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University of Gondar
College of Medicine and Health sciences
School of Nursing

Communicable Disease Control (CDC)

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2012 E.c

Gondar, Ethiopia

Course syllabus

- **Module Name:** Clinical Nursing II/
- **Course title:** Communicable Disease Control
- **Credit hour:** 2 Cr(38 hrs.)
- **Module Code:** NursM2093/
- **Course delivered to** -Surgical and Emergency Nursing 2nd year
- **Course instructor** -Chilot Desta(BScN, MPH in EPI/BIO)

Course description

- This course is designed to prepare graduate BSc.Nurse who are competent providers of community health services with regard to early case detections, management of cases, control and prevention of communicable diseases:
- Major topics includes are:
  - ✓ introduction of communicable diseases
  - ✓ Methods of communicable diseases control
  - ✓ Oro-fecal transmitted diseases

Course description …….

- ✓ Air borne diseases
- ✓ Food poisoning
- ✓ **Course objectives**
- ❖ Comprehend the basic concepts and theory regarding CDC
- ❖ Identify the common communicable disease
- ❖ Describe mode of transmission of communicable disease
- ❖ Describe factors involved in the transmission of CDC
- ❖ Explain methods of CDC

Course objectives .....

- ✓ Play an active role in the control of communicable diseases
- ✓ Organize effective heath education on CDC
- ✓ Recognizing nursing care and treatment of a patient with communicable disease

supportive objective……

For the completion of this course, students will be able

- ✓Adopt general overview of CD, its unique feature and scope
- ✓Differentiate the natural history, time course and chain of infectious disease
- ✓Identify epidemics and disease of public health importance in Ethiopia
- ✓Contribute in the disease prevention and control program
- ✓Identify, diagnose & manage of common CD

Teaching method

- ✓ Brain storming
- ✓ Interactive lecture
- ✓ Group discussion
- ✓ Case study
- ❑ **Assessment method**
- ✓ **Quizzes ……...10%**
- ✓ **Assignment with presentation……15%**
- ✓ **Mid exam…… .25%**
- ✓ **Final exam……50%**
- ✓ **Total……..100%**
- ✓ **Active participation has its on value !!!**

Attendance criteria

- ✓ Students are expected to attend all classes(100%).

4. Course content

- Part I: Introduction
- Part II: chain of disease transmission
- Part III : Surveillance and the investigation and management of outbreaks
- Part IV: Oral-fecal transmitted diseases
- Part V: Food borne diseases
- Part VI: vector born diseases
- Part VII: Sexually transmitted disease
- Part VIII: Zoonotic diseases

Chapter one : Introduction to communicable Disease

Learning Objectives:-

- Define communicable disease (CD).
- Identify different way of classifying infectious disease .
- Describe the over view of Ethiopian health situation .

Intro….

- What are infectious diseases?
- What are communicable diseases?
- What are contagious diseases?

Definition of Communicable /infectious disease

- Disease due to a specific **infectious agent /its toxic products** that **transmitted** from an **infected** person/animal/reservoir to a susceptible host.
- **Infectious diseases (IDs):** are those that result from infection or infestation.
- **Contagious diseases:** are those CDs that are transmitted directly from the patient to a susceptible individual.
- They are called so because they spread from person to person, or sometimes from animals to people.

Brain storming

- Discuss in pair the following terms(5 minute)
  - Disease
  - infection
  - Host
  - Epidemic
  - Endemic
  - Sporadic
  - Communicable disease
  - Non- Communicable disease
  - Pandemic

introduction basic term definition

- **Disease:-** a state of physiological or psychological dysfunction.
- **Infection** – the entry and development or multiplication of an infectious agent in the body of human beings or animals
- **Infestation** – presence of living infectious agent on the exterior surface of the body .
- **Host** – the person or animal that affords subsistence or lodgment to an infectious agent under natural conditions

Cont……

- **Infectious disease** – a clinically manifest disease of a man or animal resulting from an infection
- **Communicable disease** – an illness due to a specific infectious agents or its toxic products capable of being directly or indirectly transmitted from man to man, animal to animal or from environment to man or animal
- **Epidemic** – the unusual occurrence of disease in a community or region, specific health related behavior or events clearly in excess of expected occurrence

Cont. . .

- **Endemic** – constant presence of a disease or infectious agent within a given geographic area or population group; without importation from outside.
- **Sporadic** – cases occur irregularly, haphazardly from time to time and generally infrequent eg. polio, tetanus, herpes zoster
- **Pandemic** – an epidemic affecting a large proportion of the population occurring over a wide geographic area
- **Zoonosis** – an infection transmissible under natural conditions from vertebrate animals to man

Cont…

- **Nosocomial (hospital acquired)infection** – infection originating in a patient while in hospital or other health care facility
- **Opportunistic infection** – an infection by organisms that take the opportunity of defect in the host defense to infect the host and cause disease
- **Iatrogenic disease** – is the result of diagnostic and therapeutic procedures undertaken on a patient resulting from activity of physicians or health professionals.

cont..

- **Prevention** -To keep from occurring or limitation of certain events.
- **Control** – ongoing operation aimed at reducing incidence, duration and effects of disease
- **Elimination** – eradication of disease from large geographic region or political jurisdiction eg measles, polio, diphtheria
- **Eradication** – termination of infection by extermination of the infectious agent through surveillance and containment but the infectious agent is present in the lab..
- **Extinction**–eradication of infectious agent including in the lab

Classification of disease

Different ways of classifying disease

- Based on cause
- Clinical
- Time course
- Infectious agent
- Mode of transmission

The Global Burden of Infectious Diseases

- IDs are major causes of morbidity and *mortality* worldwide
- IDs contribute to 25% of death every year
- They occur at all ages but are most serious in childhood and they are to a great extent preventable
- In countries where they have been prevented/developed countries, other health conditions such as ***accident*** and ***degenerative diseases*** become the most common.

What are the Impacts of infectious disease ?

Cont..

Communicable diseases remain very important in developing countries because.

- Many of them are very common
- Some of them are serious and cause death and disability
- Some of them cause widespread **out breaks of diseases or epidemics**
- Most of them are preventable by fairly simple means
- Poor socio economic status of the individuals makes them vulnerable to a variety of diseases
- Low educational status
- Lack of access to modern health care service

Special features of CD

- A case must be a risk factor
- Each infectious disease has its own IP (incubation period)
- People may develop immunity.
- Individuals can be a sources without being recognized as a case.
- Preventive measures usually have a good scientific ground

Over view of health situation of Ethiopia

In general , communicable disease in Ethiopia situation:

- Are commonest cause of morbidity& mortality
- Are relatively easy to prevent and control
- Are epidemic prone (cause wide spread out breaks of disease).
- Put most of the population at risk
- 60-80% of the nation's health problems are due to nutritional problems & communicable disease.

Epidemiology and scope

What is the importance of Studying Communicable Diseases Epidemiology ?

- Changes in the pattern of infectious diseases
- Discovery of new infections
- The possibility that some chronic diseases have an infective origin

❖ Even though , CD is the leading cause of **mortality** and **morbidity** in developing countries in the past 70 years the incidence of CD was fall down.

❖ What are the reasons to decline incidence of CD in the past 7o years

Global Factors of Emerging Communicable Disease

- Dramatic, societal and environmental changes
- Explosive population growth
- Expanding poverty and urban migration
- International travel and commerce are increasing
- Technology is rapidly changing and increases the risk of exposure,
- Climatic change
- Emerging of new viruses
- Immuno-compromization
- Resistance of Micro organisms to antibiotics

Chapter two: Natural History of disease

Learning Objectives

- Describe the natural history of diseases
- Describe the time courses of infectious disease
- List types of carriers and explain their role in disease transmission
- Describe chain of disease transmission
- Apply level of prevention and control mechanism of CD

Discuss in Groups (5-10)minutes

- Definition of natural history of the disease
- Discus about the stages of natural history of the disease with its example
- You should have your own group Secretary

There are four stages in the natural history of a disease. These are

1. Stage of susceptibility-period of exposure:

Disease has not yet developed, but there are factors that favor occurrence.

Examples:

A person practicing casual and unprotected sex has a high risk of getting HIV infection.

An unvaccinated child is susceptible to measles.

High cholesterol level increases the risk of coronary heart disease.

2. Stage of sub clinical disease /pre-symptomatic stage:-

The disease process has already begun but , the disease is not manifested

The disease can only be detected through special tests.

Example

- Oval or intestinal parasite in the stool of apparently healthy children.
- Antibodies for polio virus are present in many adults less than one percent.

3. Stage of clinical disease :-

signs and symptoms of the disease are manifested.

4.Stage of disability or death

The disease has occurred and left over damage to the body that limits the activity of the victim(disability) or has ended with the death of the victim Disability means any limitation of physical activity.

Examples: -

- ✓ Trachoma may cause blindness.
- ✓ Meningitis may also result in death.

Time Course of an infectious disease

The different periods that are encountered in the course of infectious disease :

1. Prepatent period
2. Incubation period
3. Communicable period
4. Latent period
5. Period of illness
6. Period of decline
7. Period of convalescence.

A. Incubation period

The interval of time between infection of the host & the first appearance of symptoms & signs of the disease.

B. Pre patent period

This is the time interval between infection (or **biological onset)**, and the point at which the infection can first be detected, as measured by the time of **first shedding** of the agent by the host.

In some conditions, like the AIDS, it is the so called "window period".

C. Communicable period

The period during which an infected host can transmit the infection to others which can be measured by the time interval during which the agent is shed by the host.

D. Latent period

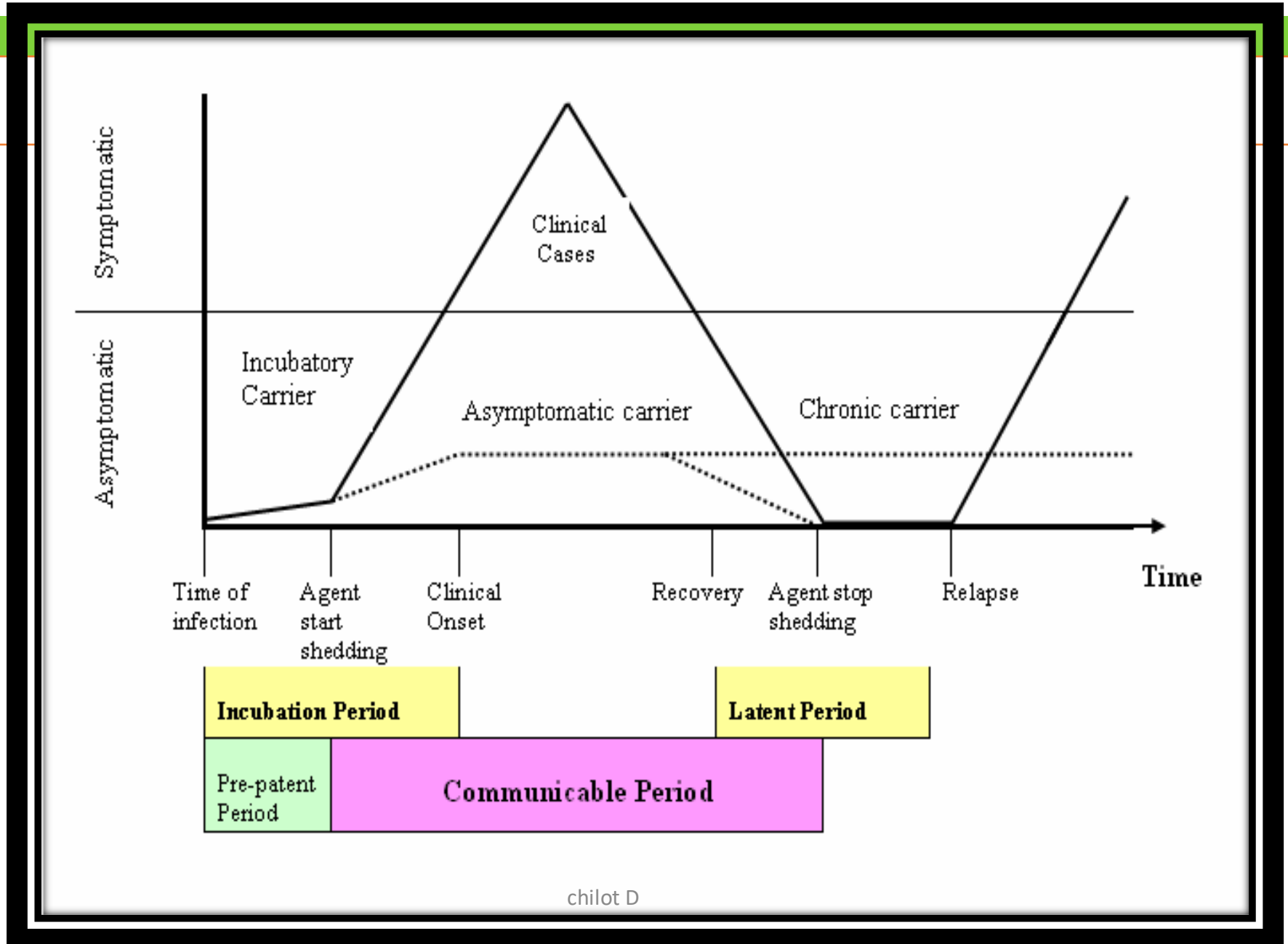
The time interval between **recovery** and the occurrence of a **relapse** or recrudescence in clinical disease, as in cases of malaria and epidemic typhus.

E. Period of illness : acute phase, ***most severe signs*** and symptoms & the immune system either overcomes pathogen or person dies.

cont.…..

F. Period of decline : signs and symptoms begin to subside.

G. Period of convalescence : host returns to pre-disease state


Symptomatic
Asymptomatic
Clinical Cases
Incubatory Carrier
Asymptomatic carrier
Chronic carrier
Time of infection
Agent start shedding
Clinical Onset
Recovery
Agent stop shedding
Relapse
Time
Incubation Period
Latent Period
Pre-patent Period
Communicable Period

Carrier and its type

- **Carrier:-** is an infected person or animal without manifestation of disease but capable of transmitting infection to others.

Three elements have to occur to form a carrier state:

1. The presence of the disease agent with in the body.
2. The absence of recognizable signs and symptoms of disease.
3. The shedding of disease agent in the discharge or excretions.

1. Healthy or asymptomatic carriers

- Persons whose infection remains unapparent. E.g. Polio virus, meningococcus, hepatitis virus, HIV infection

2. Incubatory or precocious carries

- Individuals or persons who excrete the pathogen during the incubation period (before onset of symptoms) E.g. measles, mumps, chicken pox, hepatitis

3. Convalescent carriers

- Those who continue to harbor the infective agent after recovering from the illness. E.g. diphtheria, hepatitis B virus

4. Chronic carriers

- The carrier state persists for a long period of time. E.g. typhoid, hepatitis B virus

❖ Generally, carriers are important in epidemiology because of their:

- ❑ number (may become significant reservoir);
- ❑ difficulty in recognition(detectability);
- ❑ mobility; and
- ❑ chronicity, i.e repeated reintroduction and contribute to endemicity

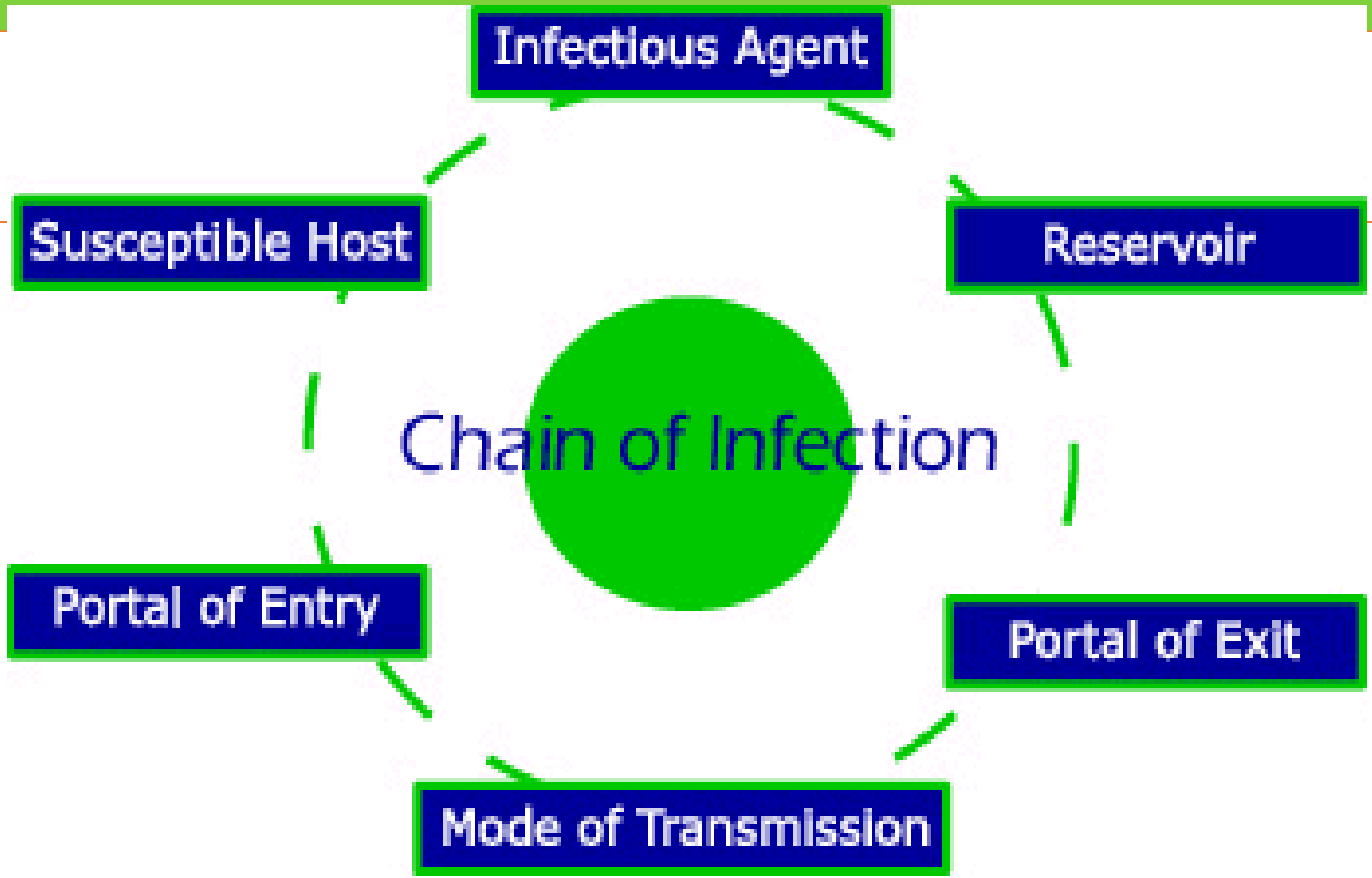
Chain of disease transmission

is a series of events , which must occur in order for disease causing organisms to cause infection .

There are six successive events implicated in the chain of disease transmission.

1. Infectious agent
2. Reservoir
3. Portal of exit
4. Mode of transmission
5. Rout of entry
6. Host

chain of the disease



1. Infectious agent

- Is pathogen (disease causing micro organism) that cause infection.
- The infectious agent is virus, bacteria, parasite or other microbes
- The agent causes infection and disease depending on its basic biological characteristics .
- The outcomes of exposure to infectious agent can be depends on:
  - Infectiousness
  - Pathogenecity
  - Virulence
  - Immunogenicity

Infectiousness (infectivity)

- The ability of an infectious agent to cause *infection* in an exposed human host
- Infectivity is measured by Infection Rate (IR):

$$IR = \frac{Number\ of\ \inf\ ectedPersons}{Number\ of\ Susceptible\ \&\ \exp\ sedPersons} \times 100$$

Pathogenicity

- The ability of an infectious agent to cause clinical disease among infected human hosts
- It is measured by clinical to sub-clinical ratio or proportion of clinical cases among infected human hosts

Examples:

- High pathogenecity: HIV, Rabies, Measles… etc.
- Moderate pathogenecity: Mumps virus and Rhino virus
- Highly infectious but less pathogenic: Poliovirus

Virulence

- It is the ability of an infectious agent to cause severe clinical disease or death among clinical cases
- Virulence of the infectious agent can be measured by:
  - Case fatality Rate (CFR)
  - Hospitalization Rate (HR)

$$CFR = \frac{Number\ of\ Fatal\ Cases}{Total\ Number\ of\ Cases} \times 100$$

$$HR = \frac{Number\ of\ Hospitalized\ Cases}{Total\ Number\ of\ Cases} \times 100$$

Immunogenicity

- infection's ability to produce specific immunity
- It is defined as the ability of a pathogen or a vaccine to inducing an immune response after an infection or a vaccination respectively
- It may lead to protection against re-infection or re-activation with the same or similar pathogen in the future
- immunity

Immunogenicity

- Immunity after infection may or may not be protective against re-infection or may last for variable periods of time
  - some infectious diseases confer lifelong immunity
  - others confer partial immunity against severe symptomatic infection, but much less against sub-clinical infection
  - some confer no or no egligible immunity

Immunity..

- Immunity can be acquired either after natural infection or indirectly
- Maternal antibodies protect the newborn child against many infections in the first few months of life
- Vaccine-induced immunity can be lifelong or temporary
- For temporary immunity, repeated booster vaccinations are necessary to ensure protection against the infection

Example: TT vaccine

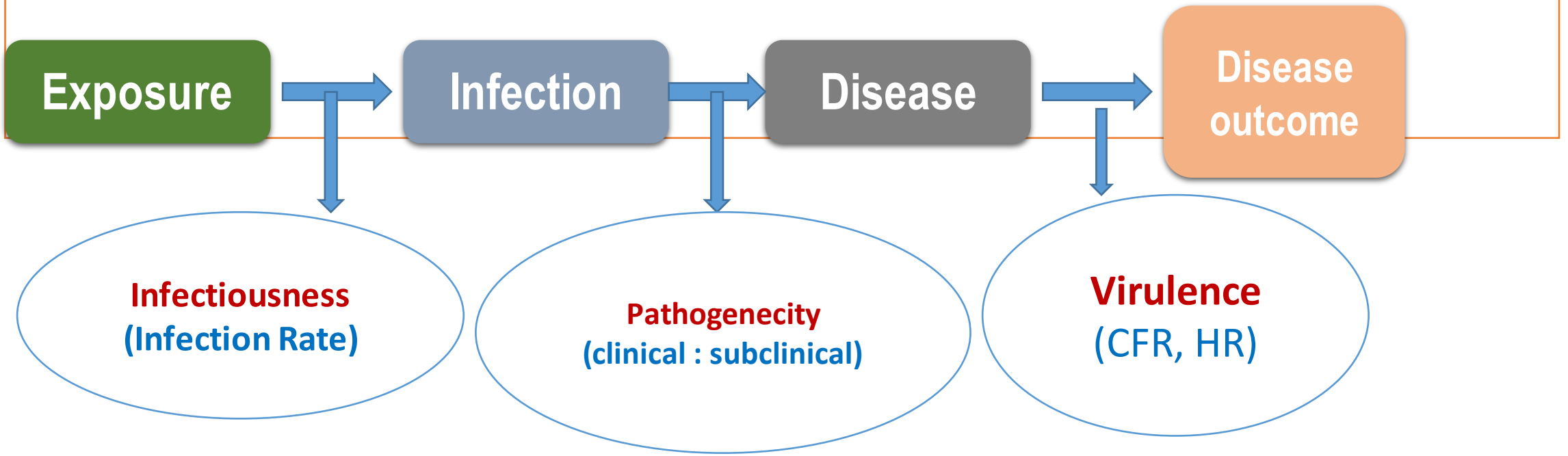
Immunity..

- Individuals gain protective antibodies in two ways:

1) They develop antibodies in response to infection, vaccine, or toxoid; immunity developed in these ways is called **active immunity**

2) They acquire their mothers' antibodies before birth through the placenta or they receive injections of antitoxins or immune globulin; immunity that is acquired in these ways is called **passive immunity**

Disease progression



cont…

- Factors that influence disease development other than infectivity, pathogenicity, immunogenecity and virulence of the agent are:
  - Strain of agent
  - Dose of agent
  - Route of infection
  - Influence of human host age
  - Influence of human host nutritional status
  - Influence of human host immune response
  - Influence of treatment
  - Influence of seasonal variation, etc…

2. Reservoir of infection

It is an organism or habitat, in which an infectious agent normally lives, transforms, develops and/or multiplies.

Types of reservoirs:

- **man** -The cycle of transmission is from man to man.
    - measles, smallpox, typhoid,
    - N.meningitis, gonorrhea, syphilis.

A person who does not have apparent clinical disease, but is a potential source of infection to other people is called a **Carrier**.

cont..

- **Animals-** cause zoonotic diseases
- **Non-living things**:- soil , food , water .

3. Portal of exit

- It is the site through which the agent escapes from the reservoir.
- Examples include:
  - Gastrointestinal tract
  - Respiratory tract
  - Skin & mucous membrane

Cont…

- **Portal of exit includes:**
  - —Body secretions and discharges (mucus, saliva and tears, breast milk, urethral secretion, semen, vaginal secretion, pus, cervical secretion, exudates etc…)
  - —Excretions (feces and urine), blood and tissues including placenta , etc…
  - —Flu or cold - mucous secretions
  - —Hepatitis A - stool

4. Mode of transmission

- Mode of transmission is the mechanism by which the infectious agent escapes from a reservoir and enters into a susceptible human host
- There are two major mechanisms of transmission

I. Direct transmission

II. Indirect Transmission

1. Direct transmission

- The immediate transfer of infectious agents from an infected host or reservoir to the appropriate portal of entry on the susceptible host.
- **What are the direct transmission and discus with your Groups**

a) **Transmission by direct contact**

b) **Transmission by direct projection**

c) **Trans-placental transmission**

d) **Blood transfusion**

e) **Organ transplantation**

2. Indirect Transmission

a) Airborne transmission

b) Vehicle borne transmission

c) Vector borne transmission

d) Non vector intermediate host

cont…

1. Air borne transmission

- Microbial agents are disseminated by air in to suitable portal of entry usually the respiratory tract
- Two particles; dust & droplet nuclei are implicated in this kind of spread

2. Vehicle-borne transmission such as:

- Bedding, soiled clothes
- Contaminated food & water
- Biological products such as blood & serum, iv fluids

3. Vector- borne transmission

- The infections agent is conveyed by an arthropod (insect) to a susceptible host

- A vector is responsible for introducing the agent into the susceptible human host through a suitable portal of entry
  - Biological vector: salivarian transmission (malaria by mosquito)
  - Mechanical vector: trachoma by common fly

cont..

- Biologic transmission -when the agent undergoes physiologic changes within the vector, the vector is serving as both an intermediate host and a mode of transmission
  - An agent undergoes part of its life cycle inside a vector before being transmitted to a new host
- Mechanical transmission - the agent does not multiply or undergo physiologic changes in the vector

4. A non-vector intermediate host

— These are hosts which are important for development of the infectious agent but don't play an active role in transporting the agent to the susceptible human host.

Example: Aquatic snail for schistosomiasis

5. portal of entry

- Portal of entry is the route through which a microorganism enters into the susceptible human host
- Whether an agent will establish infection depends on the suitable portal of entry

Example: No harm in ingesting the Clostridium tetani (it is in fact the normal flora of GIT system)

6.Susceptible host

- A person lacking sufficient resistance to a particular pathogenic agent to prevent disease if exposed.

Level of susceptibility depends up on

- Age : extreme of age
- Nutritional status
- Stress
- pre- existing medical conditions
- Immune status
- Personal behaviors like drinking, smoking etc
- Anti-microbic ant-biotics availability and effectiveness

Exercise

- Suppose 40 4th year nursing students have gone to West Africa for clinical attachment . Unfortunately 22 and 18 of them found to have SARS and Ebola infection with laboratory investigation respectively. Among SARS infected individuals 10 developed disease and 6 of them died. Among Ebola infected individuals 8 developed disease and 3 of them died. Based on the above case scenario
- 1. Calculate case fatality rate?
- 2. which disease is more pathogenic?


Thank u Very much !!!

Goal & principle of CDC & prevention

Goals

- **Eradication** :

reducing the incidence to zero level.

- **Elimination** :

reducing the incidence of disease to zero level in specified geographic areas.

- **Control**:

reducing the incidence to the level where the disease is no more public health importance.

cont..

There are three main methods of controlling communicable diseases:

1. **Elimination of the Reservoir**

a. **Man as reservoir:**

- *Detection and adequate treatment of cases*

Isolation:

- separation of infected persons for a period of communicability of the disease.

Quarantine:

- limitation of the movement of apparently well person or animal who has been exposed to the infectious disease for a duration of the maximum incubation period of the disease.

b. Animals as reservoir

Action will be determined by the usefulness of the animals, how intimately they are associated to man and the feasibility of protecting susceptible animals

- destroy
- vaccination.

For example:

Plague: the rat is regarded as a pest and the objective would be to destroy the rat and exclude it from human habitation.

Rabies: pet dogs can be protected by vaccination but stray dogs are destroyed

cont..

c. Reservoir in non-living things

Possible to limit man's exposure to the affected area (e.g. Soil, water, forest, etc.)

2. Interruption of transmission

- ✓ Improvement of environmental sanitation and personal hygiene
- ✓ Control of vectors
- ✓ Disinfections and sterilization

3. Protection of susceptible host:

. This can be achieved through:

- Immunization: Active or Passive
- Chemo-prophylaxis(e.g.Malaria,meningococcal meningitis,etc.s)
- Better nutrition
- Personal protection. (e.g. wearing of shoes, use of mosquito bed net, insect repellents, etc.)

Herd immunity

- Host resistance at the community (population) level.

I.e. the resistance of the community to the introduction and spread of infectious agent based on the immunity of high proportion of individuals in the population , thereby lessening the likelihood of a person with a disease coming in to contact with susceptible individuals.

Prerequisite for herd immunity

1. Single reservoir (the human host).
2. Direct transition (direct contact or direct projection).
3. Total immunity :partial immune hosts may continue to shed the agent

Cont..

4.No shedding of agents by immune hosts (no carrier state) .

5. Uniform distribution of immunes.

6. No over crowding.

- However , these conditions for the operation of herd immunity are seldom fulfilled .

N.B. mostly a combination of different methods are used to control a specific communicable disease .

level of prevention

A. Primary prevention

This is protection of healthy people from becoming sick.

The objectives here are to promote health , prevent exposure, and prevent disease.

Health promotion

- This consist of general non specific interventions that enhance health & the body's ability to resist disease .

  improve socio –economic status

  adequate housing , clothing &food

  Education and vocational training,

  Emotional and social support and

  relief of stress, etc.

- In short it is any intervention that promotes a healthier and happier life.

prevention of exposure

provision of safe and adequate water;

- proper excreta disposal;
- vector control;
- safe home, school, and
- street environments.

Prevention of disease

- This occurs during latency period between exposure and biological onset.
- Involves activities such as active and passive immunization

B. secondary prevention

- is a type of prevention after biological onset of diseases and before permanent damage by the disease.

The objective here is:-

- ✓ stop or slow the progression of disease.
- ✓ prevent or limit permanent damage.
- ✓ early detection and treatment of disease.

e.g. trachoma -prevention of blindness

syphilis- prevention of tertiary or congenital syphilis

C. Teritariy prevention

is a type of prevention after the disease has already occurred and left residual damage.

- **Rehabilitation** refers to the retaining of remaining functions for maximal effectiveness.
- **Limitation of disability** helps to limit or stop the damage and impact of damage.
- The impact can be physical, psychological, social (e.g., social stigma) and financial

Level of prevention	Point of intervention	Natural history of disease
Primary	Health promotion Prevention of exposure Prevention of disease	
Secondary	Early detection &treatment =screening (prevention of clinical onset) Early treatment (prevention of permanent damage)	incubation period
Tertiary	Limitation of disability Rehabilitation (prevention of deterioration in quality o f life)	

Excerscise

For each of the following intervention , indicate what level of prevention involved

1. Specials education for the handicapped children.
2. Treatment of people with a diagnosis of active TB.
3. Screening donated blood to exclude contaminating with causation agent of HBV.
4. Counseling DM patient how to take medication
5. Promoting awareness in high school students of self-protective measure again HIV/AIDS.


Thank u Very much !!!

Excerscise

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Part III: Air born diseases

OBJECTIVE

At the end of this chapter students will be able to:

- List common Air born diseases
- Identify the common clinical manifestations of Air born disease
- Participate in diagnosis and treatment of Air born diseases
- Apply preventive and control methods of Air born diseases

Group discussion

- ✓ List common air born disease
- ✓ Identify common characteristics of air born diseases
- ✓ Prevention and their control mechanism

Tuberculosis (TB)

Objectives

- Define tuberculosis(TB)
- Identify the common modes of TB transmission
- Describe different types of TB
- Participate in diagnosis and treatment of TB
- Apply preventive and control methods of TB

Part III: Air born diseases

- Are a group of diseases that are transmitted by inhalation of **infected air droplets.**
- The organisms causing these diseases enter the body via the *respiratory tract*.
- When a patient or carrier of pathogens talks, coughs, laughs, or sneezes, he/she discharges fluid droplets.
- The smallest of these remain up in the air for some time and may be inhaled by a new host.
- Droplets with a size of 1-5 microns are quite easily drawn in to the lungs and retained there.

con…

✓Droplets which are bigger in size will not remain air borne for long but will fall to the ground.

✓ however, they dry and mix with dust.

✓When they contain pathogens which are able to survive drying these may become air borne again by wind or something stirring up the dust, and they can then be inhaled.

1. Tuberculosis

- Tuberculosis (TB) is an infectious disease that primarily affects the lung parenchyma, but almost all organs can be affected.

Epidemiology

- TB is a worldwide public health problem that is closely associated with poverty, malnutrition, overcrowding, substandard housing, and inadequate health care.
- 1/3 the world's population is infected with TB.
- In 2013, **9 million** people around the world became sick with TB disease. There were around 1.5 million TB-related deaths worldwide.

Cont…

- An estimated 10.4 million people (90% adults; 65% male; 10% people living with HIV) fell ill with TB in 2016 (i.e. were incident cases).
- TB is a leading killer of people who are HIV infected.
- More than 90 % of TB cases and deaths occur in developing countries and 75 % of cases are in the most economically productive age group.
- Ethiopia has the seventh highest tuberculosis (TB) burden and one of the highest TB mortality rates in the world.

cont. …

- Incubation period- 4-12 weeks
- Period of communicability- as far as the bacilli is present in the sputum
- Susceptibility and resistance- under 3 years old children, adolescents, young adults, the very old and the immune suppressed are susceptible. Every one non-infected or non-vaccinated can be infected.

Etiology

- Mycobacterium tuberculosis- human tubercle bacilli (commonest cause)
- Mycobacterium bovis - cattle and man infection
- Mycobacterium avium- infection in birds & man
- Mycobacterium africanum

Transmission

- TB spreads from person to person by airborne transmission, droplet nuclei (usually particles 1 to 5 mm in diameter) released through talking, coughing, sneezing, laughing, or singing.
- Larger droplets settle; smaller droplets remain suspended in the air and are inhaled by a susceptible person.
- Consumption of raw milk containing M. bovis.

cont.

- ***Factors contributing for better transmission***
  - Closeness of contact
  - Poor ventilation
  - Duration of exposure
  - Bacteriological status of patient

Risk Factors

- Close contact with someone who has active TB
- Immunocompromised status (HIV infection, cancer, transplanted organs, and prolonged high dose corticosteroid therapy)
- Substance abuse (IV/injection drug users and alcoholics, drugs, tobacco)
- Pre-existing medical conditions or special treatment (diabetes, chronic renal failure,

Risk factors …

- Inadequate health care (the homeless; impoverished; minorities, particularly children under age 5 year and young adults between ages 15 and 44 year)
- Institutionalization (long-term care facilities, psychiatric institutions, prisons)
- Living in overcrowded, substandard housing
- Being a health care worker performing high-risk activities: sputum induction procedures, bronchoscopy, suctioning, coughing procedures

Natural history of TB

- Majority (90-95%) of persons got infected with MTB, but the immunological defense either kills or suppresses the inhaled bacteria causing *latent* MTB infection.
- Only about 5-10% of such persons (with primary infection) develop *active TB*.
- This reactivation leads to active TB which primarily affects the lung but can involve any other parts of the body.

Natural hx TB

- If untreated TB leads to deaths within 2-3 years in at least half of the patients.
- Without treatment about 20-25% would have natural healing and 25-30% would remain chronically ill, thus continuing to spread the diseases in the community.

Difference between latent TB infection &Active diseases

Characteristics	Latent TB infection	Active TB disease
Patient is sick	NO	YES
M.tuberculosis	present	present
Able to infect others	NO	YES
Sputum +ve for AFB	NO	YES/NO
Diagnosis	Tuberculin skin test	AFB,X-ray,etc
Treatment	INH 6 months	Multiple meds 6-8 months

Cont..

Reservoir

- human and cattle

Mode of transmission

- **Mainly airborne**
- Direct projection
- Un-boiled milk (M. bovis)
- Congenitally

pathogenesis of TB

- Primary infection occurs in persons without previous exposure to tubercle bacilli.
- Pulmonary infection occurs when TB bacilli reaches a terminal airway and succeeds in establishing infection.
- A localized granulomatous inflammatory process occurs within the lung, this is called the primary (Ghon) focus
- From the Ghon focus, bacilli drain via lymphatics to the regional lymph nodes.

cont..

- The Ghon focus with associated tuberculous lymphangitis and involvement of the regional lymph nodes is called the primary (Ghon) complex. The primary complex is asymptomatic
- From the regional lymph nodes bacilli enter the systemic circulation directly or via the lymphatic duct and hematogenous spread occurs.
- After dissemination, bacilli may survive in target organs for prolonged periods.
- The future course of the disease at each of these sites depends on the dynamic balance between host immunity and the pathogen.

Classification of tuberculosis

Pulmonary TB (80%)

- *Smear positive(75-80%)*
- *smear negative(20_25%)*

Extra pulmonary TB(20%)

- Lymph node
- Pleura
- Genitourinary tract
- Bone and joint
- Mening's
- Peritoneum and
- pericardium

Cont..

- ***Clinical Manifestations***

➢ ***General symptoms***

Fever

Night sweating

Chest pain

Weight loss

Fatigue

loss appetite/anorexia

➢ ***Pulmonary symptoms***

- Cough (usually productive)
- Chest pain
- Dyspnea
- Hemoptysis

TB HIV co-infection

Risk of active TB development is 50% in HIV positives.

Effect of HIV on TB

✓HIV is the strongest risk factor for progression from latent to active TB (Reactivation)

✓Sero-prevalence among TB patients is 50%

✓Modifies the clinical, radiological and lab findings

✓Making the dx of TB difficult

cont.

Effect of TB on HIV

- ✓TB allow HIV to multiply more quickly, increasing the viral load.
- ✓This may result in fastened rate of HIV disease progression

Diagnosis

Clinical suspicion is very important for the diagnosis of tuberculosis.

Patients who have suggestive symptoms and signs for tuberculosis should undergo further tests

1. **AFB Microscopy**

   Three sputum specimens: spot,morning,spot

2. **Mycobaterial culture(z gold standard)**

   Lowenstein-jensen media, middle-brook media

   Used for surveillance of MDR TB

Diagnosis..

3. Radiography

- Cavities in the upper third or in the apical segment of the lower lobe
- Unilateral or bilateral pleural effusion
- Miliary pattern
- Helpful in the diagnosis of smear negative pulmonary TB or childhood TB when interpreted with clinical features.

4.Histological examination

Important in the dx of EPTB

Disease classification-pulmonary TB

1.Smear positive pulmonary TB

- A patient with at least two initial smear examination positive for AFB

OR

- A patient with one initial smear positive for AFB and culture positive or X ray abnormalities suggestive of active TB as determined by a physician.
- In the current case definitions recommended by WHO, one positive result is required for a diagnosis of smear-positive pulmonary TB

2. Smear negative pulmonary TB

- A patient with 3 initial smear examination negative for AFB and who has failed to respond to a broad spectrum antibiotics and X ray abnormalities suggestive of active TB determined by the physician. **OR**
- A patient with 3 initial smear examination negative by direct microscopy but positive by culture.

Case definition

- Discuss in Groups the following terms
- New case
- Relapse
- Lost to follow up
- Treatment failure
- Transfer in(T)
- Others (O)

Management of TB

Management principles

- Chemotherapy/anti TB drugs
- Nutritional rehabilitation
- Screening of the family (other contacts)
- Follow up (adherence, response, drug side effect)

Treatment of TB

- ***Objectives***
  - ❑ To cure the TB patient and restore quality of life and productivity
  - ❑ To prevent death from active TB or its late effects
  - ❑ To prevent relapse (by eliminating the dormant bacilli)
  - ❑ To prevent the development of drug resistance (by using a combination of drugs)
  - ❑ Decrease TB transmission to others

Anti TB drugs

First Line Drugs (Essential Anti tuberculosis drugs): There are five drugs in use currently	Second Line Drugs (reserve Anti TB drugs) are used only in Chronic case of TB
•Streptomycin (S) • Ethambutol (E) • Isoniazeid (H) • Rifampicin (R) • Pyrazinamide (Z)	•Kanamycin • Amikacin •Capreomycin •Ethionamide •Protionamide •Ofloxacin •Ciprofloxacin

Treatment of TB

- **The intensive (initial) phase**: - DOTS
- Combination of 3 or more drugs is given for 2months.
- In the re treatment regimen it continued for 3 months.
- This is to decrease the bacterial load and make the patient non-infectious rapidly.

Treatment of TB

Continuation phase:-

- Two or three drugs used for 4 -5 months.
- This phase follows the intensive phase and the aim is to achieve complete cure.
- the drugs must be collected every month and self-administered by the patient.

prevention and Control

1. Chemotherapy of cases
2. Chemoprophylaxis for contacts

INH (Isoniazid) for adults and children who have close contact with the source of infection

3. Immunization of infants with BCG

prevention and Control

4. Health Education

the modes of disease transmission and

- ✓ methods of control
- ✓ Improved standard of living
- ✓ Adequate nutrition
- ✓ Health housing

Infection Control and Prevention of TB

Prevention efforts focus on the following three goals:

- ✓ Primary prevention – preventing TB infection
- ✓ Secondary prevention – preventing TB disease
- ✓ Tertiary prevention – preventing TB morbidity and mortality

Hierarchy of Infection Prevention & Control

1. Administrative controls
   - ✓ Reduce risk of exposure, infection and disease through policy and practice
2. Environmental (engineering) controls
   - ✓ Reduce concentration of infectious bacilli in air in areas where air contamination is likely
3. Personal respiratory protection
   - ✓ Protect personnel who must work in environments with contaminated air

Administrative Controls

- Develop and implement written policies and protocols to ensure:
  - ✓ Rapid identification of TB cases (e.g., improving the turn-around time for obtaining sputum results)
  - ✓ Isolation of patients with PTB
  - ✓ Rapid diagnostic evaluation
  - ✓ Rapid initiation treatment
- Educate, train, and counsel HCWs about TB
- To the extent possible, avoid mixing TB patients and HIV patients in the hospital or clinic setting

Environmental Controls: Ventilation and Air Flow

- It reduces the conc. Of infectious respiratory aerosol in the air.
- Types
  - Natural(open window &door to capture z wind)
  - Local(strategically placed fans)
  - General(centeralised air conditioning system)
- Simple measures can be effective

Personal Respiratory Protection

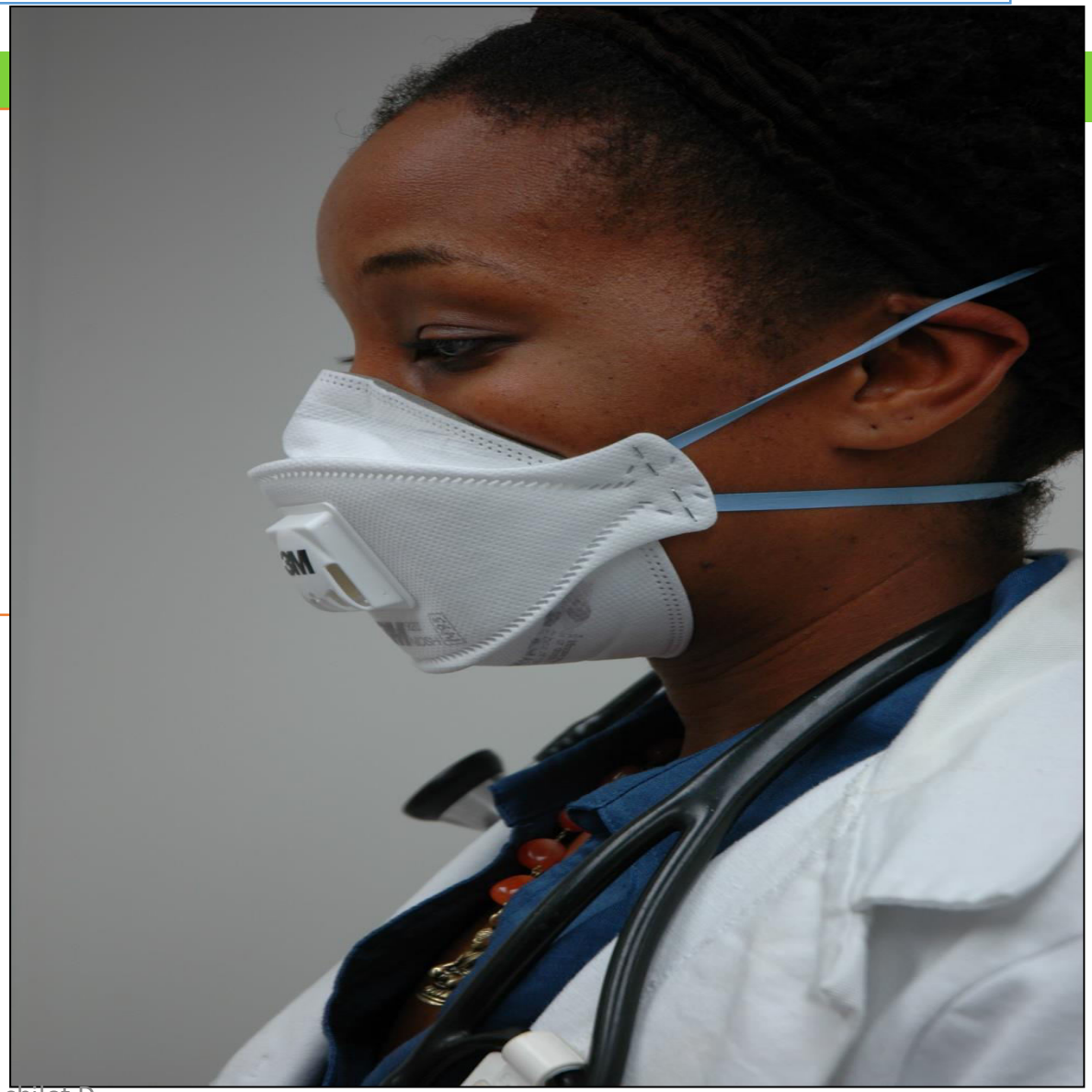
- Respirators: has only tiny pores relies on an air tight seal around the entire edge and it protects inhalation
  - Can protect HCWs
  - Should be encouraged in high-risk settings
  - May be unavailable in low-resource settings
- Face/surgical masks: has large pores and lacks air tight seal around edges
  - Act as a barrier to prevent infectious patients from expelling droplets
  - Do not protect against inhalation of microscopic TB particles.

Cont..

DO

Be sure your respirator is properly fitted!

It should fit snugly at nose and chin



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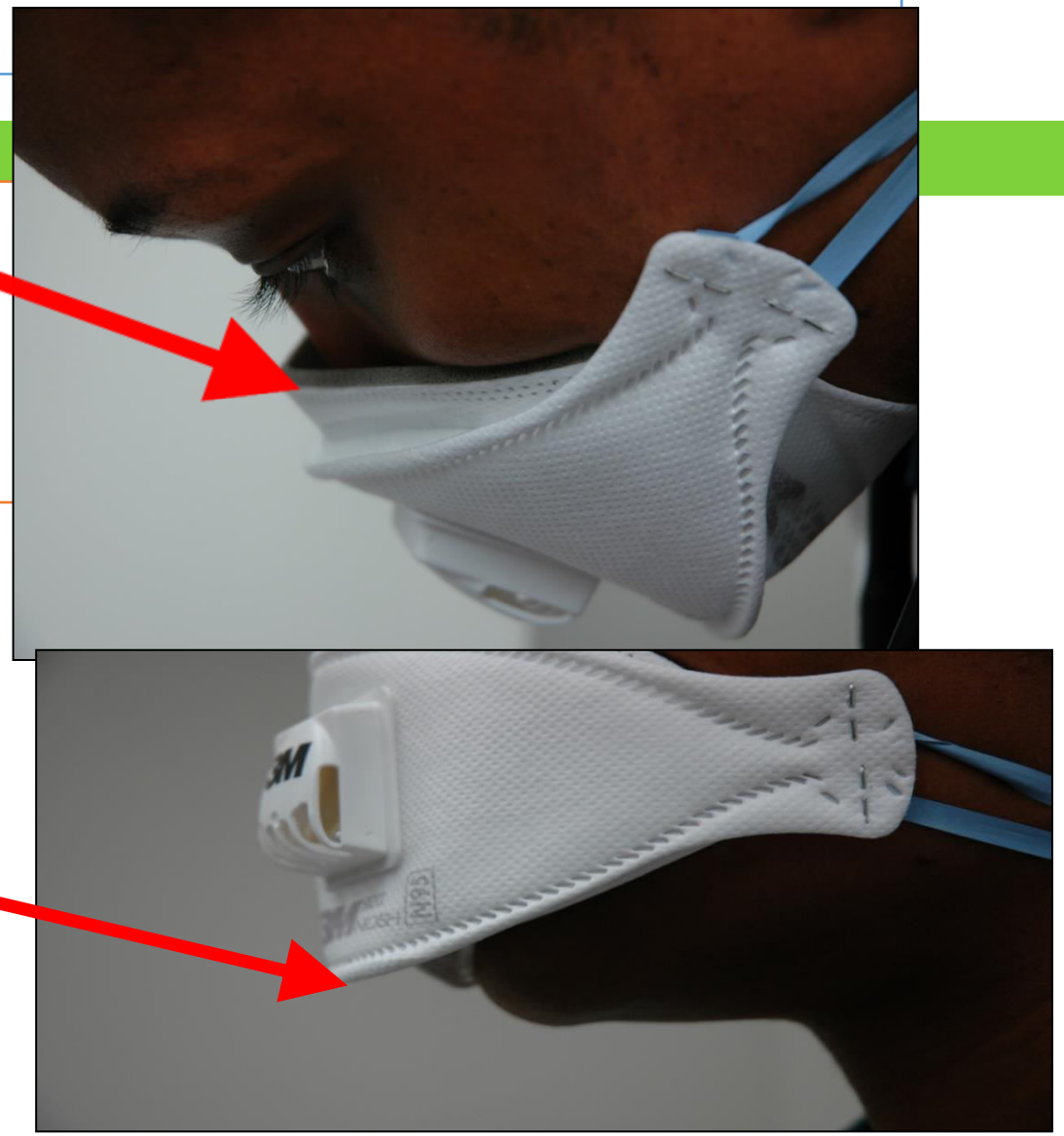
- Note poor fit at the bridge of nose
- Note poor fit at the chin
- Respirator should cover chin and create a seal



Cont..

- Don’t Forget to WEAR It!

TB Prevention & Control in the Community:

Community Role

- Recognize the early symptoms of TB
- Minimize crowded living conditions
- Allow natural light into buildings and rooms as ultra-violet rays quickly kill TB bacilli
- Open windows to air out rooms to dilute the load of infectious TB bacilli

Patient Role

- Patient should maintain a well-balanced diet to keep the immune system strong
- TB patient should stop smoking and minimize intake of alcohol
- Patient should hold a cloth or handkerchief over mouth when coughing
- Patient should not spit on the floor but in a container (preferably disposable) and dispose of properly

Infection Prevention & Control in the Workplace

- Provide a well-ventilated, sun-light environment
- Educate all staff on TB transmission & prevention
- Link with health facilities for treatment & support

TB Prevention in Special Settings

Prisons and Police Holding Cells

- Screen all prisoners
- Treat & isolate
- Implement strict DOT during entire treatment
- Refer all released prisoners under treatment to nearest healthcare facility

TB Prevention & Control Among HIV+ Patients and HCWs

- Immunosuppressed persons are much more susceptible to TB and therefore should not be housed with inpatients who have undiagnosed cough or untreated TB.
- Encourage patients to know their HIV status so they can reduce their exposure to TB infection

Contact Tracing

- The identification and diagnosis of persons who may have come into contact with an infected person.
- An important element to infection prevention and control.

TB Screening Among Contacts

- Basic screening for TB done in home by HEW or nurse
- Refer the following individuals to clinic for further evaluation and follow-up (evaluation for active TB and evaluation for INH prophylaxis or IPT):
  - Children in household < 5 years old
  - Persons in household who are HIV+
  - Persons in household who are ill

Key Points

- Prevention efforts should focus on primary, secondary, and tertiary prevention
- Attention to the potential spread of infection and disease among special populations, including among those who are HIV+ is crucial
- Contact tracing is an important component of TB control in the community

MEASLES(Rubeola)

- Measles (rubella) is among the leading causes of child morbidity and mortality worldwide. Despite remarkable progress in the control of measles, measles still continues to claim the lives of millions of children every year around the world.
- It is an acute highly communicable viral disease.

  Infectious agent: Measles virus

Cont…..

- Occurrence- Prior wide spread immunization, measles was common in childhood so that greater than 90% of people had been infected by age of 20, few went through life with out any attack.

Cont..

- **Reservoir**- Humans
- **Mode of transmission**- Air borne by droplet spread, direct contact with nasal or throat secretions of infected persons and less commonly by articles freshly solid with nose and throat secretion. Greater than 94% herd immunity may be needed to interrupt community transmission.
- **Incubation period**- 7-18 days from exposure to onset of fever.

Cont..

Measles consists of 4 phases

- Incubation period
- Prodromal illness
- Exanthematous phase
- Recovery

Prodromal phase

- prodromal phase

- ✓ begins at the onset of the first symptoms, which begin gradually and include a fever, a cough, a runny nose, and red eyes.
- ✓ Usually, the fever is the first symptom noticed by parents. The fever rises steadily and may reach maximum temperatures of 103F to 104F.
- ✓ At the height of the fever, the rash develops.
- ✓ The runny nose with a profuse watery discharge, nasal congestion, and sneezing becomes prominent.

The rash phase

Two to four days after the onset of the symptoms,

- The rash appears, marking the beginning of the rash phase.
- The symptoms of the prodromal phase worsen with the onset of the rash, but then begin to decrease in severity.
- The measles rash is a flat or slightly raised rash, and is not itchy.

The rash phase

- It first appears as irregular spots on the upper forehead or behind the ears and on the neck.
- Within 24 hours, it progresses to the entire face, head, and neck.
- Over the next two to four days, the rash extends to the chest, back, and extremities, including the palms of the hands and the soles of the feet.
- It remains most prominent on the face, especially on the cheeks.

Recovery phase

- After four to five days, the rash begins to subside, marking the beginning of the recovery phase.
- Sometimes, a very fine flaking of the skin is noted as the rash fades.
- About 10 to 14 days after developing the rash, the child is back to a normal level of activity.

Risk factors

- Young age
- Malnutrition
- Lack of immunization
- Low maternal immunity

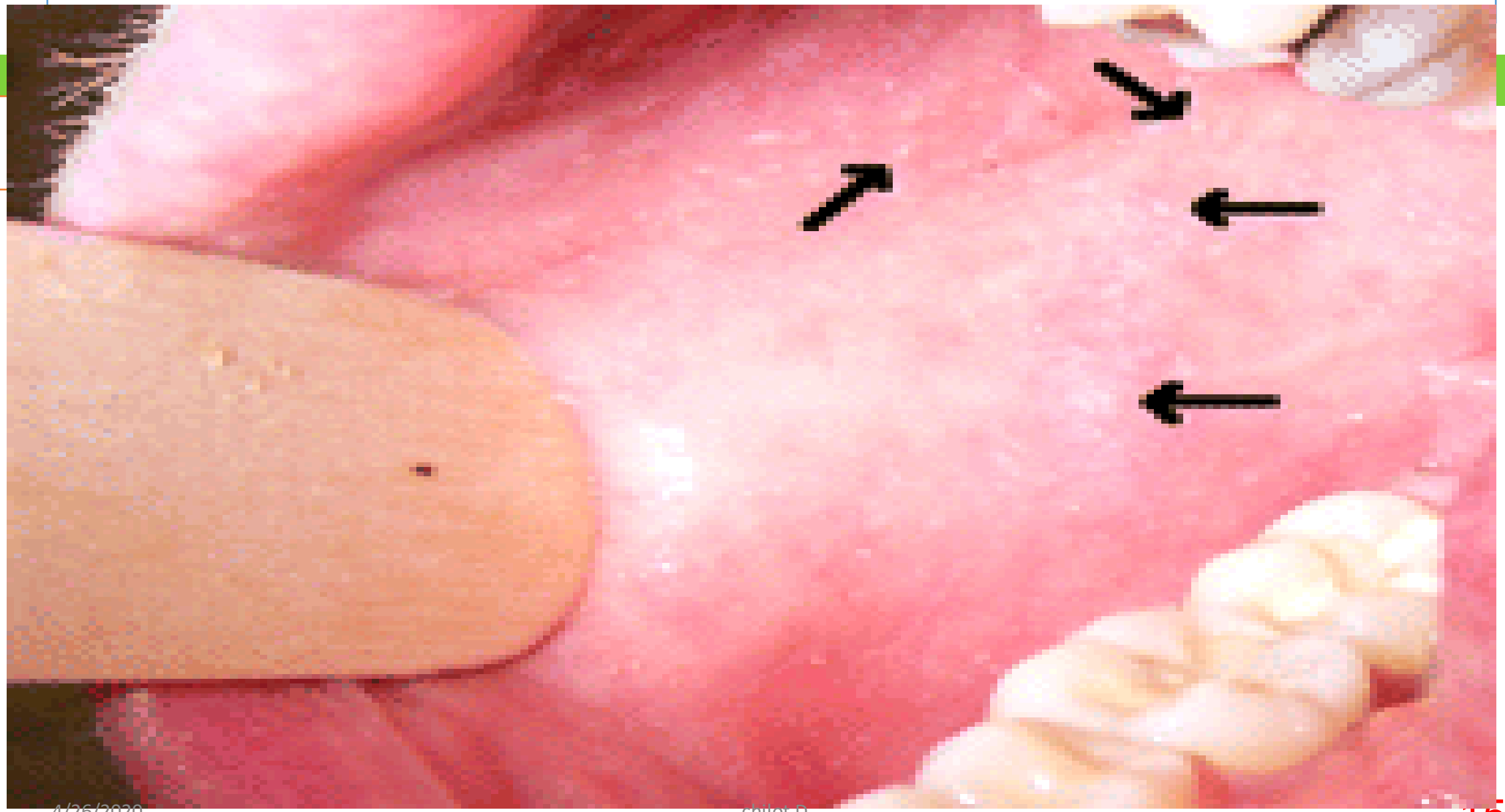
Clinical presentation

- Measles is a serious infection characterized by high fever, an enanthem, cough, coryza, conjunctivitis, and a prominent exanthema.
- After an incubation period of 8–12 days, the prodromal phase begins with a mild fever followed by the onset of conjunctivitis with photophobia, coryza, a prominent cough and increasing fever
- The onset of the disease is characterized by symptoms of the initial catarrhal (prodromal) phase that usually lasts 3-5 days and is characterized by:

Cont….

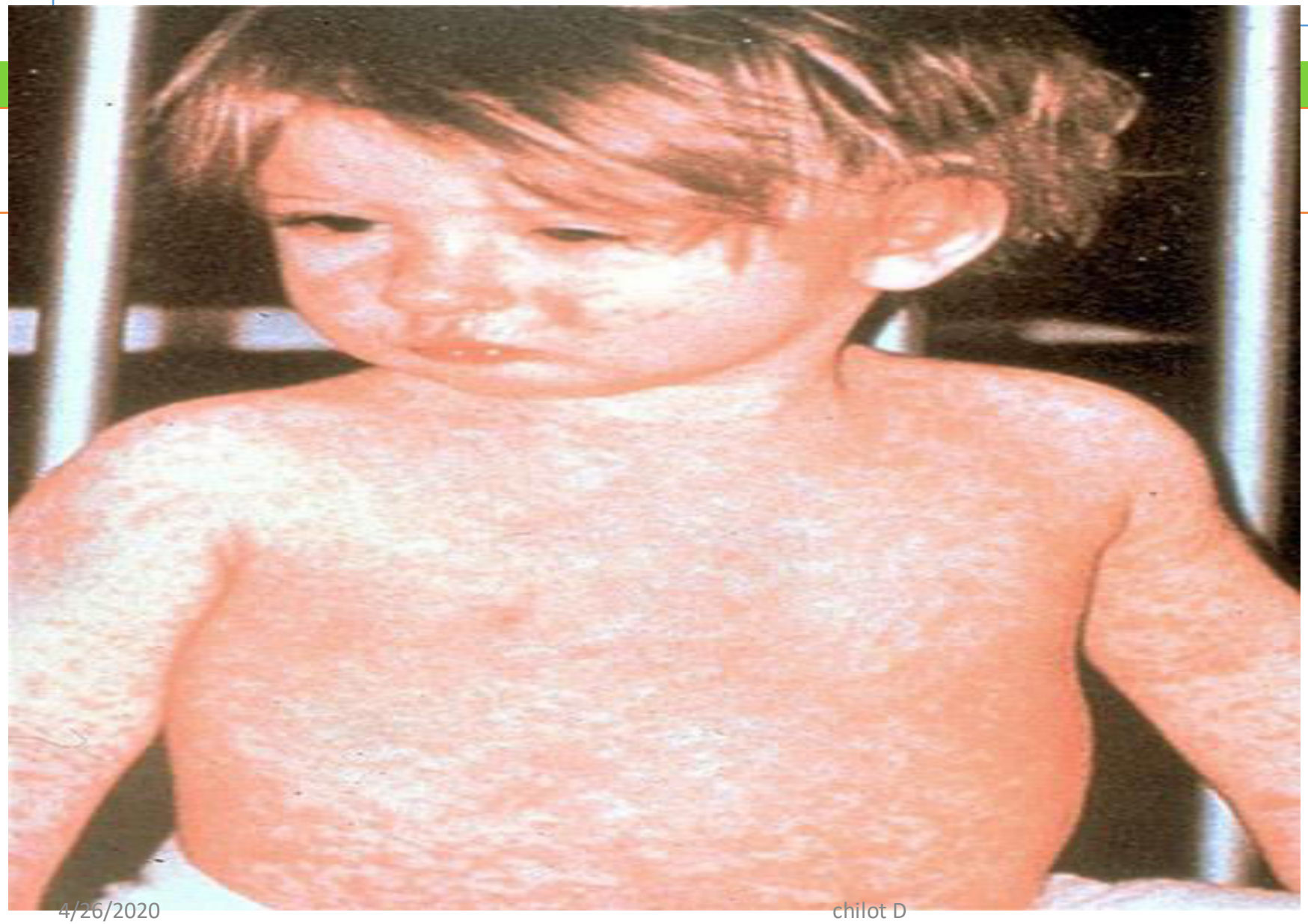
- Low or moderate fever
- Red eyes/lacrimation
- Runny nose/ Coryza
- Cough
- These symptoms nearly always precede the appearance of koplik Spots

❖ Headache, abdominal pain, vomiting, diarrhea, and myalgia may be present.

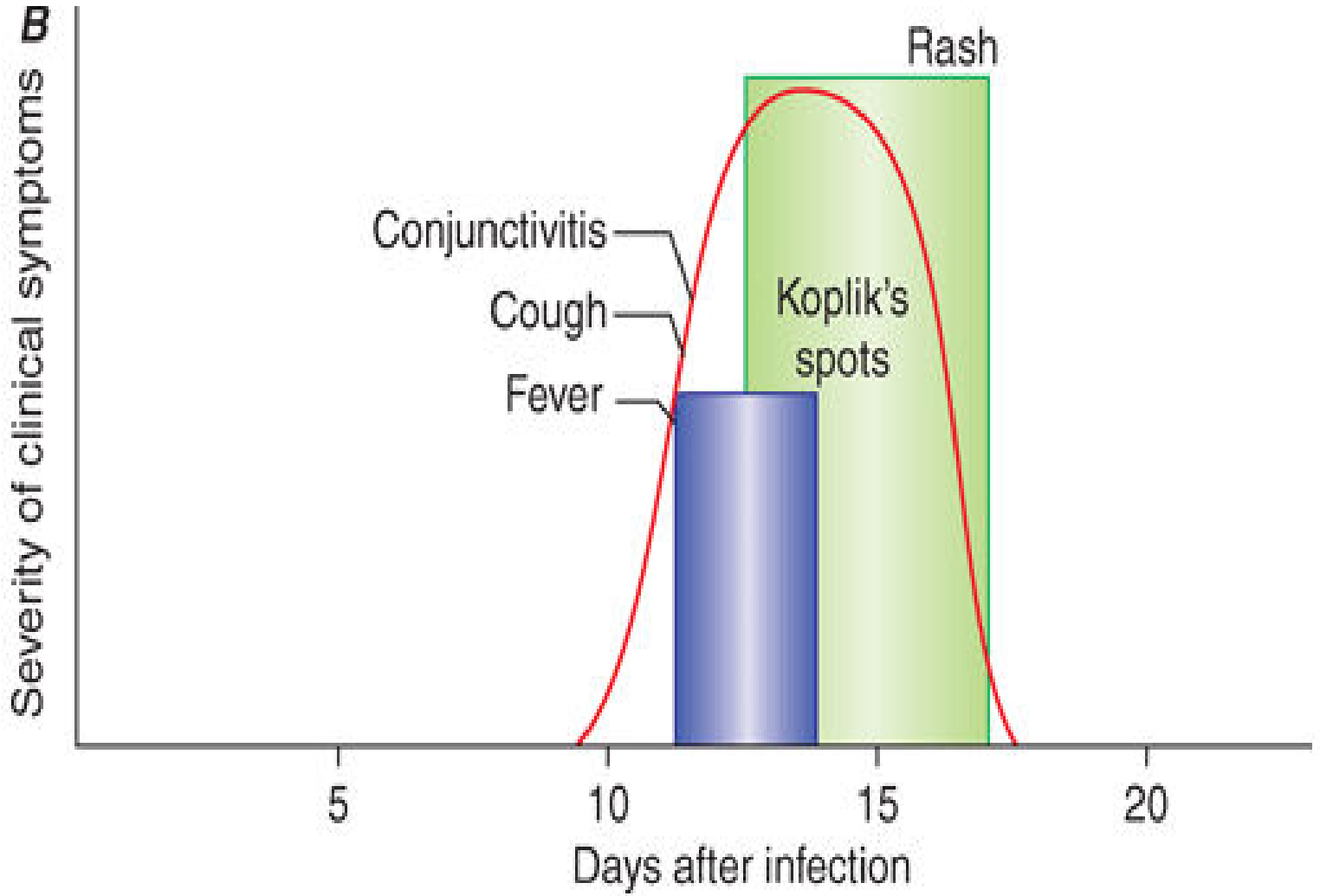


cont..

- Reddish (erythematous), maculopapular rash typically occurs in cephalocaudal (top-bottom) progression.
- The skin rash appears by the third day after the onset of fever, cough & coryza.
- The fever classically rises, often reaching 40oC,with the appearance of the rash.
- The rash usually starts as faint macules on the upper lateral parts of the neck, behind the ears, along the hairline, and on the posterior parts of the neck



Clinical pattern



Diagnosis

- The diagnosis of measles is almost always based on clinical and epidemiologic findings.
- Laboratory findings in the acute phase include reduction in the total white blood cell count, with lymphocytes decreased more than neutrophils.
- Absolute neutropenia has been known to occur, however.
- In measles not complicated by bacterial infection, the erythrocyte sedimentation rate and C-reactive protein levels are normal

cont..

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cont..

- In the absence of a recognized measles outbreak, confirmation of the clinical diagnosis is often recommended.
- Serologic confirmation is most conveniently made by identification of immunoglobulin M (IgM) antibody in serum.
- IgM antibody appears 1–2 days after the onset of the rash and remains detectable for about 1 mo.
- If a serum specimen is collected <72 hours following onset of rash and is negative for measles antibody, a repeat specimen should be obtained.

Complications of measles

- Approximately 30% of reported measles cases have one or more complications.
- The most common complications that occur are:
- Vit A deficiency
- Gastroenteritis
- Pneumonia
- Otitis media
- Encephalitis
- Different infections

Treatment

- Properly organized hygienic conditions for the patient
- Careful nursing care
- Protection from secondary infection
- Continuous feeding giving more fluids than usual
- Control fever
- Watching (actively anticipating for) complications

Treatment

- Measles cases are hospitalized when:
- They have severe and complicated measles.
- Unsatisfactory home condition or not possible to arrange proper Nursing care.

