MINISTRY OF HEALTH OF UKRAINE
POLTAVA STATE MEDICAL UNIVERSITY
DEPARTMENT OF THE GENERAL SURGERY WITH PATIENT'S CARE

CHRONIC SPECIFIC SURGICAL INFECTION

Lecture for general surgery

Chorna I.O.

Poltava

“Medical education does not exist to provide students with a way of making a living but to ensure the health of the community”

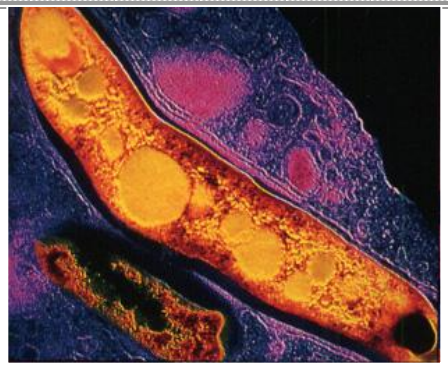
Rudolf Virchow

Plan

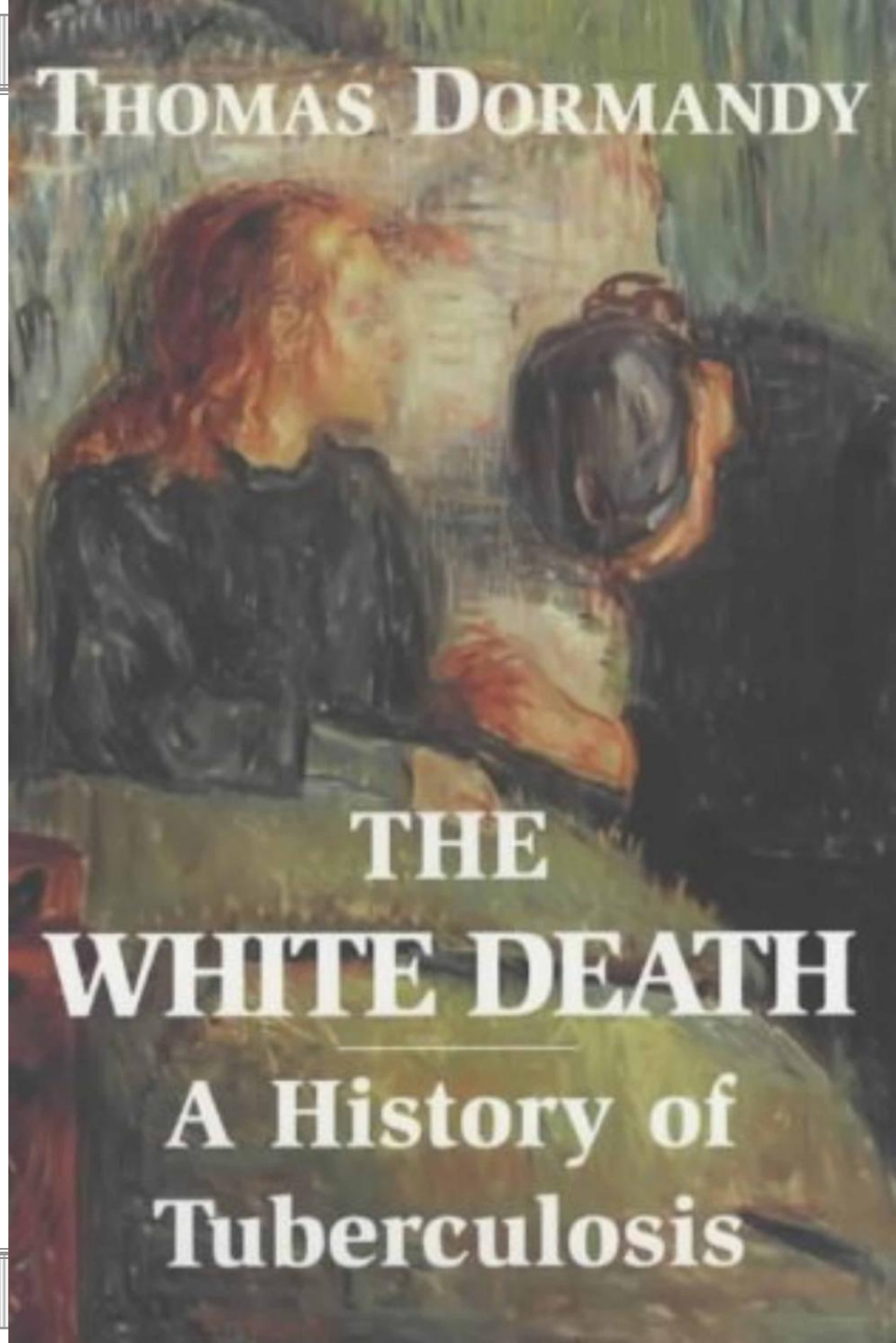
1.Tuberculosis

2.Actinomicosis

3. Sifilis



Tuberculosis
has
been killing
millions
for centuries



Symptoms of untreated active TB

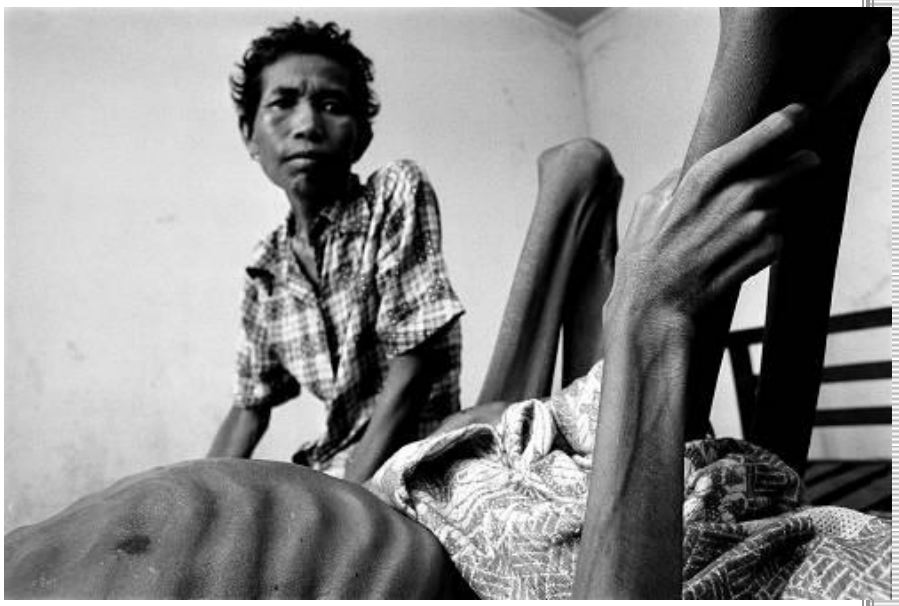
Persistent cough and Low grade-fever

Difficulty in breathing

Night sweats

Blood in sputum

Severe weight loss



Cambodian TB patient

Picture: World Lung Foundation

TB is an ancient human disease!

Recent evidence supports a diagnosis of TB
in this 500,000 year old
young male Homo erectus

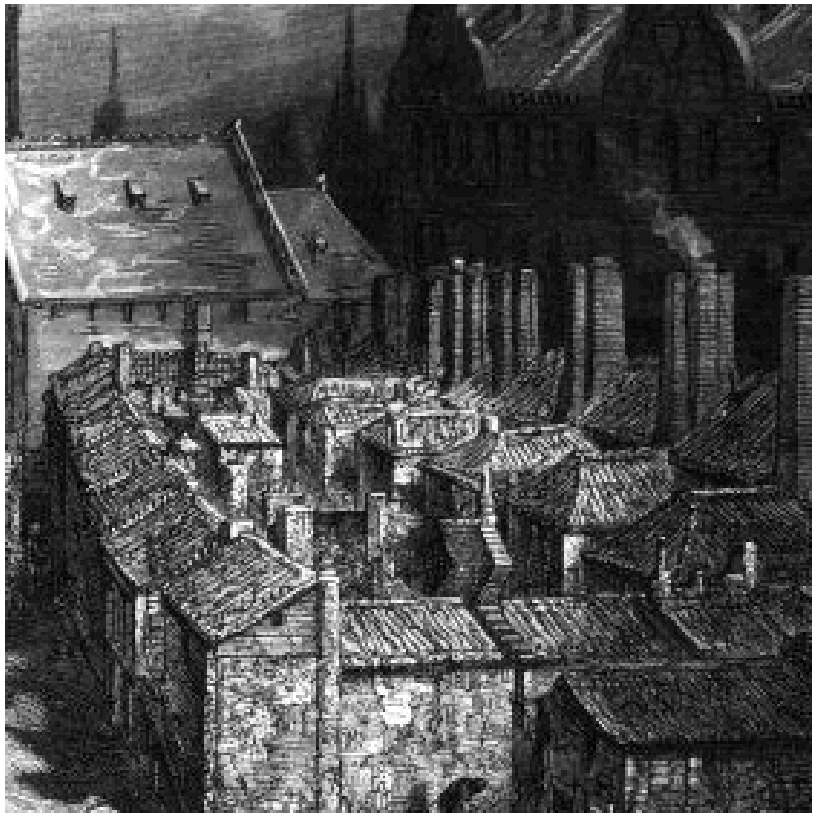
Bone lesions indicative of TB

Credit: Marsha Miller, the University of Texas at Austin

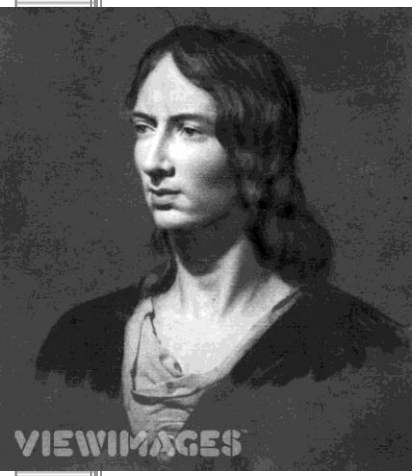
TB is an ancient human disease!

TB spinal lesions have also been found in Egyptian mummies

After urbanization as many as one-quarter of all European deaths in the 1800's may have resulted from TB



The famous and the infamous died of TB



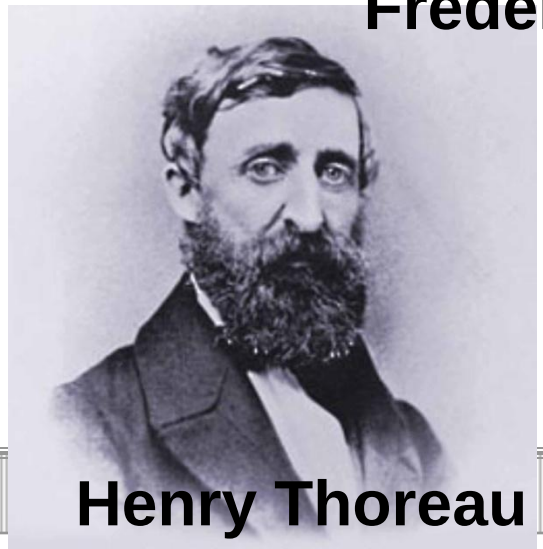
Emily Bronte

John Keats

Anton Chekhov

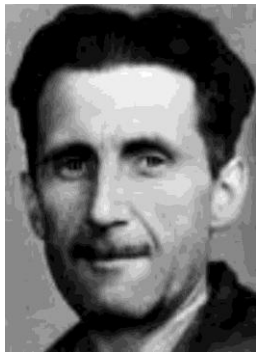
Frederic Chopin

Simon Bolivar



Henry Thoreau

The famous and the infamous died of TB along with countless millions known only to their families

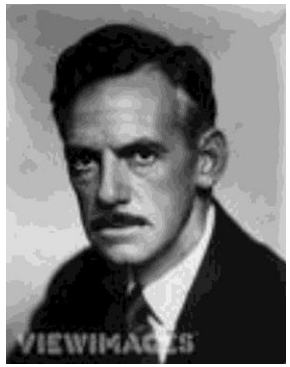


George Orwell

Eleanor Roosevelt

Franz Kafka

DH Lawrence



Eugene O'Neill

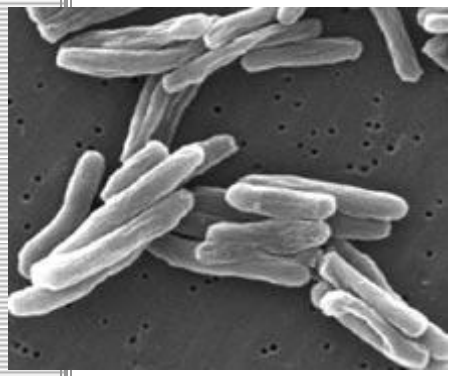
What is Tuberculosis?

Tuberculosis *is a disease caused by tiny germs that enter your lungs when you breathe them in; It is called "**TB**" for short*

TB germs are most commonly found in the lungs, but sometimes they can move to other parts of the body

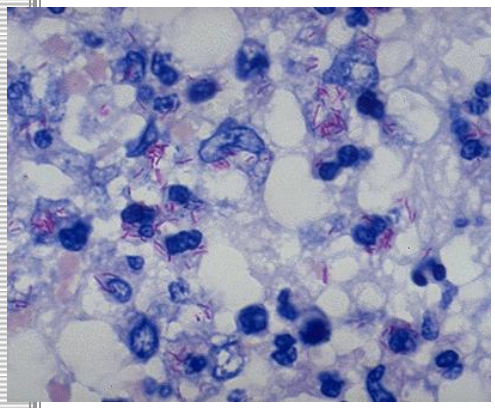
When you have TB disease of the lungs, you can spread it to other people

General Characteristics



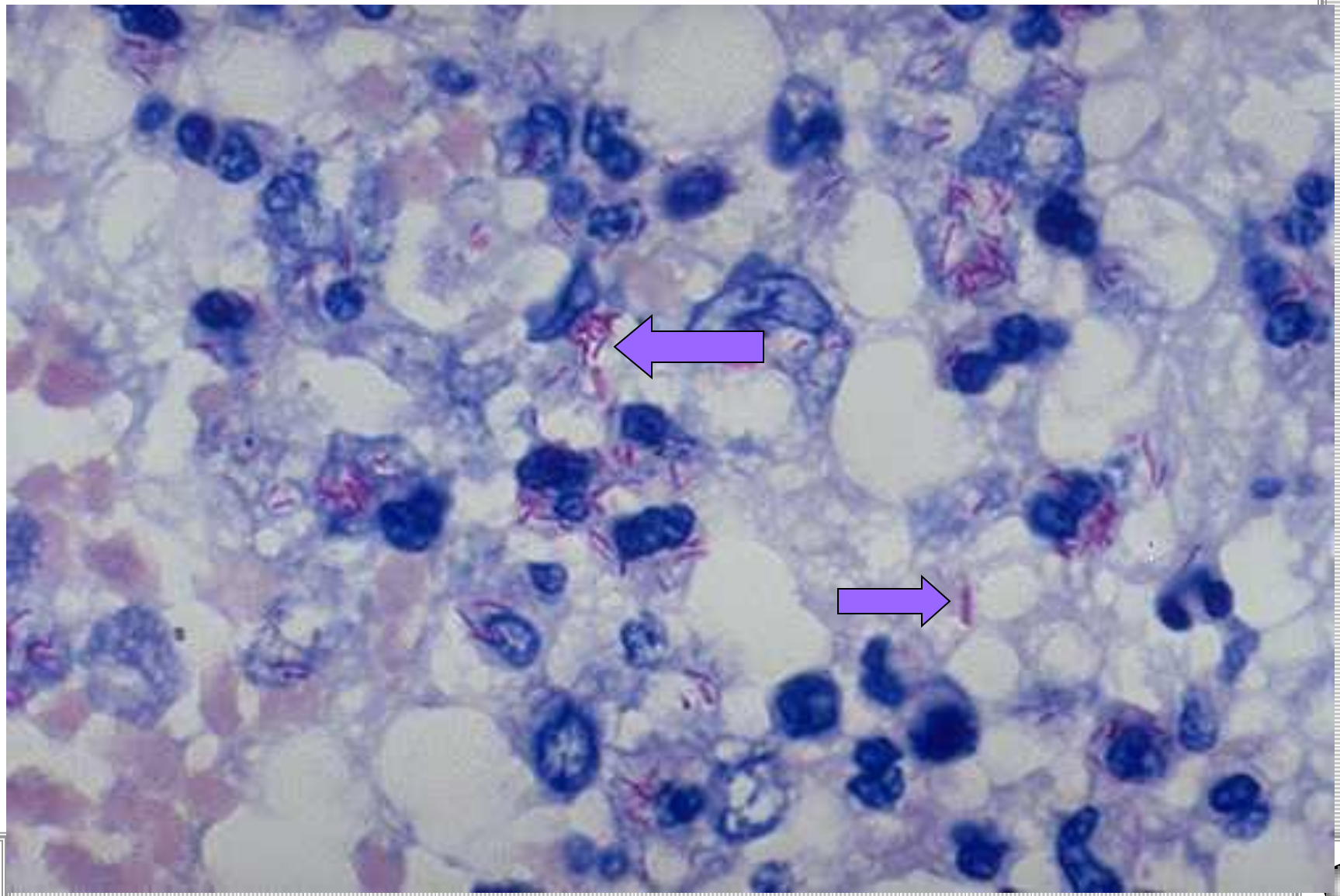
- Family – **Myobacteria**
- Gram-positive aerobic rod-shaped bacilli
- “Acid fast” bacteria
- Lack of spore formation and toxin production
- No capsule, flagellum (non-motile)
- Generation time of 18- 24 hours but requires 3-4 weeks for visual colonies

Pathological Features



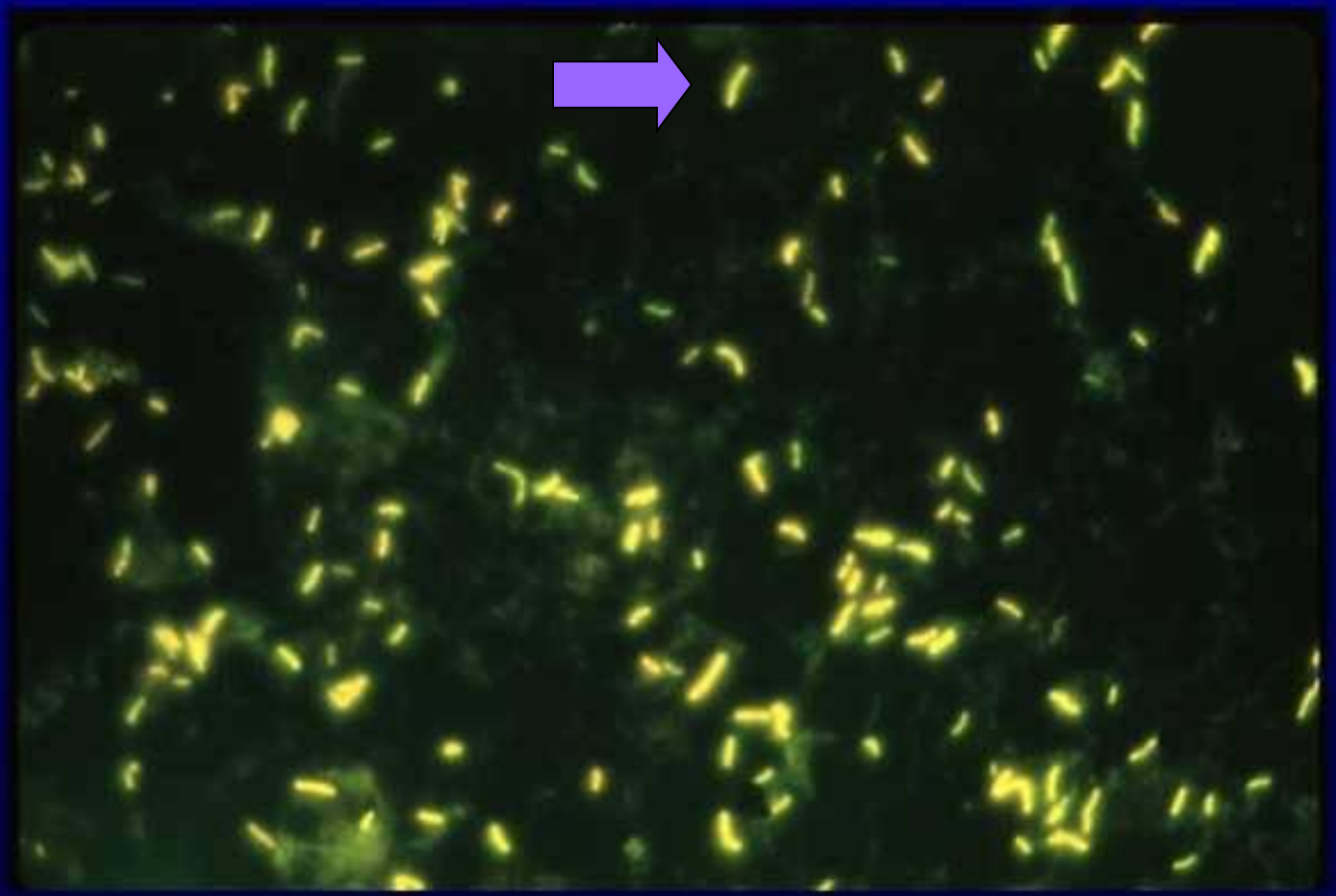
- Principle cause of Human Tuberculosis
- Intracellular pathogen (alveolar macrophages)
- Waxy, thick, complex cellular envelope
- Cell envelope components ex) sulfolipids
- Produces **tubercles**, localized lesions of *M. tuberculosis*

AFB - Ziehl-Nielson stain



Colony Morphology – LJ Slant

Sputum - TB Auromine/Rhodamine



Tuberculosis in Humans

- **Reservoir:** Humans
- **Transmission:** Airborne disease (aerosol transmission)
- **Symptoms**:

Latent TB infection:

No symptoms

*Cannot spread TB

Active TB infection:

Bad cough

Coughing up blood/sputum

Chest pain

Loss of appetite

Weight loss

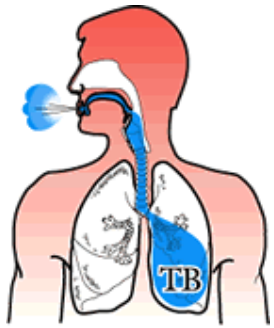
Fever

Chills

Night sweats

Swollen glands

*Contagious



Types of Tuberculous Infections

I- Pulmonary Infection

II- Extrapulmonary infections:

a-TB Pleural effusion

b- Tuberculous meningitis

c- Millary tuberculosis.

d- Renal & urogenital tuberculosis.

e- Bone &joint tuberculosis.

f- TB entritis.

Extra-pulmonary TB

– Symptoms depend on location of infection

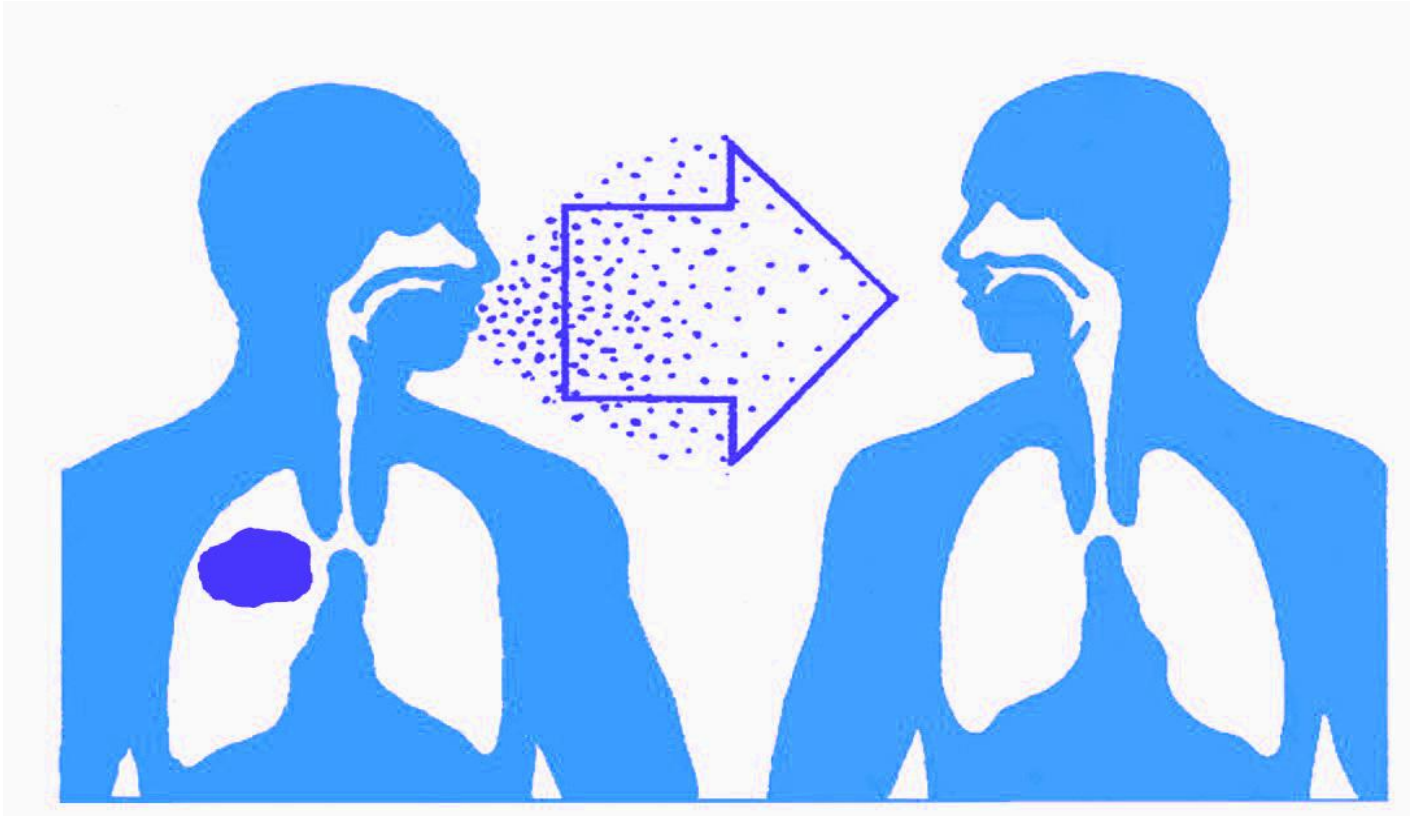
– **General symptoms:** fatigue, fever, loss of appetite, weight loss.

– TB of **lymph nodes**: swelling of lymph nodes

– TB **meningitis**: neurological symptoms including headache

– **Spinal** TB: Mobility impairments, pain

How Are TB Germs Spread?



How Are TB Germs Spread?

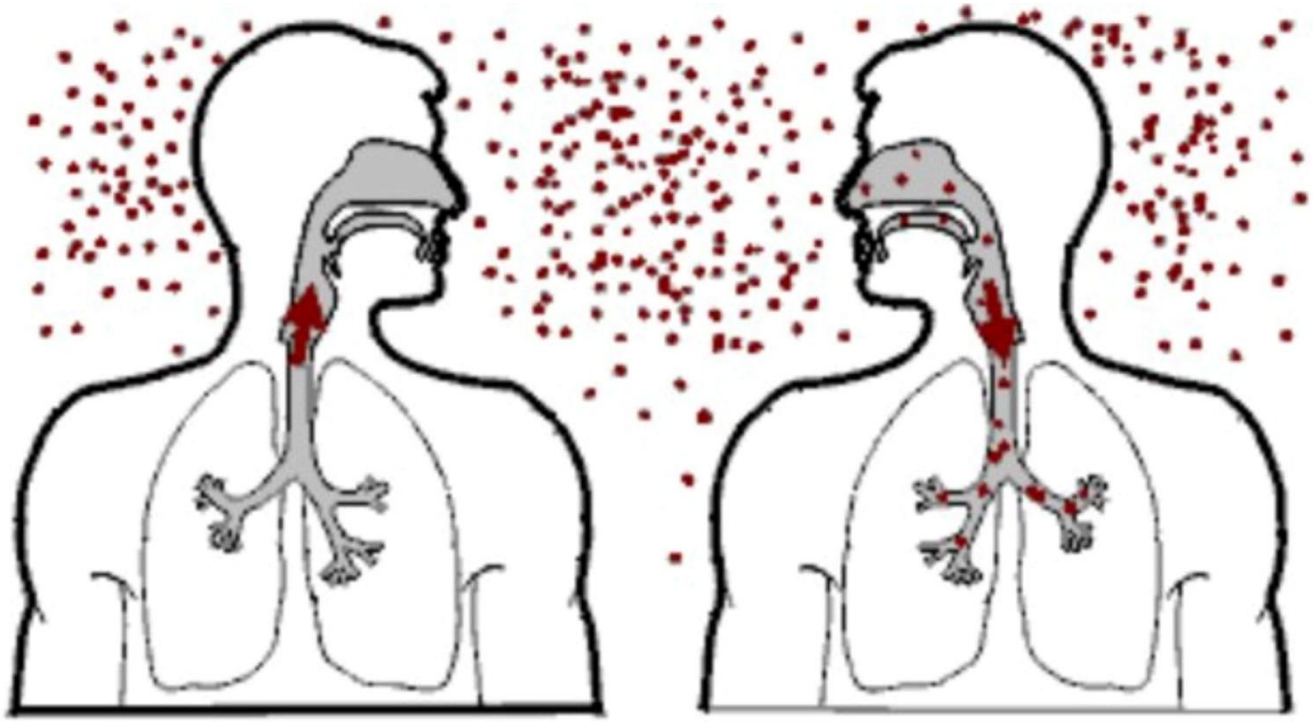
TB germs are passed **through the air** when a person who is sick with TB disease coughs, sings, sneezes, or laughs

To become infected with TB germs, a person usually needs to share air space with someone sick with TB disease (e.g., live, work, or play together)

The amount of time, the environment, and how sick the person is all contribute to whether or not you get infected

In most cases, your body is able to fight off the germs

Left untreated, a person with active TB will infect 10-15 other people per year



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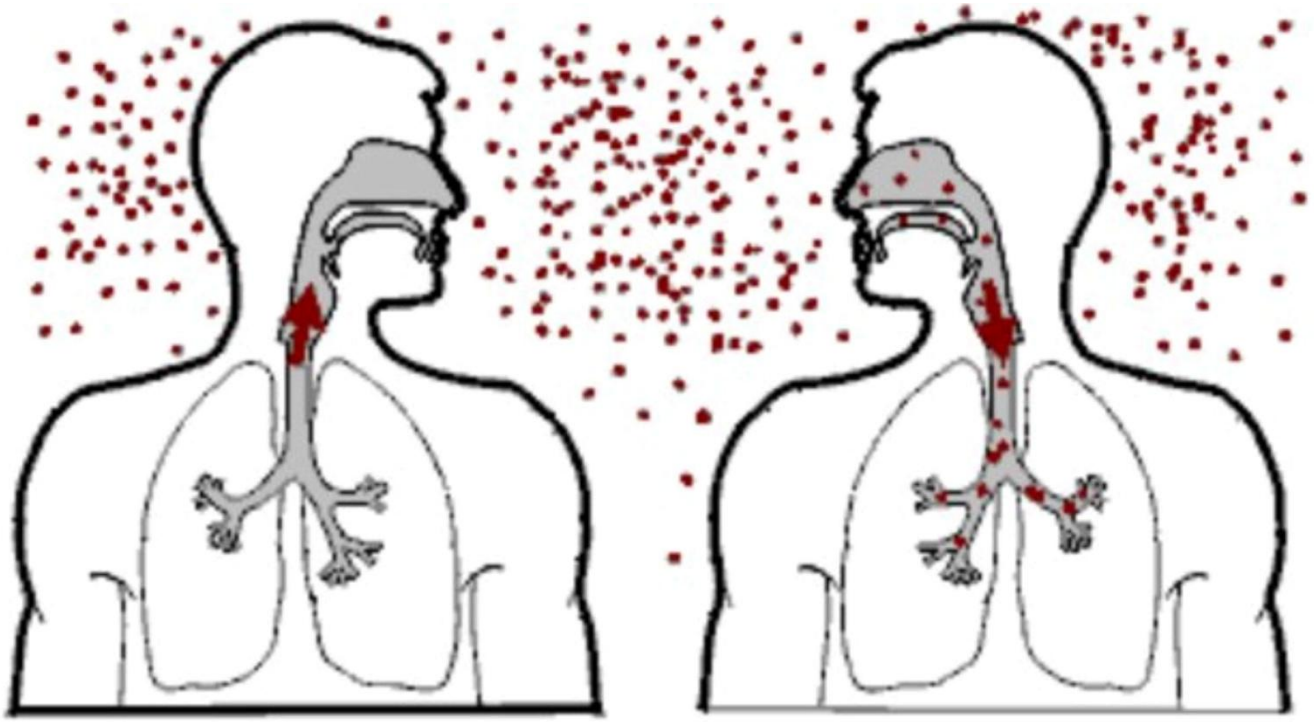
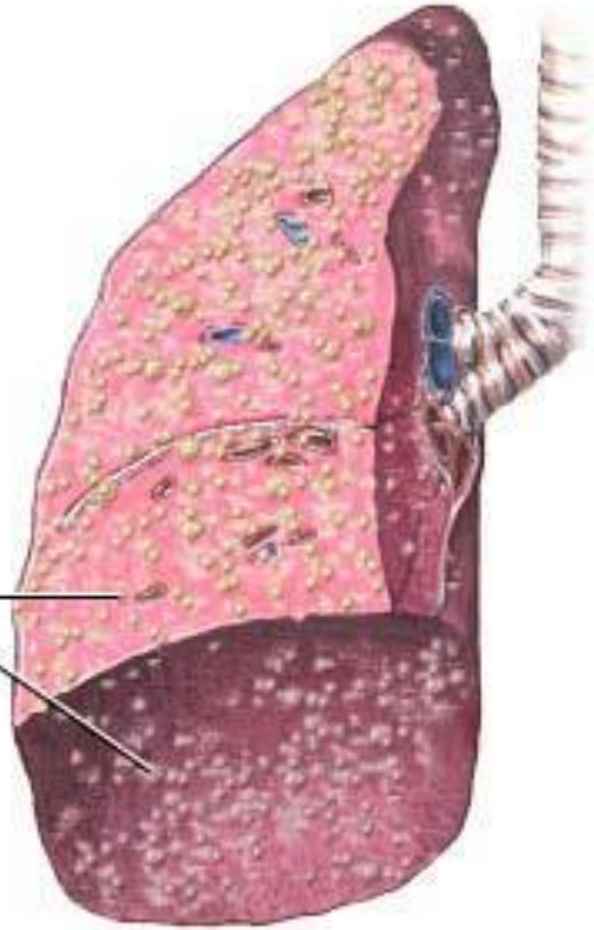
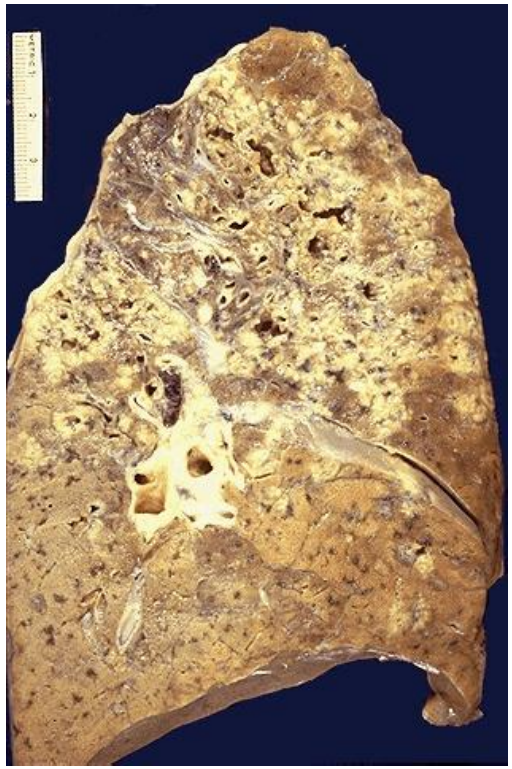


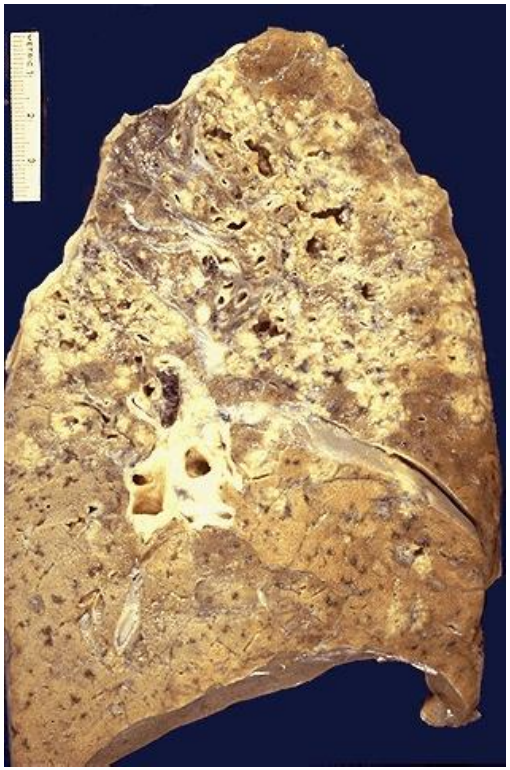
new infections occur at a rate of one per second!

