those individuals with a *T*-score ≤–2.5 [14]. Bisphosphonates are considered the mainstay of pharmacologic intervention in postmenopausal osteoporosis [15]. Other effective agents include selective estrogen receptor modulators [16], parathyroid hormone [17], and denosumab [18, 19]. Evidence is lacking or contraindications exist for estrogen replacement, calcitonin, and combination therapy.

Treatment guidelines also exist for the treatment of osteoporosis in men. As with women, diet and lifestyle changes are important in the treatment of osteoporosis. Calcium intake should reach 1,000 mg/day in younger men and up to 1,200 mg/day in older men. Vitamin D supplementation should equal 600–800 IU per day. Continued exercise, smoking cessation, and limiting alcohol consumption are also important lifestyle changes in men. Testosterone therapy is recommended in younger hypogonadal men with no contraindications to testosterone therapy [20]. In addition to the above measures, pharmacologic intervention is recommended in men with a history of fracture or *T*-score of ≤–2.5. Pharmacologic intervention may also be necessary in men with a *T*-score of –1.0 to –2.5. As with women, bisphosphonate therapy is oftentimes the drug of choice. Studies have recommended weekly treatment with alendronate or risedronate [21]. Other alternatives include the IV bisphosphonate zoledronic acid, and secondline agents such as teriparatide [22] or denosumab [23].

The Female Athlete Triad and Decreased Bone Mineral Density

Young athletes particularly vulnerable to insufficiency facture are those with the female athlete triad of amenorrhea, eating disorder, and osteoporosis. This triad is specifically observed in physically active females and is now defined as involvement of any one or more of the following components: (1) low energy availability with or without disordered eating; (2) menstrual dysfunction; and (3) low bone mineral density [24]. Risk factors for the triad that should be assessed in female athletes are menstrual irregularities, criticism of eating habits by coach, family, or peers, depression, dieting, obsessive personality, pressure to lose weight, early sport-specific training, overtraining, recurrent injuries, history of fracture, low BMI, and physical examination signs of an eating disorder [24]. In relation to triad risk factors, a 2014 study found that more triad risk factors are associated with a greater odds of bone stress injury than one factor alone [25]. Specifically, the authors found an increase in bone stress injury from 15 to 21 % for one risk factor to 30 % for two risk factors to 50 % for three triad risk factors [25]. Another 2014 study also found that multiple risk factors exhibit a cumulative risk of lower bone mineral density in young women [26].

Low energy availability in at-risk athletes often leads to menstrual dysfunction and can lead to deleterious effects on the musculoskeletal system. In cases of hypoestrogenism, increased reabsorption of calcium and decreased bone storage of calcium leads to decreased bone mineral density [27]. In terms of menstrual irregularities and bone mineral density, a 2003 study found that female runners experiencing less than 10 menstrual cycles per year had bone mineral densities 3–6 % lower than those female runners having greater than 10 menstrual cycles per year [28]. In addition to the musculoskeletal system, the reproductive, cardiovascular, endocrine, gastrointestinal, renal and neurological systems can be affected by the female athlete triad [24].

Diagnosis of the female athlete triad is multifaceted and involves a multidisciplinary approach. Low energy availability can be indicated by a BMI <17.5 kg/m^2 or in adolescents <85 % of expected body weight. While a low BMI can be an indicator of low energy availability assessing energy availability is most often a much more complex measurement. In response to this issue the Female Athlete Triad Coalition provides an energy availability calculator on their website (http://www.femaleathletetriad.org/calculators/). They report that physically active women should aim for at least 45 kcal/kg fat-free mass/day of energy intake [24].

Table 16.2 Female athlete triad risk factors [24]

High risk	Moderate risk
• History of a DSM-V diagnosed eating disorder	• Current or history of disordered eating for ≥6 months
• BMI ≤17.5 kg/m^2, <85 % estimated weight, or recent weight loss of ≥10 % in 1 month	• BMI between 17.5 and 18.5, <90 % estimated weight, or recent weight loss of 5–10 % in 1 month
• Menarche at ≥16 years of age	• Menarche between 15 and 16 years of age
• Current or history of <6 menses over 12 months	• Current or history of 6–8 menses over 12 months
• Two prior stress fractures, 1 high-risk stress fracture, or a low energy nontraumatic fracture	• One prior stress reaction or stress fracture
• Prior *Z*-score of <–2.0 (after >1 year from baseline DXA)	• Prior *Z*-score between –1.0 and –2.0 (after >1 year from baseline DXA)

Assessing amenorrhea is a complex process that should be initialized by the primary care physician with appropriate consults to both gynecology and endocrinology specialists. Pregnancy and endocrine disorders such as thyroid dysfunction, hyperprolactinemia, primary ovarian insufficiency, hypothalamic and pituitary disorders, and hyperandrogenic conditions must be ruled out as the causes of the amenorrhea.