Treatment is mainly symptomatic and supportive:

- Antipyretics (acetaminophen) for fever
- Bed rest
- Maintenance of an adequate fluid intake
- Keep the room comfortably warm

Treatment

- Vit A provision

Prevention and control

- Educate the public about measles immunization
- Immunization of all children (less than 5 years of age) who had contact with infected children
- Provision of measles vaccine at nine month
- Initiate measles vaccination at 6 months of age during epidemic and repeat at 9 month of age

Quiz

- 1. write three main control mechanism of CDC
- 2. write at a least four factors for the transmission of TB
- 3. write at least three complications of measles
- 4. Define Treatment failure in Tuberclusis

- Pertussis, Diphtheria, Meningitis and leprosy

Pertussis

Learning objectives

- Define pertussis
- Differentiate the clinical manifestation of pertussis from other air born disease
- Describe the diagnosis modalities of pertussis
- Write the treatment approaches of pertussis and Nursing intervention

Pertussis

Group discussion (5 minute)

- 1. What is pertussis
- 2. How to differentiate pertussis from other air born disease

Pertussis(Whooping Cough)

- An acute bacterial disease involving the respiratory tract.
- Infectious agent
  - ✓ Bordetella pertusis
- Epidemiology
  - ✓ An endemic disease, common in young children everywhere in the world.
- Reservoir: Humans

Mode of transmission of Pertussis(Whooping Cough)

- Primarily by direct contact with discharges from respiratory mucus membranes of infected persons by airborne route, by droplets.
- Indirectly by handling objects freshly soiled with nasopharyngeal secretions.

Incubation period

- 1-3 weeks
- The most contagious disease with an attack rate of 75-90%
- One attack usually confers prolonged immunity but may not be lifelong.

Clinical manifestation

The disease has insidious onset and 3 phases

1. Catarrhal phase

- Lasts 1-2 weeks
- Cough and rhinorrhea, sneezing, low-grad fever and similar with common cold .

2.paroxysmal phase

- Explosive, repetitive and prolonged cough
- Child usually vomits at the end of paroxysm
- Whoop (inspiratory whoop against closed glottis) between paroxysms.
- Child looks healthy between paroxysms
- Paroxysm of cough interferes with nutrition
- Cyanosis and sub conjunctiva hemorrhage due to violent cough.

3. Convalescent phase

- The cough may diminish slowly or may last long time.
- After improvement the disease may recur

Diagnosis

- History and physical examination
- Antibody test
- CBC -Marked lymphocytosis.
- Culture –isolation of B. pertussis

Treatment

- Erythromycin
- Prevention
- Active immunization –DPT
- Health education
- Isolation :exclude suspected case from the presence of young children and infant
- Disinfection

prevention and control

- Educate the public about the dangers of whooping cough and the advantages of initiating immunization at 6 weeks of age.
- Consider protection of health workers at high risk of exposure by using erythromycin for 14 days.

Diphtheria

Learning objectives

- Define diphtheria
- Differentiate the clinical manifestation of diphteia from other air born disease
- Describe the diagnosis modalities of diphtheria
- Write the treatment approaches of diphtheria
- Write prevention and control mechanism of Diphteria

Diphtheria

- What is diphtheria ?
- It is an acute bacterial disease involving primarily tonsils, pharynx, nose, occasionally other mucus membranes or skin and sometimes the conjunctiva or genitalia.

Infectious agent

- Corynebacterium diphtheria

Epidemiology

- Occurrence- Disease of colder months in temperate zones, involving primarily non-immunized children under 15 years of age. It is often found among adult population groups whose immunization was neglected. Inapparent, cutaneous and wound diphtheria cases are much more common in the tropics.
- Reservoir- Humans
- Mode of transmission- contact with a patient or carrier. i.e. with oral or nasal secretions or infected skin.

cont..

- Asymptomatic respiratory tract carriage is important in transmission. Where diphtheria is endemic, 3-5% of healthy individuals can carry toxigenic organisms
- Incubation period- usually 2-5 days
- Period of communicability- variable, until virulent bacilli have disappeared from discharges and lesion, usually 2 weeks or less.

cont..

- ❖ Susceptibility and resistance- Susceptibility is universal.
- ✓ Infants borne to immune mothers are relatively immune,
- ✓ protection is passive and usually lost before 6 months.
- ✓ Recovery from clinical disease is not always followed by lasting immunity.
- ✓ Immunity is often acquired through inapparent infection.
- ✓ Prolonged active immunity can be induced by diphtheria toxoid.

cont..

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Clinical manifestations

- ❖ Influenced by the anatomic site of infection,
- ❖ the immune status of the host and
- ❖ the production and systemic distribution of toxin
- ✓ Unilateral nasal discharge is quite pathognomic of nasal diphtheria
- ✓ The presence of a pharyngeal pseudomembrane or an extensive exudate should prompt consideration of diphtheria

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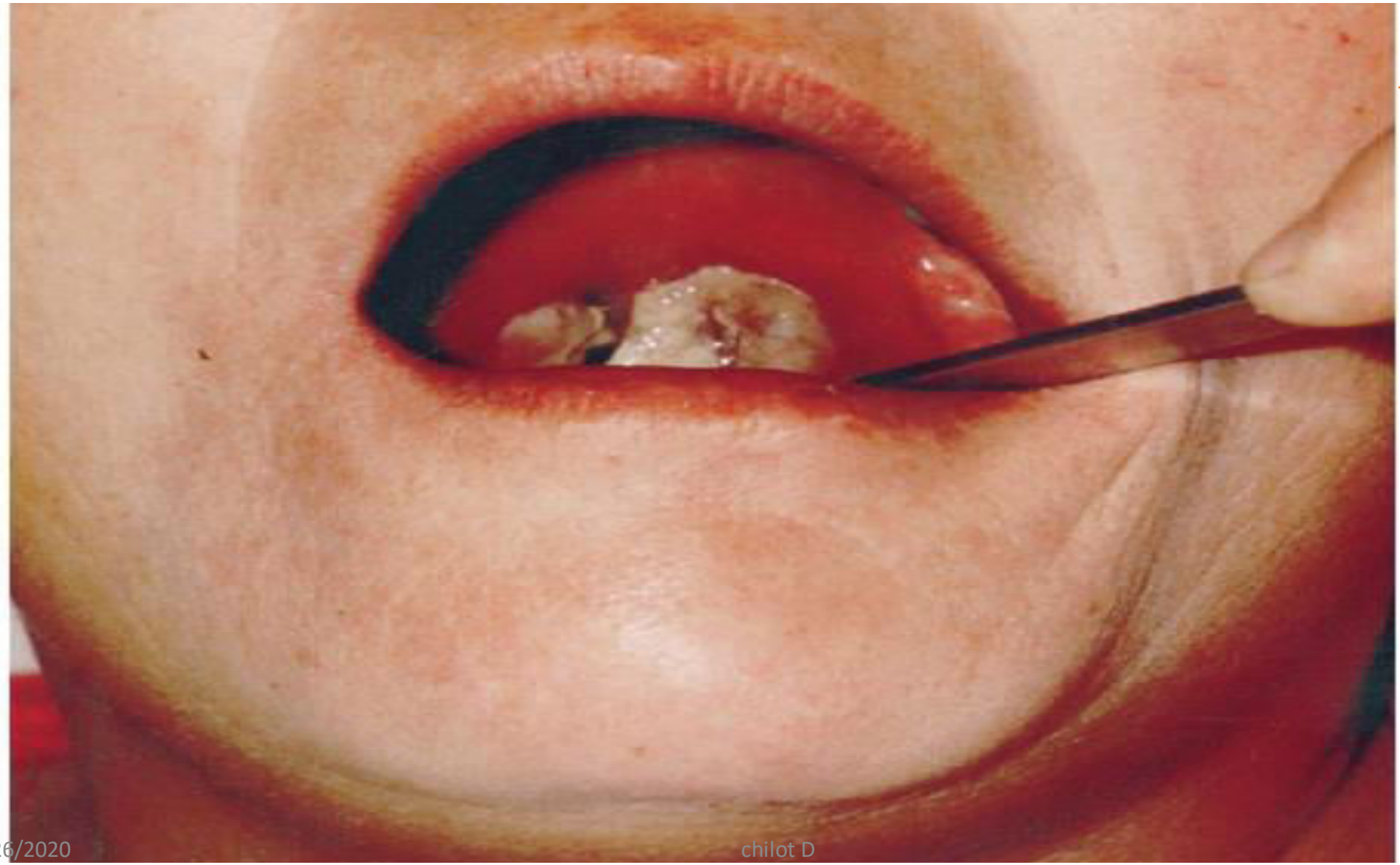
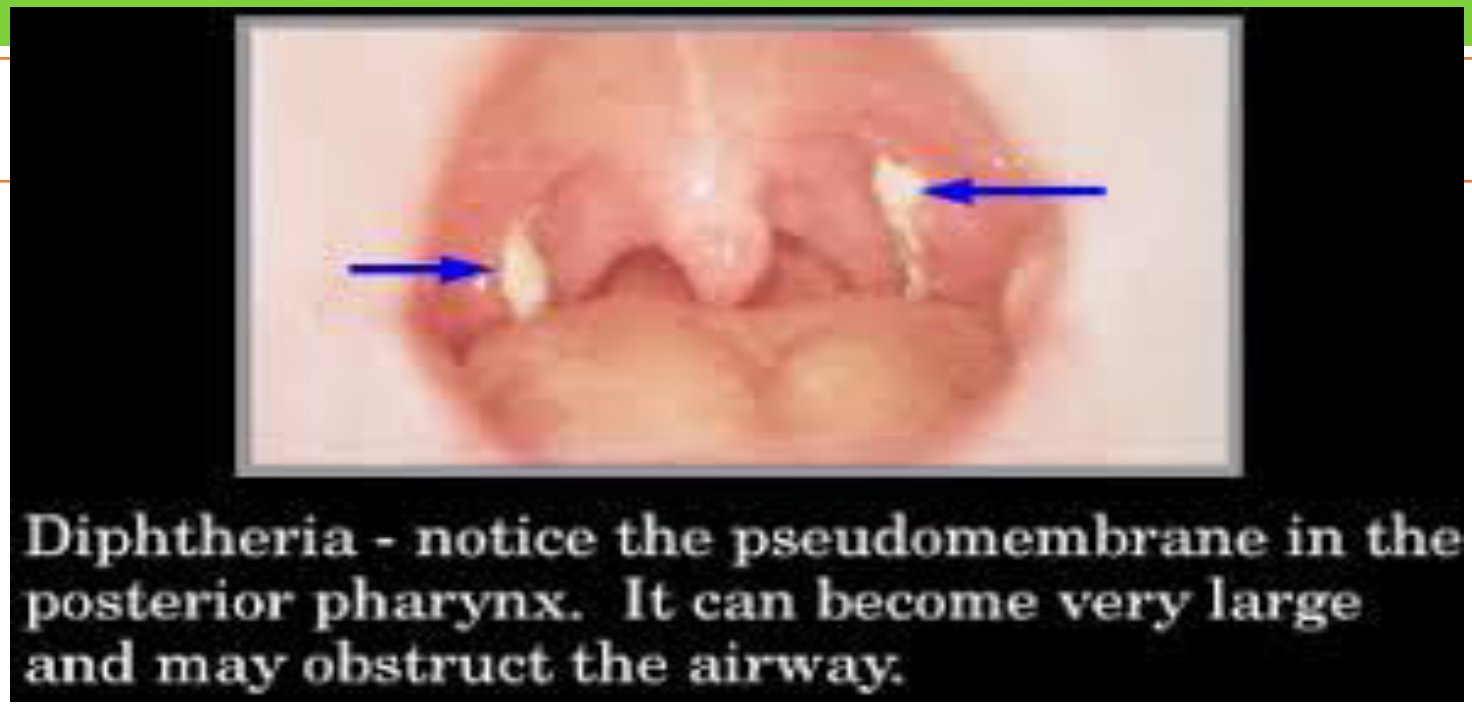


Clinical manifestations

sore throat is the universal early symptom

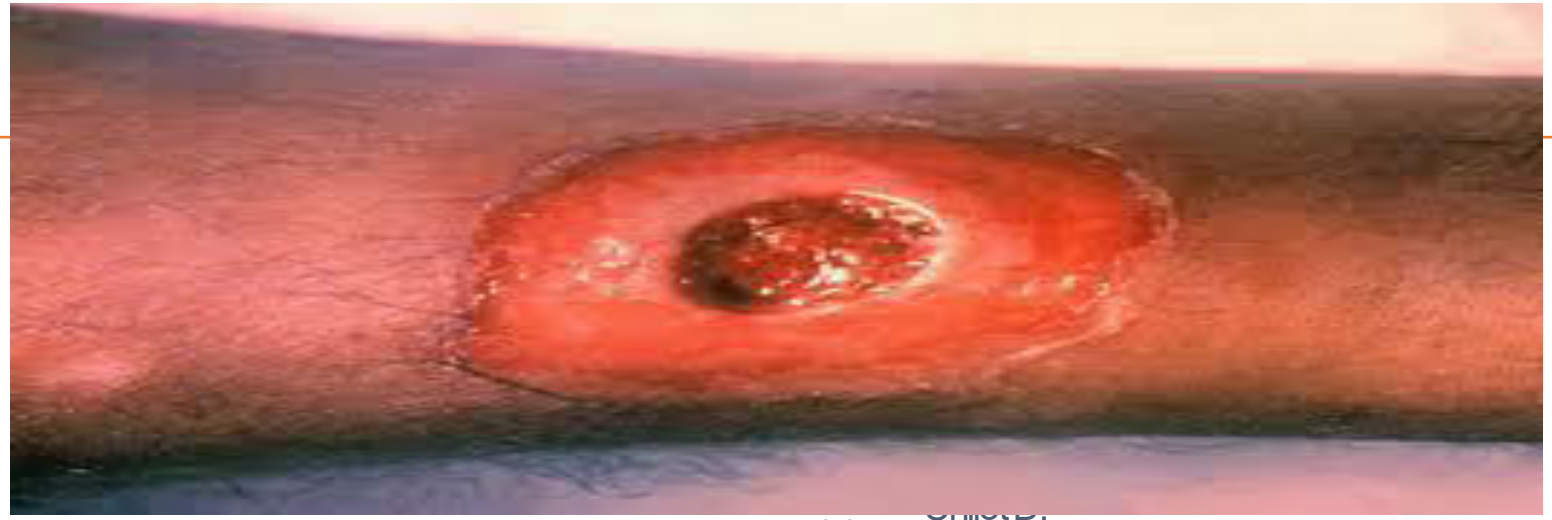
- Only half of patients have fever and fewer have dysphagia, hoarseness, malaise, or headache
- Mild pharyngeal injection ⟹ unilateral or bilateral tonsillar membrane formation ⟹ extend to involve the uvula, soft palate, posterior oropharynx, hypopharynx, or glottic areas
- Underlying soft tissue edema and enlarged lymph nodes: ***bull-neck appearance***

Clinical manifestations



Clinical manifestations

- Laryngeal diphtheria: at significant risk for suffocation because of local soft tissue edema and airway obstruction by the diphtheritic membrane
- Classic cutaneous diphtheria is an indolent, nonprogressive infection characterized by a superficial, ecthymic, nonhealing ulcer with a gray-brown membrane



Diagnosis

- Clinical features
- Culture: from the nose and throat and any other mucocutaneous lesion
- Hypoglycemia, glycosuria, BUN, or abnormal ECG for liver, kidney and heart involvement

Treatment

- Diphtheria antitoxin
- Erythromycin for 2 weeks but 1 week for cutaneous form or
- Procaine penicillin for 14 days or single dose of Benzathin penicillin

N:B Primary goal of antibiotic therapy for patients or carriers is to eradicate C.diphtheriae and prevent transmission from the patient to susceptible contacts.

Prevention and Control

- Educate the public, and particularly the parents of young children, of the hazards of diphtheria and the necessity for active immunization
- Immunization of infants with diphtheria toxoid
- Concurrent and terminal disinfection of articles in contact with patient and soiled by discharges of patient
- Single dose of penicillin (IM) or 7-10 days course of Erythromycin (PO) is recommended for all persons exposed to diphtheria

Meningitis

Learning objectives

- Define Meningitis
- Differentiate the signs of Meningitis
- Describe the diagnosis modalities of meningitis
- Write the treatment approaches of meningitis
- Write nursing management of meningitis

Pair discussion questions

- What is meningitis
- Discuss about the clinical manifestation and signs of meningitis
- List the predisposing factors of meningitis

Meningitis

- An inflammation of meninges /the pia and arachnoid membrane/ which cover the CNS.
- **It may be either:**
  - **Acute or chronic**
  - **Sterile or infective / aseptic or septic meningitis/**

Reservoir

- Humans

Infection agent, risk groups and predisposing conditions of meningitis

Causative organism	Age group most at risk	Predisposing condition
Pneumococci Streptococci	Adults	Mastoiditis, otitis media, sinusitis, pneumonia, head injury, puerperium, pregnancy
H. influenza	Children	Respiratory tract infection, otitis media, mastoditis
Salmonellae	<2 years	Diarrhea or septicemia
Meningococcus /N. meningitides/	Children, young adults	Overcrowding
M. tuberculosis	Children	Malnutrition
Virus	Children	Epidemics of mumps, measles, polio, chicken pox, and other viruses

Occurrence and importance

- Meningococcal meningitis can occur sporadically or in the form of epidemic outbreaks especially in crowded institutions such as barracks, camps, and prisons.
  - In the cold dry season, especially in the highlands, the temperatures drop to very low levels at night.
  - This makes people crowd together in badly ventilated rooms.
  - Their mucous membranes are also irritated by dust and by the smoke of firewood.
  - Under such conditions the meningococci spread easily.

cont..

- Other forms of meningitis always occur sporadically as they are usually complications of other diseases
- The acute forms of meningitis may be caused by several **viruses and bacteria**.
- Chronic meningitis may be caused by **Tb**.

cont..

- Aseptic or viral meningitis differs from bacterial meningitis because it is usually a self-limiting disease or associated with other clinical entities such as mumps, poliomyelitis, measles and others.
- All forms of meningitis can obstruct normal CSF flow and so cause **hydrocephalus**,
- and can damage cranial nerves proximally by adhesions which can result in paralysis or loss of senses (deafness, blindness).

Cont…

- If meningitis is properly treated, mortality should not exceed **5%**
- If treatment is delayed, mortality may be over **30%** and commonly those who do survive have permanent damage. .
- Meningitis should be differentiated from meningism or meningeal irritation as is seen for example in malaria, by doing a lumbar puncture.

Cont…

- **Meningism** is sign and symptom of meningitis associated with acute febrile illness or dehydration but without actual inflammation of the meninges

Meningitis ..

Reservoir

- 25 % of healthy people may carry meningococci and other organisms, therefore healthy carriers are common

Mode of transmission /for meningiococcus/

- Transmission occurs by direct contact and droplet spread of discharges from nose and throat of infected persons (mostly carriers).

Incubation period

- 2-10 days, commonly 3-4 days

Clinical Manifestation

- Sudden onset of fever , intense headache, nausea & often vomiting, neck stiffness and frequently a petechial rash with pink macules
- The headache becomes sever and spreads down the neck.
- There may be pain in the back and limbs.
- In children convulsions are common at the onset.
- The patient is irritable, becomes confused and drowsy, and later comatose.

Cont

- Signs of meningeal irritation are clear and present at an early stage: neck rigidity, kernig's sign and Brudzinski's signs are positive
- **Kerning's sign –**
  - ✓ patient feels back pain when one of the lower limbs is flexed at the knee joint & extended forward in an elevated position.
- **Brudinski's sign**
  - ✓ when the patient's neck is flexed; the two lower extremities get flexed or raised up.
- In babies the anterior fontanelle may bulge.

Diagnosis

- Based on clinical & epidemiological grounds
- Lumbar puncture must be done with out delay to confirm the diagnosis and start appropriate therapy immediately
- Increased CSF pressure.
- CSF gram stain and culture can be done to confirm the diagnosis

CSF analysis from LP

Disease	CSF	Cells	Protein	Glucose
Acute bacterial	Always turbid	Increased polymorphs	Raised	Low
Tb	Opalescent **/milky/** or clear	Lymphocytes increased	Raised	Low
Aseptic or viral	Opalescent or clear	Lymphocytes increased	Raised	Normal
Meningism	Clear	Normal	Normal	Normal

Treatment…

For commonly acquired etiology known

- N.meningitides, pneumococci and S. pneumonia
  - ➢ Benzyl penicillin 20-24miu /day iv 4-6x/day for 7-10 days
- H. influenza
  - ➢ Chloramphenicol 100mg /kg/day iv 4x/day for the first 48-72 hrs, then 50mg/kg /day 7-10 days

For resistant strains of N.meningitides, S. pneumonia and H. influenza

- ➢ Ceftriaxone IV 4gm/day 2x/day for 10-14 days

Nursing management

- Tepid sponging , paracetamol or ASA for fever
- Diazepam or phenobarbitone for convulsions
- Suction to keep air way clear
- NG tube with nasogastric feeds if the pt. is in coma
- Patient isolation is not recommended b/s spread mainly occurs by healthy carriers
- Maintains fluid balance (input & output)
- Timely administration of antibiotics
- Monitor vital sings

Prevention & control

- Educate the public on the need to reduce direct contact & exposure to droplet infection
- Reduce overcrowding in schools, camps, etc..
- Vaccines containing group *A,C,* and Y strains of meningococcus
- Chemotherapy of cases
- Chemoprophylaxis e.g. ciprofloxacine, rifampicin
- Report to the concerned health authorities

Case study

A 7- year old male child presented with fever of 3 days' duration associated with headache, neck stiffness, vomiting and loss of consciousness. CSF analysis finding shows leukocytosis with neutrophil predominance and increased protein (500mg/dl). What is the most likely diagnosis?

A. tuberculosis meningitis B. Chemical meningitis

C. Viral meningitis D. Bacterial meningitis

LEPROSY (HANSEN'S DISEASE)

- It is a chronic bacterial disease of the skin, peripheral nerves, eyes and in lepromatous patients the upper air way

Infectious agent

- Mycobacterium leprae
- It is acid fast bacilli (AFB)

Occurrence and importance

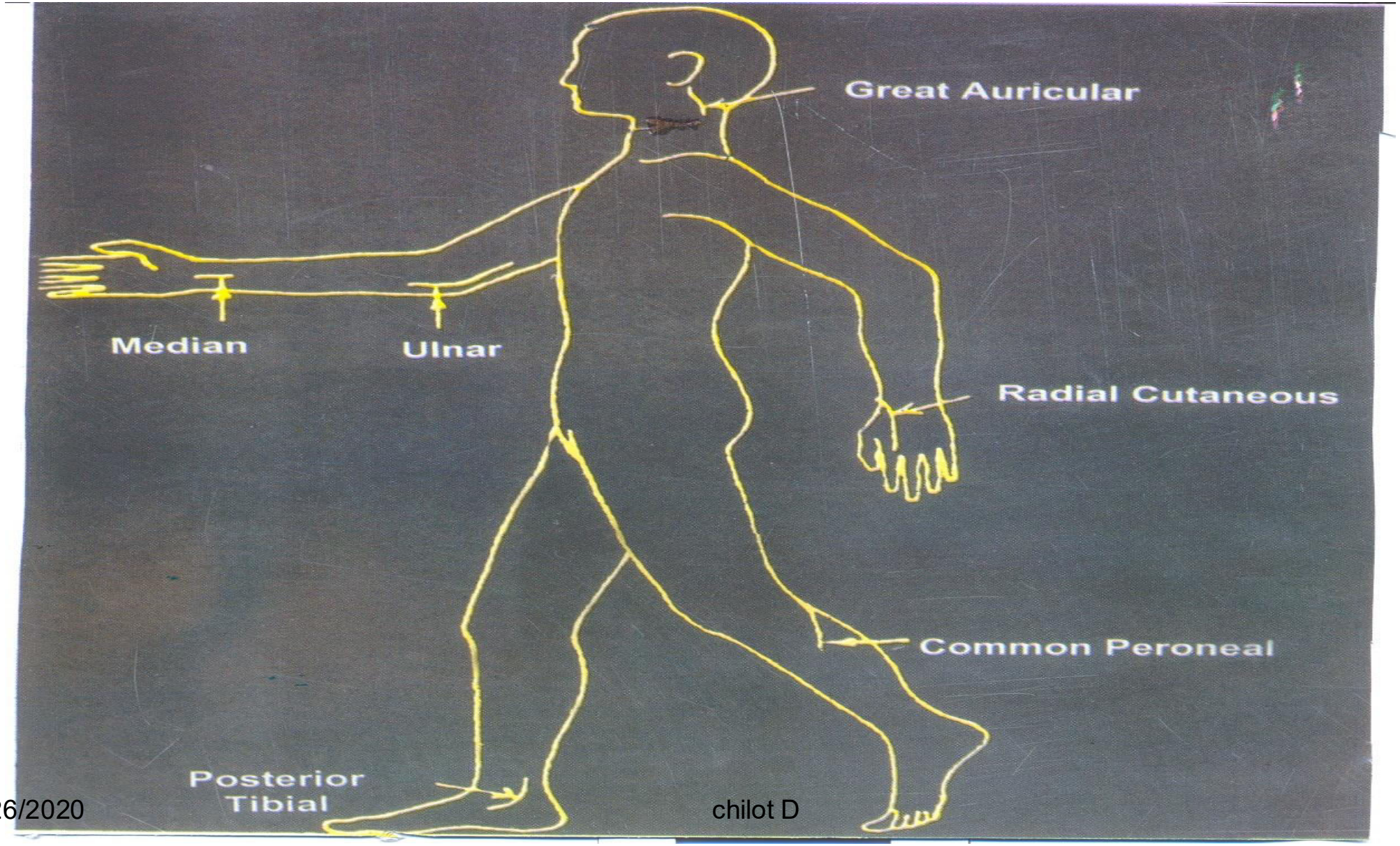
- It is a very **disabling** and **deforming** disease
- Deformity occurs in hands, feet and face. eye involvement may cause blindness
- Often the patient suffering from leprosy find that the community avoid him
- The disease is common in rural tropics & subtropics
- Endemic in south & southeast Asia, tropical Africa and central America

Pathogenesis

- The leprae bacilli multiply in macrophages of the skin and nerve fibers
- The organism multiplies best in the

  cooler parts of the body
- So that the skin of the face and limbs and their more superficial nerves are invaded first.
- As a result of the invasion of bacilli there is an inflammatory response.
- Clinically patients develop
  - **Hypopigmented /pale/ or**
  - **Reddish or**
  - **Copper color flat or raised or nodular** patches

Places where nerves can be felt



Pathogenesis…

Nerve damage and disability

- The primary cause of **disability** is **the destruction of nerves**

1. Damage to sensory nerves → anesthesia

- Minor injuries are not noticed
- Wounds fill with dirt & infection is driven deeper in to the tissue
- Pressure is not noticed

2. Damage to motor nerves → paralysis

- Cause muscular imbalance & leads to abnormal position of fingers and toes (deformity) → injury → infection

Pathogenesis…

- **Ulnar** nerve damage results in weakness of **4th and 5th fingers** and lumbricoid position impossible
- **Median** nerve damage results in **claw hand**, dryness and insensitivity of inner hand
- **Radial** nerve damage results in **wrist drop**
- **Peroneal** nerve damage results in **foot drop**; cock's gate

3. Damage to autonomic nerves

- Impaired circulation → slow wound healing
- Loss of sweating → skin cracks→ infection

Pathogenesis…

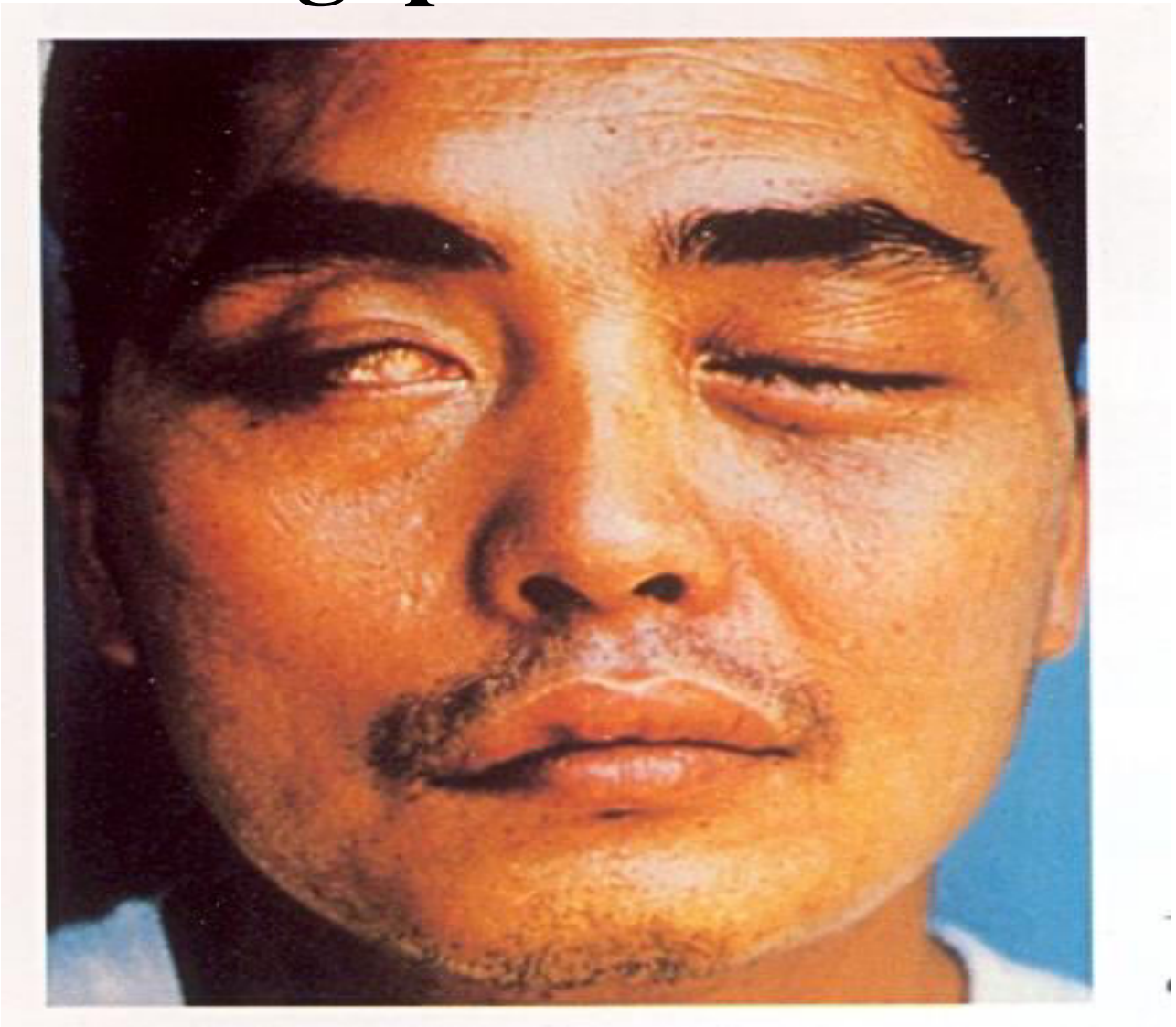
Eye problems

1) Anesthesia of the cornea → foreign bodies in the eye not noticed →corneal ulcers → blindness

2) Paralysis of the eye lids → lagophthalmos → dryness of the cornea → corneal ulcer→ blindness

3) Leprosy reactions → irritis → adhesions → glaucoma → blindness.

Lagophthalmos



Leprosy pts are likely to get three types of ulcers on their feet

- Those due to injuries & burns – because of anesthesia. Usually unnoticed until secondarily infected
- Crack of the heel and or lateral borders of foot due to absence of normal sweating
- The specific plantar ulcer which is due to walking on anesthetic foot

Mode of transmission and IP

- Not clearly established
- Leprosy transmission is favored by overcrowding, poor housing and low socio economic conditions
- It is very likely that leprosy is transmitted by sneezing, coughing, spitting & unhygienic nose cleaning habits- this is by droplets
- House holds (10x higher) and prolonged close contact are important
- Leprosy bacilli are able to enter through small wounds (unable to penetrate intact skin)

Incubation period

- 9 months – 20 years (most often 3-5 yrs)

Clinical manifestations

Vary between clinical **classifications** of the disease

1. **Lepromatous (multibacillary –MB) leprosy**

Skin lesions

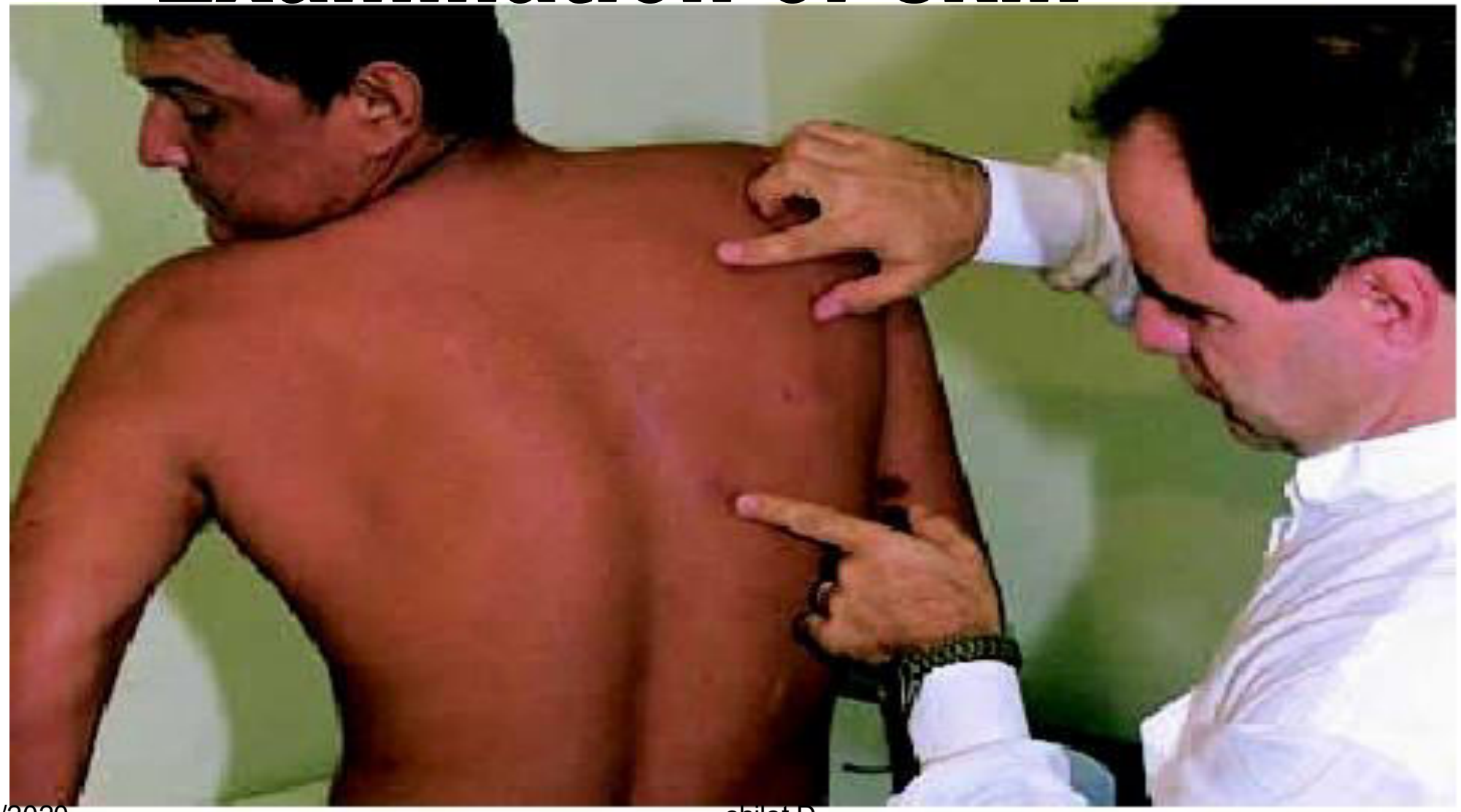
- Include macules (flat lesions), papules (raised lesion) and nodules
- More than 5 lesions

2. Tuberculoid (paucibacillary –PB) leprosy

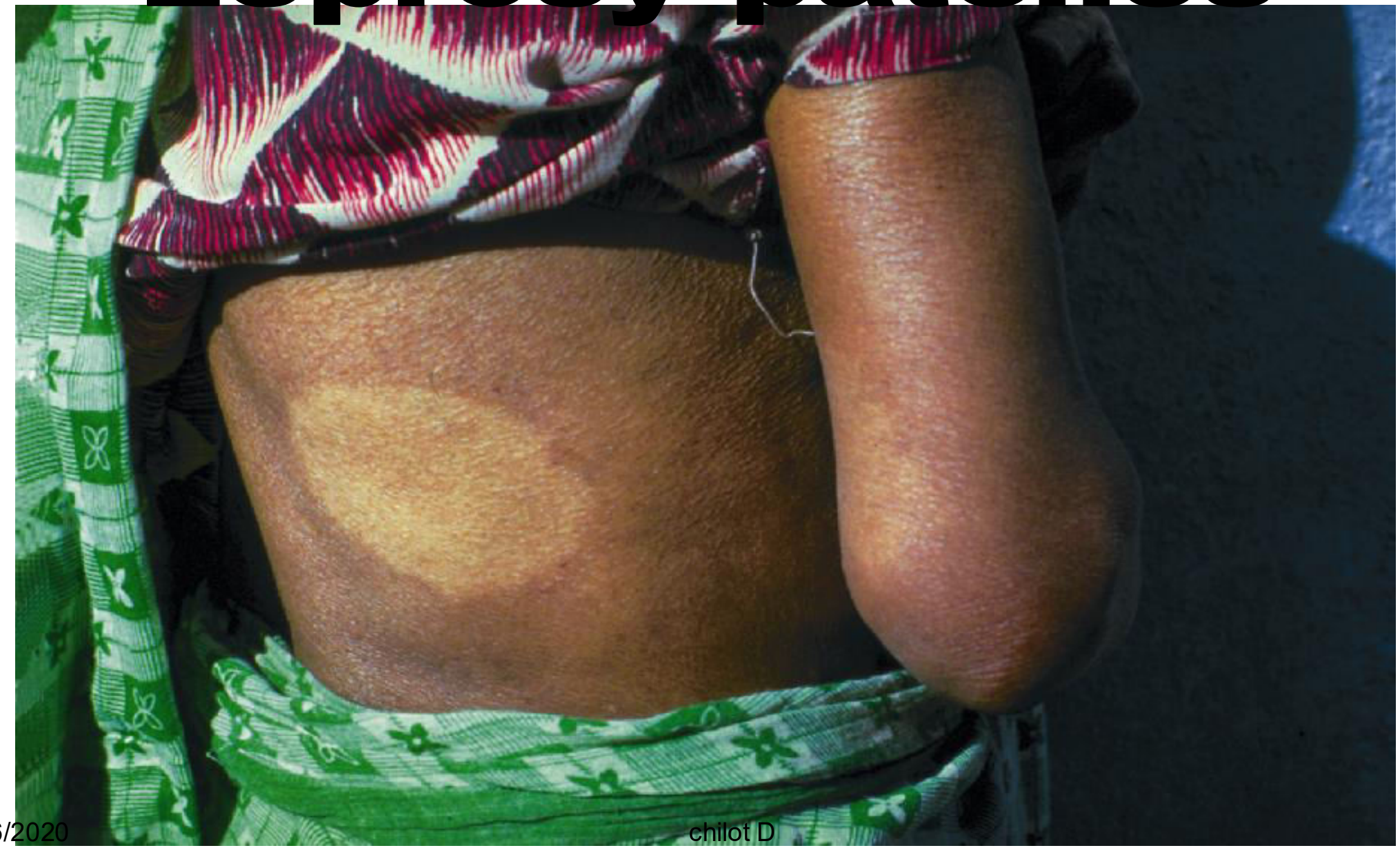
Skin lesions

- Include macules (flat lesions), papules (raised lesion) and nodules
- 1-5 lesions

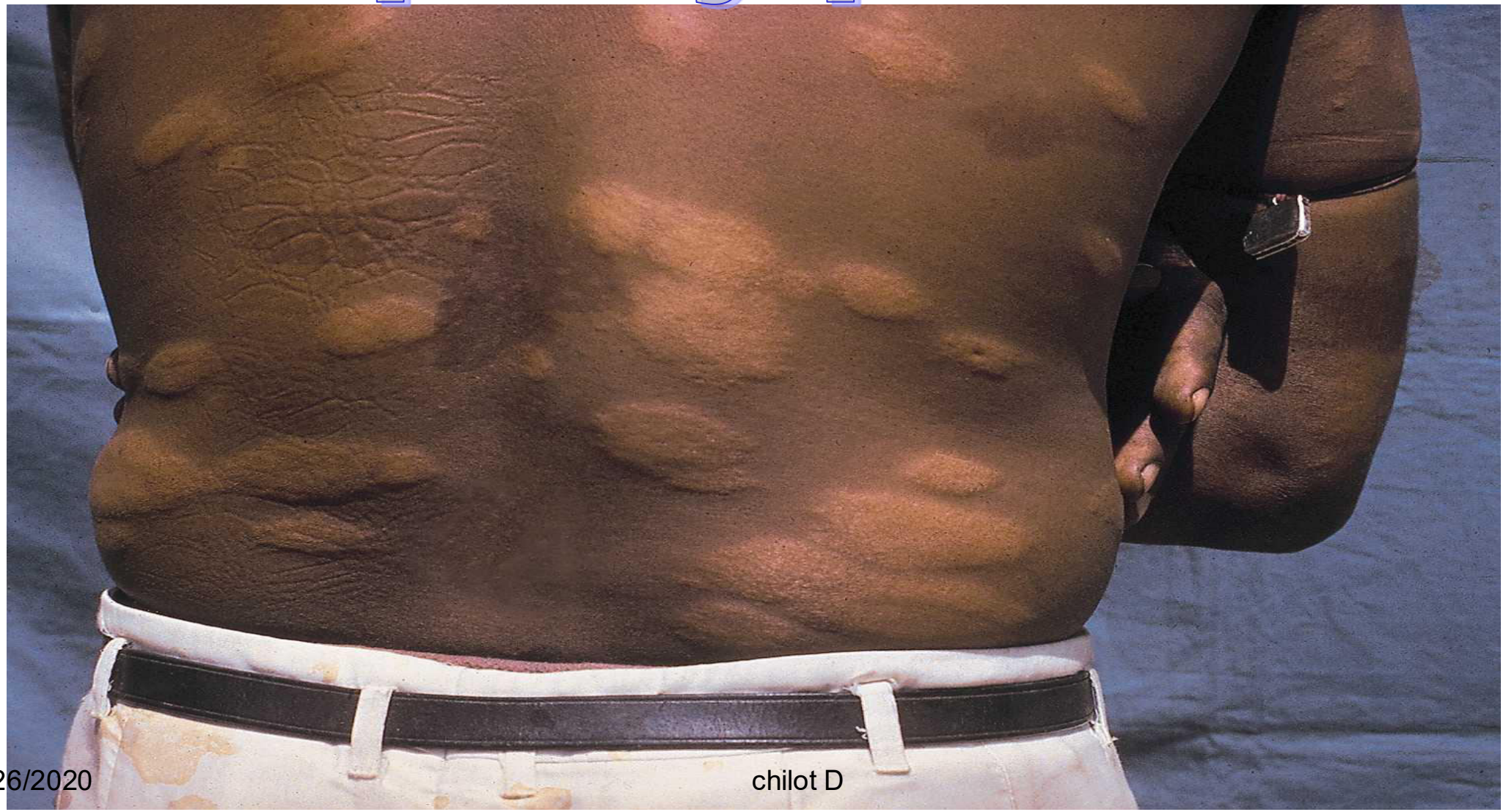
Examination of skin



Leprosy patches



Leprosy patches



Leprosy patches

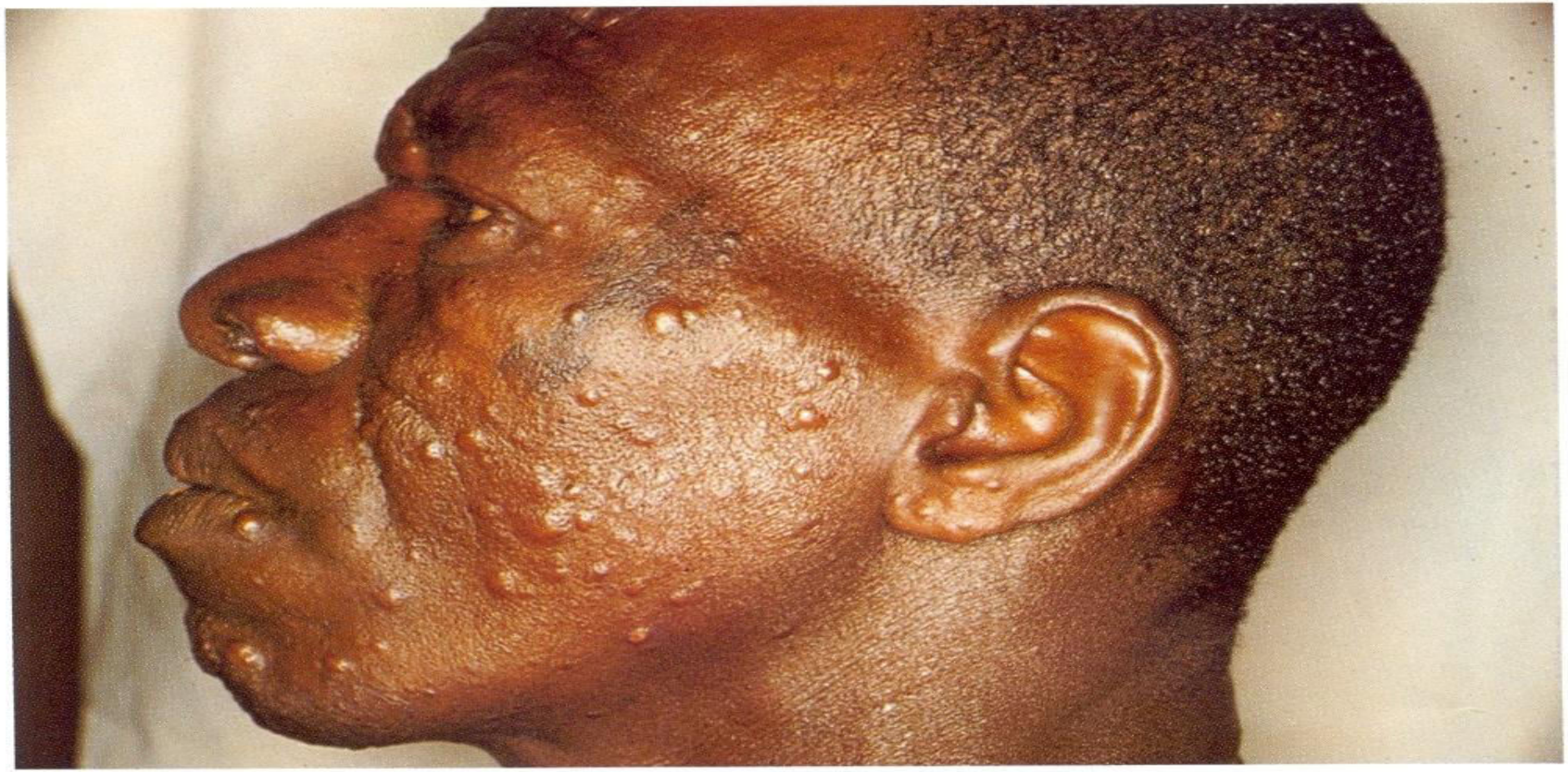
Leprosy patches...

...can be pale or reddish
or copper-coloured.

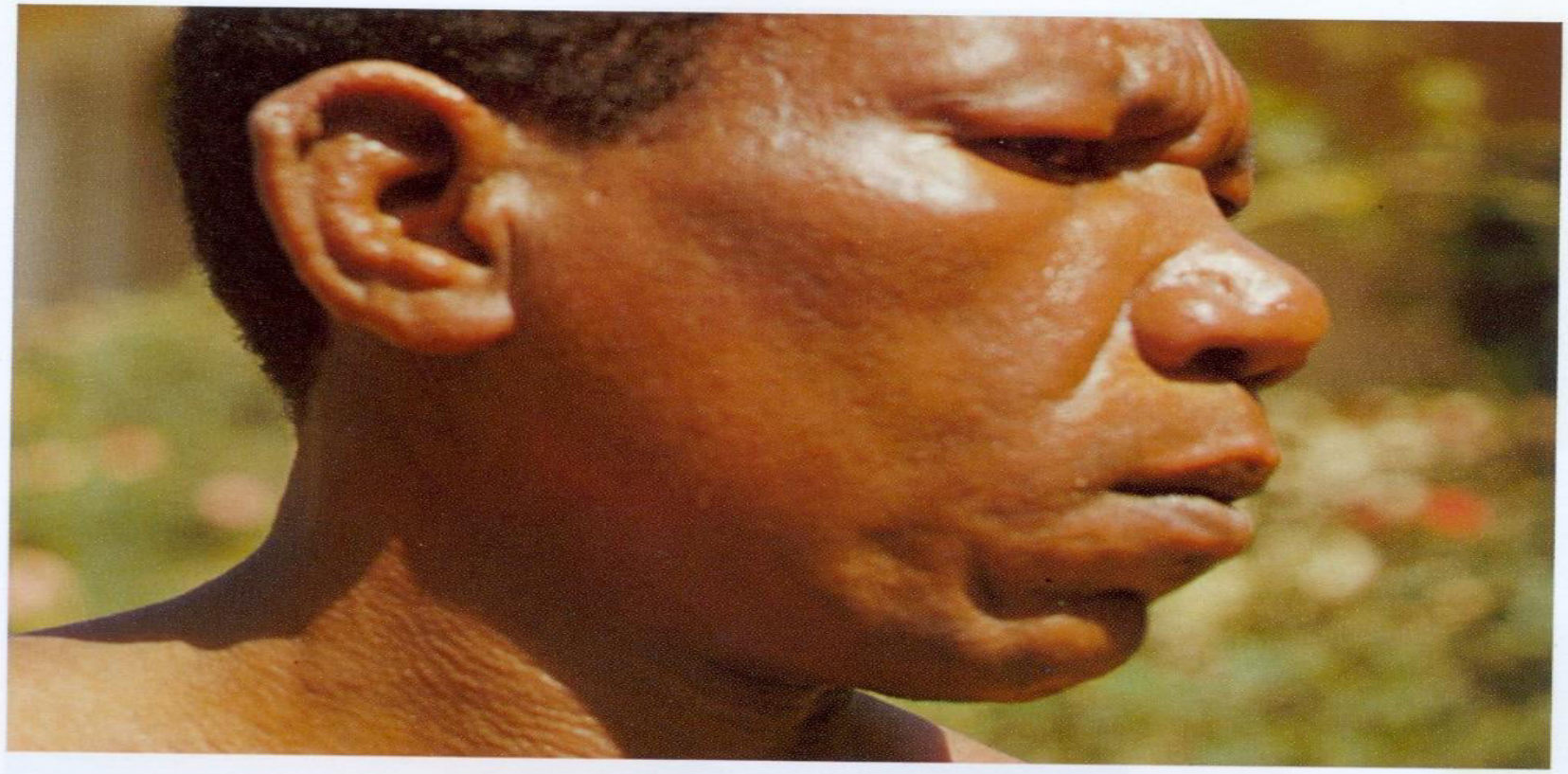
...can be flat or raised.

Skin patch

Leprosy nodules



Infiltration of the skin



25. LEPROMATOUS LEPROSY, LL

Diffuse infiltration of the face, particularly prominent on the ear and glabella. Madarosis (loss of eyebrows and eyelashes) BI - 6.

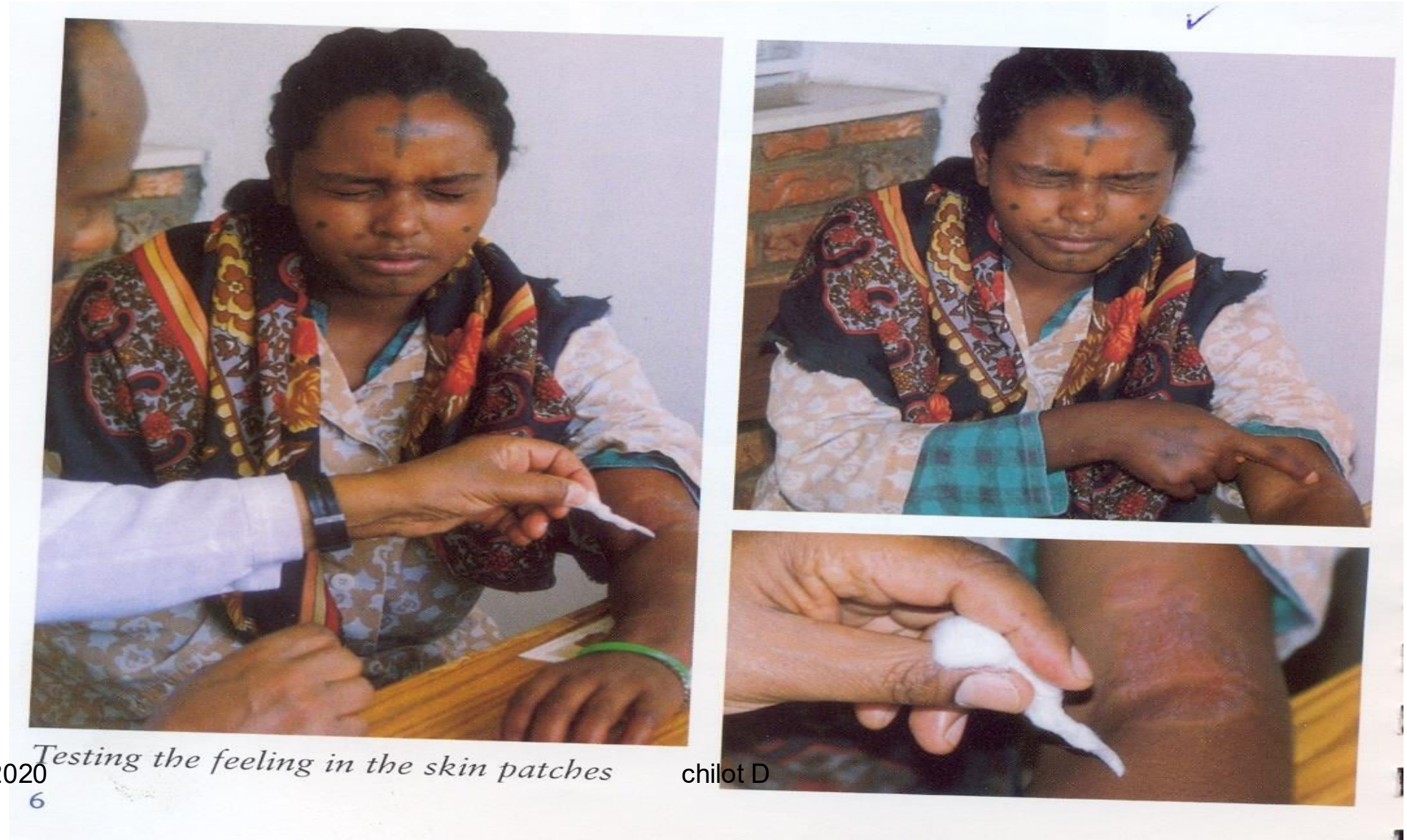
Diagnosis

- Leprosy should never be diagnosed in the absence of definite evidence
- Most commonly based on clinical signs & symptoms /over 95%/
- **Laboratory diagnosis is** indicated for doubtful cases or cases difficult to diagnose

Diagnosis…

- A case of leprosy is a person, having **one or more** of the following cardinal signs of leprosy
  - ❑Hypopigmented or reddish skin lesion (s) with definite loss of sensation
  - ❑Damage to the peripheral nerves , as demonstrated by loss of sensation and weakness of the muscles of hands , feet or face
  - ❑Positive skin smear (AFB)

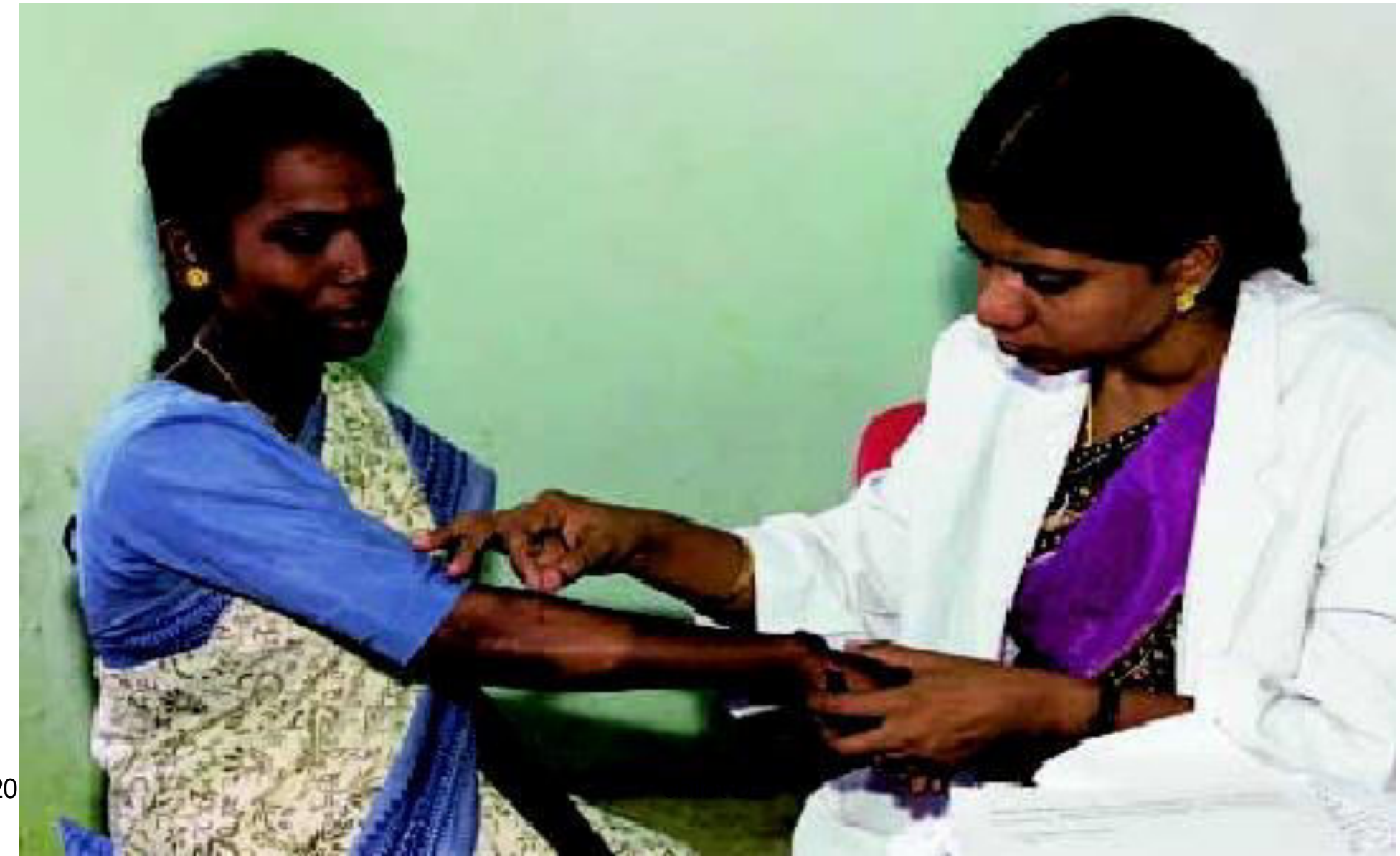
Testing feeling in skin patch with cotton wisp



Testing the feeling in the skin patches

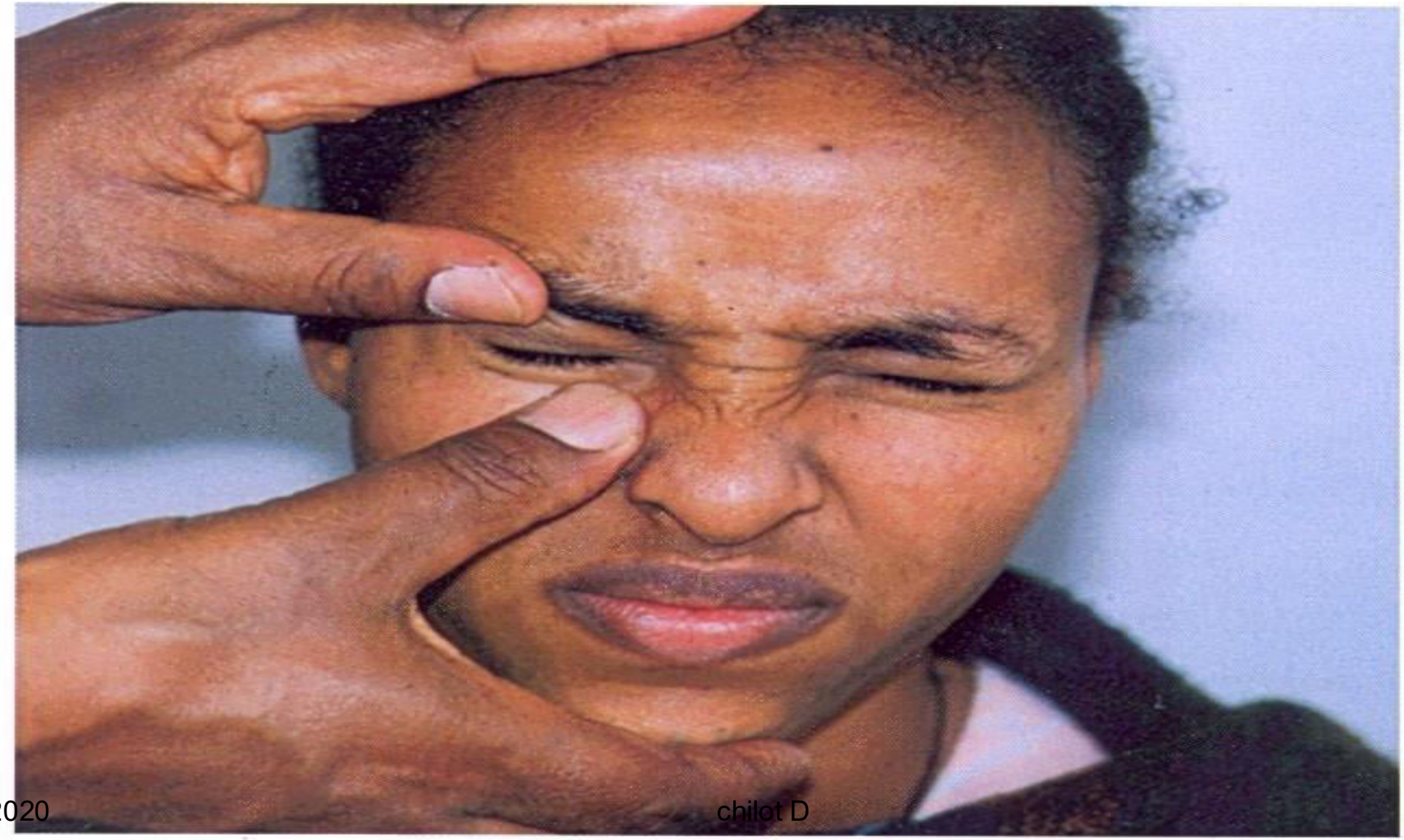
6

Testing feeling in skin patch



Testing feeling in skin patch

Testing the eyelid muscles

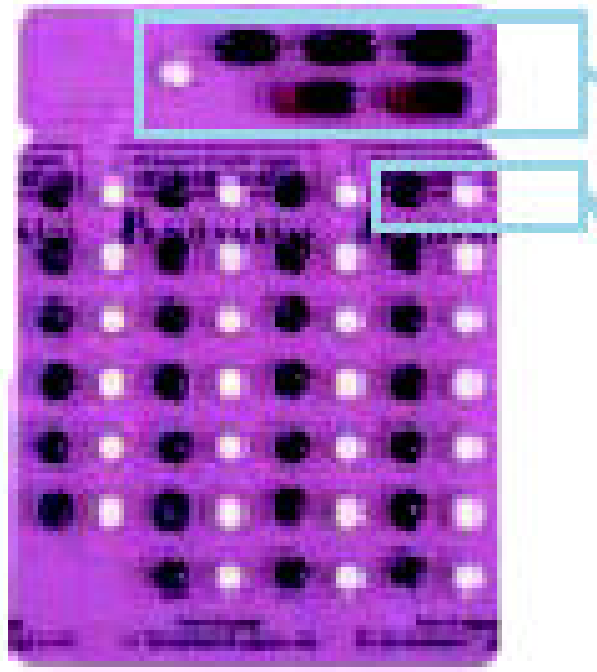


Treatment

- Any patient showing a positive skin smear, irrespective of the clinical classification, should be treated with the **MDT regimen for MB leprosy**
- All registered and newly detected cases must be started on an appropriate MDT (multi drug therapy) regimen as the diagnosis is confirmed

Treatment…

- The drugs used in WHO- MDT are combination of:
  - **Rifampicin, clofazimine and dapson for MB** patients
  - **Rifampicin and dapson for PB** patients
- Rifampicin is given once a month but dapsone is administered daily



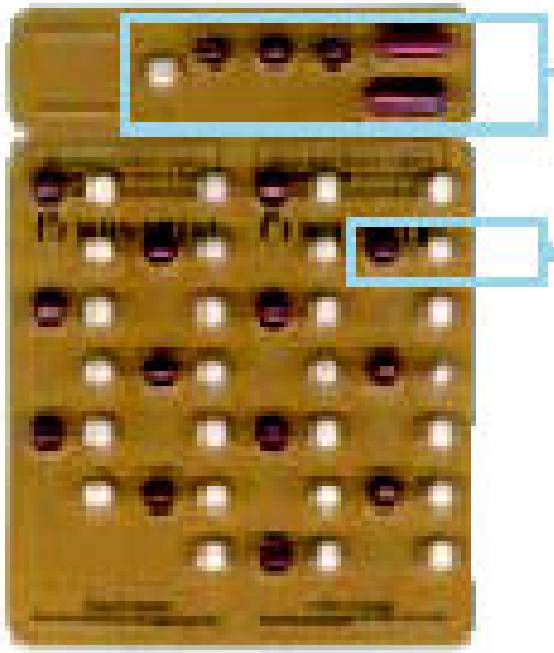
MB adult blister pack

MB adult treatment:

Once a month: Day 1
- 2 capsules of rifampicin (300 mg X 2)
- 3 capsules of clofazimine (100mg X 3)
- 1 tablet of dapsone (100 mg)

Once a day: Days 2–28
- 1 capsule of clofazimine (50 mg)
- 1 tablet of dapsone (100 mg)

Full course: 12 blister packs



MB child blister pack

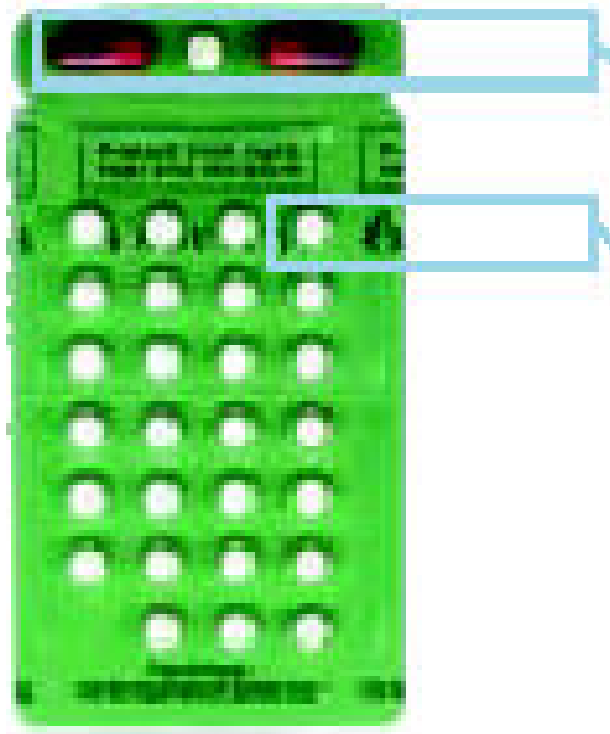
MB child treatment (10–14 years):

Once a month: Day 1
- 2 capsules of rifampicin (300 mg+150 mg)
- 3 capsules of clofazimine (50 mg X 3)
- 1 tablet of dapsone (50 mg)

Once a day: Days 2–28
- 1 capsule of clofazimine every other day (50 mg)
- 1 tablet of dapsone (50 mg)

Full course: 12 blister packs

For children younger than 10, the dose must be adjusted according to body weight.



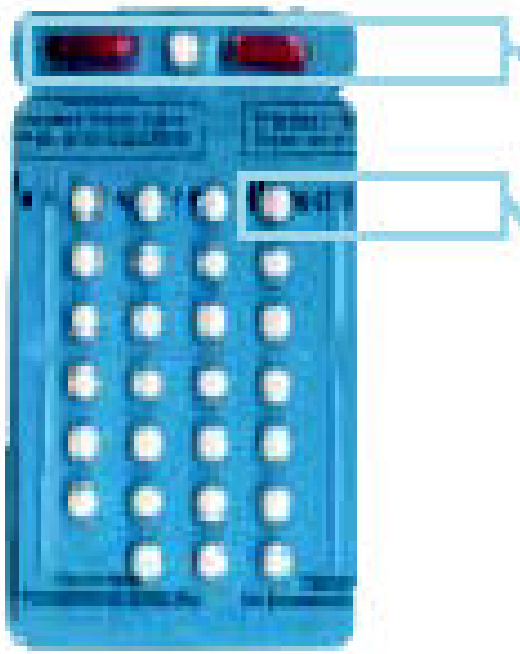
PB adult blister pack

PB adult treatment:

Once a month: Day 1
- 2 capsules of rifampicin (300 mg X 2)
- 1 tablet of dapsone (100 mg)

Once a day: Days 2–28
- 1 tablet of dapsone (100 mg)

Full course: 6 blister packs



PB child blister pack

PB child treatment (10–14 years):

Once a month: Day 1
- 2 capsules of rifampicin (300 mg+150 mg)
- 1 tablet of dapsone (50 mg)

Once a day: Days 2–28
- 1 tablet of dapsone (50 mg)

Full course: 6 blister packs

For children younger than 10, the dose must be adjusted according to body weight.