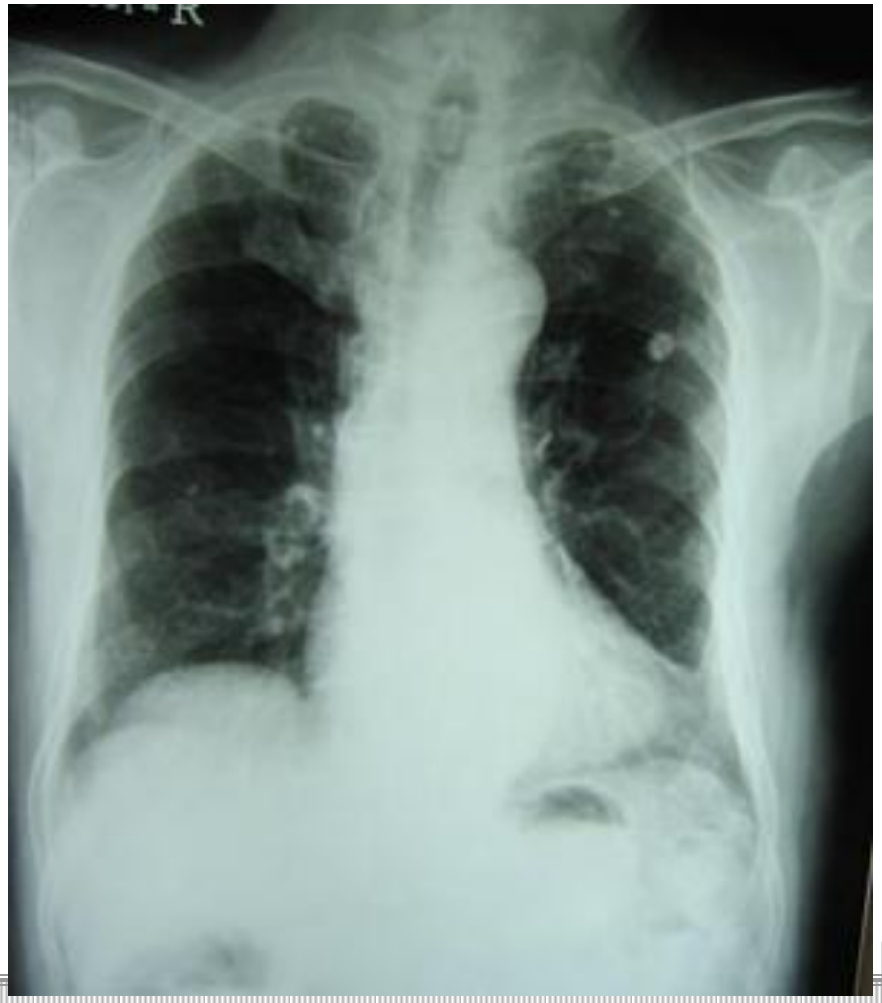
In most cases TB starts as an infection of the lungs = pulmonary TB



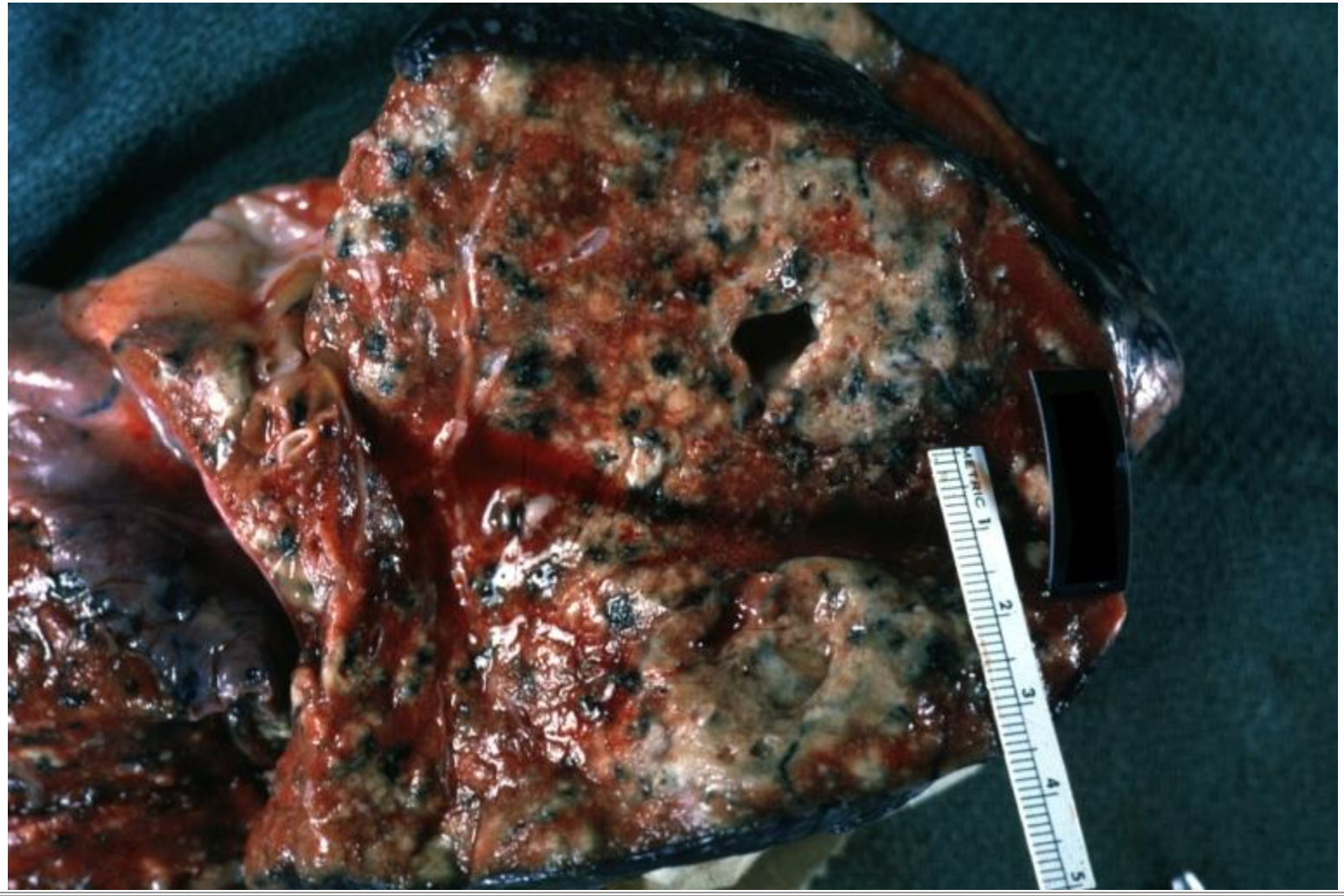


X-rays used to be the primary means of diagnosis

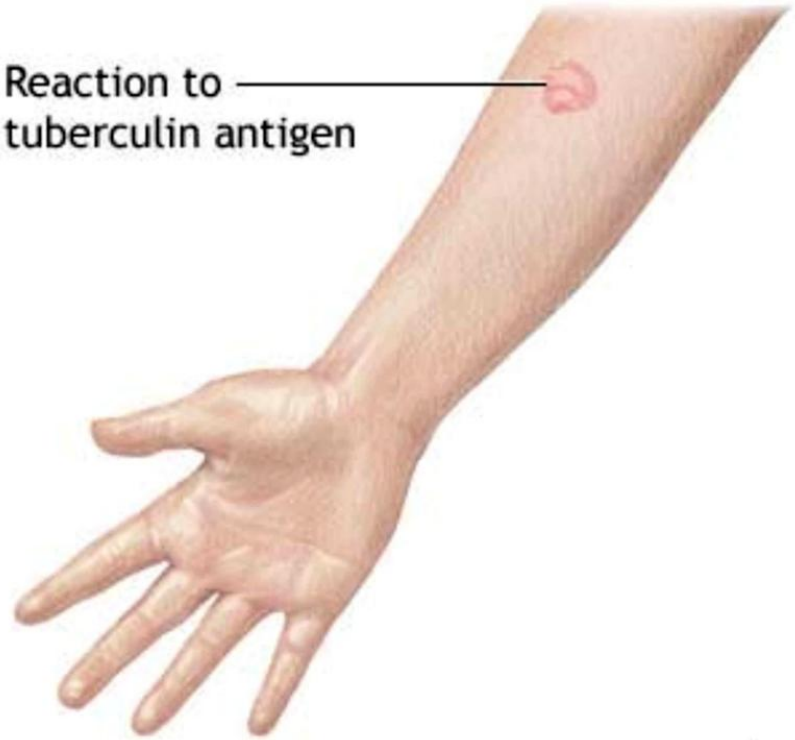




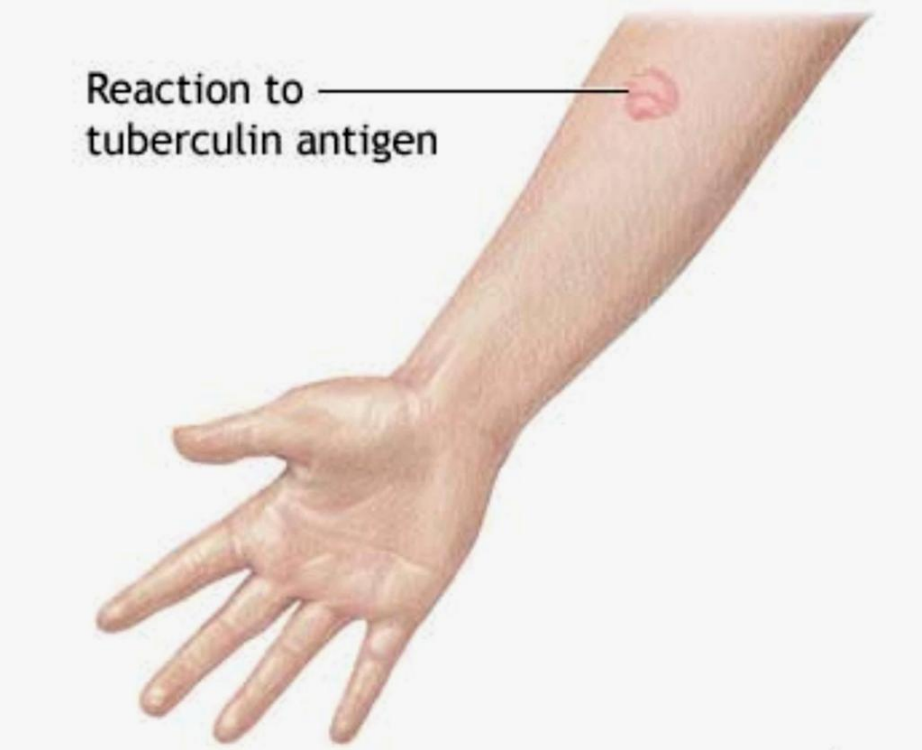
Lung TB - Cavitation



Now TB exposure is diagnosed by a skin test

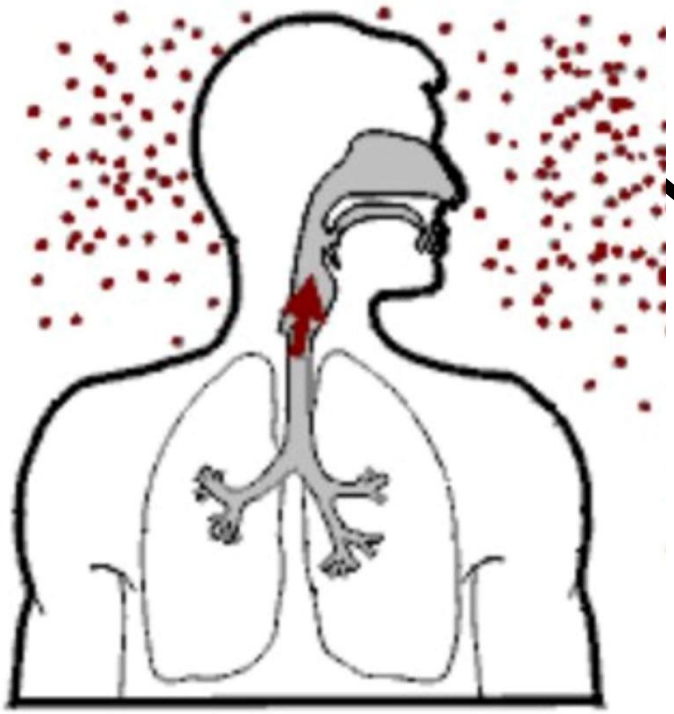


A positive test result simply means you have been exposed to TB* at some point and have developed antibodies to it



*=Active case
Previously “cured”
Inactive case
Vaccinated with BCG

The next step is a sputum culture



The next frontier: DNA testing to identify different strains of TB and assess drug resistance

The next frontier: DNA testing to identify different strains of TB and assess drug resistance

July 2008: WHO unveils

$26 million program

to create labs in poor nations

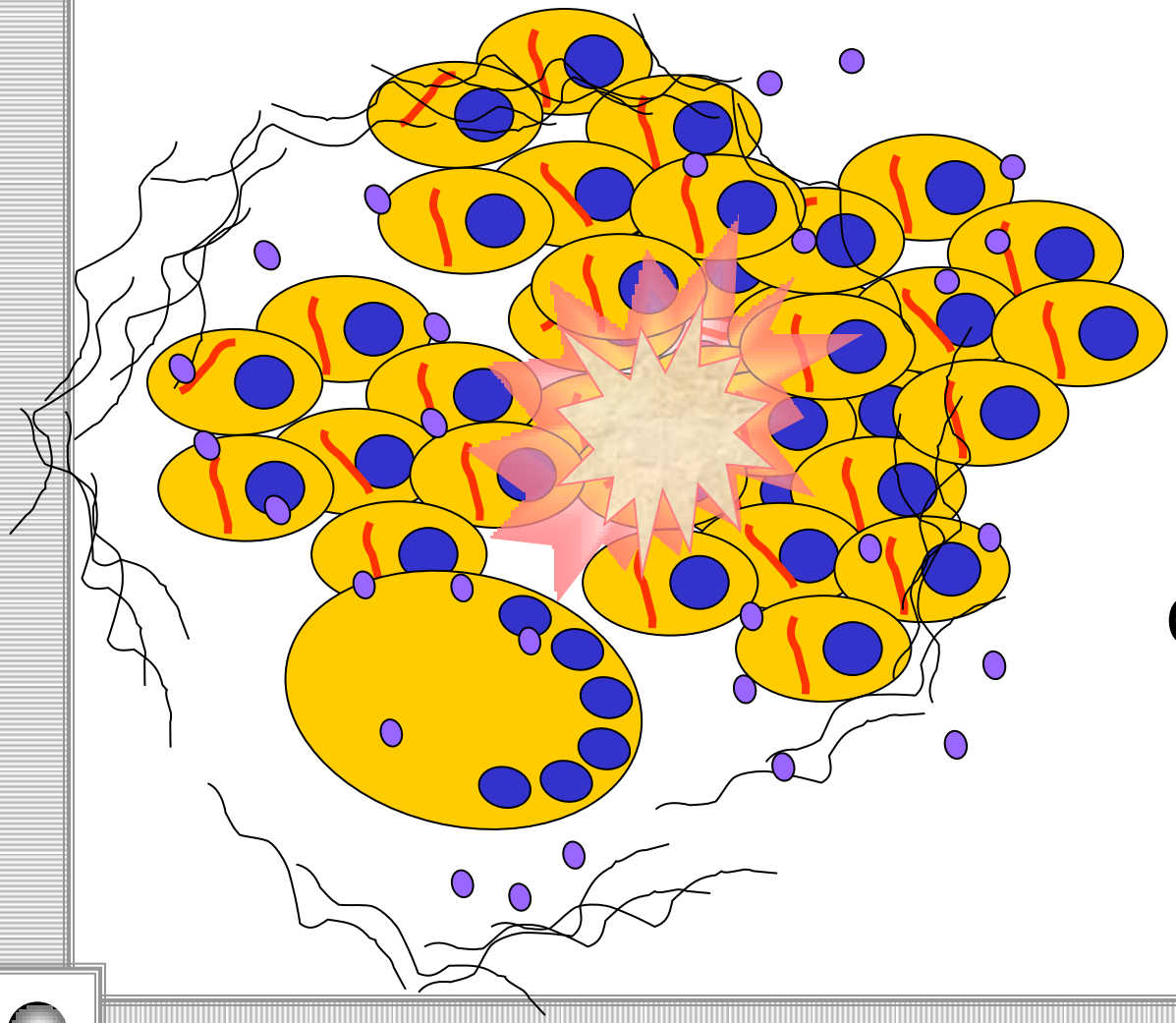
that can do DNA tests

Cost: $5 per test

Time frame: 24 hours

Rather than weeks to months!

TB Pathogenesis



Bacterial entry

T Lymphocytes.

Macrophages.

Epitheloid cells.

Proliferation.

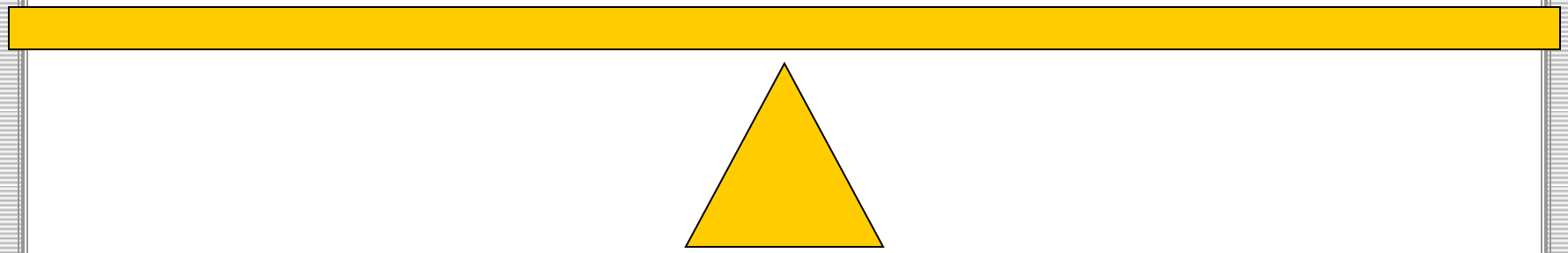
Central Necrosis.

Giant cell formation.

Fibrosis.

Pathogenesis of TB:

Infection - Immunity



Morphology of Granuloma

Rounded tight collection of chronic inflammatory cells.

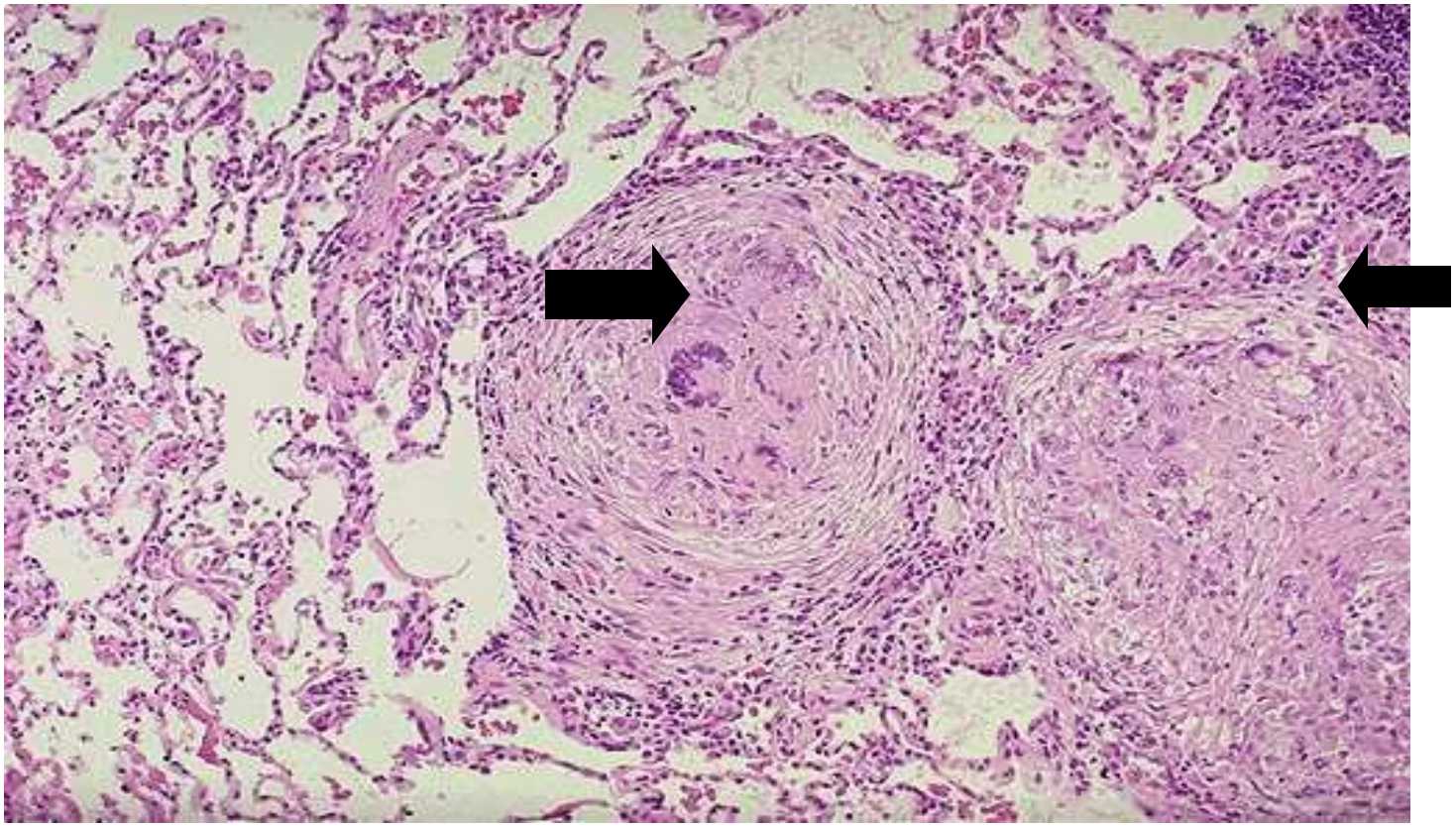
Central Caseous necrosis.

Active macrophages - epithelioid cells.

Outer layer of lymphocytes, plasma cells & fibroblasts.

Langhans giant cells – joined epithelioid cells.

Tuberculous Granuloma



Two forms -
Primary, Secondary

Primary tuberculosis

In a non immunized individual – children* adult*

Lesion in subpleural zone of lung – can be at other sites*

Brief acute inflammation – neutrophils.

5-6 days invoke granuloma formation.

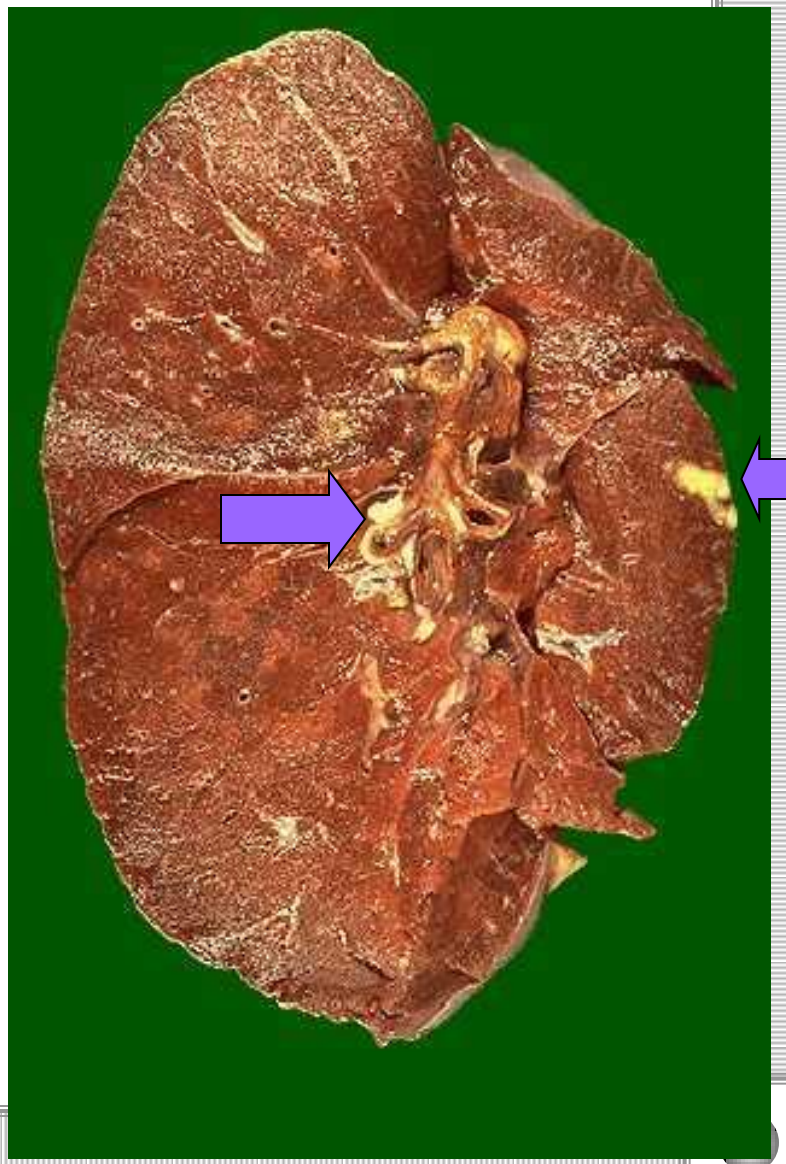
2 to 8 weeks – healing – Ghon focus (+ lymph node → Ghon complex)

Develop immunity – Mantoux positive

Primary or Ghon's Complex

Primary tuberculosis is the pattern seen with initial infection with tuberculosis in children.

Reactivation, or secondary tuberculosis, is more typically seen in adults.



Primary Tuberculosis

In Non Immunized individuals (Children)

Primary Tuberculosis:

- Self Limited disease
- Ghons focus, complex or Primary complex.

Primary Progressive TB

- Miliary TB and TB Meningitis.
- Common in malnourished children
- 10% of adults, Immuno-suppressed individuals

Secondary Tuberculosis:

Post Primary in immunized individuals.

Cavitary Granulomatous response.

Reactivation or Reinfection

Apical lobes or upper part of lower lobes – O_2

Caseation, cavity - soft granuloma

Pulmonary or extra-pulmonary

Local or systemic spread / Miliary

- Vein – via left ventricle to whole body
- Artery – miliary spread within the lung

Secondary Tuberculosis:

Reactivation occurs in 10-15% of patients.

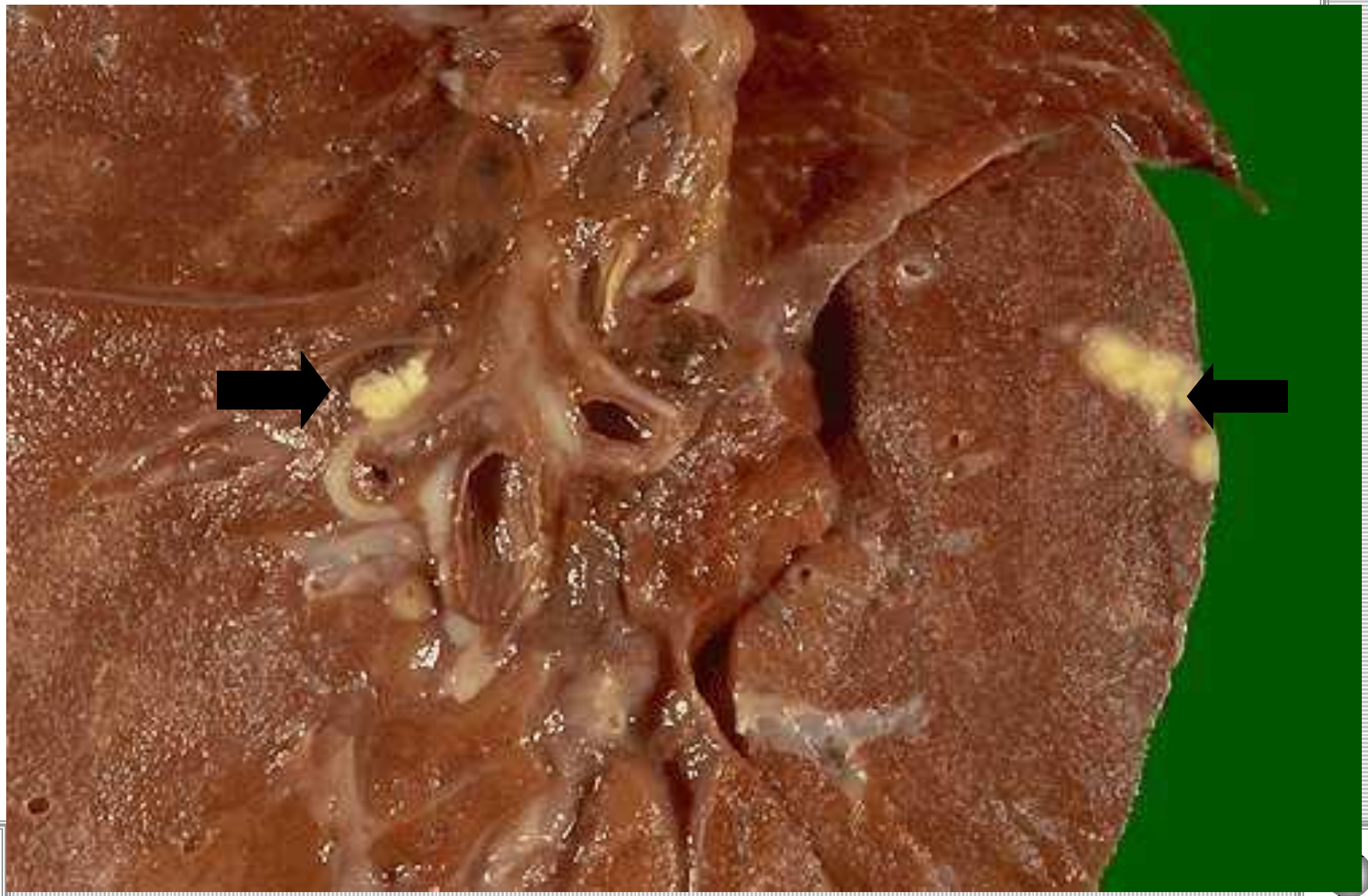
Most commonly males 30-50 y

Slowly Progressive (several months)

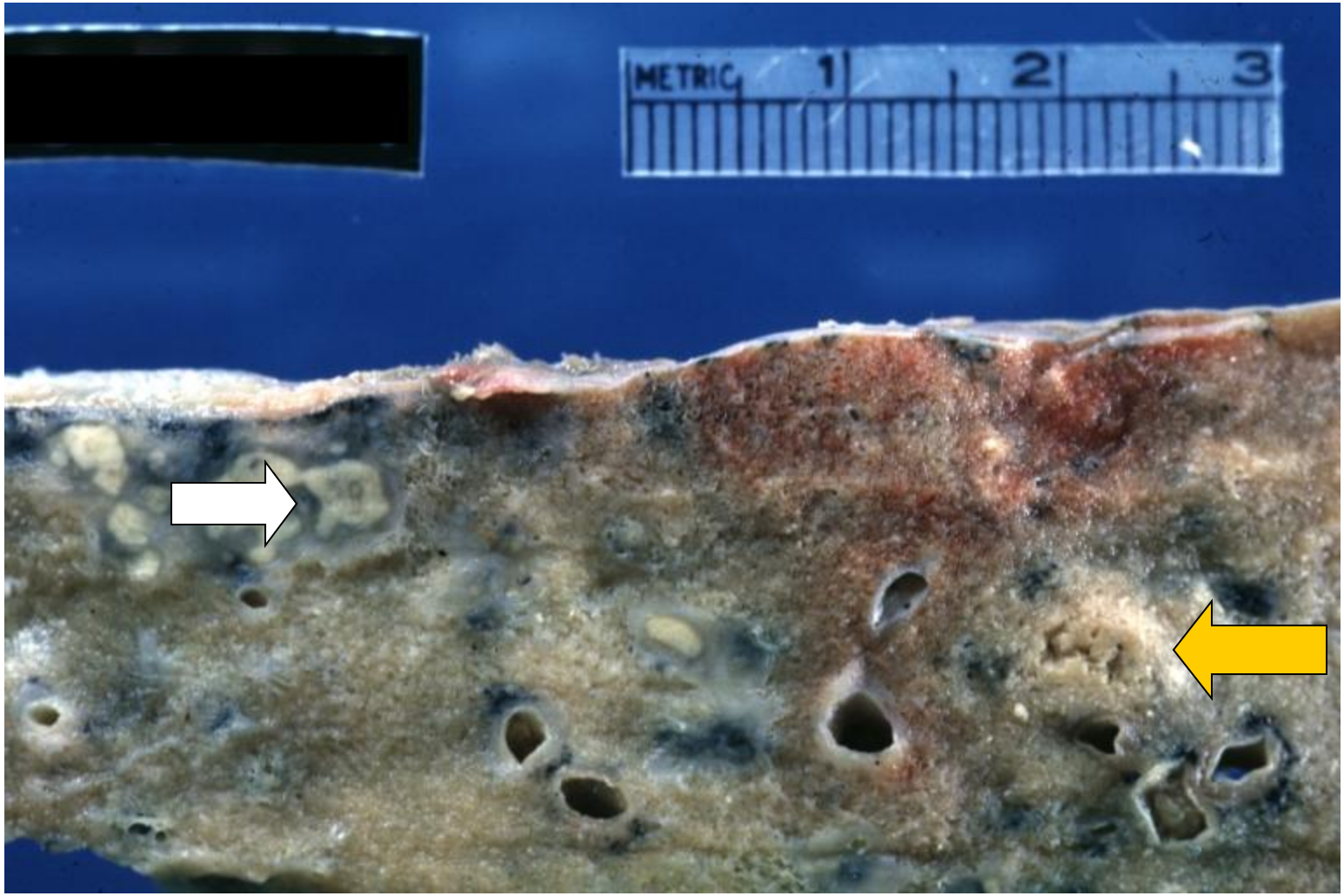
Cough, sputum, Low grade fever, night sweats, fatigue and weight loss.