Because low bone mineral density is the direct contributing factor to an insufficiency fracture, criteria have been established for obtaining DEXA testing in young woman and girls. The Female Athlete Triad Coalition recommends DEXA testing in athletes with one or more high-risk factors or two or more moderate risk factors (Table 16.2). The Coalition also recommends DEXA testing in athletes with a history of two or more peripheral long bone traumatic fractures when 1 or more high or moderate triad risk factors are identified [24]. Results from DEXA scanning should be interpreted carefully and may need to be repeated every 1–2 years in individuals with ongoing indications for testing. The International Society for Clinical Densitometry (ISCD) provides guidelines for interpreting DEXA testing in children and adolescents. In their 2013 position statement the ISCD maintained that a vertebral compression fracture is indicative of osteoporosis in children and adolescents while densitometry alone is not adequate to diagnose osteoporosis. Total body less head and the posterior–anterior spine are the preferred skeletal areas when performing DEXA testing. In children and adolescents without vertebral compression fracture, osteoporosis is diagnosed with a significant fracture history and a *Z*-score of ≤–2.0 on densitometry. The ISCD also reports that a *Z*-score of >–2.0 does not necessarily preclude skeletal fragility [29, 30]. Young individuals with osteoporosis most often present with fracture before the diagnosis is confirmed; therefore early recognition and prevention of the female athlete triad is instrumental in avoiding insufficiency fractures.

Osteomalacia/Rickets

Osteomalacia, termed rickets in children, is defective bone mineralization most often caused by a chronic deficiency in vitamin D or phosphate. Consequently, individuals with osteomalacia have a softening of bones which predisposes to fracture. While it is extremely rare in athletes, it should be considered as an underlying cause of insufficiency fracture in athletes with generalized osteopenia. A 2013 study found that DEXA scanning may detect osteoporosis in up to 70 % of individuals with osteomalacia [31]. Patients with osteomalacia often present with generalized bone pain and osteopenia. Generally the best way to prevent osteomalacia induced insufficiency fracture is ensuring adequate vitamin D intake throughout life. In a 2013 position statement, The Society for Adolescent Health and Medicine recommended vitamin D supplementation of 600 IU daily in healthy adolescents and at least 1,000 IU for adolescents at risk for vitamin D insufficiency, in addition to dietary intake. Other guidelines for vitamin D supplementation and management for adolescents are presented in Table 16.3. Some conditions that are potential factors associated with vitamin D deficiency are

Table 16.3 Vitamin D guidelines as recommended by the Society of Adolescent Health and Medicine [32]

• Vitamin D supplementation of 600 IU daily in healthy adolescents
• Vitamin D supplementation of at least 1,000 IU daily for adolescents at risk for vitamin D insufficiency
• Serum 25(OH)D concentration in at-risk adolescents
• Serum 25(OH)D concentration of 30–50 ng/mL is optimal in adolescents
• In adolescents with <20 ng/mL 25(OH)D, supplement 50,000 IU of vitamin D once per week for 8 weeks
• In adolescents with 20–29 ng/mL 25(OH)D, supplement 1,000 IU per day for at least 3 months
• Use a vitamin D3 preparation if available
• Vitamin D supplementation should be taken with dinner if possible

25(OH)D 25-hydroxyvitamin D

increased skin pigmentation, frequent use of sunscreen, obesity, specific diets such as vegan, cultural body coverage requirements, chronic GI diseases, amenorrhea, pregnancy or lactation, immobilization, bariatric surgery, chronic kidney or liver disease, certain medications such as steroids, anticonvulsants, and HIV medications, and known low bone density status [32]. In older adults the International Osteoporosis Foundation (IOF) states that, on average, 800–1,000 IU of vitamin D are required per day to maintain a serum level of 25-hydroxyvitamin D (25(OH)D) of 30 ng/mL. The required intake also varies per individual as 800 IU per day may be sufficient in healthy individuals with regular sun exposure. On the other hand, obese individuals, those with low sun exposure, with osteoporosis, malabsorption, and in populations such as those of Middle Eastern or Southern Asian decent may need upward of 2,000 IU of vitamin D intake per day [33]. The IOF advises that 100 IU of vitamin D will increase the serum 25(OH)D by about 1.0 ng/mL.