Give the patient the first dose at the health centre. Show them which drugs from the MDT blister pack should be taken once a month and which every day.

Give the patient **enough blister packs** to last until their next visit. Arrange the time and place of the visit.

The best way to prevent the spread of leprosy is to treat all patients with MDT.

Prevention and control

Prevent nerve damage

- Most nerve damage is caused by reactions
- Early diagnosis, treatment and accurate management of reactions prevent all disabilities

Prevent deformity

- Proper care of skin
- Proper care of minor wounds & skin infections
- Provision of shoes
- Physiotherapy
- Health education

Prevention and control…

Prevent blindness

- Check for proper eye closure
- Test the sensation of the cornea
- Refer patients with red eyes immediately

Prevent defaulting

Reasons why the patient gives up his treatment

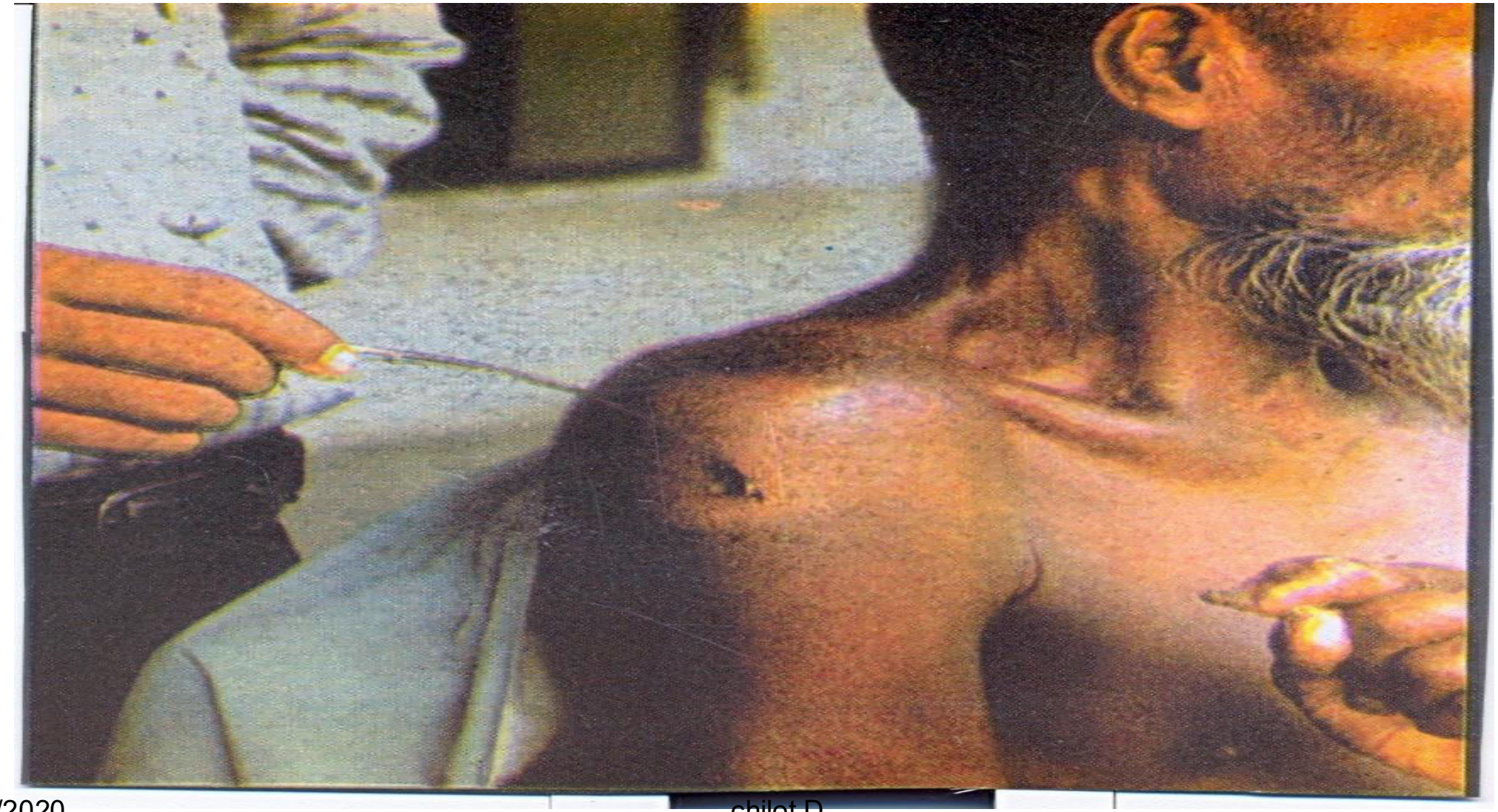
- No confidence in treatment
- Clinic too far
- Shame
- Disability
- Thinks he is cured
- Thinks he has not got leprosy
- Treatment too long

A leprosy case is categorized as:

- **New case (N)** : A patient who has never had treatment for leprosy before.
- **Relapse after MDT (R)** : A Pat. who develops active leprosy after completing a full course of MDT treatment.

  NB: Same classification as the original classification.
- **Return after default (D)** : An **MB** patient returning for treatment after missing > 3 four-weekly doses of MDT.
- **Transfer in (T):** A patient coming to another woreda to continue treatment.

Testing feeling skin patch for loss of sensation



Examination of the nerves (1)

General

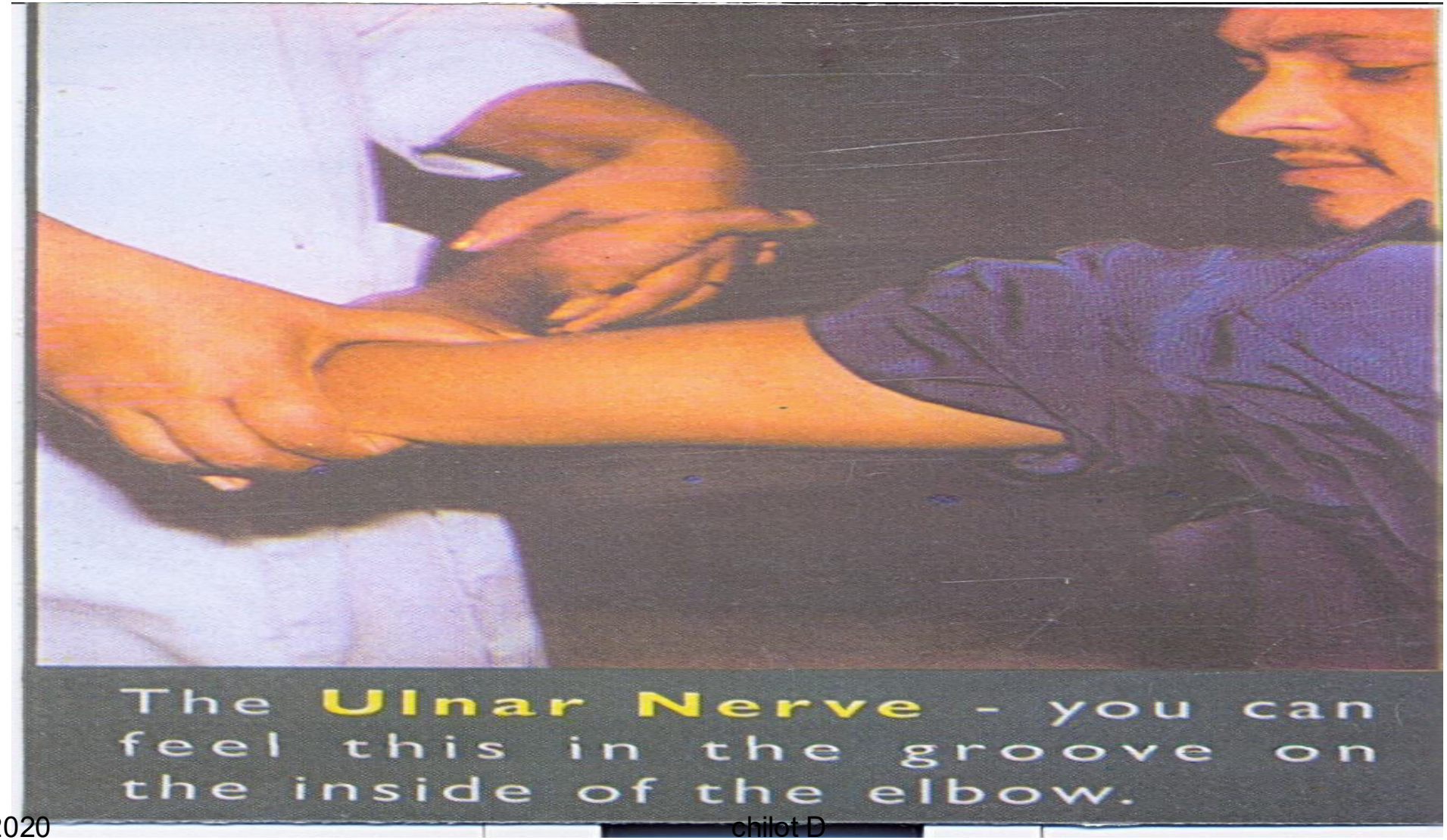
- Nerves that are affected by leprosy are:
  - Great Auricular
  - Radial Cutaneous
  - Ulnar
  - Median
  - Common Peroneal
  - Posterior Tibial
- Ulnar & Peroneal nerves are most commonly enlarged.

Examination of the nerves (3)

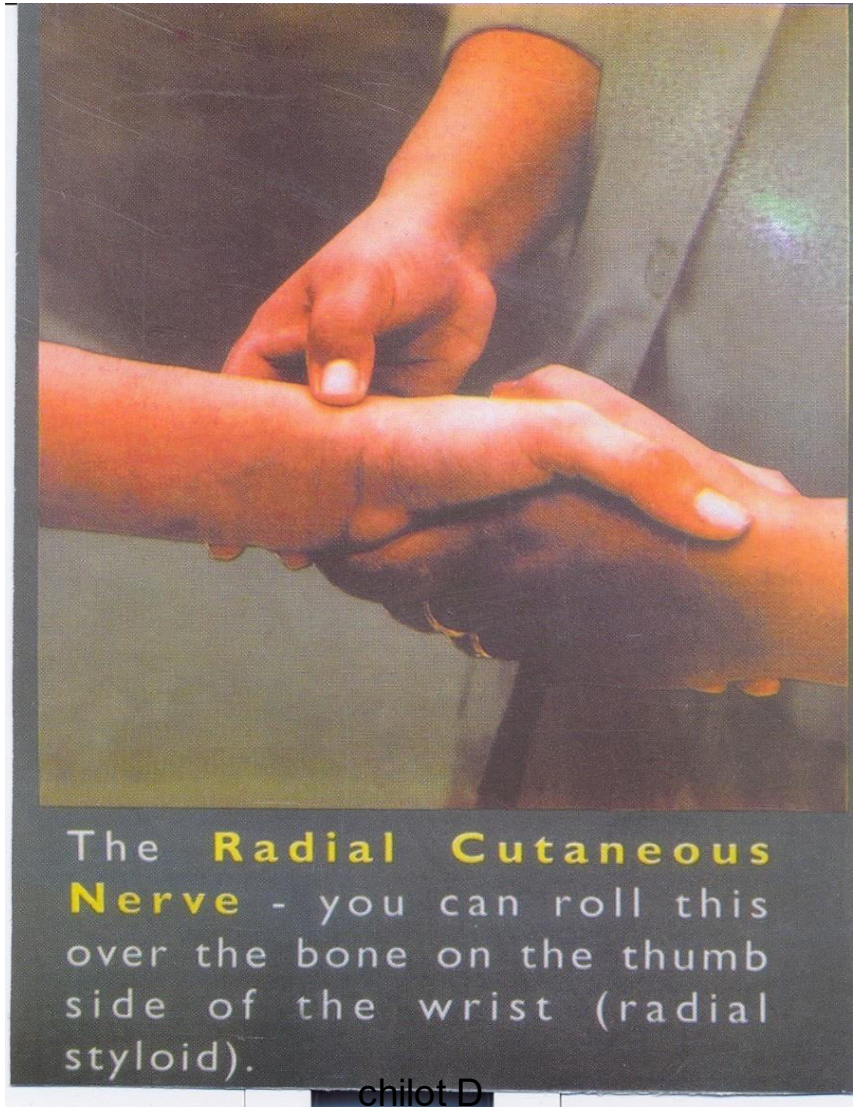
Nerve palpation

- Palpate the nerves for enlargement and tenderness (size, consistency (firm/soft), tenderness)
- When palpating a nerve trunk:
  - always use 2 or 3 fingers to do the palpation.
  - roll over the surface of the underlying bone
  - compare the left and right side

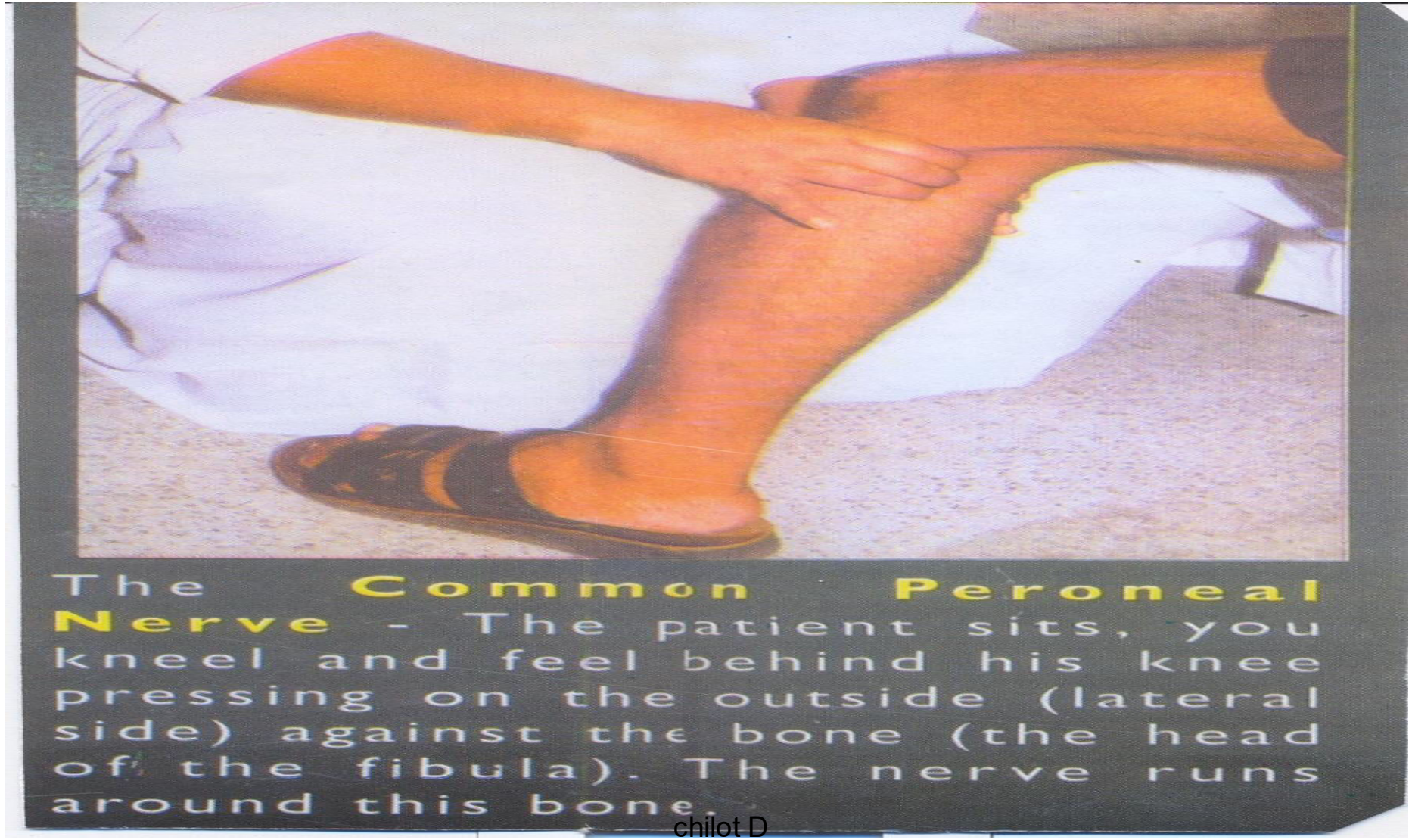
The Ulnar nerve



The Radial Cutaneous nerve



The Common Peroneal nerve



- **Test the following peripheral nerve fibers.**

a) Test Sensory Nerves by Sensory Testing (ST)
b) Test Motor nerves by Voluntary Muscle Testing (VMT)
c) Test Autonomic nerves by checking palms and soles for dryness.

Examination of the nerves (4)

Nerve function testing

- **Sensory nerve fibers:** are involved early and most severely affected. Cause aneasthesia which is the most important cause of disability.
- **Motor nerve fibers:** paralysis, joint stiffness, clawing,
- **Autonomic nerve fibers:** sweating impaired, skin dryness, skin fissures, ulceration, scaring

1- Voluntary Muscle Testing (VMT)

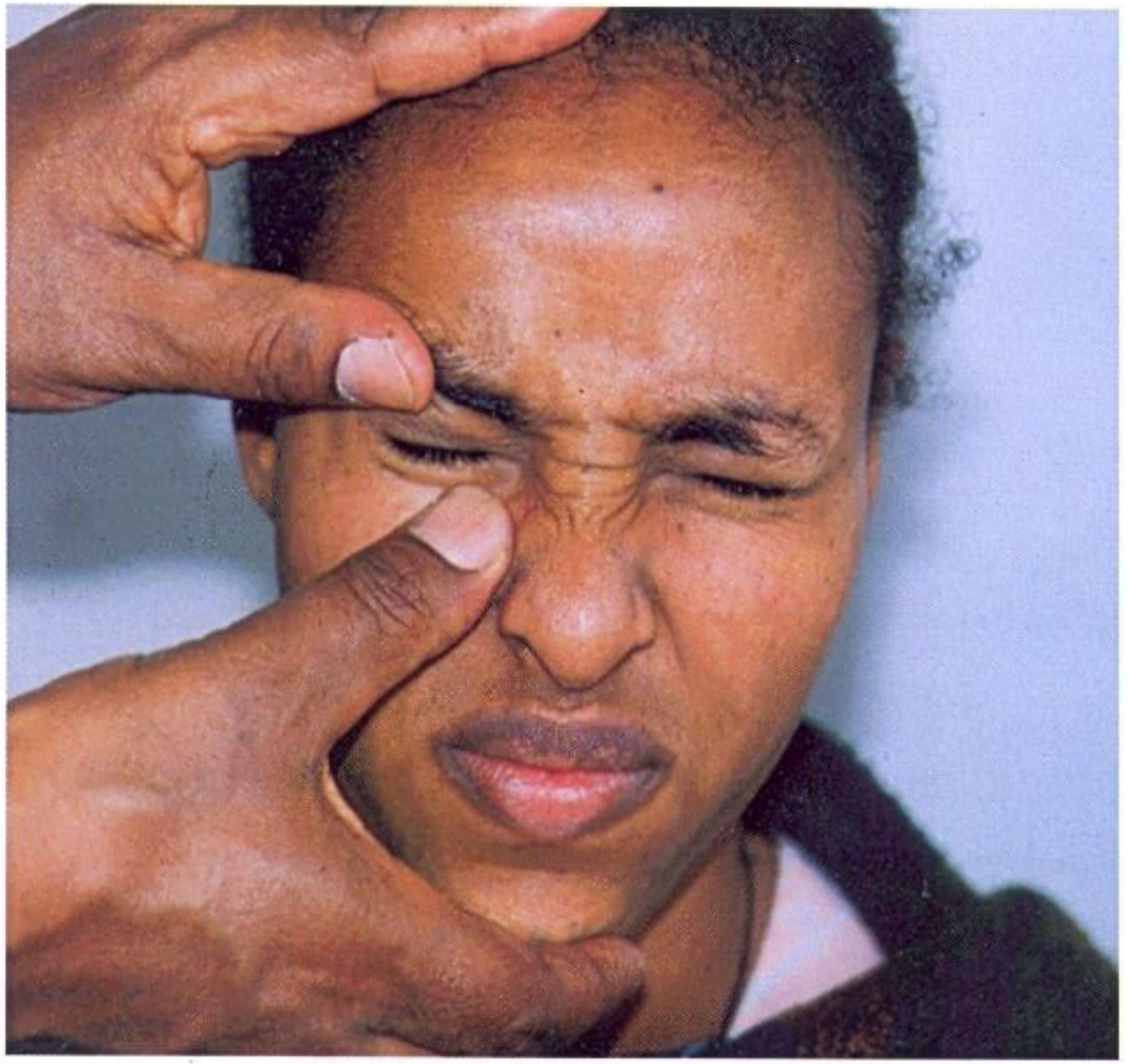
- Test the strength of muscles of:

- the **eyes, hands and feet.**
  - **Grade strength as: Strong (S), Weak (W) or Paralyzed (P)**

a) VMT of the EYE:

- Eye closure : Check movement of eye lid (normal, reduced or absent);
- Look for completeness of eye lid closure: lagophthalmus (inability to fully close the eye);
- if there is lagophthalmus measure the lid gap in mm.
- If movement is normal: Check for strength (resistance) of eye lid muscles: Is resistance (S), (W) or (P)?

Testing the eyelid muscles

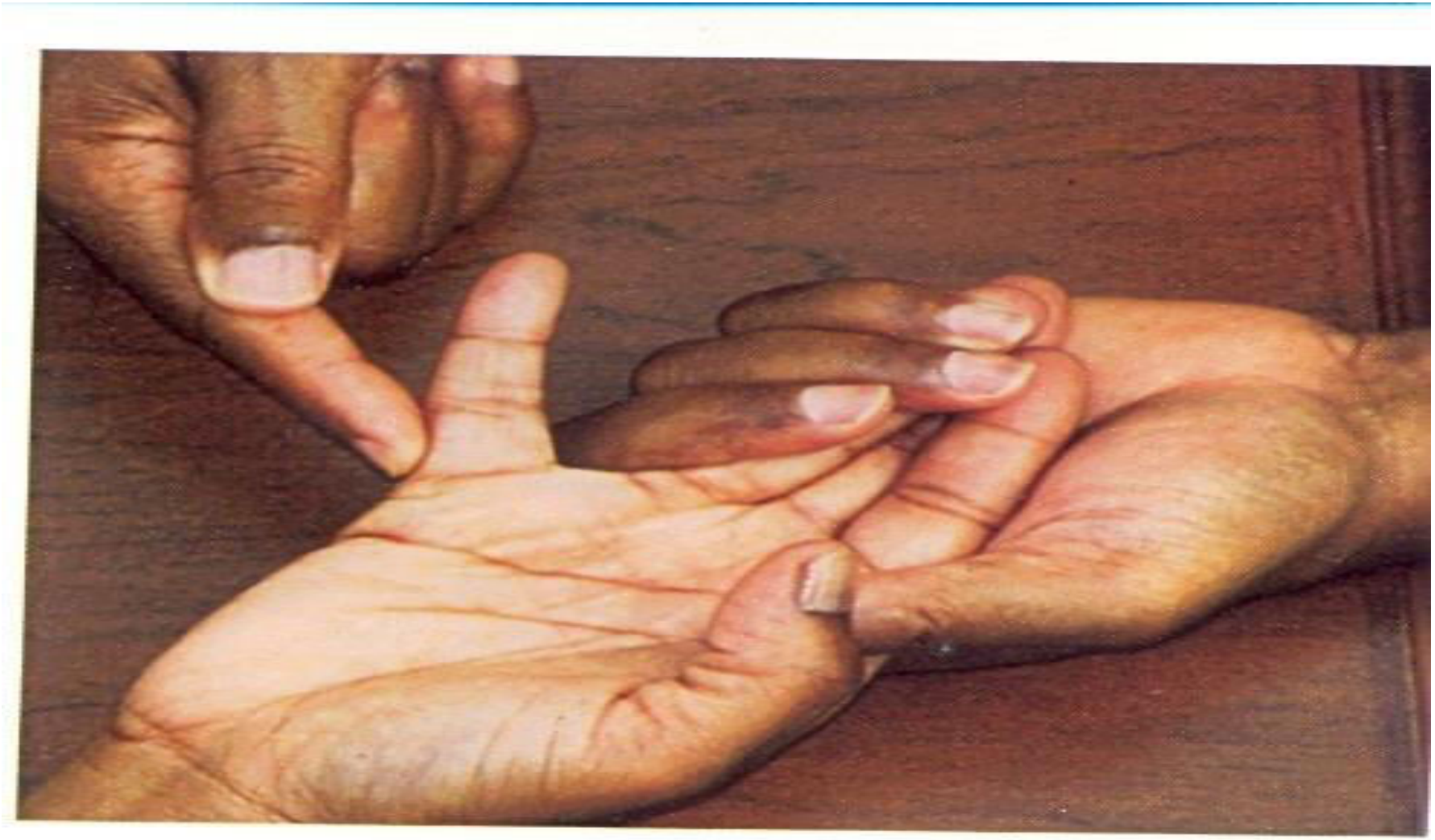


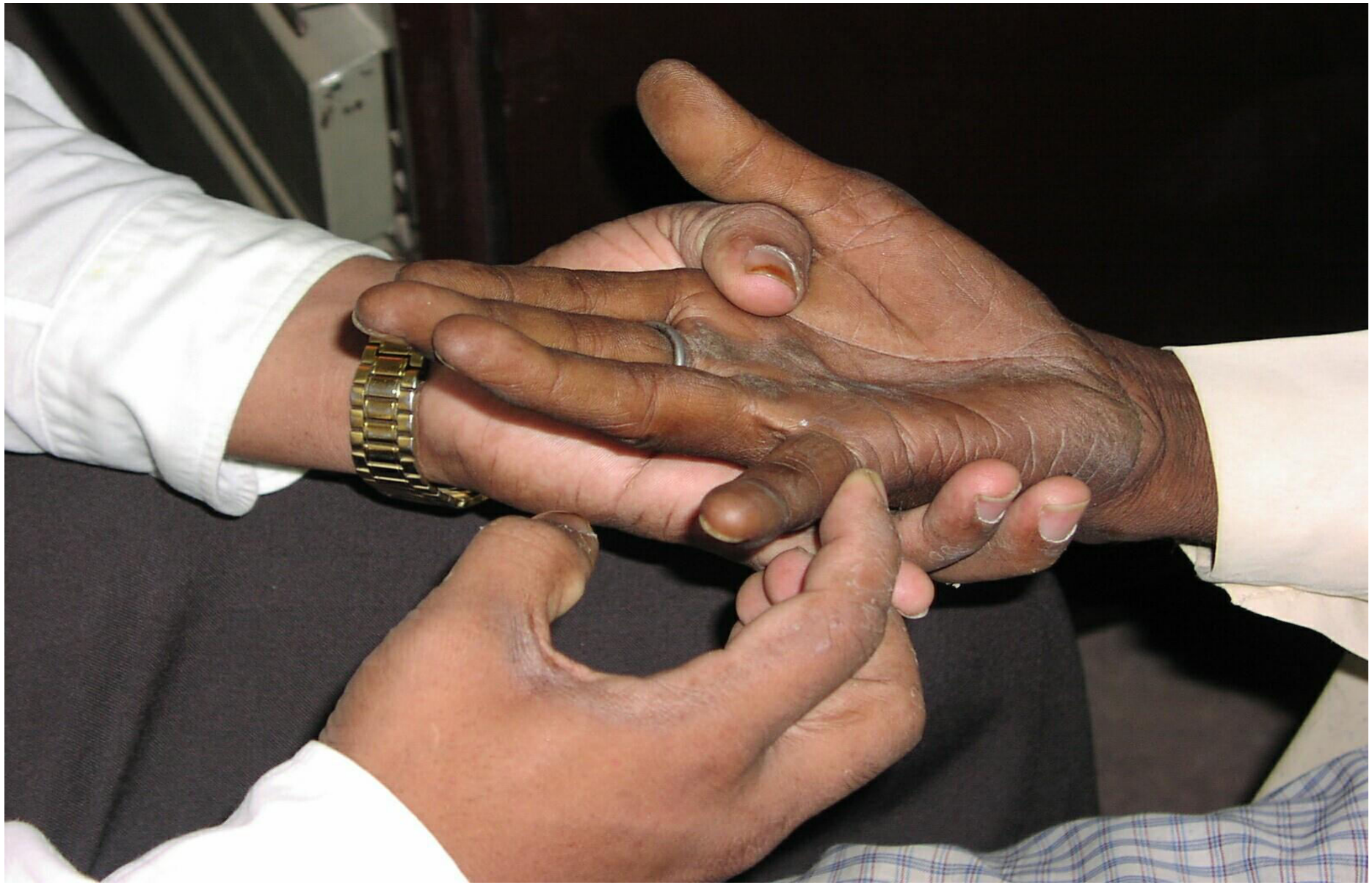
b) VMT of hands and feet

- Check range of **movement**:
- Is movement normal, reduced or absent because of muscle paralysis .
- If movement is normal: test for resistance; Press gently while asking the patient to maintain the position by resisting the pressure.
- Is the resistance (S), (W) or absent (P)

- Test for: - **Ulnar** nerve function :
  (little finger out test).
  - **Median** nerve function:
    (Thumb up test)
  - **Radial** nerve function:
    (Wrist up test)
  - **Peroneal** nerve function:
    (Foot up test)

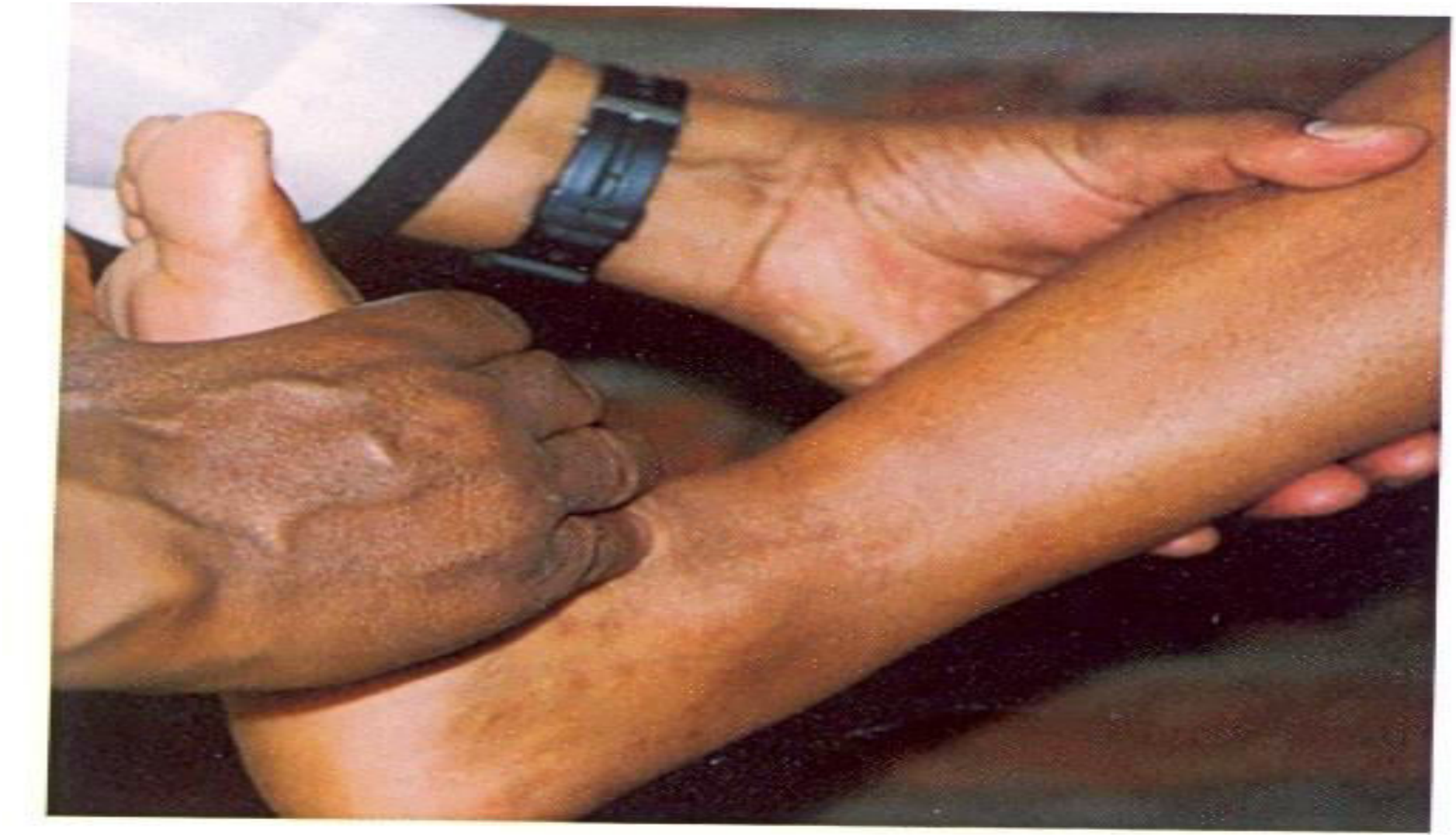
Finger out test



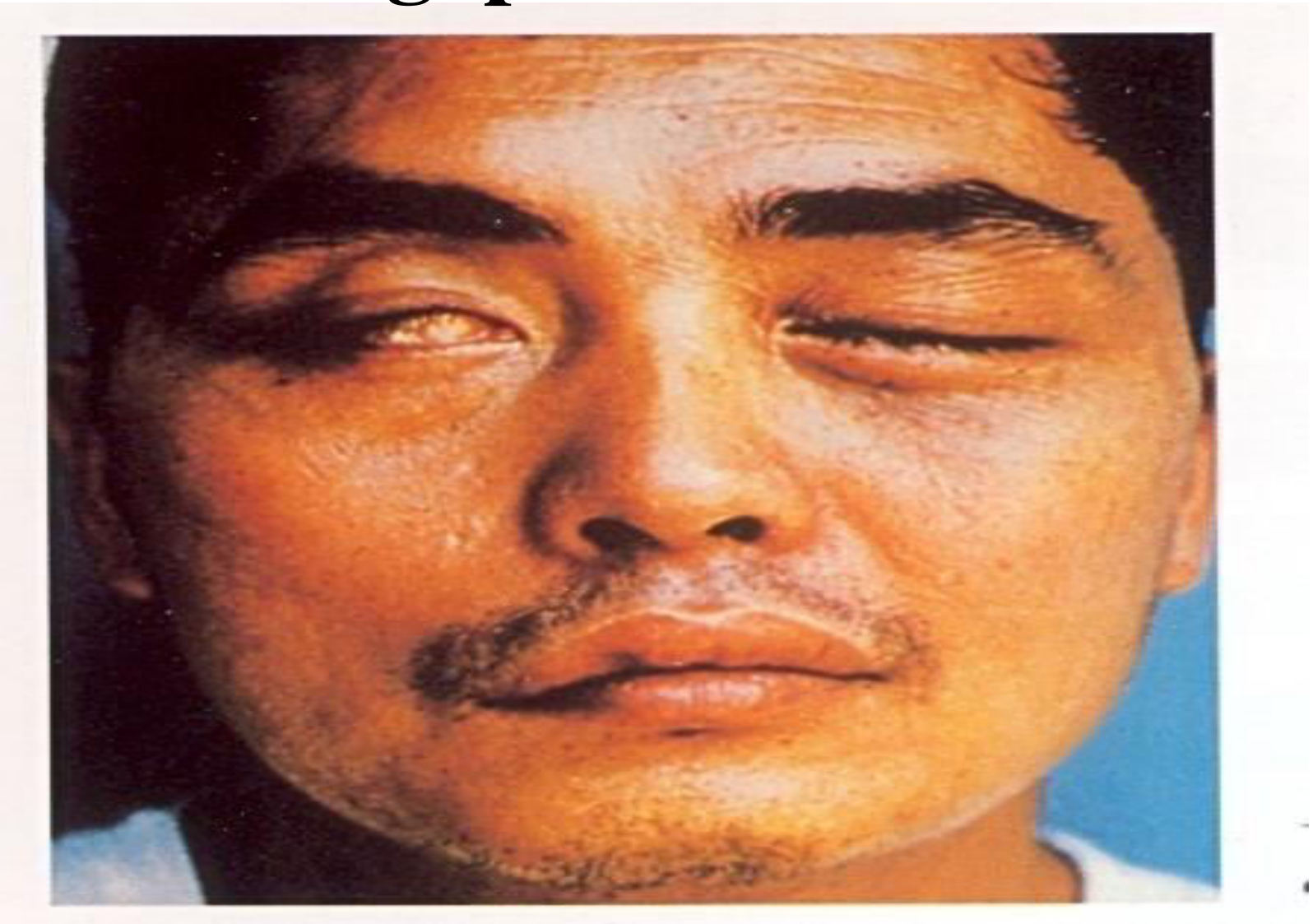


Thumb up test

Foot up test



Lagophthalmos



Examination of Eyes, Hands and Feet

Assessment of the eyes: Visual Acuity

- Test at 6 metres asking the patient to cover one eye and then count the number of fingers that the assessor holds up.

  If the patient cannot see at 6 meters, re-test at 3 meters.

- Check for other problems/complications: Corneal injury, vision loss due to incomplete blink and/or lagophthalmus.

Examination of Eyes, Hands and Feet (cont'd)

Look for:

- Skin cracks & wounds on palms & soles
- Clawed finger & toes
- Shortening/absorption of fingers & toes
- Wrist & foot drops

N.B. The above complications result from nerve damage.

Leprosy case definition

A leprosy case is categorized as:

- **New case (N)** : A patient who has never had treatment for leprosy before.
- **Relapse after MDT (R)** : A Pat. who develops active leprosy after completing a full course of MDT treatment.

  NB: Same classification as the original classification.
- **Return after default (D)** : An **MB** patient returning for treatment after missing > 3 four-weekly doses of MDT.
- **Transfer in (T):** A patient coming to another woreda to continue treatment.
- **Other (O):** A patient not fitting in any of the above categories.

Give the patient the first dose at the health centre. Show them which drugs from the MDT blister pack should be taken once a month and which every day.

Give the patient **enough blister packs** to last until their next visit. Arrange the time and place of the visit.

The best way to prevent the spread of leprosy is to treat all patients with MDT.

steps to start MDT

- Classify as PB or MB leprosy
- Inform the patient about the disease
- Explain the MDT blister pack - show drugs to be taken once a month and every day
- Explain possible side effects (e.g. darkening of skin) and possible complications and when they must return to the health centre
- Ask the patient when it is convenient for him/her to come back to the health centre. Give enough MDT blister packs to last until the next visit.
- Fill out the patient treatment card

Information about the patient

- Caused by a bacteria
- Affects skin and sometimes nerves
- Progresses slowly
- Easy to diagnose and cure
- Lead normal life, do not change life style

Information to patients about possible problems

- Skin discoloration due to clofazimine
- Urine discoloration due to rifampicin
- In case of fever, pain in the nerves, muscle weakness, joint pains they must return immediately to the health centre
- In case of eye problems
- Appearance of new skin patches
- How to protect insensitive hands/feet

Why integrate leprosy into the general health services?

- ❑ Integration means to provide "comprehensive" essential services from one service point
- ✓ to improve patients' access to leprosy services and thereby ensure timely treatment
- ✓ to remove the "special" status of leprosy as a complicated and terrible disease
- ✓ to consolidate substantial gains made
- ✓ to ensure that all future cases receive timely and correct treatment
- ✓ to ensure that leprosy is treated as a simple disease


Thank u Very much !!!

Reading assignment

- SARS
- Common cold
- Swine flu

Intestinal parasitic disease and infection

Learning Objective:

- Define diseases that spread with contaminated food and water
- Describe the epidemiologic distribution of each disease
- Describe the clinical manifestations and diagnosis modalities of each disease
- Identify the five important ''Fs'' in oral fecal disease transmission
- List diseases commonly transmitted by having direct contact with feces
- Implement preventive and control methods of oro-fecally transmitted diseases

Brain storming questions

List Intestinal parasite disease ?

What do you think their mode of transmission ? And what are their prevention mechanism ?

Intestinal parasitic disease and infection

Introduction

Causative organisms are excreted in the stools of infected persons

The portal of entry for those diseases is the mouth

Five "Fs" which play an important role in fecal oral disease transmission include: **finger, flies, food, fomites** and **fluid.**

Intestinal parasitic disease and infection

1. Typhoid fever

- Bacteria of the genus Salmonella are highly adapted for growth in both humans and animals and cause a wide spectrum of systemic disease
- Salmonella spp. > 2400 serotypes
- Motile, Non-spore forming Gram-ve bacilli
- A systemic infectious disease characterized by high continuous fever, malaise /feeling of uneasiness/ and involvement of lymphoid tissues and spleen

Intestinal parasitic disease and infection

Infectious agent

- Salmonella typhi (S.typhi)

Occurrence

- World wide, particularly in poor socio economic area
- Most common in preschool & school aged children (5-19 yrs) in endemic areas

Intestinal parasitic disease and infection

Reservoir

- Humans /patients and carriers/
- After convalescence typhoid bacilli often settle in the gall bladder and are excreted in the faces for a very long time
- Urinary carriers are seen particularly in areas where shistosoma hematobium infections occur

Intestinal parasitic disease and infection

Mode of transmission

- Water and food contaminated by feces & urine of patients and carriers
- Flies may infect foods
- Incubation period
- 1-3 wks

period of Communicability

- As long as the bacilli appears in excreta
- Usually from the first week through out convalescence
- 10% of untreated patients will discharge bacilli for 3 months after onset of symptoms
- 2 - 4% becomes chronic carriers
- Susceptibility increased in individuals with gastric achlorhydria or in HIV positive individuals

Clinical manifestaions

First week

Mild illnesses characterized by:

- Fever rising stepwise (ladder type)
- Anorexia
- Lethargy, malaise
- General aches
- Dull and continuous frontal head ache

- Splenomegaly
- Nose bleeding /epistaxis
- Vague abdominal pain
- Patients are usually constipated
- Cough (due to bronchitis)

- Relative bradycardia (slower than expected)

Clinical manifestations..

Second week

- ✓ Sustained temperature (fever)
- ✓ Sever illness with weakness
- ✓ Mental confusion and delirium with hallucination
- ✓ Bronchopneumonia
- ✓ Abdominal discomfort and distension due to ulcers in the lymphatic tissues of intestine
- ✓ Diarrhea (half of the patients)

Clinical manifestations…

- Fever and exhaustion will increase
- Lymphatic ulcer bleeds and perforation to intestine will result
- Improvement and reduction of temperature **if no complication**

Dx and D.Dx

Diagnosis

- Clinical manifestation
- Widal test:
  - Becomes positive on the 10th day of disease
  - It must be repeated after one week
  - An increase in titre between the first and the second widal tests is **diagnostic of typhoid fever**

Clinical manifestations…

- Culture/ blood, stool and urine/
  - Blood culture is positive during the first week and for a variable period after this
  - Stool culture is positive after the first week
  - Urine culture may be positive at any time
- WBC count: leucopenia (<4000/mm3 of blood) with relative lymphocytosis
- Stool exam: in 100% of the cases occult blood is present in the stool
  - occult blood is the presence of blood in patients stool identified by chemical testing

RX

First line

- Chloramphenicol 500mg po every 6 hr for 14 days, for children 25mg/kg

Alternative:

- Ciprofloxacin 500mg 2x/d for 10 days
- Ceftriaxone 1gm every day as single dose or 2 divided doses im or iv for 5-7 days. For children 20-50mg/kg/day
- Ampicillin and co-trimaxazole for carriers and mild cases.
- Antipyretics to control fever

prevention and Control

- Treatment of patients and carriers
- Education on hand washing – particularly – food handlers, patients and child care giver.
- Sanitary disposal of feces and control of flies
- Provision of safe and adequate water
- Safe handling of food
- Exclusion of typhoid carriers & patients from handling of food
- Immunization for people at special risk (e.g. travelers to endemic areas)
- Regular check up of food handlers in food and drinking establishments

2. AMOEBIC DYSENTERY (AMOEBIASIS)

- An infection with pathogenic amoebae which causes both acute and chronic diarrhea

Infectious agent

- Entamoeba histolytica (protozoan parasite)

Occurrence

- World wide, but most common in tropics & subtropics

Reservoir

- Human /lumen of large intestine/

Amoebiasis..

Mode of transmission

- Feco- oral transmission
- Ingestion of food or water contaminated by feces containing the cyst

Incubation period

- Few days to several months or years
- Commonly 2-4 weeks.

Life cycle of Ameobiasis

Life cycle

Transmission

1. cysts ingested in food, water or from hands contaminated with feces

Human host

2. cysts excyst, forming trophozoites
3. multiply in intestine
4. Trophozoites encyst
5. infective cysts passed in feces

Environment

6. Feces containing infective cysts contaminate the environment

***trophozoites passed in feces disintegrate**

Cont..

Trophozoite: any stage in a protozoan's life cycle which can ingest food. In practice also refers to the **motile form**

Cyst: the non motile form which is protected by a distinct membrane or cyst wall. This is an **infective stage of the parasite**

Excystation: the process of emergence of the trophozoite from the cyst (vs.encystations)

Pseudopod: literally means false foot; **temporary cytoplasmic processes** at the **surface of the trophozoite**

Clinical manifestations…

- Infection is usually asymptomatic but under certain circumstances which are not fully understood, the amoeba may change in to tissue parasites /trophozoites/ and invade the bowel wall causing the disease amoebiasis./bottle shaped ulcers/
- With dysentery, feces are generally watery, containing mucus and blood **and is foul smelling**
- The infection can be spread to other organs, specially the liver
- Clinical manifestation starts with a prodormal episode of diarrhea, abdominal cramps, nausea, vomiting and tenasmus

spectrums of amoebiasis

1. asymptomatic carrier state
2) acute amoebic dysentery

Intestinal ameobiasis

3) amoebic liver abscess

4) amoeboma

Amoebic dysentery

Intestinal amoebiasis

- Mostly asymptomatic cyst passage (90%)
- Symptomatic cases (10%):
- Presentation
  - - **Bloody, mucoid diarrhea**
  - - **Fever< 40 %,chills**
  - - **lower Abdominal pain**
- Diagnosis
  - Amoebic (**hematophagous trophozoites**) in stool (repeated stool exam increases the yield)
  - Mixed WBCs in stool
  - Patchy inflammation seen on colonoscopy
  - Stool PCR or antigen capture

Amoebic dysentery

- Acute presentation in young pts with prominent symptoms <10 days
- Chronic /sub acute presentation in older pts with Hepatomegaly /Wt loss/anorexia ≥ 6 months

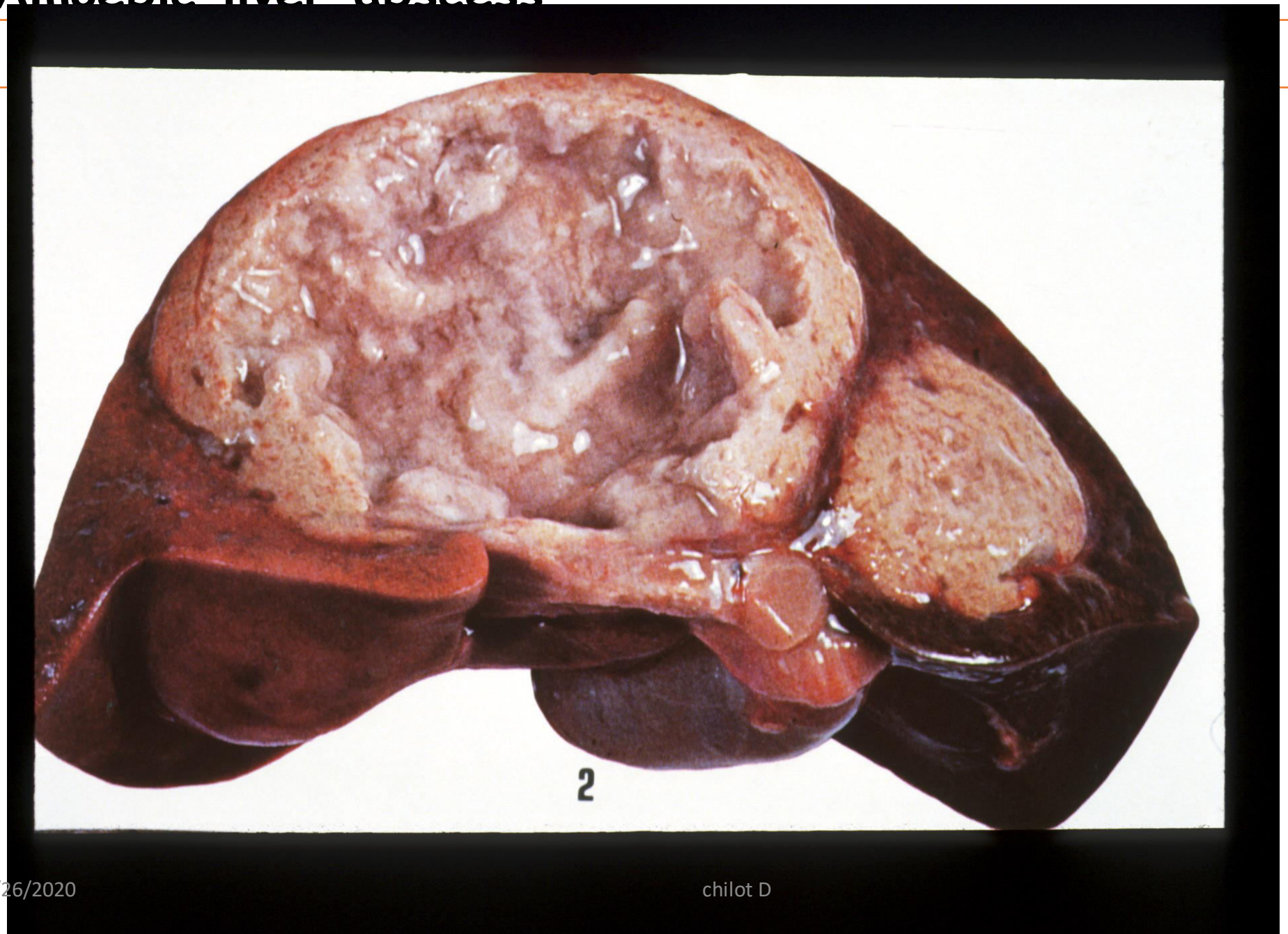
Presentation

1. **Persisting fever**
2. **RUQ or epigastric pain and/or shoulder pain**
3. **Rarely diarrhea**

Diagnosis

1. Ultrasound(U/s)
2. raised WBC
3. serology
4. aspirate microscopy
5. response to metronidazole 750mg t.i.d.

Amoebic liver abscess



Treatment of Amebiasis

Disease	Tissue agent	Luminal agent
Amebic liver abscess	Metronidazole 750mg po tid X 10d Or Tinidazole 2g po/d X 5 d	Paromomycin 10mg/kg/d po tid X 5-10 d Or Diloxanide furoate 500 mg po tid X 10d
Amebic Colitis	Metronidazole 750mg po tid X 5-10d	As above
Asymptomatic intestinal	none	As above

Prevention and control

- Adequate treatment of cases
- Provision of safe drinking water
- Proper disposal of human excreta and hand washing following defecation
- Cleaning and cooking of local foods (e.g. Raw vegetables) to avoid eating food contaminated with feces
- Ordinary chlorination will not kill the cyst (boiling is safe)

D.DX	Bacillary dysentery	Amoebic dysentery
Incubation time	Short : less than1 week	Long: 3 weeks or more
Onset	Acute	Insidious
Occurrence	Epidemic	Endemic
Fever	Common	Only in complications
Clinical picture	'Lying down dysentery '	"Walking dysentery'
Tenderness	Whole abdomen	More local (sigmoid)
Tenasmus	Very sever	Not usual
Stool macros.	Mucus and blood only	Stools with blood & mucus
Stool microscopic	•Numerous red cells •Numerous polymorphs •Few bacteria •Macrophages	•Numerous red cells in clumps •Polymorphs scanty •Many bacteria •E. Histolytica trophozoites with ingested red cells


Thank u Very much !!!

Giardiasis

- It is a protozoan infection principally of the upper small intestine

Infectious agent

- Giardia lamblia /Intestinalis/

Occurrence

- World wide
- Children are more affected than adults
- Highly prevalent in areas of poor sanitation

Reservoir

- Humans

Epidemiology

Giardia

- fecal oral spread

- prevalence **30-40% carrier rate**, more in children, and seasonal (increase during rainy seasons)

- **zoonosis** - found in most mammals; esp. beaver ("**beaver fever**"), cattle, cats, dogs, etc.

Clinical manifestations

- Ranges from asymptomatic infection to sever failure to thrive and malabsorption
- Young children usually have diarrhea
- Steatorrhoea /excess fat in a stool/: indication for failure of absorption
- Abdominal distension and bloating /tympanicity/ are frequent
- Abdominal cramps, foul-smelling diarrhea, anorexia, nausea, malaise, bloating,
- Many patients complain of sulphur testing (belching)-noisy emission of the gas from stomach via esophagus

Diagnosis and treatment

Diagnosis

- Giardia lamblia cyst or trophozoite in feces

Treatment

First line

Metronidazole

- For adults: 250 - 500mg 3x/day for 5 days
- For children:
  - 1-3 years, 500mg daily
  - 3-7 years, 600-800mg daily
  - 7-10 years, 1gm daily, all for 3 days

Alternative

Tinidazole

- For adults: 2 gm po stat
- For children: 50-75mg /kg stat

Brain storming questions

- Do you know about Cholera, clinical findings, mode of transmission and treatments?

prevention and control

- Good personal hygiene and hand washing
- Sanitary disposal of feces
- Protection of public water supply from contamination of feces
- Case treatment
- Safe water supply

CHOLERA /Acute watery diarrhea– AWD/ "ATET

- It is an acute intestinal disease characterized by sudden onset of:
  - Profuse watery stools/diarrhea/
  - Vomiting
  - Rapid dehydration and circulatory collapse.
- It is an acute illness caused by an enterotoxin elaborated by vibrio cholerae.
- It is not a systemic infection. The vibrios are confined to the intestinal canal

Infectious agent

- Vibrio cholerae (comma bacillus)
  - ✓ it is a gram -ve, very small, curved motile organisms.
  - ✓Vibrios can live in water for two weeks.
  - ✓They prefer brackish (salty water
  - ✓In sea water they may survive for 8 months
  - ✓Vibrios readily multiply in certain food such as milk and boiled rice especially when salt fish or meat is added

occurrence and importance

- Has made periodic out breaks in different parts of the world and given rise to pandemics.
- Endemic predominantly in children.
- Fatality rates of untreated classical cholera exceed 50%.
- But with adequate rehydration this can be below 1%.

cont..

Reservoir

- Humans
- For every clinical case of cholera there may be 50-100 or more asymptomatic carriers

Incubation period

- From few hrs to five days, usually 2-5 days.

Mode of transmission

- Almost all cholera infections are water-borne
- Ingestion of food or water directly or indirectly contaminated with feces or vomitus of infected person.

cont.

Period of communicability

- For the duration of stool positive stage
- Only for few days after recovery
- Antibiotics shorten the period of communicability.

Susceptibility and resistance

- Gastric achlorhydria increases risk of illness
- Because cholera vibrios are very sensitive to gastric acid, a large number of vibrios have to be ingested in order to cause disease
- For this reason cholera is **not a very infectious disease**
- Breast feeding infants are protected.

Clinical manifestations

- Most infections are asymptomatic or cause only simple self limiting diarrhea
- The clinical syndrome is due to water & electrolyte loss
- Abrupt, painless, non-offensive, profuse watery diarrhea
- The diarrhea looks like gray or **rice water**
- Fever is absent
- Sever muscular cramps in the abdomen & limbs develop from salt loss
- Vomiting follows the diarrhea
- Loss of fluid continues

cont.

- In sever cases; several liters of liquid may be lost in **few hrs** leading to:
  - Cyanosis
  - Sunken eyes and cheeks
  - Scaphoid abdomen
  - Poor skin turgor
  - Thready or absent pulse
  - Body becomes cold
  - The blood pressure is low or unrecordable
  - Stoppage of urine production and finally the patient may die of **hypovolemic shock**

cont.

Diagnosis

- Based on clinical grounds
- Stool culture for confirmation

D.DX

- Food poisoning

Treatment

- Patients are treated on "cholera beds"- beds with a central hole through which the continuous stools can pass in to a bucket & can be measured
- Monitor out put including stool out put
- Prompt replacement of fluids and electrolytes
- Rapid IV infusion of large amounts
  - isotonic sodium lactate /ringer lactate/ altering with Isotonic saline solutions /normal saline/ 50- 100 ml/min

First line

- Doxycycline 100mg 2x/day for 3 days for children 6mg/kg daily for 3 days

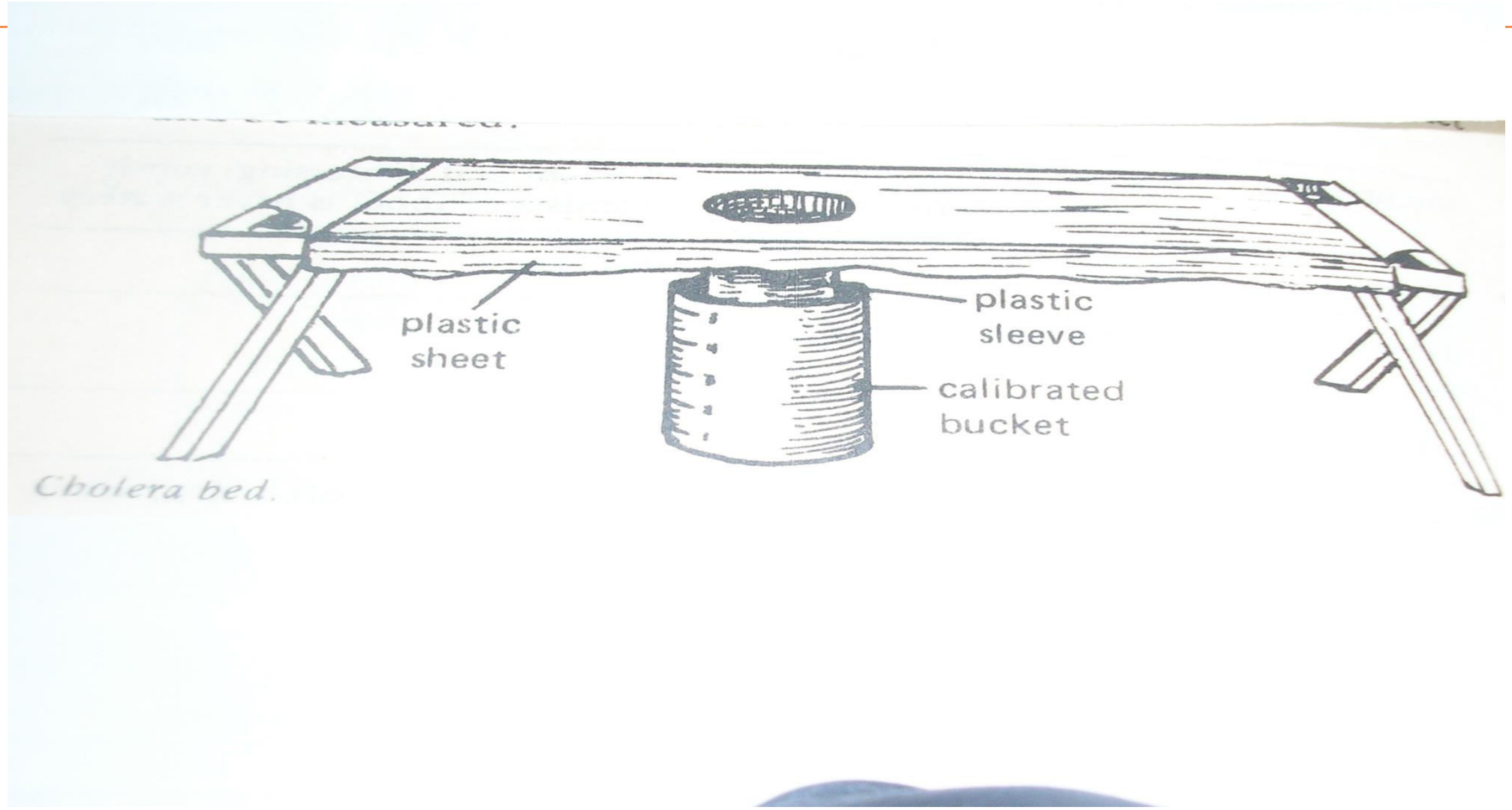
Alternative

- Tetracycline 500mg po 4x/d for 3-5 days
- Cotrimaxazole 960mg po 2x/d for 5 days
- Ciprofloxacin 500mg po 2x/d for 3-5 days

Complications

- dehydration is not corrected adequately & promptly it can lead to hypovolemic shock, acute renal failure & death.
- Electrolyte imbalance is common.
- Hypoglycemia occurs in children.
- Complications of therapy like over hydration & side effects of drug therapy are rare.

Cholera bed



Cholera bed.

prevention and Control

- Case treatment
- Safe disposal of human excreta & control of flies
- Safe public water supply (chlorination or boiling)
- Health education on hand washing and sanitary handling of food
- Protect the patient family by administering tetracycline
- Wear gown and glove to protect yourself

- Isolation
- Investigation and management of contacts
- Specific treatments
- Report cases to nearby authority

Shigellosis(bacillary dysentery

It is an acute bacterial disease involving the large and distal small intestine caused by the bacteria of the genus shigella

- **Infectious agent:** Gram-ve bacilli of shigella

Comprised of 4 species or serotypes

- Shigella dysentraie= group A (most common cause)
- Shigella flexneri = group B
- Shigella boydie = group C
- Shigella sonnei = group D

Bacillary dysentery….

Occurrence

- World wide
- Endemic in both tropics & temperate climates
- Out breaks commonly occur in over crowding and poor personal hygiene, refuge camps, mental hospitals and day care centers.

Reservoir

- Humans /children are the main reservoir/

Mode of transmission and IP

- oral transmission from a patient or carrier
- Through contaminated water and milk
- The main sanitary aspects for transmission include:
  - Faces disposal
  - Availability of water
  - Fly population
  - Seasonal influence

Incubation period

- Usually 1-3 days

Clinical manifestations

- Fever, rapid pulse, vomiting & abdominal cramp are prominent
- Diarrhea usually appears after 48 hrs with dysentery
- Generalized abdominal tenderness
- Tenasmus is present and feces are bloody, mucoid & of small quantity.
- Dehydration is common & dangerous = cause muscular cramp, oliguria and shock

Diagnosis

- Clinical grounds
- Stool microscopy (presence of pus cells and erythrocytes)
- Stool culture confirms the diagnosis

management

- Relieve pain and fever if necessary
- Fluid and electrolyte replacement. rehydration is the first priority
- Anidiarrheals should be avoided as they may slow the clearance of the organism

First line

- Ciprofloxacin 500mg po 2x/day for 3 -5 days

Alternative

- Cotrimazazole 960 mg po bid for for 5-7 days

For children:

  - 6wks- 5 months 120mg
  - 6 months – 5 yrs 240 mg
  - 6 -12 yrs 480 mg, all bid for 5-7 days
- Ceftriaxone 1-2 gm stat or 2 divided doses im or slow iv

For children:

  - 20-50mg/kg/day as single dose or 2 divided doses im or iv slowly

prevention and control

- Detection of carriers & treatment of cases
- Hand washing after toilet and before handling or eating food
- Proper excreta disposal
- Adequate and safe water supply
- Control of flies
- Cleanliness in food handling and preparation.

- Isolation
- Investigation and management of contacts
- Report cases to nearby authority

Acute Gastro Enteritis (AGE)

It is an inflammation of mucous membranes of the stomach and the intestina tract

Occurrence

- World wide; sporadically & in out breaks
- 50% of hospitalized cases of diarrheal illness is in 6-24 month of age

Causative agents

1. Viral

- Example is rotaviruses - the commonest causes in children of < 2yrs.

AGE….

2. Bacterial

- Example is escherichia coli (E.coli)
  - a resident of bowel
  - responsible for many cases of traveler's diarrhea & is still common cause o
    gastroenteritis.

3. Other causes could be:

- **Alcohol, certain drugs, food allergy and contaminated food**

Mode of transmission

- Fecal-oral route- either directly or indirectly

AGE..

Incubation period

- 2 days for E. coli
- 1 day for viral GE

Clinical manifestations

- Fever, vomiting and diarrhea (watery)
- Dehydration in sever cases
- Could be mild and self limiting or sever and prolonged

AGE…

- ELISA to detect rota virus antigen
- Electron microscopy of stool to demonstrate the virus
- Stool culture to isolate E. coli

AGE..

Treatment

- Give analgesics if required
- It is mainly directed at maintenance of correcting fluid balance
- Antibiotic treatment is indicated for sever cases

First line

- Cotrimaxazole 960 mg 2x/d for 5-7 days.

  /For children dose: see the previous dose/

Alternative

- Ciprofloxacine 500mg po 2x/d for 3-5 days
- Chloramphenicol 500 mg po 4x/d for 7 days. For children: 25mg/kg/day

Prevention and control

- Hygienic measures applicable to diseases transmitted via fecal – oral route.

Cases

1. A-5- year old male child presented with repetitive and prolonged cough with inspiratory whoop and he also vomits at the end of cough. On P/E the child is cyanotic and pulse rate is 140beat/min. what is the most likely diagnosis?

   A. Pneumonia
   B. Relapsing fever
   C. Pertussis
   D. Tuberculosis

2. Based on above scenario which of the following is correct statement?

   A. Streptococcus pneumonia is causative agent
   B. passive immunization is the prevention mechanism
   C. One attack usually confers lifelong immunity.
   D. It is contagious disease with high attack rate.

3. You were assign in medical OPD and a 14 years old adolescent present with fever bloodydiareha, abdominal cramp and tenesmus for the investigation. what is the most likely dx of the patient?

4. Based on the above scenario what is its medical treatment ?

Helminthes/parasitic worms/

Two phylums of parasitic worms:

1. Phylum Aschelminthes
   - ➢Class nematoda
     - ✓Nematodes or round worms e.g. Ascaris
2. Phylum platyhelminthes /Flat worms/
   - ➢ Class trematoda /trematodes or flukes/
     - ✓Blood flukes e.g. schistosomes
     - ✓Liver flukes
     - ✓Intestinal flukes
     - ✓Lung flukes
   - ➢Class cestoda
     - ✓Cestodes or tapeworms

General features of nematodes

. Adult location in the body

1. Intestinal nematodes
   - Small intestine: ascaris, hook worm, strongyloides, trichinella
   - Large intestine: enterobius, trichuris

2. Tissue nematodes
   - Lymphatic: wuchereria, brugia
   - Subcutaneous:Loa loa, onchocerca, drucunculus

3. Mesentry: masonella

4. Conjunctiva: Loa loa

General features of nematodes…

Based on whether they lay eggs or larvae

- Oviparous – laying eggs
  - ascaris, hook worm, enterobius, trichuris
- Viviparous- producing larvae
  - wuchereria, brugia, drucunculus, trichinella
- Ovoviviparous: laying eggs containing fully formed larvae which hatch ou immediately
  - strongyloides

General features of tape worms

- Do not have body cavity or alimentary canal
- Hermaphrodites / male and female organ/
- Segmented worms /tape-like/
- The adult consists of three parts: the scolex /head/, neck and trunk
- Humans are definite hosts except for dog and pork tape worms/

General features of trematodes /flukes/

- Trematodes are unsegmented helminths
- They are flat and broad shape resembling the leaf of a tree or a flatfish /fluke means flatfish/
- They are hermaphroditic except for schistosomes
- They are oviparous

ASCARIASIS (ROUND WORM

- It is a helminthic infection of the small intestine generally associated with few or no symptoms
- The adult male measures 15-30cm, while the female is 20-40cm long.