Hemoptysis or pleuritic pain = severe disease

Ghon Complex



Typical cavitating granuloma



Miliary TB

Millet like – grain.

Extensive micro spread.

Through blood or bronchial spread

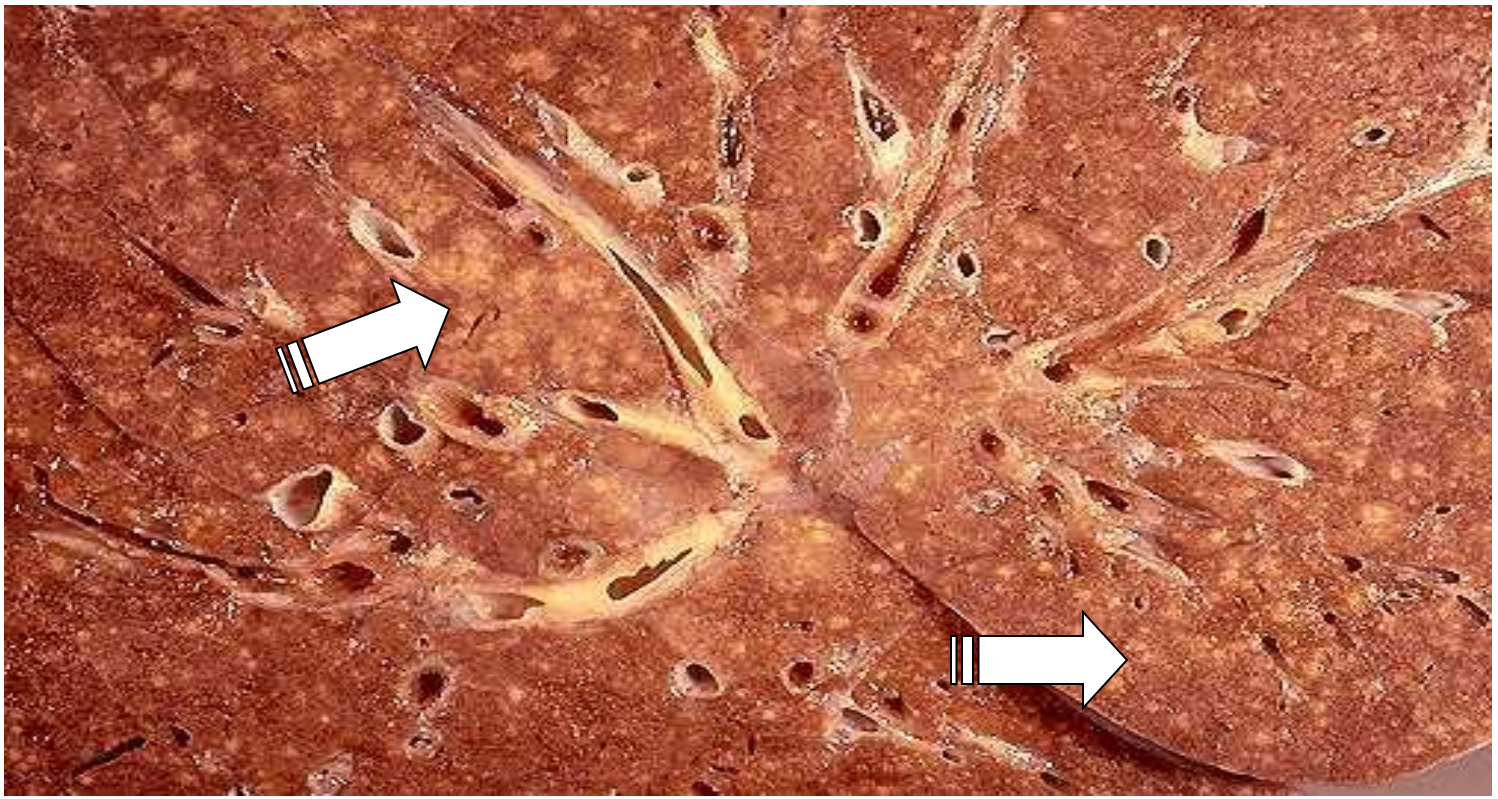
Low immunity

Pulmonary or Systemic types.

Miliary TB

Miliary spread TB

Miliary TB Lung

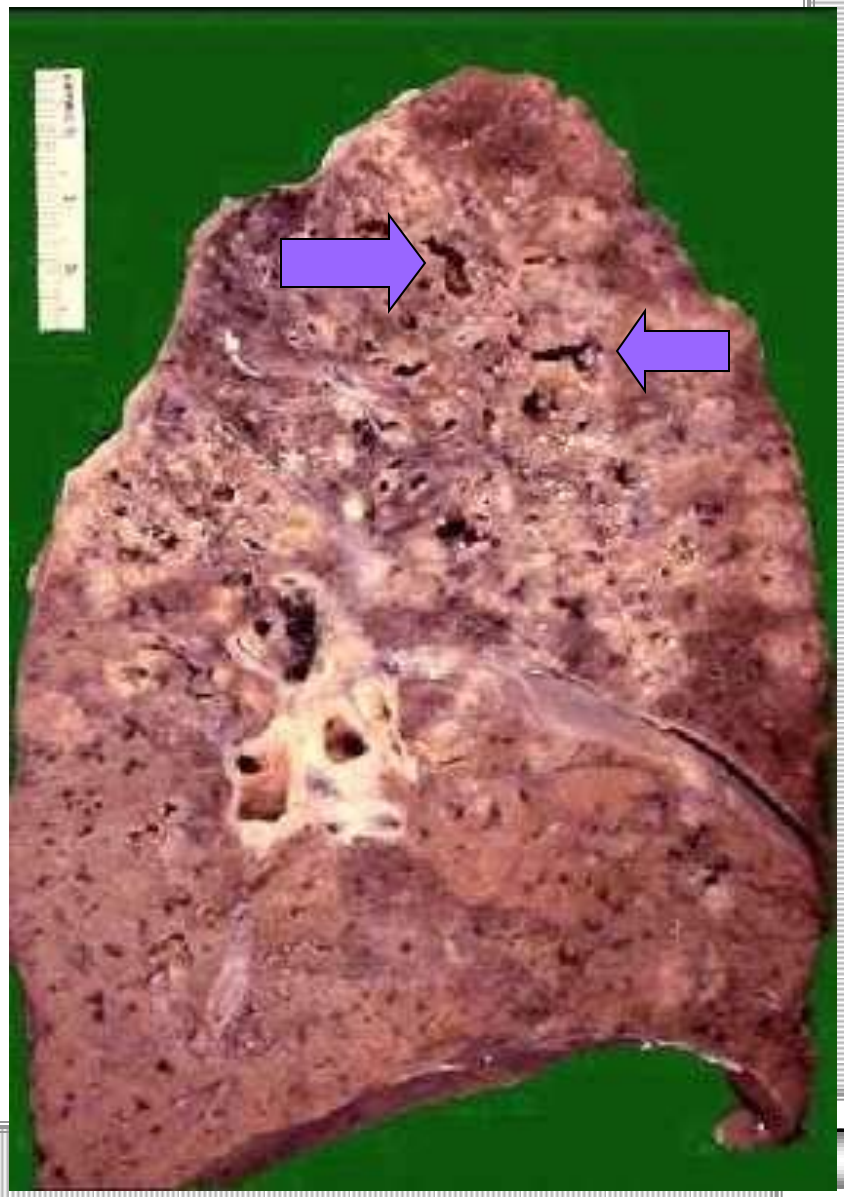


Cavitary Tuberculosis

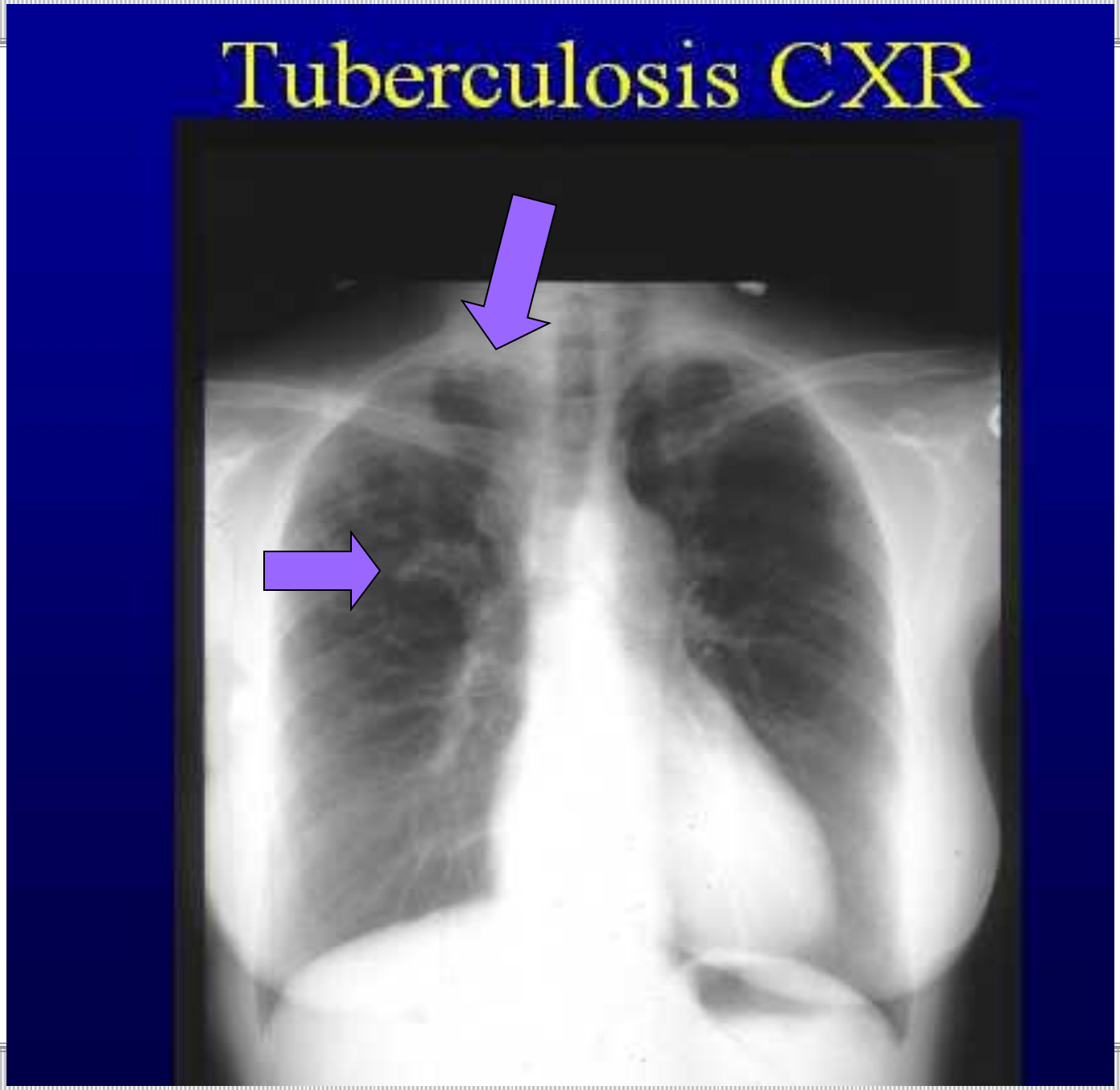
When necrotic tissue is coughed up → cavity.

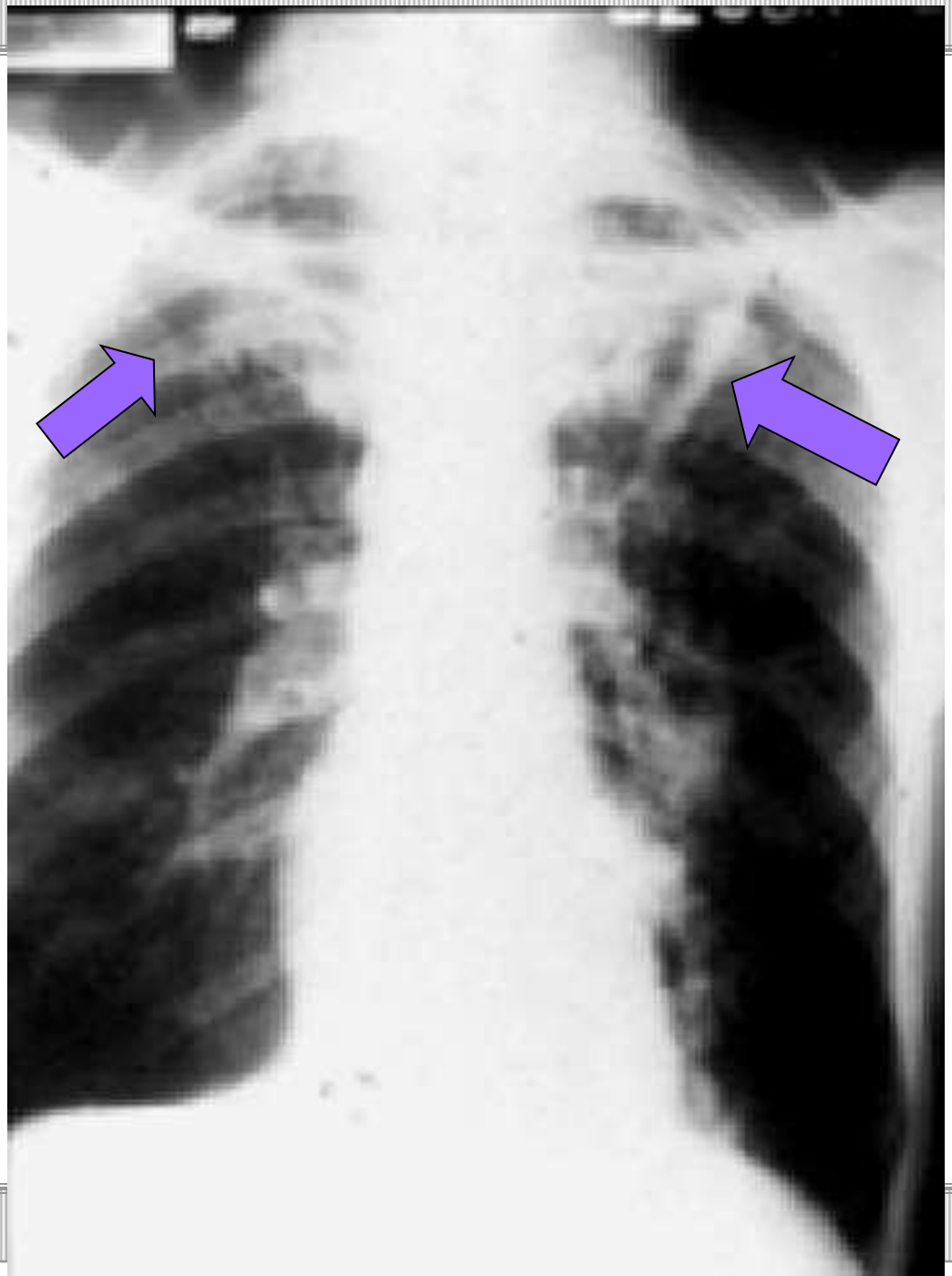
Cavitation is typical for large granulomas.

Cavitation is more common in the secondary reactivation tuberculosis - upper lobes.