Paget's Disease of Bone

Paget's disease of bone is generally a disease of older individuals and is characterized by abnormal bony remodeling and resultant disorganized bony architecture. The disorganized bone growth associated with Paget's disease of bone can initially lie clinically silent and may lead to bone pain, bone deformity, fracture, osteoarthritis, spinal stenosis, cranial nerve compression, tinnitus, deafness, and in a small number, osteosarcoma. Paget's disease of bone is thought to have genetic influences and potentially environmental triggers such as viral infection and low calcium and vitamin D intake. The most common bones affected by Paget's disease of bone are the pelvis, femur, lumbar spine, skull, and tibia [34, 35]. Patients with Paget's disease of bone often have normal calcium, phosphate, and PTH levels on laboratory testing. Variable but often elevated levels of alkaline phosphatase may be observed and depend on the stage of the disease [35]. Another potential presentation finding of Paget's disease of bone is an abnormal radiograph while investigating for other pathologies. Pseudofractures on the convex aspects of affected bones also should raise suspicion of Paget's disease of bone [35]. Patients may also report pain with use of the affected area, with rest, and at night [35]. Other factors useful in diagnosing Paget's disease of bone are localized pain in areas with continued uptake on bone scan and pain improvement with a bisphosphonate trial. Pain that originates in joints is less likely to be due to Paget's disease of bone. While Paget's disease of bone is an extremely rare cause of insufficiency fracture in younger individuals, it should be ruled out as a potential underlying cause of insufficiency fracture in aging athletes as treatment of Paget's disease involves medical and surgical management.

Radiation-Induced Fractures

While fracture associated with tumor is termed pathological fracture, insufficiency fracture can occur in previously irradiated bone in which a tumor has since resolved. Ionizing radiation is effective as a treatment means for cancer because it causes cell death through DNA strand breaks. This radiation disrupts the bone's blood supply and decreases the number of osteoblasts while increasing the activity and number of osteoclasts. This leads to bone marrow suppression and

abnormal bony remodeling, which in effect lowers bone mass and density predisposing affected individuals to insufficiency fracture. These effects are dose dependent and can remain permanent with higher radiation dosages. Therefore, athletes with a history of radiation therapy must be monitored for the development of insufficiency fractures [36].

Presentation of Insufficiency Fractures

Patients with insufficiency fractures often present with acute pain in a commonly affected area such as the back, groin, or foot. A history of trauma is usually lacking. Depending on the severity of the fracture, the patient may present in a non-ambulatory state. Physical examination of a suspected insufficiency fracture involves localization of pain and inspection of the area for warmth and swelling and palpation for tenderness. Range of motion, the fulcrum test, flexion–abduction–external rotation (FABER test), and Flamingo test may assist in evaluation of areas not readily accessible to direct palpation [3].

Imaging of Insufficiency Fractures

Obtaining proper imaging is instrumental in early recognition and treatment of suspected insufficiency fractures. As with any suspected skeletal injury, plain radiographs should be obtained. Plain radiographs may assist in the diagnosis of various insufficiency fractures though magnetic resonance imaging (MRI), computed tomography (CT), or bone scintigraphy may be necessary in cases where plain radiographs are inconclusive and a patient's pain persists.

Radiography

The most common radiographic imaging finding in patients with insufficiency fracture is a sclerotic band in the affected bone. Other imaging findings on radiography of insufficiency fractures include bone resorption along the fracture line, bony expansion, callus, and osteolysis. Radiography is often more successful in detecting insufficiency fractures in long bones, pubic rami, and peripheral bones while sacral and pelvic insufficiency fractures are more often elusive on standard radiographs [37].

Multidetector CT Scanning

Multidetector CT (MDCT) scanning may be the imaging modality of choice in the detection of insufficiency fractures though the high dose of ionizing radiation limits its usage to some extent. MDCT allows for thin slices and detection of otherwise non-visible fracture lines. Callus development can also be readily observed. While bony edema on MRI can be suggestive of fracture as well as other processes such as tumor, high resolution CT scanning has the ability to rule out lytic lesions and those extending into the adjacent soft tissue [37].

Magnetic Resonance Imaging

Due to the absence of ionizing radiation, MRI is commonly used as the imaging modality of choice in the diagnosis of insufficiency fracture when plain radiographs prove inconclusive. Hypointense signal to adjacent bone is seen on T1 weighted images while hyperintense signal is observed on T2 weighted images with the possibility of observing the fracture line within bony edema. A commonly cited radiographic and MR grading system of stress fractures can be found in Table 16.4 [38]. Care must be taken as to not confuse an insufficiency fracture with underlying avascular necrosis or tumor.