Infectious agent

- Ascaris lumbricoides

occurrence and importance

- The most common parasite of humans where sanitation is poor
- 1.47 billion people infected world wide
- School children from 5-10 yrs are most affected
- Highly prevalent in moist tropical countries (not too dry but loose soil)
- A female may produce up to 200,000 eggs daily

Occurrence and importance…

- Two types of eggs are passed by the worms:
  - The **Fertilized eggs**, laid by females inseminated by mating with a male, are embryonated and develop in to the infective eggs
  - The uninseminated female also lays eggs, but these are non-embryonated and cannot become infective. These are called ***unfertilized eggs***
- The eggs are resistant to adverse conditions and can survive for several years

Ascaris

Reservoir

- Humans /eggs in soil/

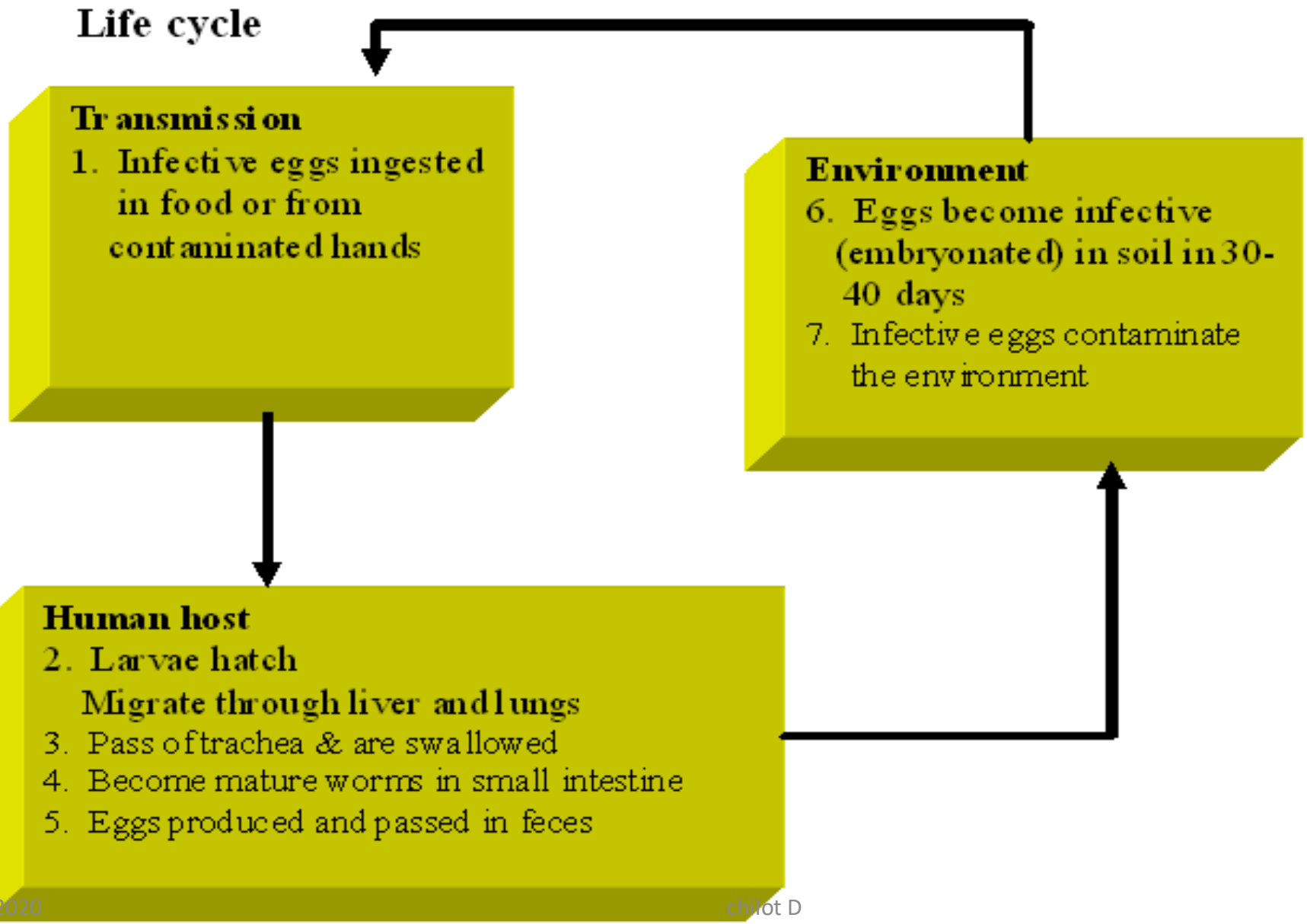
Mode of transmission

- Ingestion of infective eggs /embryonated eggs/ from the soil contaminated with human feces.
- Uncooked vegetable (e.g. salad) contaminated with soil containing infective eggs (especially when the field has been manured with human feces)
- No transmission from fresh feces
- No direct transmission from person to person

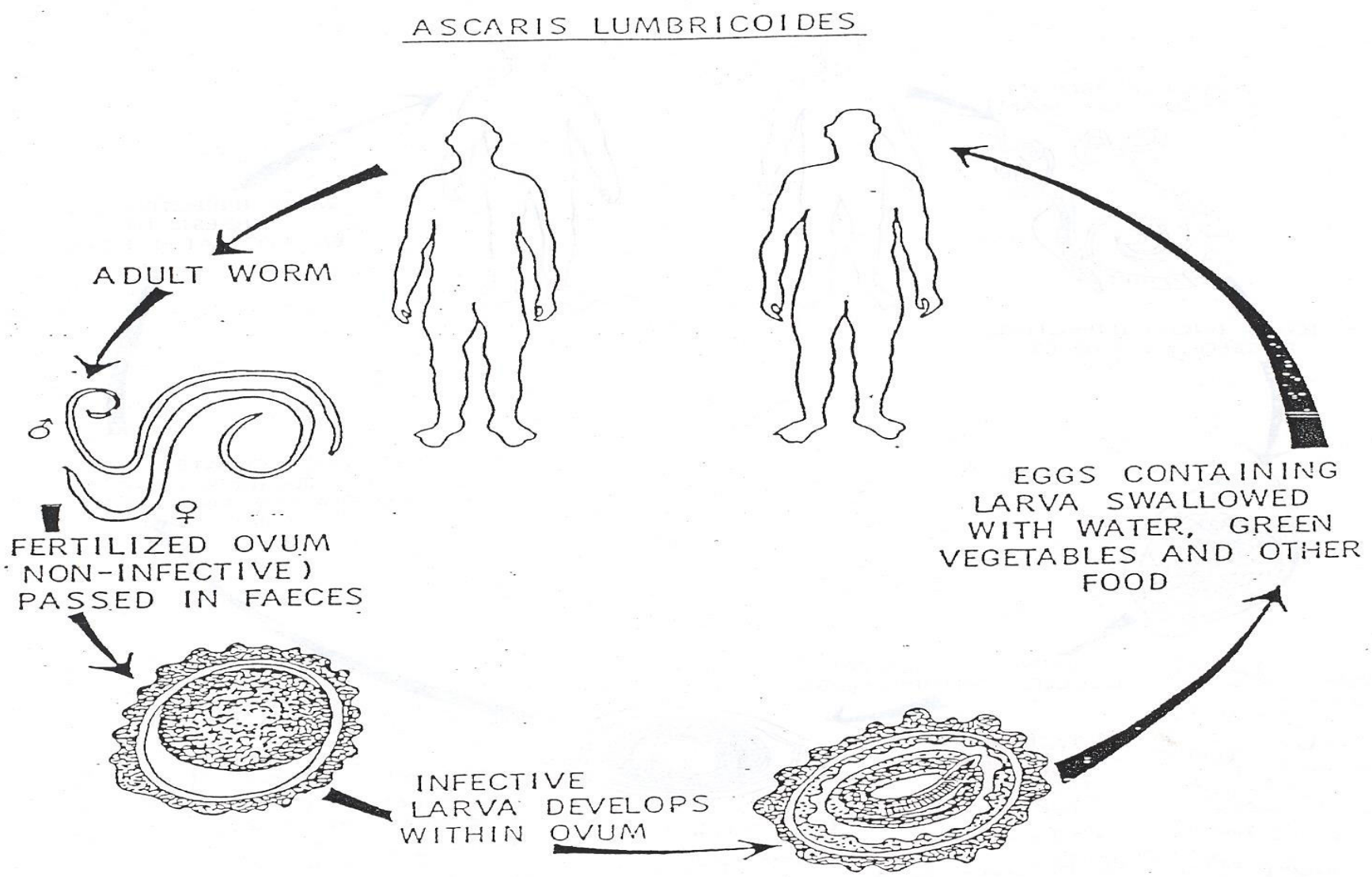
Incubation period

- 4-8 wks.

Ascaris


Life cycle
Transmission
1. Infective eggs ingested in food or from contaminated hands
Human host
2. Larvae hatch
Migrate through liver and lungs
3. Pass of trachea & are swallowed
4. Become mature worms in small intestine
5. Eggs produced and passed in feces
Environment
6. Eggs become infective (embryonated) in soil in 30-40 days
7. Infective eggs contaminate the environment

Ascaris…


ASCARIS LUMBRICOIDES
ADULT WORM
♂
♀
FERTILIZED OVUM
NON-INFECTIVE)
PASSED IN FAECES
INFECTIVE
LARVA DEVELOPS
WITHIN OVUM
EGGS CONTAINING
LARVA SWALLOWED
WITH WATER, GREEN
VEGETABLES AND OTHER
FOOD

Clinical manifestation

- Most infections go unnoticed until large worm is passed in feces and occasionally through the mouth & nose
- Migrant larvae may cause: itching, wheezing, dyspnea, fever & cough /productive of bloody sputum may occur/
- Abdominal pain may arise from intestinal or duct (billary, pancreatic) obstruction

Clinical manifestation

- The worm may wonder up or down along the gut.
- Going up, it may enter the opening of the biliary or pancreatic duct causing acute biliary obstruction or pancreatitis
- It may enter the liver parenchyma, where it may lead to abscess
- The worm may go up the esophagus and come out through the mouth or nose
- It may crawl into the trachea and the lung causing respiratory obstruction or lung abcess

Clinical manifestation

- Migrating downwards, the worm may cause obstructive appendicitis.
- It may lead to peritonitis when it perforates, generally at weak spots such as typhoid or tuberculosis ulcers or through suture line
- Wondering of the worms which may also be provoked by treatment
- Serious complications include **bowel obstruction** due to knotted interwined worms at ileo-caecal junction which could be partial or complete
- The adult worms interfere with proper digestion and absorption of food leading to malnutrition

Diagnosis and treatment

Diagnosis

- Microscopic identification of eggs in a stool sample
- Adult worms passed from anus, mouth or nose.

Treatment

First line

- Piperazine 4gm single dose
  - for cildren:
    - 9 -12 yrs 3.7gm
    - 6 - 8yrs 3gm
    - 4 -5 yrs 2.25gm
    - 1-3 yrs1.5gm
    - Less than 1 yr 120mg/kg, all as a single dose

Alternative

- Albendazole – 400mg po stat. for children 1-2 yrs: 200mg po stat
- Mebendazole – 500mg single dose stat
- Levamisole (ketrax) -2.5 mg/kg stat

Prevention and control

- Treatment of cases
- Sanitary disposal of feces
- Prevent soil contamination in areas where children play
- Promote good personal hygiene

HOOK WORM DISEASES (ANCYLOSTOMIASIS, NECATORIASIS)

- is a common chronic parasitic infection with a variety of symptoms usually in a proportion of the **degree of anemia**
- The male Ancylostoma duodenale worm is about 8-11mm long where as the female is 10-13mm long
- The male Necator Americans worm is about 7-9mm long where as the female is 9-11mm long

Infectious agent

- Ancylostoma duodenale and Necator americanus
- In Greek: Ankylos – hooked. Stoma - mouth
- In Latin: necator: Murderer

Hook worm larva, filariform

HOOK WORM DISEASES….

Occurrence and importance

- Widely endemic in tropical & subtropical countries
- Nector is more truly tropical than ancylostoma as its larva require a higher temperature to develop
- Necator is more common species
- Primarily a rural disease associated with all types of cultivation (e.g. coffee, banana plantation)

Reservoir

- Humans

HOOK WORM DISEASES….

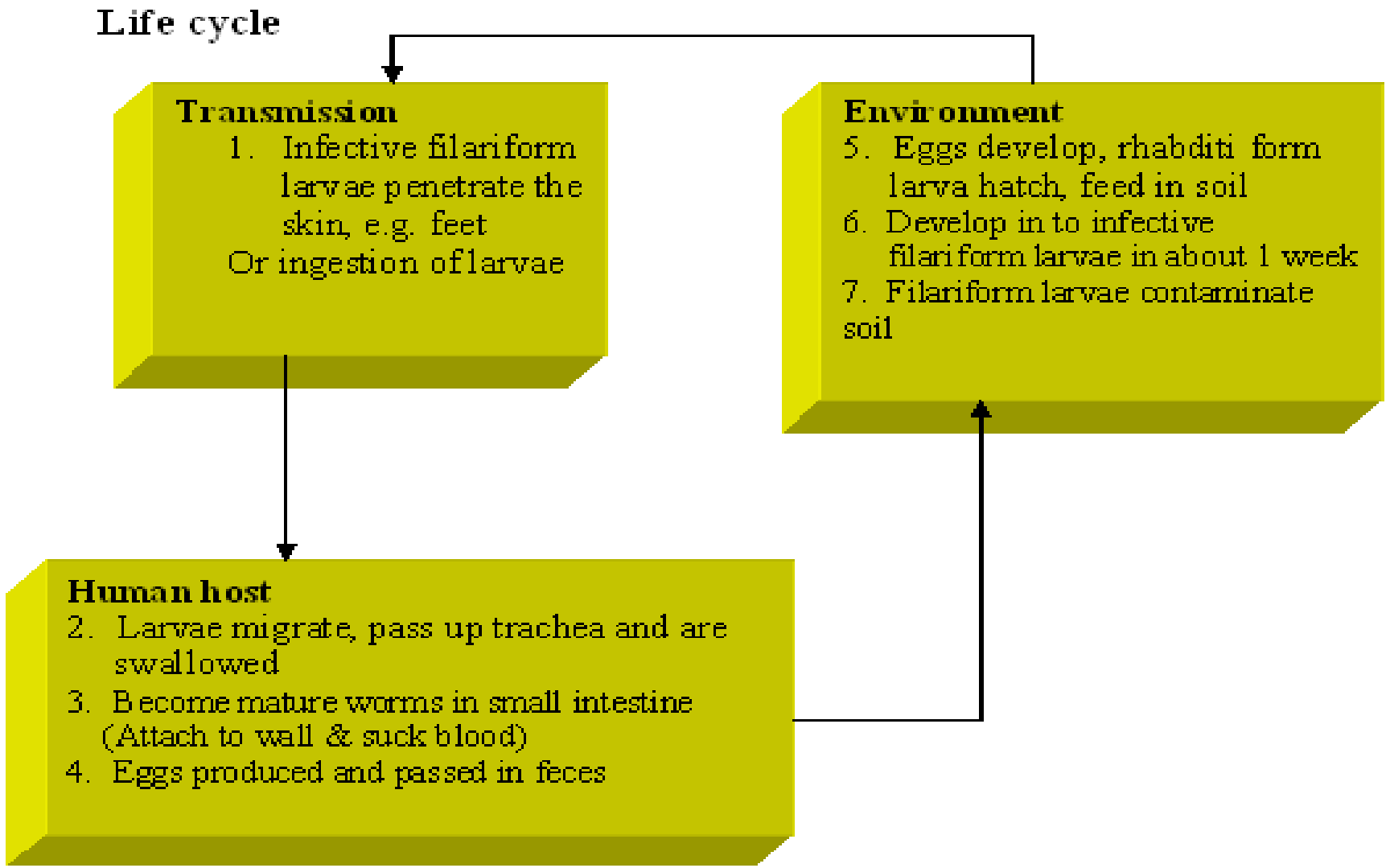
Mode of transmission

- Penetration of **unbroken** skin by infective larvae (L3)
- Oral ingestion on unwashed vegetables (L3) -more common with A.d than with N.a

Incubation period

- Few weeks to many months
- Depends on intensity of infection & iron intake of the host
- r

HOOK WORM DISEASES …



Clinical manifestation

- Larval migration produces transient localized maculo papular rash associated with itching, **ground itch**
- Migration of larvae to lungs produces cough, wheezing & transient pneumonitis
- In light infection /up to 100 worms/ there is no symptoms & blood loss is 3 ml/day
- In heavy infection /500- 1000 or more worms there is epigastric pain and tenderness & blood loss is 100ml/day which results significant blood loss and anemia
- Egg counts of 20 or more in the feces are associated with significant anemia

HOOK WORM DISEASES (ANCYLOSTOMIASIS, NECATORIASIS)

- Anemia→ manifested by exertional dyspnea, weakness and light headedness which results cardiac insufficiency
- Physical and mental stunting in children
- **Blood loss** is the most significant consequence of hook worm infection

Diagnosis

- Demonstration of eggs in stool specimen

HOOK WORM DISEASES….

Treatment

First line

- Mebendazole 100mg po 2x/d for 03 days

Alternative

- Albendazole 400mg po stat
- Levamisol 2.5 mg/kg stat repeated after 7 days for Nector. A
- Treat anemia with $Feso_4$ 1 tab 3x/d po for 1-2 months

Prevention and control

- sanitary disposal of feces, proper hand and vegetable washing
- wearing of shoes and case treatment

TRICHURIASIS (WHIP WORM)

- It is a nematode infection of the large intestine, usually asymptomatic in nature
- The male 30- 45 mm, while the female is about 40-50mm long
- The fertilized female lays about 5000 eggs per day
- In Greek, trichuris is to mean hair like tail

Infectious agent

- Trichuris trichuria

TRICHURIASIS…

Occurrence

- World wide, especially in warm moist regions
- Common in children 3-11 yrs of age

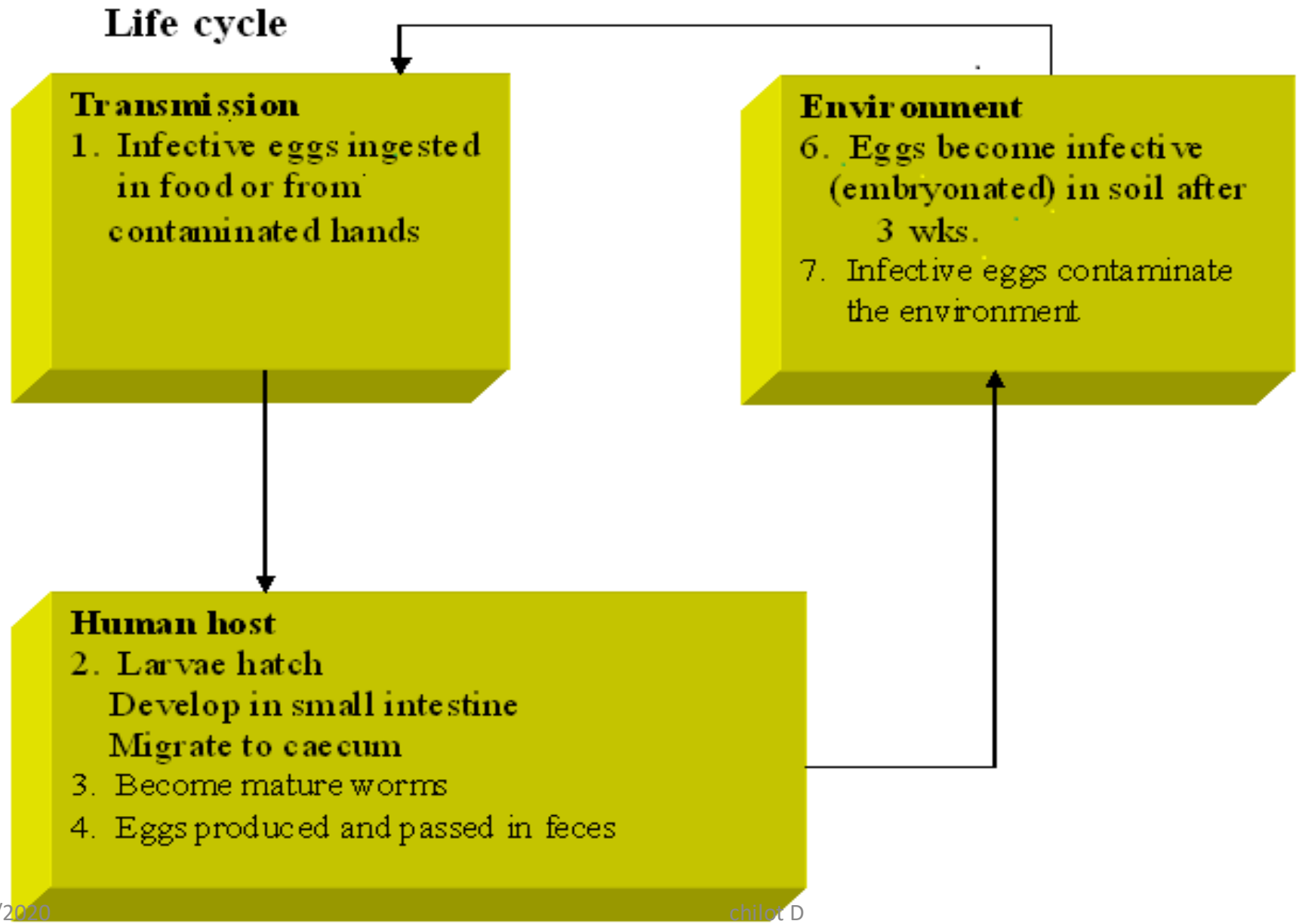
Reservoir

- Humans

Mode of transmission

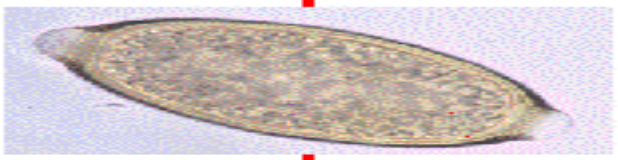
- Indirect, particularly through pica or ingestion of contaminated vegetables
- Not immediately transmissible from person to person

TRICHURIASIS, life cycle



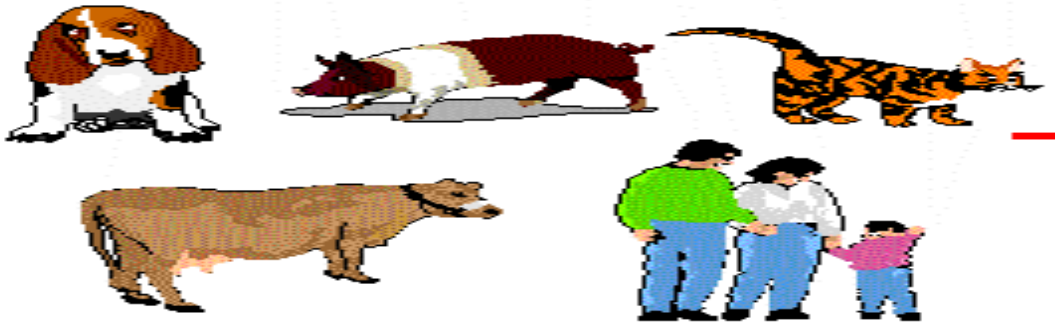
THE LIFE CYCLE OF *TRICHURIS* SPP. (THE WHIPWORMS)

The adult males and females occur in the host's large intestine.



Eggs are passed in the host's feces.

The juveniles within the eggs mature into infective juveniles.



The eggs are ingested by the appropriate host.

The eggs hatch in the host's small intestine.

The juveniles migrate into the large intestine.

Males and females reach sexual maturity and mate.

(Parasites and Parasitological Resources)

Clinical Manifestation

- Severity is directly related to the number of infective worms
- Most infected people are asymptomatic
- Abdominal pain, tiredness, nausea & vomiting
- Mucus diarrhoea, chronic dysentery, loss of weight, anemia
- **Rectal prolapse** in heavily infected very young children

TRICHURIASIS...

Diagnosis

- Demonstration of eggs in feces

Treatment

- Albendazole- 400mg po stat or
- Mebendazole- 100 mg 2xld for 03 days

Prevention and control

- Sanitary disposal of feces
- Maintaining good personal hygiene
- Cutting nails especially in children
- Treatment of cases

ENTEROBIASIS (OXYURIASIS, PIN WORM INFECTION, THREAD WORM)

- It is a common intestinal helminthic infection that is often asymptomatic
- The adult male is 2-5 mm long, while the female is 8-13mm long
- The word enterobius vermicularis in Greek is to mean tiny worm living in the intestine

Infectious agent

- Enterobius vermicularis

ENTEROBIASIS...

Occurrence

- World wide
- Prevalence is high in school aged children followed by pr-school children
- Infection usually occurs in more than one family member

Reservoir

- Human

Incubation period

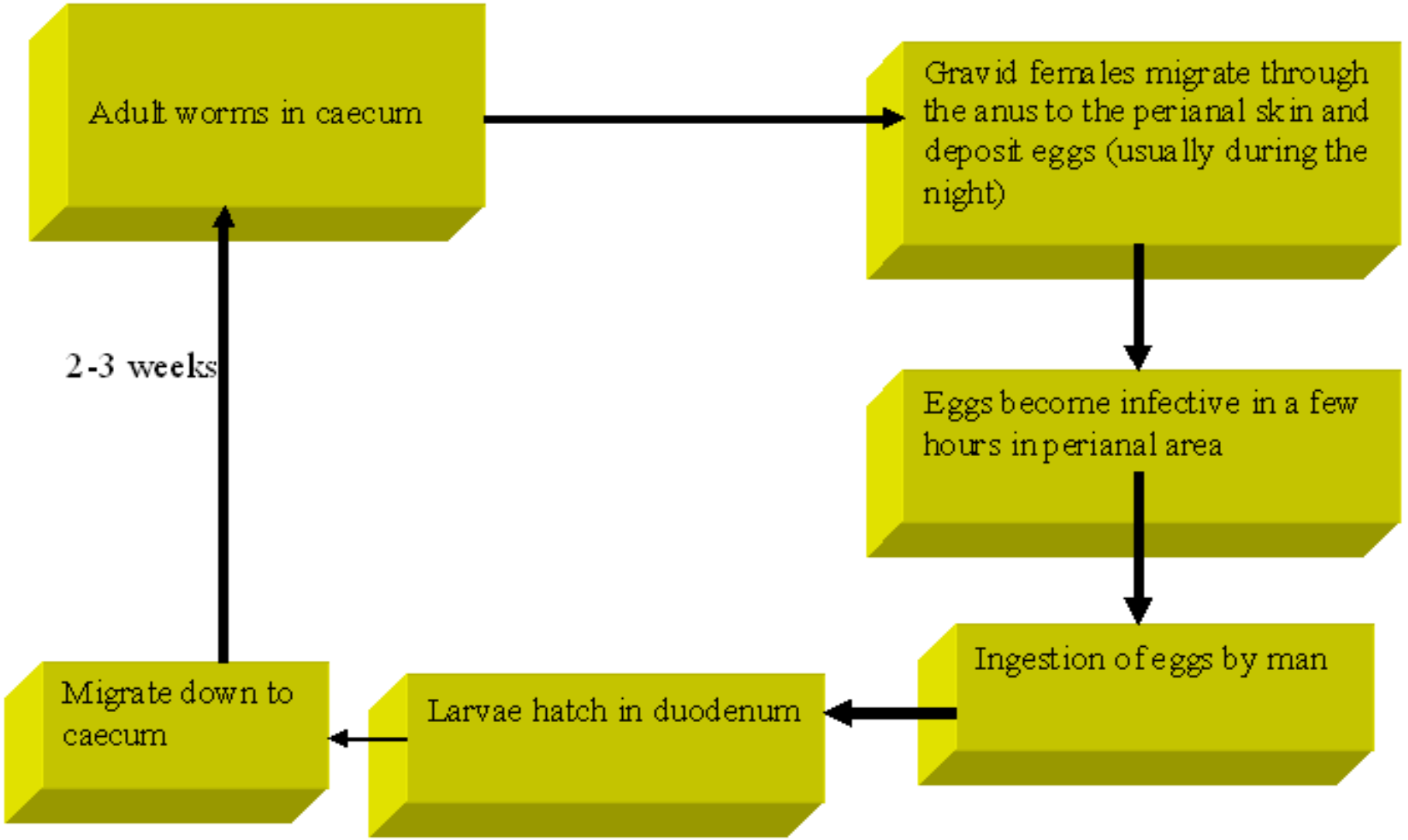
- 2-6 weeks

ENTROBIASIS...

- Mode of transmission
- Direct transfer of infective eggs by hand from anus to mouth of the same person (auto re- infection) or another person
- Indirect transmission through clothing, bedding, food or other articles contaminated with eggs.
- Retro infection- in moist conditions eggs on the peri anal skin may hatch – larvae-enter the host via anus.
- Air born infection through inhalation of dust containing eggs

ENTROBIASIS…

Life cycle



THE LIFE CYCLE OF *ENTEROBIUS VERMICULARIS* (THE HUMAN PINWORM)

Humans are infected when they ingest eggs containing infective juveniles.

Eggs hatch in the small intestine and male and female worms migrate to the large intestine and reach sexual maturity.

Females crawl out of the anus (usually during the early morning hours) and deposit eggs on the perianal skin

RETROINFECTION

If eggs remain on the perianal skin long enough they will hatch, and the juveniles will crawl back into the anus and mature into adults.

Eggs are deposited on the perianal skin.

Eggs become infective within six hours.

Hands, bed clothing, bed linens, floors, drapes, kitchen counters, clothing, school rooms, desk tops, etc.,are contaminated with infective eggs.

Clinical manifestation

- Peri anal itching, appendicitis
- Lose of appetite, nausea, disturbed sleep & diarrhea
- Some times secondary infection of the scratched skin

Diagnosis

- Stool microscopy for eggs or female worms
- Applying transparent adhesive tape over the anus in early morning- search the eggs under microscope
- Eggs are not usually found in the faeces
- Adults may be seen on the surface of stools

Enrobiasis....

Treatment

- Treat the whole family
- Single dose of mebendazole or albendazole
- Repeated treatment after 2-4 weeks is needed because of re-infection

Prevention and control

- Educate the public about hygiene: hand washing, keeping nails short, discourage nail biting
- Treatment of cases
- Reduce over crowding in living accommodations
- Provide adequate toilets.

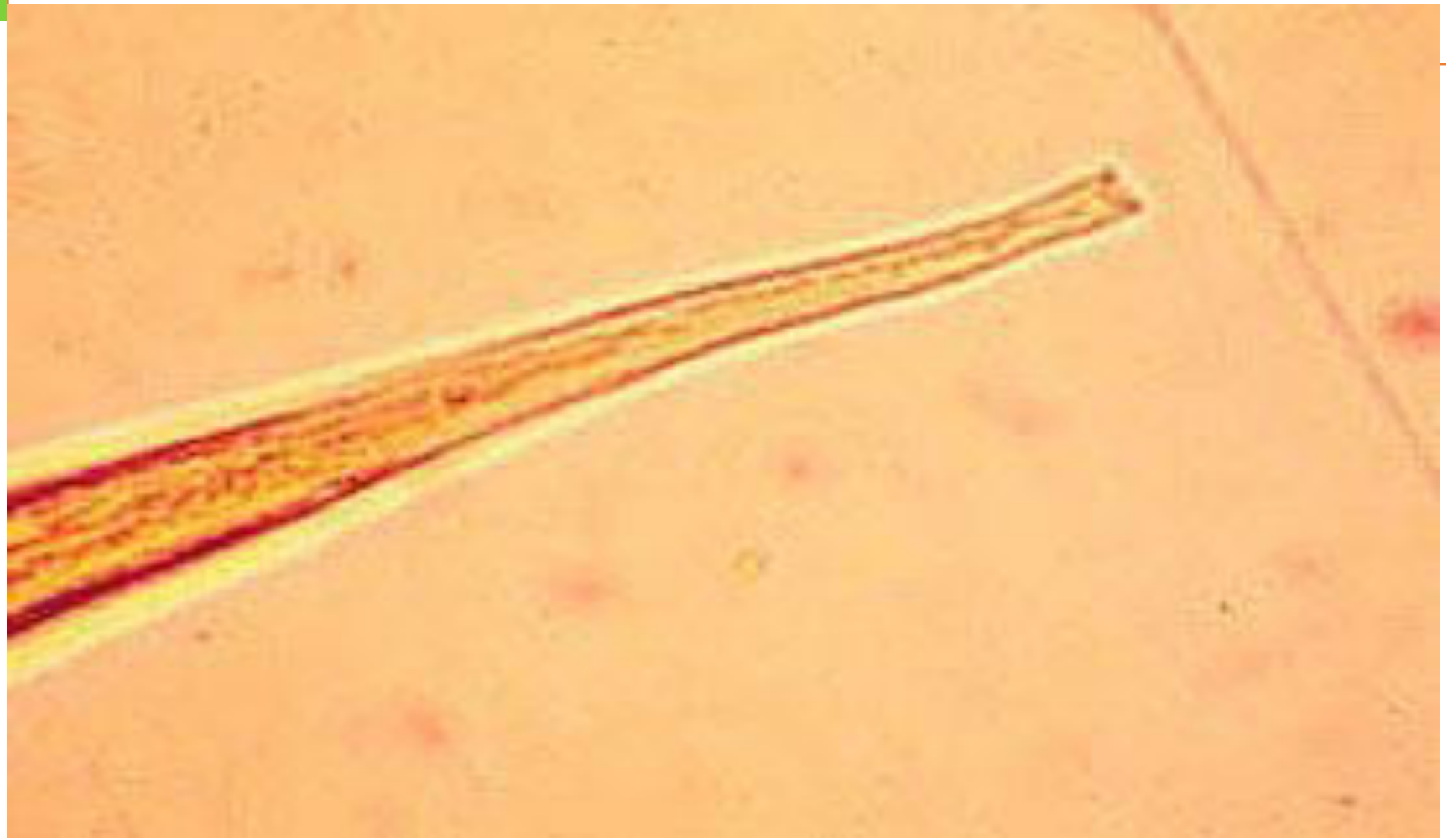
STRONGYLOIDIASIS

- It is often asymptomatic helminthic infection of the duodenum and upper jejunum
- Only the female worms are seen in the intestine
- They are parthenogenetic /produce offspring without being fertilized by the male/
- The female worm is about 2.5mm long

Infectious agent

- Strongyloides stercolaris

Strongyloides larva, filari form



STRONGYLOIDIASIS ….

Occurrence

- In tropical and temperate areas
- More common in warm and wet regions

Reservoir

- Human

STRONGYLOIDIASIS ….

Mode of transmissions

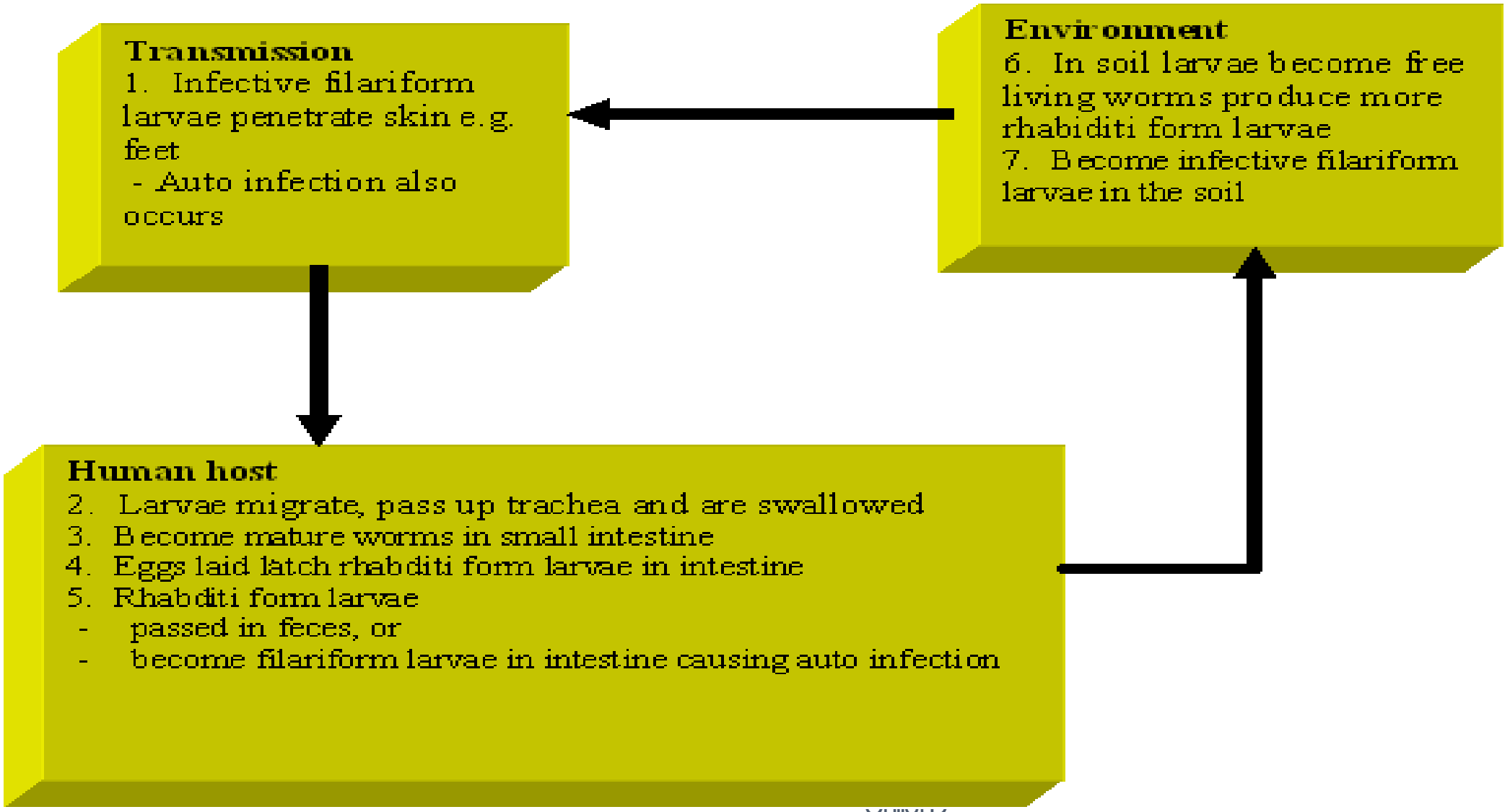
- Penetration of skin (usually feet), perianal region and intestine by infective (filariform, L3) larvae

Incubation period

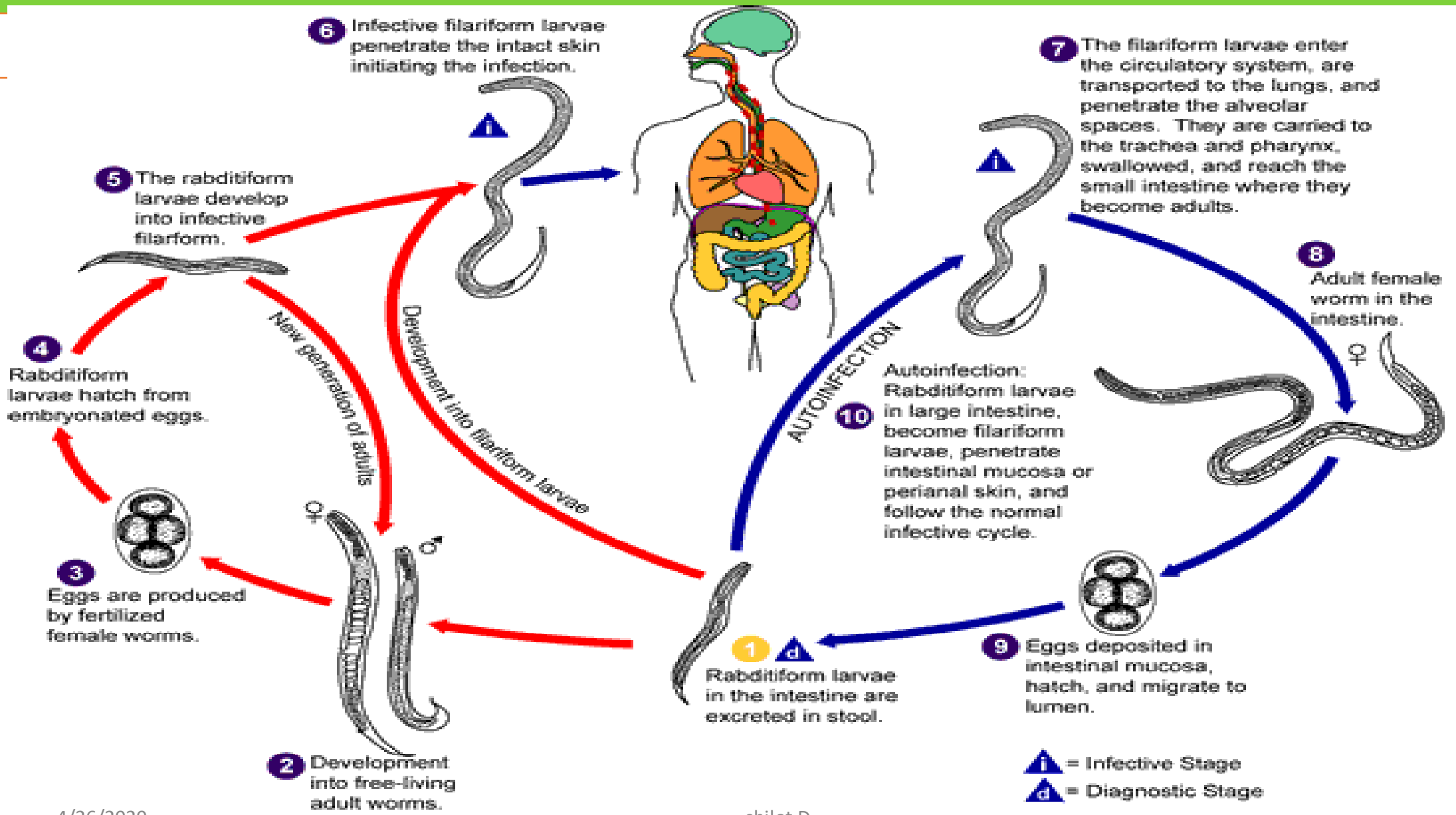
- 2-4 weeks (from skin penetration up to appearance of rhabditi (L1) form larva in the feces

STRONGYLOIDIASIS ….

Life Cycle



Strongyloides, life cycle



STRONGYLOIDIASIS ….

Clinical manifestations

- Pneumonia occurs during heavy larval migration
- Mild peptic ulcer like epigastric discomfort to severe watery diarrhea
- Heavy infection may result in malabsorption syndrome

Diagnosis

- Identification of larvae in stool specimen

Treatment

First line

- Thiabendazole (Mintezol)
  - 1500mg po bid, for children: 25mg/kg po for 2 days
  - For disseminated strongyloidiasis in AIDS: monthly treatment is needed

Alternative

- Albendazole 400mg 2x/d for 03 days
- Ivermectin 200 micro- gram/kg single dose – repeated after 2 wks.

Prevention and control

- Proper disposal of human excreta
- Personal hygiene including use of foot wear
- Case treatment


Thank u Very much !!!

Epidemiological surveillance

OUTLINES

- ✓Definition
- ✓Purpose and Characteristics of Public Health Surveillance
- ✓Types of surveillance
- ✓Activities in surveillance
- ✓The integrated disease surveillance system

Definition

- Surveillance is a continuous and systematic collection, analysis, interpretation and dissemination of health data in an ongoing basis.
- Surveillance provides "information for action" which can be used to investigate, prevent, and control disease in communities.
- Surveillance can be conducted globally, regional, national, or institutional.

Purpose of Public Health Surveillance

Purpose

- To identify diseases, injuries, hazards and other health related factors as early as possible,
- To predict early detection of outbreaks.
- To provide scientific baseline data and information for priority setting, planning, implementing
- To follow secular (long-term) trends of a disease.

Cont…

- To define the magnitude and distribution of diseases by time, person and place dimension.
- To evaluating disease control program for health problems.
- To identify changes in agent, host and environmen factors
- To test hypothesis

Types of surveillance

- The are three common types of surveillance such as passive, active and sentinel surveillance.

Passive surveillance

- Passive surveillance may be defined as a mechanism for routine surveillance based on passive case detection and on the routine recording and reporting system.

Count…

- The information provider comes to the health institutions for help, be it medical or other preventive health services.
- It involves collection of data as part of routine provision of health services.

Advantages of passive surveillance

- ❑covers a wide range of problems
- ❑does not require special *arrangement*
- ❑ it is relatively cheap
- ❑ covers a wider area

Disadvantages of passive surveillance

- The information generated is to a large extent unreliable, incomplete and inaccurate

Disad…

- passive surveillance is not available on time
- you may not get the kind of information you desire
- It lacks representativeness of the whole population.

B. Active surveillance

- Active surveillance is defined as a method of data collection usually on a specific disease, for relatively limited period of time.
- It involves collection of data from communities such as in house-to-house surveys or mobilizing communities to some central point where data can be collected.

Active surveillance..

- This can be arranged by assigning health personnel to collect information on presence or absence of new cases of a particular disease at regular intervals.

Example: investigation of out-breaks

Advantages of active surveillance

- the collected data is complete and accurate
- information collected is timely.

Disadvantages of active surveillance

- it requires good organization,
- it is expensive
- it requires skilled human power
- it is for short period of time
- it is directed towards specific disease conditions

C. Sentinel surveillance

- Sentinel surveillance uses a **pre-arranged sample of reporting sources** to report all cases of one or more conditions.

This is carried out by:

- Selecting sample sources most likely to see cases of the specified condition.
- It provides a practical alternative to population-based surveillance, in developing countries.

Cont…

- Identifying institutions that serve the population subgroups and that can obtain data regarding the condition of interest.

Advantage

- Relatively inexpensive
- Provides a practical alternative to population-based surveillance
- Can use of data collected for other purposes

Disadvantage

- The selected population may not be representative of the whole population
- use of secondary data may lead to data of lesser quality and timeliness

Steps in Planning surveillance

- Establish objectives
- Develop case definitions
- Determine data source
- Develop data collection instruments
- Collection of Data
- Compilation and Analysis of Data
- Formulation of Recommendation

Criteria for identifying disease for surveillance

- **Frequency**
  - Incidence
  - Prevalence
- **Severity**
  - Case fatality ratio
  - Hospitalization rate
  - Years of potential life lost etc…

Cont...

- Cost
- Preventability
- Communicability
- Public Interest

Activities in Surveillance

- The different activities that carried out under surveillance are:

➢ Data collection and recording

➢ Data compilation, analysis and interpretation

➢ Reporting and notification

➢ Dissemination of information

Data collection and recording

Basic techniques of data collection include the following:

- Record review
- Interviews
- Surveys using questionnaires
- Observation.

Sources of data for surveillance

The major sources summarized by the WHO in 1968 are:

- Mortality and Morbidity registration
- Reports of laboratory utilization
- Reports of individual case investigations
- Reports of epidemic field investigations
- Information on animal reservoir and vector distribution
- Report of biologics and drug utilization
- Knowledge of the population and environment

Data compilation, analysis and interpretation

- The data should be collected at each level of the health care delivery system.
- Quality of data collected should be accurate, complete, reliable, and submitted on time.
- surveillance data is analyzed in terms of **time, place** and **person**
- Analysis at the health facility level helps to recognize problems timely and to take appropriate action immediately.

Reporting and notification

- Reporting formats must be clear and easy to use.
- Any report must be clear and answer questions like what, where, when, to whom, for what and why.

Types of reports

- Oral
- Radio or telephone
- Written

Dissemination of information

To ensure motivation and active involvement there must be:

- Preparation of regular weekly, monthly, quarterly and annual reports
- Regular feedback from higher levels
- Publication of newsletters

Features of a good surveillance system

- Using a combination of both active and passive surveillance techniques.
- Timely notification
- Timely and comprehensive action taken in response to notification.
- Availability of a strong laboratory service for accurate diagnoses of cases.

Attributes of surveillance

- Simplicity,
- Flexibility,
- Acceptability,
- Sensitivity,
- Predictive value positive,
- Representativeness,
- Timeliness and
- stability.

The integrated disease surveillance system

- The integrated disease surveillance system is a relatively new strategy, which is being implemented in Ethiopia.
- In this strategy several activities from the different vertical programs are coordinated and streamlined in order to make best use of scarce resources.

Integ…

- The activities are combined taking advantage of similar surveillance functions, skills, resources, and target population.
- Integrated disease surveillance strategy recommends coordination and integration of surveillance activities for diseases of public health importance.

Diseases included in the integrated disease surveillance system

- Among the most prevalent health problems of communicable diseases some of them are selected for integrated disease surveillance to be implemented in Ethiopia.
- The diseases are recommended because they fall into one or more of the following categories:

Count…

- Are top causes of high morbidity and mortality in Ethiopia (for example, malaria, pneumonia, diarrheal diseases, tuberculosis, and HIV/AIDS).
- Have epidemic potential (for example yellow fever and cholera)
- Surveillance required internationally (for example plague, yellow fever and cholera)

Count …

- Have available effective control and prevention interventions for addressing the public health problem they pose (for example schistosomiasis, onchocerciasis and trypanosomiasis)
- Can easily be identified using simple case definition.

List of Priority Disease in Ethiopia

A. Epidemic-Prone Diseases

- Cholera
- Diarrhea with blood (Shigella)
- Yellow fever
- Measles
- Meningitis

Count…

- Plague
- Viral hemorrhagic fevers
- Typhoid fever
- Relapsing fever
- Epidemic typhus
- Malaria

B. **Diseases Targeted for Eradication and Elimination**

- Acute flaccid paralysis (AFP)
- Dracunculiasis (Guinea worm)
- Leprosy
- Neonatal tetanus

C. Other Diseases of Public Health Importance

- Pneumonia and Diarrhea in children less than 5 years of age
- New AIDS cases
- Onchocerciasis
- Sexually Transmitted Infections (STIs)
- Tuberculosis

VECTOR-BORNE COMMUNICABLE DISEASES

5.1 Learning objectives

At the end of this unit the learners should be able to:

- describe what arthropod or intermediate vector-borne disease means
- identify the common vectors which transmit disease to man
- list the common vector-borne diseases
- participate in diagnosis and treatment of vector-borne diseases
- implement the common preventive and control methods of vector-borne diseases

Introduction

- **Generally** speaking a vector is any carrier of disease
- But in case of the vector- borne diseases, we restrict the word to those invertebrate hosts (insects or snails) which are an essential part of the life cycle of the disease organism.
- Insect vectors usually acquire the disease organism by **sucking blood** from infected persons, and pass it on, later by the same route.
- There are other routes, however, infection may enter **skin cracks**, or **abrasions** either from infected feces deposited when feeding, or from body fluid when an insect is crushed

Introduction …

- Arthropod borne communicable diseases included in this unit:
  - Malaria
  - Filariasis
  - Guinea worm
  - Typhus
  - Relapsing fever
  - Schistosomiasis
  - Yellow fever
  - Leishmaniasis
  - Trpanosomiasis

MALARIA

- An acute infection of the blood caused by protozoa of the genus plasmodium

Infectious agent

- P. falciparum (malignant tertian malaria) – invades all ages of RBCs
- P. vivax (benign tertian malaria)- invades reticulocyts only
- P. ovale (benign tertian malaria)- invades reticulocyts only
- P. malariae (quartan malaria) - invades reticulocytes only.

Occurrence and importance

- Malaria is endemic in tropical & sub tropical countries of the world
- It affects 40% of the world population
- Globally about 300-500 million people suffer from malaria each year
- More than 90% of the cases are from sub-Saharan Africa
- Each year 1.7 to 2 million people die from malaria. Majority of these deaths occur in Africa
- Children less then 5 yrs of age, pregnant women, and travelers to endemic areas are risk groups.
- It causes abortion, still birth, premature labour, anemia and maternal death in pregnant mothers
- Two-third of Ethiopian population is at risk of malaria epidemic
- Malaria accounts 60% P. falciparum and 40% P.vivax in Ethiopia. The other two account less than one percent

- Case fatality rate of sever and complicated malaria is about 10% in hospitalized adults & 33% in children less than 12 years

Pathogenesis

- Infection follows the entry of plasmodia sporozoites from the salivary gland of female anopheles mosquitos in to the blood stream of human beings during blood meals.
- The parasites are carried by the blood stream in to the liver where they invade the paranchymal cells of the liver and reproduce asexually.
- This is known as intrahepatic or exo-erythrocytic phase or schizogony.
- Each sporozoit produces many thousands of daughter merozoites.
- This process leads to swelling of the invaded liver cells and soon after follows bursting of the cells that is accompanied by release of motile merozoites in to the blood stream
- This is the time where the symptomatic stage of infection occurs.

Pathogenesis…

- The merozoites rapidely invade red blood cells and become trophozoites. This development phase is called intra-erythrocytic phase.
- The parasites consume hemoglobin and occupy most of the red cell.
- Multiple divisions occur especially of P. palciprum in the erythrocytic stage.
- This multiplication takes place every 48 hours. That is, each attacking one RBC and then produces 8-24 merozoites, which on their turn would repeat same cycle.
- This justifies the importance of early treatment for malaria

Epidemiological pattern / types / of malaria in Ethiopia

- There are different epidemiological patterns of malaria in different communities
- Areas ranging between 1500-2500m have a different malaria transmission pattern with the temperature being the most important determinant factor
- High land areas above 2500m from sea level are free from local malaria transmission
- The major malaria transmission season usually occurs from September to December following the end of the rains and a minor transmission season occurs in April and May
- The patterns are classified as **stable and unstable malaria**

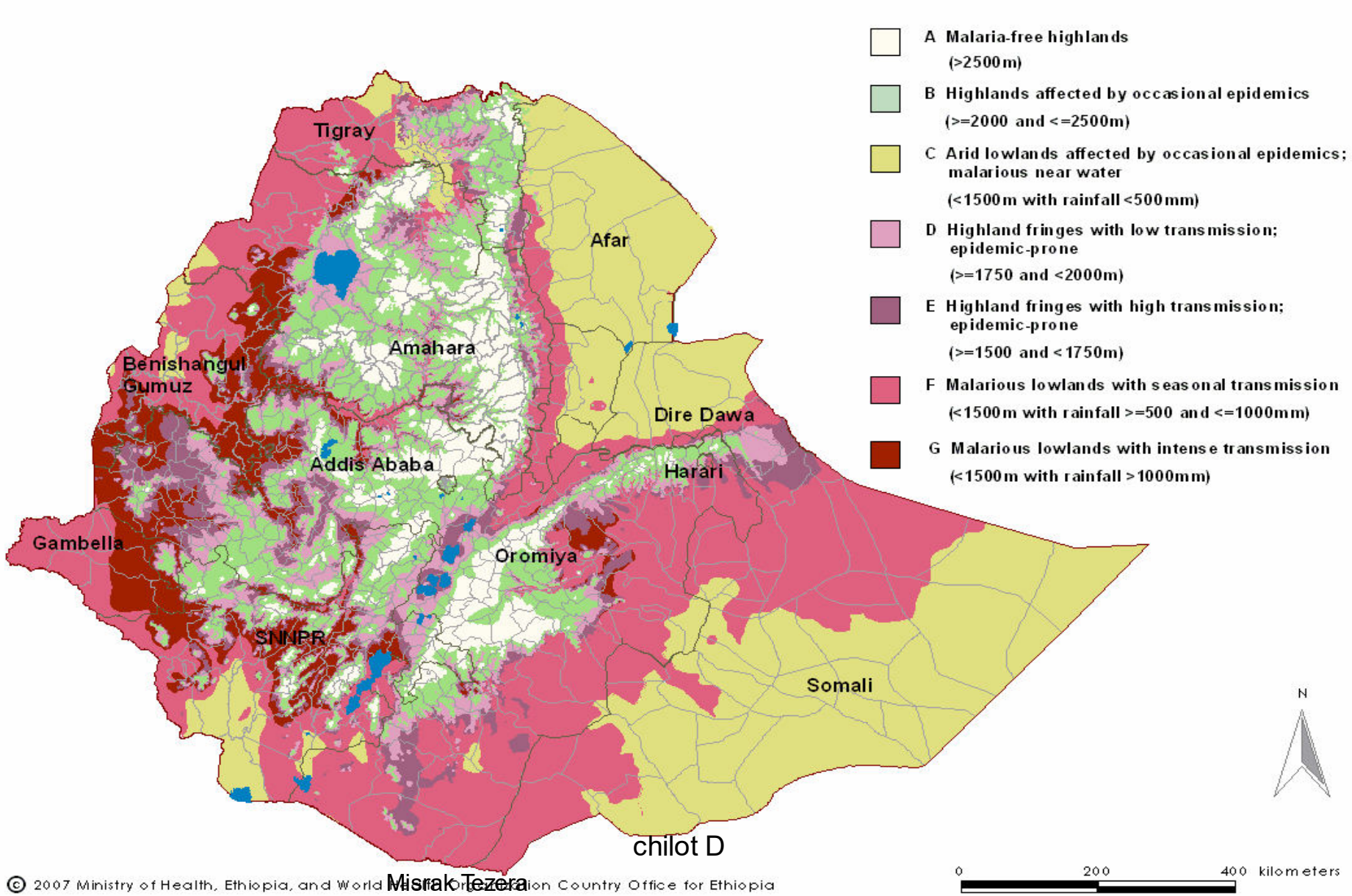
Unstable malaria

- Occurs in high lands or high land-fringe areas between 2000-2500 meters
- Uneven /in epidemics/, less intense transmission /liable to flare up in to dramatic epidemics/
- Short living and less frequently biting vectors present
- Due to the low level of transmission, the immunity status of the community is very low and all age groups are affected equally.
- Eradication is much easier than stable malaria

Stable malaria

- Occur in low land areas located below 1500m of altitude
- Transmission occurs throughout the year and the intensity is fairly uniform
- Long living and frequently biting vectors are present
- There is a high resisistance in the community due to the prevailing intense transmission
- The main impact of the disease is in young children and pregnant women
- Eradicating such type of malaria is usually very difficult

Malaria situation in Ethiopia



Factors affecting the epidemiology of malaria

1. Environmental factors

- Temperature, humidity and rainfall
  - ➢P. falciparum requires around16-20^{0}c / p. vivax even lower tempreture/ with humidity of 65%, which shortens the sexual cycle in mosquito and longevity of mosquito increases
- **Note:** heavy rain damages the larva production

2. Factors related to vectors

- Life expectancy of the mosquitoes
  - When there is favorable temperature and humidity the longevity of the mosquito increases and the life cycle of sexual reproduction period shortens
- Anthropophyllia /desire to bite humans/
- Presence of large numbers of mosquitoes
- The resting habit of mosquito
  - After entering houses at dusk, mosquitoes feed on human blood then they rest on the walls for about 7-8 hrs

3. Parasitic factors

- Longevity of the parasite in human host /P. falciparum for one year and P. vivax for 3-5 years/
- Multiplication pattern of the parasite in humans differs from species to species /P. falciparum multiplies very fast/

4. Host factors

- Resistance to malaria such as frequency and occurrence of protective genetic changes
  - **Sickle cell traits are resistant to P.falciparum**.
    - P.falciparum does not multiply properly in sickle red cells containing the abnormal haemoglobin. /common in Africa/
  - **Duffy blood group deficiency.**
    - Duffy antigen negative red blood cells lack receptor for P. vivax.
    - It is absent in the native population of west Africa and hence no vivax in this area
- Acquired immunity in endemic areas
- Susceptible particularly of migrant worker, children and pregnant women

5. Other factors

- Availability and accessibility of health services
- Preventive behaviors of communities

Malaria…

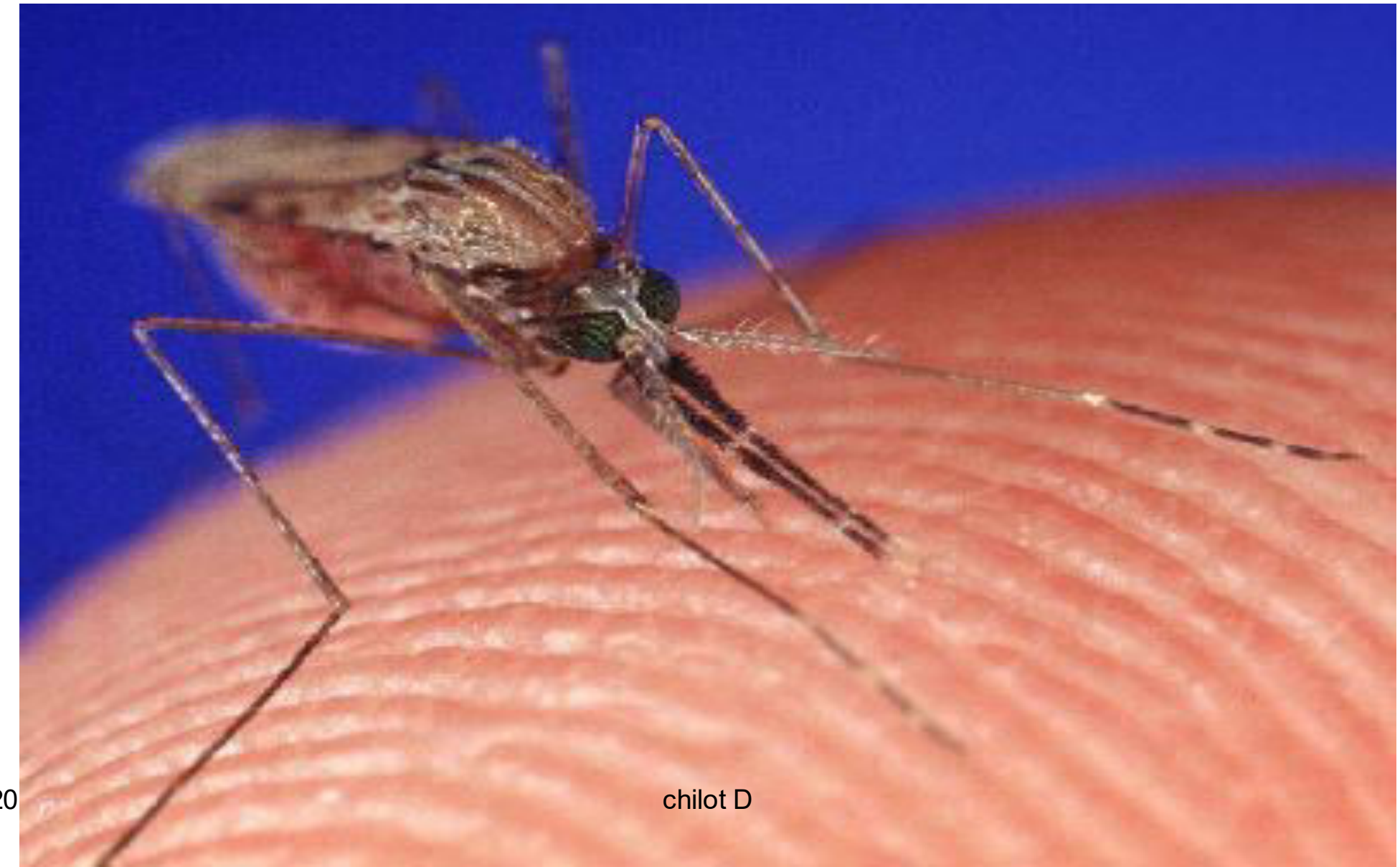
Reservoir

- Humans

Mode of transmission

  - By the bite of an **infective** female anopheles mosquito, which sucks blood for egg maturation
  - Blood transfusion
  - Hypodermic needles
  - Organ transplantation
  - Mother to child transmission
- *An. gambae* (***An. gambae arabiansis***) is common vector in Ethiopia

Mosquito- fed



Malaria…

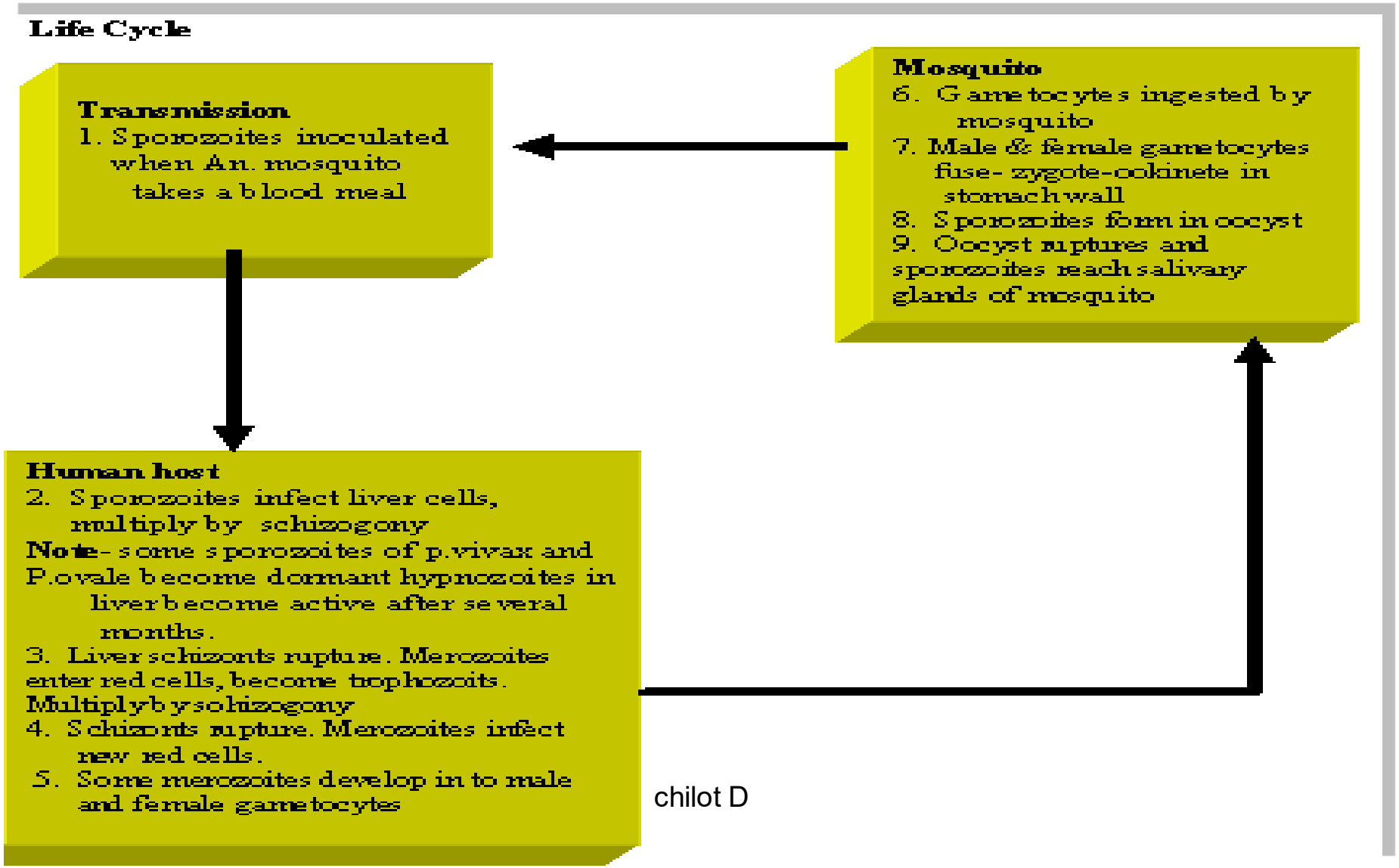
Incubation period - varies with species

- P. falciparum-7-14 days
- P. vivax – 8-14 days
- P. ovale – 8-14 days
- P. Malariae - 7-30 days

Period of communicability

- Mosquitoes are infective as long as infective gametocytes are present in the blood of the pts.
- Once infected, mosquito remains infective for life

Life cycle


Life Cycle
Transmission
1. Sporozoites inoculated when An. mosquito takes a blood meal
Mosquito
6. Gametocytes ingested by mosquito
7. Male & female gametocytes fuse- zygote-ookinete in stomach wall
8. Sporozoites form in oocyst
9. Oocyst ruptures and sporozoites reach salivary glands of mosquito
Human host
2. Sporozoites infect liver cells, multiply by schizogony
Note- some sporozoites of p.vivax and P.ovale become dormant hypnozoites in liver become active after several months.
3. Liver schizonts rupture. Merozoites enter red cells, become trophozoits. Multiply by schizogony
4. Schizonts rupture. Merozoites infect new red cells.
5. Some merozoites develop in to male and female gametocytes

Transmission

Malaria parasites enter the human body via the bite of a malaria-carrying mosquito of the genus *Anopheles*. The parasites invade the liver via the bloodstream and

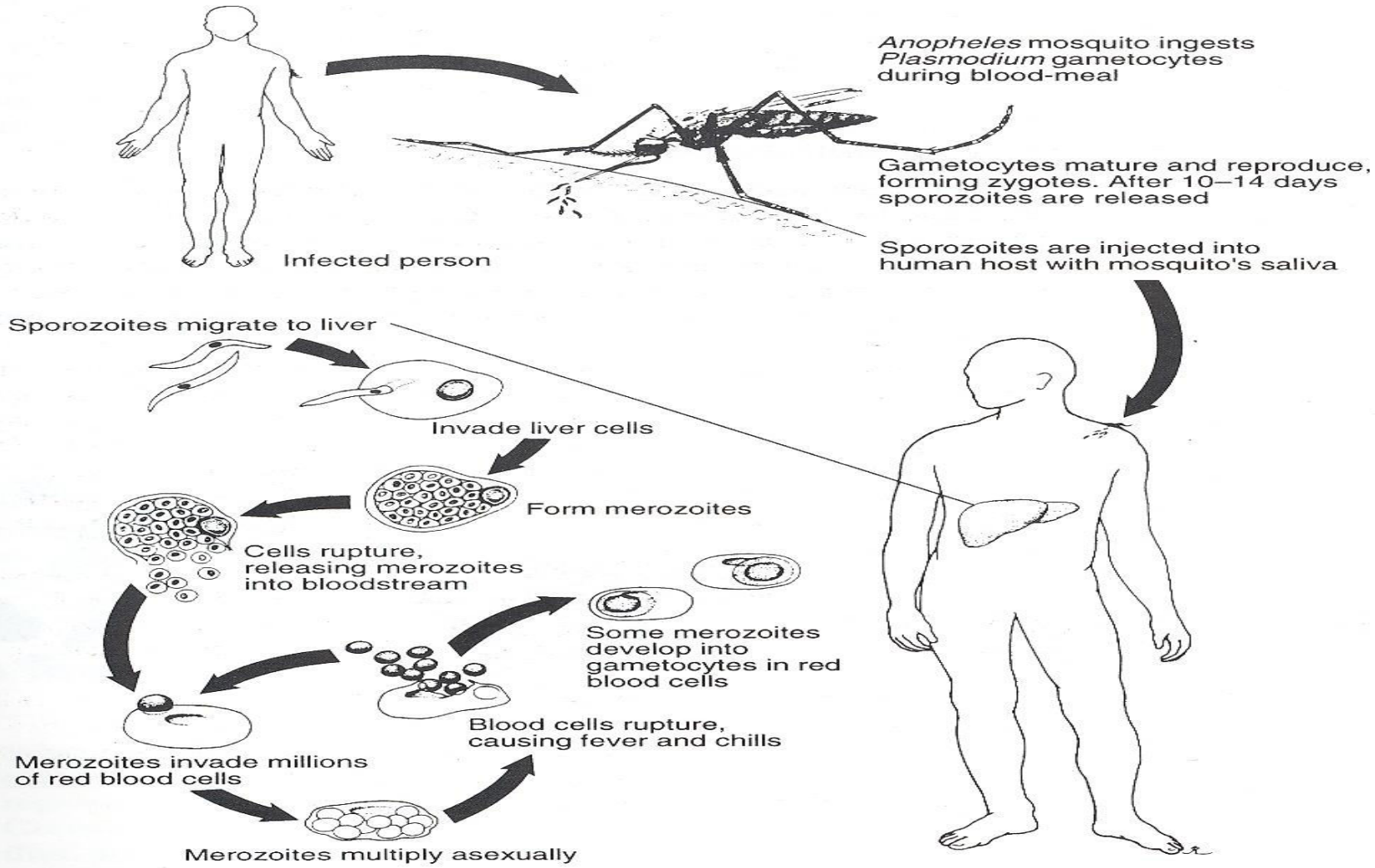
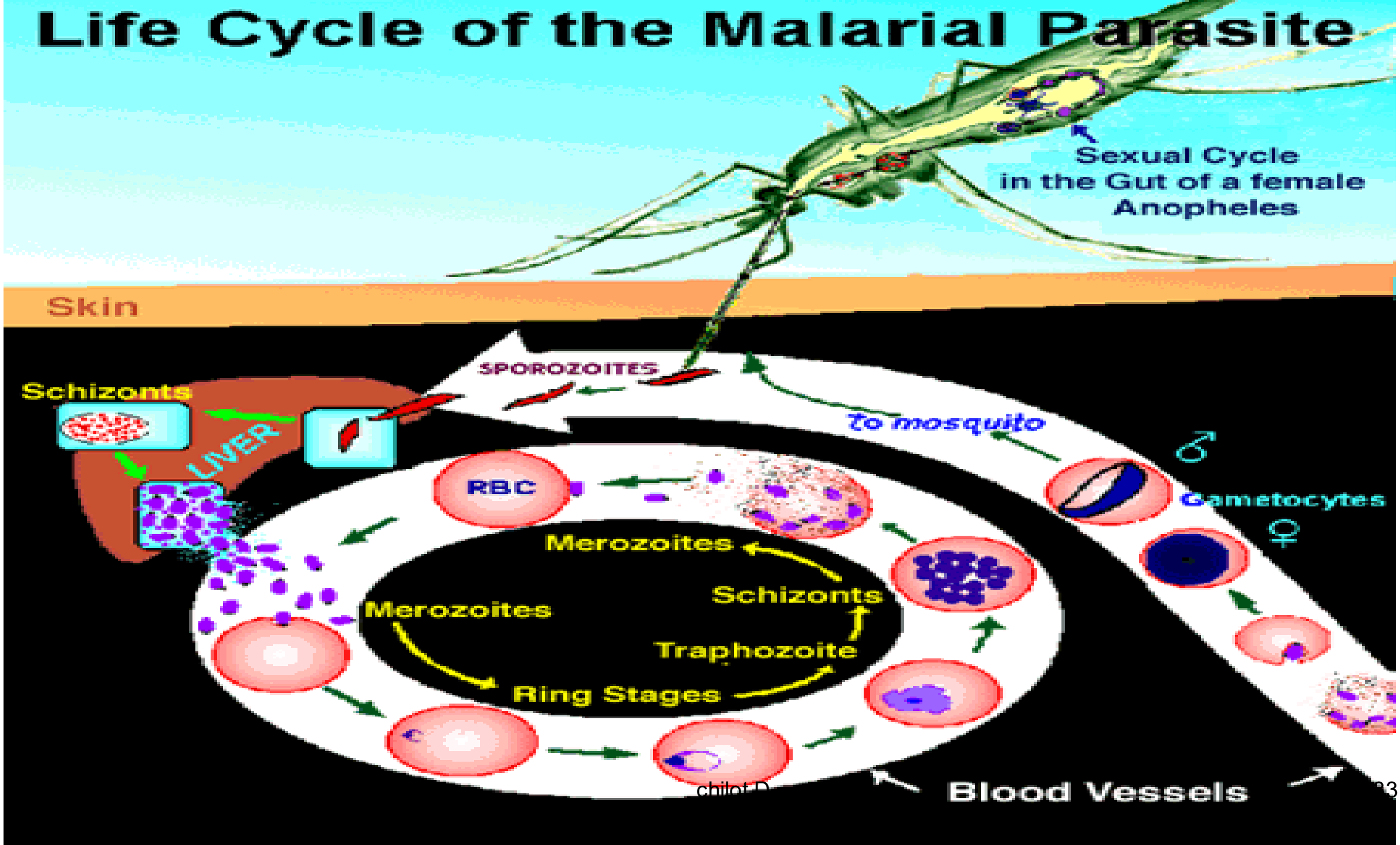
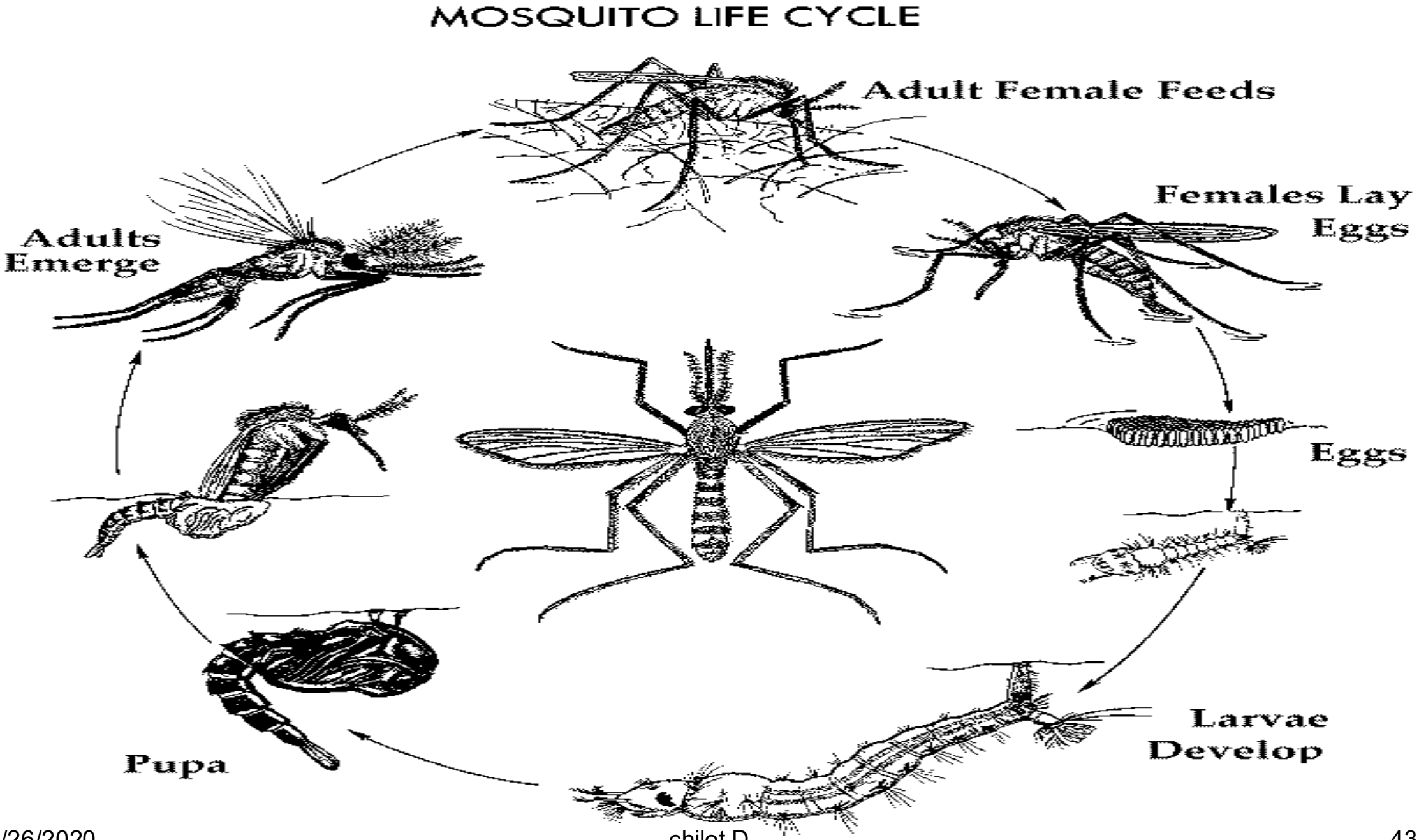


Fig. 1.17
Life cycle of the malaria parasite (by Taina Litwak for the United States Agency for International Development's VBC Project).