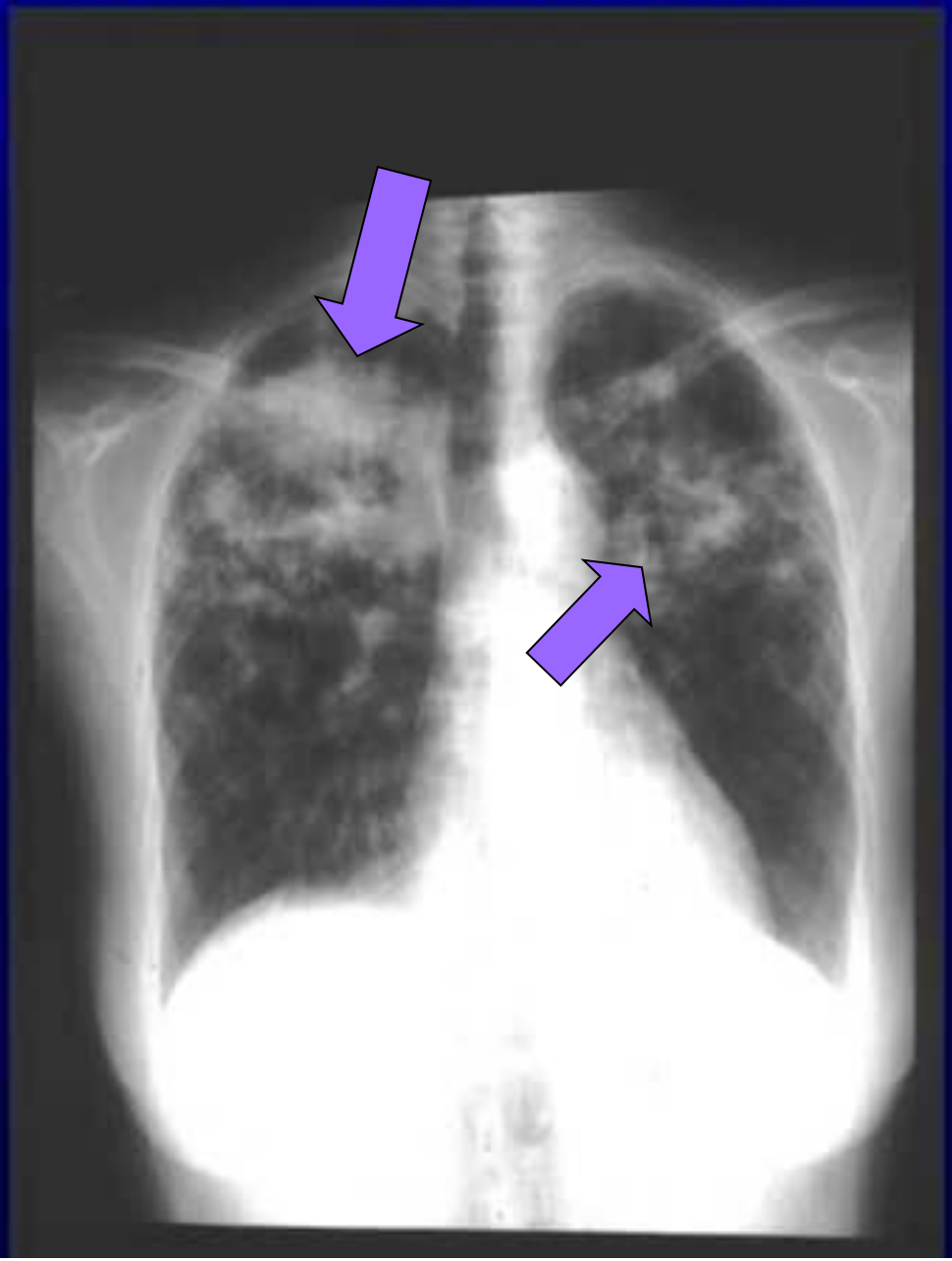


Tuberculosis CXR

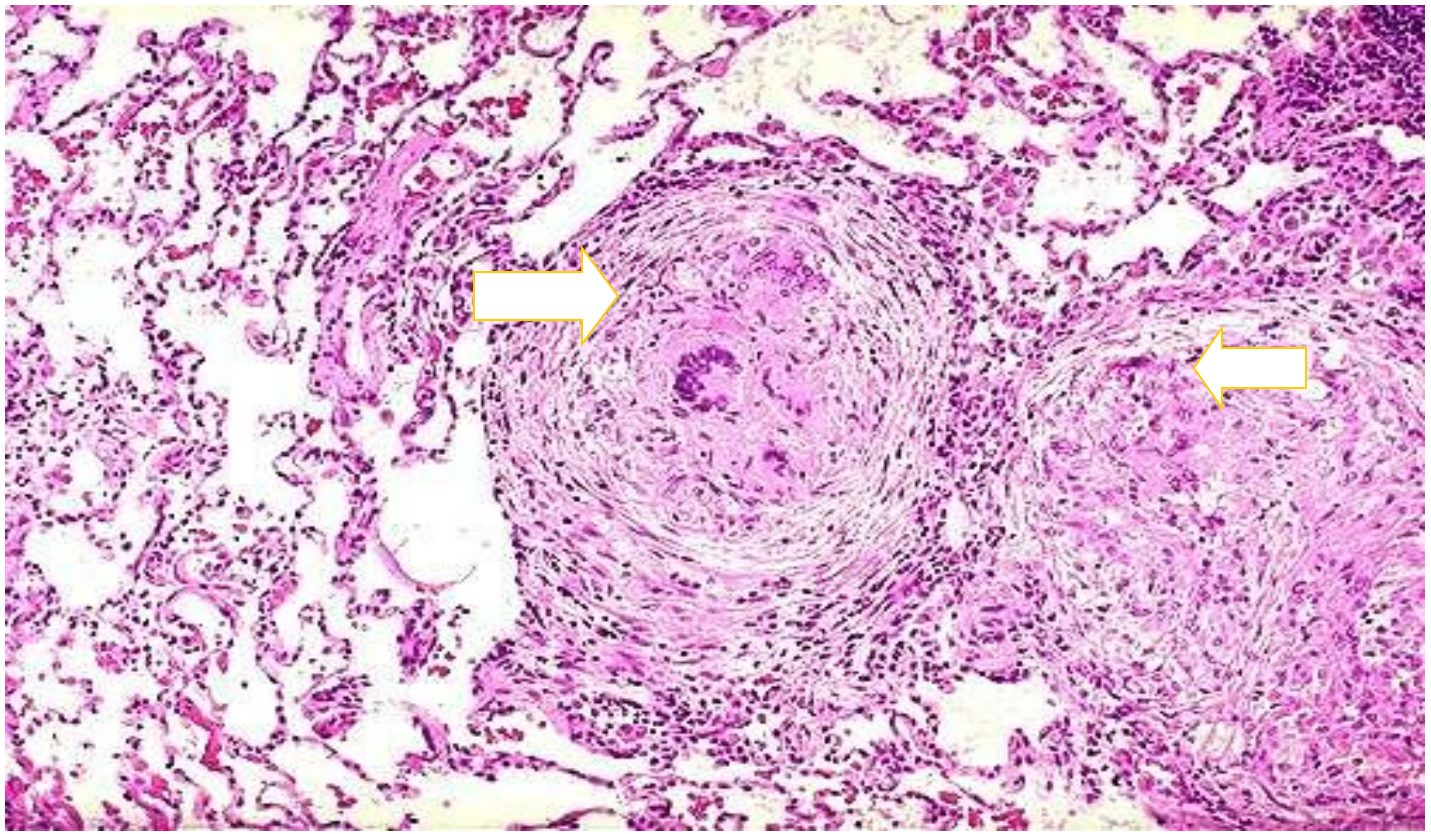




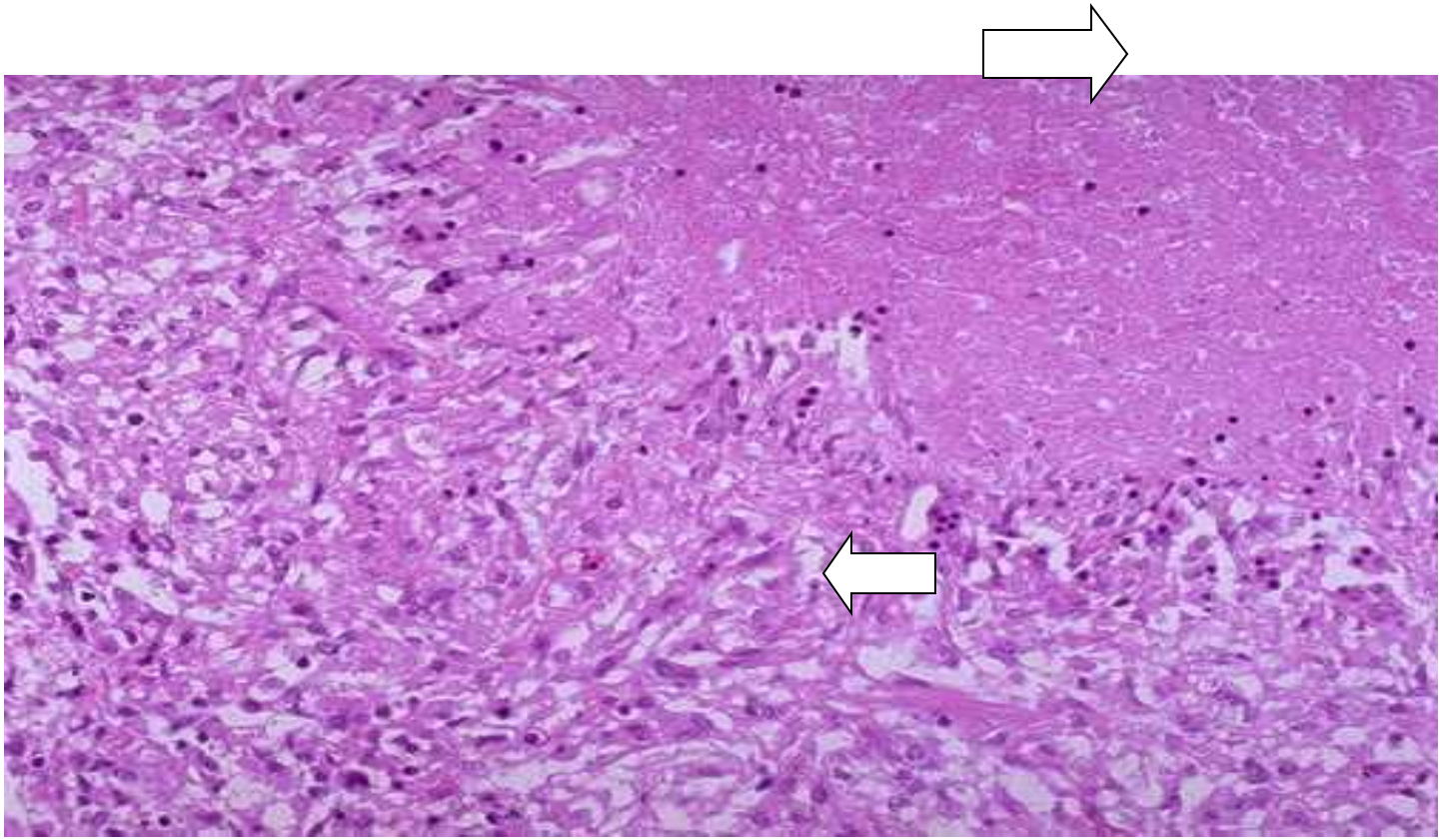
Untreated Tuberculosis CXR



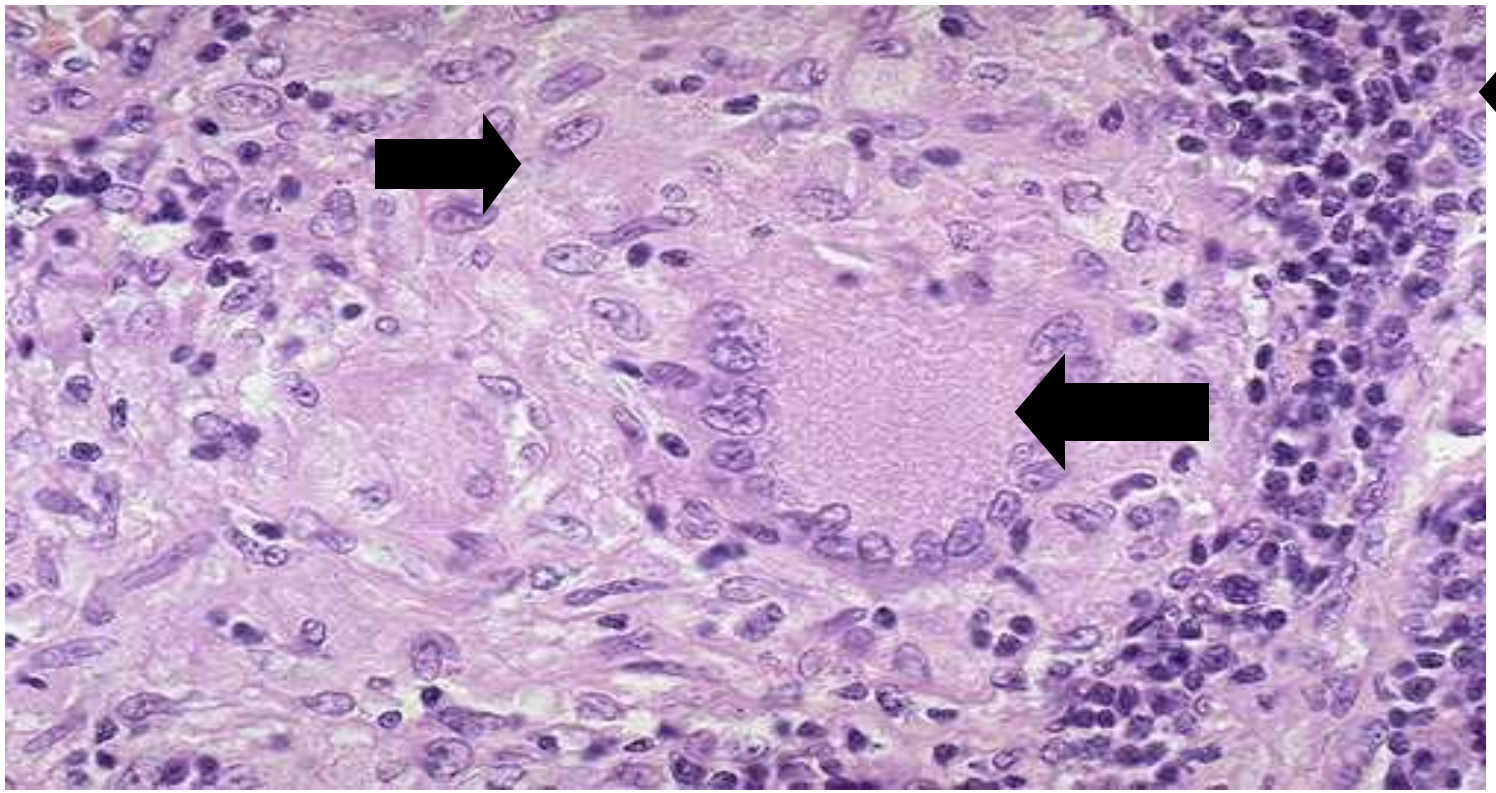
Tuberculous Granulomas



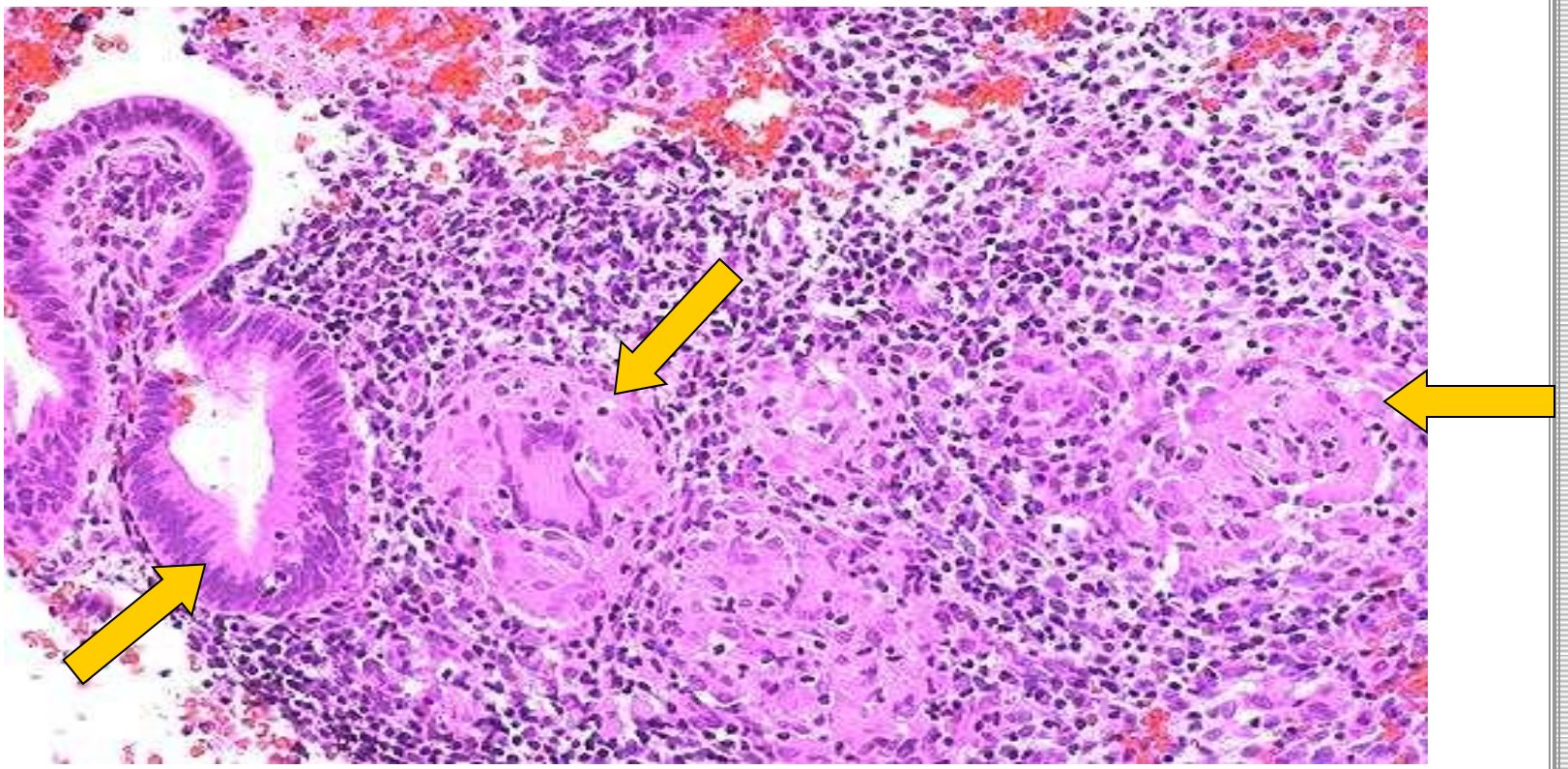
Caseation Necrosis



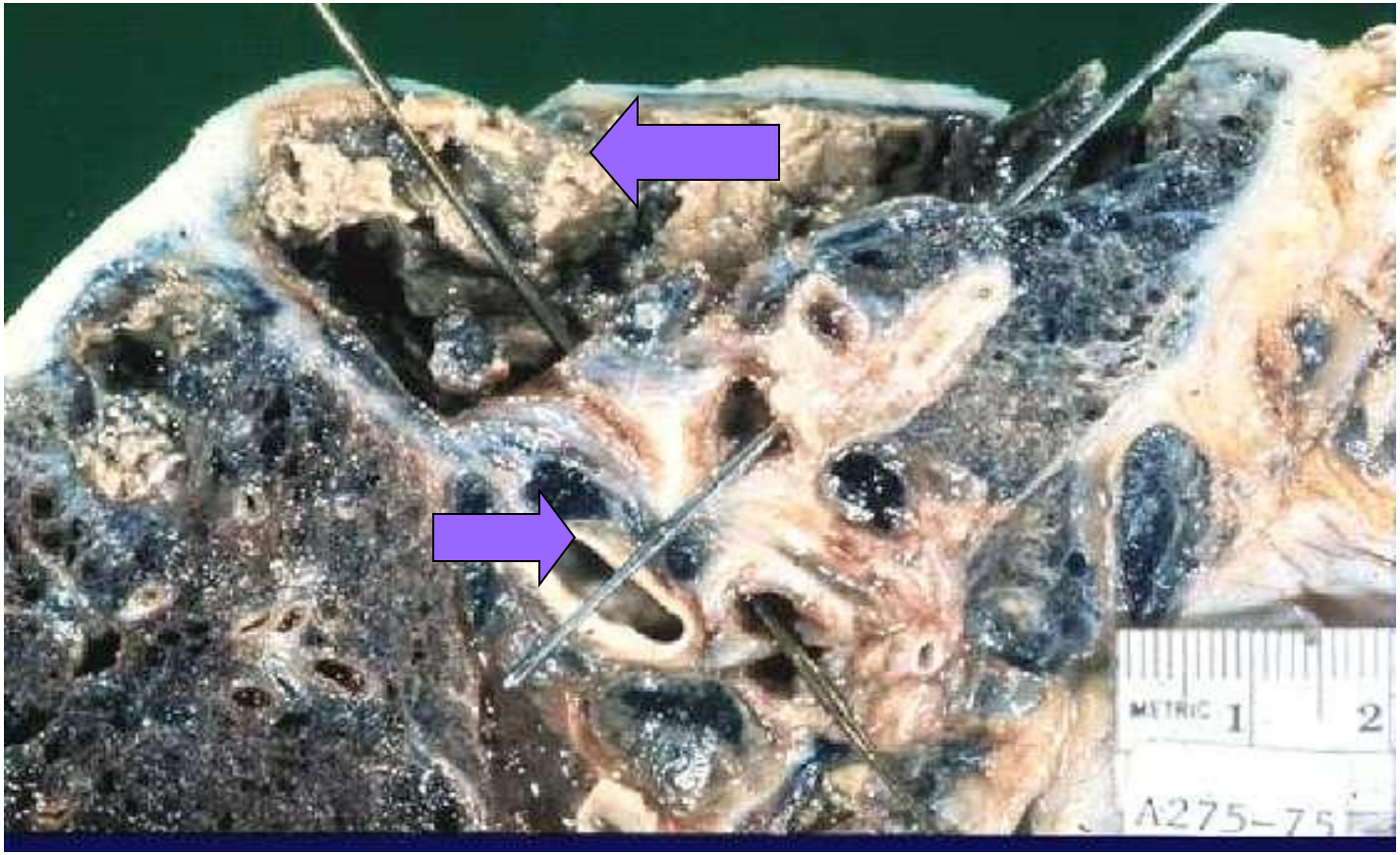
Epitheloid cells in Granuloma



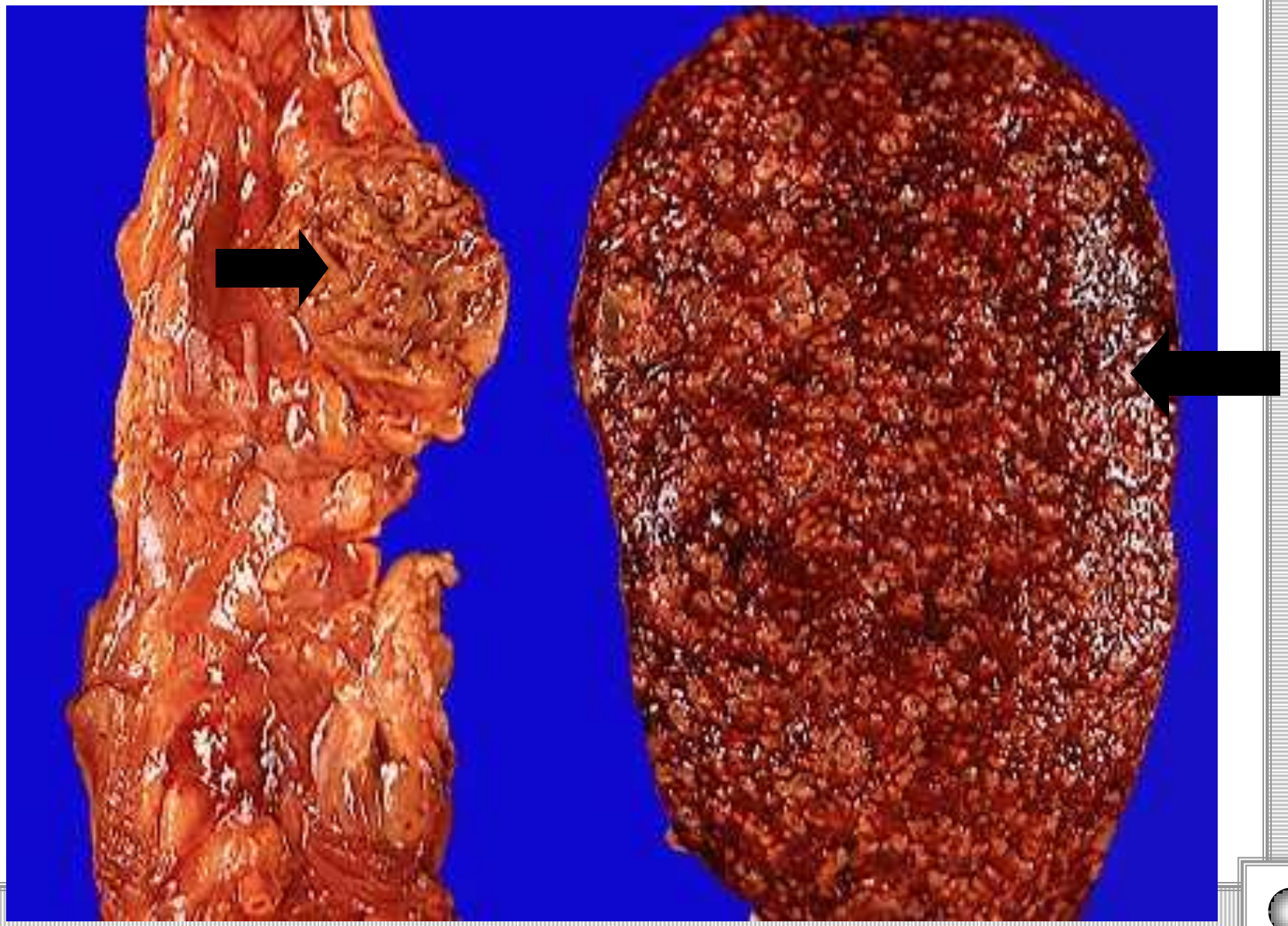
Cells in Granuloma



Cavitary Secondary TB



Systemic Miliary TB



Extra-pulmonary TB

a-TB Pleural effusion

b- Tuberculous meningitis

c- Millary tuberculosis.

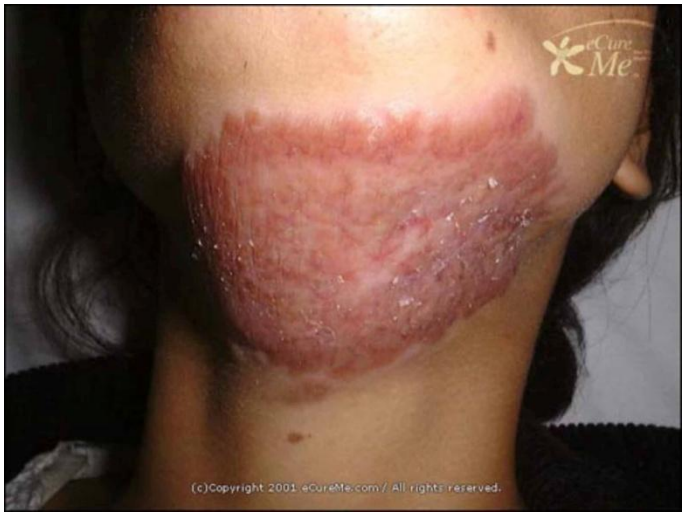
d- Renal & urogenital tuberculosis.

e- Bone &joint tuberculosis.

f- TB entritis.

However, the TB bacterium can also Infect many other organs, causing distinct “diseases

Renal TB

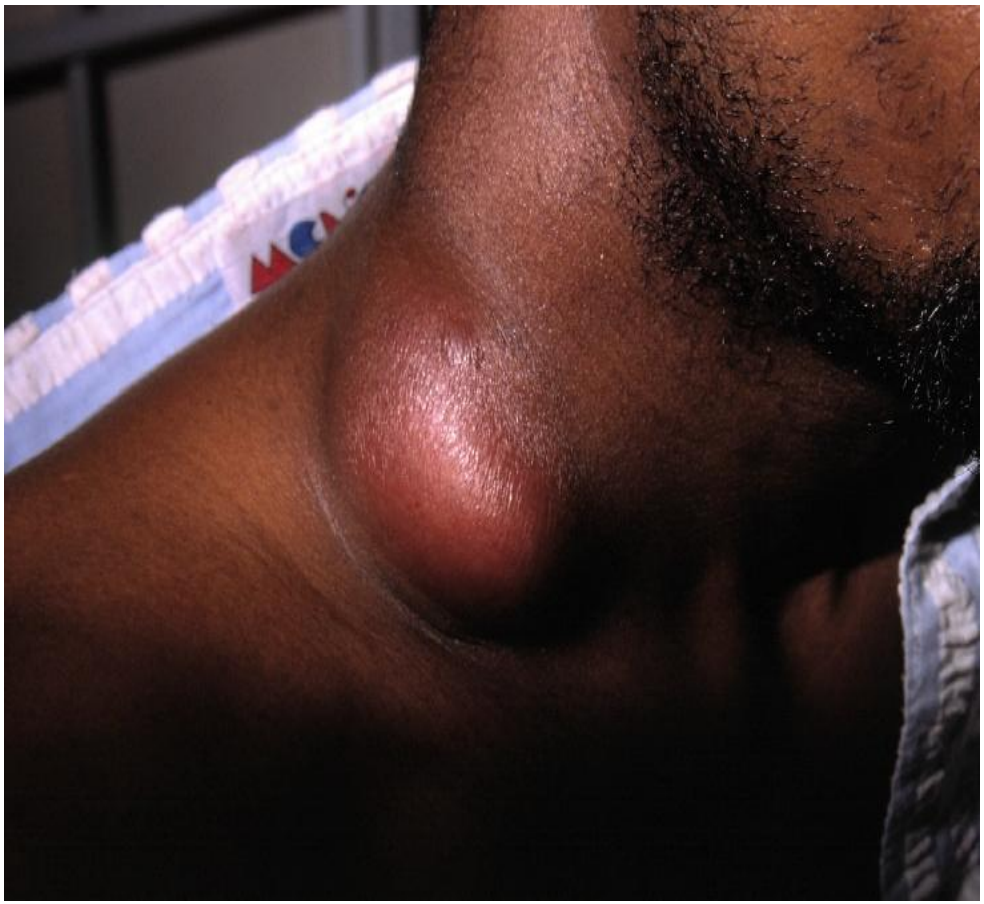


Lupus vulgaris

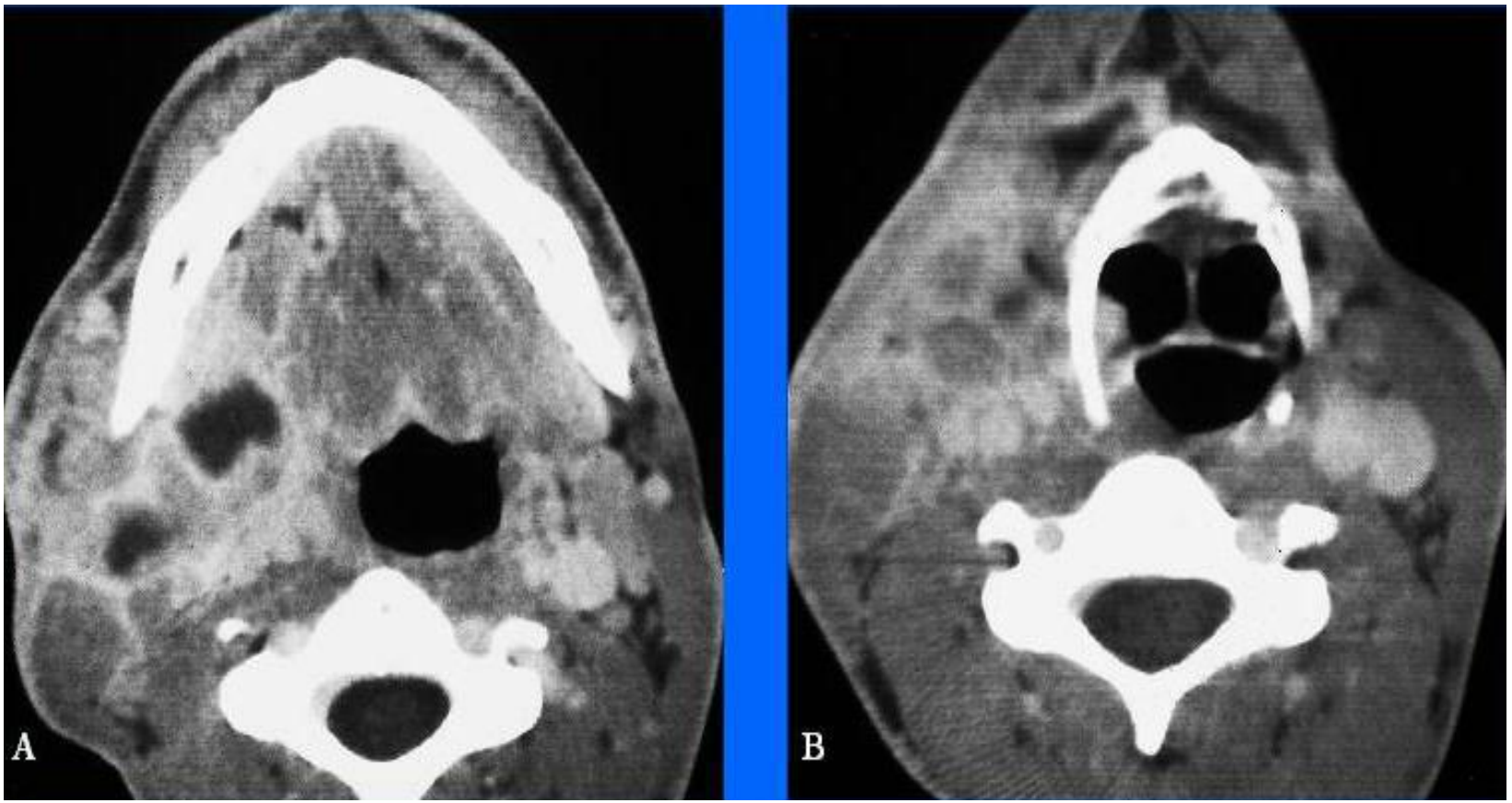
http://www.ecureme.com/atlas/data/Tuberculosis_of_Skin550_ab.htm

Skin and soft tissue

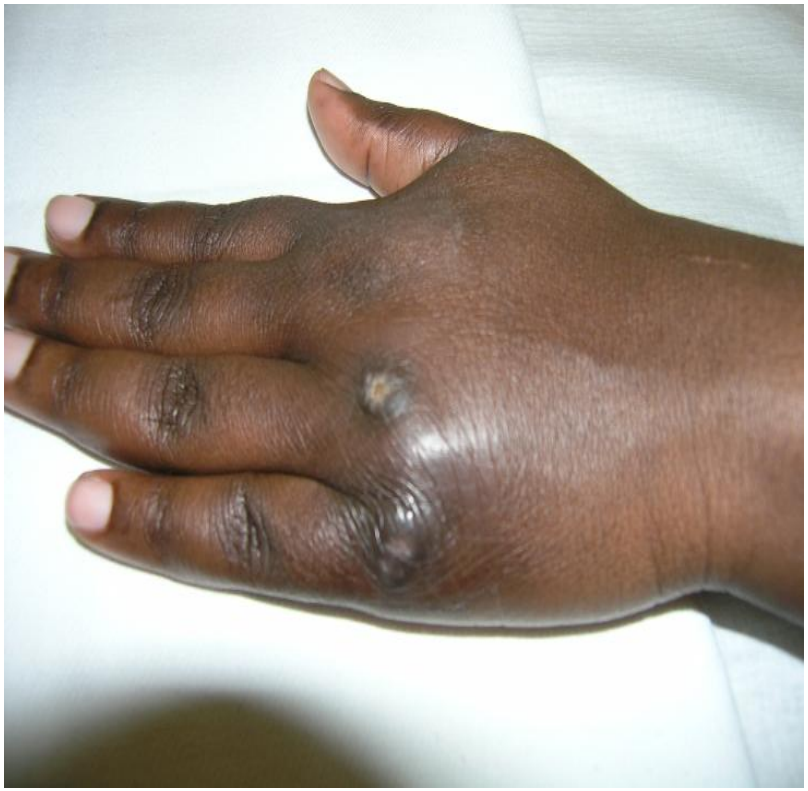
25 male African. Expanding non painful lesion in neck - Cervical lymph node TB progressing to abscess (beware deep extension – collar stud abscess)

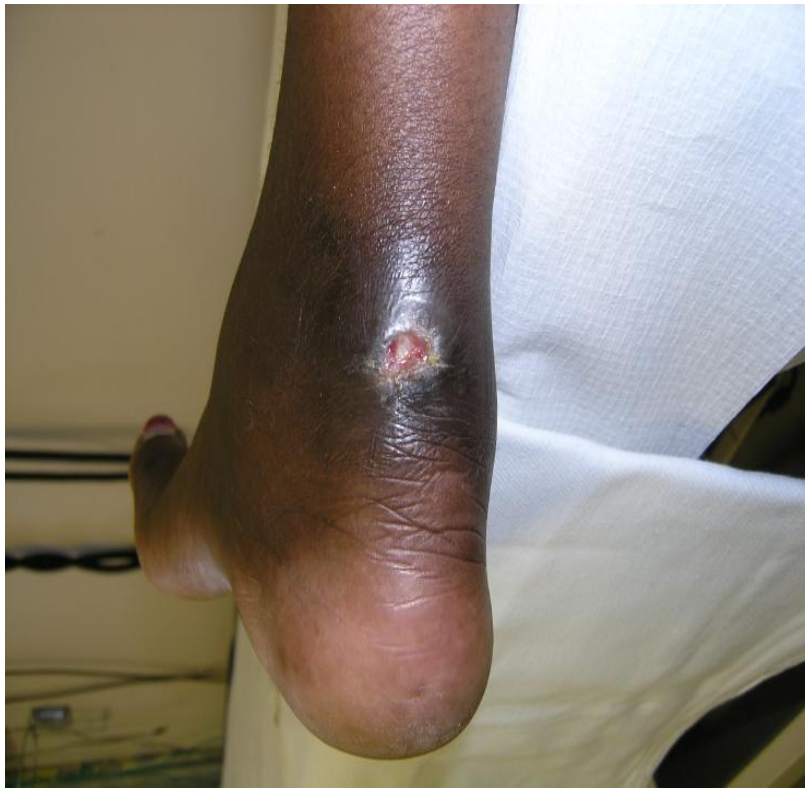


TB node in neck with deep extension



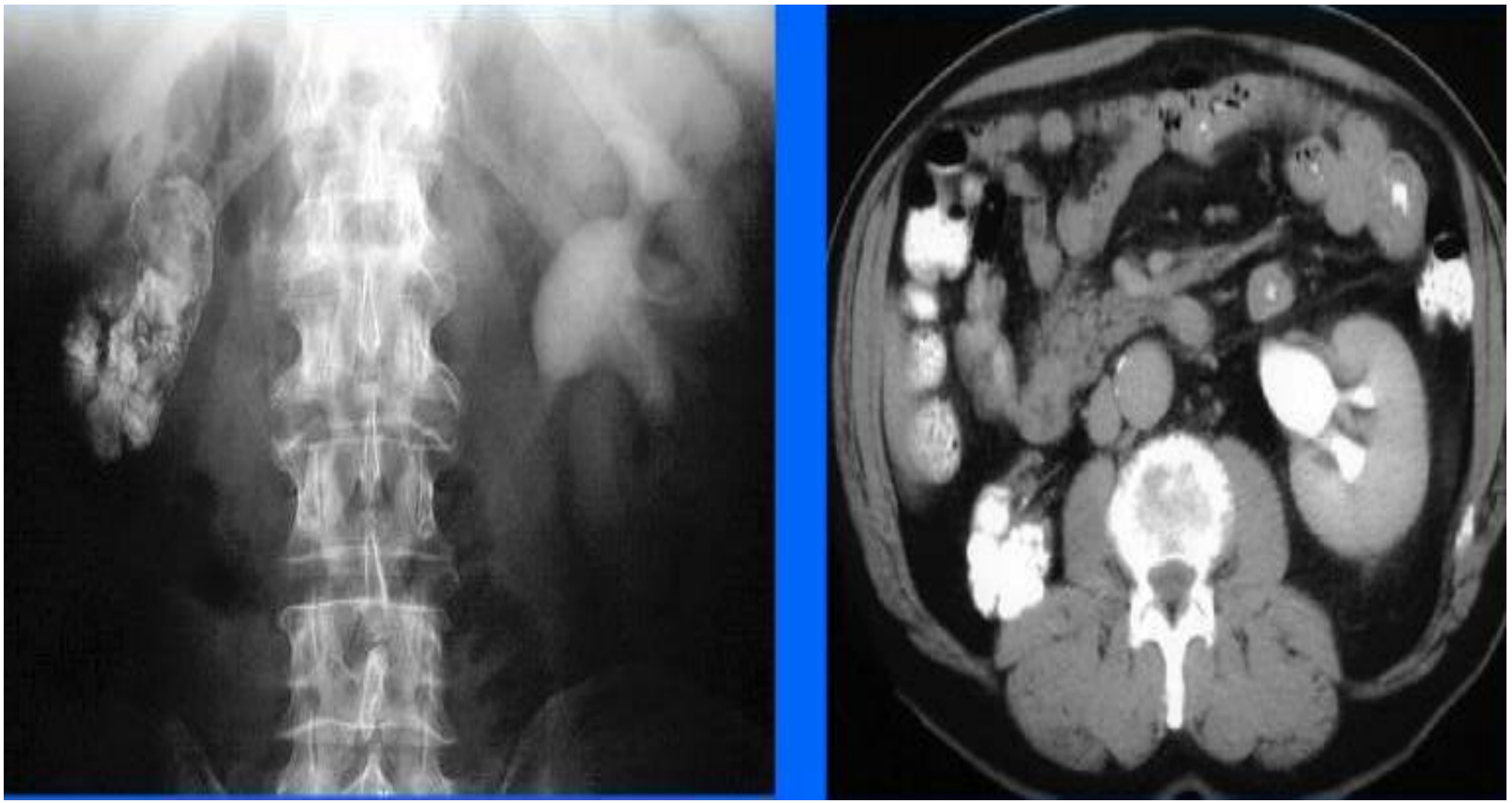
35 female African – systemically well - hand and foot lesions present for 6 months – MTB grown on biopsy by plastic surgeons (HIV neg)



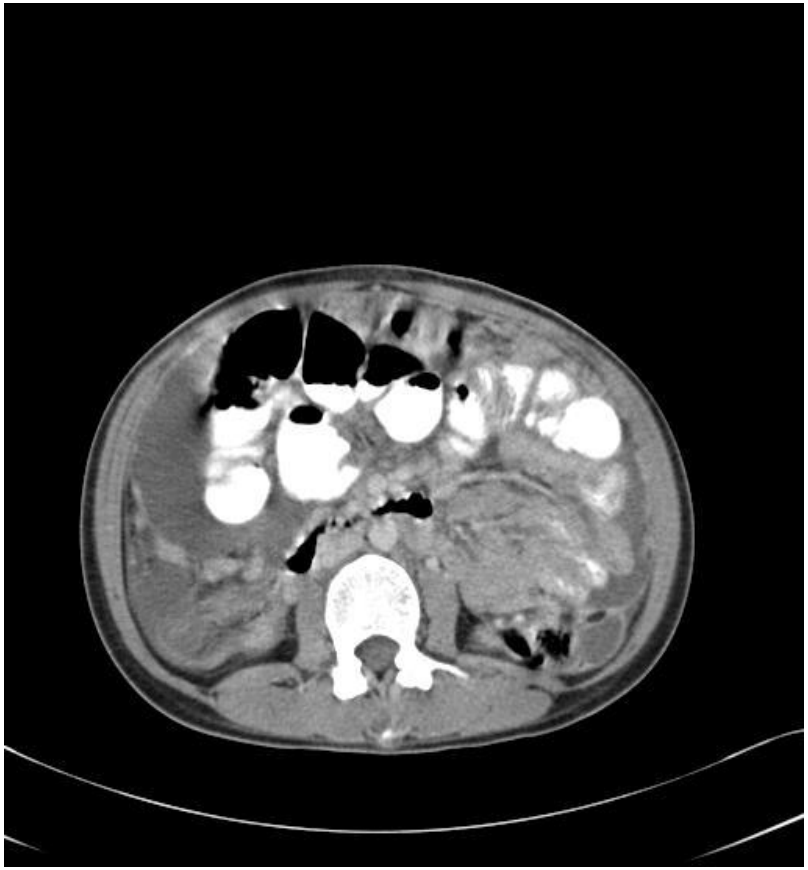


Abdominal Tuberculosis

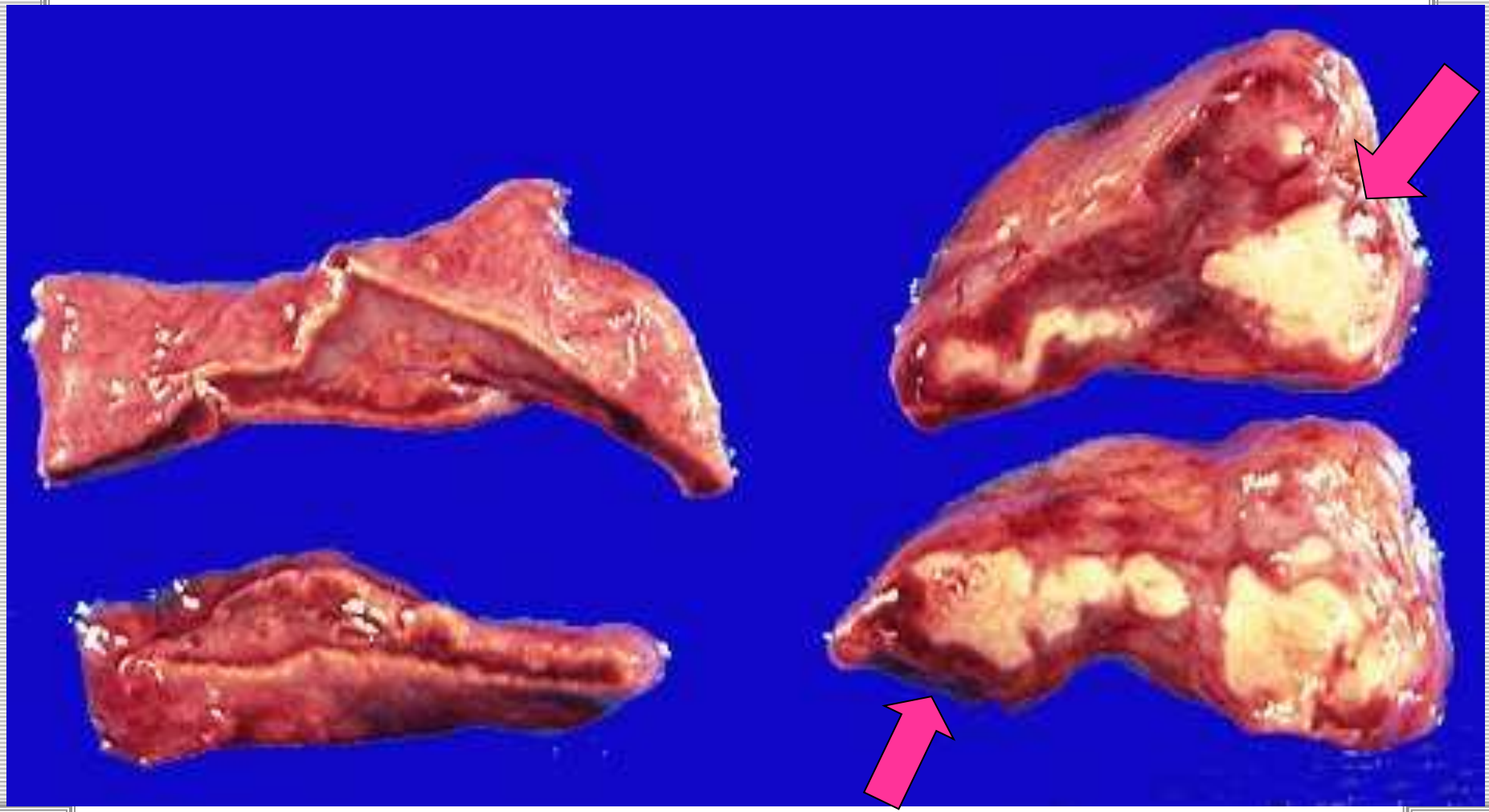
Renal tuberculosis (may have few or no symptoms) leading to autonephrectomy



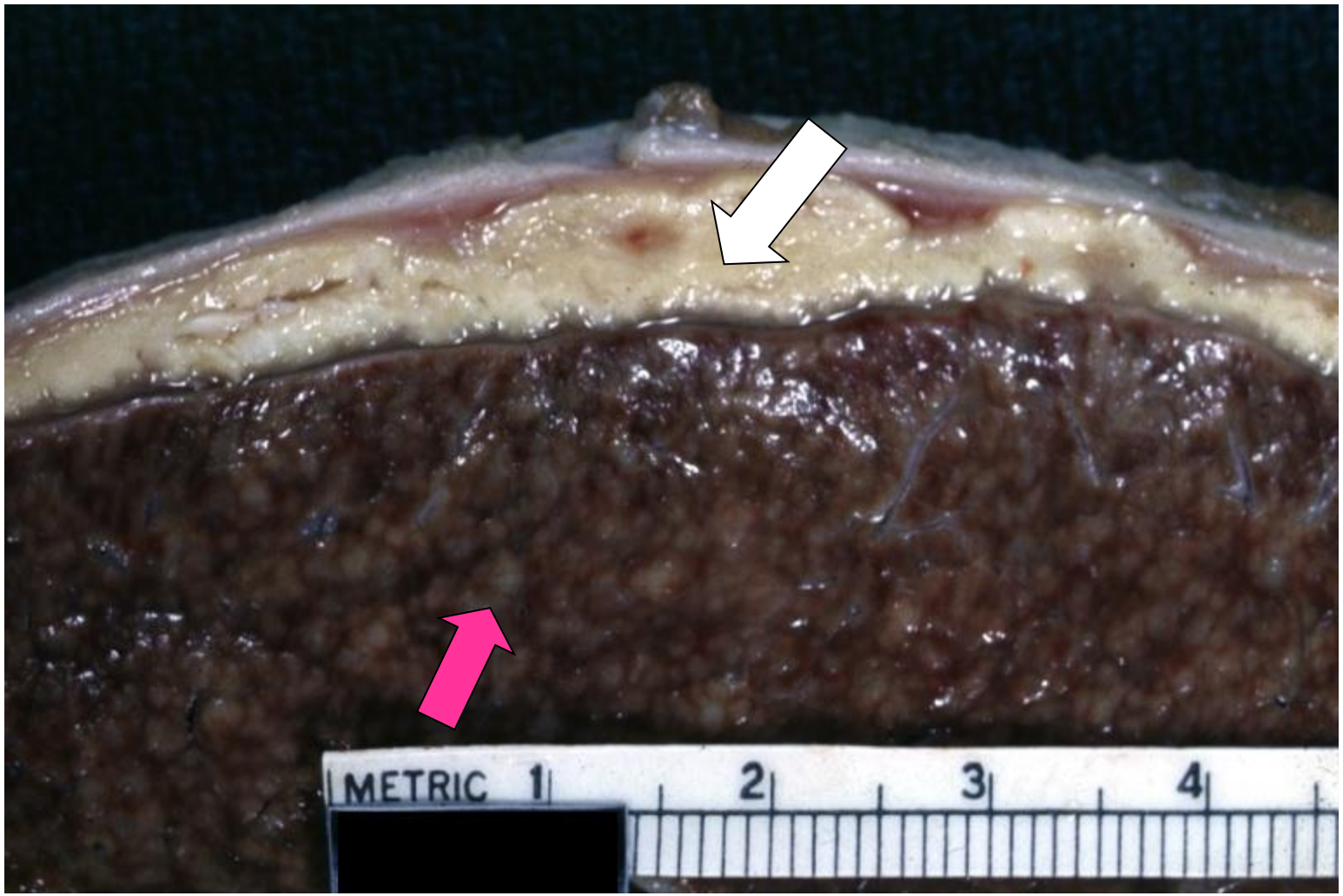
30 middle eastern asylum seeker - abdo pain, fever, sweats – CT scan - peritoneal TB confirmed on biopsy – may mimic malignancy