Bone Scintigraphy

Bone scintigraphy, also termed radionucleotide scanning or bone scanning is very sensitive for insufficiency fractures though generally it is non-specific. Various uptake patterns can be difficult to interpret and can remain positive well after a fracture occurs. Bone scintigraphy has clinical utility in the diagnosis of insufficiency fractures of the sacrum and pelvis as two or more areas of

Table 16.4 Radiographic grading of stress fractures[a]

Grade	Radiograph findings	MRI findings
Normal	Normal	Normal
1	Normal	Positive STIR image
2	Normal	Positive STIR, plus positive T2-weighted
3	Periosteal reaction	Positive T1 and T2 weighted, STIR without definite cortical break visualized
4	Injury or periosteal reaction	Positive injury line on T1 or T2 weighted scans

[a]Adapted from Clin Sports Med, 16/2, Arendt EA, Griffiths HJ, The use of MR imaging in the assessment and clinical management of stress reactions of bone in high-performance athletes, 291–306, 1997, with permission from Elsevier

increased uptake in the sacrum and another in a pelvic site are diagnostic of insufficiency fractures [37]. Grading of stress fractures on bone scans has been proposed as grade 1, a small, ill-defined cortical area of mildly increased activity; grade 2, larger well-defined cortical area of moderately increased activity; grade 3, wide to fusiform involvement, cortical-medullary area of highly increased activity; and grade 4, transcortical area of intensely increased activity [39]. This grading system has also been correlated to MRI findings as Grade 1, mild to moderate periosteal edema on T2 with normal marrow; grade 2, moderate to severe periosteal edema on T2 with marrow edema on T2; grade 3, moderate to severe periosteal edema on T2 with marrow edema on T1 and T2; and grade 4, moderate to severe periosteal edema on T2 with a clearly visible fracture line and marrow edema on T1 and T2 images [40]. It should be noted that a more recent study made note that periosteal edema is often observed on MR but not always present in a bone stress injury [41].

Specific Sites of Insufficiency Fractures and Management

Pelvis

Pelvic insufficiency fractures are more often seen in elderly individuals with osteoporosis though any condition that leads to premature osteoporosis can predispose to the development of an insufficiency fracture. Pelvic insufficiency fractures are also associated with a history of radiation therapy in postmenopausal women. In younger women, a pelvic insufficiency fracture is most often observed in the inferior pubic rami [42]. Insufficiency fractures in the pelvis can often present as low back pain making the proper diagnosis more difficult to determine. Because plain radiographs oftentimes do not visualize these fractures with certainty, MRI of the pelvis is often necessary to confirm the diagnosis. Patients may have previously had an extensive lower back workup with the causative fracture escaping previous imaging attempts. In a 2008 study of MRI and CT imaging of 145 patients with pelvic and proximal femur stress fractures, it was found that 70.3 % of patients had a stress fracture at more than 1 site. In patients with pubic stress fractures 89.2 % had concomitant stress fractures, most commonly in the sacrum and acetabulum. In patients diagnosed with acetabular stress fractures, 76 % had concomitant fractures [43].

Pelvic insufficiency fractures are generally considered low-risk for malunion or nonunion in active individuals, therefore treatment generally consists of conservative management [44]. As mentioned previously, any underlying medical cause must be investigated and treated appropriately. A period of rest with gradual return to activity is usually utilized for pelvic insufficiency fractures. Limited or non-weightbearing is prescribed for 2–6 weeks with gradual progression to full weightbearing. Prolonged low-impact activities should be achieved without pain before resumption of high-impact exercises [44].

Sacrum

Like the pelvis, sacral insufficiency fractures can be difficult to diagnose. Bone scanning is generally very sensitive for detecting sacral insufficiency fractures as a characteristic "H" pattern or the combination of concomitant sacral and parasymphyseal uptake being typical of fractures in this region [45]. Sacral insufficiency fractures are also more common in elderly individuals and can be a cause of significant back pain. As with other

insufficiency fractures, osteoporosis and conditions that are associated with osteoporosis such as hyperparathyroidism and renal osteodystrophy are the most common underlying causes though a history of radiation therapy and Paget's disease of bone are other possibilities [46, 47]. It is also important to rule out tumor as a cause of the fracture. A 2013 study found that menstrual irregularities were noted in 75 % of female athletes with trabecular bone injuries, which includes the sacrum [41]. This presents further evidence that female athlete triad risk factors are associated with insufficiency fractures normally found in elderly women.