Life Cycle of the Malarial Parasite
Sexual Cycle
in the Gut of a female
Anopheles
Skin
SPOROZOITES
Schizonts
LIVER
To mosquito
RBC
Merozoites
Schizonts
Merozoites
Traphozoite
Ring Stages
Gametocytes
Blood Vessels


MOSQUITO LIFE CYCLE
Adult Female Feeds
Females Lay Eggs
Eggs
Larvae Develop
Pupa
Adults Emerge

Clinical manifestations

- **1. Uncomplicated malaria**

The first symptoms and signs of malaria which are non specific to the disease include:

- High grade fever and headache
- Malaise
- Chills, shivering and sweating
- Muscle ache and joint pain
- Pallor and enlargement of spleen and liver
- Sometimes nausea, vomiting, loss of appetite, abdominal pain, diarrhea and thirst

2. Sever malaria /complicated malaria/

- Sever malaria is **falciparum malaria** that is sufficiently serious to be an immediate threat to life.
- A patient is regarded to have **sever malaria** if there are asexual forms of P. falciparum on a blood film and he has one or more signs and symptoms of the followings sever illnesses:
    - Cerebral malaria: defined as unarousable coma
    - Sever anemia – Hematocrite < 15%
    - Hypoglycemia <60mg%
    - Fluid, electrolyte and acid -base disturbance
    - Pulmonary edema or difficulty of breathing

Sever malaria /complicated malaria/…

- Acute renal failure or oliguria
- Circulatory collapse or shock /algid malaria/ - because of gm -Ve sepsis
- Haemoglobinurea –black water fever /hematuria/ -because of RBC hemolysis
- Change of behavior, confusion, drowsiness
- Jaundice
- Spontaneous bleeding tendency /disseminated intravascular coagulation/
- Prostration/ unable to eat, sit or stand/
- Hyper parasitemia / > 5% RBCS affected/
- Hyperpyrexia / ≥ to 39^0c

Possible mechanisms of sever disease

Anemia

- Destruction of RBC
- Bone marrow suppression
- Abnormal bleeding
- Renal failure
- Sequestration in spleen (extra vascular hemolysis)

Possible mechanisms of sever disease…

Pulmonary edema

- Iatrogenic- excessive fluid replacement by IV infusion especially if there is ARF
- Respiratory distress syndrome- direct effect of sequestrated parasites

Renal failure

- Due to acute tubular necrosis
- Diagnosis is urine out put <17 ml/hr for adult and <0.3 ml/kg/hr for children
- It is fully reversible

Possible mechanisms of sever disease…

Hypoglycemia

- Impaired gluconeogenesis in the liver
- Maturing parasites consume large quantity of glucose
- Quinine induced hypoglycemia – because of hyper insulinism
- Anorexia- period of fasting

Chronic complications of malaria

Hyper active malarial spleenomegally

- Also called TSS (tropical spleenomegally syndrome)
- Seen in malaria endemic areas
- Results from abnormal immunologic response to repeated infection by malaria parasites characterized by:
  - Enormous spleenomegally />10cm/
  - High titers of circulating antimalaria antibody
  - Absence of malaria parasites in peripheral blood smear

Quartan malaria nephropathy

- Chronic or repeated infections with P. malariae resulting in:
  - Immune complex injury to the renal glomeruli
  - Nephrotic syndrome (NS)

Relapses and recrudescence

- Malaria due to p. vivax may relapse months or years after the original infection because the exo-erythrocytic cycle /hypnozoites/ has persisted.
- This doesn't happen with p. falciparum
- Further febrile episode/recrudescence/ with high parasitemia may develop upto a year after p. falciparum infection.
- This is due to exacerbation of a low-grade persistence of the erythrocytic cycle

Diagnosis

- Clinical manifestation and epidemiological ground
- Blood film for hemoparasites
- Rapid diagnostic tests/Dip stick/
- Blood glucose
- Hematocrit
- White blood cell count to role out other infections
- Blood culture to role out sepsis
- Chest X-ray to role out pneumonia
- Lumbar puncture to exclude meningitis

Quantity estimate of the parasite from blood film examination

- + = 1-10 parasites per 100 thick films fields
- + + = 11-100 parasites per 100 thick films fields
- + + + = 1-10 parasites per each thick film field
- + + + + = More than 10 parasites per each thick film field

Treatment

1. Management of uncomplicated malaria

a)1st line drug

- Artemether-lumefantrine /coartum/ 4tabs. 2x/d for 03 days (for P. falciparum)
- Quinine – 3×/d for 7days for infant <5kg and pregnant women (for P. falcipurm).
- Chloroquine for P. vivax, P. malariae or P. ovale
- Primaquine- 1 tab. daily for 14 days for hypnozoites in malaria free areas

Note: **Coartum** is contraindicated for infant <5kg, pregnant woman and sever malaria

b) 2nd line treatment

- Quinine if P. falcipurum positive patient returns back

Treatment …

2. Management of sever /complicated/malaria

- ✓ For all pts with sever malaria, iv quinine should be given at least for the first 48 hrs
- ✓ Quinine – Initial loading dose 20mg/kg by IV infusion over 4 hrs for 8-12hrs
- ✓ Maintenance dose 10 mg/kg in DW 4 hrly
- ✓ If patient requires more than 48 hrs of parentral therapy, reduce the quinine maintenance dose by **1/3** (i.e. 5-7 mg/kg every 8 hrs)
- ✓ As soon as the patient starts to take orally, change quinine to po to complete 7 days treatment

Treatment…

- ✓ Correct hypoglycemia – 50% Dextrose, 1ml/kg for children
- ✓ Reduce body temperature if > 39^{o}c
- ✓ Control convulsion
  - ➢ Phenobarbital sodium single dose of 200mg for adults by im injection (10-15mg/kg for children)
- ✓ Consider the need for blood transfusion- if HCT <15%
- ✓ Catheterize if either ARF or pulmonary edema is suspected to monitor fluid balance
- ✓ Maintain clear air way
- ✓ NG tube feeding if unconscious

Note: if the diagnosis of malaria is made on clinical bases, combined treatment of **coartum and chloroquine** is recommended

Prevention & control of malaria

- **Chemotherapy of cases**
- **Body protection against mosquito bite:**
  - Wearing clothes
  - Fixing screen on windows and doors
  - Use of impregnated mosquito bed net /ITN/
  - Application of mosquito repellents
- C**homoprophylaxis** especially for <5 yrs, pregnant mothers and travelers to endemic area
  - Mefloquine 5mg/kg weekly and chloroquine can be used

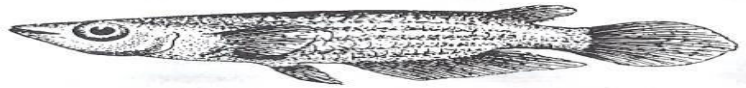
Prevention & control of malaria…

- **Vector control**
  - ➢Eliminating mosquito breeding sites
    - ✓Cutting vegetation around residence
    - ✓Clearing the environment
    - ✓Disposing garbage's and wastes in prepared pits
    - ✓Draining & filling stagnated water frequently
    - ✓Clearing marshy areas
  - ➢Use of insecticides such as deltamethrine spray
  - ➢Biological control such as use of larvivorous fish
- **Health education**

Larvivorous fish

The panchax, Aplocheilus panchax

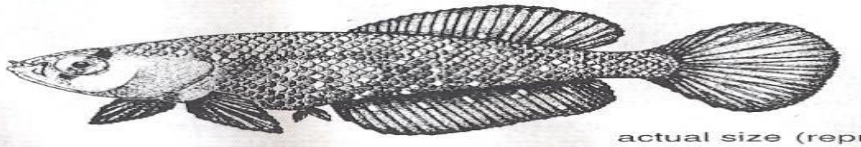
This egg-laying tooth carp is found in the Indian subcontinent, Indonesia, Malaysia and Sri Lanka, where it commonly occurs in paddy fields and ditches and is important in the control of mosquitos (*154*). The fish can withstand pollution and water temperatures between 20 °C and 45 °C.



actual size (reproduced from *154*)

The Argentine pearlfish, Cynolebias bellotii

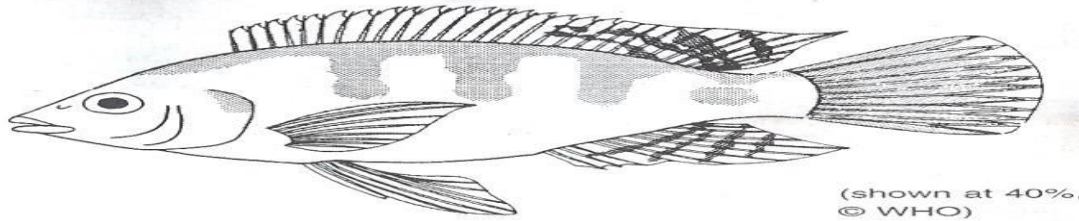
This is one of the annual fishes that occur in South America and Africa, known as instant fish. They cannot reproduce in permanent water bodies and occur only in habitats where the water disappears every 2–3 months or at least once a year. The eggs, which survive the dry period buried in the soil, may be concentrated, transported and dispersed in slightly damp material. They hatch within a few hours after flooding. Although not extensively evaluated, these fish may be useful in borrow-pits and temporary dry pools as well as in rice fields and irrigated pastures where other fish cannot survive (*153*).



actual size (reproduced from *153*)

The Mozambique mouthbrooder, Oreochromis (Tilapia) mossambicus

This cichlid fish occurs in East Africa. It has been reared successfully in irrigated rice fields where it was used both to control mosquitos and as a source of food. With an optimal temperature of 22 °C it reproduces very rapidly. The species can live and reproduce in fresh and brackish water (*146*).



(shown at 40%, actual size is 20 cm; © WHO)

FILARIASIS

- It is a nematode disease caused by the reaction of the body to the worms
- 8 species of filariae are natural parasites of human
- 5 species are important
  - Wuchereria bancrofti - lymphatic or bancroftian filariasis
  - Brugia malayi - lymphatic or Malayan or brugian filariasis
  - Brugia timori –lymphatic or timoran filariasis
  - Loa loa – loiasis, eye worm and cause of calabar swelling
  - Onchocerca volvulus- onchocerciasis, river blindness

LYMPHATIC FILARIASIS

- It is a disease caused by the reaction of the body to the presence of worms in the lymphatic system
- The size of the adult worm is 4-8cm long

Infectious agents

- W. bancrofti
- Brugia malayi
- Brugia timori

Occurrence and importance

- 120 million cases in the world , 107 million due to W. bancrofti
- Half of the world's filariasis occurs in India (vector culex)
- Widely prevalent in tropical & subtropical areas of Africa, central and south America
- Found in Gambella region of Ethiopia

Lymphatic filariasis…

Reservoir

- Humans are definite hosts

Mode of transmission

- By the bite of mosquito harboring infective larvae

Vectors of Lymphatic filariasis include:

- Culex, Anopheles and Aedes species for w. bancrofti
- Mansonia species for B. malayi
- Anopheles for B. timori

Lymphatic filariasis …

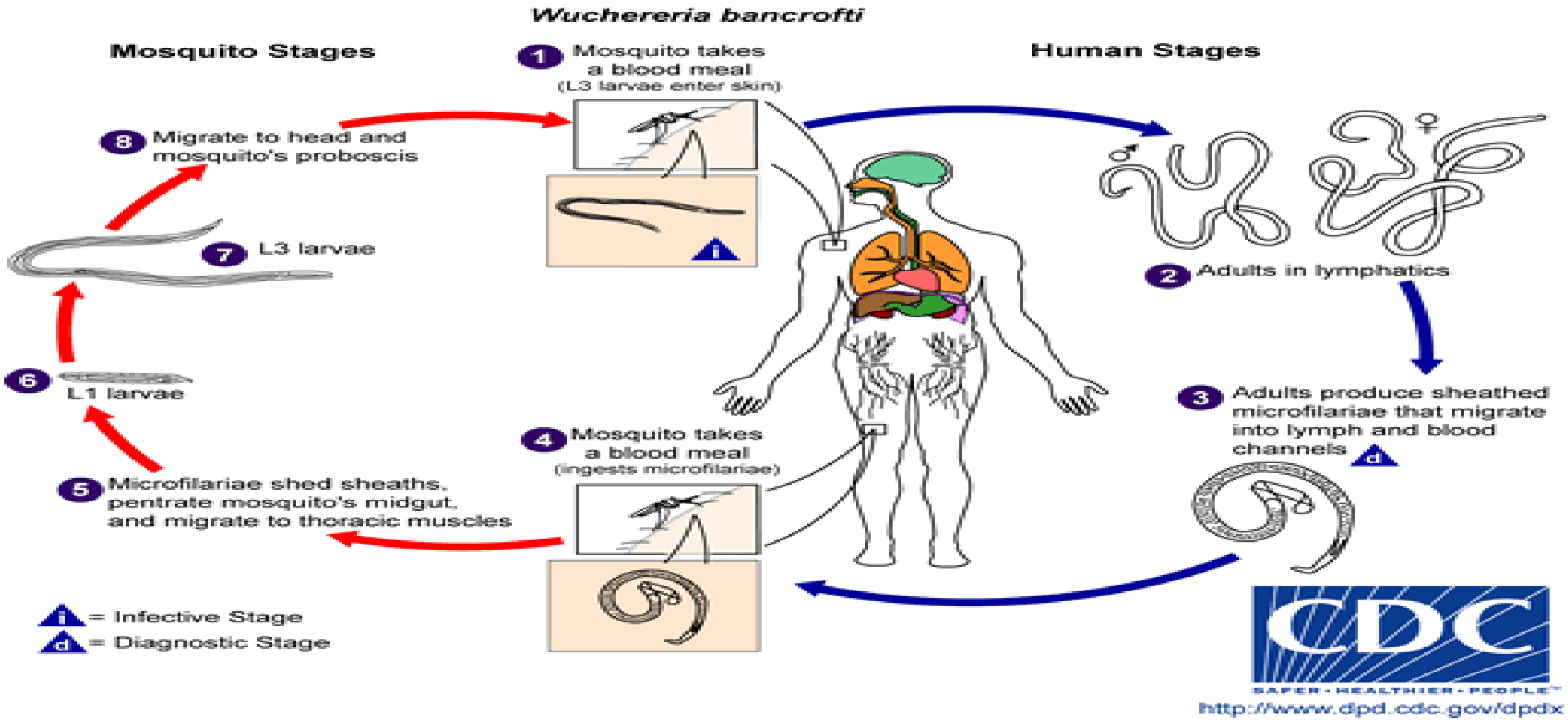
Incubation period

- One month, while allergic inflammatory manifestations may appear.

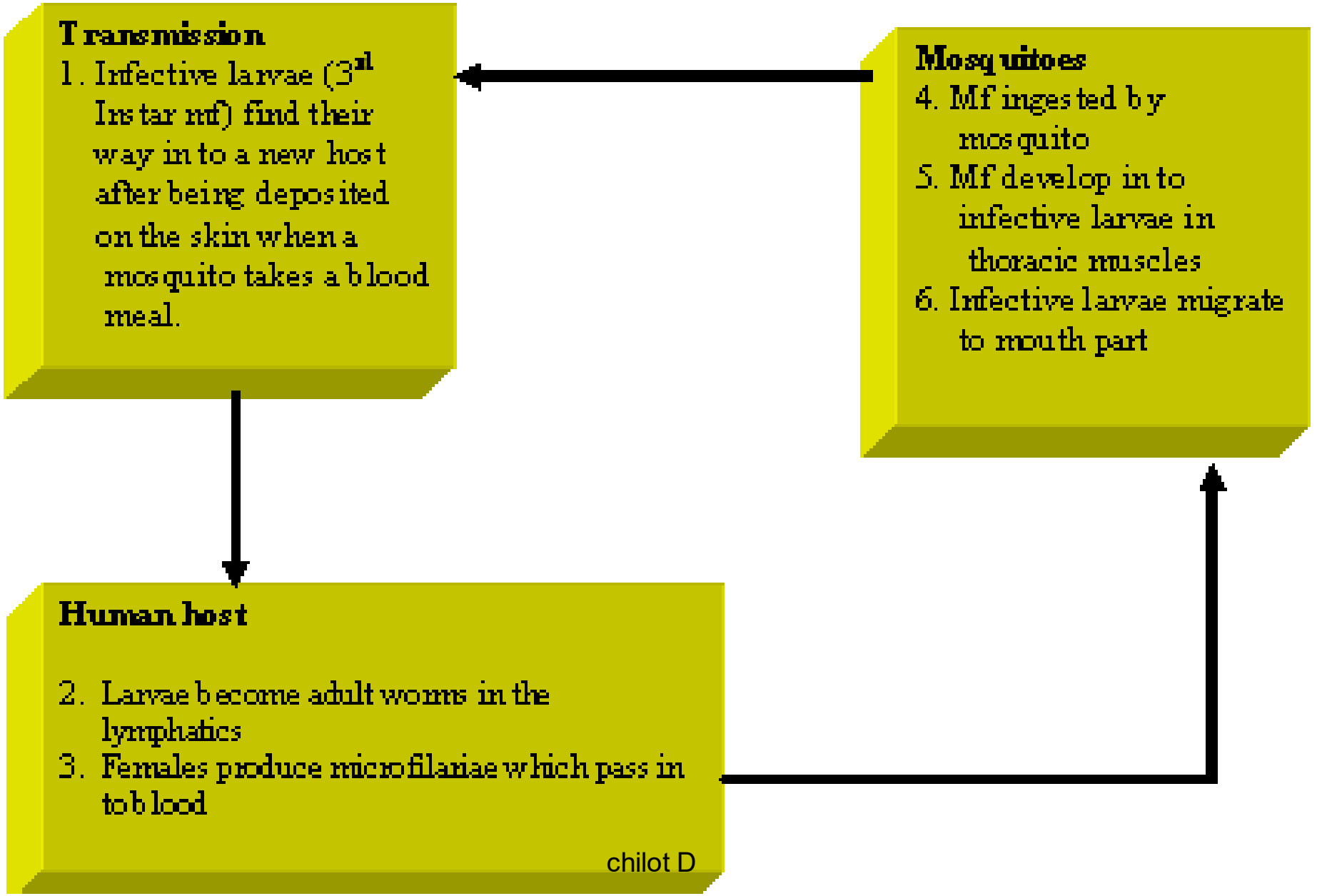
Period of communicability

- Humans may infect mosquitoes when the microfilaria are present in the peripheral blood(at night when vectors come to bite)
- Microfilaremia may persist for 5-10 years or longer
- The mosquito becomes infective about 12-14 days after an infective blood meal.

W_bancrofti_Life Cycle



Life cycle


Transmission
1. Infective larvae (3rd Instar mf) find their way in to a new host after being deposited on the skin when a mosquito takes a blood meal.
Mosquitoes
4. Mf ingested by mosquito
5. Mf develop in to infective larvae in thoracic muscles
6. Infective larvae migrate to mouth part
Human host
2. Larvae become adult worms in the lymphatics
3. Females produce microfilariae which pass in to blood

Clinical manifestations

- The presence of adult worms in the lymphatic vessels gives rise to foreign body reaction which leads ultimately to the **pathological effects**

- Death of adult worm →↑ protein release →↑ reaction

Clinical manifestations…

- It has three phases:

1. Acute phase

- This phase is mainly due to hypersensitivity reaction
- Starts with in a few months after infection
- Lymphadenopathy
- Fever
- Eosinophilia
- Mf are not demonstrable in the peripheral blood because worms are not yet mature

Clinical manifestations …

2. Sub acute phase

- Occurs after about one year
- Mf present in the peripheral blood
- Reactions to the adult worms cause attacks of fever with lymph adenitis, funiculitis or epididymitis
- Recurrent attacks lead to **hydrocele**

Clinical manifestations…

3. Chronic phase

- After many years of repeated attacks
- Lymph glands & lymph vessels become obstructed → lymph edema

Lymph edema

- ➢ Legs or scrotum (elephantiasis)- most common
- ➢Vulva, breasts or arms

- Mf are not seen in the blood, because of adult worm death

Fig. 1.21
Elephantiasis (permanent swelling) of the leg due to lymphatic filariasis.

Note

- *Elephantiasis of the lower legs is not seen in Ethiopia.*
- *But elephantiasis of the foot (big foot disease) as a result of accumulation of silica & other minerals in the leg (lymphatics) causes* ***silicosis***
- *Mostly occurs in* ***bare-footed*** *individual.*

Diagnosis

- Clinical & epidemiological ground
- Best established by identifying mf in the blood film
- Blood sample is taken during night (nocturnal) when mf appears /in most parts of the world/
- Single dose of **DEC** causes the sequestrated mf to emerge to blood, 45-60 minutes later (**mazoti test**)

Treatment

- DEC- results in rapid disappearance of most mf from blood
- Ivermctin & albendazole in combination with each other or with DEC and given annually, virtually eliminate mf from the blood
- Refer the patient for surgical treatment of **hydrocele**

Prevention & control

- Mass and selective treatment
- Organized free supply of ivermectin & albendazole is the basis of the **WHO** plan to eliminate filariasis
- Personal protection against mosquito bite
- Reducing the vector population
  - ➢E.g. polystyrene beads to a pit latrine for culex mosq. (inhabit dirty water)

ONCHOCERCIASIS (RIVER BLINDNESS)

- It is a chronic disease caused by a filarial nematode worm
- The size of adult female and male worm is about 40cm and 4cm long respectively

Infectious agent

- Onchocerca volvulus

Occurrence & importance

- Major cause of blindness in tropical Africa (0.27 million)
- 17.8 million cases in Africa, 0.14 in central & S. America

Reservoir: Humans are definite hosts

Mode of transmission

- Transmitted by small black fly named **simulium** (female)
- The fly lays eggs in fast running rivers where there is enough oxygen to make eggs develop in to the larvae
- The fly attacks out doors during the day around sun rise or sun set or on cloudy days or in the shade
- When the fly takes blood from an infected person the mf develop in to infectious larvae.

Black flv

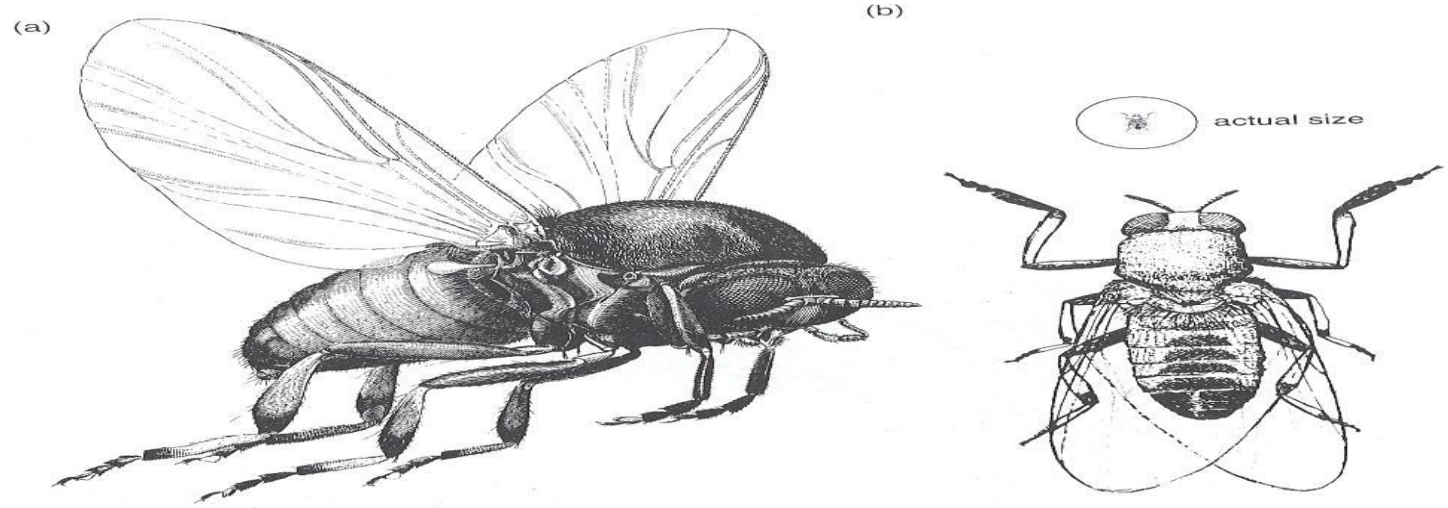
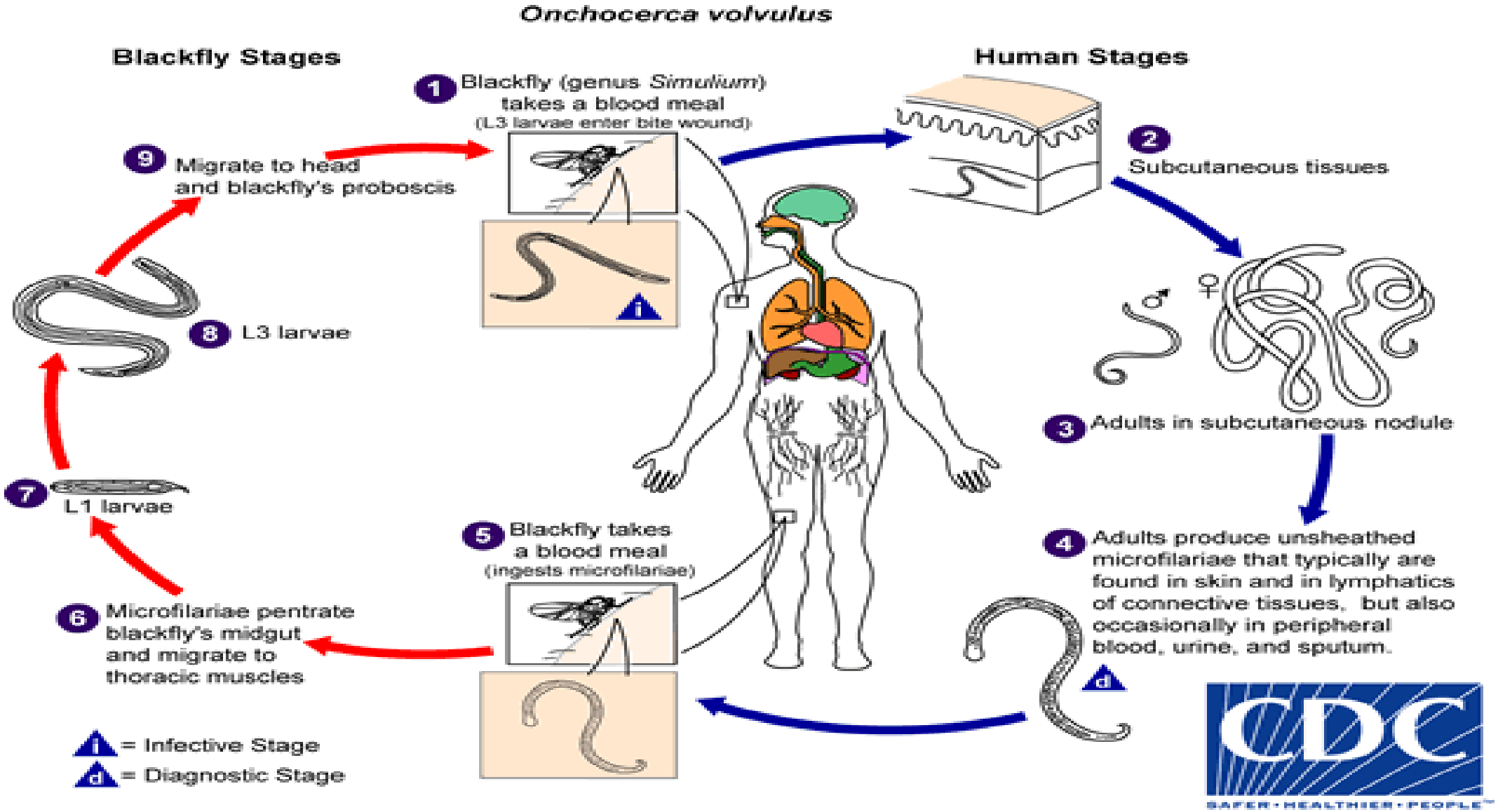


Fig. 1.9
Blackfly: (a) in flight (© WHO); (b) at rest (by courtesy of the Natural History Museum, London).

O. VoLVULUS LIFE CYCLE



Pathogenesis

- The infective larvae enter human host through bite wound and develop in to adult worms in subcutaneous tissue
- The mature males and females then collect in balls, bound together by fibrous tissue which forms the typical nodules
- Nodules are best seen in places where the bone is near to the skin, e.g. elbow, shoulder, scapula, skull, ribs, iliac crests.
- After one year the adult worms start giving birth to mf /micro filarae/.
- The mf migrates in the epidermis, subcutaneous tissues and in the eye.

Clinical pictures

Presents in **3** different clinical pictures

1. Nodules

- Caused by adult worms: non tender, rubbery, firm nodules, 3mm-3cm in size

2. Dermatitis

- Caused by reaction to the presence of mf in the epidermis
- The skin is itchy, which after words becomes loose, atrophic and depigmented (leopard skin)

Clinical pictures …

3. River blindness

- The mf invade the cornea, anterior chamber of the eye, causing photophobia, chronic conjunctivitis followed by secondary glaucoma and cataract which can make cornea opaque & lead to blindness
- Invasion of the retina by mf can also lead to blindness

Diagnosis and treatment

Diagnosis

- The mf are not found in the blood
- Diagnosis is made by examination of **skin snip**, to see moving mf under microscope
- DEC causes sever inflammatory reaction (don't treat with this drug)

Treatment

- Ivermectin- given once a year to kill mf
- Surgical removal of the nodules

Prevention & control

- Mass treatment with ivermectin- annually
- Personal protection against fly bite
- Vector control
  - ➢ adding insecticides (larvicide) continuously to the water of the rivers known to be breeding places of simulium fly.

Aerial application of insecticide to simulium breeding sites in a river

Fig. 1.30
Aerial application of insecticide to *Simulium* breeding sites in a river.

LOIASIS /AFRICAN EYE WORM/

- It is a tissue nematod filarial disease
- The adult worm is 2.5 - 5 cm long

Infectious agent

- Loa loa.

Mode of transmission & life cycle

- It is transmitted by **deerflies** of the genus **chrysops**
- Its life cycle resembles that of *onchocerca volvulus*

Occurrence and pathogenesis

Occurrence

- It occurs only in woods and forests in west and central Africa, from Benin to Uganda and Southern Sudan where **10 million** people are affected

Pathogenesis

- The adult worm lives in the subcutaneous tissues under the skin
- Migration of the worms under the skin may cause a pricking & itching sensation.
- Infection sometimes may cause swelling of the various parts of the body **called calabar swelling /** fugitive swelling – disappear in a few days, only to reappear elsewhere/

Diagnosis and treatment

Diagnosis

- Adult worm is seen especially about the orbit and even under the conjunctiva
- Microfilariae may be seen in peripheral blood collected during the day

Treatment

- Treatment is possible with **ivermectin**
- DEC is active against the worm, but has to be used with caution as sever adverse reactions may develop following the sudden death of large numbers of micrifilariae
- Surgical removal of the adult worms when they come to accessible sites

GUINEA WORM DISEASE (DRACUNCULIASIS)

- It is an infection of the subcutaneous and deeper tissues by large nematode
- The adult female worm is **60-120cm** long
- In Greek, Draco – dragon or serpent / seems long snake/
- Medinens – prevalent in Medina

Infectious agent

- Dracunculus medinensis

Occurrence and importance

- The worm was present in tropical Africa, Middle east and Pakistan
- About 50 million people were estimated to be infected with the worm

Reservoir

- Humans

Mode of transmission and life cycle

Site suitable for transmission are accumulation of water where:

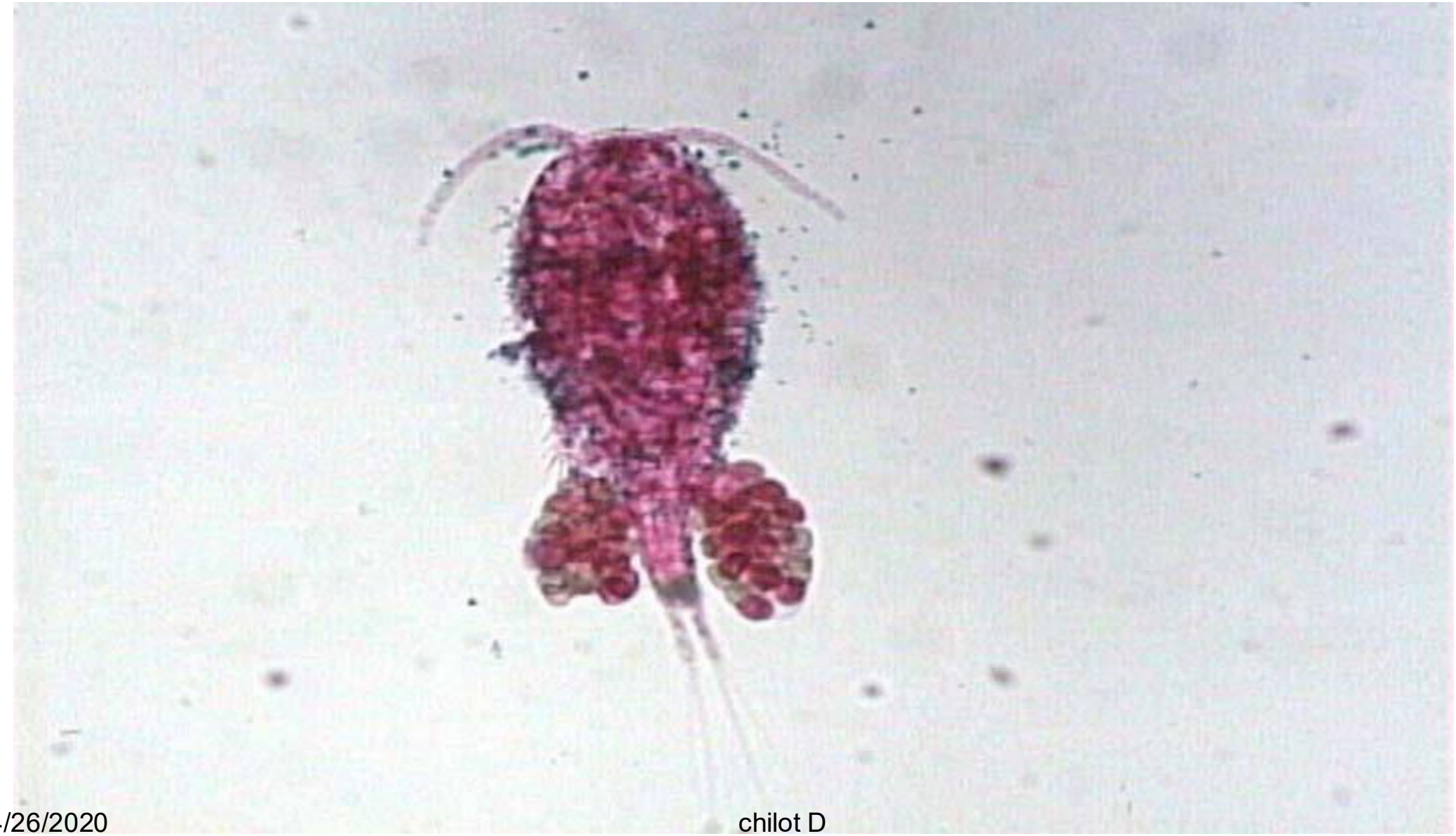
- Infected people enter the water
- The water is stagnant and Cyclops species, which can transmit the parasite, are present
- The water is used regularly as drinking water

Mode of transmission and life cycle…

- Humans are the definite host
- Larvae discharged by the female worm in to stagnant water are ingested by minute crustacean copepods (**Cyclops species**)
- In about 2 weeks, the larvae develop in to the infective stage
- People swallow the infected copepods in drinking water from infested step wells and ponds
- The larvae are liberated in the stomach, cross the duodenal wall, migrate through the viscera and became adults
- The female after mating, grows and develops to full maturity, then migrates to the subcutaneous tissues (most frequently of the legs - 90%)

Incubation period: About 12 months.

Cyclops species



Life cycle of guinea worm

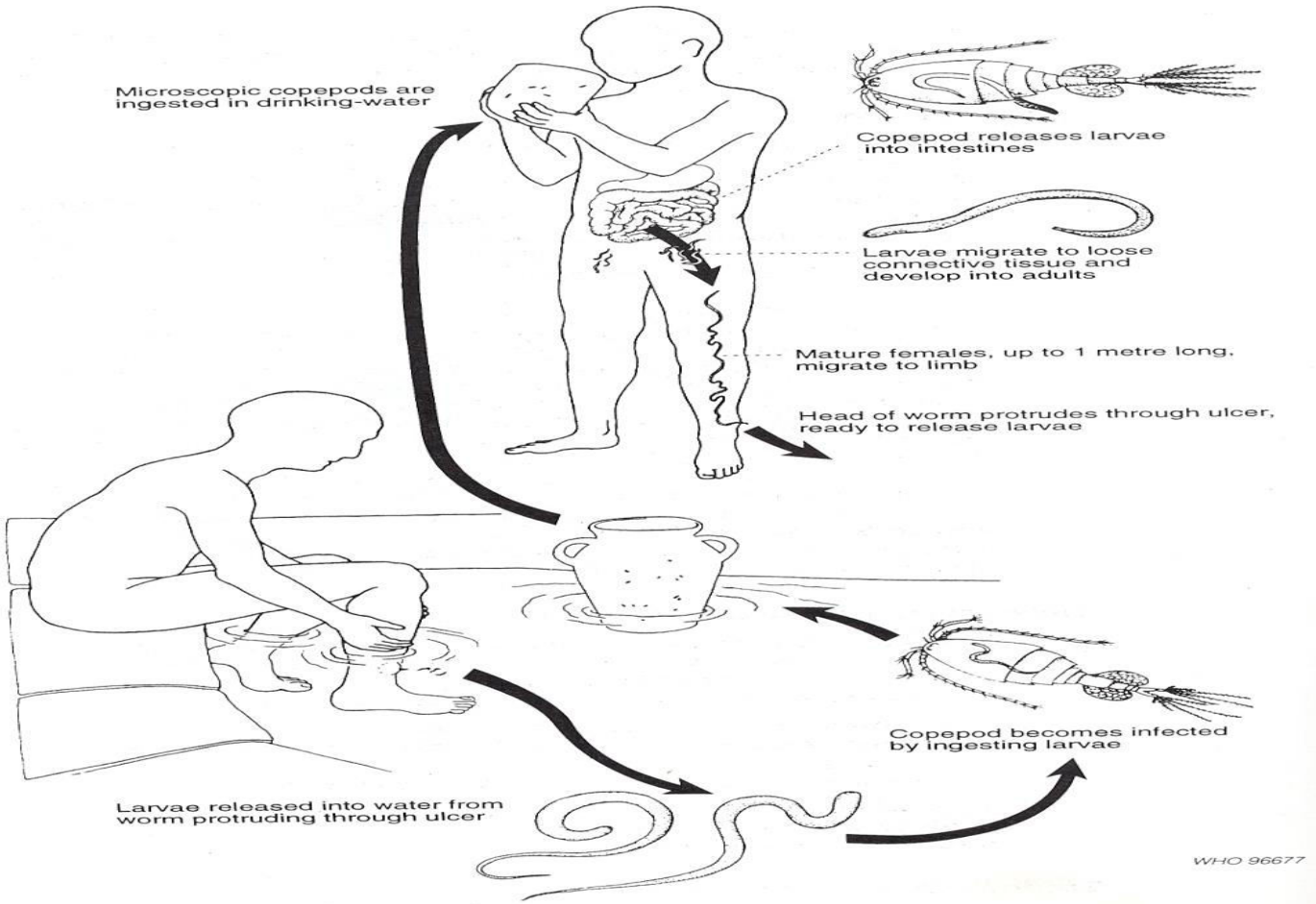


Fig. 7.3
Life cycle of guinea worm (by Taina Litwak for the United States Agency for International Development's VBC Project).

Period of communicability

- From rupture of vesicle until larvae have been completely evacuated from the uterus of the gravid worm, usually 2-3 wks.
- The worm subsequently dies & is eliminated from the body over a period of 3-8 weeks
- The released larvae can remain active in water for about 3 days & die unless they are swallowed by a cyclops

Clinical manifestations

- Few or no clinical manifestations are evident until just before the blister forms
- Fever and generalized allergic symptoms
- Local pain and swelling- associated with **emergence** of the worm
- Relief of symptoms-when blister ruptures and release larva-rich fluid
- The shallow ulcer surrounding the emerging adult worm heals over weeks to months
- Secondary infections such as **abscesses**, **tetanus**, **septicemia** and **arthritis**

Diagnosis

- Based on clinical & epidemiological grounds
- Tip of the worm projects from the base of the ulcer
- By bathing the ulcer with water, the worm can be induced to release the embryos, which can be examined under the microscope
- Calcified worms can be seen by radiography

Treatment

- Antihistamines and steroids
- Metronidazole, niridazole and thiabendazole are useful
- Gradual extraction of the worm by winding of a few centimeters on a stick each day remains the common and effective practice
- Worms may be excised surgically
- Treat secondary infection
- **Provide** TAT

Management of guinea worm disease

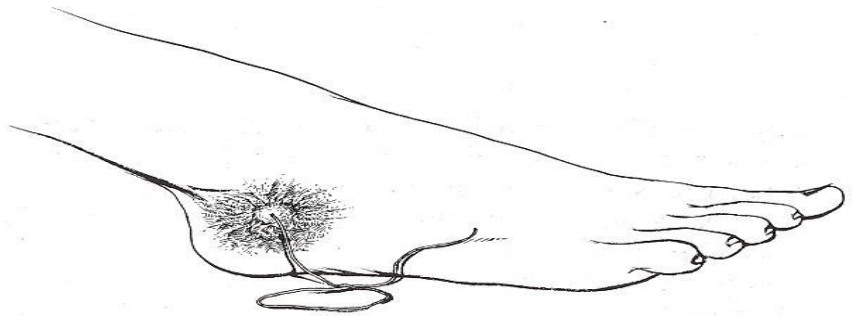


Fig. 7.9
Infection with guinea worm results in an itching and burning swelling on a leg or other part of the body, developing within a few days into a blister and an open sore. The worm emerges at the bottom of the ulcer.

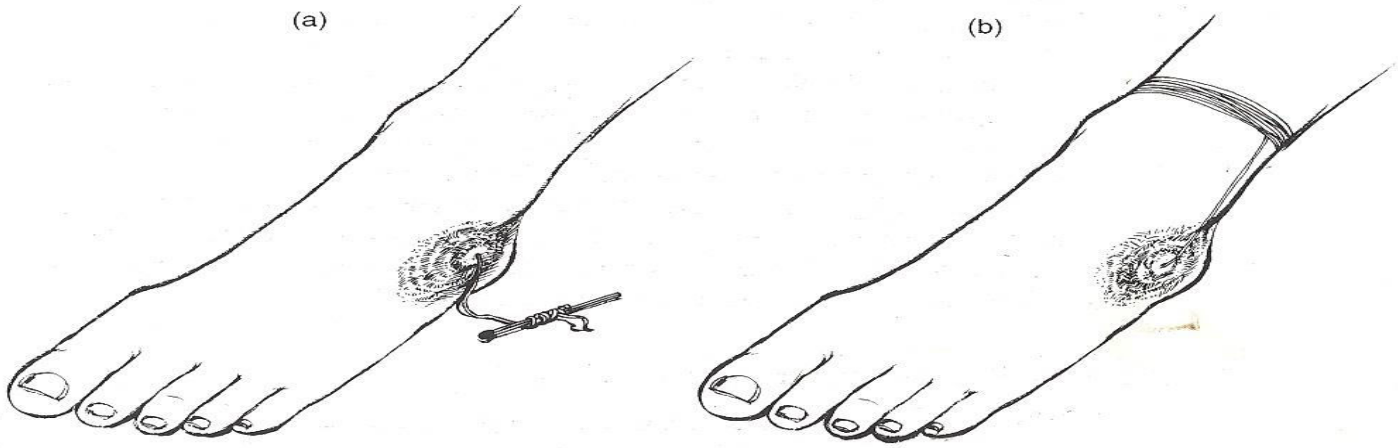


Fig. 7.10
Guinea worms can be extracted slowly by attaching to a string or rolling around a match or small stick to prevent them from withdrawing inwards.

Prevention and control

- Health education to convey three messages
  - ➢The guinea worm infection comes from their drinking water
  - ➢Villagers with blisters or ulcers should not enter to any source of drinking water
  - ➢Drinking water should be filtered through **fine mesh cloth** to remove copepods
- Provision of safe drinking water
- Boiling water is safe

Preventive technique of guinea worm

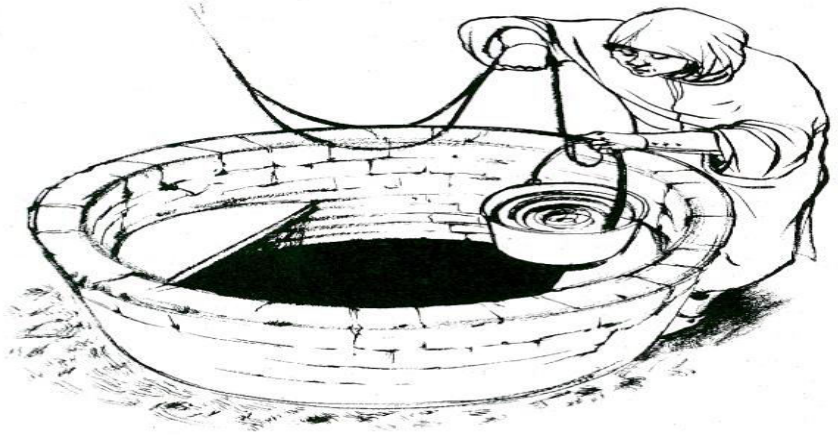


Fig. 7.11
Draw- or pit-well with rim: a safe source of drinking-water.

Fig. 7.12
Cyclops can be removed from drinking-water by pouring it through a gauze filter.

TYPHUS FEVER

- It is an acute rickettsial disease often with sudden onset
- Two types
  - Epidemic typhus fever or louse- borne- typhus fever
  - Endemic typhus or flea-borne typhus fever or murine typhus fever. Murine is to mean affecting mice or rats

Epidemic typhus

– An acute highly infectious disease caused by micro organism, Rickettsia prowazekii

Occurrence and importance

– In colder areas where people may live under unhygienic conditions and are louse- infected

– It occurs sporadically or in major epidemics, for example during **wars** or **famine**, when personal hygiene deteriorates and body lice flourish.

– May be fatal in **10-40% of untreated cases**

Reservoir and **mode of transmission**

Reservoir

- Humans
- Infected lice die & don’t serve as reservoir

Mode of transmission

- The body louse is infected by feeding on the blood of a patient with acute typhus fever.
- Infected lice excrete rickettsiae in their feces and usually defecate at the time of feeding
- People are infected by rubbing feces or crushed lice in to the bite or in to superficial abrasions (scratch inoculation)

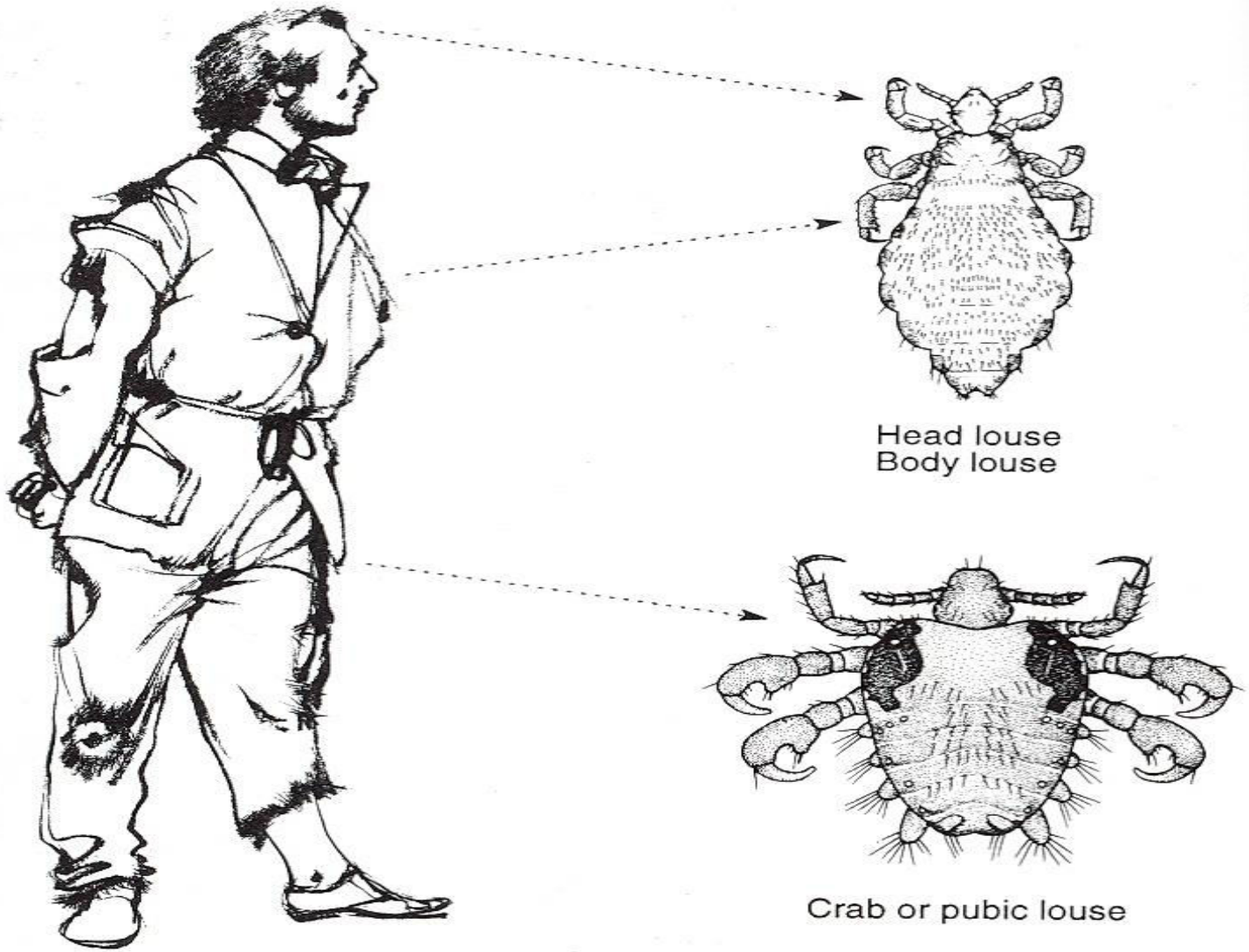
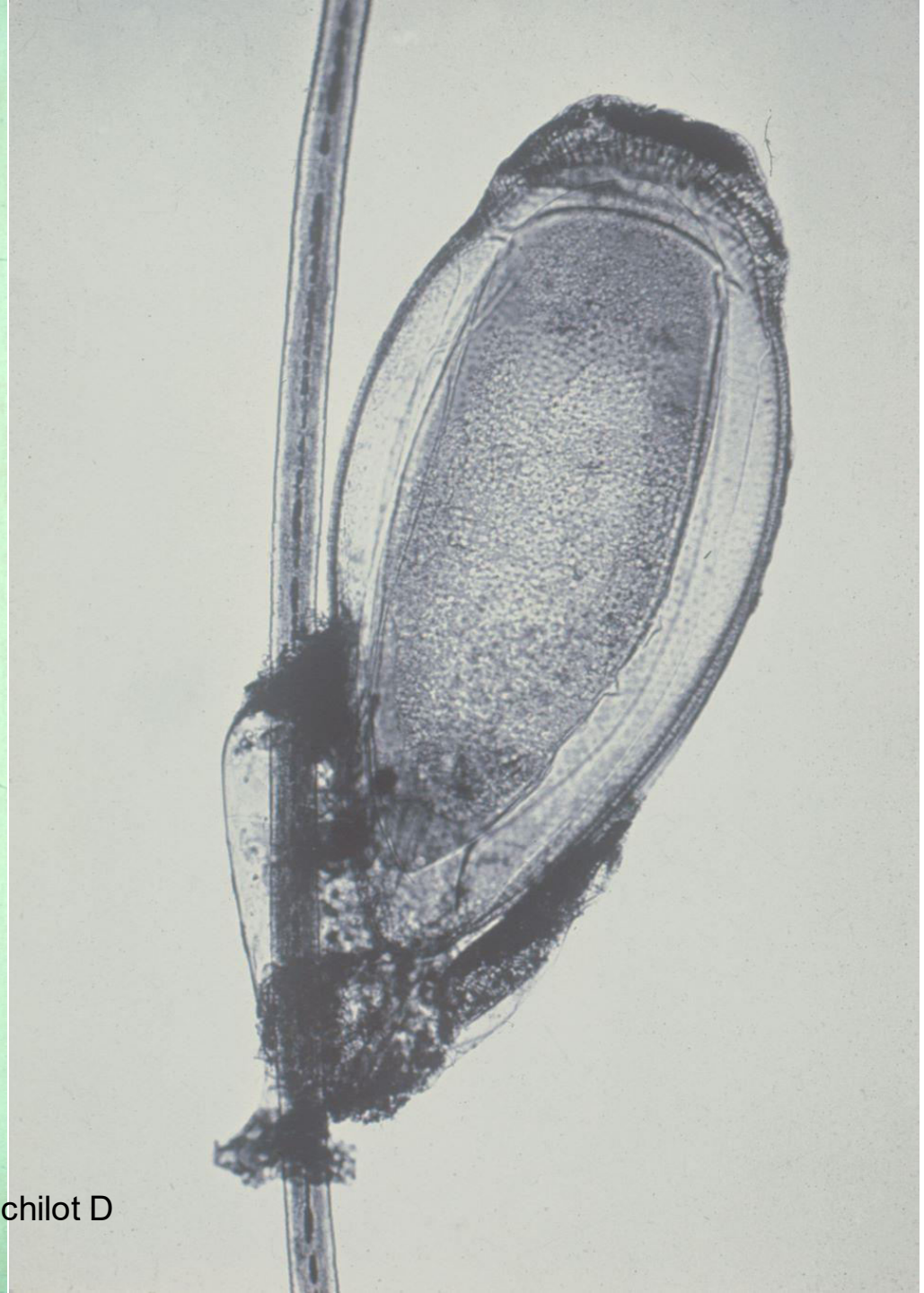
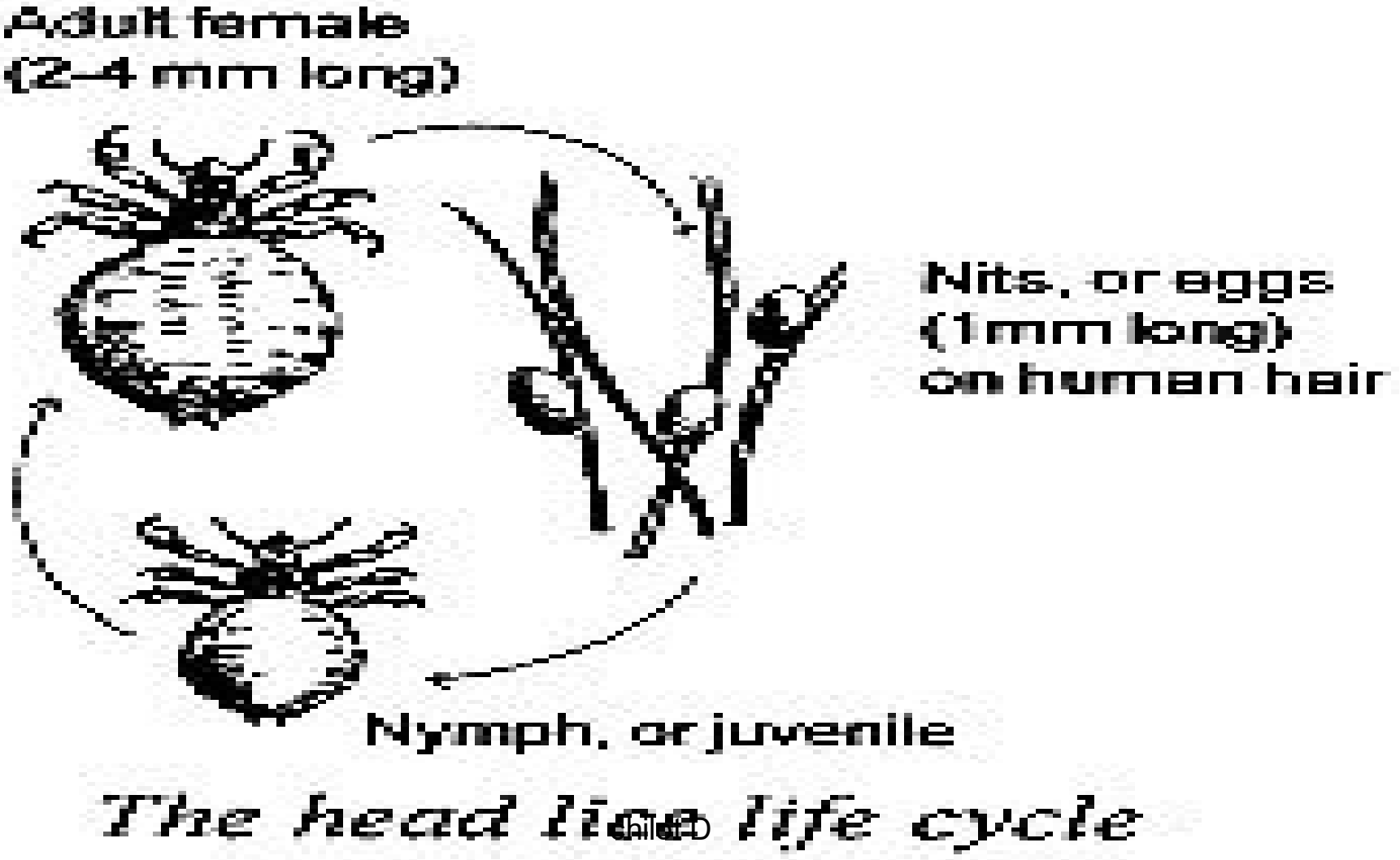


Fig. 4.15
Human sucking lice are flat wingless insects with legs adapted for grasping hairs (infested man © L. Robertson; lice © WHO).



ADULT LICE & NETS

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Typhus…

Incubation period

- From 1-2 weeks, usually 10 days

Period of communicability

- Patients are infective for lice during febrile illness and possibly for 2-3 days after the temperature returns to normal
- The louse die with in 2 weeks after infection
- Rickettsiae may remain viable in the dried louse faces for at least 2 months

Susceptibility & resistance

- Susceptibility is general
- One attack usually confers long- lasting immunity

Clinical manifestations and dx

Clinical manifestations

- Early symptoms of fever, headache, myalgia and macular eruption appear on the body
- Patients may have pneumonia, renal or CNS involvement, gastrointestinal disease
- Disease usually terminates by rapid lysis after 2 weeks of fever

Diagnosis

- Based on clinical and epidemiological grounds
- Serologic test -**Weil-felix agglutination test**

Treatment and prevention & control

Treatment

- Tetracycline 250mg 4x/day for 7 days or
- Doxycycline 200mg 2x/day for 7 days or
- Chloramphenicol 500mg 4x/day for 7 days, for children 25mg/kg

Prevention & control

- Delousing of clothes by insecticides (DDT) or dipping in to boiling water
- Public education on personal hygiene
- Treatment of cases
- Chemoprophylaxis for contacts.