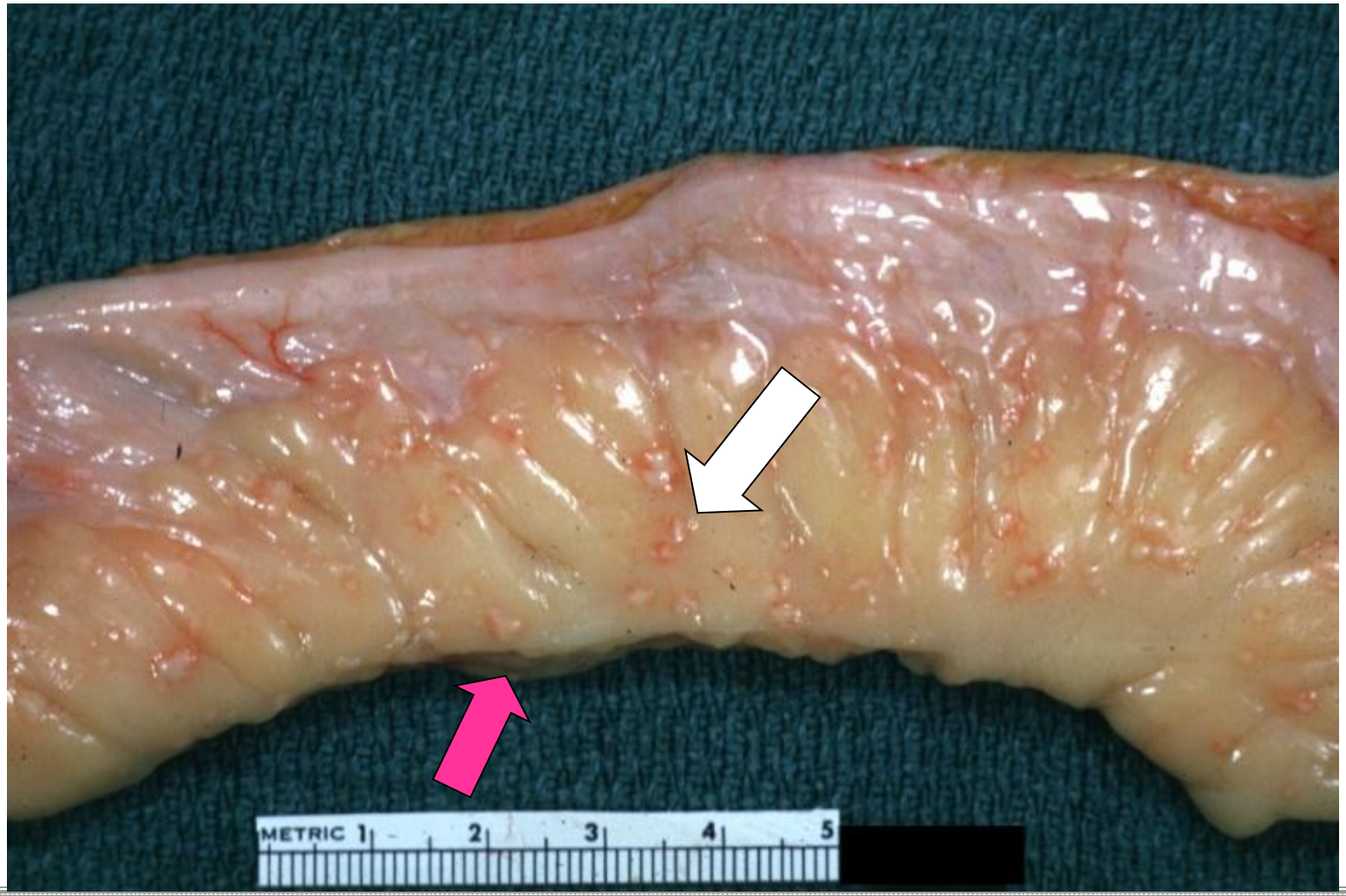
Adrenal TB - Addison Disease



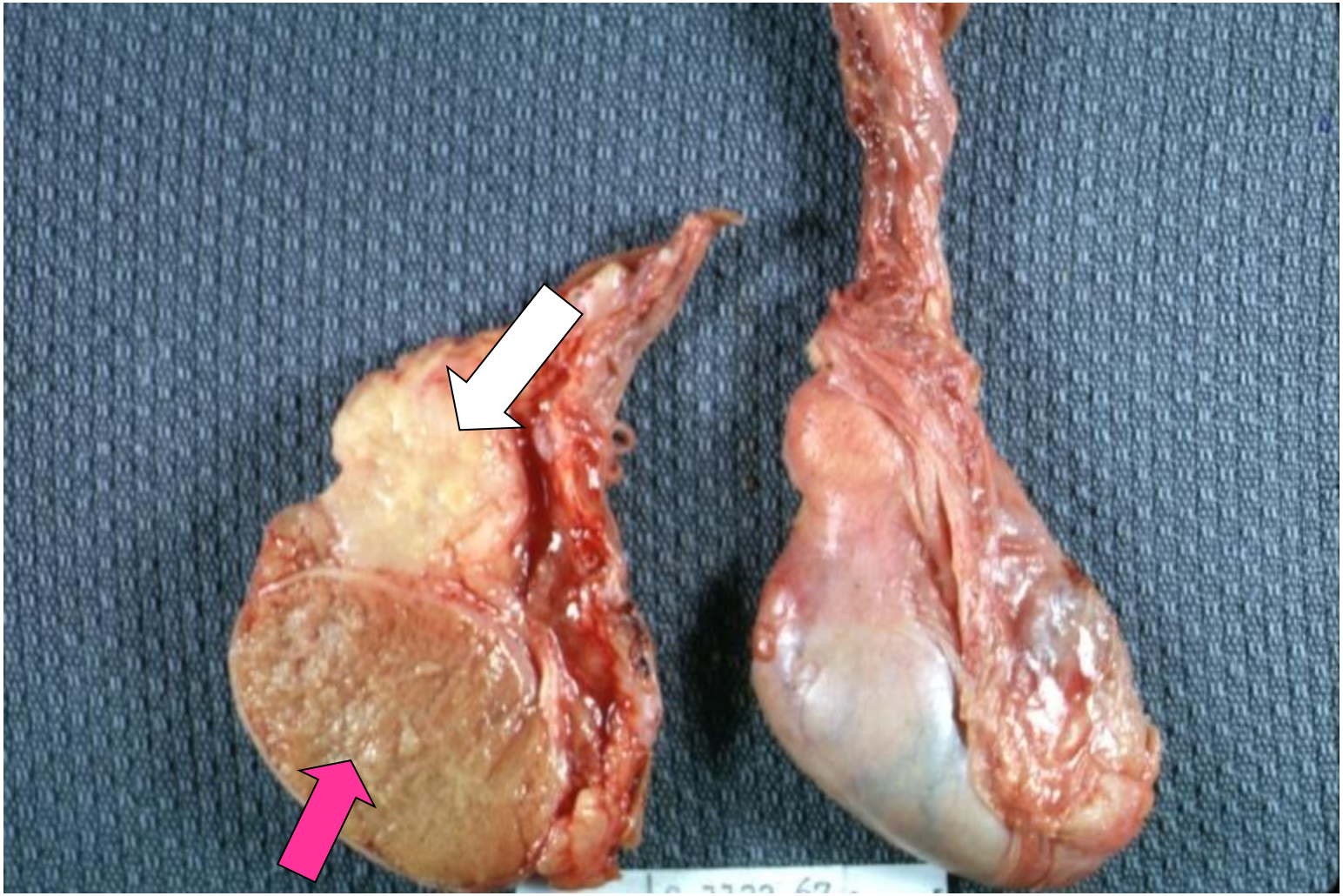
TB Peritonitis + liver Miliary TB



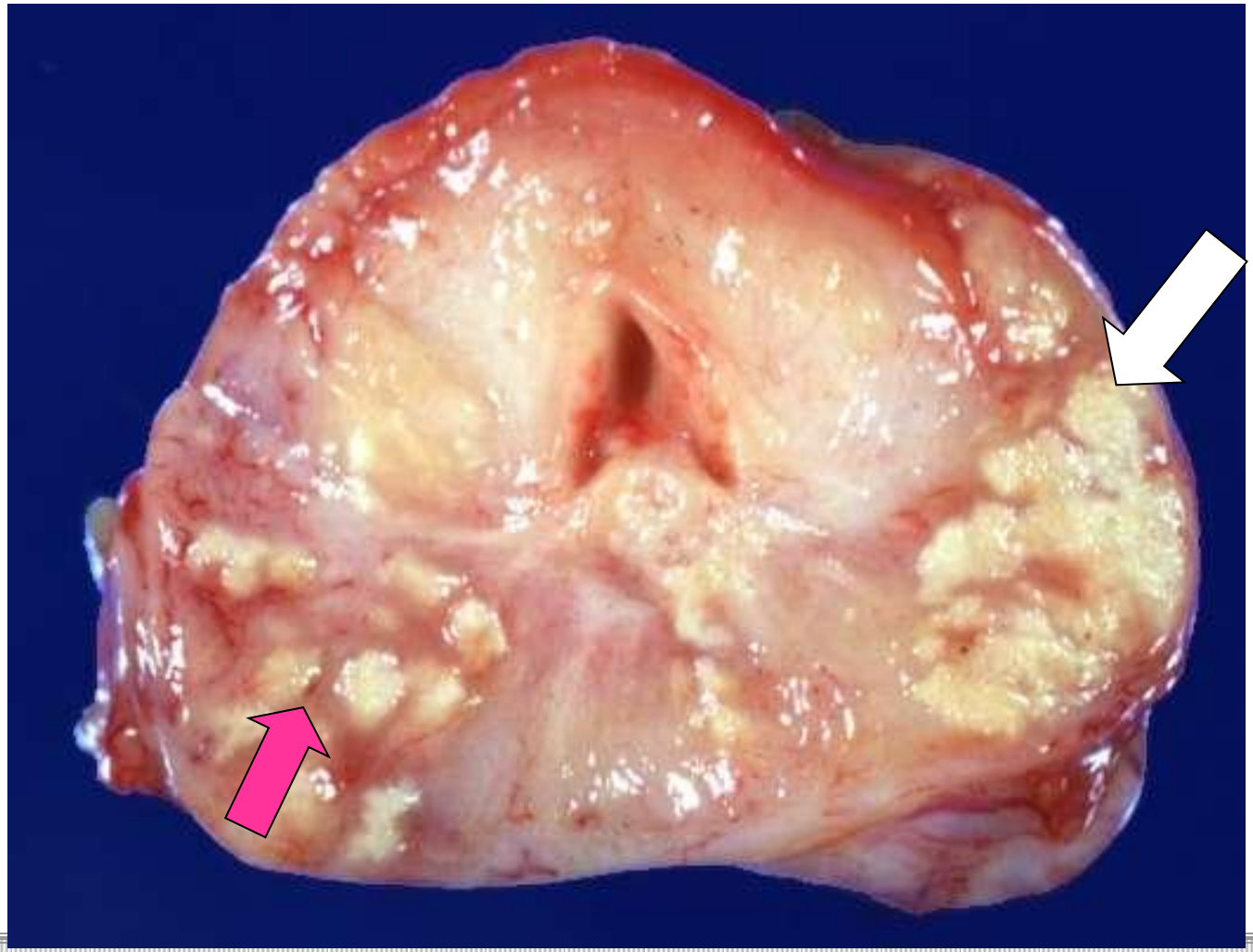
TB Intestine



Testes TB Orchitis.

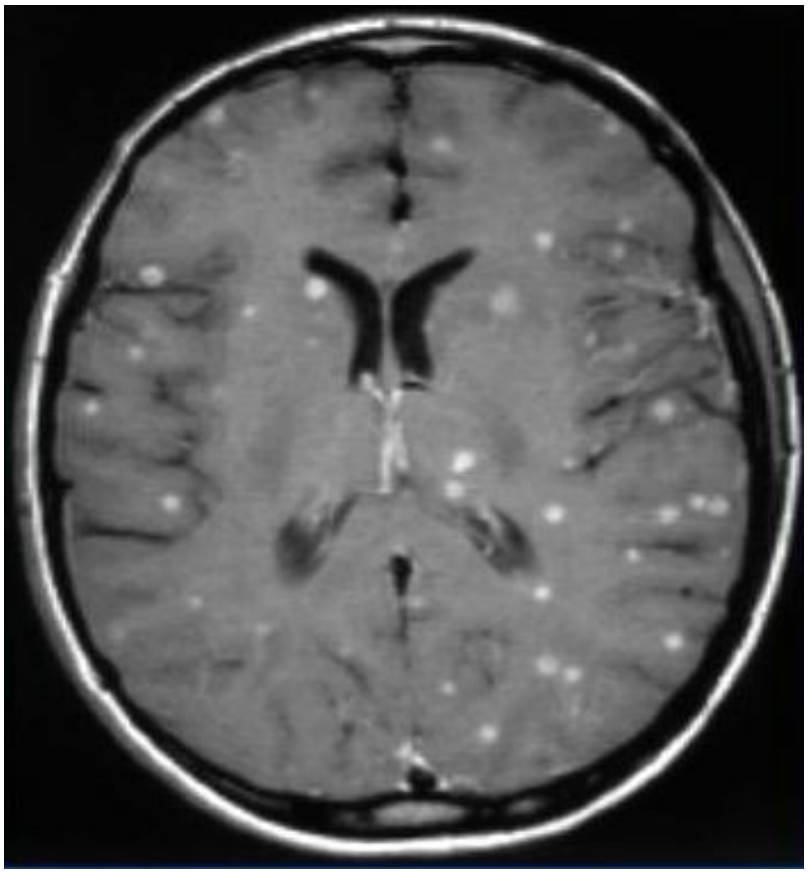


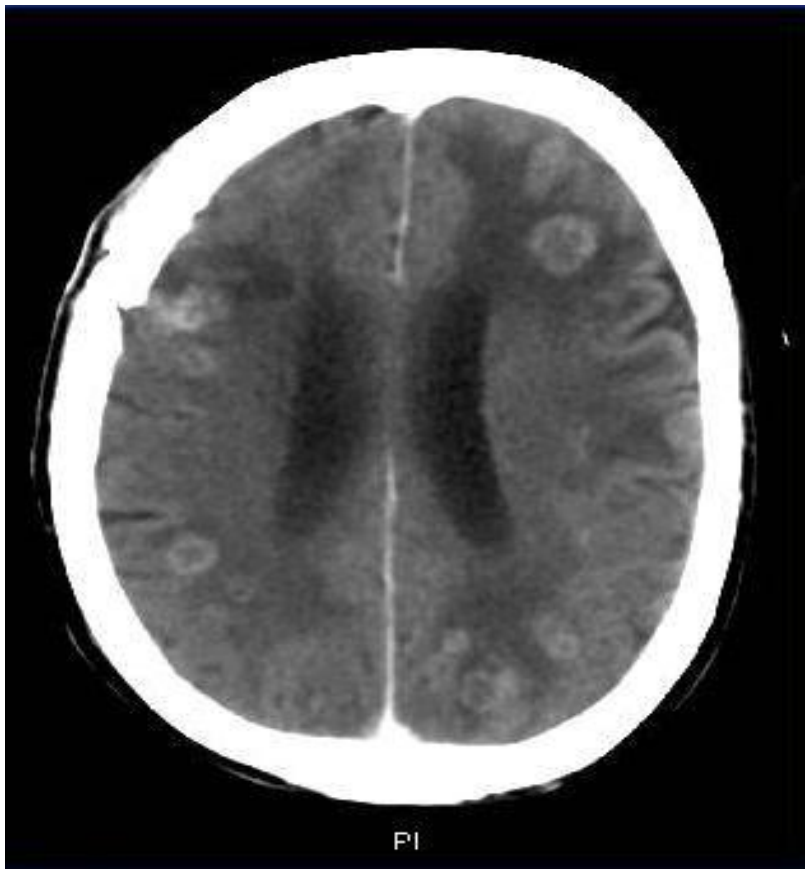
Prostate TB



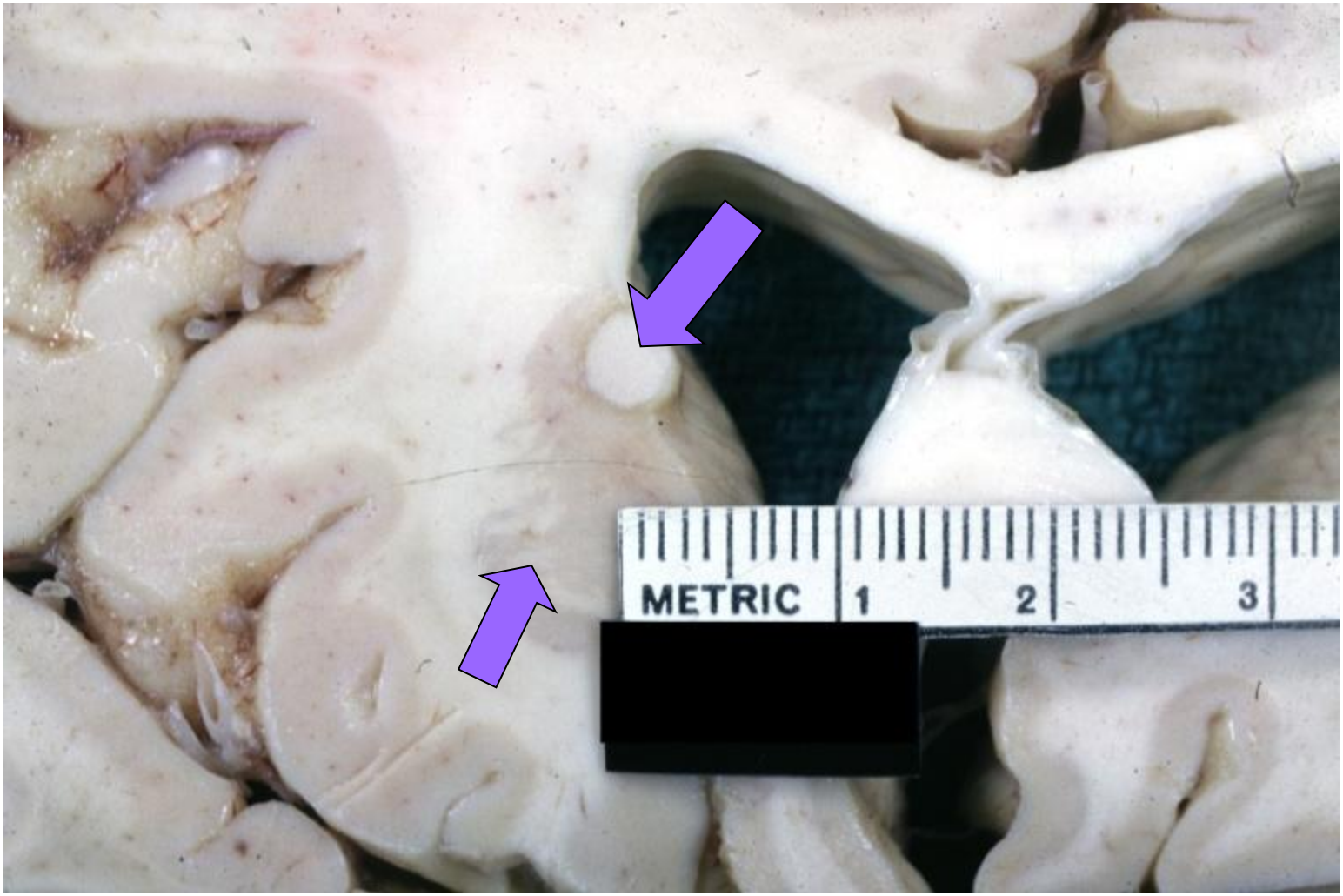
Intracranial TB

miliary TB on MRI scan
tuberclomas on CT scan

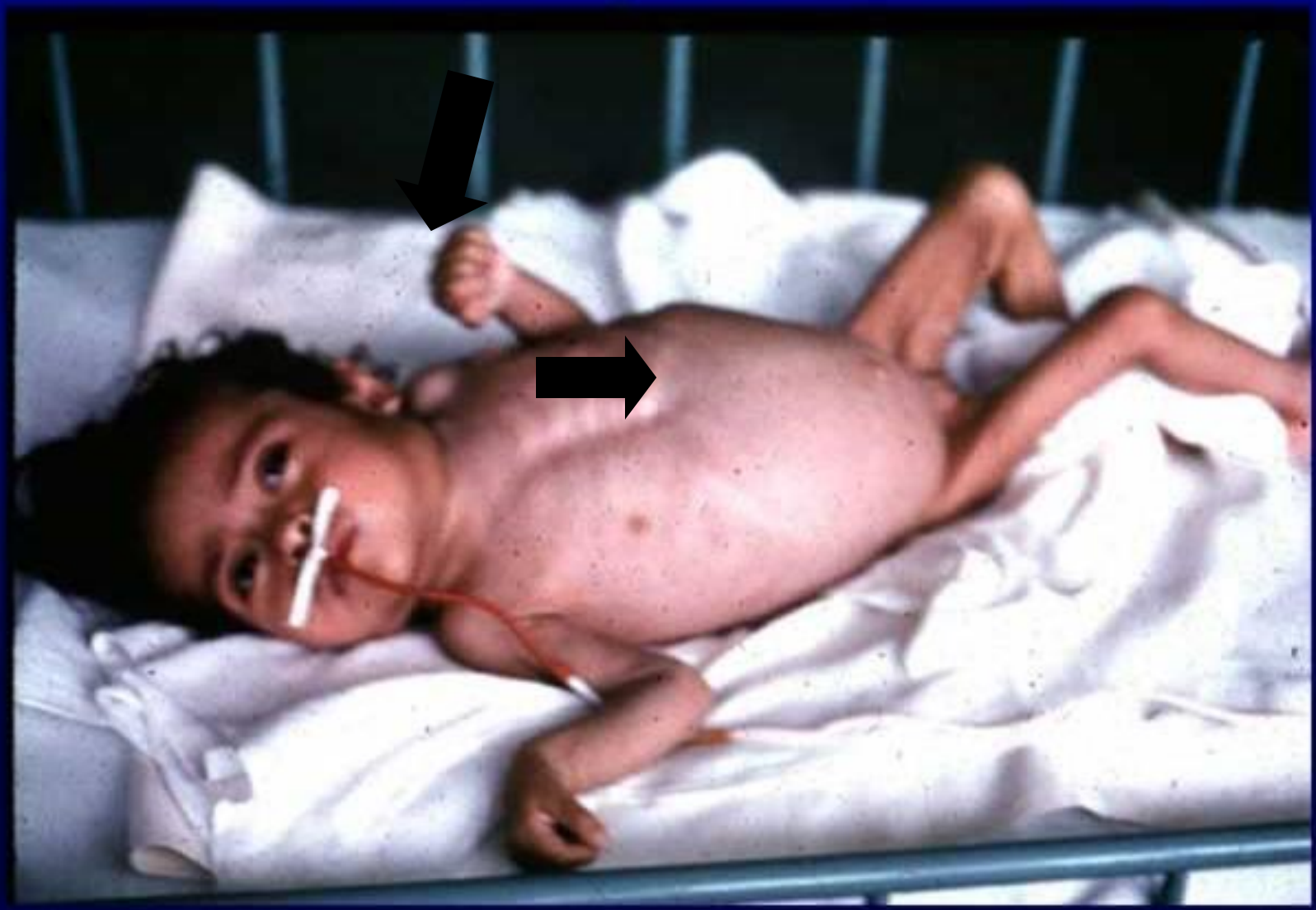




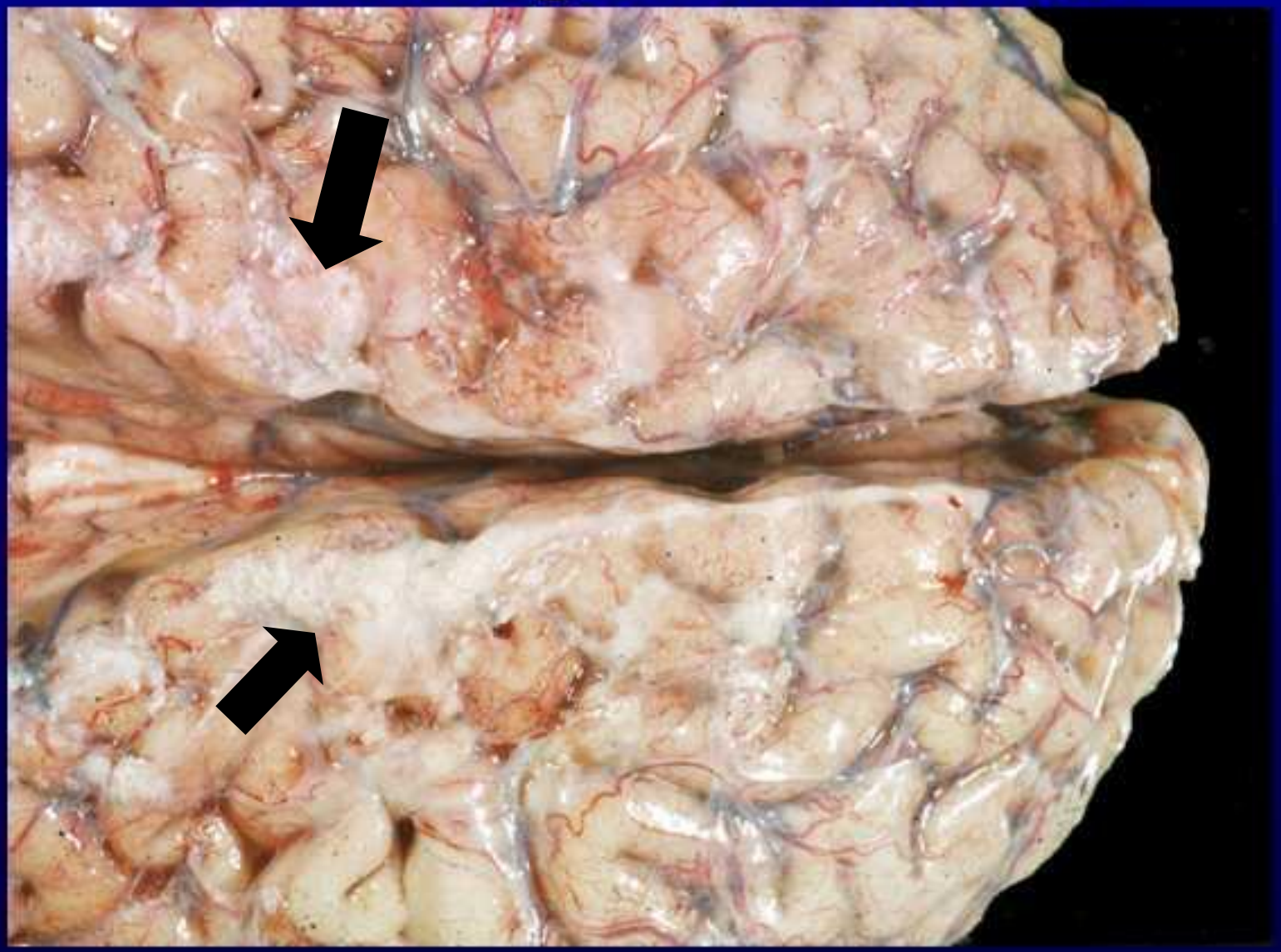
TB Brain – Caudate n.



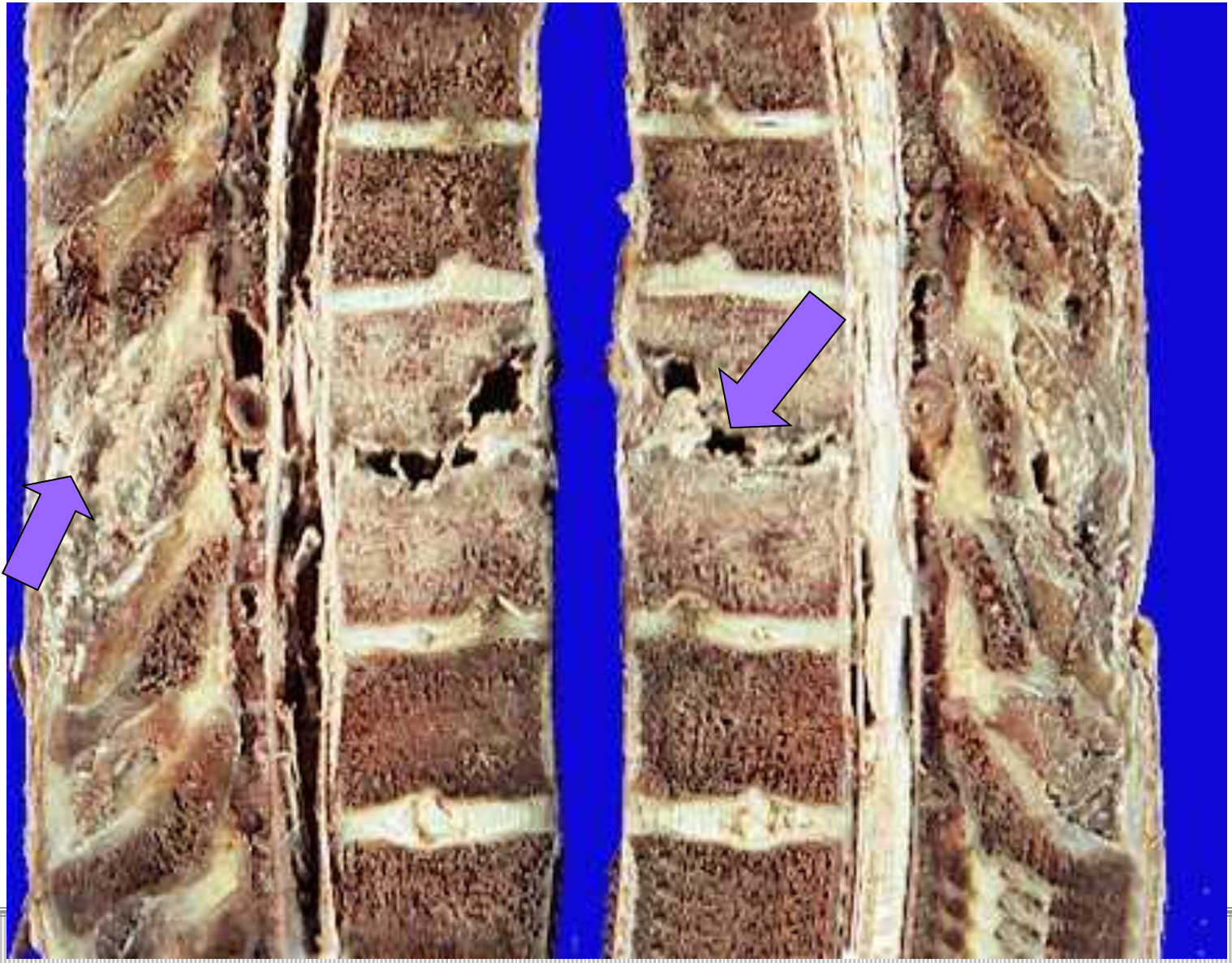
TB Meningitis Patient



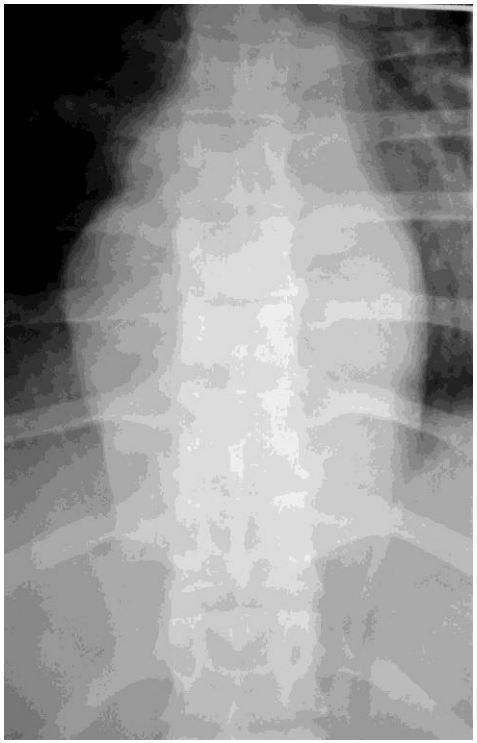
TB Meningitis Gross Brain



Spinal TB - Potts Disease



TB infecting the spine

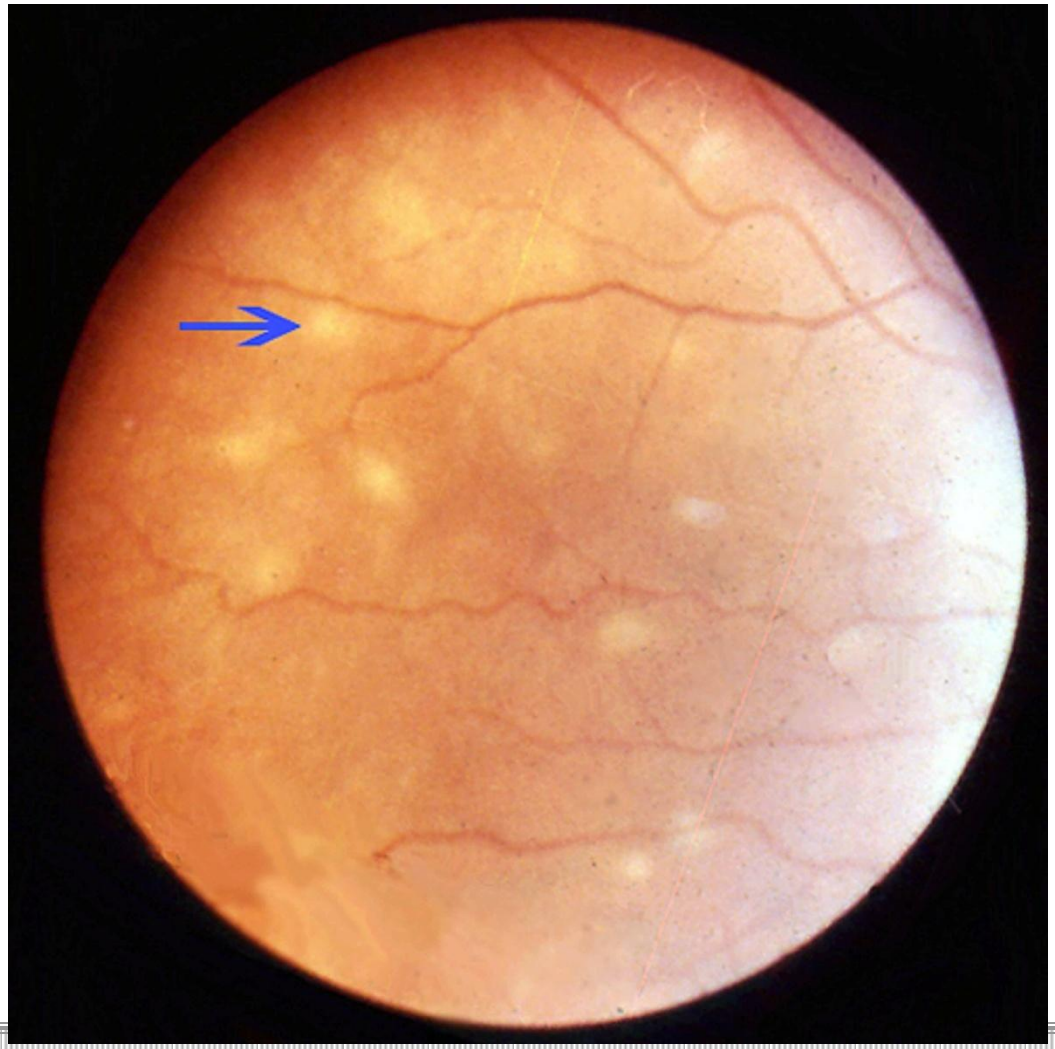


Pott's disease = tuberculous spondylitis

Credit: Dr Laughlin Dawes

www3.shropshire-cc.gov.uk/. ../med/med_i03.htm

“Miliary TB” is disseminated throughout the body
Here it is presenting in the eye