Similar to pelvic insufficiency fractures, sacral insufficiency fractures are generally low-risk in terms of malunion/nonunion [44]. As such, many authors advocate initial conservative management consisting of rest, pain control, and modified weightbearing. Conservative management is usually the preferred treatment method in younger individuals with sacral insufficiency fracture and return to sport has been reported between 3 and 6 months in distance runners with stress fracture, many of whom exhibited characteristics consistent with the female athlete triad [48, 49]. Resolution of symptoms can be a lengthy and risky process with conservative management in older individuals; therefore, surgical treatment techniques are sometimes the preferred treatment method. Surgical management is usually only considered in younger athletes after failed conservative management. A variation to vertebroplasty, sacroplasty, is one possible surgical treatment method which involves injection of polymethylmethacrylate cement under fluoroscopic guidance into the fracture. The benefits of sacroplasty as compared to conservative management include earlier mobilization and symptom relief as well as reduction of risks associated with prolonged immobilization [47, 50, 51]. Techniques of this procedure vary per surgeon as posterior approach, long axis approach, and midline approach have been advocated. The most significant complication is extravasation of cement outside of the fracture which may cause neurological sequelae [52].

Spine

Spinal insufficiency fractures can occur in various regions of the spine and in different areas of the vertebrae but are most often associated with the vertebral body. While spinal insufficiency fractures such as wedge or burst fractures are a common cause of back pain in the elderly with osteoporosis, literature is lacking in terms of vertebral insufficiency fractures in the younger athlete. Theoretically any condition that leads to decreased bone density in an athlete could predispose an athlete to sustain spondylolysis or a vertebral insufficiency fracture similar to an elderly osteoporotic patient. As such, care must be exercised in evaluating an athlete with back pain and risk factors for decreased bone density.

Hip and Femur

Insufficiency fractures of the femur can occur at several locations including the femoral head, femoral neck, femoral diaphysis, and femoral condyles. An insufficiency fracture of the hip can be a catastrophic injury in an athlete and as with other insufficiency fractures prevention and treatment of underlying risk factors are of monumental importance in minimizing time-lost to these injuries. Patients presenting with stress fractures in the hip often report pain in the anterior groin and pain with internal and external rotation of the hip [53, 54]. Radiographs and oftentimes MRI are of great importance in distinguishing these conditions because hip pathologies such as femoroacetabular impingement often present with similar anterior groin pain and pain with hip internal rotation.

One possible insufficiency fracture of the hip is subchondral insufficiency fracture of the femoral head. Initially, radiographs may be negative until callus formation is viewed in resolving cases [55]. In patients that progress to collapse of the femoral head, a fracture line termed a "crescent sign" may be observed. Because subchondral insufficiency fracture of the femoral head often escapes early detection by radiographs,

MRI is necessary in suspected cases. Bone marrow edema and a low-signal intensity line on T1 images parallel to the subchondral bone are often observed [37]. Subchondral insufficiency fracture is oftentimes confused with osteonecrosis of the femoral head. A history of corticosteroid use or alcohol abuse may raise suspicion of osteonecrosis while a history of osteoporosis may raise suspicion of subchondral insufficiency fracture [56]. Radiographic appearance and MR findings may be similar between subchondral insufficiency fracture and osteonecrosis. While not always reliable, a high signal intensity of the proximal segment divided by the fracture line on a T2 or gadolinium enhanced image suggests subchondral insufficiency fracture while in osteonecrosis, before healing occurs, the subchondral bone segment proximal to the low intensity band is of lower signal intensity. Histopathology is the diagnostic confirmatory test to discern the two entities.

It has been suggested that if femoral head collapse is not present conservative management may be utilized. In cases of anterosuperior femoral head collapse in young patients a transtrochanteric rotational osteotomy may be a treatment option while hemiarthroplasty or total hip arthroplasty is utilized in elderly patients [57].

Another possible insufficiency fracture of the hip occurs at the femoral neck. On MRI, fracture lines and bone marrow edema are often appreciated, especially on coronal images [43]. It is important to distinguish the affected area of the femoral neck as insufficiency fractures occurring at the superior femoral neck (tension side) are at significant risk for malunion, nonunion, fracture migration across the femoral neck with subsequent displacement, and consequently avascular necrosis [53]. While strict non-weightbearing may successfully treat these fractures, their propensity for displacement oftentimes leads to cancellous lag screw fixation of the femoral neck [58]. In addition, these patients require 6 weeks of non-weightbearing followed by 6 weeks of partial weightbearing postoperatively. In contrast, an insufficiency fracture occurring at the inferior femoral neck (compression side) is at significantly lower risk for nonunion and displacement and can most often be treated non-operatively [53, 59] (Fig. 16.1).