Endemic typhus

- Caused by Rickettsiae typhi (Rickettsiae Mooseri)
- Also called flea-borne typhus (murine typhus fever)
- Resembles louse borne typhus, but is milder
- Reservoirs are rats and mice
- Dx and Rx are the same as louse borne typhus

RELAPSING FEVER (R.F), RECURRENT FEVER

- It is an acute infectious disease characterized by alternating febrile periods (recurrent pyrexial attacks)

Infectious agent

- Borrelia recurrentis - cause of louse borne relapsing fever
- Borrelia duttoni - cause of tick- borne relapsing fever

Occurrence and importance

- It occurs in Asia, South America and limited areas of Africa such as: Ethiopia, Sudan, Burundi)
- Occurs in epidemic form when it is spread by lice and in endemic form when it spreads by ticks
- In both species of relapsing fever, usually, about 2-10% of untreated persons die
- But the mortality rate may be as high as 50% during epidemics of louse borne relapsing fever
- The bacteria pass into the ovary of the tick and offsprings of an infected tick are automatically infected without having sucked infectious blood themselves
- Ticks live in crackes and crevices of walls and floors

Reservoir and mode of transmission

Reservoir

- Humans for Borrelia recurrentis
- Wild rodents and soft ticks through **trans ovarian transmission** for tick borne relapsing fever

Mode of transmission

- Tick- borne relapsing fever is transmitted through bite wound deposited with saliva **and coxal fluid** of **soft ticks** which usually feed quickly at night in or near houses and then leave the host
- Louse borne relapsing fever is acquired by crushing an infected **body louse** between the finger nails or the teeth so that it contaminates the bite wound or an abrasion of the skin or the mucous membrane of the mouth

Soft ticks

Fig. 4.23
A soft tick, *Ornithodoros moubata*, vector of relapsing fever in Africa (by courtesy of the Natural History Museum, London).

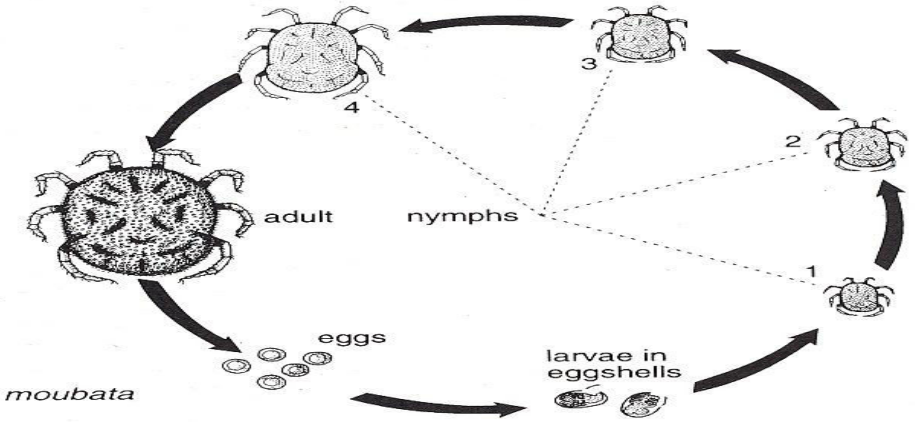


Fig. 4.24
Life cycle of the soft tick, *Ornithodoros moubata* (*30*). Copyright Blackwell Science Ltd.

Hard ticks

RF…

Incubation period

- 5-10 days usually 8 day

Period of communicability

- Louse becomes infective 4-5 days after ingestion of blood from an infected person and remains so for life (20-40 days)

Susceptibility & resistance

- Susceptibility is general
- Immunity after clinical attacks is not known, repeated infection may occur

Clinical manifestations

- Sudden onset of illness with chills, fever and prostration, head ache and arthralgia
- There may be nausea and vomiting, jaundice and liver swelling
- After 4-5 days the temperature comes down, the patient stays free for 8-12 days and then a relapse follows with the same signs but less intense.
- In untreated cases there may be up to **ten relapses**

Diagnosis and treatment

Diagnosis

- Clinical and epidemiological grounds
- Giemsa or Wright stain (blood film)

Treatment

- Admit the patient
- Secure an iv line before administering penicillin
- Administer 400,000-600,000 iu pro.penicillin im stat
- Tetracycline during discharge for 03 days
- Chloramphenicol in infants & children can be used in place of tetracycline

Patient care

- Maintain body temperature
- Close vital sign monitoring for 3 hrs after medication
- Check whether there is reaction or not & report
- Comfort the patient by providing antipain
- Shaving of hair & delousing of clothes

Prevention and control

- Control of vectors
- Personal hygiene
- Health education about hygiene & of disease transmission
- Delousing of patient's clothes and his/her family
- Chemotherapy of cases and chemoprophylaxis for contacts.

SCHISTOSOMIASIS (BILHARZIA)

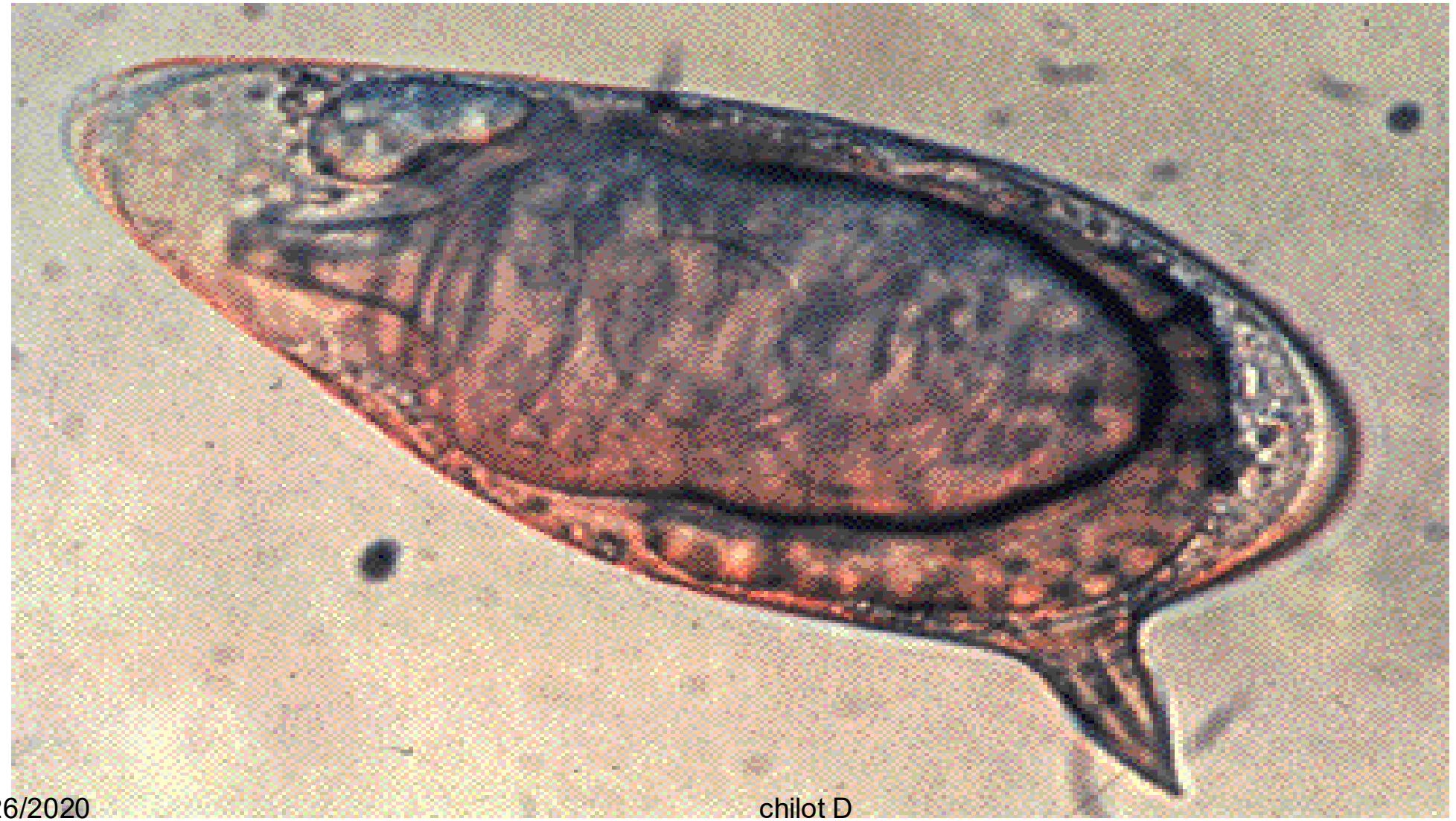
- It is a blood fluke (trematode) infection
- Adult worms live with in mesenteric or vesicle veins of the host over a life span of many years /up to **10 years/**
- It is a chronic disease caused by the reaction of the body to the **eggs** of a worm.

Infectious agent

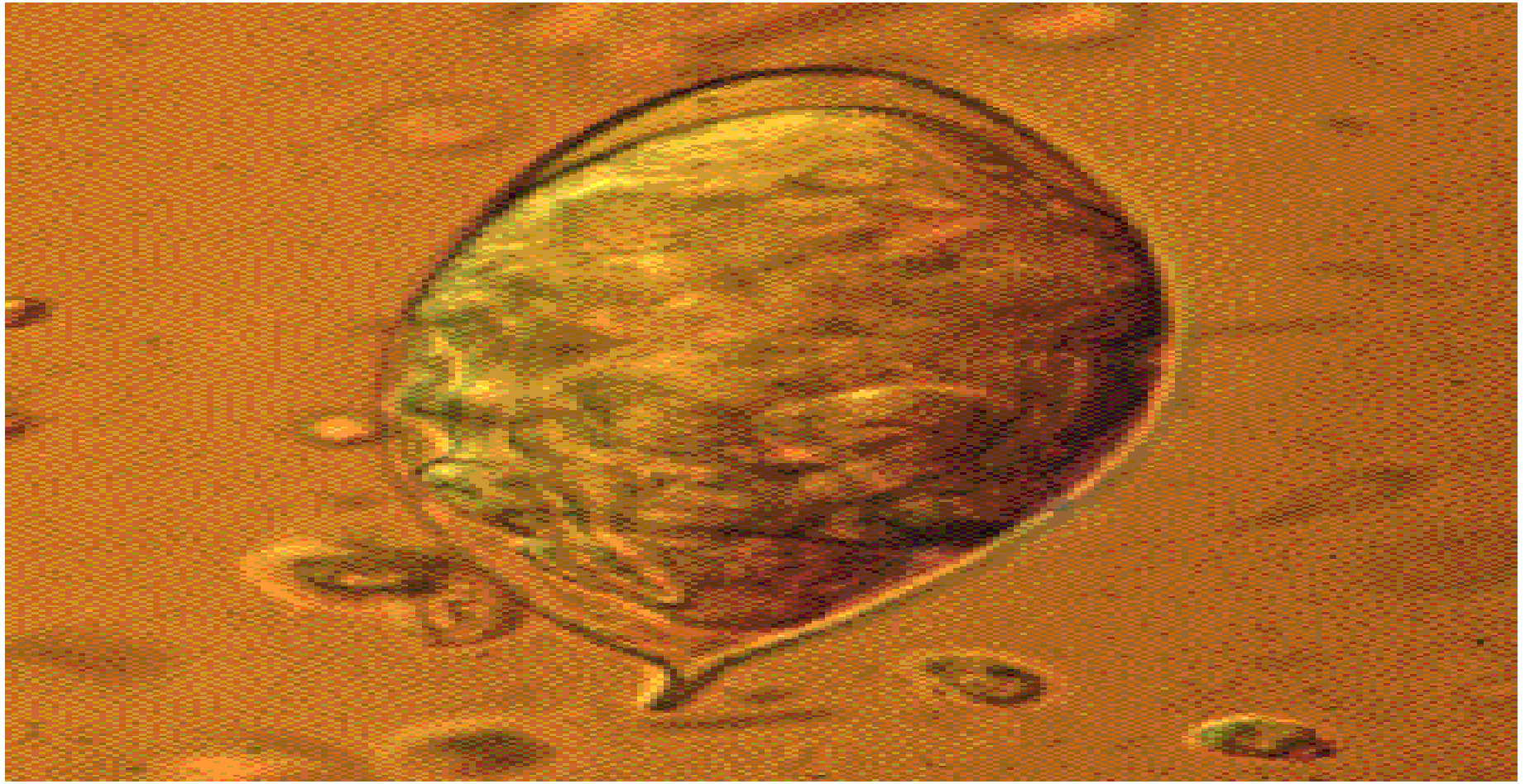
- The major schistosoma species that cause schistosomiasis of human are:
  - Schistosoma mansoni *
  - Schistosoma hematobium *
  - Schistosoma japonicum

*most prevalent species in Africa

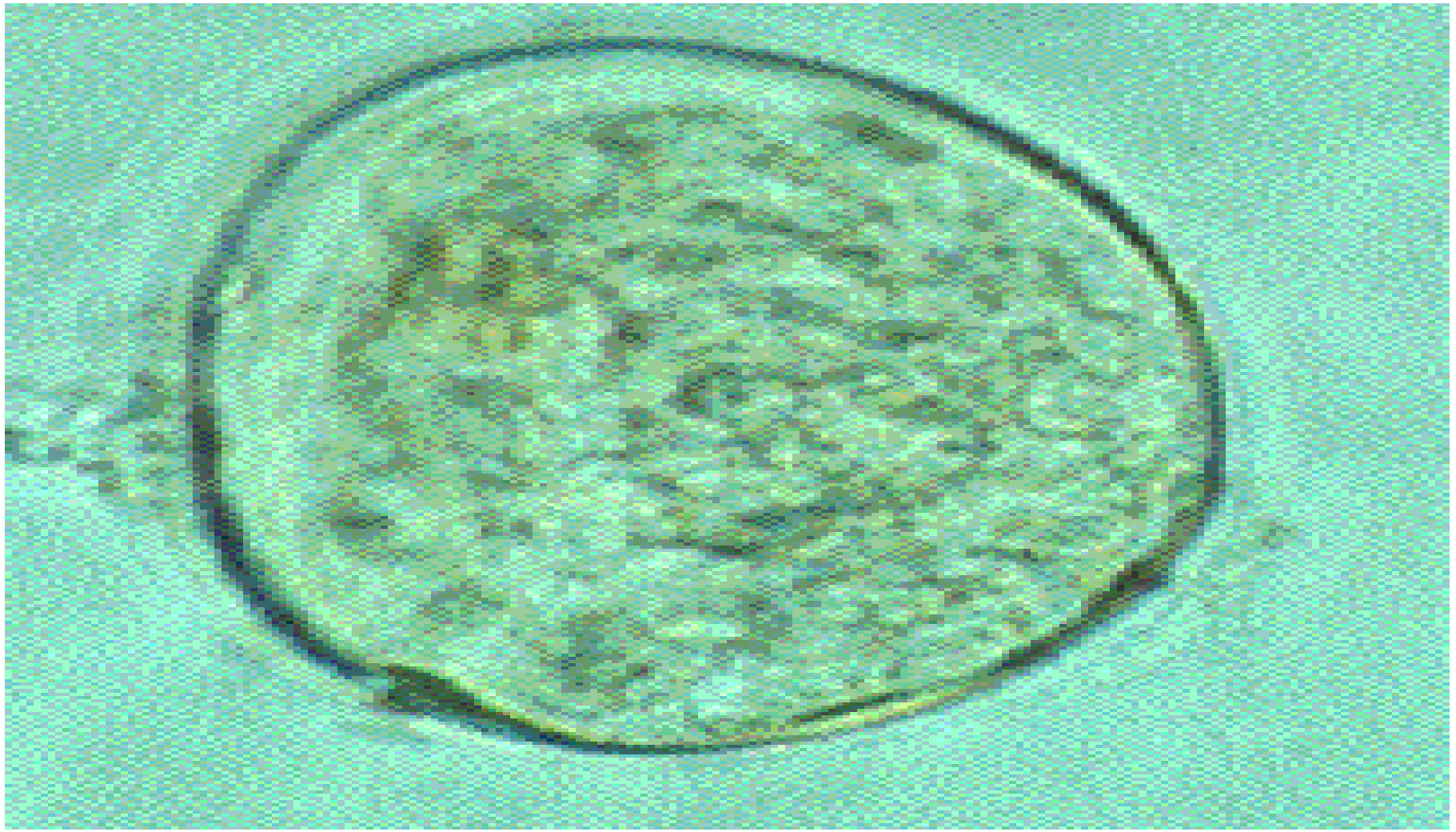
Schistosoma-mansoni-egg



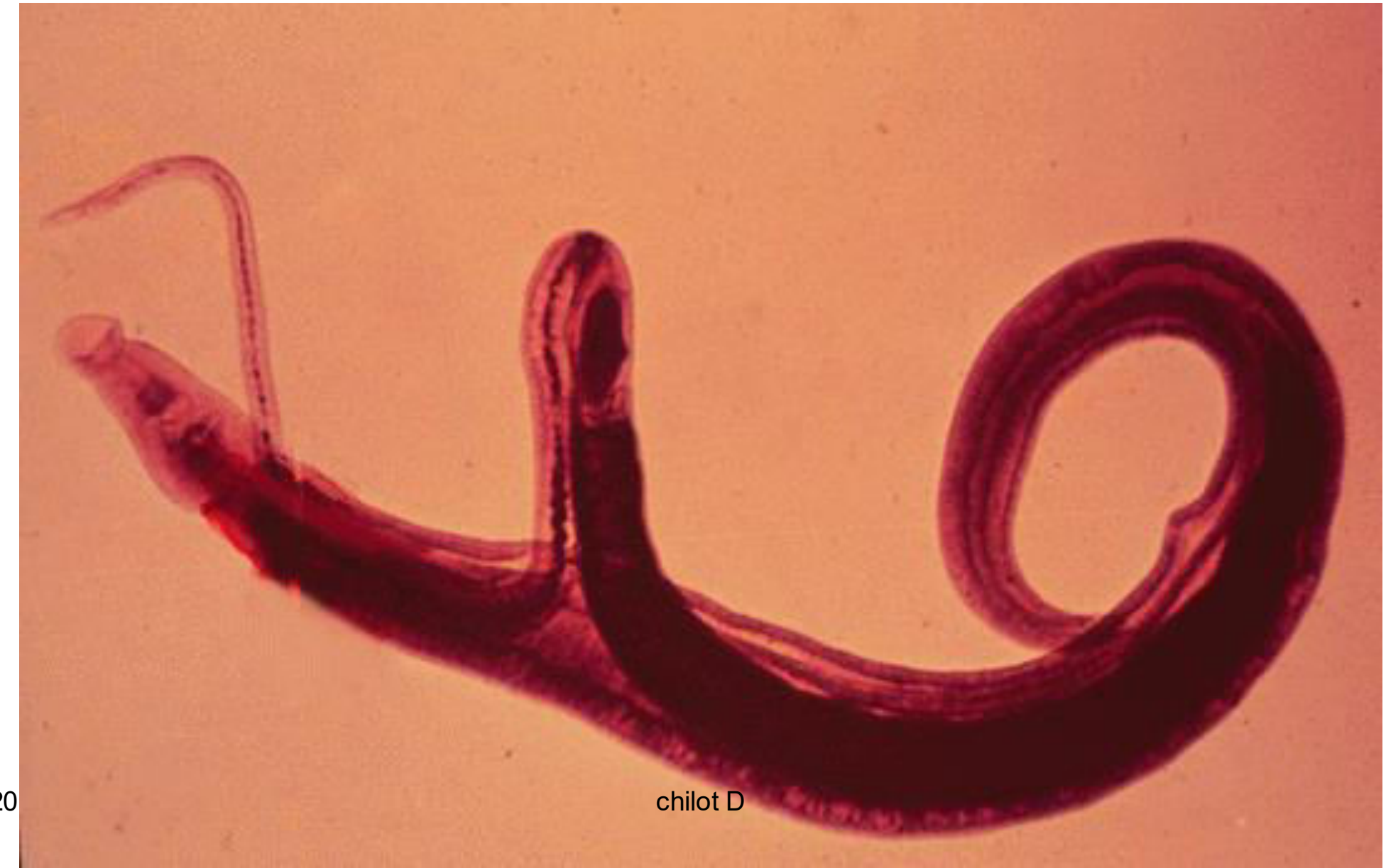
Schistosoma-hematobium-egg



Schistosoma-japonicum-egg



Adult schistosomes



Vectors of schistosomiasis

Snail vectors (intermediate host) of Schistosomiasis that harbor the asexual stages include:

- Balinus for S. hematobium
- Biomphalaria for S. mansoni
- Onchomelania for S. japonicum

Occurrence and importance

- S. mansoni is found in south America, Caribbean islands, Africa and Middle east
- S. hematobium is found in Africa and the middle east
- S. japonicum is found in the far East
- Most infected individuals show few or no signs and symptoms, and only a small minority develop significant disease

Reservoirs

- Principal reservoir for S. mansoni and S. hematobium is **man**
- Other animals such as dog, cat, pig, cattle water buffalo, horse and wild rodents are hosts for S. japonicum

Schistosomiasis…

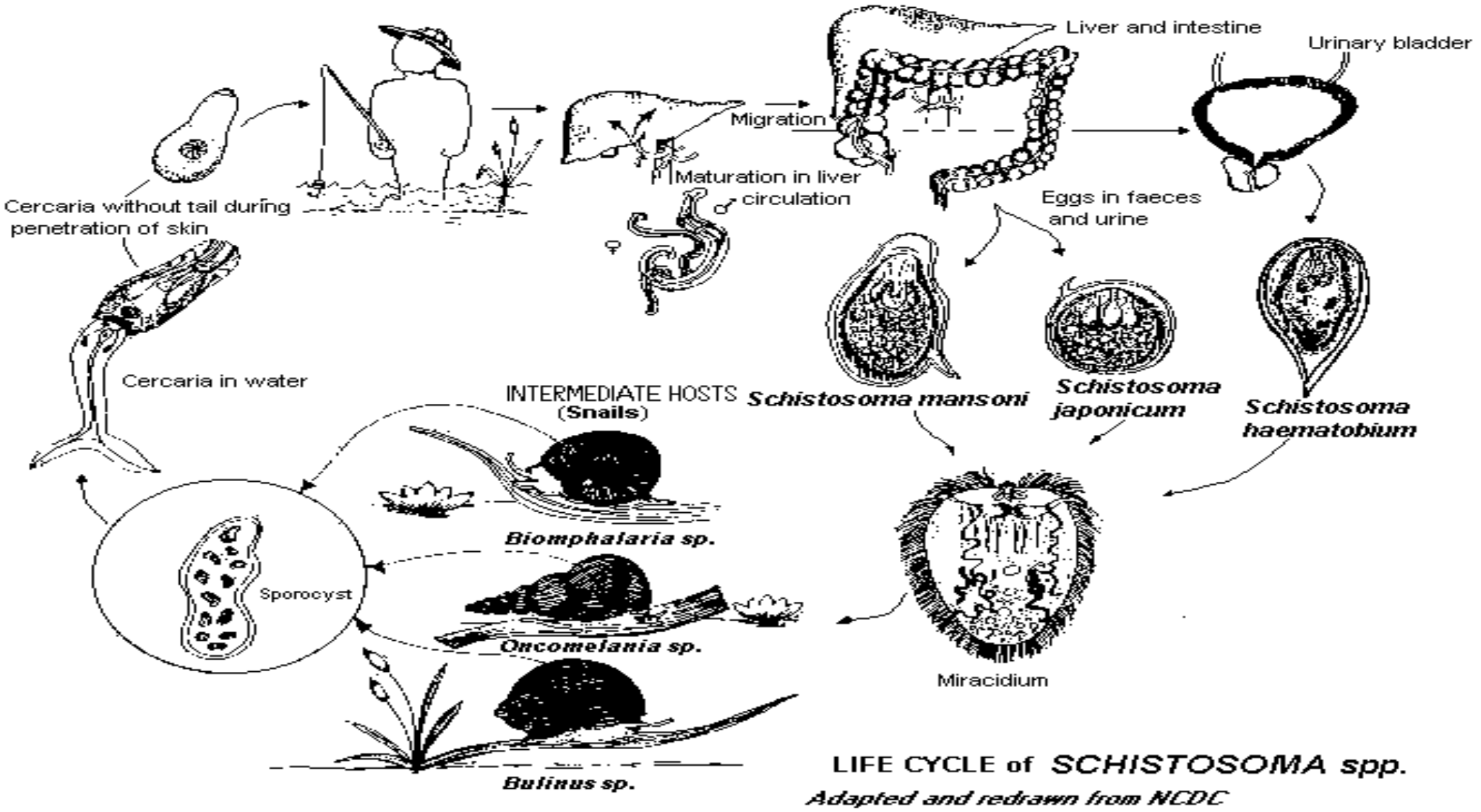
Mode of transmission

- Infection is acquired from water containing free swimming larval forms (cercariae) that have developed in snails

Incubation period

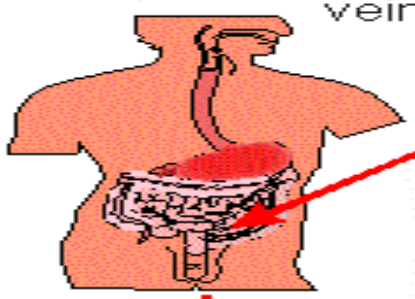
- Acute systemic manifestations (kateyama fever) may occur in primary infections 2-6 weeks after exposure

Schistosoma life cycle



The Life Cycle of *Schistosoma* spp.
(the causative agent of schistosomiasis)

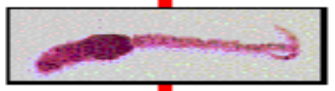
Worms reach sexual maturity in veins of abdominal cavity, females produce eggs, eggs enter intestinal tract or bladder.



Eggs passed in urine or feces into fresh water

Miracidium hatches from egg, infects first intermediate host (a snail)

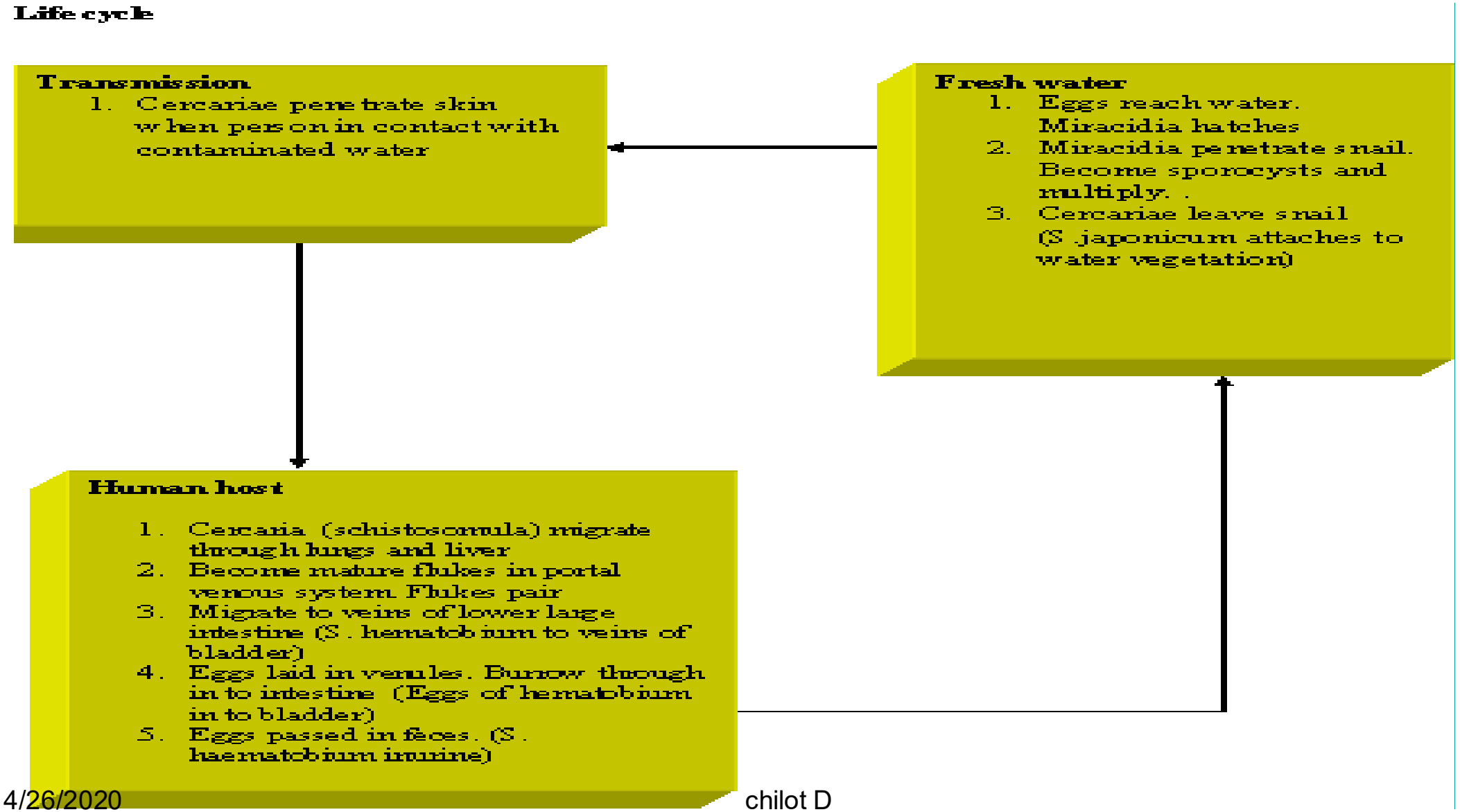
Cercariae leave snail, penetrate skin of human who is in water



Immature worm enters the blood stream and eventually ends up in veins near the intestine or bladder

(Parasites and Parasitological Resources)

Schistosomiasis, cont'd…



Clinical manifestations

4 stages:

1. **Invasion stage**

- Cercariae penetrate skin & cause dermatitis with itching papules (**swimmer's itch**) and local edema
- Cercariae remain in skin for 5 days before they enter the lymphatic system and reach the liver

Clinical manifestations…

2. Maturation

- Schistosoma mature in the liver
- Fever, easinophilia, abdominal pain and transient generalized urticaria /skin reaction with sever itching/- **(katayama syndrome)**
- Worms descend the portal vein & migrate to mesenteric veins in the intestinal wall and to bladder plexus.

Clinical manifestations …

3. Established infection

- This is a stage of egg production and eggs reach to the lumen of bladder and bowel
- Some eggs penetrate the tissue; reach the bladder and intestinal wall and are discharged with urine and feces.
- Eggs that couldn’t penetrate the tissue are carried with blood to the liver & lungs

Established infection…

- Other eggs that fail to reach the lumen of the bladder or bowel provoke an inflammatory reaction
- The inflammatory reaction, resulting in fibrosis causes signs and symptoms of schistosomiasis
- Sign of colitis with bloody diarrhea and cramps in S. mansoni infection.

Clinical manifestations…

4. Late stage

- This is the stage of fibrosis, which occurs where there are eggs in the tissues.
- Eggs of urinary schistosomiasis cause damage to the urinary tract leading to painless terminal hematuria
- There is progressive damage to the bladder, ureters and kidneys resulting in:
  - ➢ Stricture of urethra /around bladder/ leading to urine retention or fistula
  - ➢ Dilation of ureters (**hydroureter**) and kidney (**hydronephrosis**) possibly leading to kidney failure
  - ➢ Calcification of bladder that can result in **bladder carcinoma**

Late stage…

- In the liver portal hypertension leads to
  - Hyperspleenism and anemia
  - Esophageal varices and bleeding
  - hepatospleenomegally and ascitis
- In the lungs fibrosis results in pulmonary hypertension, which leads to congestive cardiac failure

Diagnosis and treatment

Diagnosis

- Demonstration of ova in urine or feces
- Serology - ELISA tests (using soluble egg antigen) - useful in many chronic cases

Treatment

- Praziquantel- effective in a single dose against all forms of schistosomiasis: 40 mg/kg po stat
- Oxamniquine- used exclusively to treat intestinal schistosomiasis. S. mansoni is less susceptible
- Metrifonate- safe & effective for treatment of urinary schistosomiasis -7.5 mg/kg po stat

Prevention and control

- Treatment of cases
- Intermittent irrigation
- Drainage of water bodies
- Clearing of vegetation in water bodies to deprive snails of food and resting place.
- Educating the public about the mode of transmission and ways of prevention
- Proper disposal of human feces and urine
- Avoid swimming in water bodies known to have the infection
- Use rubber boots to prevent exposure to contaminated water

YELLOW FEVER /JUNGLE FEVER/

- An acute infectious viral disease characterized by sudden onset of fever, hepatic and renal failure, and jaundice
- It is a zoonosis of forest monkeys.

Infectious agent

- Yellow fever virus

Reservoir

- Urban areas - humans and Aedes aegypti mosquitoes.
- Forest areas - vertebrates other than human (mainly monkeys) and forest mosquitoes.

Incubation period

- **3-6 days**

Transmission and distribution

- The disease exists in two transmission cycles; namely:
  - ➢ The sylvatic or jungle cycle
  - ➢ An urban cycle

The sylvatic or jungle cycle

- The yellow fever virus mainly occurs in populations of monkeys in dense forests and gallery forests in Africa and South and Central America
- In Ethiopia the disease is found in south west areas (Gambella region).
- It is transmitted from monkey to monkey by forest-dwelling mosquitoes
  - Aedes species in Africa
  - Haemagogus and Sabethes in South and Central America
- These mosquitoes occasionally bite humans when they enter forests and may thus transmit the virus from the monkey reservoir to the human population.

An urban cycle

- In Africa, monkeys sometimes leave the forest in search of bananas implantations and may then infect the local mosquito species, which in turn infect humans living or working on the plantations.
- People infected in or near forests can carry the virus to rural or urban areas where Aedes aegypti or related mosquitoes can pick it up and transmit it among the human population.
- Such situations can result in serious epidemics and many deaths.

Sylvatic, rural and urban transmission cycles of yellow fever in Africa

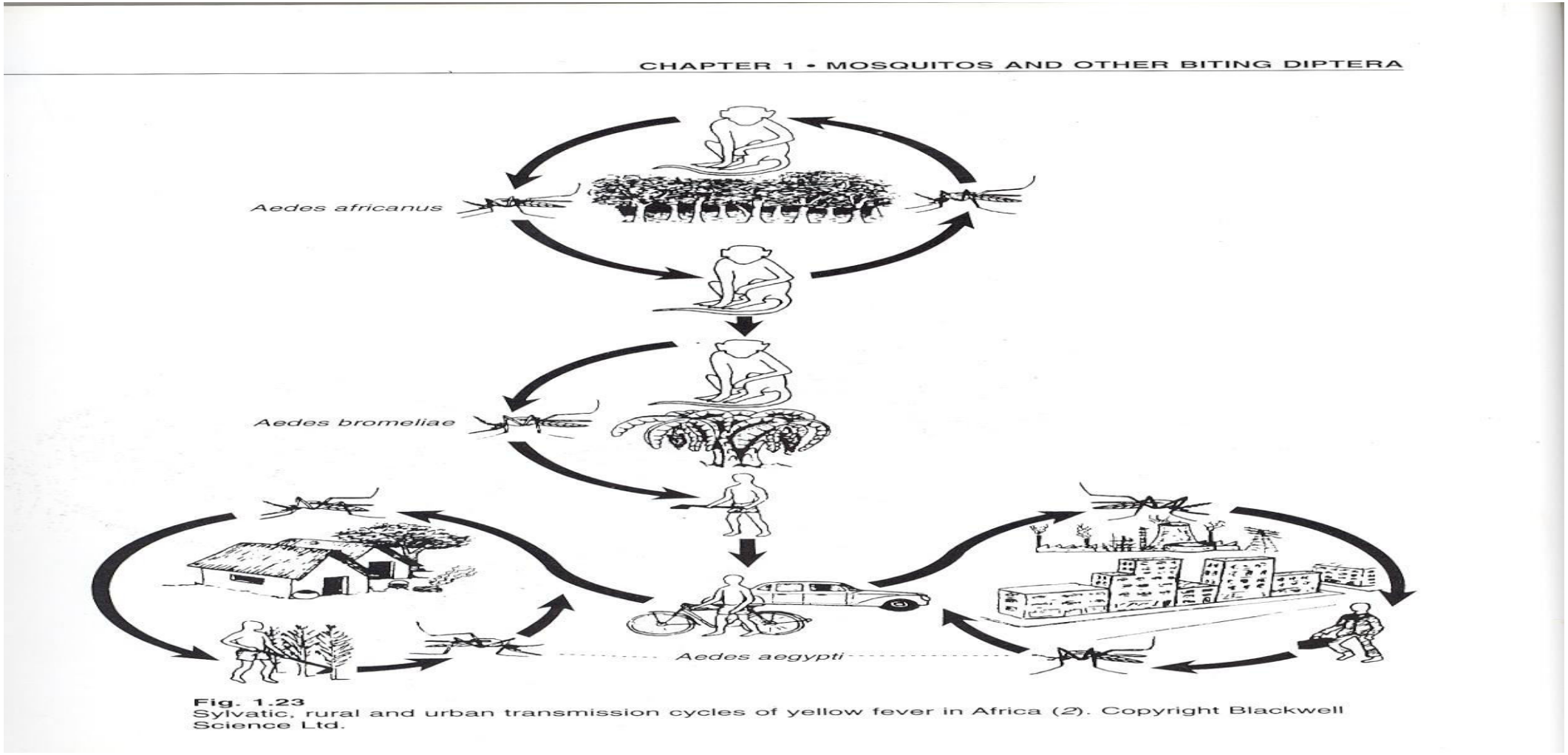


Fig. 1.23
Sylvatic, rural and urban transmission cycles of yellow fever in Africa (*2*). Copyright Blackwell Science Ltd.

Clinical manifestations

- Typical attacks are characterized by sudden onset of fever, chills, headache, backache, generalized pain, prostration, nausea and vomiting.
- Slow and weak pulse
- Bleeding tendency is common resulting in epistaxis, bleeding of gums, hematemesis, and melaena.
- Jaundice occurs due to liver cell necrosis and this may result in liver failure and death.
- Albumin urea occurs due to nephrosis and this may result in kidney failure and anuria.
- Patients surviving the seventh day of the disease usually recover.

DX, D.DX and R_X

Diagnosis

- Clinical manifestation
- History of residence and/or travel to endemic area
- Yellow fever should be suspected when patients die from a feverish disease with jaundice especially when there was albuminuria

Differential diagnosis

- Viral hepatitis
- Malaria
- Leptospirosis
- Relapsing fever

Treatment

- No specific treatment.

Nursing management and prevention and control

Nursing management

- Monitor vital signs regularly
- Maintain body temperature to normal
- Monitor input and output balance
- Keep patient in screened rooms or under mosquito nets to avoid further infection.

Prevention and control

- Active immunization of all people greater than 9 months of age gives good protection for at **least 10 years**.
- Eradication or control of Aedes aegypti mosquitoes in urban areas
- Notification of the disease to the concerned health authorities.

LEISMANIASIS

- It is a polymorphic protozoan disease of the skin and mucous membrane **or**
- A chronic **systemic disease** caused by a number of species of the genus leishmania.
- It is common where dog populations are high.
- Generally more common in rural than urban areas.

Infectious agents and reservoir

Infectious agents:

For cutaneous leishmaniasis:

- Leishmania tropica
- Leishmania major
- Leishmania infantum
- Leishmania aethiopica*

For visceral Leishmaniasis:

- Leishmania donovani*
- Leishmania infantum (Leishmania chagasi)*

*Common agents in Ethiopia.

Reservoirs

  - Locally variable: include human beings, wild carnivores and domestic dogs.

Types, occurrence and importance of leishmaniasis

Visceral leishmaniasis (Kala-azar: meaning black sickness)

- It is a disease of the **internal organs** and is often fatal if left untreated.
- Most important immunological feature is marked suppression of cell mediated immunity
- It is endemic in East Africa, the Indian subcontinent and South America, and occurs sporadically in China, the Mediterranean region, south-west Asia and the countries of the southern part of the former USSR

Mucocutaneous leishmaniasis

- It is a disease of the skin and mucosal tissues in the nose and mouth, and can lead to gross deformities.
- It occurs in Central and South America
- Oronasal leishmaniasis due to other leishmania species has been recorded in Ethiopia and Sudan.

Cutaneous leishmaniasis

- It results in ulcers of the skin and is the **most common** form of leishmaniasis
- Occurs in Africa, South America, the Indian subcontinent, south-west Asia, the Mediterranean region and the countries of the southern part of the former USSR.

Transmission

- Most forms of leishmaniasis are primarily infections of small mammals.
- Humans are often infected by **sandflies** which previously fed on infected animals.
- The risk of being infected is higher for people who sleep outdoors or have outdoor activities at night.
- An increased risk also occurs in places where there are infected rodents or other host animals.
- Person to person transmission by blood transfusion, and sexual contact has been reported, but rare.

Sand fly, vector of leishmaniasis

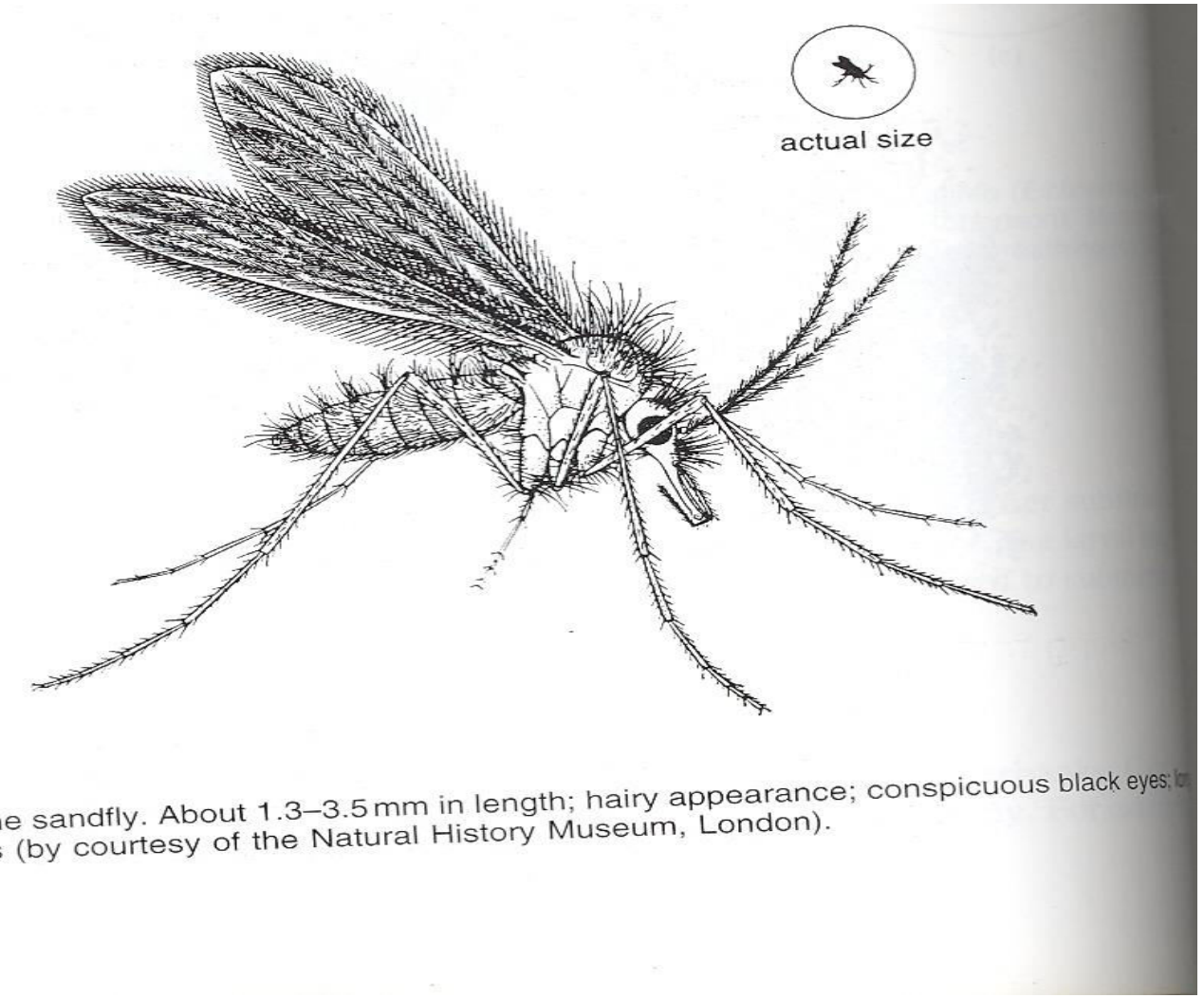


Fig. 1.11
Phlebotomine sandfly. About 1.3–3.5 mm in length; hairy appearance; conspicuous black eyes; long stilt-like legs (by courtesy of the Natural History Museum, London).

Life cycle of leishmania

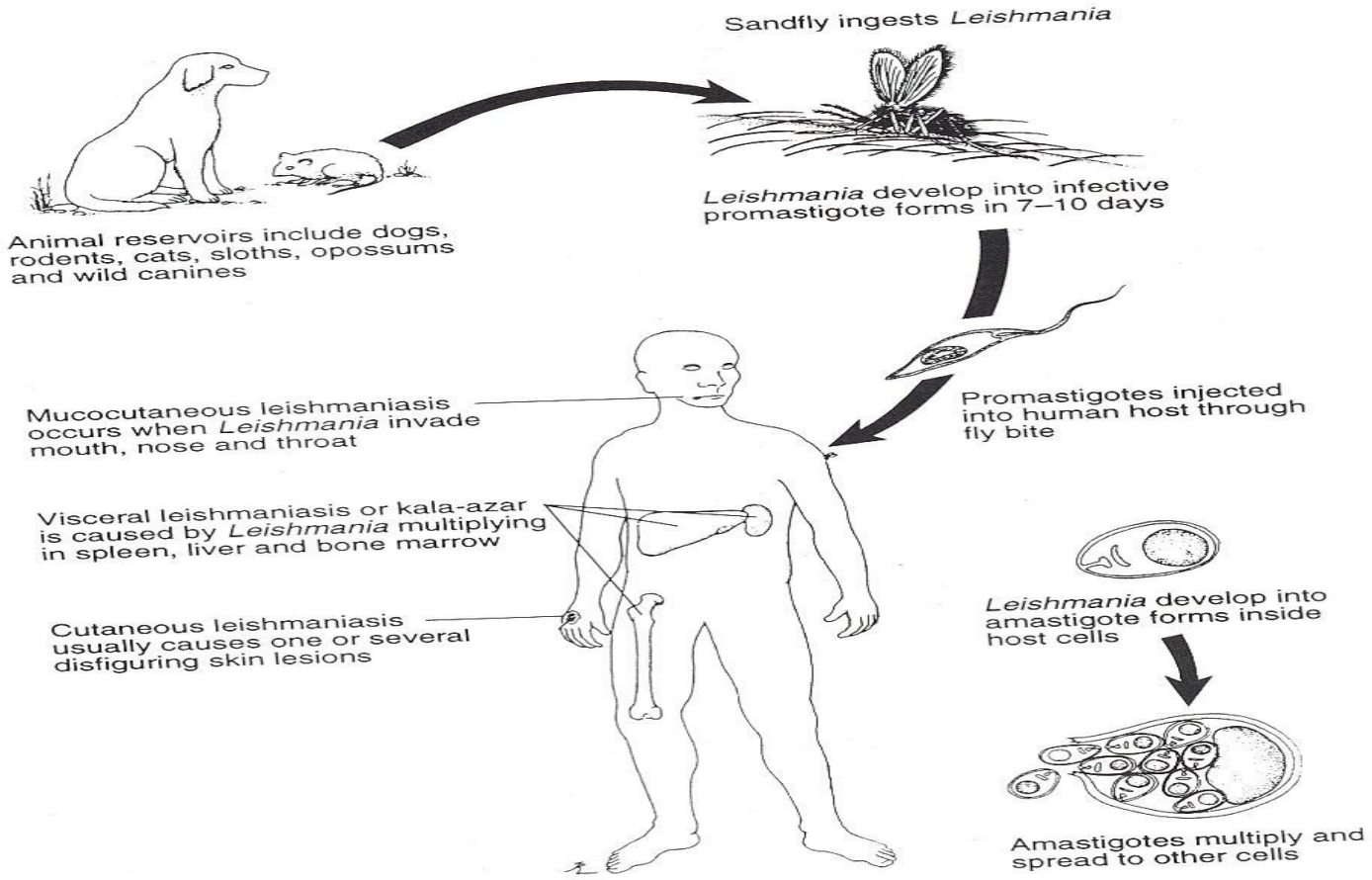
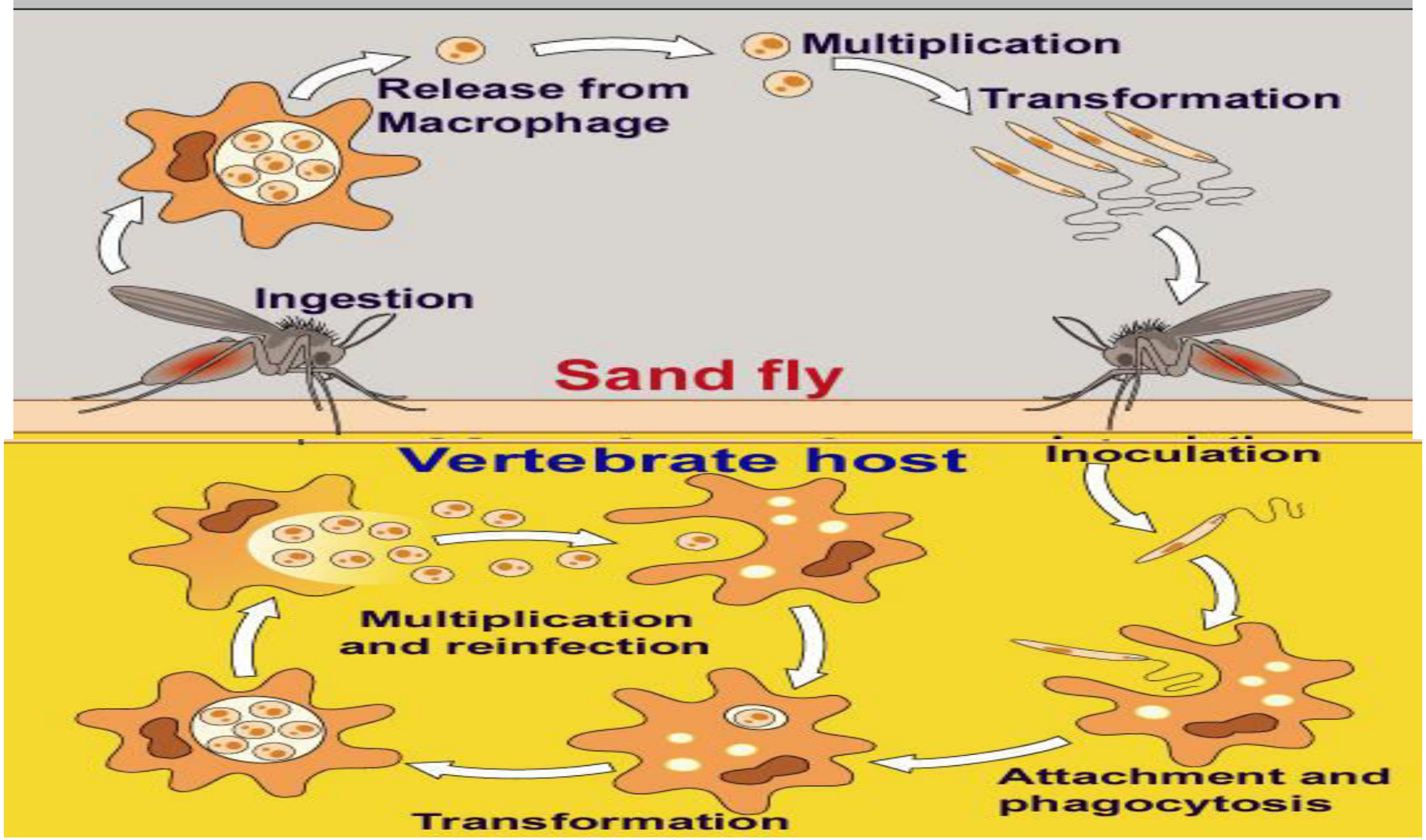


Fig. 1.32
Life cycle of *Leishmania* (by Taina Litwak for the United States Agency for International Development's VBC Project).


Multiplication
Release from Macrophage
Transformation
Ingestion
Sand fly
Vertebrate host
Inoculation
Multiplication and reinfection
Attachment and phagocytosis
Transformation

Clinical symptoms

Visceral leishmaniasis (Kala-azar)

- Children are most affected, and twice as many males are affected as females.
- The disease starts slowly with fever, malaise, loss of weight and, in many cases, cough and diarrhea.
- A major clinical sign is enlargement of the spleen which starts early and is progressive and massive
- Hepatomegaly and LAP also occur but are not so prominent.
- Prothrombin production is commonly decreased

Visceral leishmaniasis...

- The skin becomes dry, rough arid darkly pigmented /hence the name **Kala-azar**/ .
- The hair becomes thin and brittle.
- Epistaxis and bleeding gums are common
- Anemia occurs as a result of infiltration of bone marrow as well as by the increased destruction of erythrocytes due to hypersplenism
- Most untreated patients die in about 2 years due to some intercurrent diseases such as dysentery or tuberculosis

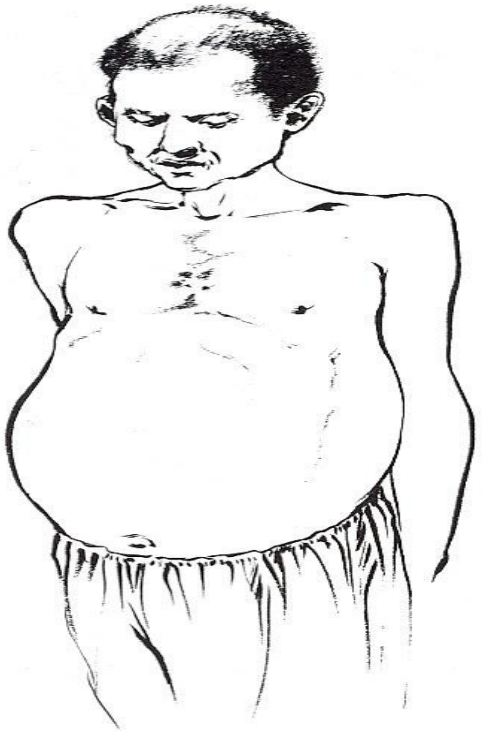


Fig. 1.33
A typical clinical sign of visceral leishmaniasis is enlargement of the spleen and liver.

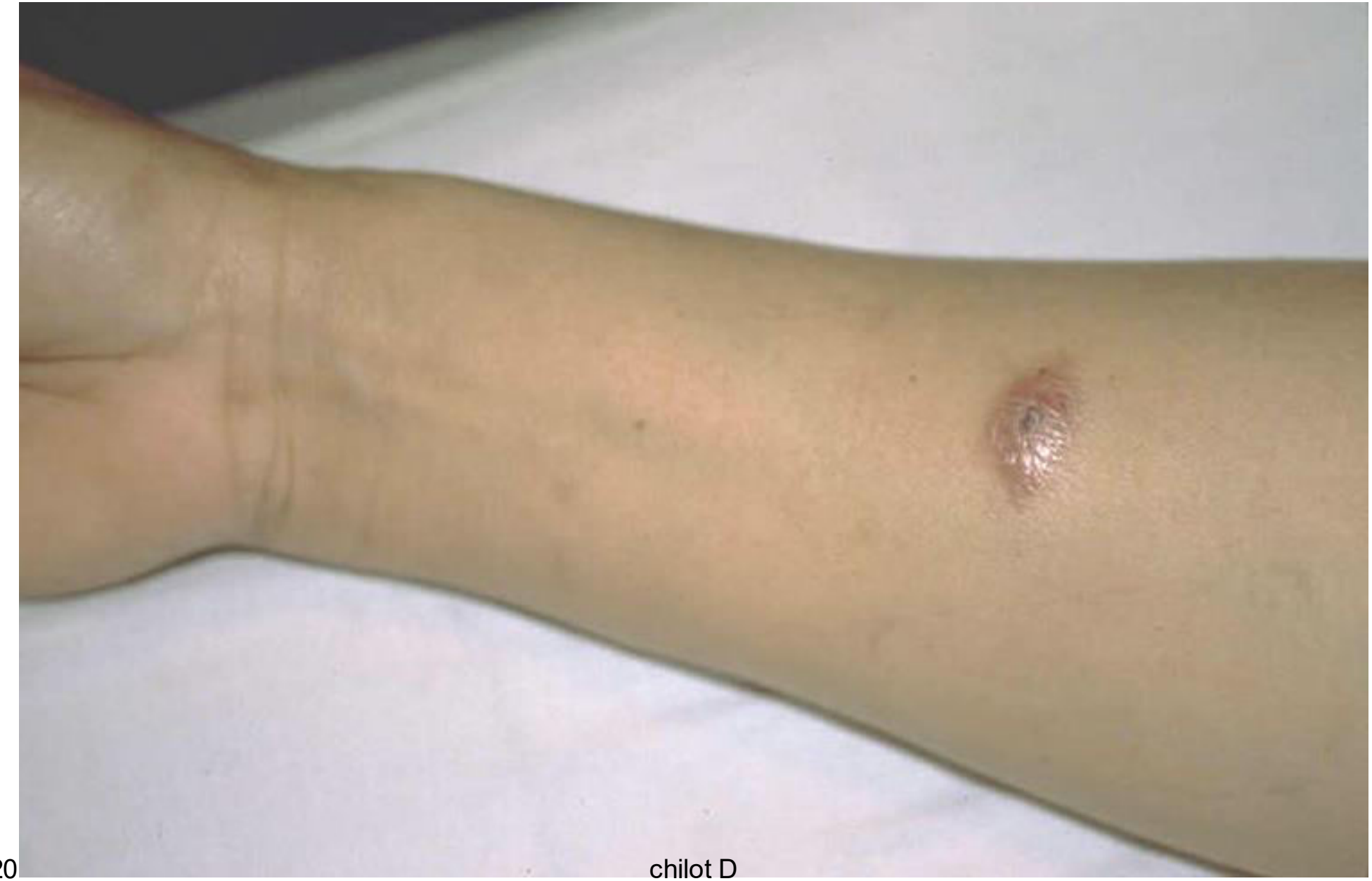
Fig. 1.34
Cutaneous leishmaniasis may typically cause ulcers which, after healing, leave permanent depressed scars.

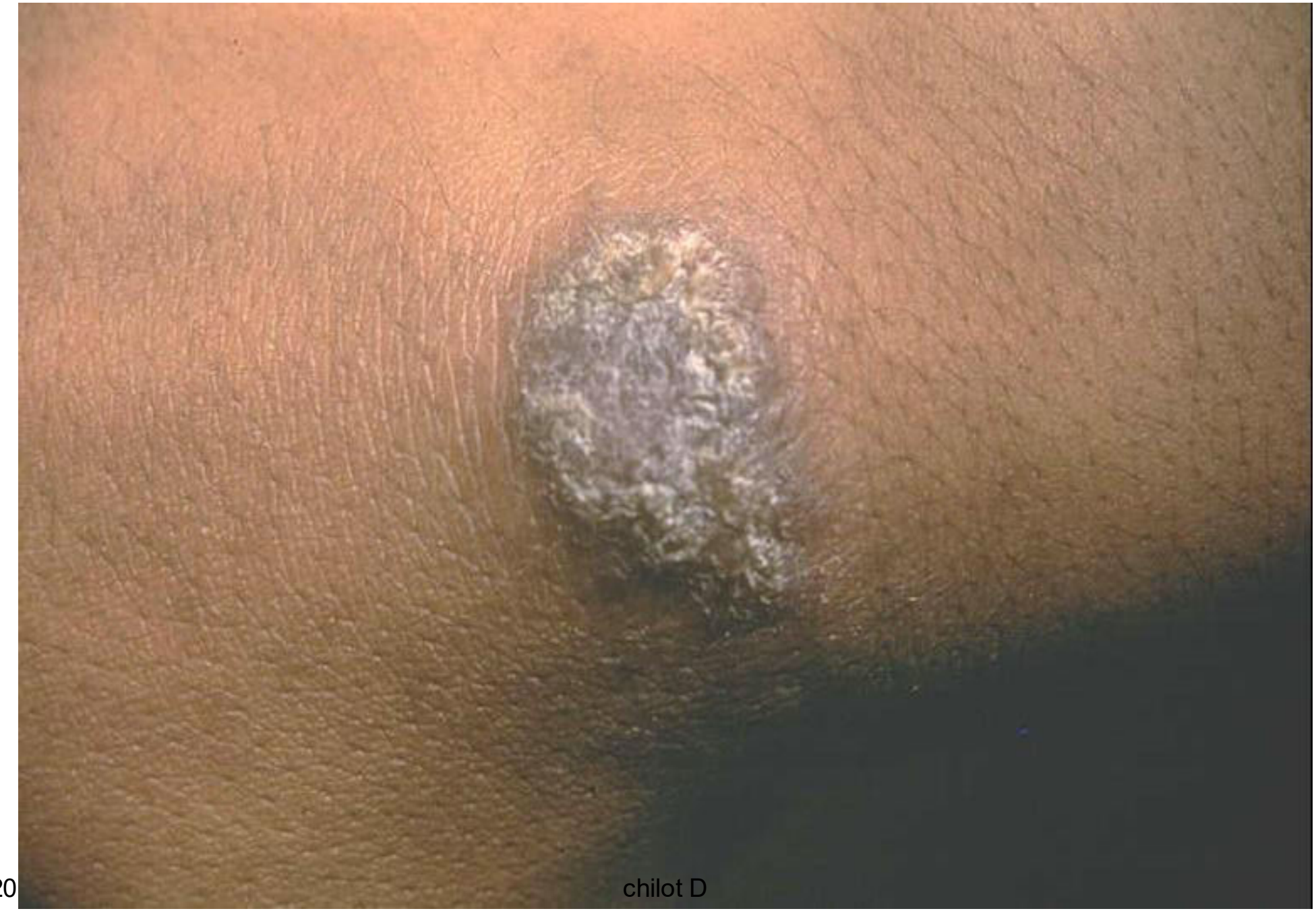
Cutaneous leishmaniasis

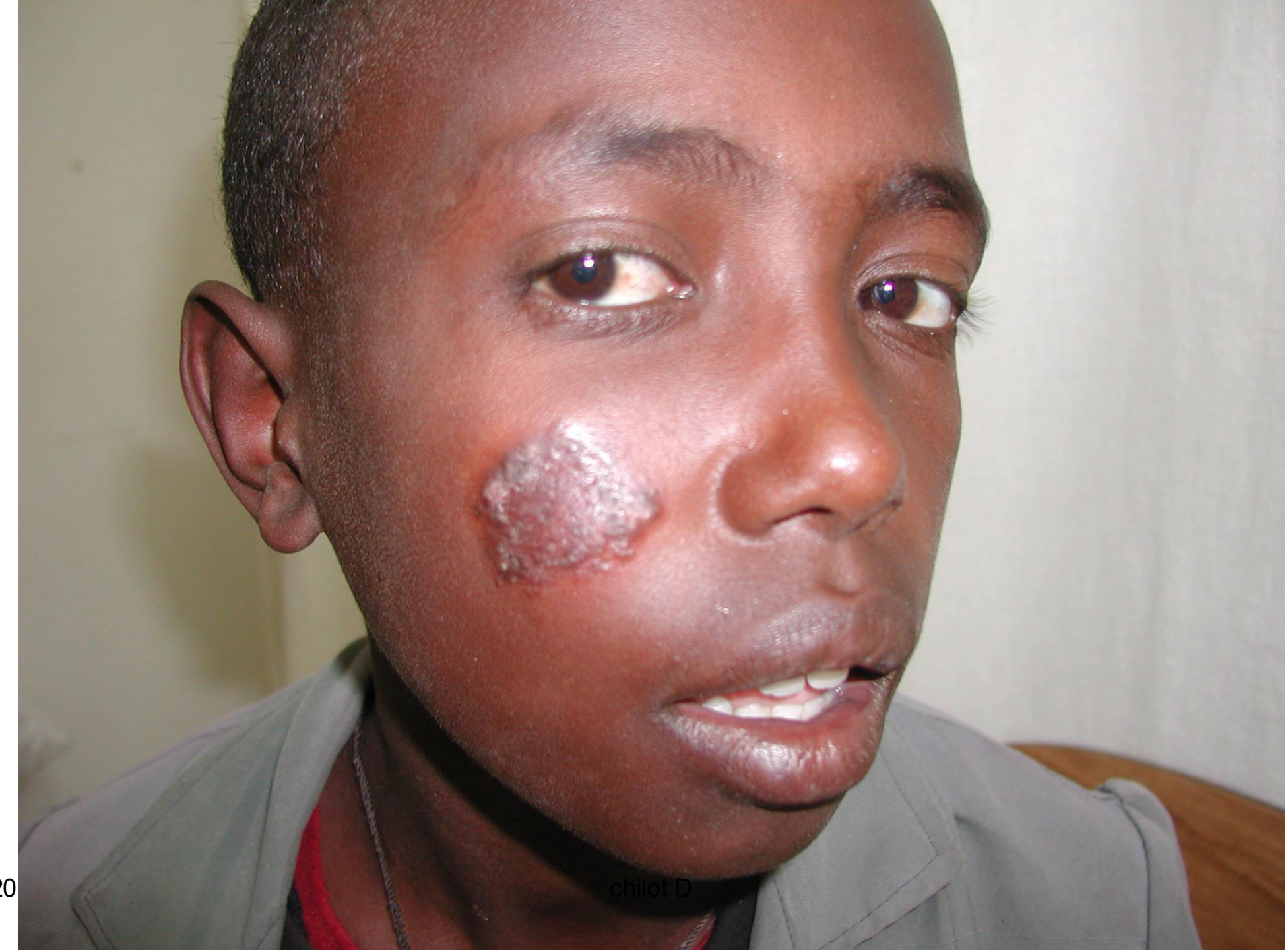
- A typical ulcer starts as a nodule at the site of the sand fly bit; a crust develops in the middle which, if it falls away, exposes the ulcer
  - The ulcer heals gradually and leaves a permanent depressed scar different in color from the surrounding skin.
  - Depending on the parasite species, healing takes place spontaneously in periods ranging from two months to several years.
  - Some types do not heal without treatment and may develop in to mucocutaneious leishmainiasis
  - Sometimes the disease spreads via the lymphatic system and causes ulcers all over the body.