www.aippg.net/forum/ viewtopic.php?t=11673

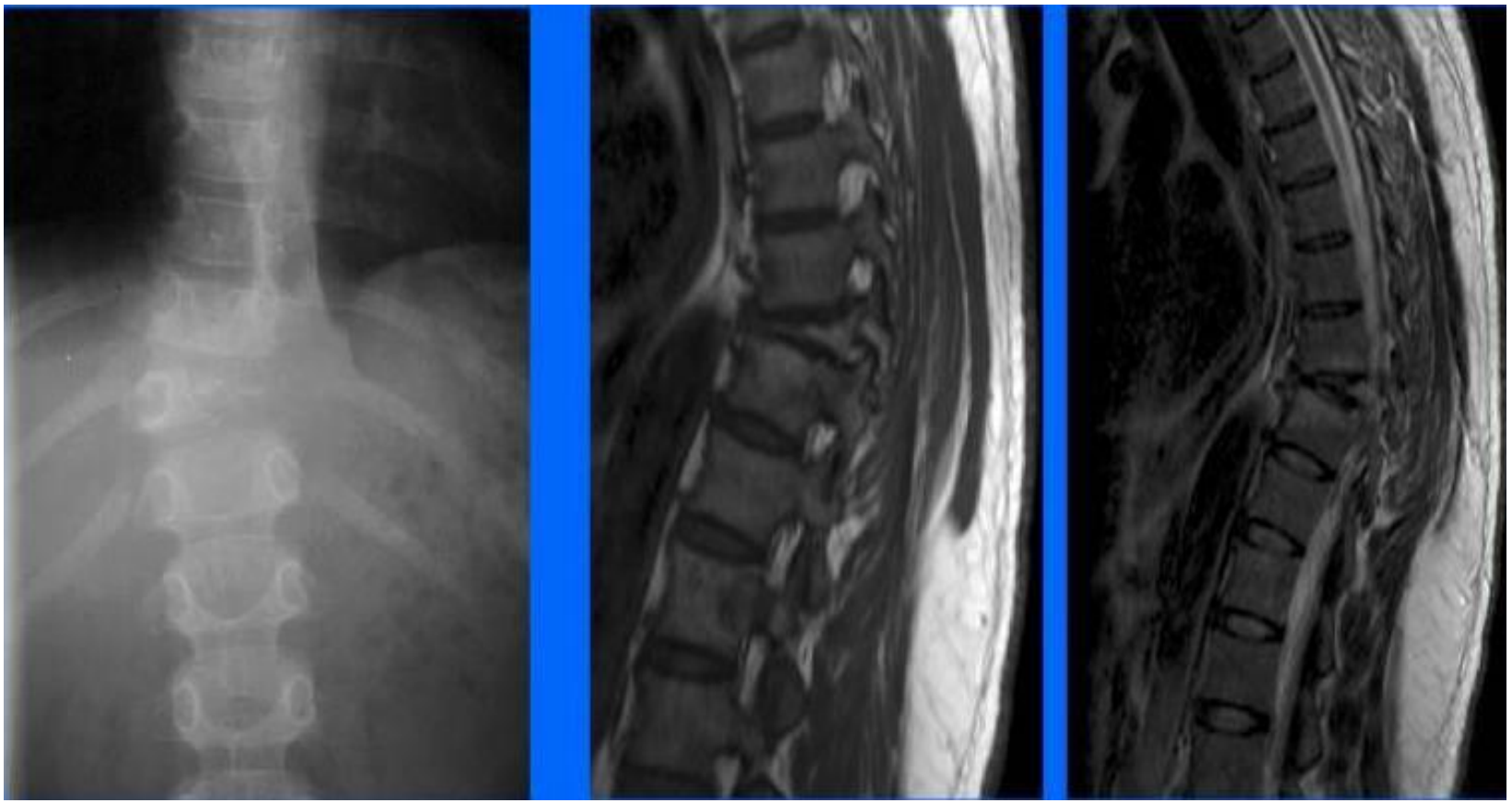
Scrofula: TB of the lymph nodes of neck

King Henry IV of France touching sufferers of scrofula. André de Laurens, 1609

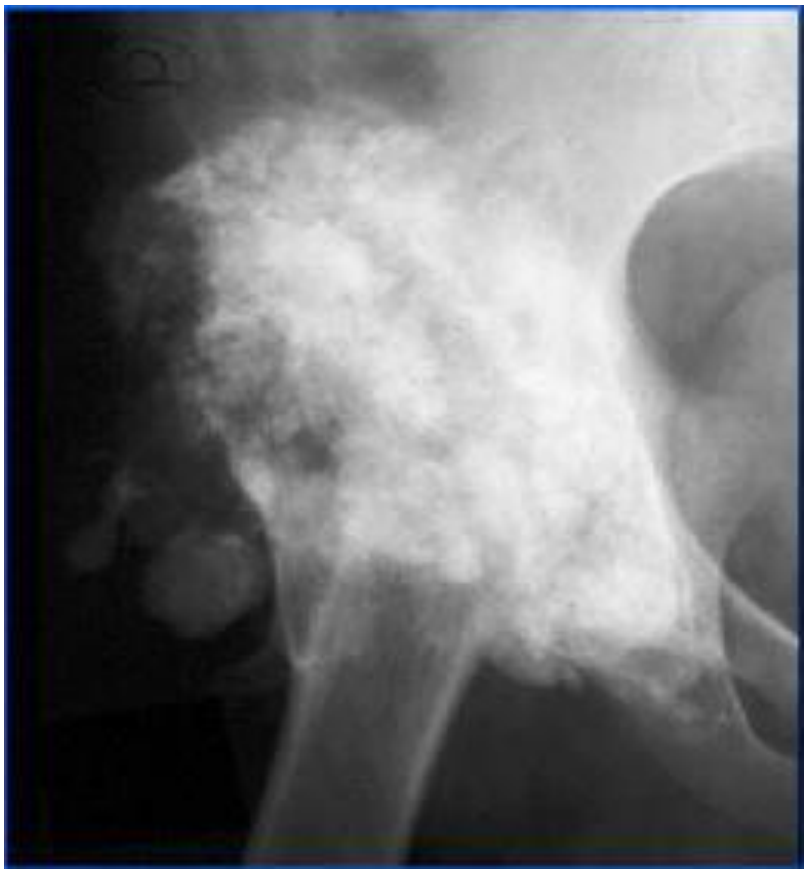
TB - Scrofula (Mexico)

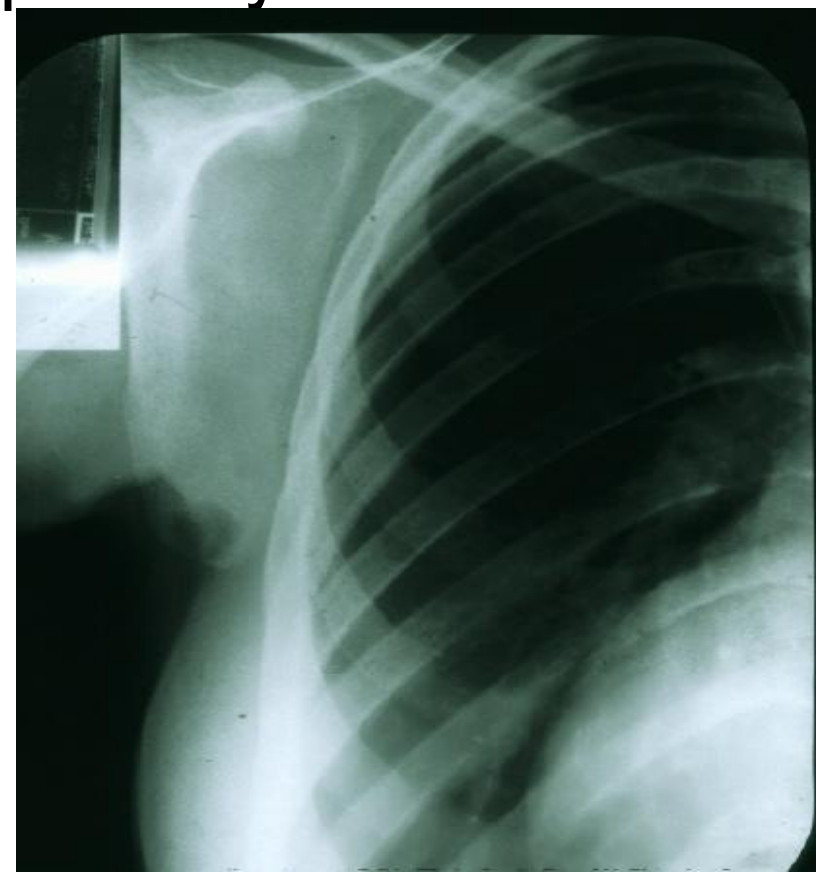
Bony tuberculosis

Astute radiologist should enable the appropriate further investigation

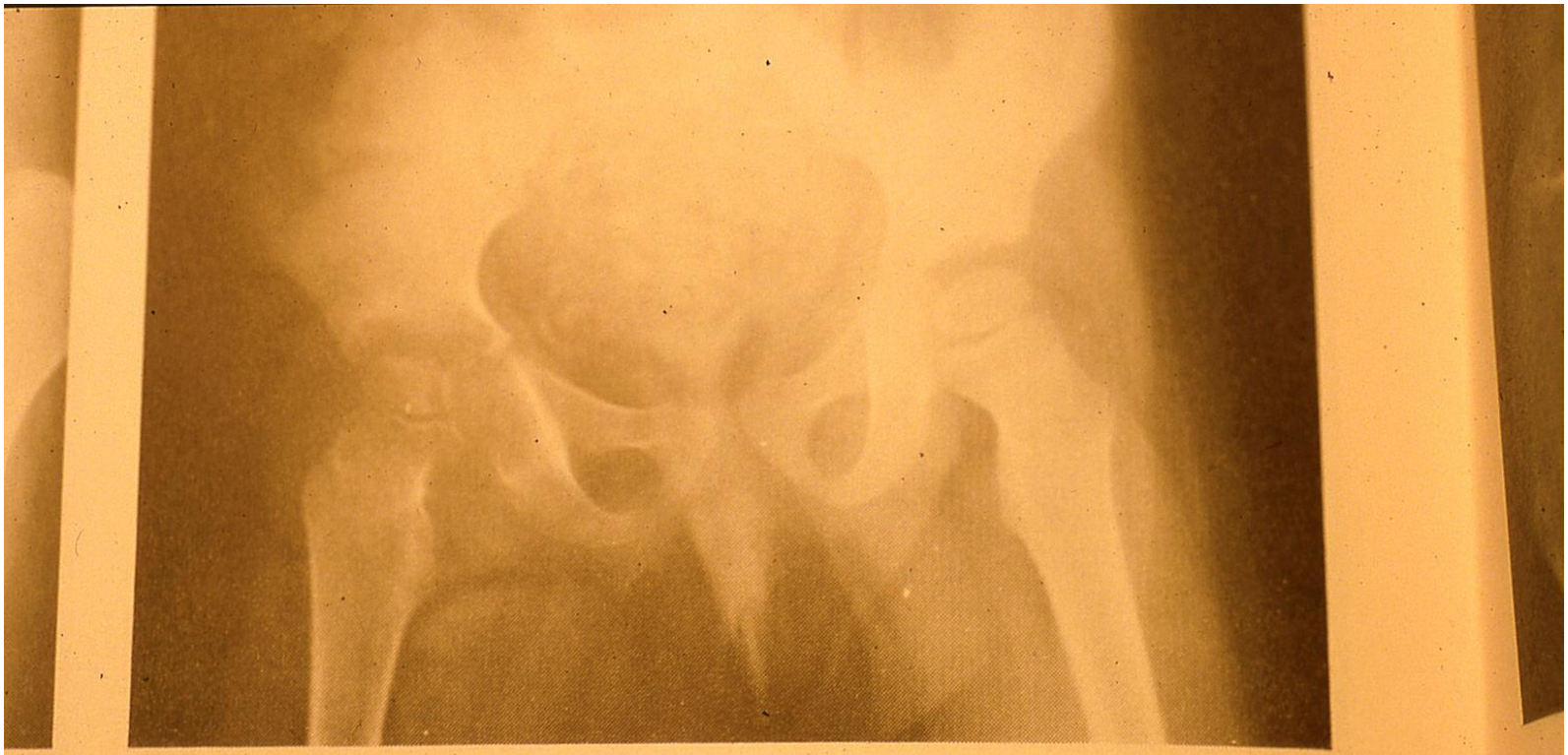


Often associated with delay in diagnosis – **any** chronic discharging lesion must be considered possibly TB



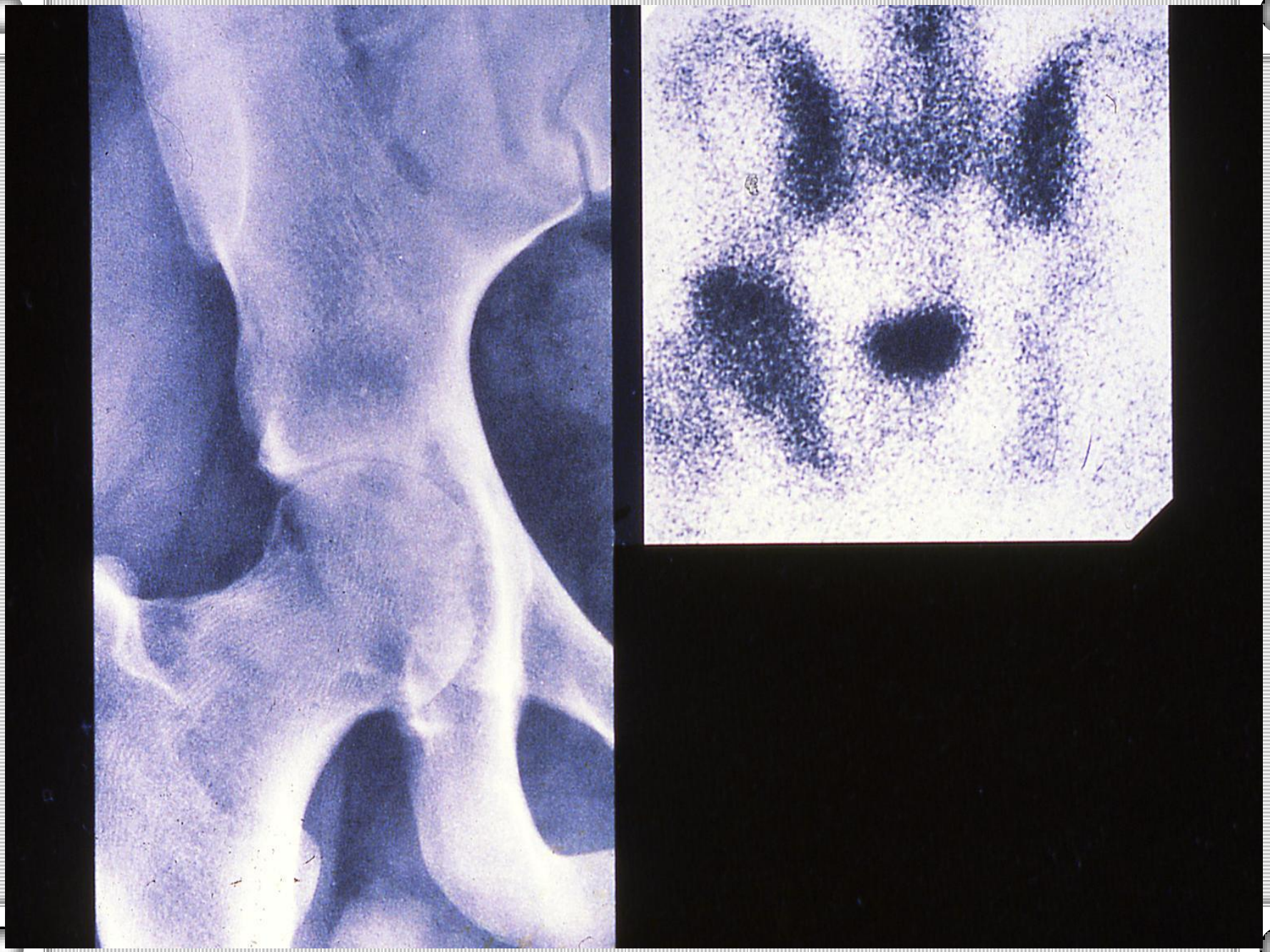


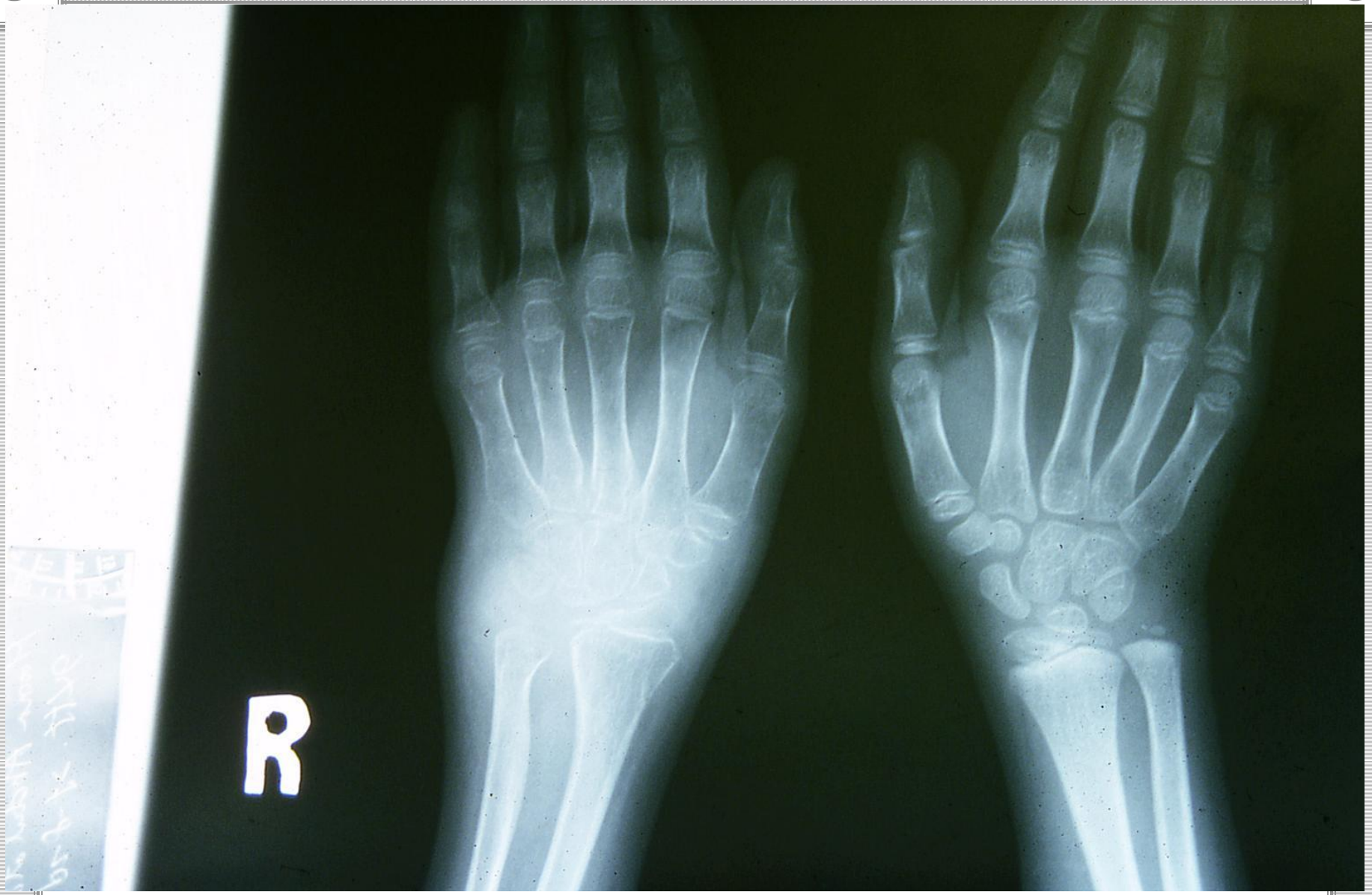
Tuberculosis of bones



38. Radiograph of a three year old female child with a three month history of a limp. She was noted to be fretful and cried at nights. Clinically there was a flexion deformity of the right hip, muscle wasting in the right leg, restriction of movements in the right hip with pain at the extrêmes, and an increased lumbar lordosis.

40. Radiograph of a 14 year old boy with a nine month history of a limp, with progressive pain and stiffness in the right hip. He was noted to have a fixed flexion deformity of the hip with an associated increase in lumbar lordosis. Movements in the right hip were grossly restricted, with pain at the extremes.

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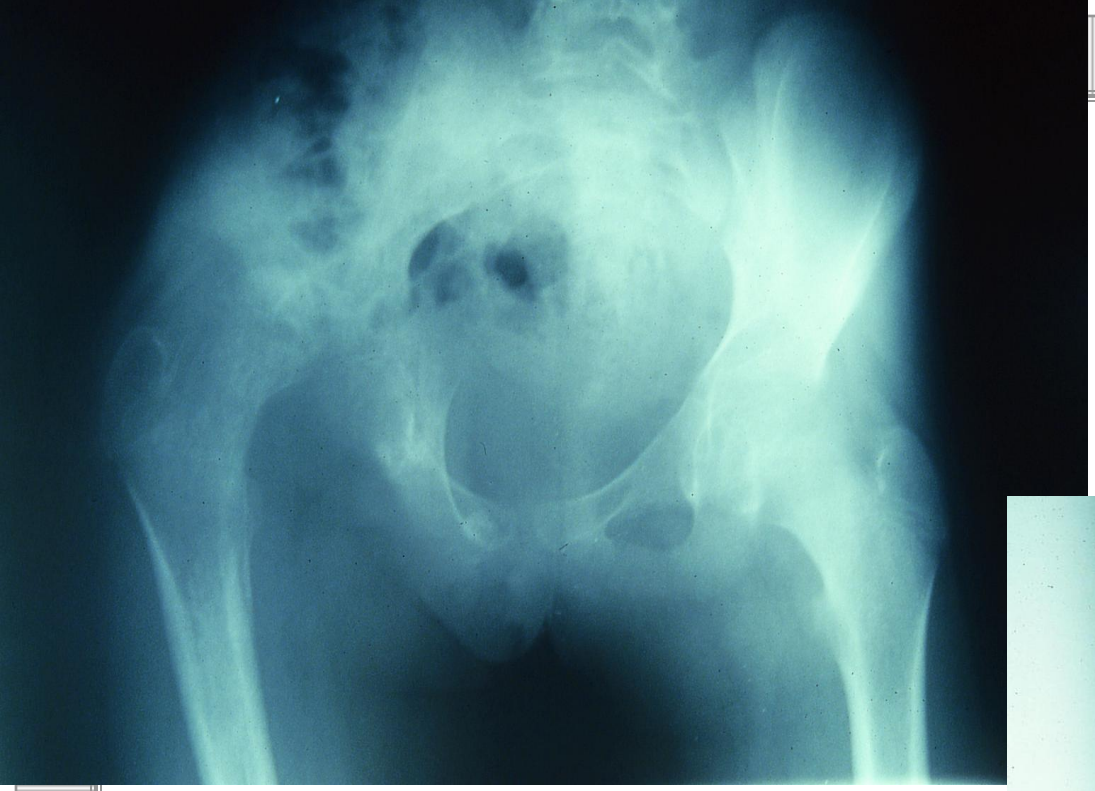