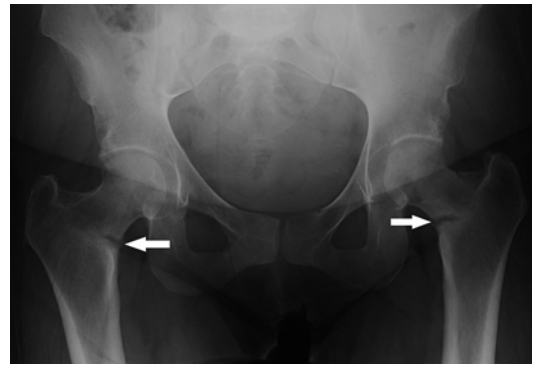

Fig. 16.1 Bilateral insufficiency fractures on the compression (inferior) side of the femoral neck in a patient with suspected osteomalacia. Fractures indicated by *arrows*. Image courtesy of Joanna Costello, M.D.

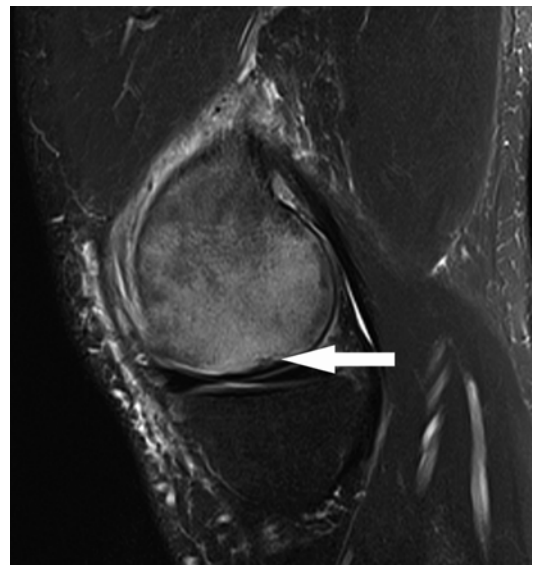

Fig. 16.2 T2 sagittal MRI image of a subchondral insufficiency fracture with subchondral collapse of the medial femoral condyle in a previously active 64-year-old male. Note the significant bone marrow edema and subchondral collapse indicated by the *arrow*. Image courtesy of Joanna Costello, M.D.

It should be noted that subchondral insufficiency fracture can also occur in the femoral condyles and may be termed spontaneous osteonecrosis of the knee (SONK or SPONK) (Fig. 16.2). These insufficiency fractures have also been associated with low bone mineral

density [60]. Subchondral insufficiency fractures in this region are initially treated with pain management, protected weightbearing, and possibly bisphosphonates [61] with refractory cases necessitating high tibial osteotomy [62], unicompartmental arthroplasty [63], or total knee arthroplasty.

Insufficiency fracture can also occur at any point along the femoral diaphysis. Clinical presentation often includes pain in the thigh with weight bearing. These fractures tend to have a lower risk of nonunion and displacement so management typically consists of conservative measures.

Tibia

The tibia is a common location of both insufficiency and fatigue stress fractures in the athlete. These stress fractures tend to present in distance runners and can be a source of continuous pain and debilitation. A 2005 study of imaging findings in early tibial stress injuries in active young patients found that 21/50 of the tibiae studied were osteopenic [64]. This finding highlights the fact that many tibial stress fractures can be classified as insufficiency fractures rather than fatigue fractures due to the presence of underlying abnormality. This also highlights the fact that prevention of osteopenia in young patients is of significant importance in terms of fracture prevention.

Due to the possibility of nonunion and extension of the fracture line, anterior tibial diaphyseal stress fractures are classified as high-risk. Patients often present with complaints of pain with weightbearing activities and tenderness over the anterior tibia [53]. Oftentimes, tibial stress fractures may be appreciated on plain radiographs as a cortical thickening or in persistent fractures, a transverse fissure line in the cortex [65] (Fig. 16.3a). On occasion, multiple fissures corresponding to more than 1 stress fracture may be observed. If left untreated these small defects can progress to a complete fracture through both cortices. With inconclusive radiographs in high-level athletes, MRI is a standard imaging technique. T2 and STIR MRI sequences most often confirm cortical abnormalities, a round or oval area of high signal intensity and a hyperintense line through the cortex.