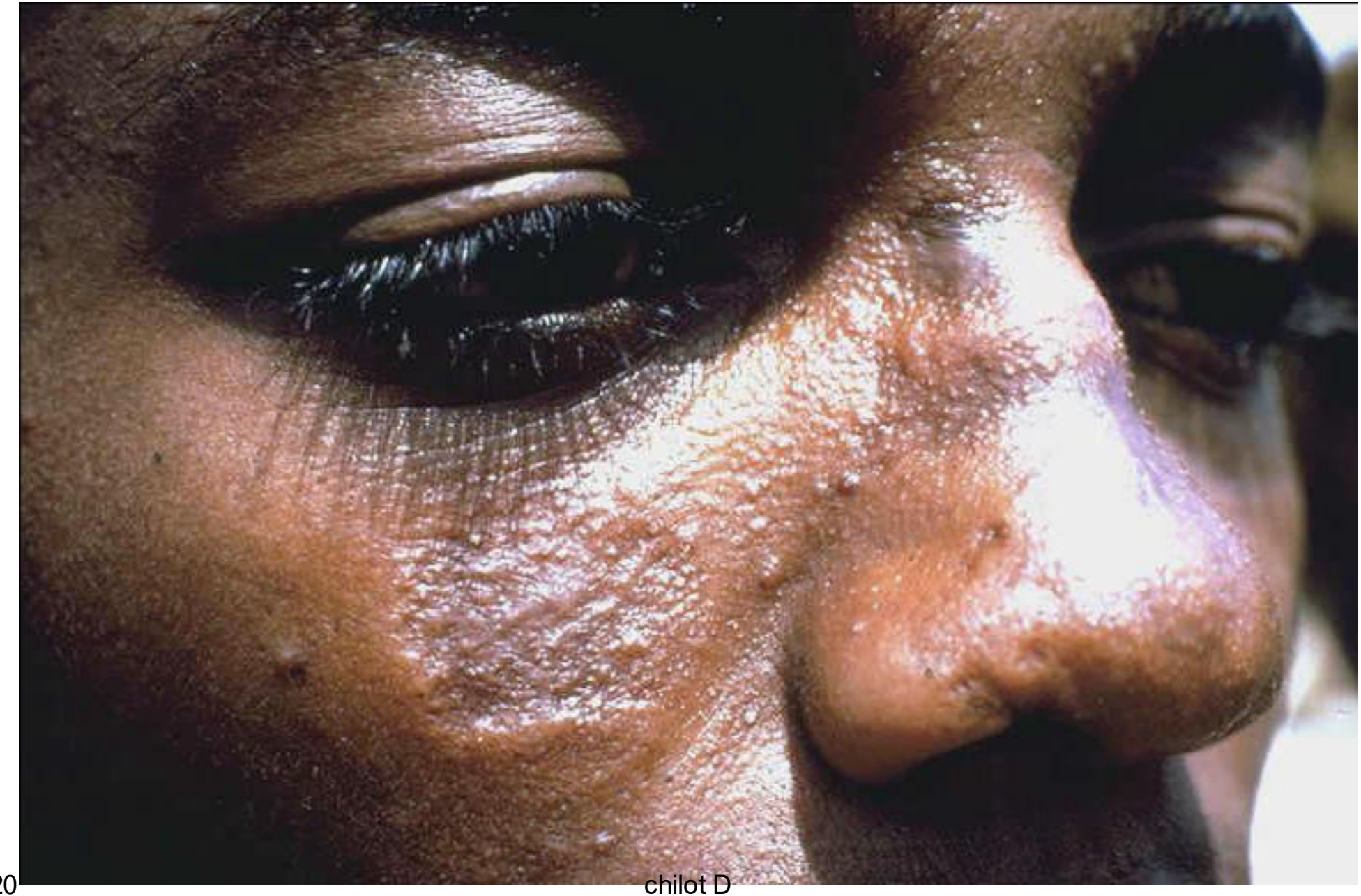
Mucocutaneous leishmaniasis

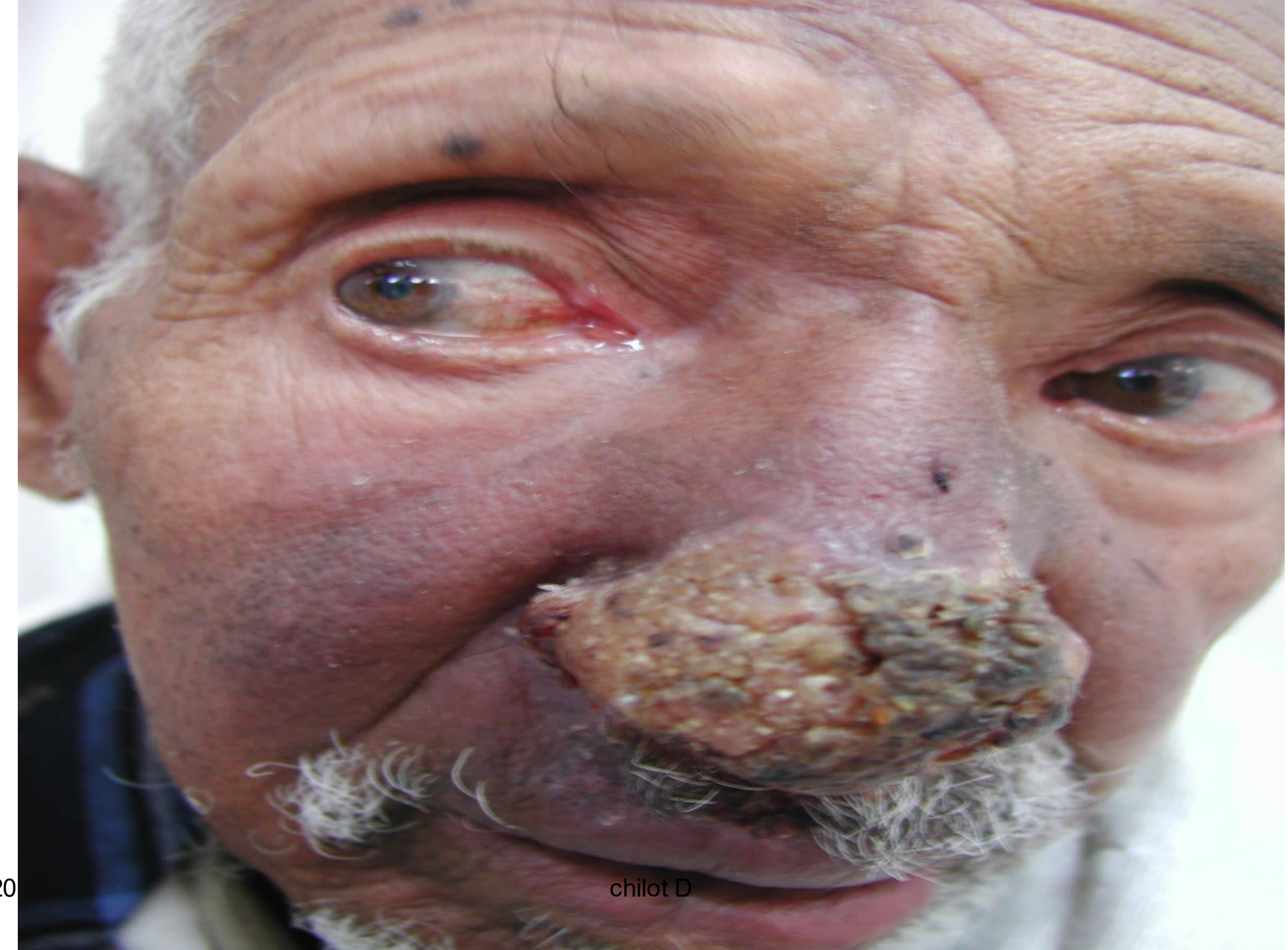
- The first symptoms of mucocutaneous leishmaniasis are similar to those of cutaneous leishmaniasis but the parasites may spread to the mucosa in the oronasal and pharyngeal cavity.
- The soft tissues and cartilage in these areas are then progressively destroyed by ulcers and erosion
- Swelling of the lips and nose may produce a so-called "**tapir nose**".
- Mutilations are severe and occasionally result in death due to malnutrition and bronchopneumonia.