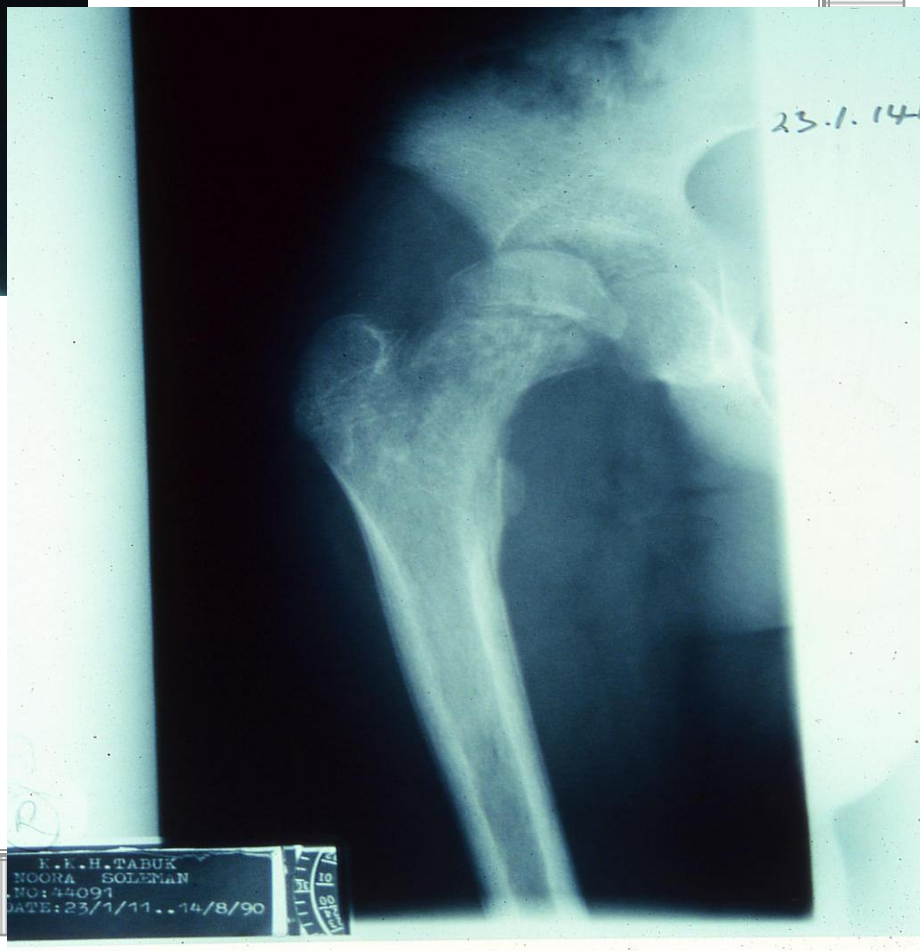
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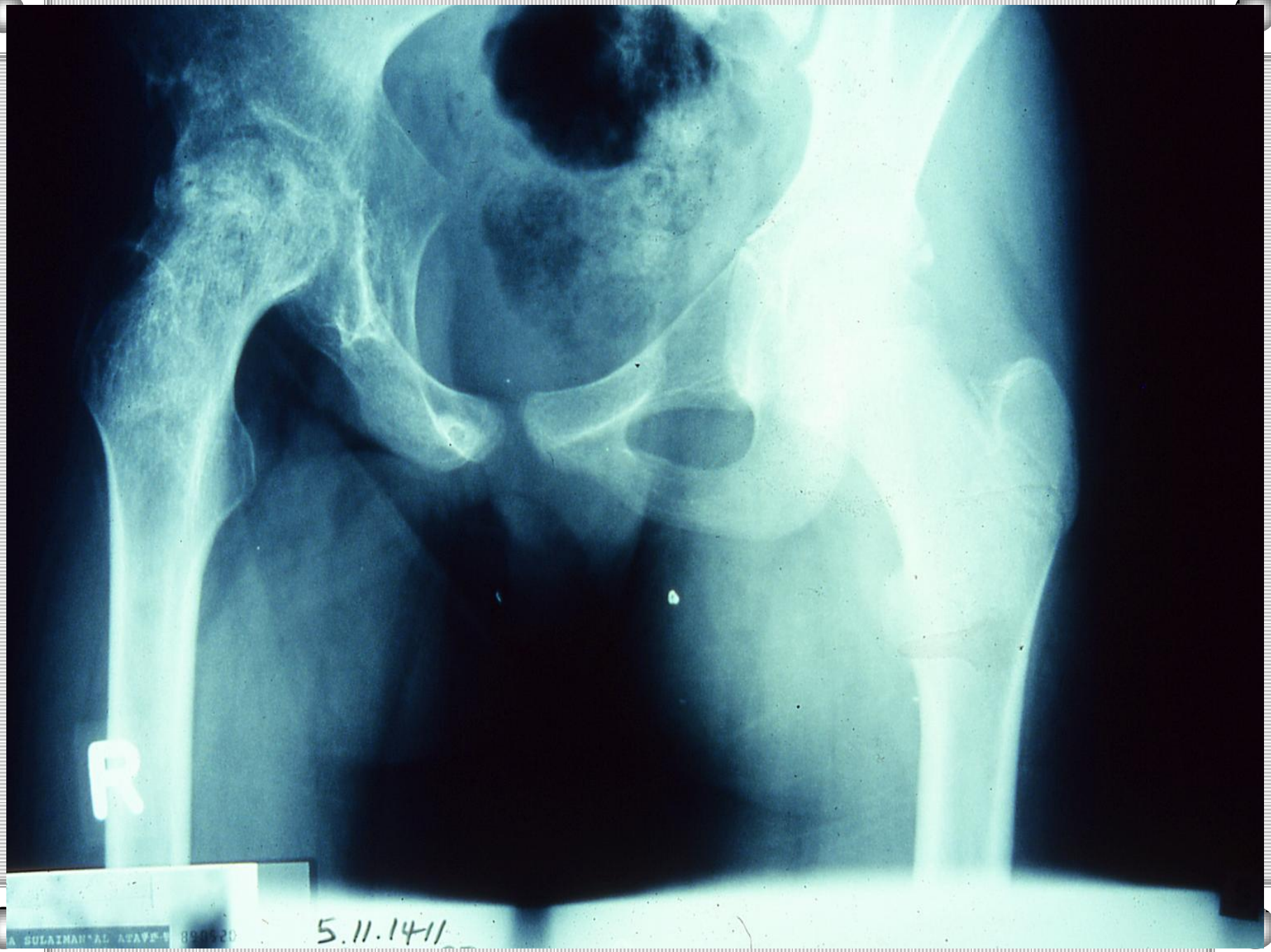
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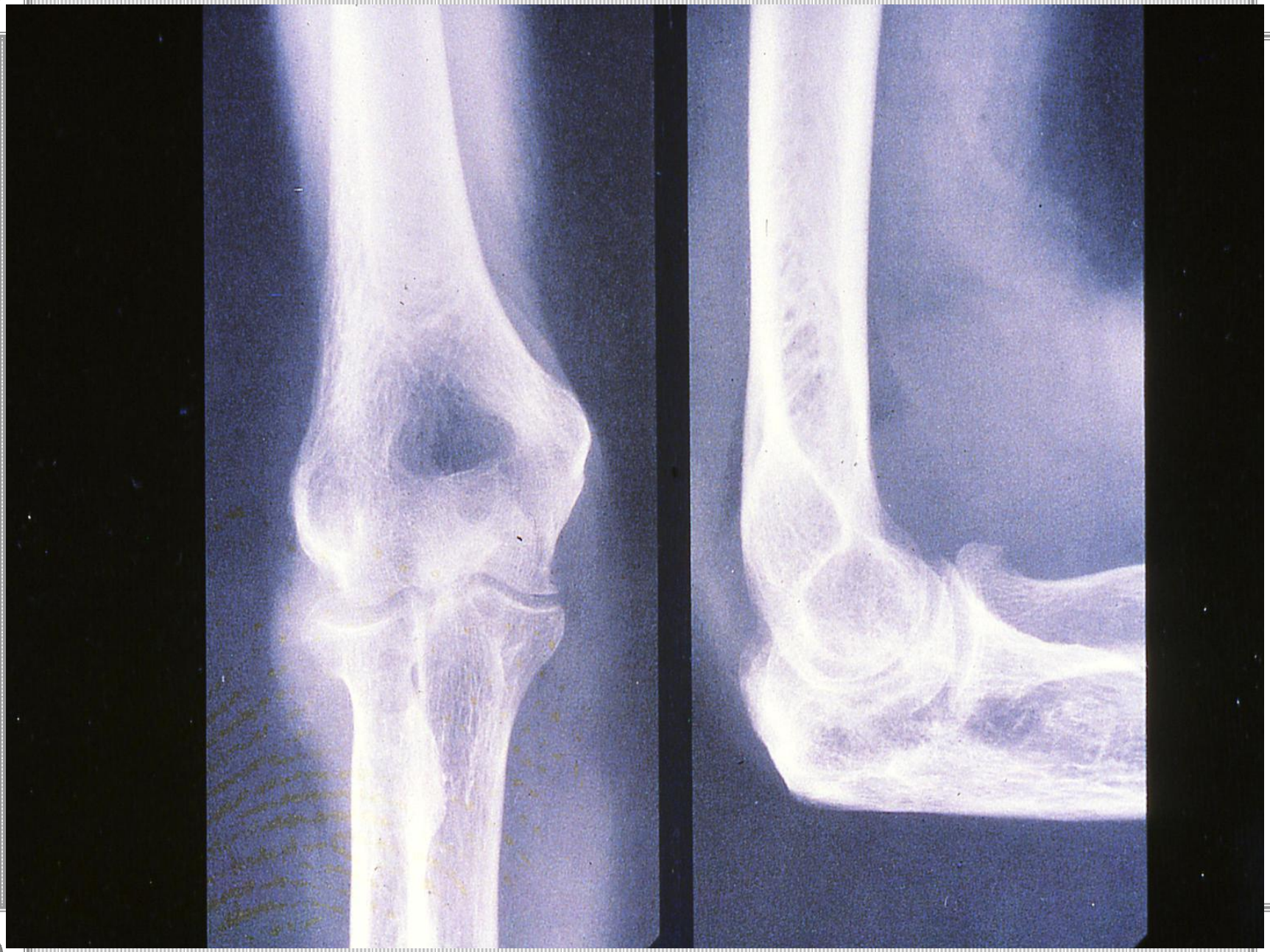
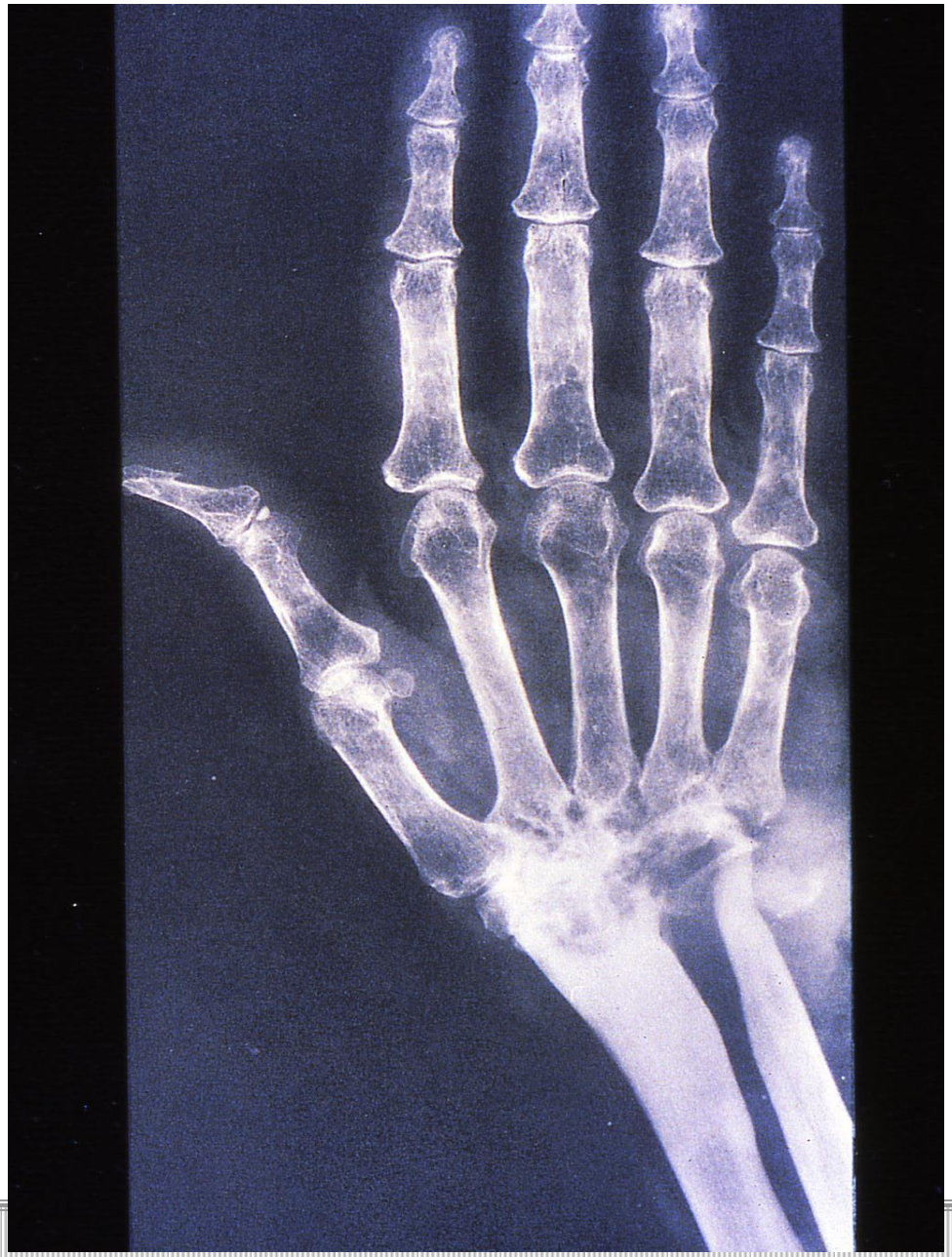
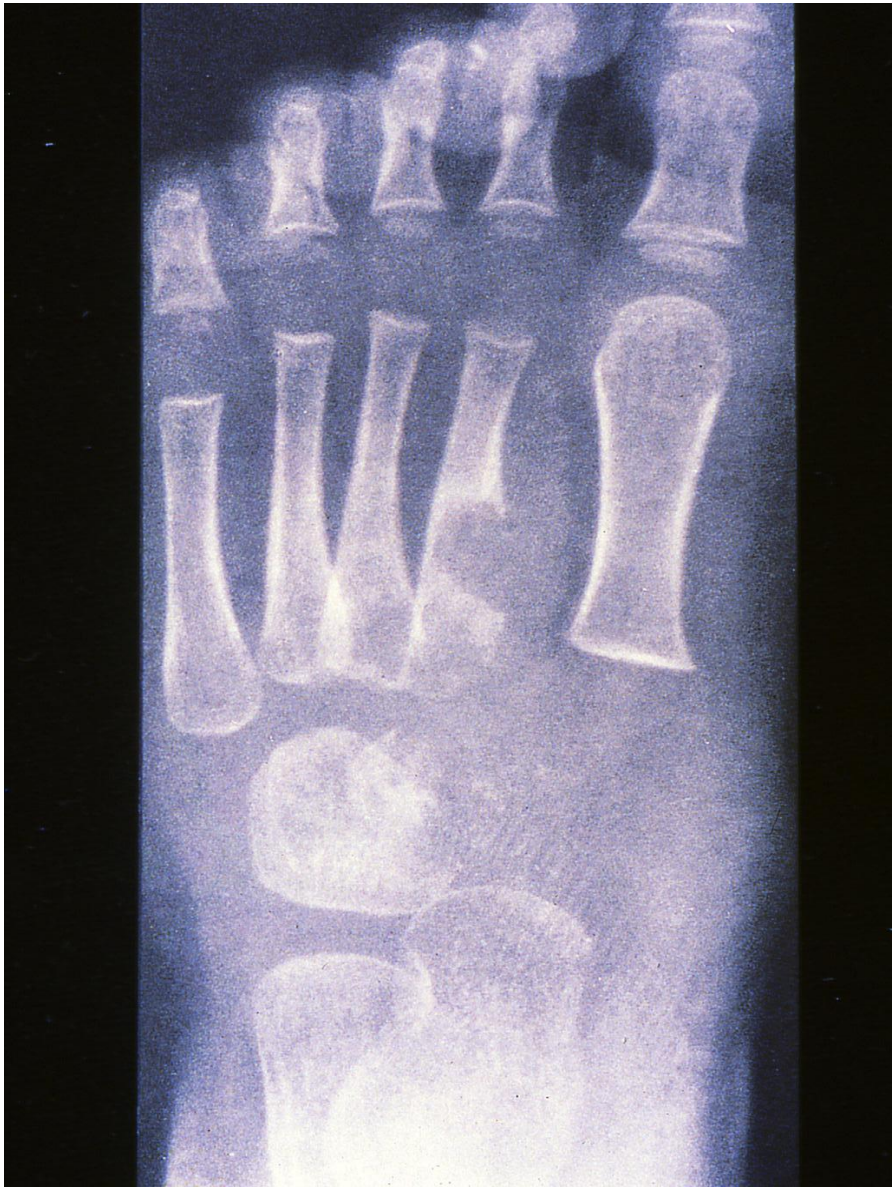
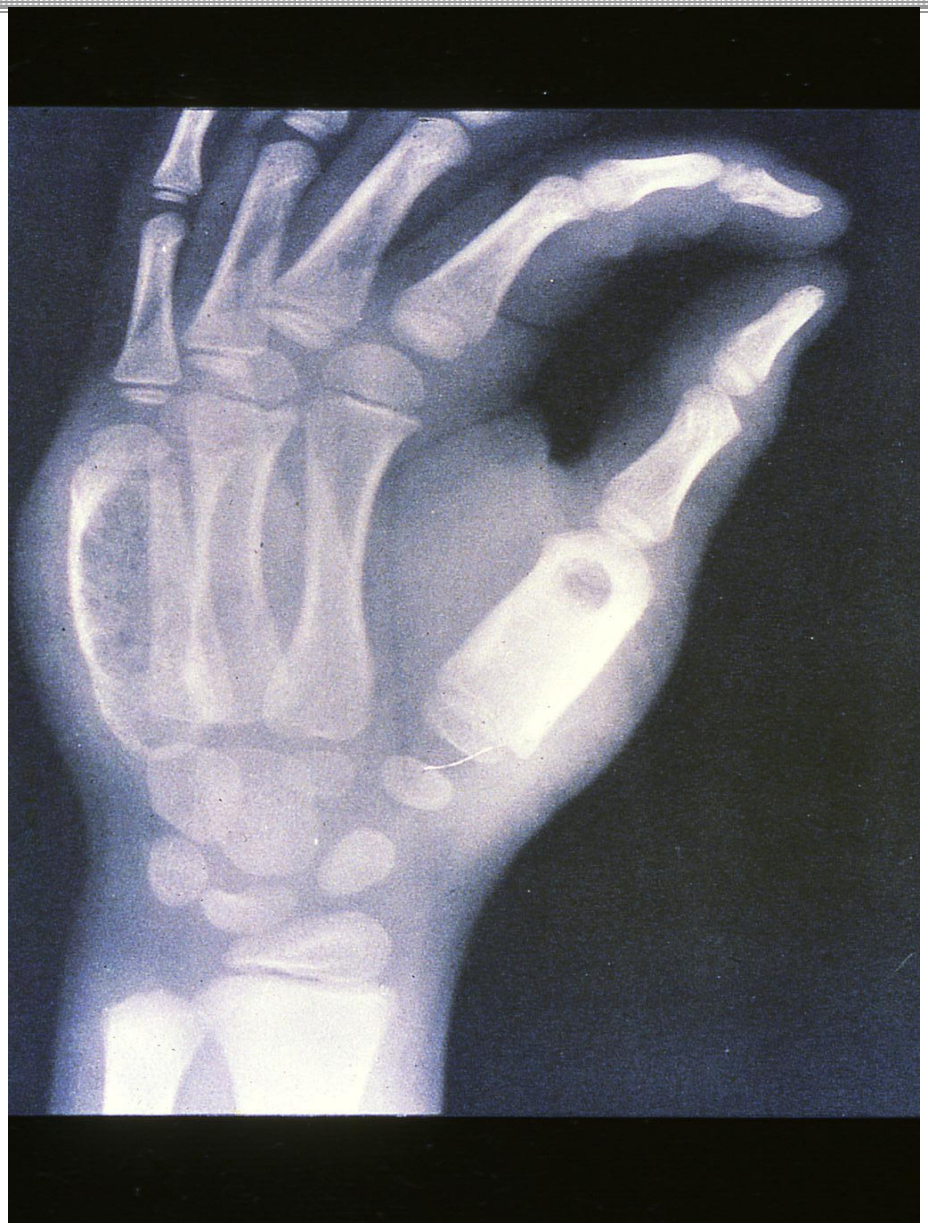








TB SPINE
SURGICAL PATHOLOGY

FIRST THREE DECADES

THORACO-LUMBAR

CENTRAL SPINE

SPARKS POSTERIOR ELEMENTS

SPREADEDS UP/DOWN
ANT./POST. LONG. LIGS.

LESIONS COALESCE – COLLAPSE

KYPHUS FORMATION

TB SPINE
SURGICAL PATHOLOGY

PARA VERTEBRAL ABSCESS

CERVICAL : RETROPHARALYGEAL

THORACIC : P.V. & ALONG RIBS

LUMBAR: PSOAS ABSCESS

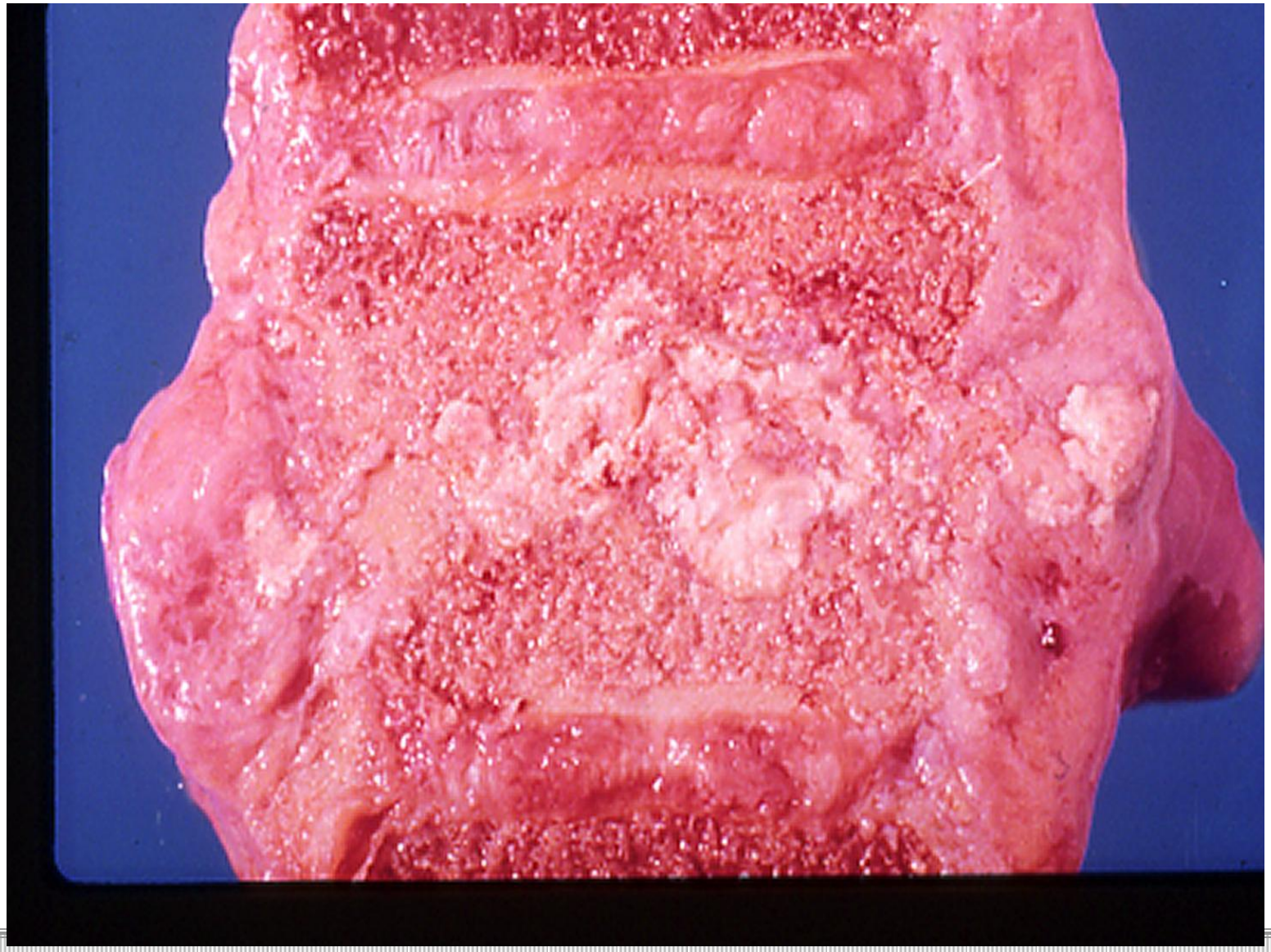
POSTERIOR:LUMBAR TRIANGLE

ANTERIOR: ILIAC FOSSA

BELOW ING. LIG.

NEUROLOGICAL COMPLICATION

MORE IN THORACIC (NARROWEST CANAL)



TB SPINE
CLINICAL FEATURES

GENERAL: INSIDIOUS ONSET

CONSTITUTIONAL

LOCAL: PAIN – FIRST INDICATION

LOCAL – REFERRED

STIFFNESS – SPASM

WEAKNESS – NEUROLOGICAL

SIGNS OF TB SPINE

MUSCLE SPASM

KHPHUS – GIBBOUS

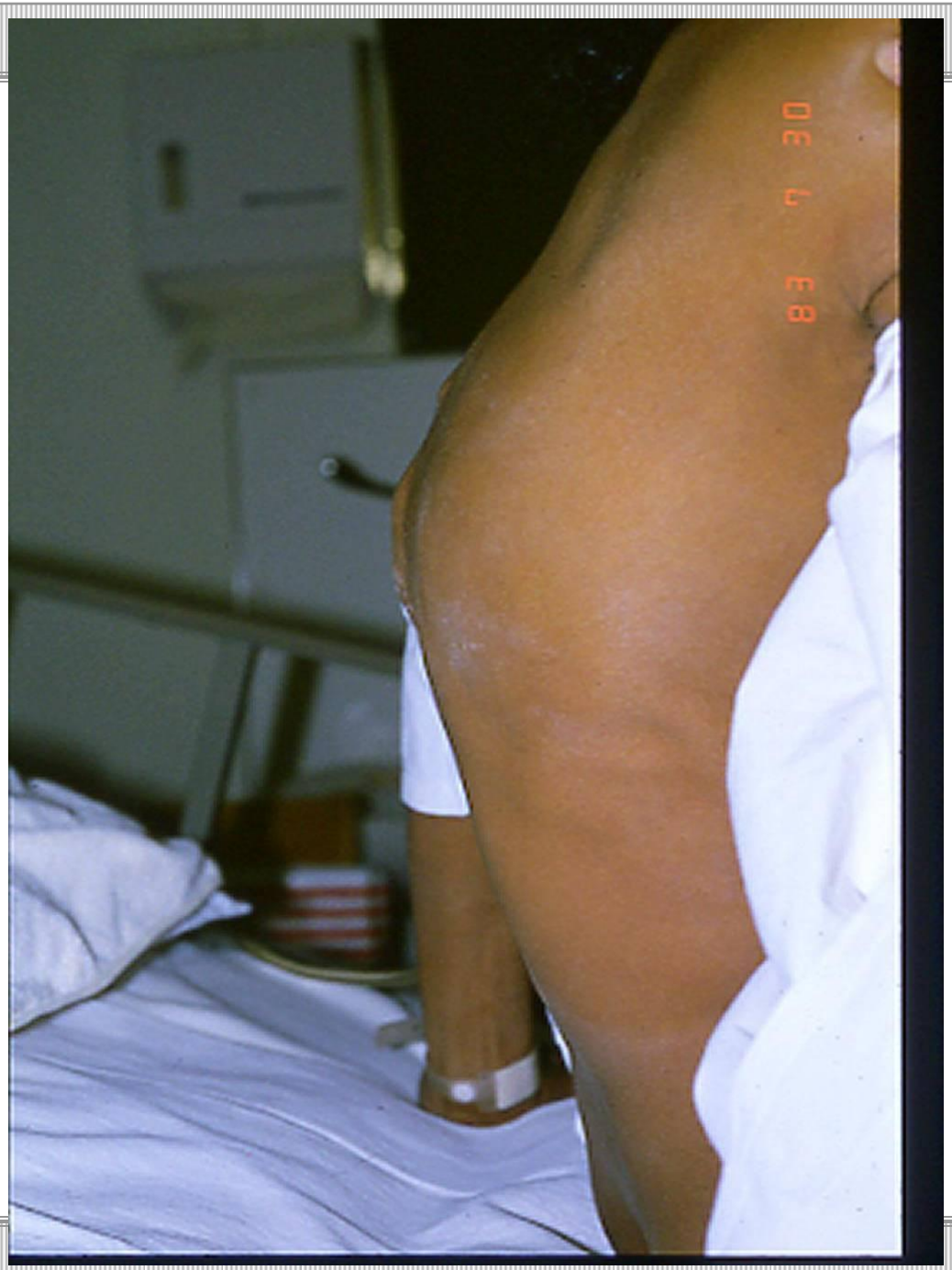
TENDERNESS

STIFFNESS

PARA VERTEBRAL ABSCESS

NEUROLOGICAL – WEAKNESS

PARAPLEGIA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TB SPINE
RADIOLOGICAL FEATURES

DISC NOT INVOLVED PRIMARILY

NARROWING OF DISC SPACE

BONE DESTRUCTION

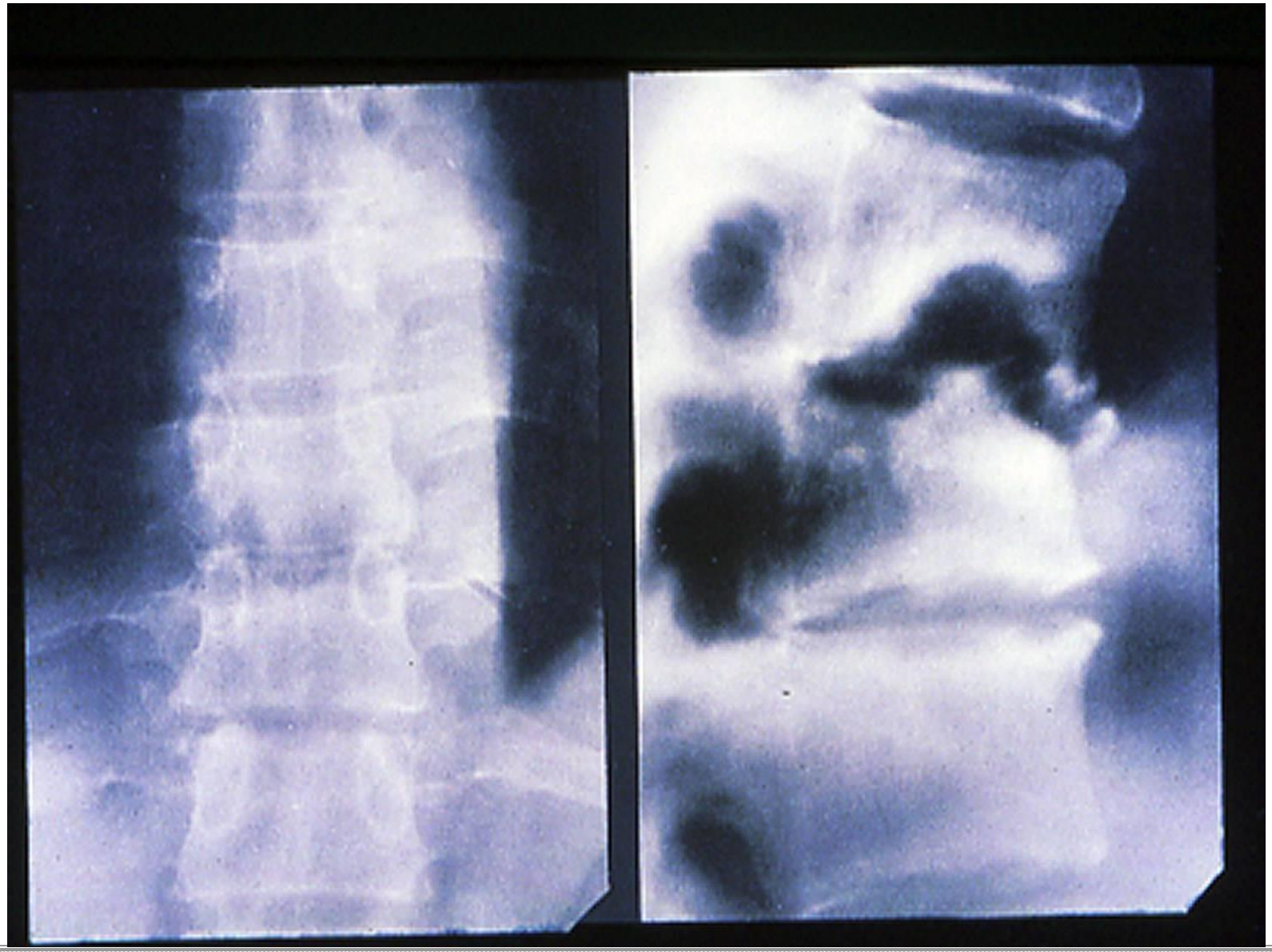
USUALLY TWO ADJACENT VERTEBRAE

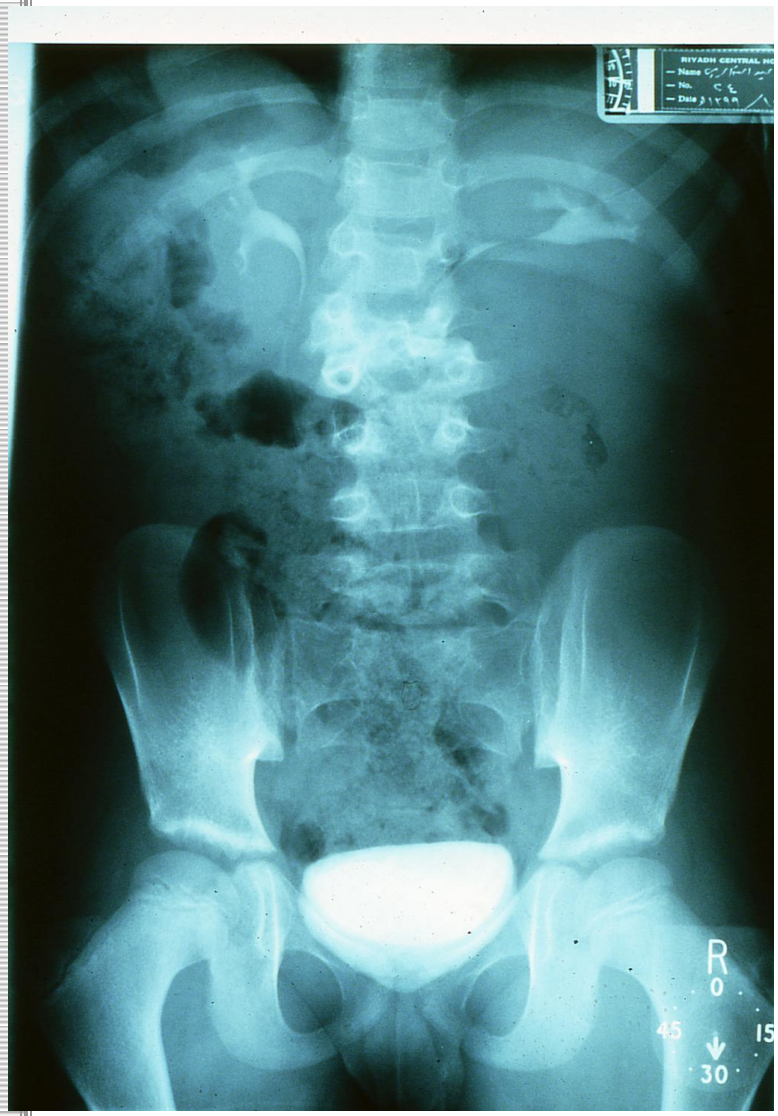
MAY SHOW SKIP LESIONS

PARA VERTEBRAL ABSCESS

KHYPUS

CT/MYELOGRAM/MRI IN PARAPLEGIA

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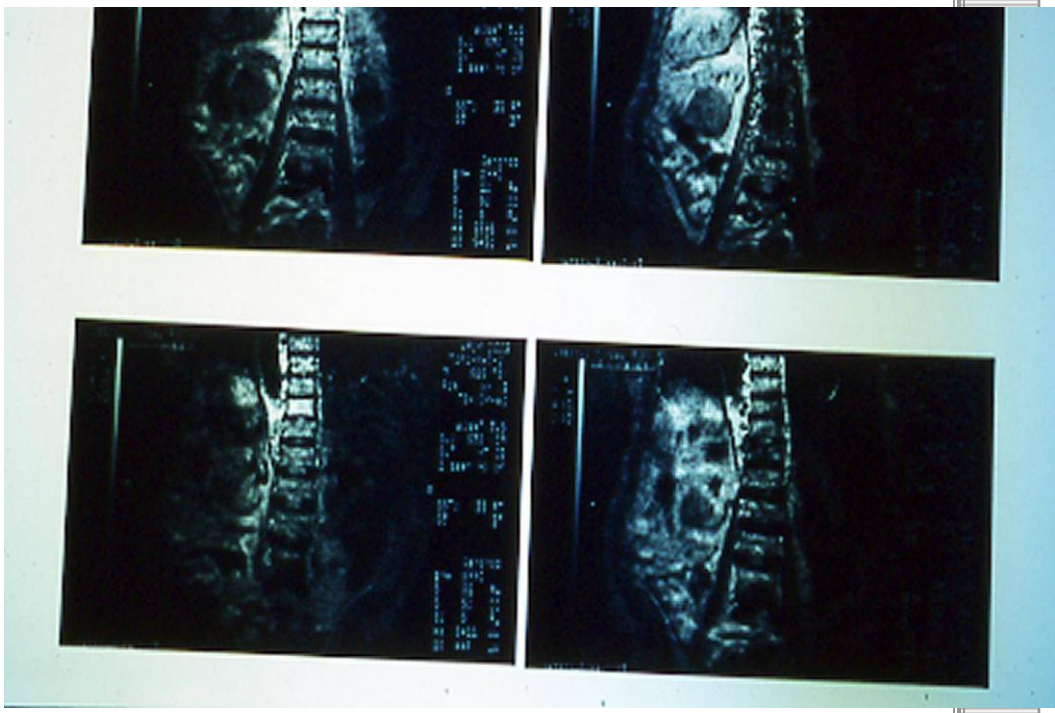