Athletes that fail a period of non-weightbearing with crutches and possible pneumatic bracing may necessitate surgical treatment. Successful union, resolution of pain, and return to play at as early as 4 months has been reported with intramedullary

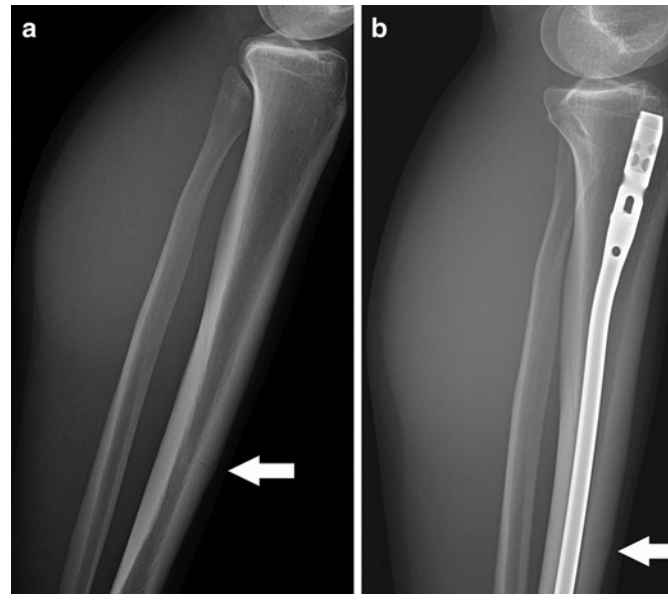

Fig. 16.3 (**a**) Anterior tibial diaphyseal stress fracture in a 19-year-old gymnast with risk factors for the female athlete triad. Fracture indicated with *arrow*. (**b**) Postoperative radiograph demonstrating successful treatment with an antegrade intramedullary nail following a failed course of non-operative management as well as calcium and vitamin D supplementation. The fracture line is less visible 2.5 months postoperatively. Location of original fracture line indicated with *arrow*. Images courtesy of Joanna Costello, M.D.

nailing (Fig. 16.3b) [66], anterior tension band plating [67–70], or drilling with bone grafting of the cortical defect [71, 72].

Foot

Stress fractures of the foot have been reported in every bone except the lesser toes. Commonly affected sites include the calcaneus, talus, navicular, and metatarsals. Because of the large number of bones in the foot, localization of the potential fracture may be difficult. Insufficiency fractures of the foot are especially common in the neuropathic foot associated with diabetes mellitus and in the elderly with osteoporosis though they may occur in younger athletes with underlying bone density issues.

Like other stress fractures, calcaneal stress fractures are more likely to be fatigue fractures in young patients or insufficiency fractures in the elderly though they do occur as insufficiency fractures in younger athletes. Insufficiency fractures of the calcaneus commonly present with pain and tenderness, especially in the posterior superior region [44]. The pain may mimic Achilles tendinitis/bursitis so proper imaging is necessary to differentiate between the two. Radiographs may be useful in the imaging of these lesions and the fractures are usually viewed as a sclerotic line that lies in a vertical-type orientation [44]. MRI is also useful in confirming these fractures and differentiation from Achilles pathology and more severe lesions such as tumor. Marrow edema is often present on T2 sequencing and a vertically oriented fracture line may be appreciated [73]. Calcaneal stress fractures are classified as low-risk for nonunion, therefore they may successfully heal with nonoperative management which can include nonweightbearing progressed to a boot and subsequently adequate heel lift and padding. Fracture healing may be affected if underlying medical pathologies are not treated as well. Figure 16.4a, b presents a case of insufficiency fracture of the calcaneus in a 26-year-old female runner that healed after prolonged conservative and medical management.

The talus is another hindfoot bone that may experience insufficiency fracture. These fractures are associated with decreased bone density [74] and rheumatoid arthritis [75]. Talar stress fractures