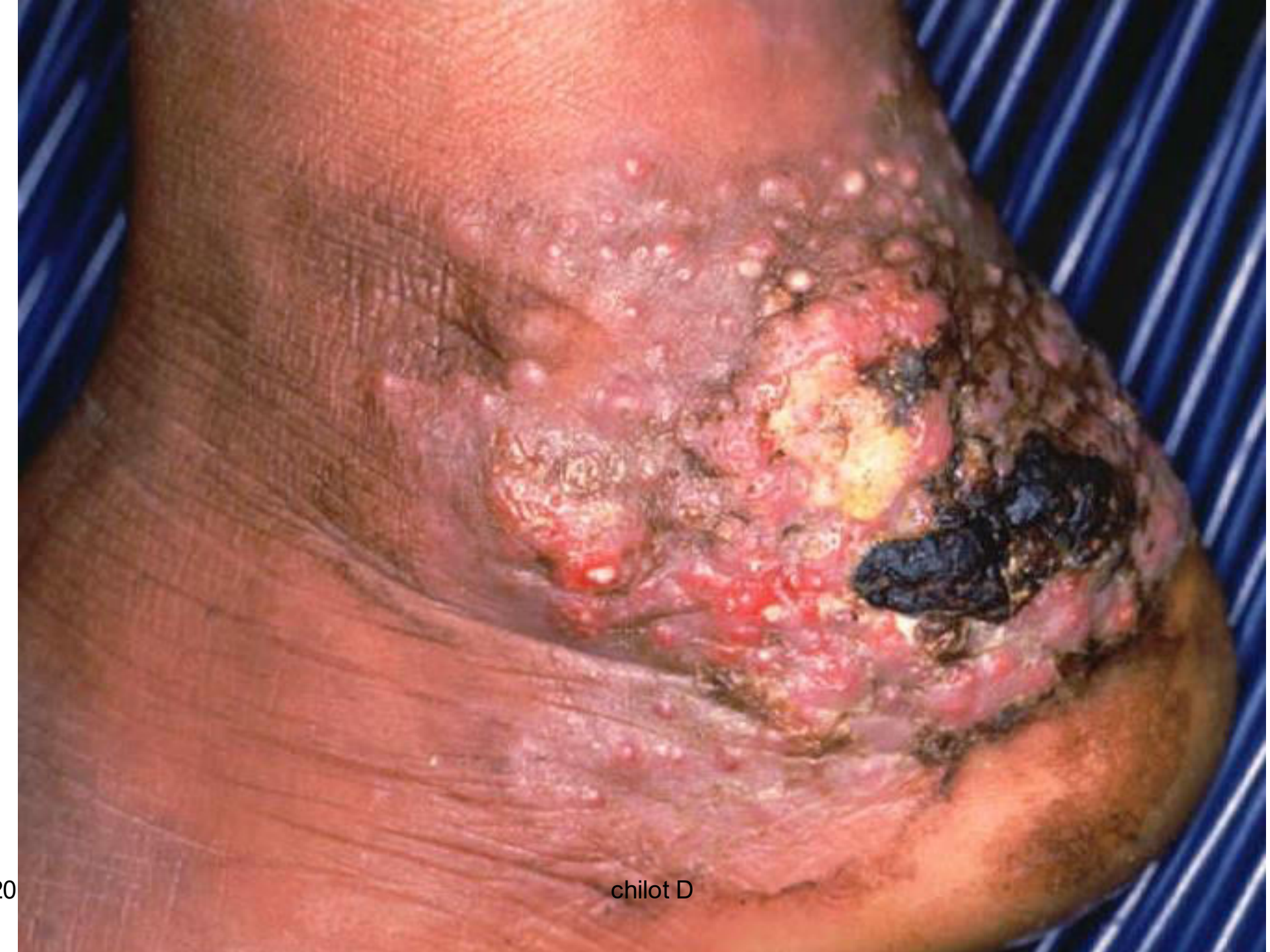


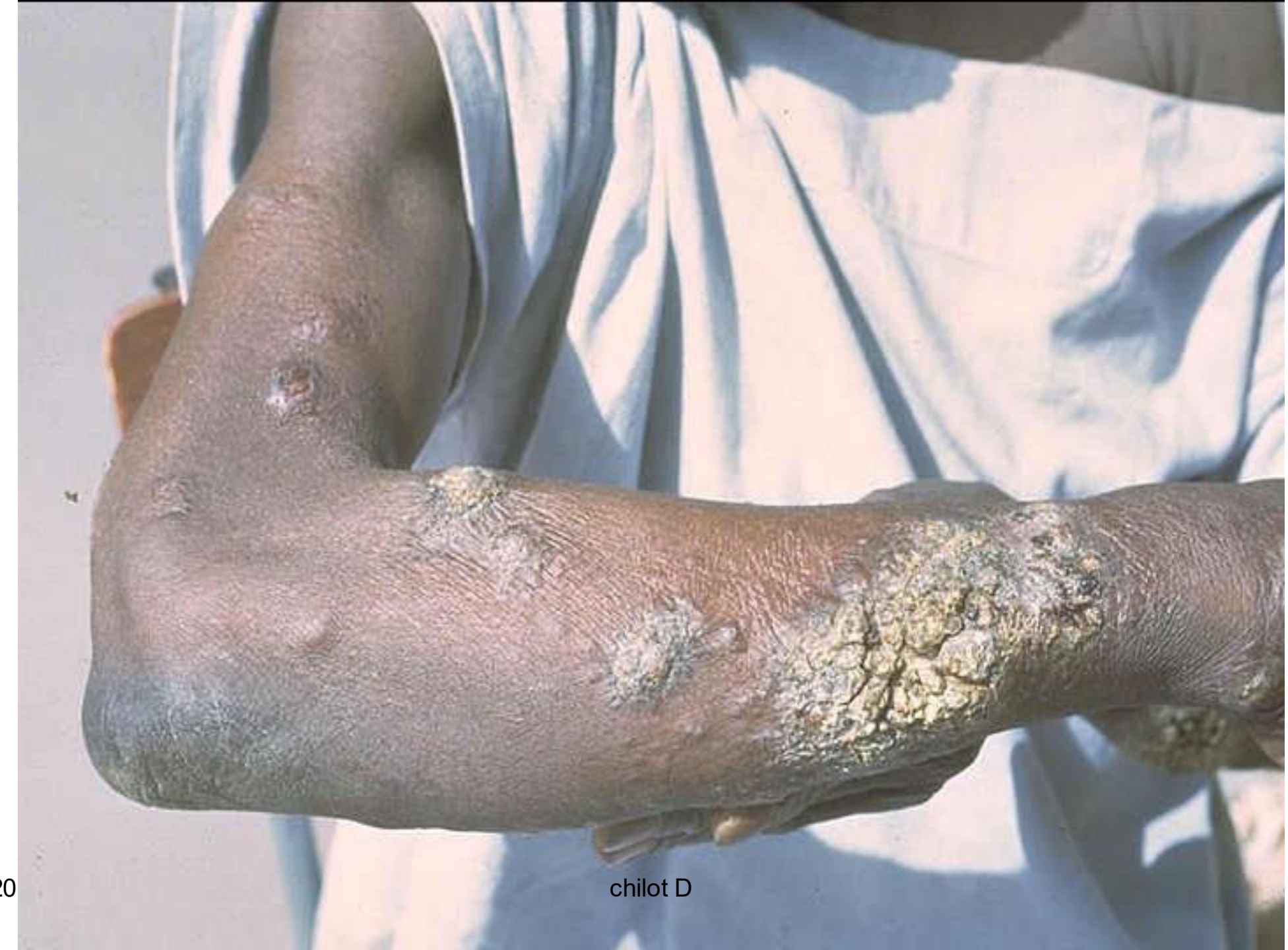


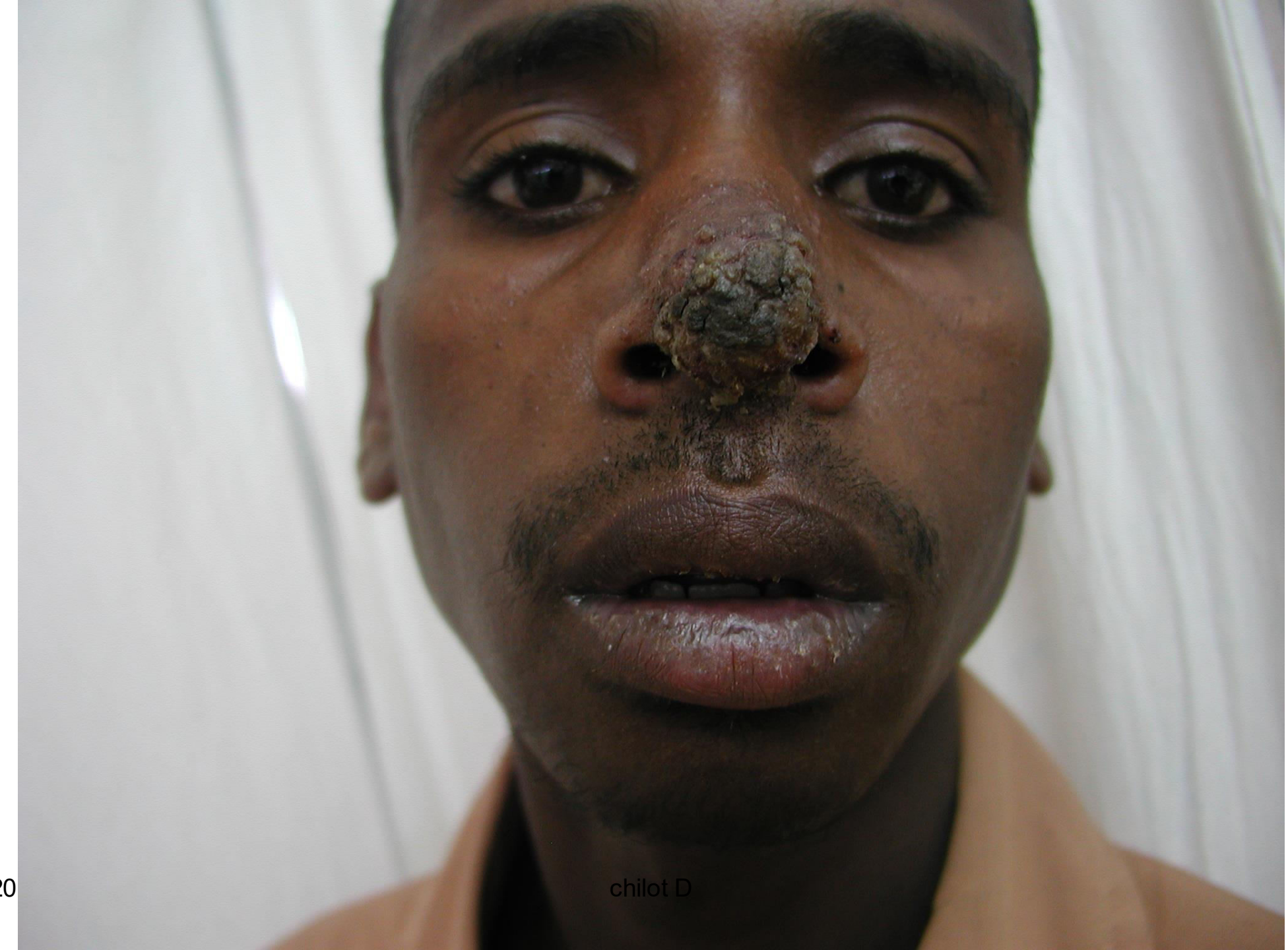


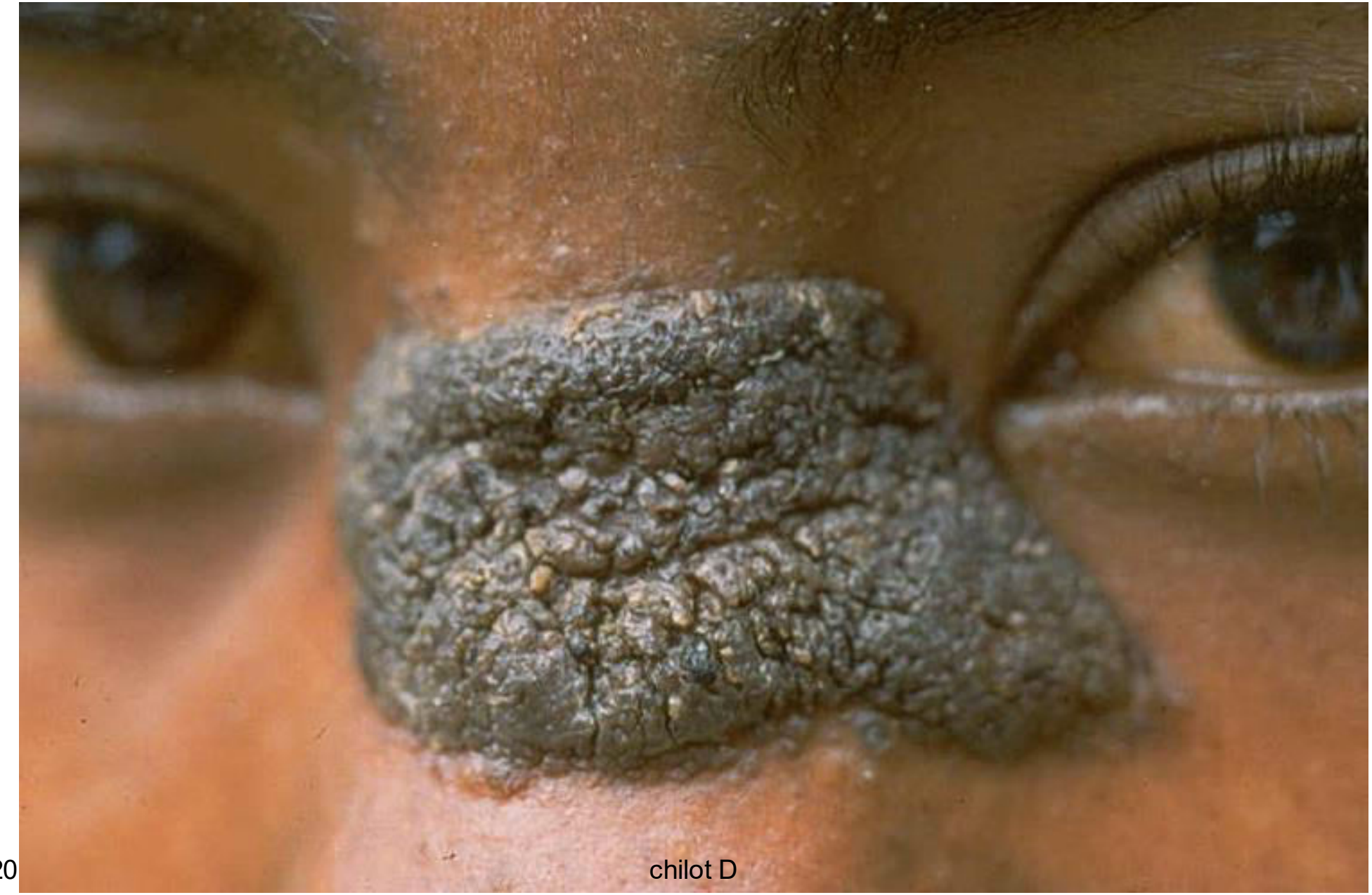


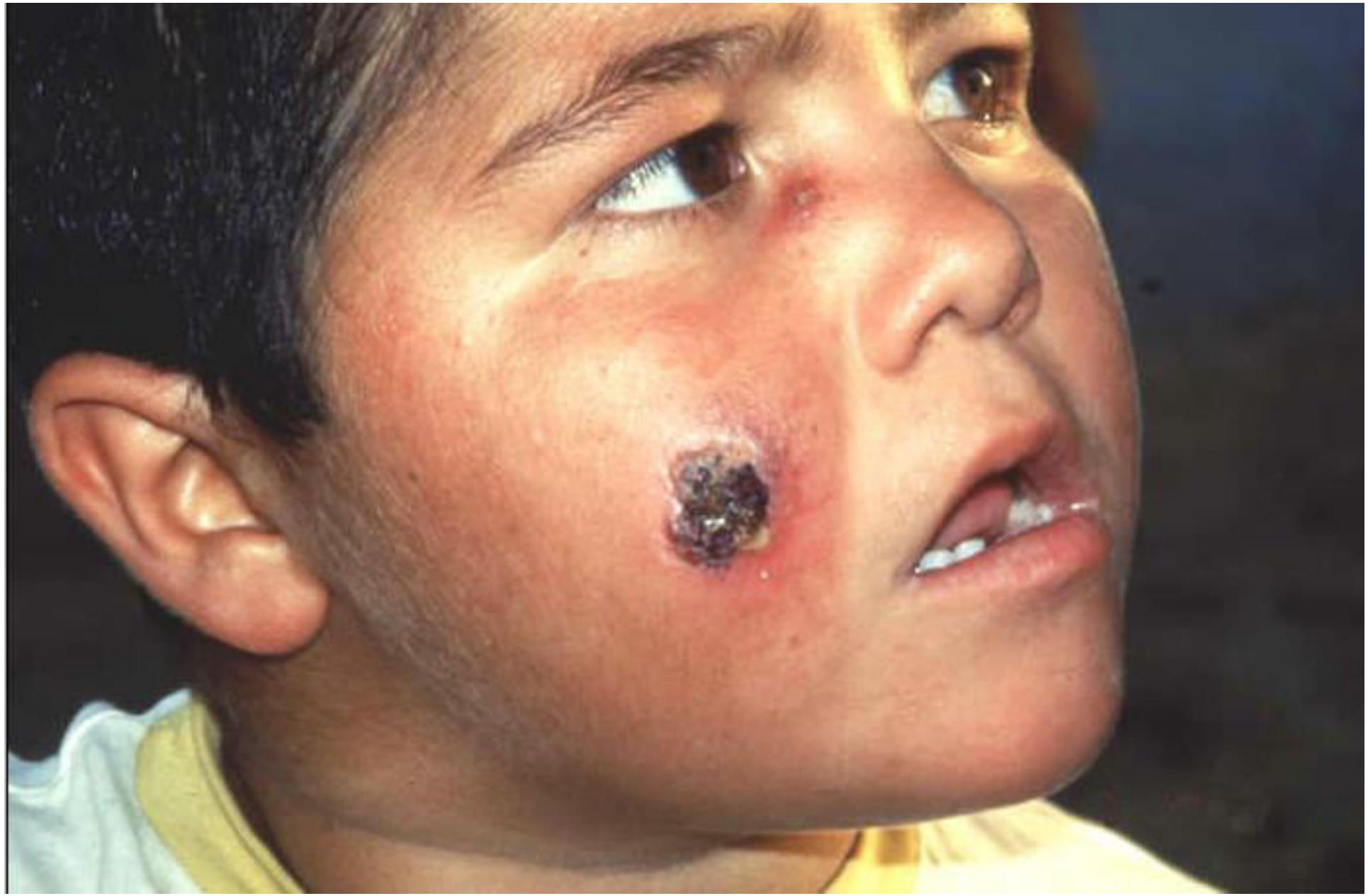


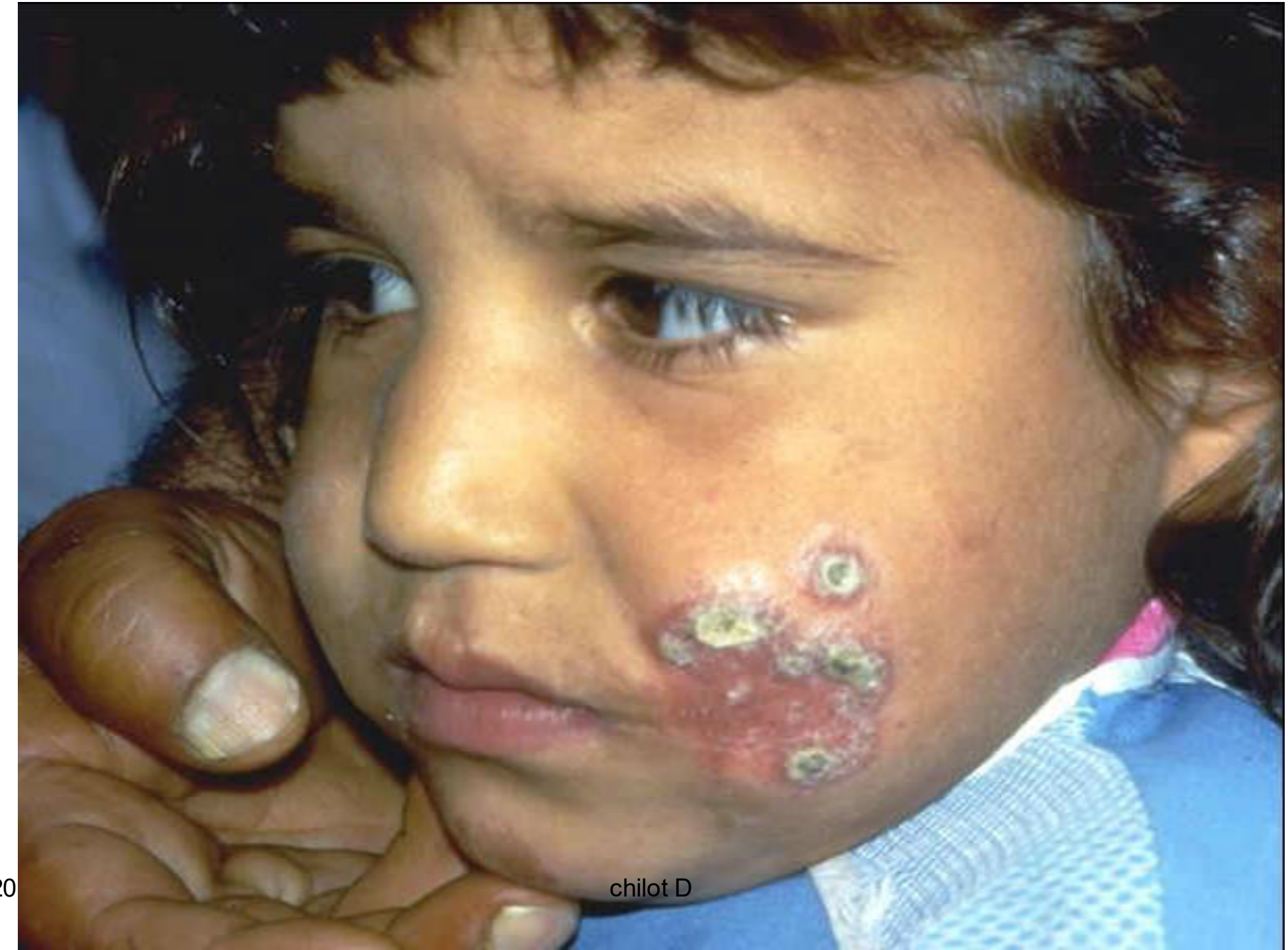


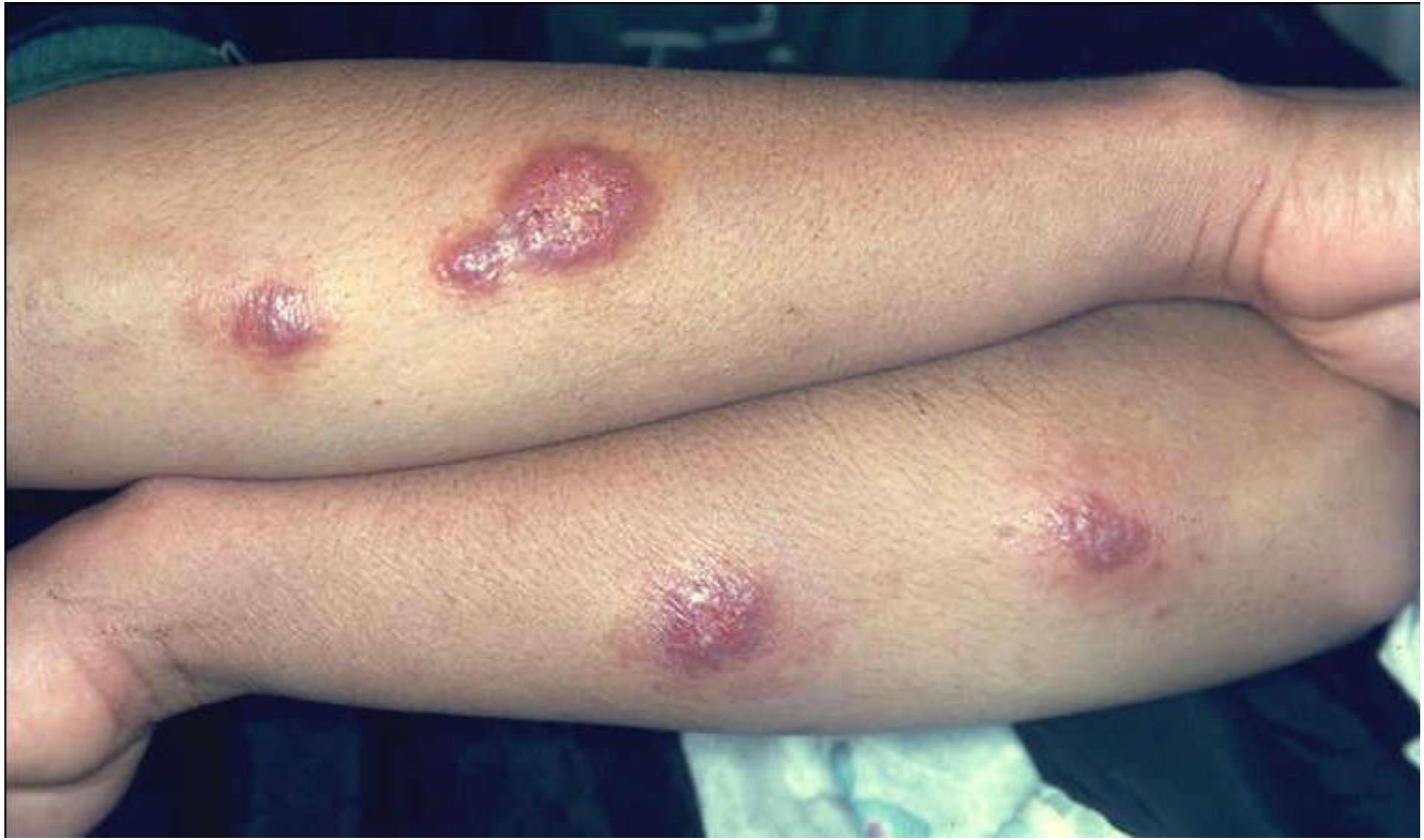


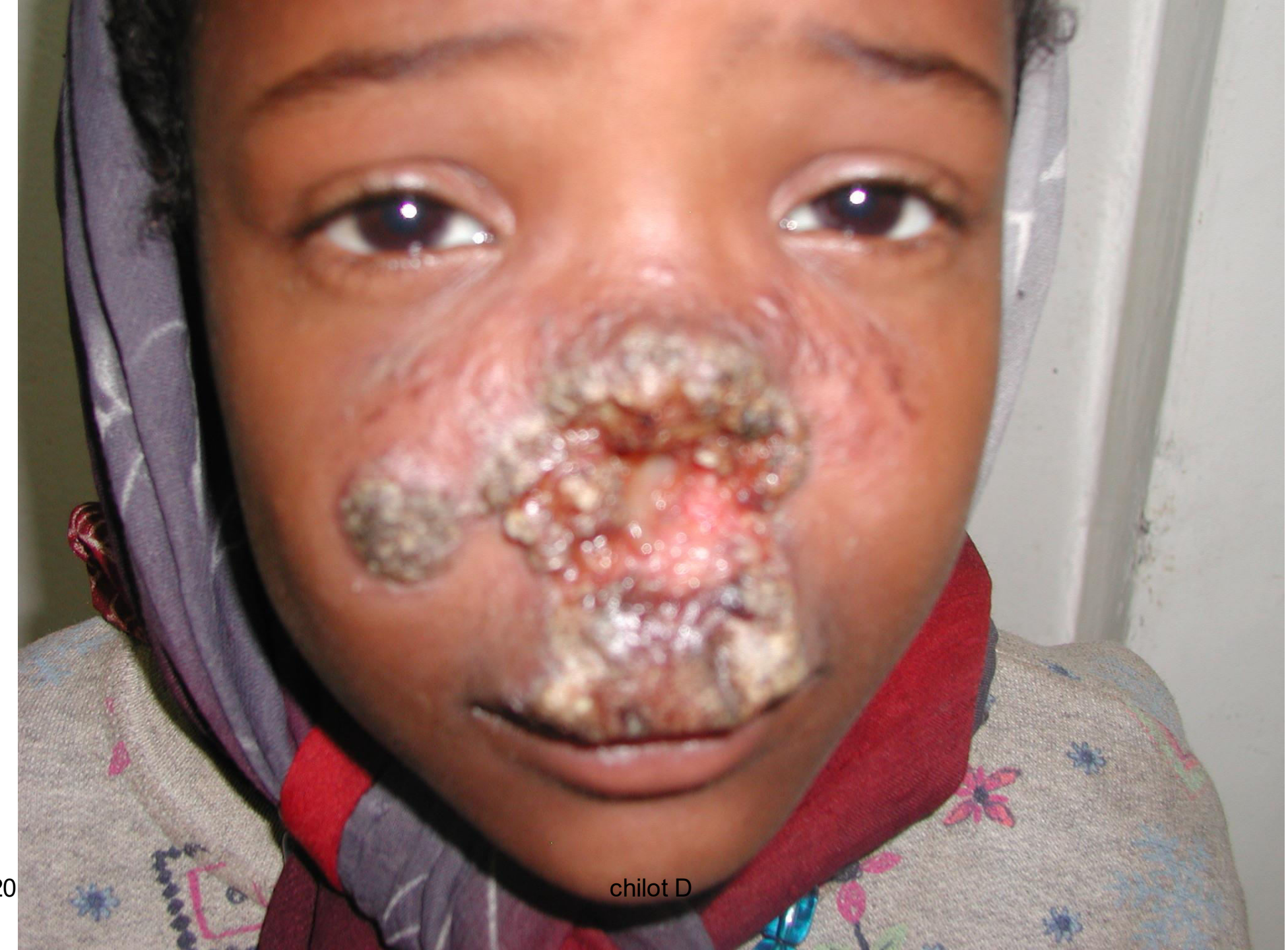


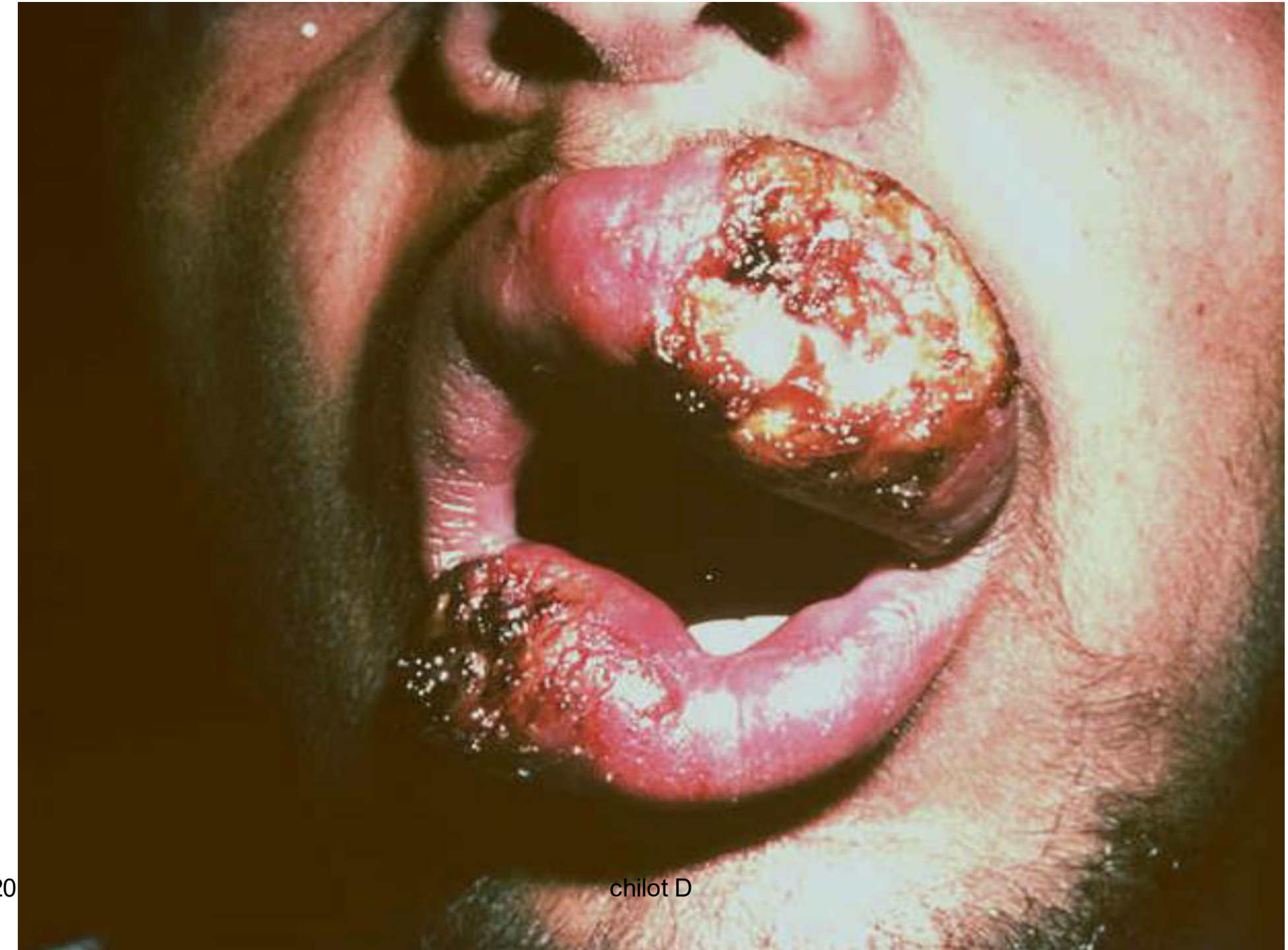


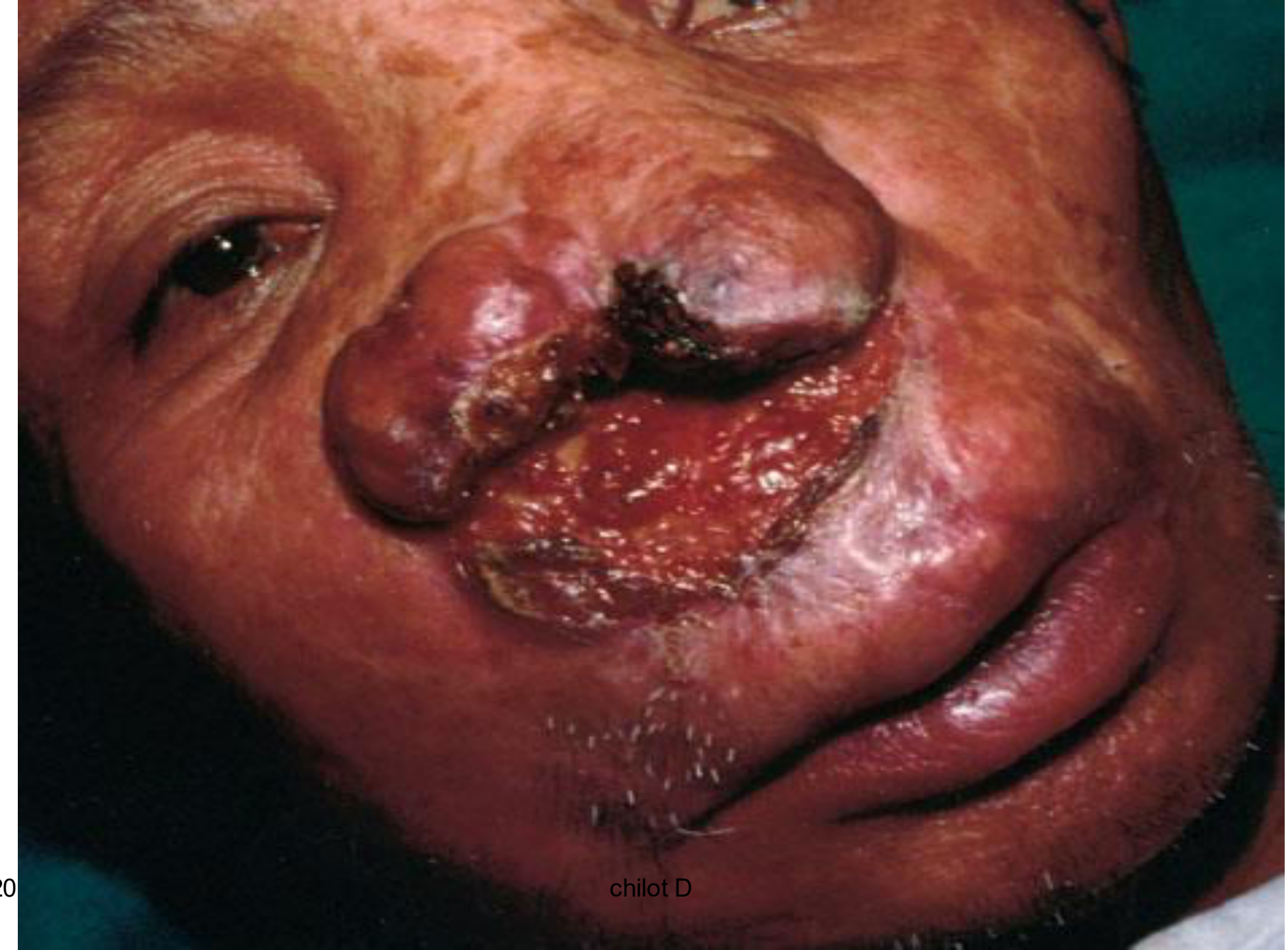


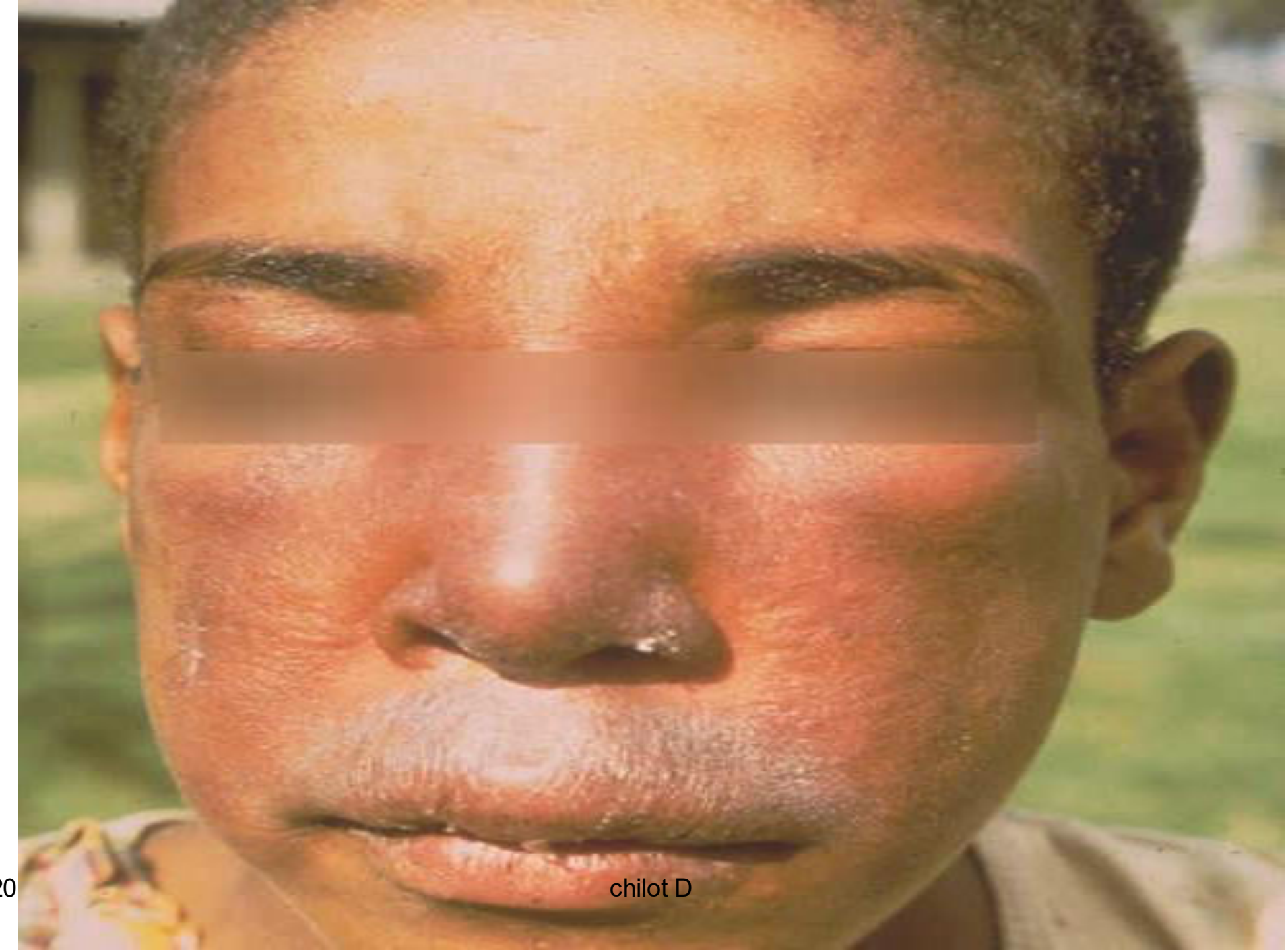


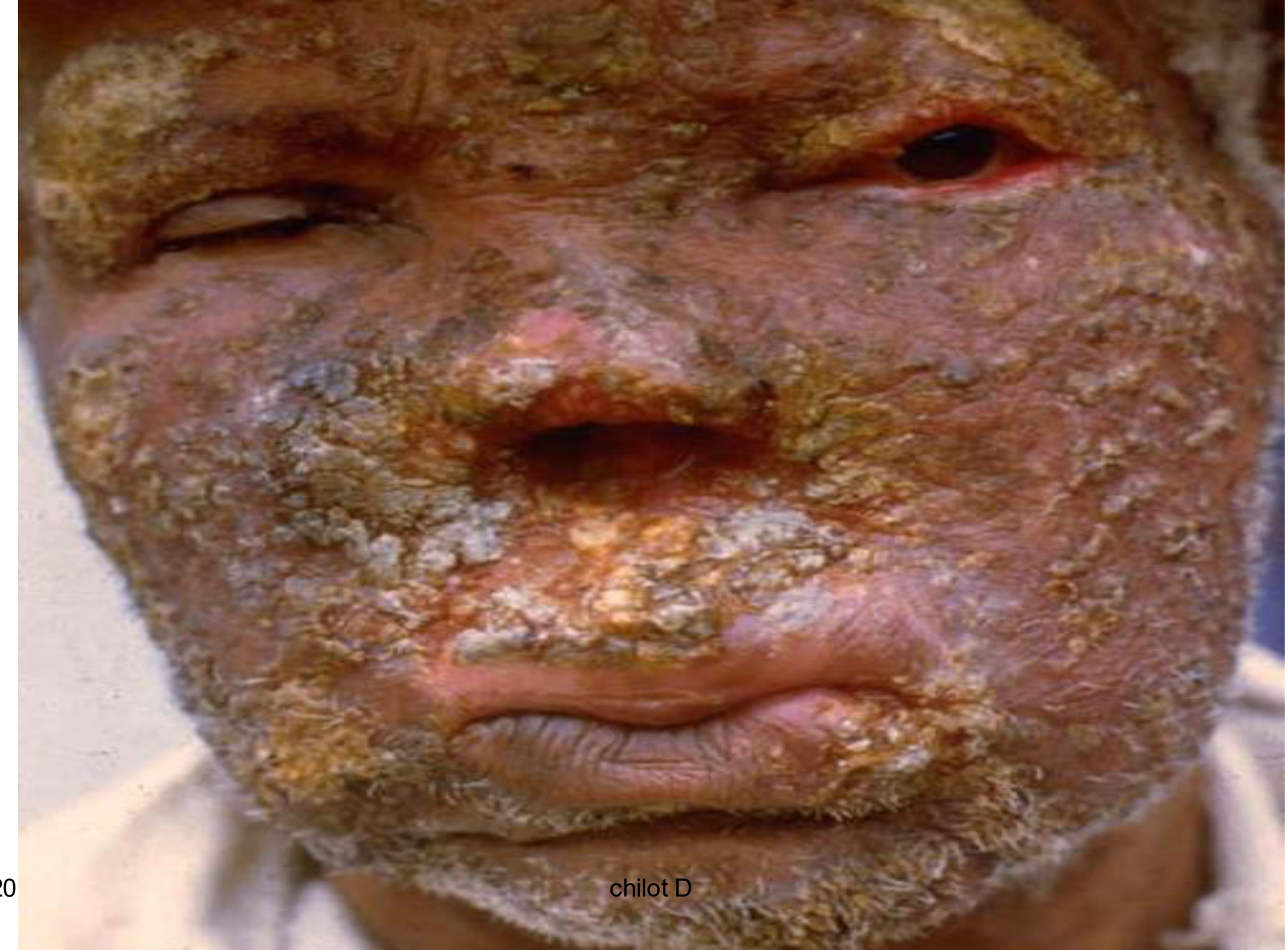


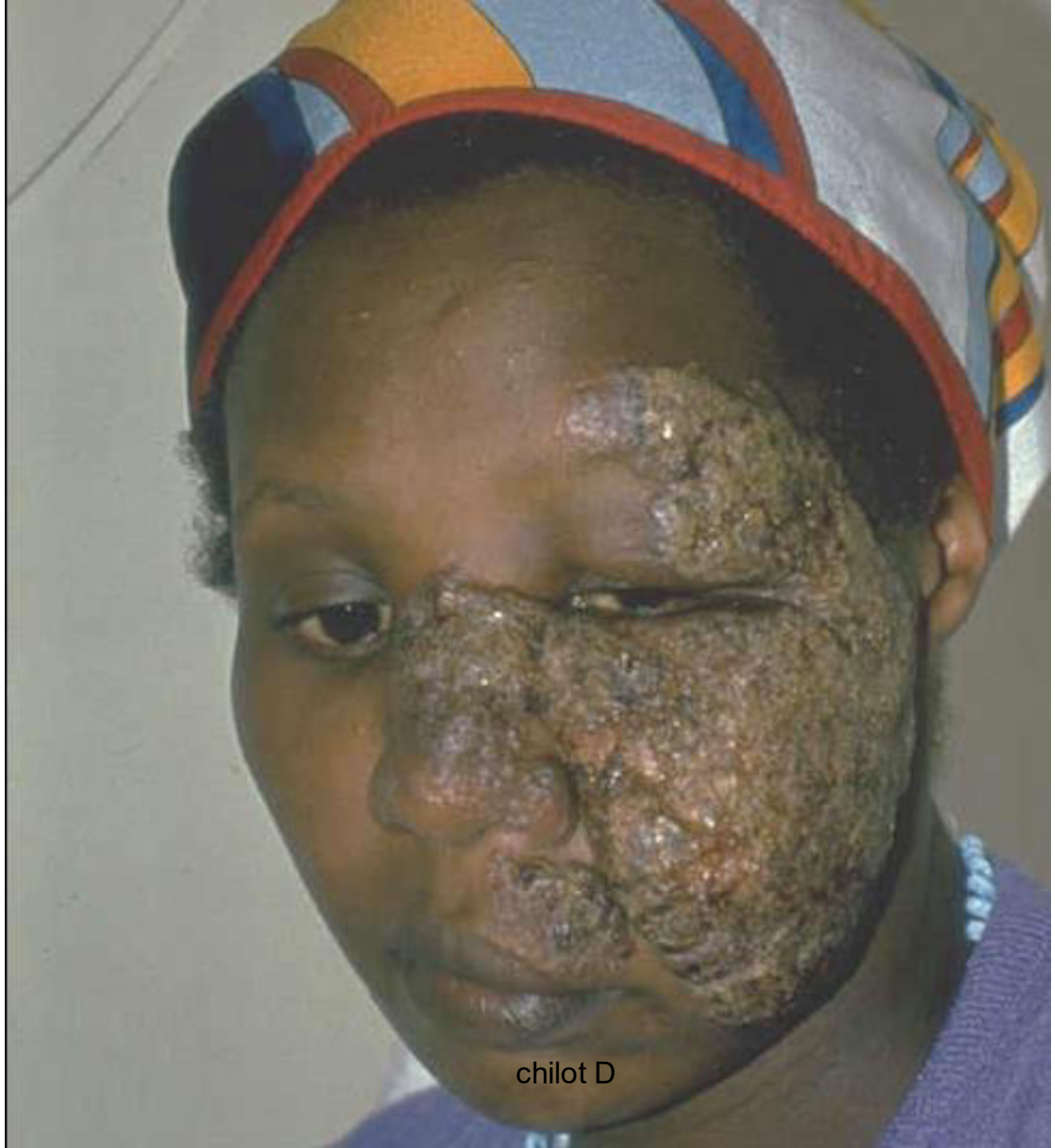


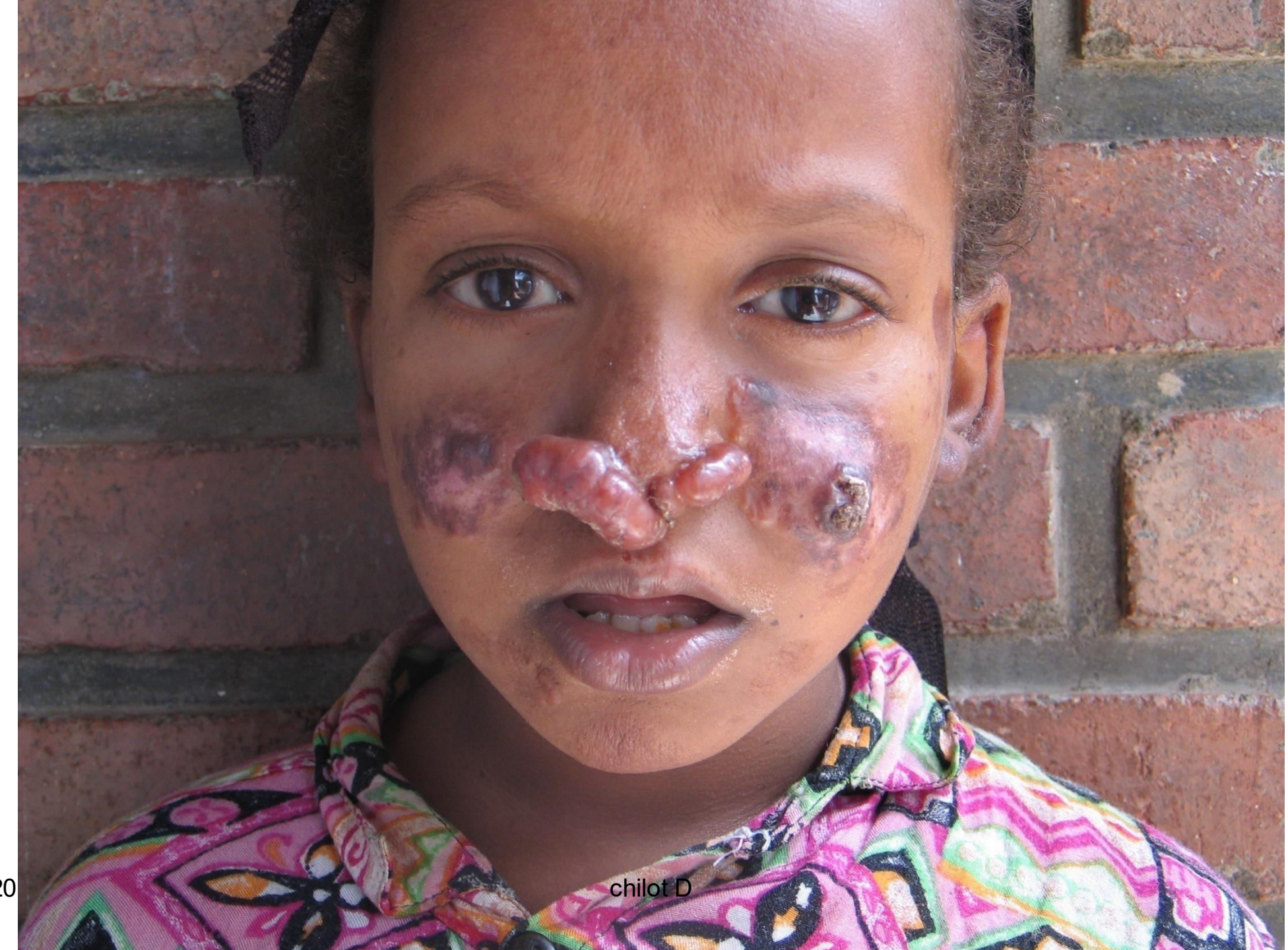


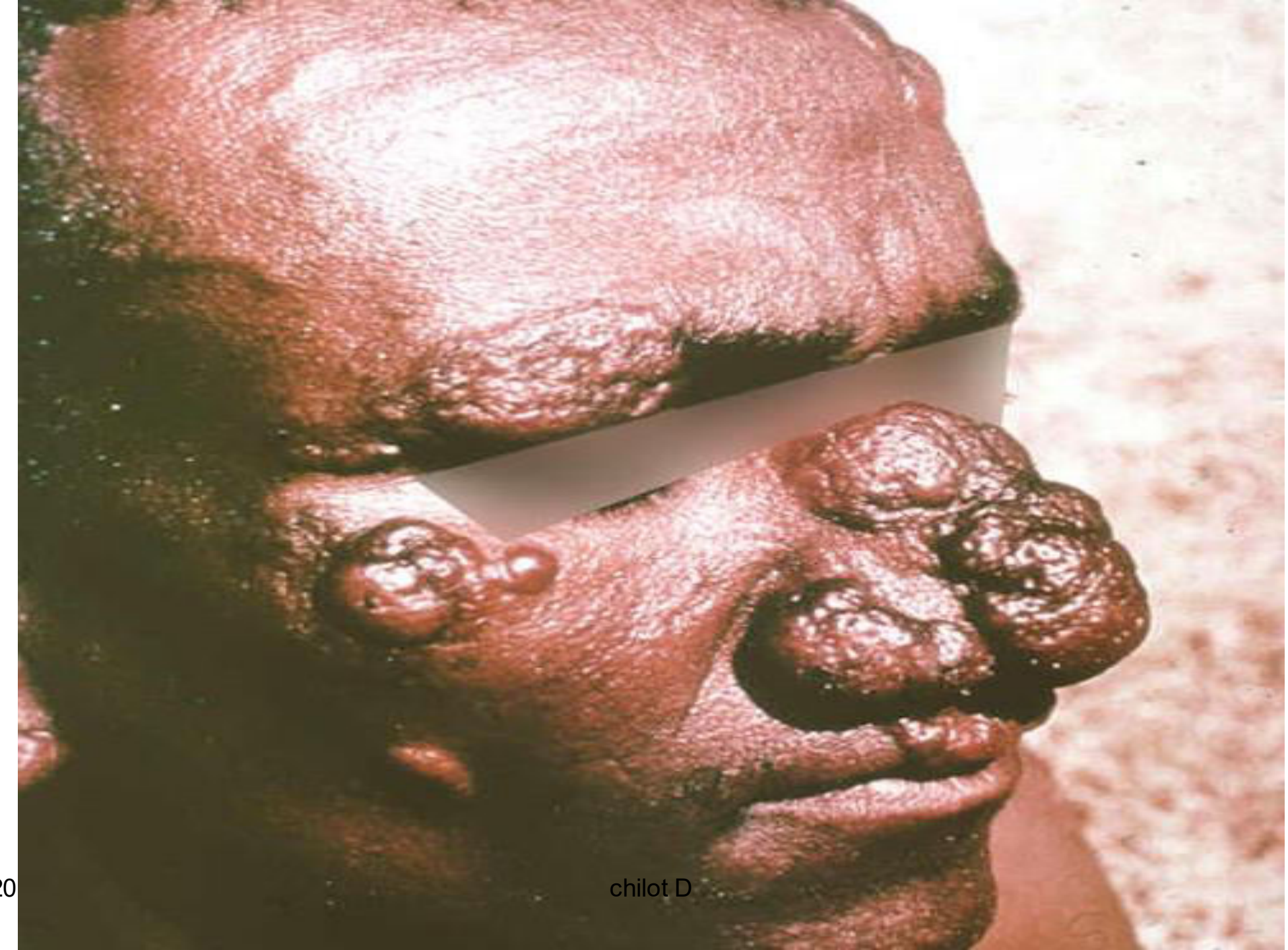


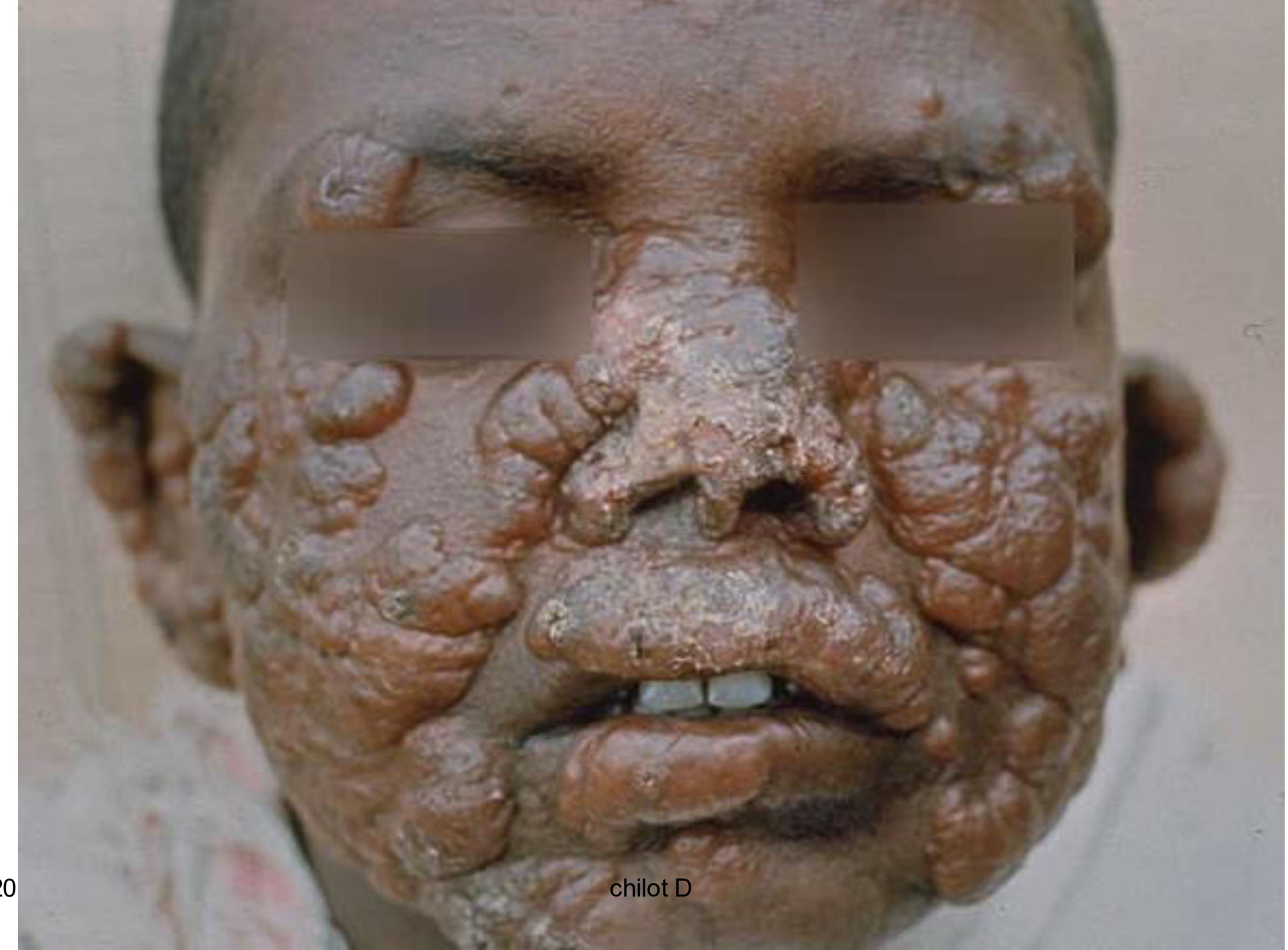


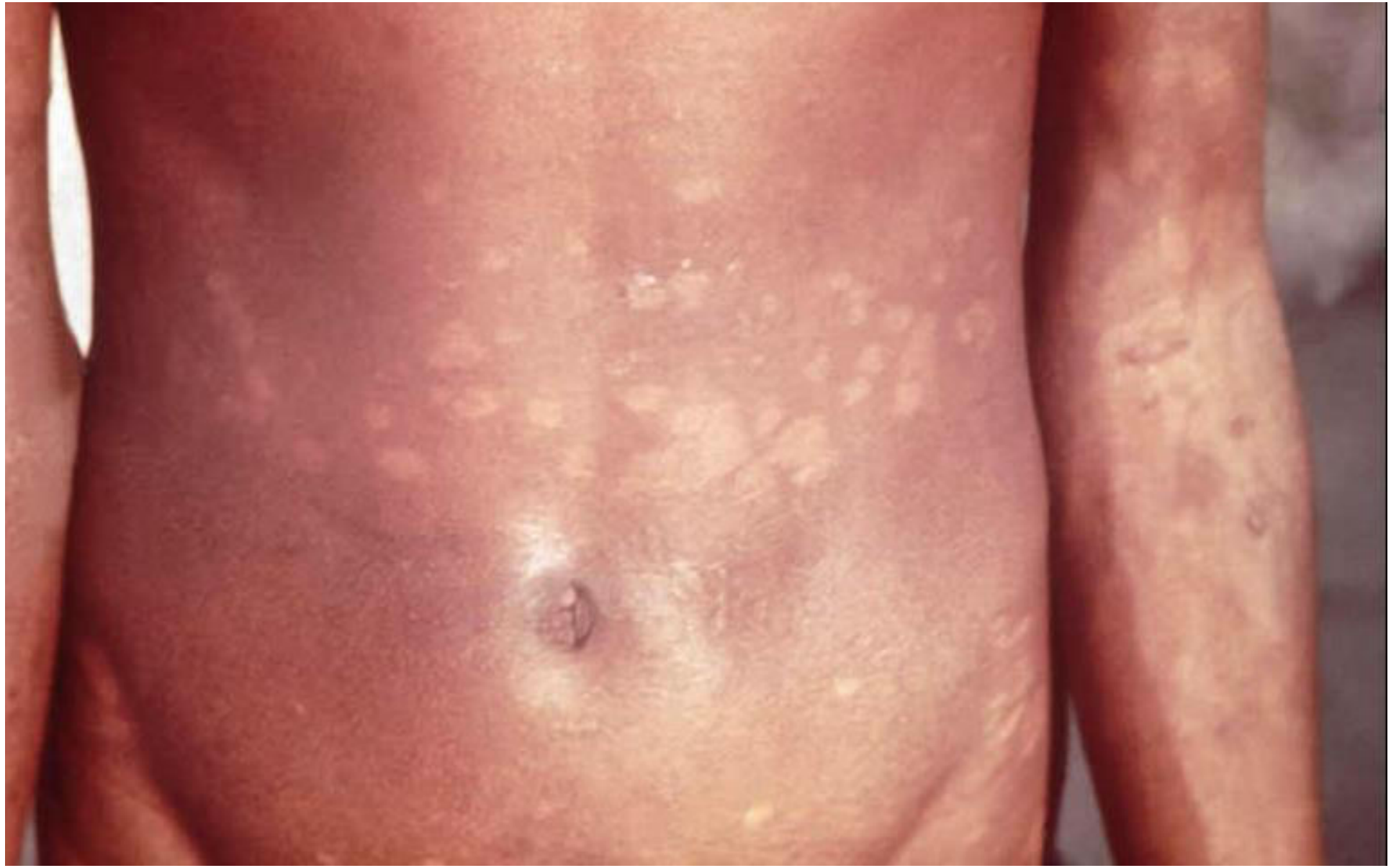


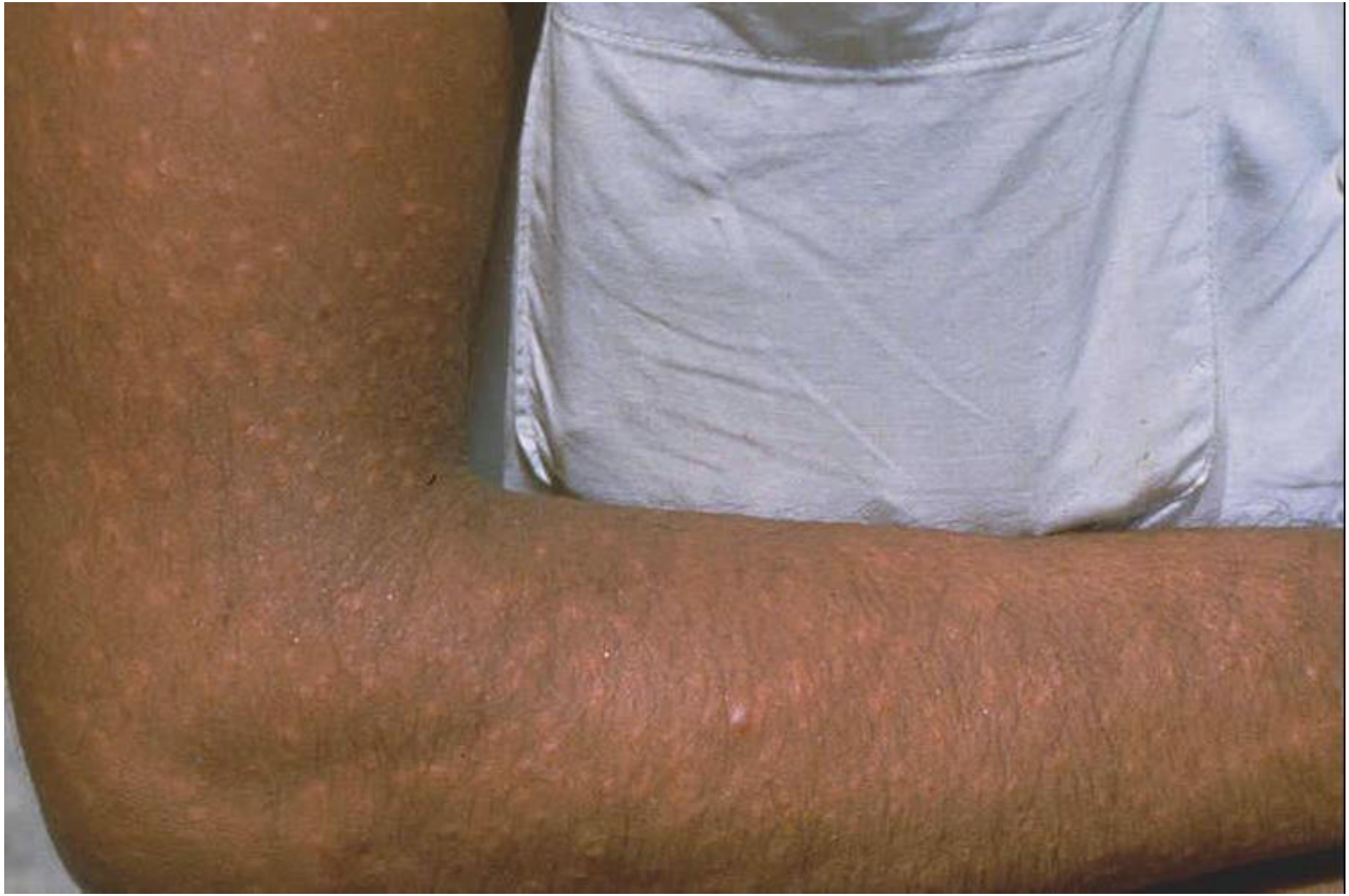


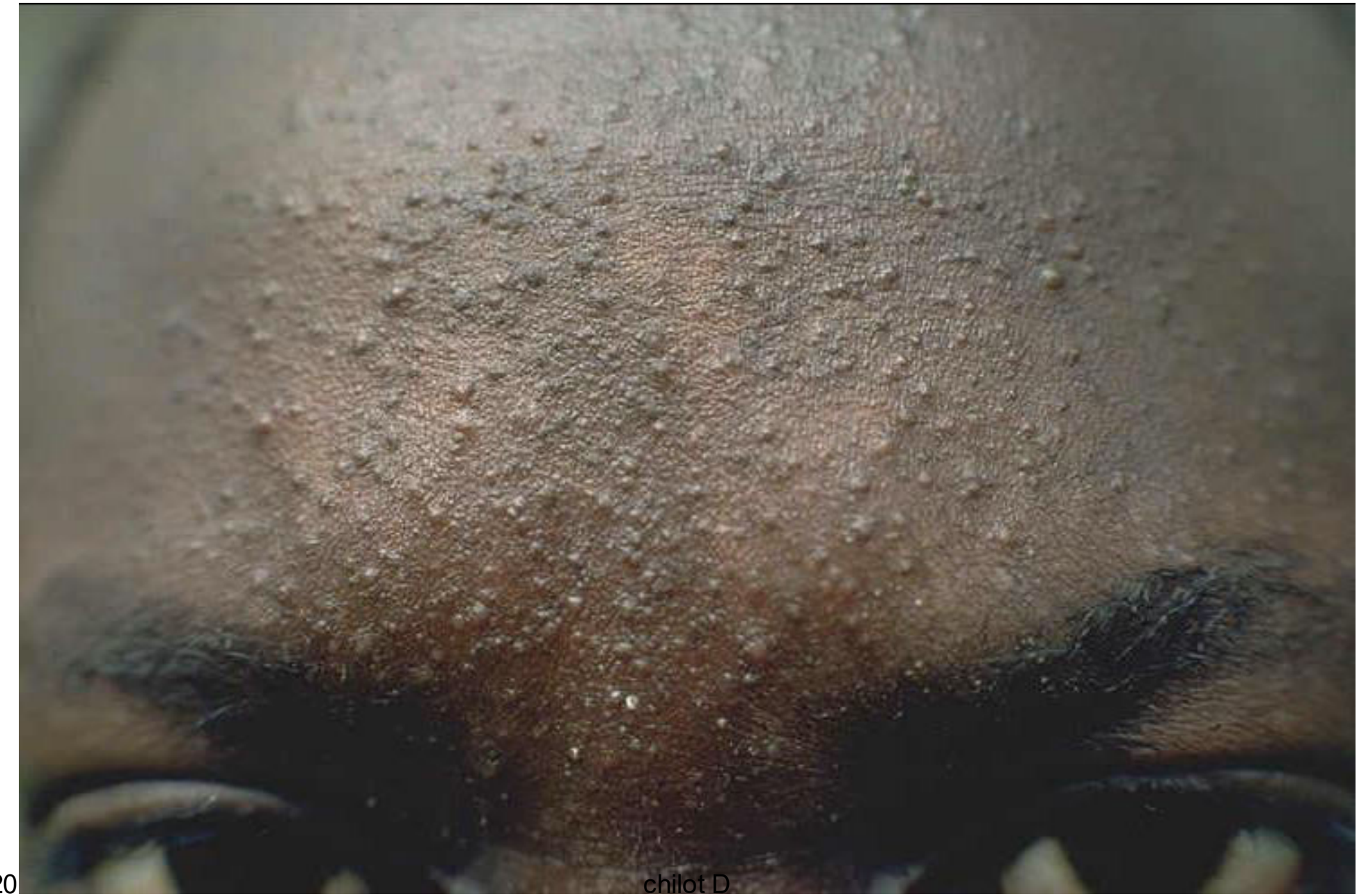


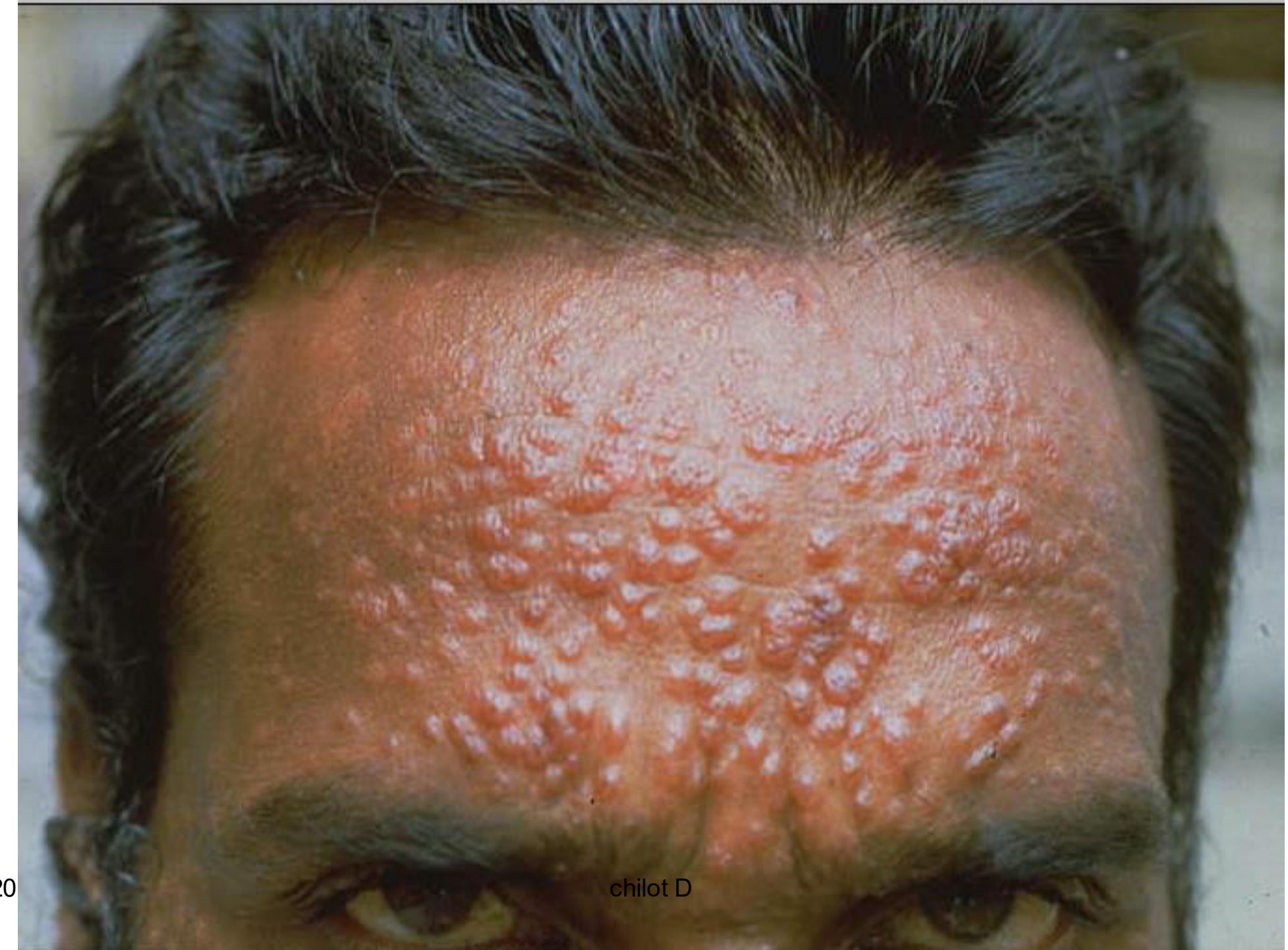


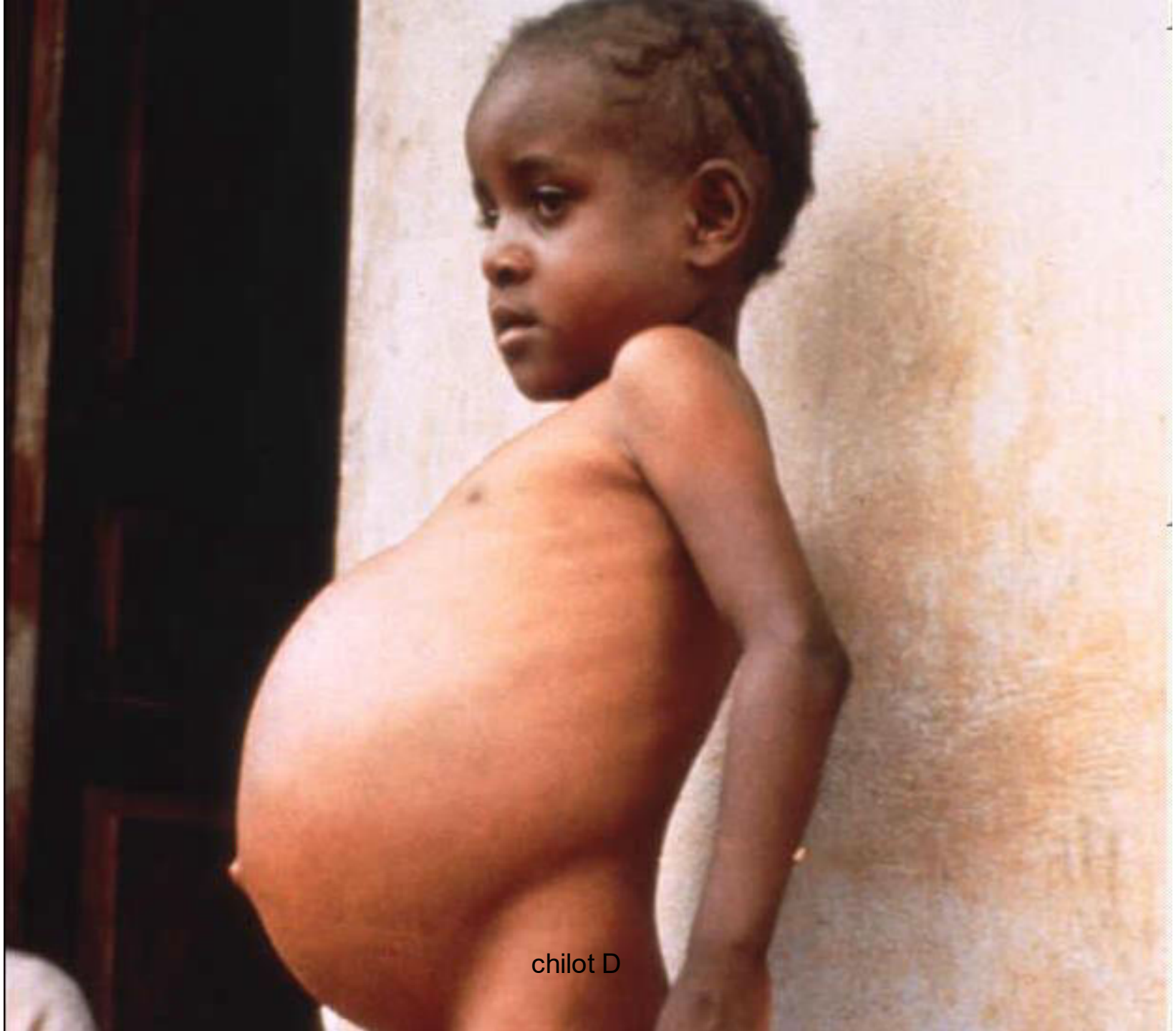












Diagnosis

- Demonstration of the parasite from:
  - ➢ Peripheral blood
    - ✓Using direct blood smear amastigotes are present in circulating monocytes and less often in neutrophiles, but the numbers are so scanty that smear may not show them
  - ➢ Bone marrow
    - ✓It is the most common diagnostic specimen.
    - ✓The sternal marrow is aspirated by puncturing the sternum at the level of 2nd or 3rd intercostals space, using a sternal puncture needle
  - ➢ Splenic aspirate
    - ✓It is richer in parasites and so more valuable for diagnosis.
    - ✓But the procedure can sometimes cause dangerous bleeding so should be done carefully
- By culture of the motile promastigote using **NNN** /Novy-McNeal-Nicolle/ medium
- Serologic test to demonstrate antibodies or antigens

Management

- Supportive care includes treatment of concomitant infections and blood transfusions
- Simple cutaneous leishmaniasis usually heals without treatment and renders the person immune to other infections with the same parasite species

Management---

For visceral leishmaniasis

First line

- **Sodium stibogluconate**: 20mg/kg/d iv or im in a single dose for 28 days. /40-60 days in patients with relapse or incomplete response/

Alternative

- **Amphotericin B**: 0.25 to 1 mg/kg slow infusion QD or QOD or three times a week for up to 8 wks depending on the response
- **Pentamidine**: 3-4mg/kg/ im QD or QOD for up to 15 days

For cutaneous leishmaniasis

- **Sodium stibogluconate**: im or iv QD for 10 days

Prevention and control

- The avoidance of outdoor activities
- The use of mechanical barriers such as screens and bed nets
- Wearing of protective clothing
- Application of insect repellent
- Treatment of cases
- Vector control and elimination of reservoir host (e.g. domestic dogs).

AFRICAN TRYPANOSOMIASIS /SLEEPING SICKNESS/

- It is a systemic disease caused by protozoa characterized by fever followed by general weakness and cerebral involvement leading to death.

Infectious agent

- The commonest agents are:
  - T.Brucei rhodesiense
  - T.Brucei gambiense
- Other species which is less important is T.Cruzi- causes American Trypanosoniasis

Occurrence and importance

T. rhodesiense

- Cause the more severe trypanosomiasis **without sleeping sickness.**
- Occurs mainly in East Africa
- It causes disease in humans and in cattle

T. gambiense

- Causes sleeping sickness in West And Central Africa.
- No animal reservoir known

❖ In Ethiopia, the distribution of trypanosomiasis is mostly found in Jinca, Afar, Setit Humera, Konso, Moyale and Dilla.

Reservoir and mode of transmission

Reservoir

- For T.brucei gambiense it is only humans
- For T. brucie rhodesiense the reservoir is dog, cattle, fox, wolf and human beings.

Mode of transmission

- By the bite of infective glossina **tsetse fly** during blood meal.
- Congenital transmission can occur in humans.

Incubation period

- T. brucei rhodensiense: 3 days to few weeks.
- T. brucei gambiense: several months up to one year.

TSETSE FLY

Life cycle of T.brucei

- Elongated trypomastigote form in midgut of tsetse fly→epimastigote in salivary gland →metacyclic trypomastigote, the infective form for vertebrates

Pathology, clinical feature and diagnosis of trypanosomiasis

Time after bite	Parasites found	Pathology	Clinical features	Diagnosis
1-3 weeks local stage	Only at place of bite	Trypanosome chancre	Local swelling painless; spontaneous recovery	• Examination of aspirated fluid from boil
1-2 months fever stage	In blood and lymph	Parasitaemia	Irregular fever;headache; General weakness	• Thick blood film • Aspiration of lymph gland fluid
From 4 months	In organs: Heart	Pancarditis	Quick irregular pulse Oedem, CHF	• Repeated examination of fresh blood smear
	Bonemarrow	Anemia	Headache, Anaemia signs CHF	• Bone marrow biopsy
	Lymphoid tissues		Lymphadenopathy Splenomegaly	• Lymph gland fluid
	Kidney	Glomerulonehritis	Oedema, Proteinuria	
	Brain	Meningitis and Encephalitis	Severe head ache, Apathy Dizziness, Difficulties in walking, speaking Tremors of fingers, tongue, Day time somnolence	CSF: raised protein & cell count

A patient with African trypanosomiasis

Fig. 2.7
African sleeping sickness usually starts with headaches, irregular fevers, swollen tissues and joint pains. At a later stage the brain becomes affected, which results in mental deterioration, coma (the sleeping stage) and death.

Treatment

In the early stage of the disease, where the CNS is not involved:

- Suramin sodium is used for rhodesiense infections
- Pentamidine is used for gambiense infections

In the late stage of the disease, where the CNS is involved:

- The chance of achieving cure is diminished
- Melarsoprol is used for both gambiense and rhodesiense

Prevention and control

- Public education on personal measures to protect against insect bite.
- Eradication of vectors
- Drug treatment of infected humans
- Avoiding areas to be known by harboring infected insects
- By wearing protective cloth and by using insect repellents
- Reducing tests fly number by
  - Identifying and studying the breeding habits of local vector
  - Selectively clearing the bush and wooden areas especially around game reservoirs, water holes, bridges and along rivers bank
  - Using and maintaining insecticide impregnated tsetse fly traps.
- Spraying vehicles with insecticide as they enter and leave tsetse fly infested areas
- Prohibit blood donation from those who have visited or lived in endemic areas.

SEXUALLY TRANSMITTED INFECTIONS

Learning objectives

At the end of this unit the learners should be able to:

- list the common sexually transmitted infections
- identify the diagnostic symptoms of sexually transmitted diseases
- Introduced about syndromic management of sexually transmitted infections
- state the preventive and control measures for sexually transmitted infections

Introduction

- The diseases belonging to this group are usually transmitted during **sexual intercourse**
- During sexual intercourse there is close body contact, which is ideal situation for transmission
- STIs are very **common in adults**, but they are often hidden for fear of the opinion of others
- A person who has **unsafe sex** is at risk of several sexually transmitted diseases

STIs included in this unit

- Syphilis
- Chancroid
- Herpes genitalis
- Gonorrhea
- Trichomoniasis
- Chlamydia
- Lymphogranuloma venurum /LGV/

The transmission of STIs

- The most common mode of transmission is **unprotected sex**
- Other forms of transmission include:
  - ❖Mother to child:
    - ➢During pregnancy (HIV & Syphilis)
    - ➢At delivery (Gonorrhea ,Chlamydia &HIV)
    - ➢Through breast feeding (HIV)
  - ❖Unsafe (unsterile) use of needles or injections or contact with blood or blood products (syphilis , HIV & hepatitis)

Factors increasing transmission of STIs

Biological factors

- Age, young and old age in women are more susceptible
- Gender, women more easily infected than males
- Immune status

Behavioral factors

- Changing sexual partners frequently
- Having **more than one sexual partner**
- Having sex with ‘casual’ partners, sex-workers or their clients
- Use of alcohol or other drugs before or during sex

Socio-cultural factors

- In most cultures women have **very little decision** making power over sexual practices and choices, including use of condoms
- Women tend to be **economically dependent** on their male partners and are therefore more likely to tolerate men's risky behavior
- **Sexual violence** tends to be directed more towards women by men
- Women have difficulties to discuss with their male counterparts
- In some societies the **girl-child** tends to be married off to an **adult male** at a very young age, thus exposing the girl to infections

Socio-cultural factors…

- In some societies a permissive attitude is taken towards men allowing them to have **more than one sexual partner**
- Harmful traditional practices such as:
  - Skin-piercing
  - The use of un-sterile needles to give injections or tattoos
  - Scarification or body piercing
  - Circumcision using shared knives

Epidemiology of STIs

- STIs are major public health problems in all countries
- **Globally 340 million** new cases of curable STIs occur every year (69 million are in sub-Saharan Africa
- In many developing countries STIs are among the **top five disease**
- There is little information on the incidence & prevalence of STIs in Ethiopia
- Total of **451,686** cases of STIs were reported b/n June 1988 &June 2002 in Ethiopia

Epidemiology…

- Except for adult prevalence of HIV (2.1%) & syphilis (2.7%),there is no actual information or estimate on other STIs in Ethiopia
- Prevalence higher in urban than rural
- **Higher in unmarried & young adults** (15-44 yrs)
- More frequent among **females** than males between the ages of 14-19
- After the age of 19, there is slight male preponderance

SYPHILIS

It is a disease characterized by:

- A primary lesion **/Hard chancre/**
- A later secondary eruption on the skin & mucus membrane
- Then a long period of latency, finally
- Late lesions of skin, bones, viscera , CNS & CVS /tertiary syphilis/

Syphilis…

Infectious agent

- Treponema pallidum, a spirochete

Occurrence

- World wide
- Primarily involves **sexually active** young people between 20-29 yrs
- More common in urban than rural areas

Syphilis…

Reservoir

- Humans

Mode of transmission

- Direct contact with lesion mainly during sexual intercourse
- Accidentally by touching infective tissues
- Blood transfusion
- Congenitally

Incubation period

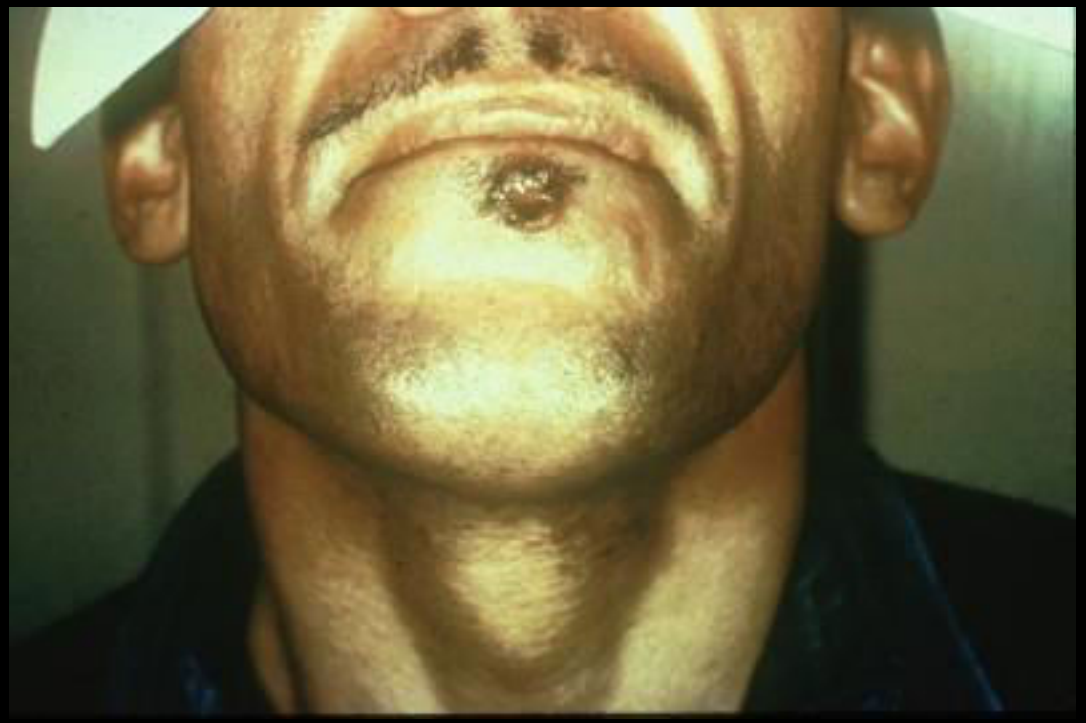
- 10 days to 3 months, usually 3 weeks

Clinical manifestations of acquired syphilis

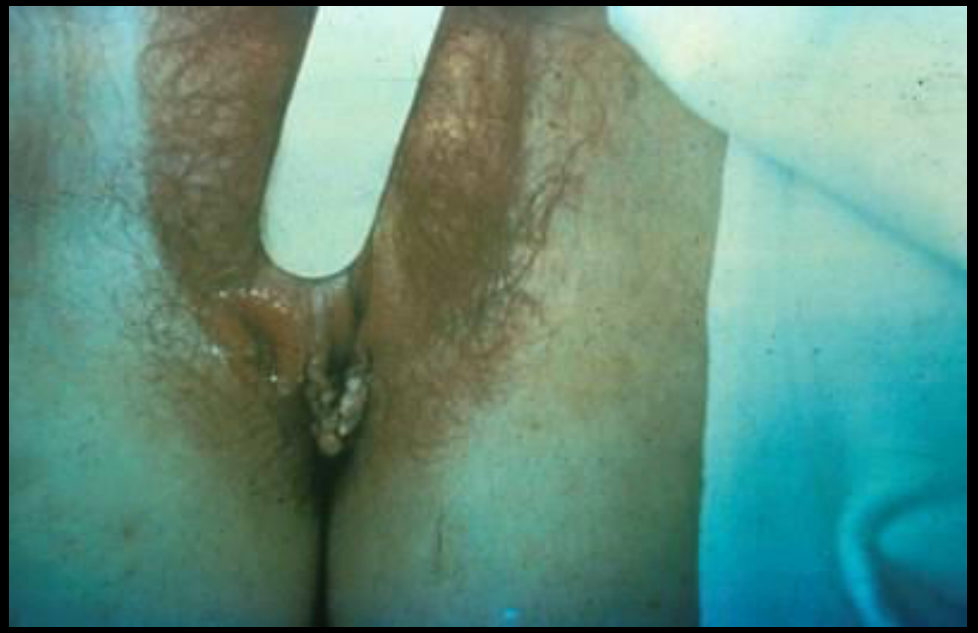
- Divided in to :
  - Primary syphilis
  - Secondary syphilis
  - Latent syphilis
  - Tertiary syphilis

Primary syphilis /Hard chancre/

- Consists of **hard chancre** - primary lesion of syphilis that occurs at the site of entry of infection, together with **regional lymphadenitis**
- The hard chancre is a single, **painless ulcer** on the genitalia or elsewhere (lips, tongue, breasts) and heals spontaneously in a few weeks without treatment.
- Lymph glands are **bilaterally enlarged** & **not painful.**
- There will be **no suppuration** /pus/



Primary syphilis - chancre



Primary syphilis - chancre

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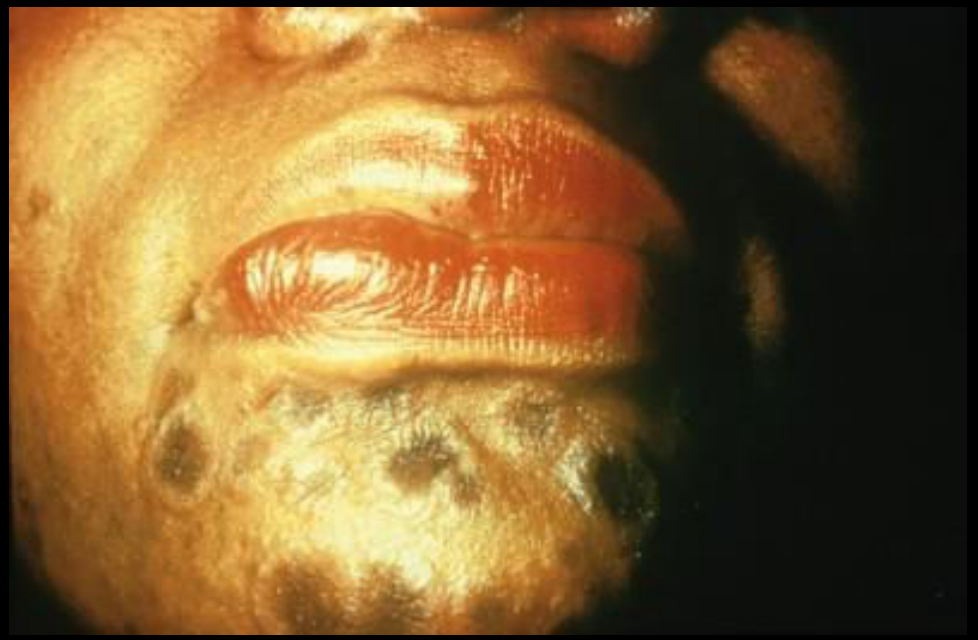
Secondary syphilis

- Disseminated **spirochetemia**
- After 4-8 weeks of the primary infection, generalized secondary eruption appears
- **Early rashes /skin lesions/ are**:
  - ➢ symmetrical
  - ➢ none itchy
  - ➢ highly infective & many spirochetes are demonstrated in them

Secondary syphilis…

- **Alopecia**; moth-eaten appearance
- Atypical facial plaques /patch/
- Mucosal ulcerations
- Condylomata lata /elevated wart like lesions/
- Painless generalized lymphadenopathy



Secondary syphilis

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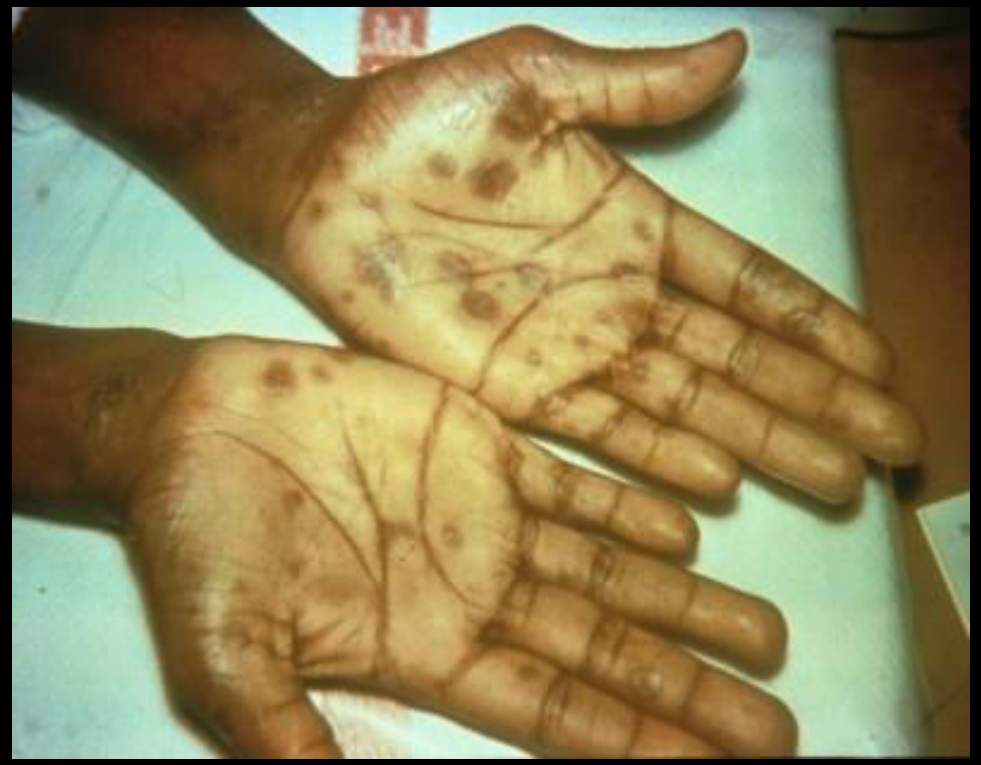


Secondary syphilis

Latent syphilis

- No clinical sign but syphilis serology is reactive
  - **Early latent** = infection less than one year
  - **Late latent** = infection occur for over one year

Tertiary syphilis

Gumma

- destructive, **non infectious** lesion of the skin, bones, viscera and mucosal surfaces

Cardiovascular

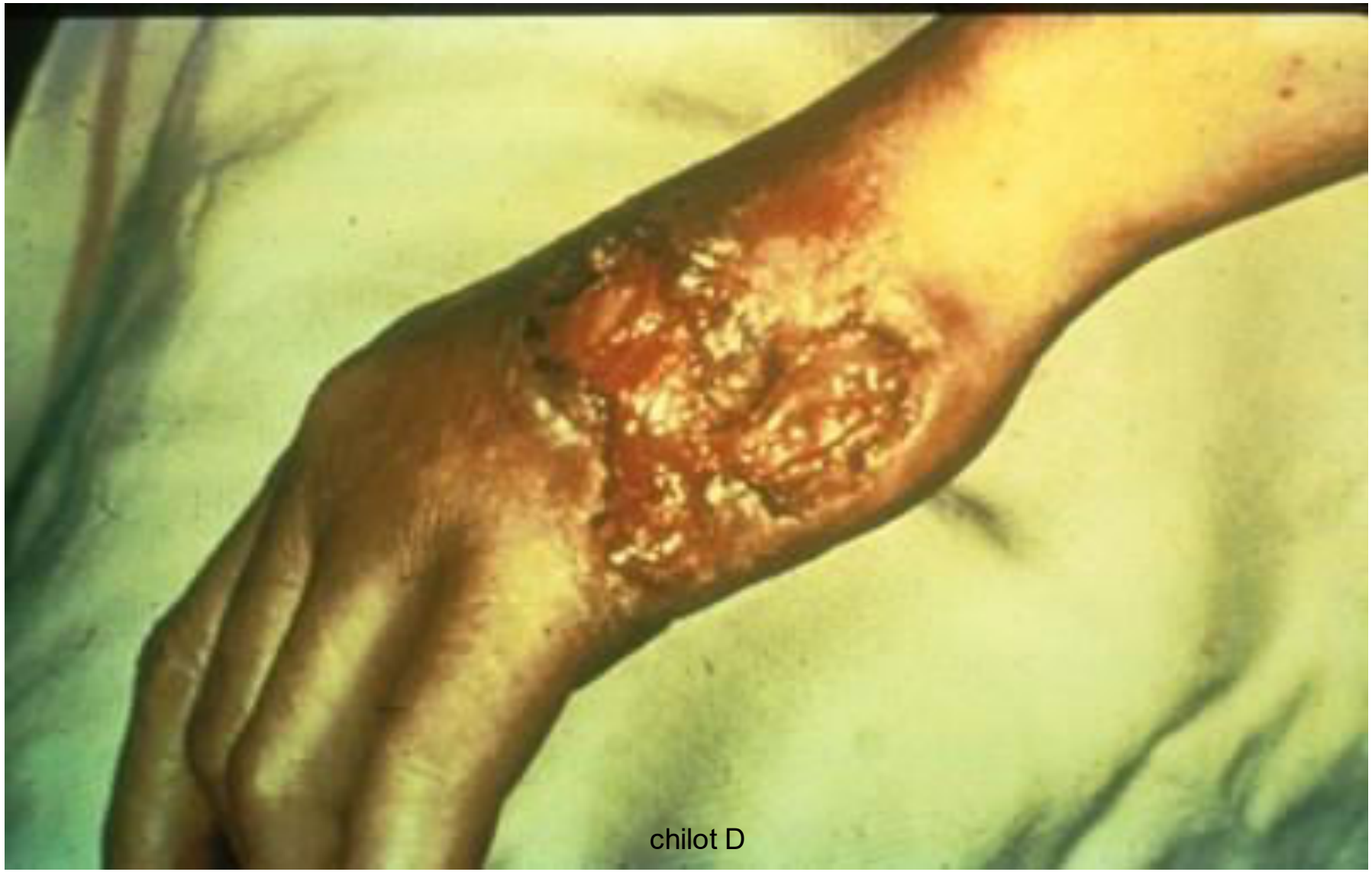
- aortitis leading to valve incompetence and aneurysm

Tertiary syphilis…

Neurosyphilis:

- Stroke like presentation (meningovascular)
- Asymptomatic but positive VDRL on CSF
- Tabes dorsalis/degeneration of the dorsal column of the spinal cord & sensory nerve trunk
- Paralysis of the insane/inability to be responsible/
- Cranial palsy (cranial nerve VIII, III, optic atrophy)

Ulcerating gumma



Syphilis in pregnancy

It can result in:

- Congenital abnormalities such as:
  - ➢deformity
  - ➢ blindness
  - ➢ deafness
  - ➢ paralysis
- Still birth
- Repeated spontaneous abortions

Diagnosis

- Clinical manifestation
- Serological tests based on antibodies formed (**VDRL test**)
- VDRL /RPR/ will be positive 6-8 weeks after infection
- Dark field microscopy of smears from primary lesion or from skin lesions in the early secondary stage will show the spirochetes

CHANCROID (SOFT CHANCRE)

- An acute bacterial infection localized in the **genital area**
- It is characterized clinically by single or multiple, **ragged painful**, **necrotizing ulcer** frequently accompanied by inflammatory swelling & **suppuration** of the regional lymph nodes (usually unilateral)
- Ulcer is with soft margins described as **soft chancre**

Infections agent

- Haemophilus ducreyi, the ducrey bacillus

Chancroid…

Occurrence

- Endemic in many developing countries
- Most frequently diagnosed in men, especially those who frequently prostitutes

Reservoir

- Humans

Mode of transmission

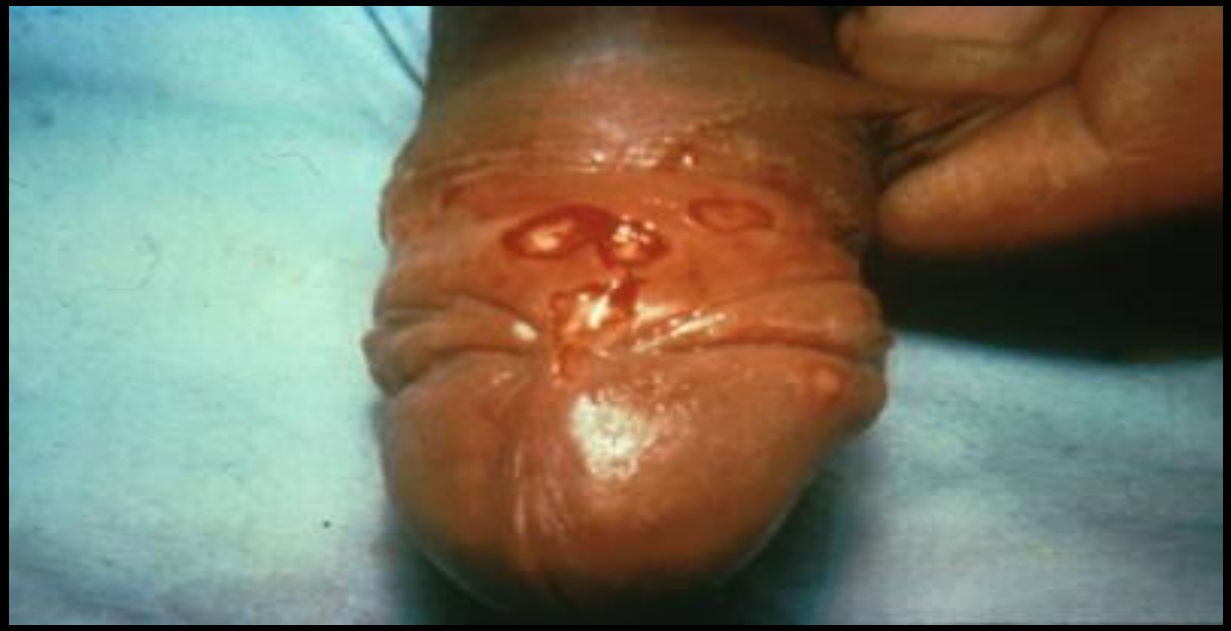
- Direct sexual contact with discharges from open lesion and pus from buboes

Incubation period

- 2-10 days

Clinical manifestations

- Classic chancroid ulcer begins as a **tender papule** that ulcerates with in 24 hours
- The ulcer is painful, irregular & sharply demarcated from the nearby skin
- About 50% of men will have single ulcer
- It can complicate as penile auto-amputation



Chancroid ulcers

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Chancroid Male - regional adenopathy

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Diagnosis

- Clinical, but always rule out syphilis
- Gram stain of smear shows typical **rods** in chain
- Culture

HERPES GENITALIA

- It is a viral infection characterized by a localized primary lesion, latency and a tendency to localized recurrence

Infectious agent

- Herpes simplex virus type two (HSV_2)

Occurrence

- World wide
- Prevalence is greater (up to 60%) in lower socioeconomic groups & persons with multiple sexual partners
- **The commonest cause** of genital ulcer world wide and in Ethiopia

Herpes genitalia…

Reservoir

- humans

Mode of transmission

- usually sexual contact
- transmission to neonate usually occurs via the infected birth canal but less common in intra uterine

Incubation period

- 2-12 days

Clinical manifestations

- primary genital herpes is characterized by fever, headache , malaise & myalgias
- pain, itching, dysuria, vaginal and urethral discharge and tender inguinal LAP-predominant local symptoms
- lesions may be vesicles, pustules, or painful erythematous ulcers.

Clinical manifestations…

- Cervix and urethra are involved in more than **80% women**
- A **clear mucoid discharge & dysuria** are characteristic symptoms of urethritis
- Occasionally, endometritis & salpengitis in women and prostatitis in men.

GONORRHEA

- It is an acute or chronic purulent infection of the **urogenital tract**

Infectious agent

- Neisseria gonorrhea, the gonococcus

Occurrence & importance

- World wide
- Affects both genders, especially sexually active adolescents and young adults.
- Causes sterility in both males and females
- Gonococal ophthalmia of the new born may cause blindness

Gonorrhea…

Reservoir

- Humans

Mode of transmission

- Almost always as a result of sexual activity

Incubation period

- Usually 2-7 days

Clinical manifestations

In males:

- Urethritis with **thick yellow purulent** discharge dysuria and frequency
- Epididymitis, prostatitis and infertility
- Urethritis may cause urethral stricture resulting in urine retention and eventually hydronephrosis and kidney failure.

Clinical manifestations …

In females:

- Females are usually asymptomatic
- Vaginal discharge is common
- Most common site of **infection is cervix**, followed by urethra, anal canal and pharynx.
- Bartholinitis occurs unilaterally
- Salpengitis or pelvic peritonitis can give tubal obstruction with the danger of ectopic pregnancy or infertility

Clinical manifestations & diagnosis

- In both **males and females:**
  - ➢ Gonorrhea can be complicated by septicaemic spread, resulting in arthritis, endocarditis or meningitis, but these are rare

Diagnosis

- Gram stain of discharge (urethral, cervical , conjunctival)
- Culture on selective media

TRICHOMONIASIS

- It is a common and persistent **protozoal disease** of the genitourinary tract.

Infectious agent

- Trichomonas vaginalis, a flagellate protozoan, likes an acid environment (PH =4)

Occurrence

- World wide
- Highest incidence- among females 16-35 yrs

Reservoir

- Humans
- Men are symptom free carriers for years

Trichomoniasis…

Mode of transmission

- By contact with vaginal and urethral discharges of infected people during sexual intercourse
- Indirect through contact with contaminated articles and clothes

Incubation period

- 4-20 days, average 7 days

Clinical manifestations

- Most men remain asymptomatic although some develop, urethritis and a few have epididymitis or prostitis
- Infection in women is usually symptomatic & manifests with:
  - Malodorous vaginal discharge **often yellow**
  - Vulvar erythema and itching
  - Dysuria or urinary frequency
  - Dyspareunia

Diagnosis

- Detection of **motile trichomonads** by microscopy of wet mounts of vaginal or prostatic secretions (fresh specimen)
- Culture (most effective), takes 3-7 days
- Gram stain to exclude gonococcus for a patient complaining of urethral discharge

CHLAMYDIAL INFECTIONS

- It is an infection caused by Chlamydia trachomatis, an **obligate intracellular bacterium**
- **Incubation period**
  - One week to several months, usually 1-3 week
- **Transmission**
  - Anal or vaginal intercourse
- **Clinical manifestations**
  - Mucoid or mucopurulant discharge
  - Occccasionaliy erythematous urethral meatus

LYMPHO GRANULOMA VENEREUM /LGV/

- It is a veneral disease caused by chlamydia microorganisms, most commonly manifested by **acute inguinal lymphadenitis**

Infectious agent

- Chlamydia trachomatis (L_1, L_2 & L_3) gm -ve bacterium

Occurrence

- World wide
- Very common in tropical & subtropical Africa & Asia
- Its incidence is lower than gonorrhea & chancroid

Lympho granuloma venereum…

Reservoir

- Humans
  - ➢Often asymptomatic, particularly in females

Mode of transmission

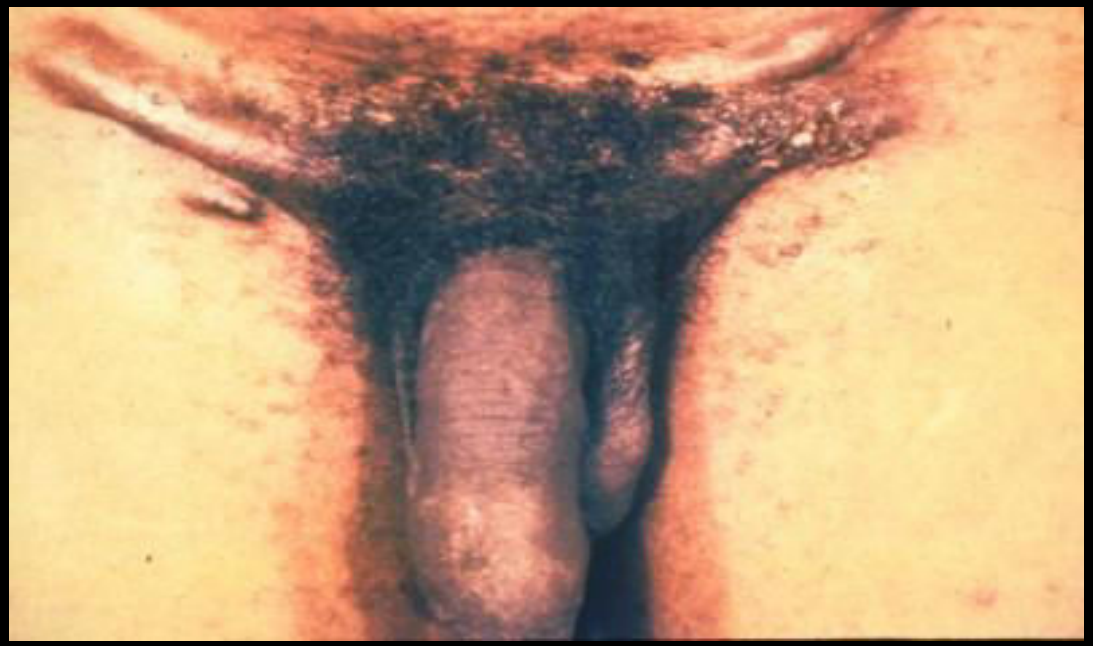
- Direct contact with open lesions of infected people, usually during sexual intercourse

Incubation period

- 3-30 days

Clinical manifestations

- **Lymphadenopathy** with non- specific symptoms of fever, chills, head ache, malaise, anorexia & weight loss
- Regional lymph nodes under go suppuration, followed by adjacent tissue involvement
- In the female, inguinal nodes are less frequently affected and involve **mainly pelvic nodes** with extension to the rectum & rectovaginal septum, resulting in proctitis, stricture of the rectum & fistula.



LGV lymphadenopathy

Division of STD Prevention

CDC Centers for Disease Control and Prevention

Diagnosis

- Clinical presentation (presence of bubo)
- Culture of bubo aspirate

Approaches to STIs management

1. Classical approaches

- Etiologic diagnosis
- Clinical diagnosis

2 . Syndromic approach

1. Classical approaches to STIs management

Etiologic diagnosis

- Using lab to identify the causative agent

Clinical diagnosis

- Using clinical experience to identify causative agent

Etiologic management

Advantages:

- ✓Avoids over treatment
- ✓Conforms to traditional clinical training
- ✓Satisfies patients who feel not properly attended to
- ✓Can be extended as screening for the asymptomatic patients

Problems of etiologic approach

- Requires **skilled personnel** &consistent supplies
- Treatment does not begin until results are available
- It is time consuming & expensive
- Testing facilities are not available at **primary level**
- Some bacteria are fastidious &difficult to culture (H.ducrey, C.trachomatis)
- Lab. results often not reliable
- Mixed infections often overlooked
- Miss-treated/untreated infections can lead to complications and continued transmission

Clinical management

Advantages:

- ✓Saves time for patients
- ✓Reduces laboratory expenses

Disadvantages:

- ✓Requires high clinical acumen
- ✓Most STIs cause similar symptoms
- ✓Mixed infections are common &failure to treat may lead to serious complications
- ✓Doesn’t identify asymptomatic STIs

2. Syndromic approach

- Syndrome – is group of symptoms patient complains & clinical signs you observe during examination
- There are seven syndromes (aim is to identify &manage accordingly)

Identifying syndromes

SYNDROME	MOST COMMON CAUSE
Vaginal discharge	Vaginitis(trichomniasis, candidisis, bacterial vaginosis, Gardenella vaginalis) Cervicitis(gonorrhea, chlamydia)
Urethral discharge in men	Gonorrhea, chlamydia
Genital ulcer	Syphilis, chancroid, herpes
Lower abdominal pain	Gonorrhea, chlamydia, mixed anaerobes
Scrotal swelling	Gonorrhea, chlamydia
Inguinal bubo	LGV, chancroid
Neonatal conjunctivitis	Gonorrhea, chlamydia

Syndromic management key features /advantages/

- Problem oriented (responds to patient's symptoms)
- Highly sensitive & does not miss mixed infections
- Treats the patient at first visit
- Can be implemented at primary health care level
- Use flow charts with logical steps
- Provides opportunity & time for education &counseling

The four steps in syndromic STI case management

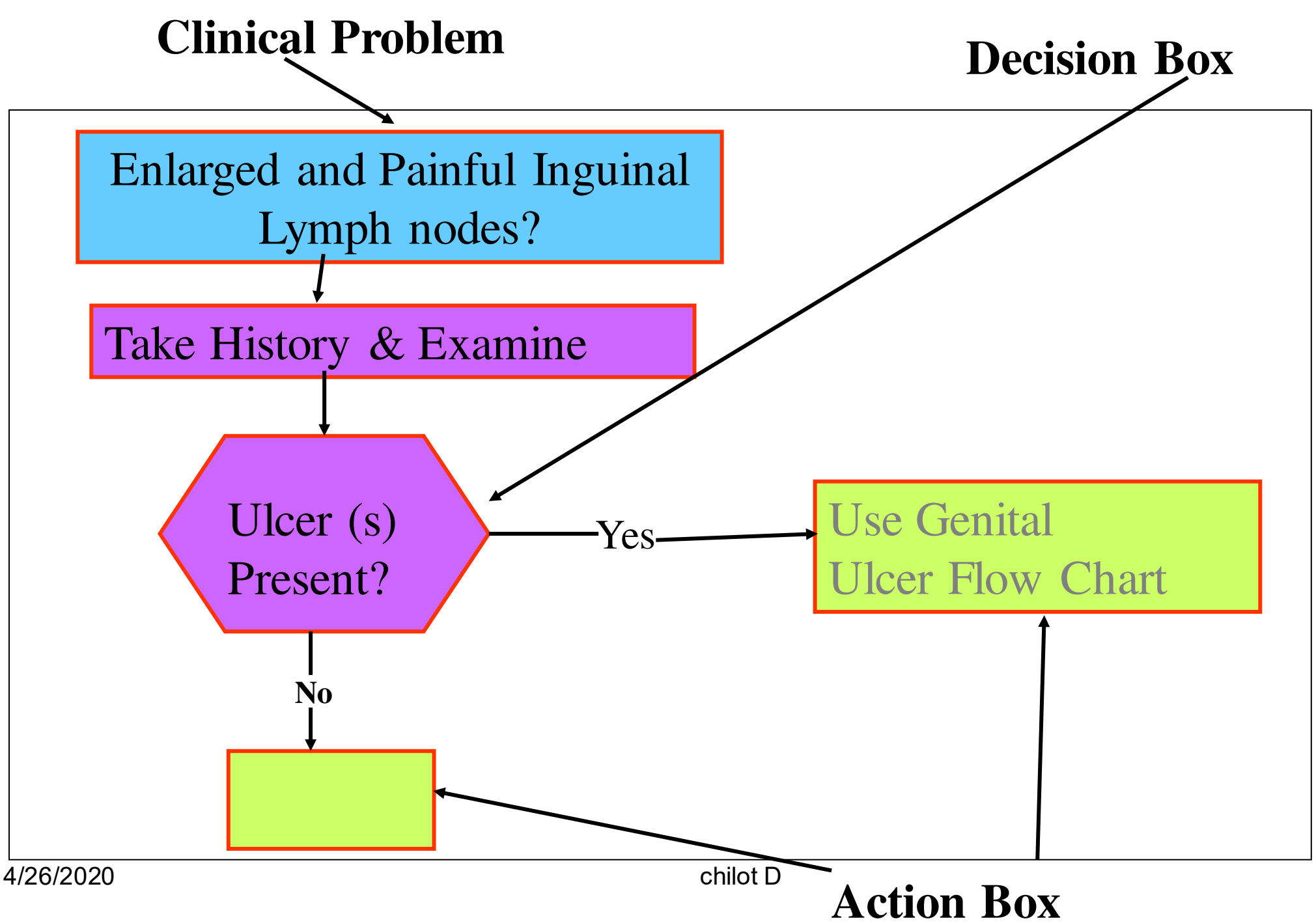
- History taking and examination
- Syndromic diagnosis and treatment, using flow charts
- Education and counseling on STI and HIV testing and safer sex, including condom promotion and provision
- Management **of sexual partners**

Limitations of syndromic management

- Misses sub-clinical infection
- Needs validation study
  - Require prior research to determine the common causes of particular syndromes
- Needs training

Syndromic flow- charts

- A flow chart is a diagram (map) representing steps to be taken through a process of decision making
- Can be used at **any health facility**
- Each flow chart is made up of three steps
- The clinical problem (patient's presenting symptom)
  - **Problem box**
- A decision to make usually by answering yes or no to a question
  - **Decision box**
- An action to take (what you need to do)
  - **Action box**


Clinical Problem
Decision Box
Enlarged and Painful Inguinal Lymph nodes?
Take History & Examine
Ulcer (s) Present?
Yes
Use Genital Ulcer Flow Chart
No
Action Box

Urethral discharge

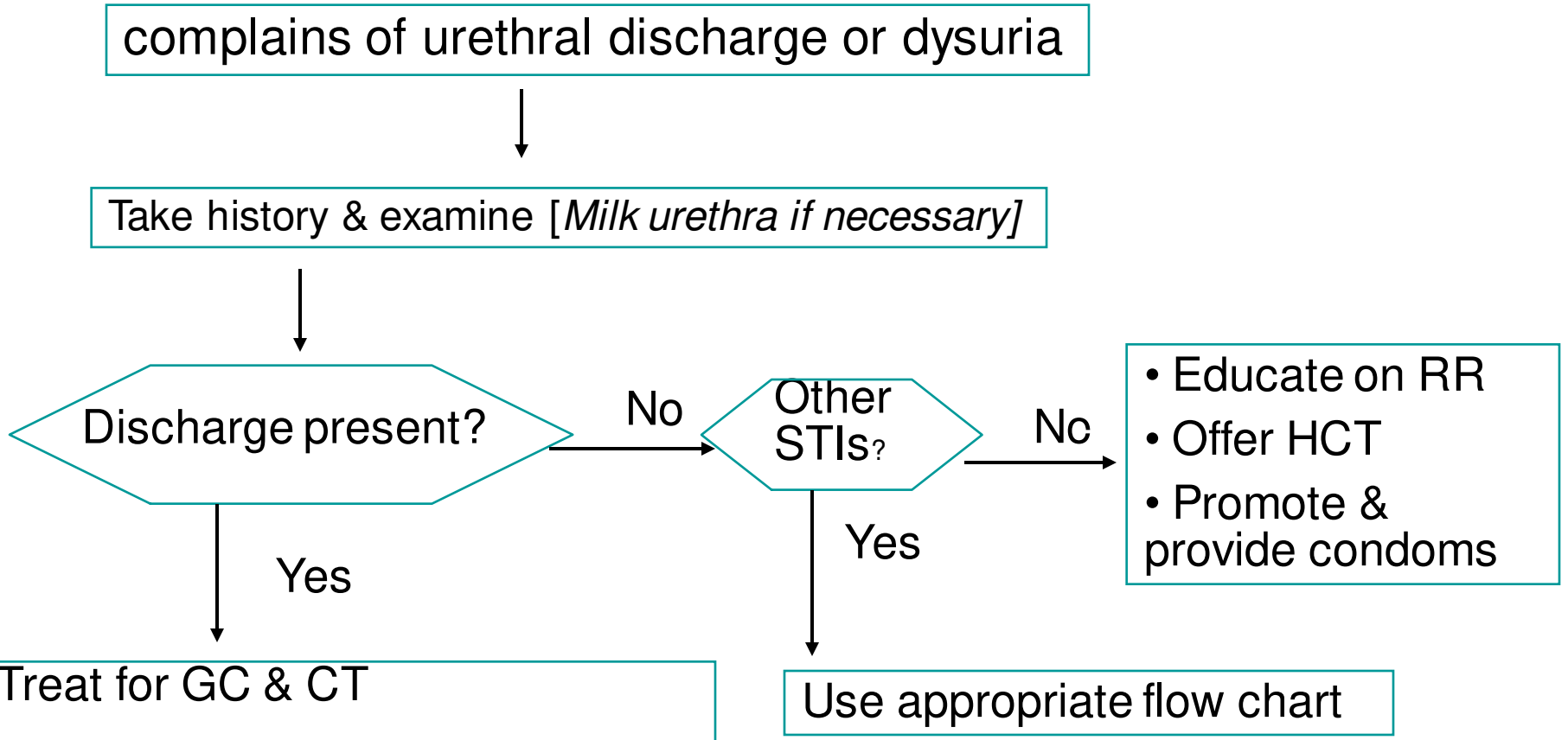


Figure 1

SEVEN SYNDROMES OF STIs AND THEIR SYNDROMIC TREATMENT

1. urethral discharge syndrome

Clinical presentations

- Burning sensation on urination & urethral discharge are common symptoms of urethritis in men
- N.gonorrhea & C.trachomatis are common causes
- T.vaginalis is found to be the second most common cause exceeding C.trachomatis in Ethiopia

Management of recurrent/persistent urethritis

- Recurrence could be due to inadequate treatment or poor compliance, re-infection or infection by drug-resistant organisms
- Look for objective signs of urethritis
- Re-treat with initial regimen if non-compliant or re-exposure occurs
- *Trichomonas vaginalis* could be treated with Metronidazole
- Patient should be warned to avoid use of alcohol while taking metronidazole

2. Genital ulcer syndrome

Causes include:

- H.simplex type two (genital herpes)
  - the commenest cause
- T.pallidum(syphillis)
- H.ducrey(chancroid)

3. Vaginal discharge syndrome

Common causes of vaginal discharge

- **Sexually transmitted**
  - ***Neisseria gonorrhoeae*[4]**
  - ***Chlamydia trachomatis*[5]**
  - ***Trichomonas vaginalis*[3]**
- **Endogenous infection**
  - ***Gardnerella vaginalis*[1]**
  - ***Candida albicans*[2]**

Vaginal discharge…

Initial evaluation of patients with vaginal discharge include:

- Risk assessment
- Clinical speculum examination to determine site of infection

Risk factors for cervicitis:

- Age less than 25 years
- Having multiple sexual partner in the last three months
- Having new partner in the last three months
- Having ever traded for sex

Vaginitis & cervicitis

VAGINITIS	CERVICITIS
Trichomoniasis, candidiasis, bacterial vaginosis	Gonorrhea & chlamydia
Most common cause of vaginal discharge	Less common cause of vaginal discharge
Easy to diagnose	Difficult to diagnose
No complications	Major complications
Partner treatment unnecessary	Partner treatment needed

Complication of vaginal discharge syndrome

- Pelvic inflamatory disease
- Premature rupture of membrane
- Pre -term labor
- Infertility
- Chronic pelvic pain

4. Inguinal bubo syndrome

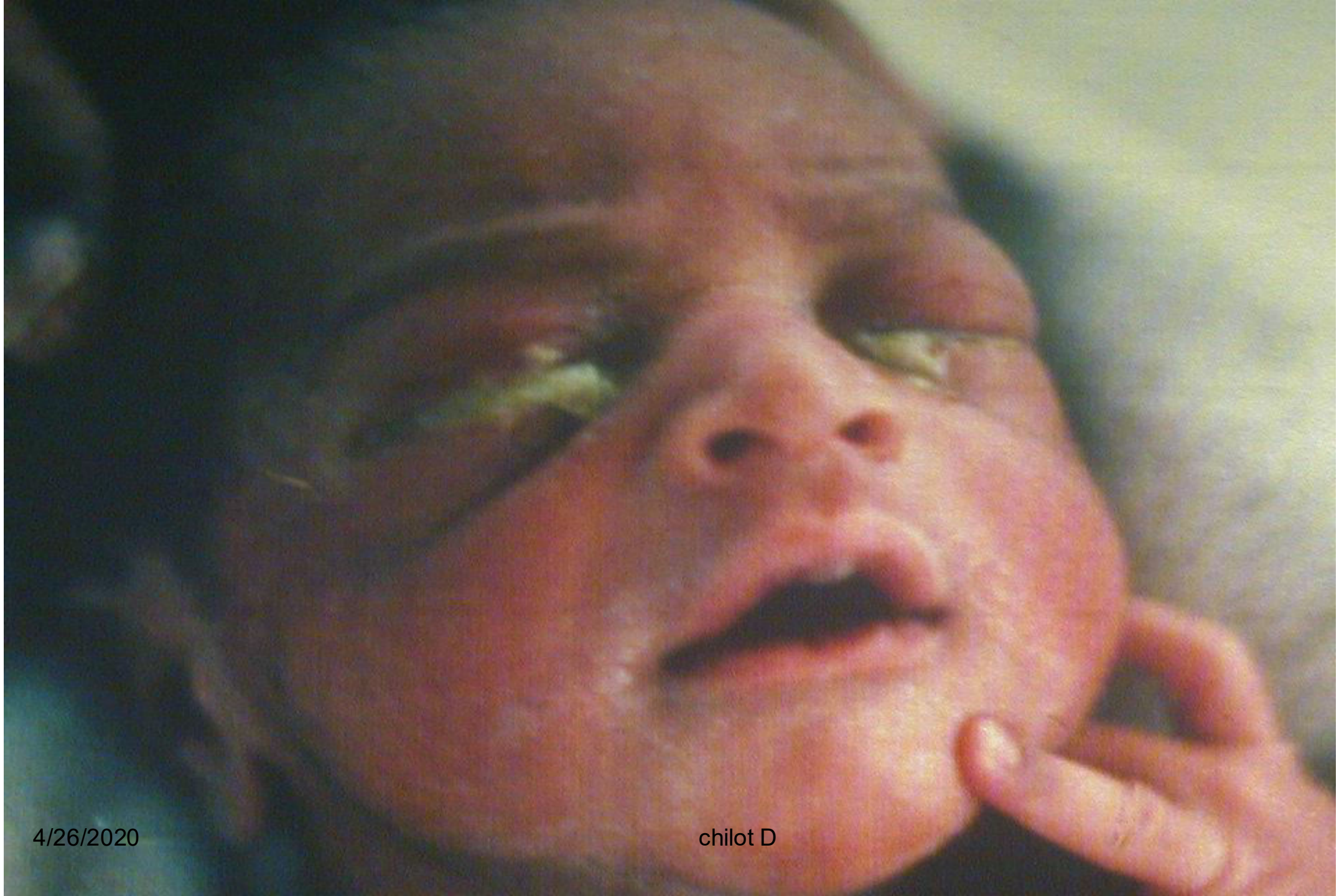
- Inguinal bubo is **a painful**, often fluctuant, swelling of the lymph nodes in the inguinal region (groin)
- The common sexually transmitted pathogens that are associated with inguinal bubo include:
  - *C. trachomatis (serovar L1, L2, and L3)-LGV*
  - *H. ducreyi*
  - *Klebsiella granulomatis for granuloma inguinale /GI/ or Donovanosis*

5. Neonatal conjuctivitis (ophthalmia neonatorum) syndrome

- It is defined as purulent conjuctivitis occurring in a baby less than **one month of age.**
- Sight-threatening condition
- The most important causes are *gonorrhoea* and *chlamydia*
- If caused by gonorrhoea**, blindness** often follows

Neonatal conjuctivitis …

- Common presentation are redness, swelling of the eye lid &discharge from the eye (**sticky eye**)
- For babies older than one month, the cause is **unlikely** to be an STI
- In developing countries, gonorrhoea accounts for **20-75%** and chlamydia for **15-35%** of cases of neonatal conjuctivitis



Preventions of neonatal conjuctivitis

- As soon as the baby is born, carefully wipe both eyes with dry, clean cotton wool, then apply:
- **1% tetracycline eye ointment into the infant's eyes or**
- 1% silver nitrate solution or
- Other options:
  - ➢0.5% Erythromycin ointment or
  - ➢2.5% povidone iodine solution;

6. Scrotal swelling syndrome

- C.trachomatis & N.gonorrhea are common causes in pts <35 yrs
- In patients > 35 yrs, it is commonly caused by gram negative bacteria
- Other infectious causes of scrotal swelling could be brucellosis, mumps, onchocerciasis
- In pre-pubertal children the usual etiology is coliform, pseudomonas or mumps virus
- Mumps epidedimorchitis is usually noted within a week of parotid enlargement

Scrotal swelling syndrome…

- Other causes of scrotal swelling include, testicular torsion, trauma, tumor and incarcerated inguinal hernia
- Complications of scrotal swelling caused by STI:
  - Epididymitis
  - Infertility
  - Impotence
  - Prostatitis

Syndromic management…

Syndromes	Causes	Treatment
.ower abdominal pain	Gonorrhea, Chlamydia, Mixed anaerobes	Ceftriaxone+Azithromycin+metronidazole
-crotal swelling	Gonorrhea, Chlamydia	Ceftriaxone+Azithromycin
nguinal bubo	LGV, Chancroid	Ciprofioxacin+Doxycline /Erythromycin
Neonatal conjunctivitis	Gonorrhea, Chlamydia	Ceftriaxone+Azithromycin

Syndromic management…

syndrome	Common causes	Sign and symptoms	treatment
Vaginal discharge with STI risk assessment +ve	N.Gonorrhea C.Trachomatis T.Vaginalis C.albicans	Vaginal discharge	Ceftriaxone+Azithromycin+metronidazole
Vaginal discharge with _ve STI risk assessment	Bacterial vaginosis T.vaginosis		Metronidazole+clotrimazole
Urethral discharge	N.Gonorrhea C.Trachomatis	Urethral discharge dysuria	Ceftriaxone+Azithromycin
Genital ulcer	HSV2 Syphilis C.Trachomatis chancroid	Non-vesicular	Bezanthine peniciline+Doxicyclinc+Ciprofloxacin +acyclovir
		vesicular	acyclovir

Thank you