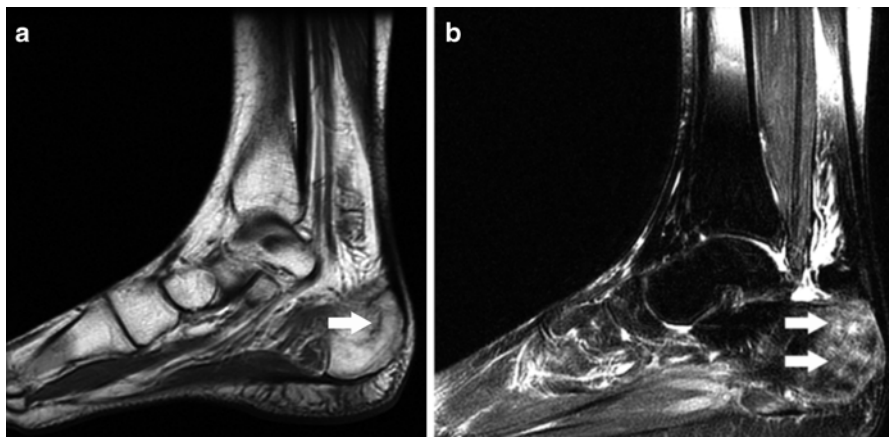

Fig. 16.4 (**a**) T1 weighted sagittal MRI image of a curvilinear calcaneus insufficiency fracture in an active 26-year-old runner with progestin only birth-control induced amenorrhea, vitamin D deficiency and DEXA confirmed osteopenia. Fracture line indicated by *arrow*. (**b**) T2 weighted sagittal MRI image demonstrating bone marrow edema (indicated by *arrows*) in the area of the fracture. The patient was treated with estrogen containing contraceptive medication, calcium and vitamin D supplementation, extended non-weightbearing and was eventually transitioned to a boot and full weightbearing. Two-year follow-up DEXA scanning demonstrated improvement in bone density to *Z*-scores within normal ranges. Images courtesy of Joanna Costello, M.D.

can occur in the talar body, talar neck, or lateral process with the majority occurring in the talar head in young patients [76]. Patients may complain of pain in various areas of the foot, making these fractures difficult to localize [74]. Standard radiographs may not appreciate these lesions therefore MRI and/or CT are usually necessary. On MR imaging, bone marrow edema is often observed on T2 weighted imaging and a subchondral linear fracture line may be viewed on T1 weighted images. In contrast to stress fractures of the calcaneus, talar insufficiency fractures are at high-risk for nonunion and often necessitate surgical treatment though healing with protected weightbearing and casting has been reported [53, 74].

Metatarsal stress fractures, specifically 5th metatarsal stress fractures are a well-studied injury in athletes. While metatarsal stress fractures are most often of the fatigue type, insufficiency fractures have been reported. According to the most common anatomical classification system, fifth metatarsal stress fractures are found at the proximal diaphysis and are not to be confused with the avulsion (Zone 1) fracture or Jones (Zone 2) fracture. As opposed to the Jones fracture which is located at the metaphyseal-diaphyseal junction and enters the 4th and 5th metatarsal articulation, the stress fracture lies distal to the 4th–5th articulation [77, 78]. Patients often report pain exacerbated by inversion and tenderness over the lateral foot. These fractures can most often be observed on standard radiographs as a radiolucent line making further imaging unnecessary in most cases. Second metatarsal base and fifth metatarsal fractures are considered high-risk and are prone to nonunion and refracture after conservative management making intramedullary screw placement the preferred treatment method [77–79]. A 2014 study of 5th metatarsal fractures, foot stress fractures, and ankle fractures found that 47 % of the patients studied had a vitamin D level below the recommended level, suggesting that a number of these fractures may have a component of insufficiency rather than solely being fatigue fractures [80].

Upper Extremity

Upper extremity stress fractures are relatively uncommon injuries. Most literature reports examining upper extremity stress fractures explore fatigue type injuries rather than the less common insufficiency fracture in a younger athlete. Sites of reported stress fracture include the shoulder girdle, humerus, ulna, radius, scaphoid, and metacarpals. Throwing athletes, swimmers, gymnasts, weight lifters, and rowers are the more commonly reported athletic populations that sustain upper extremity stress injury [81]. In general upper extremity stress fractures are considered low-risk for nonunion and can be successfully treated with non-operative management [44]. Li and colleagues reported the case of a 12-year-old male baseball pitcher with secondary hyperparathyroidism and vitamin D deficiency that sustained a proximal humeral stress fracture and subsequently was diagnosed with a proximal ulnar stress fracture as well as spondylolisthesis. Interestingly, the athlete had a *Z*-score of 2.76 consistent with significantly elevated bone mineral density. The authors concluded that this increased bone mineral density may have translated into bone changes similar to osteopetrosis. The athlete was treated non-operatively and with medical management of his vitamin D levels and subsequently returned to baseball participation [82]. Being that a stress fracture associated with abnormal bone is an insufficiency fracture, it is clear that upper extremity insufficiency fractures do occur in athletes and at times may be misclassified as fatigue fractures. As with other stress fractures, underlying medical diagnoses must be ruled out before concluding the presence of fatigue fracture rather than insufficiency fracture in the young athlete.

Summary

Insufficiency type stress fractures are less commonly reported than fatigue type stress fracture but do in fact occur in athletes. At particular risk are aging athletes with osteopenia or osteoporosis

and younger athletes with decreased bone density as a result of underlying causes such as the female athlete triad. Recognition of the causes of abnormal bone predisposing an athlete to insufficiency fracture is essential in the prevention of this type of injury. Proper imaging, correction of underlying medical pathologies, and possible surgical intervention in refractory and high-risk cases are necessary in order to maximize an athletes' potential to return-to-play.

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