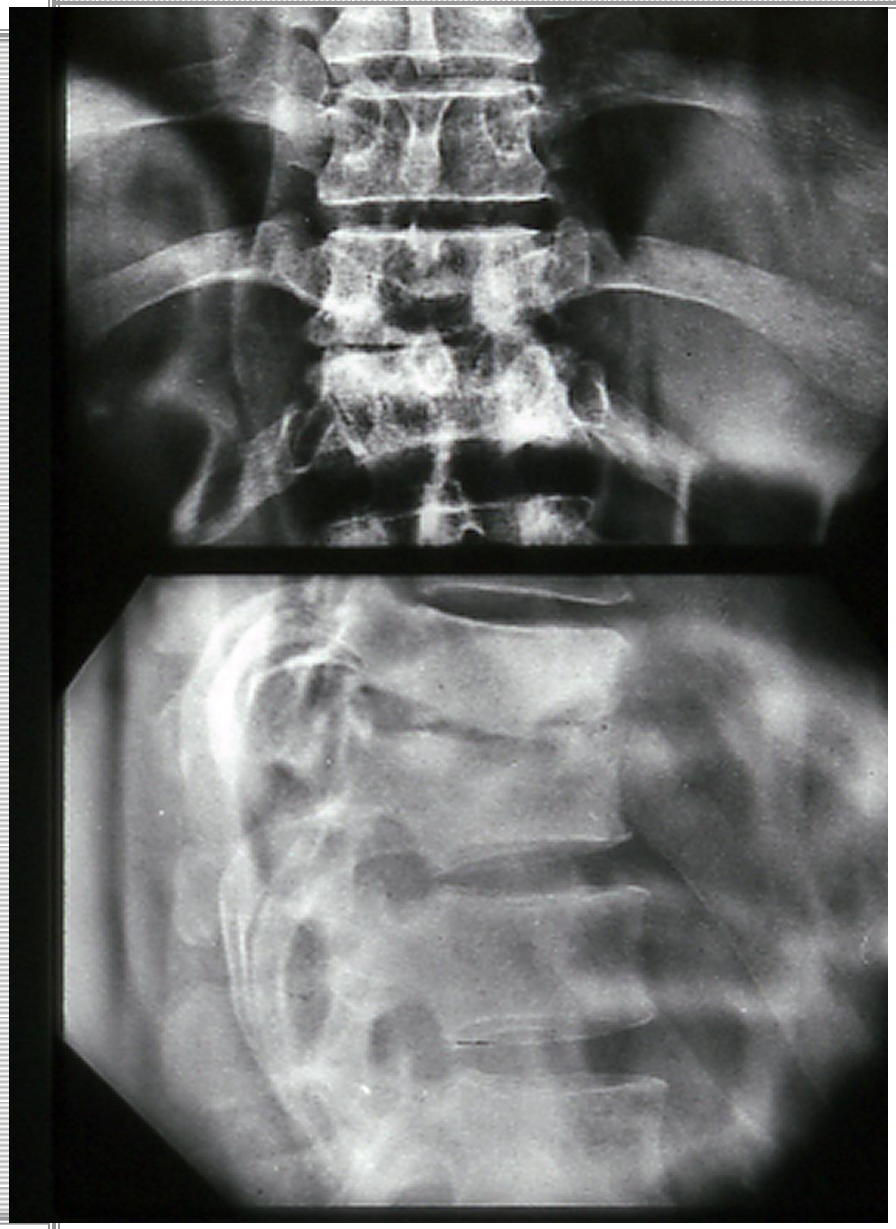

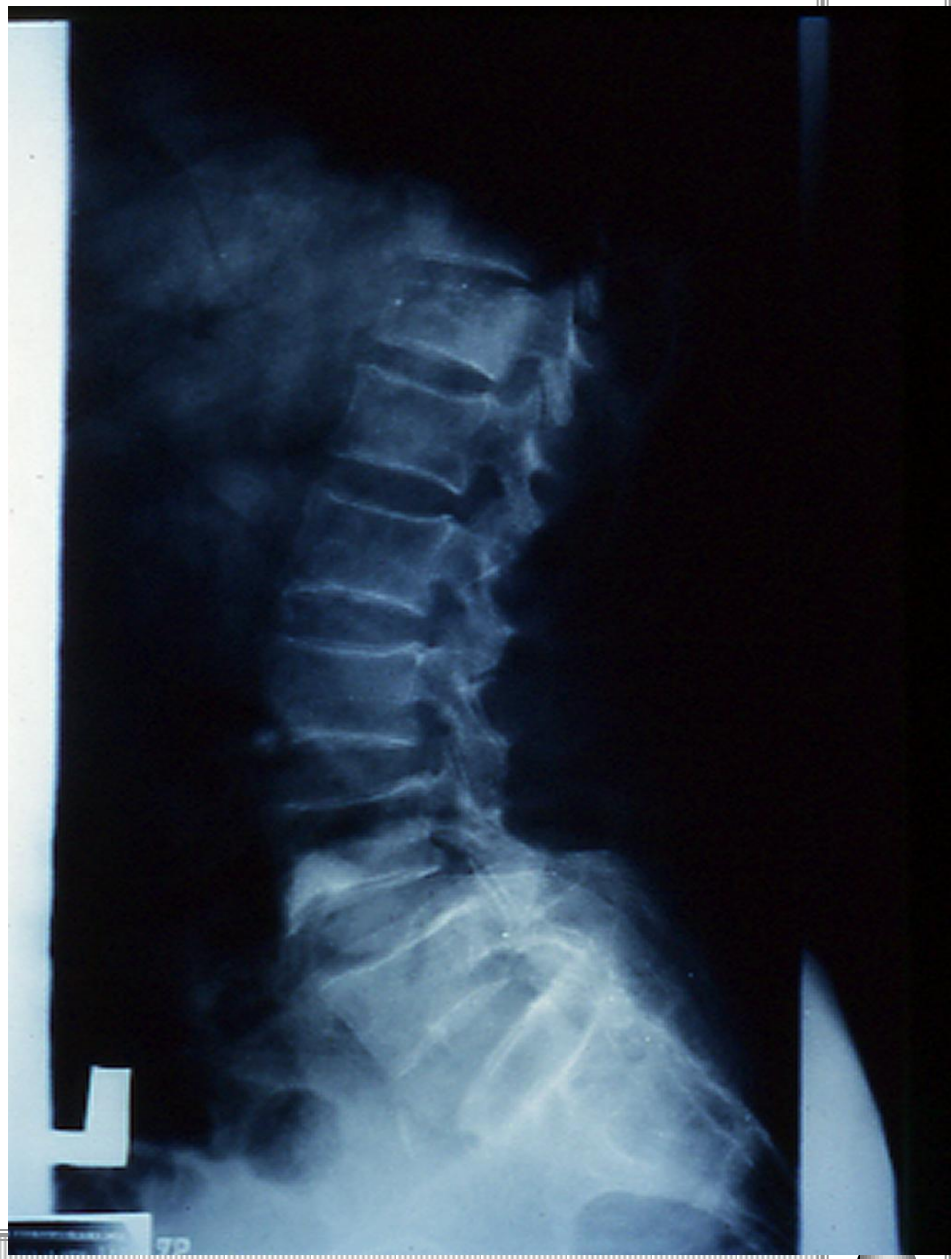

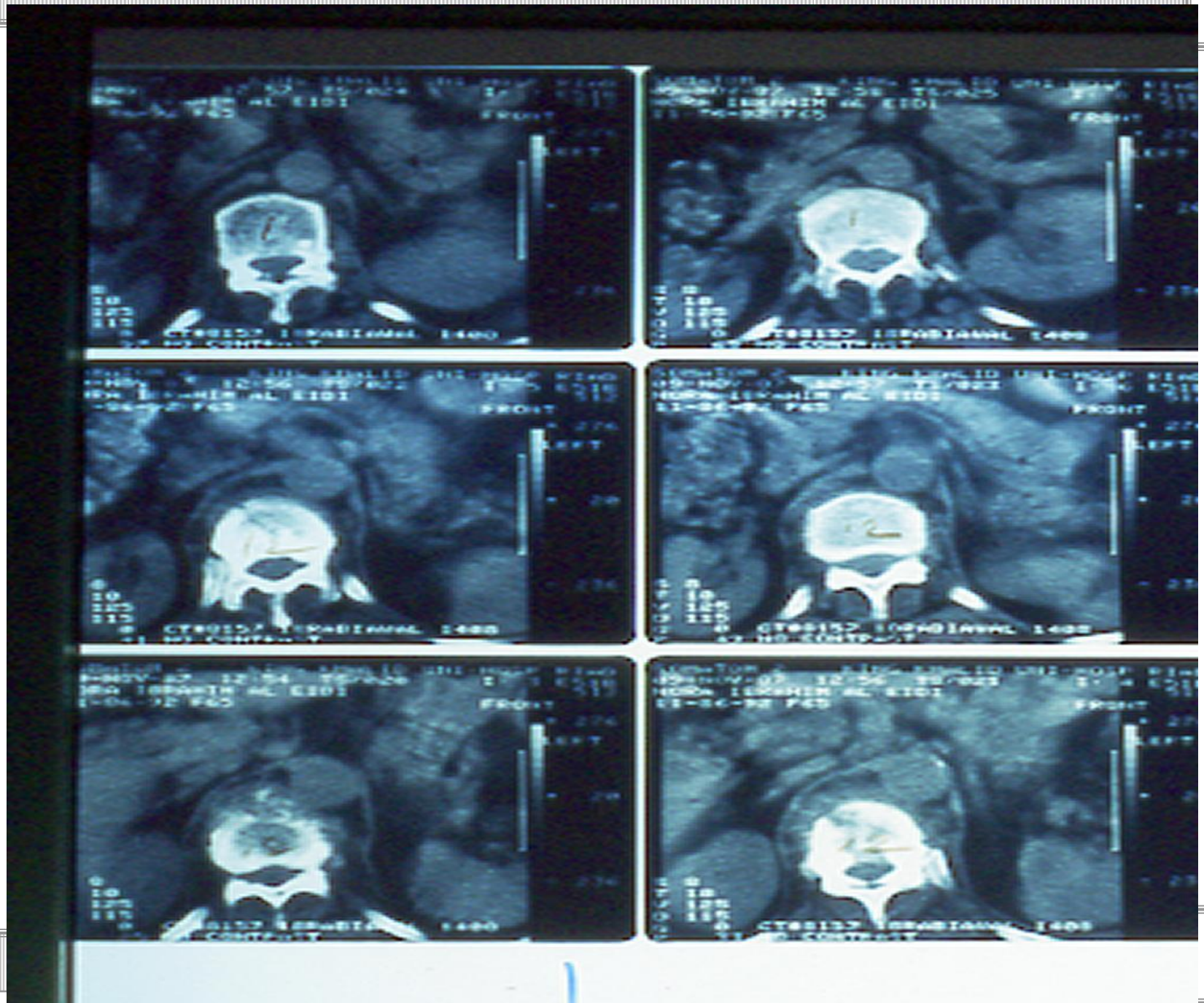

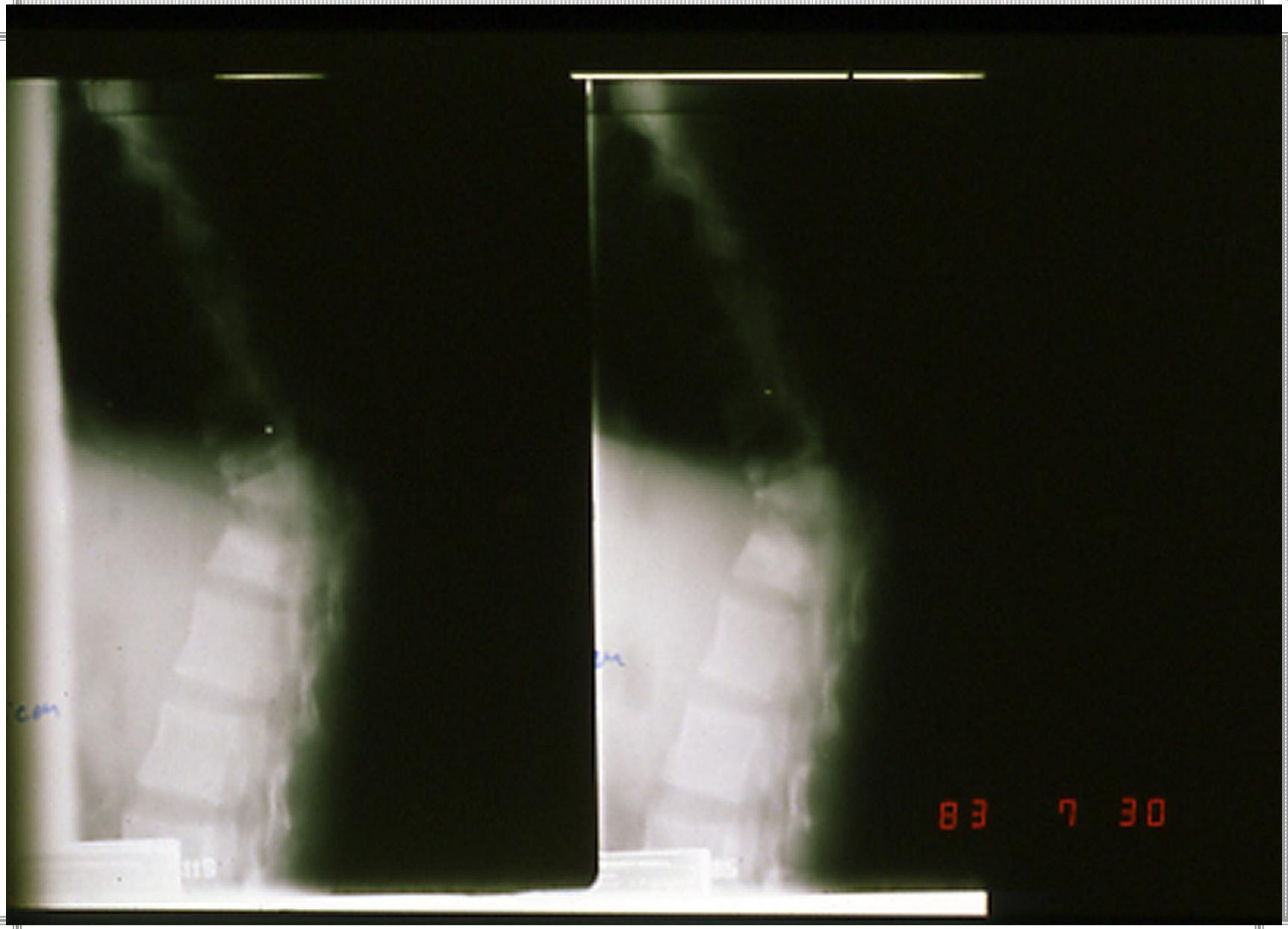
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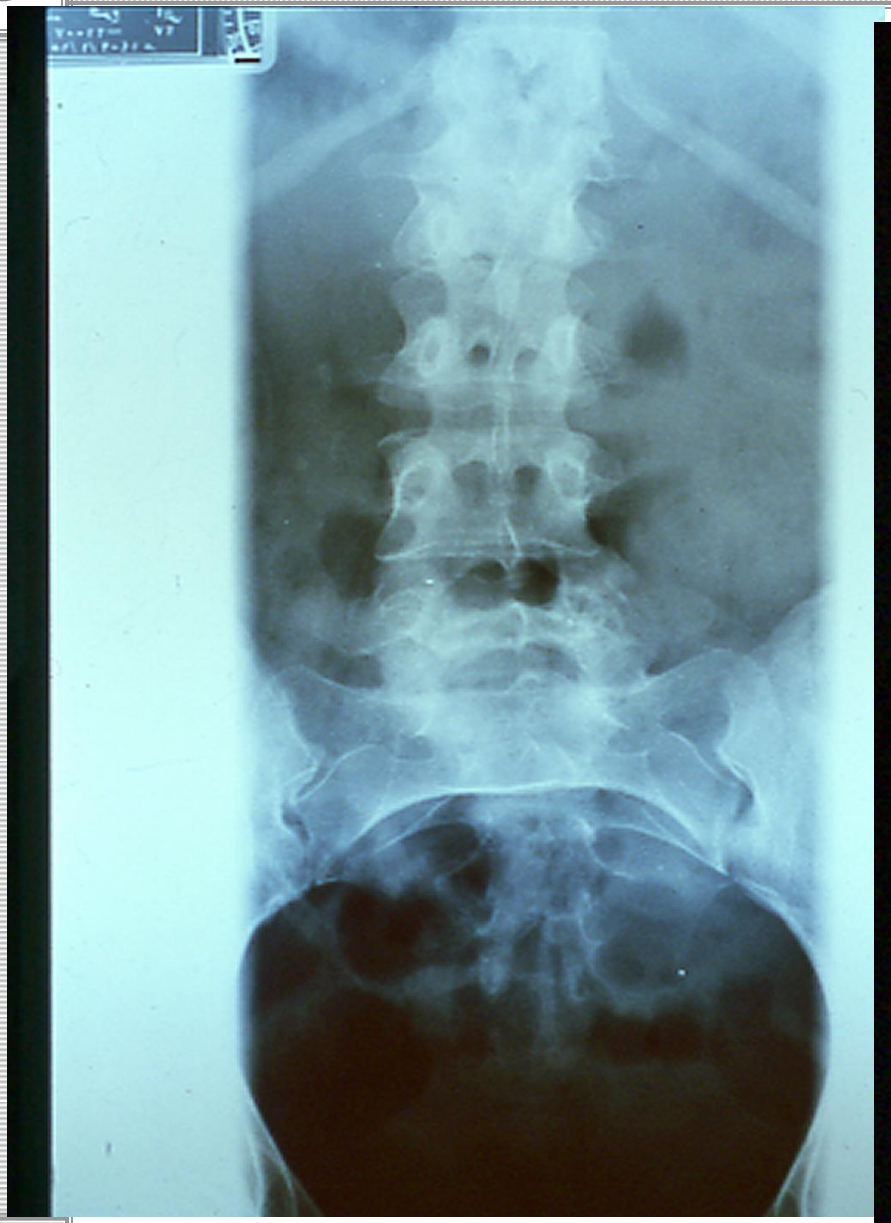

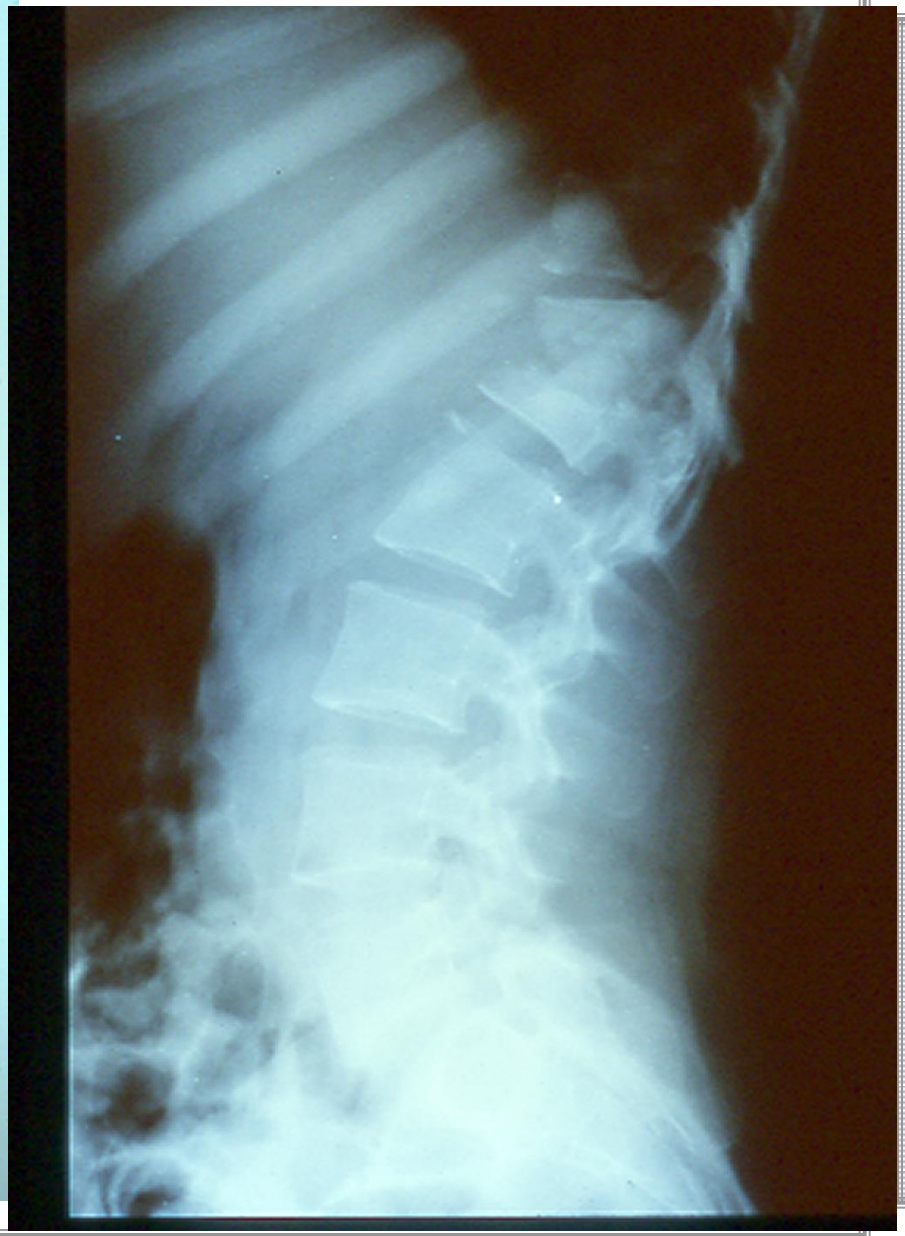

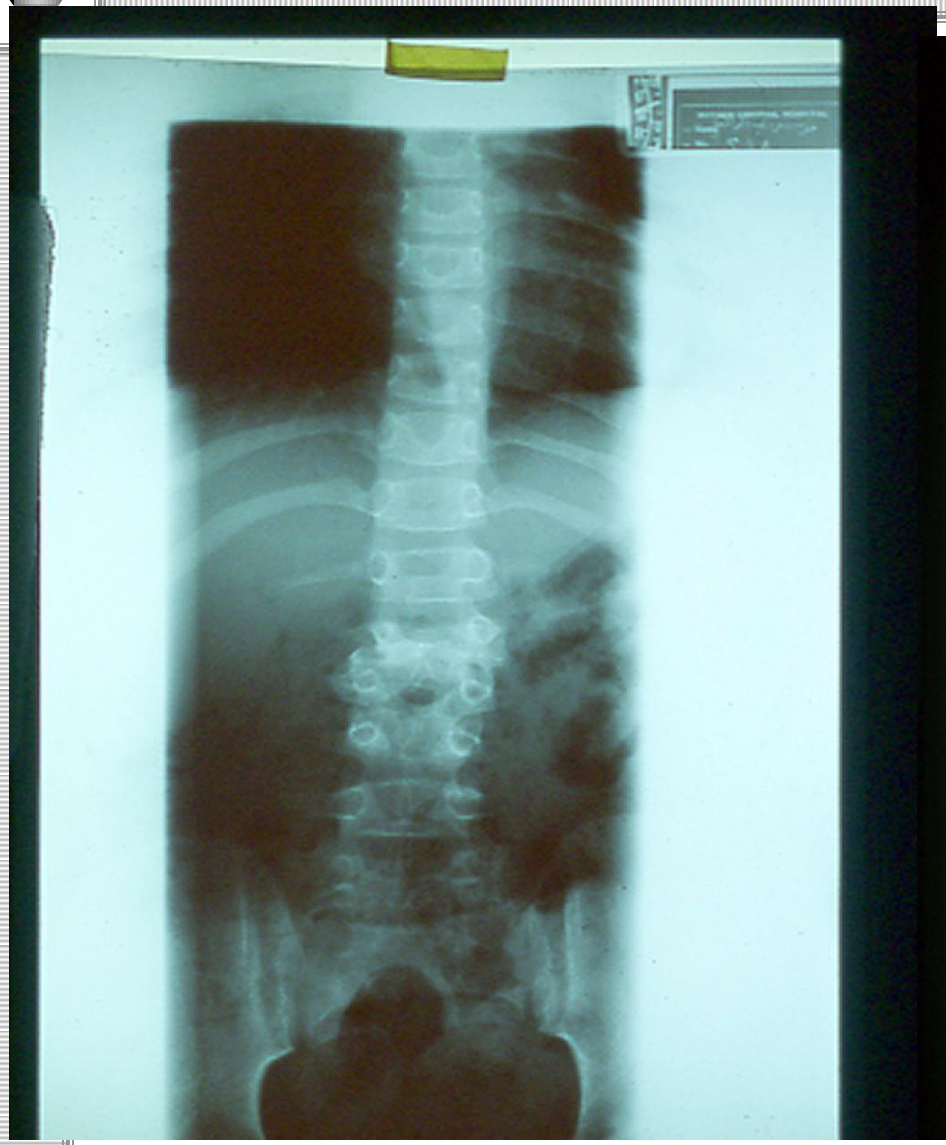

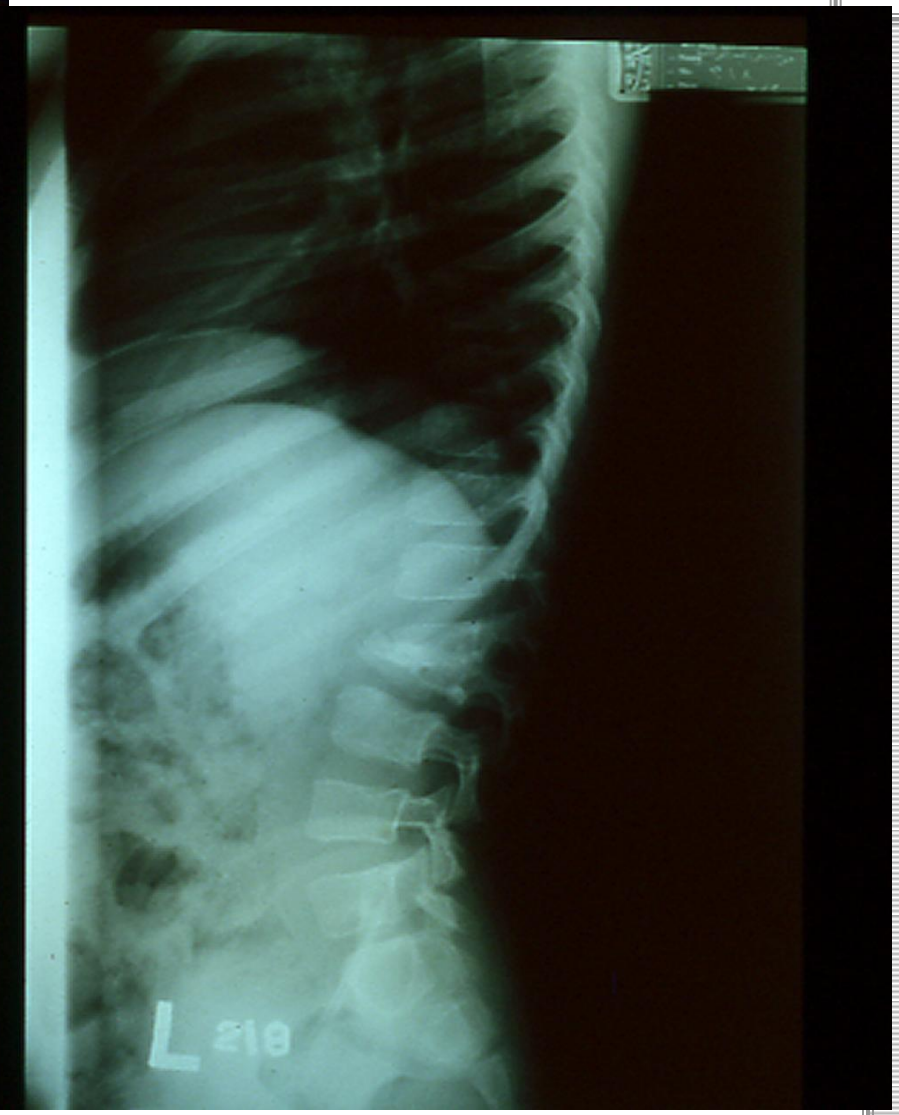
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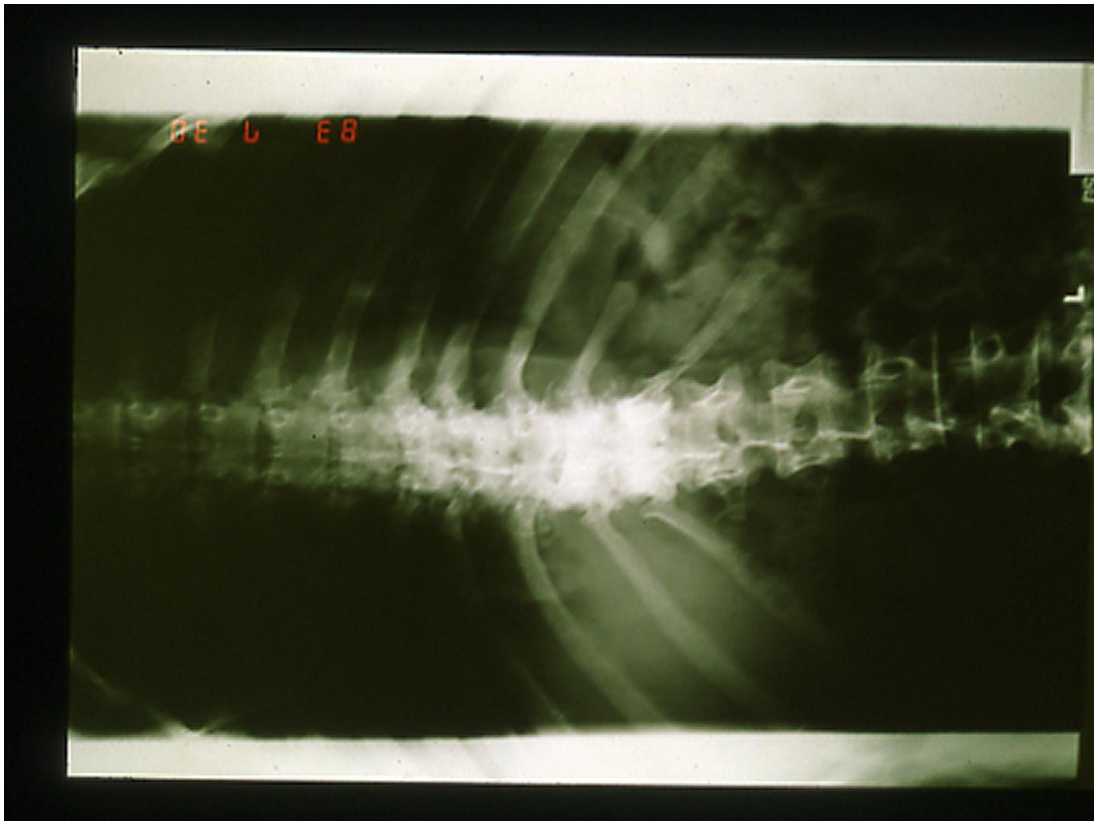
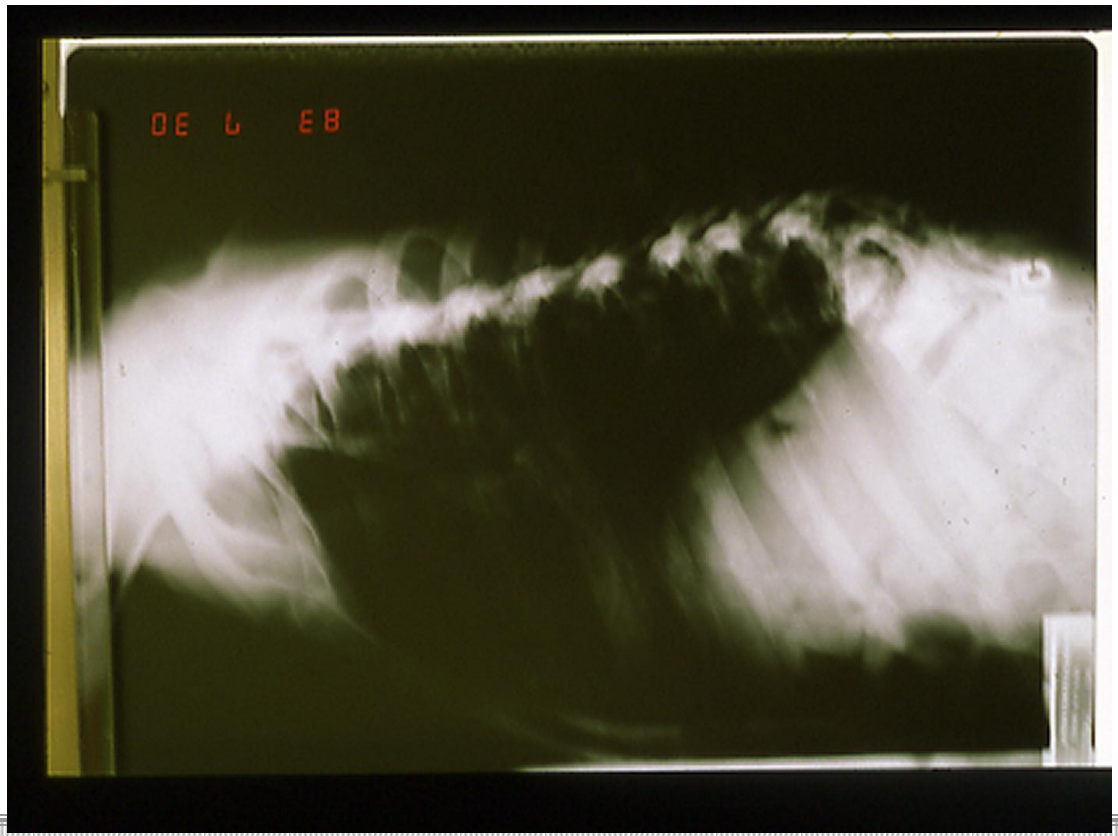
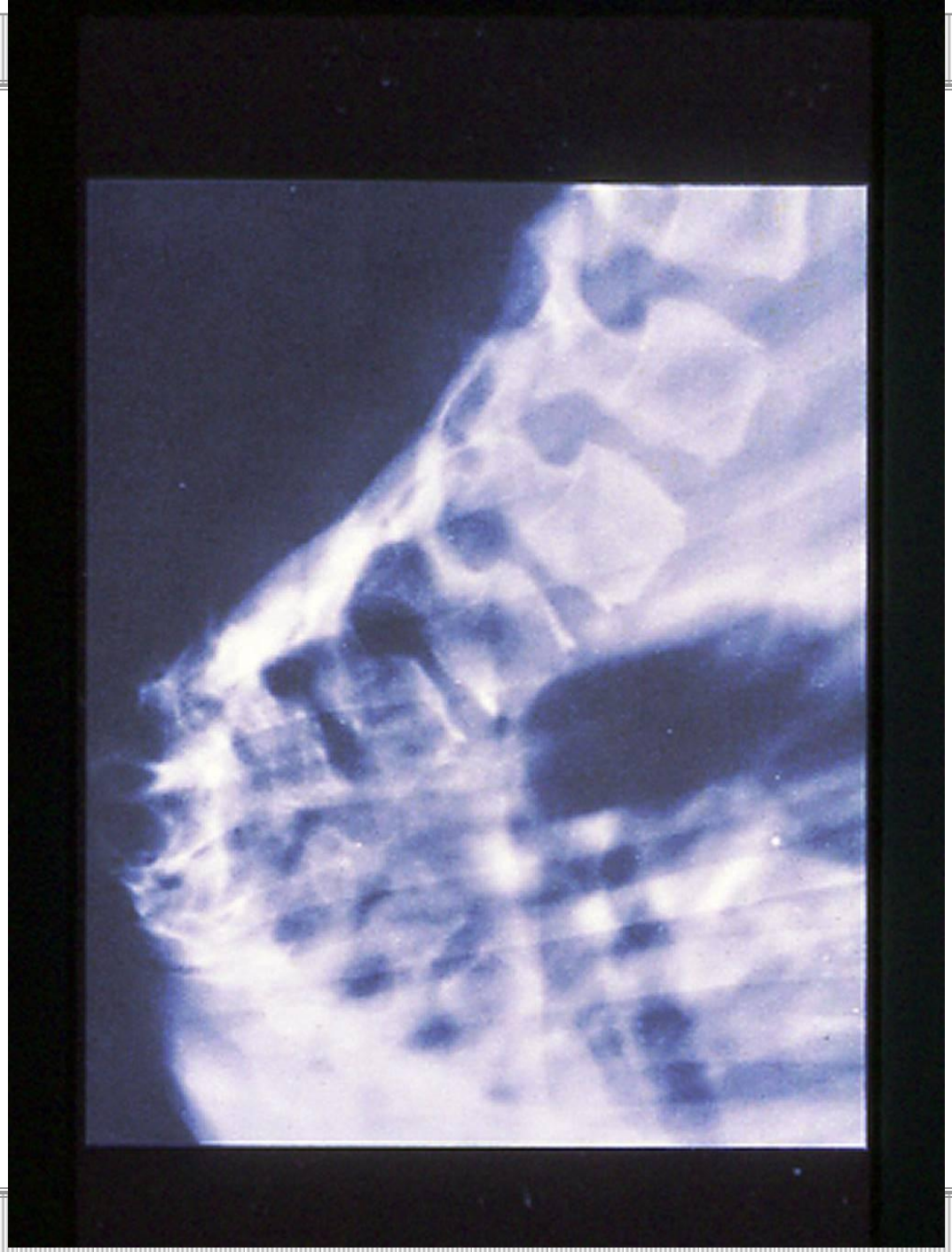
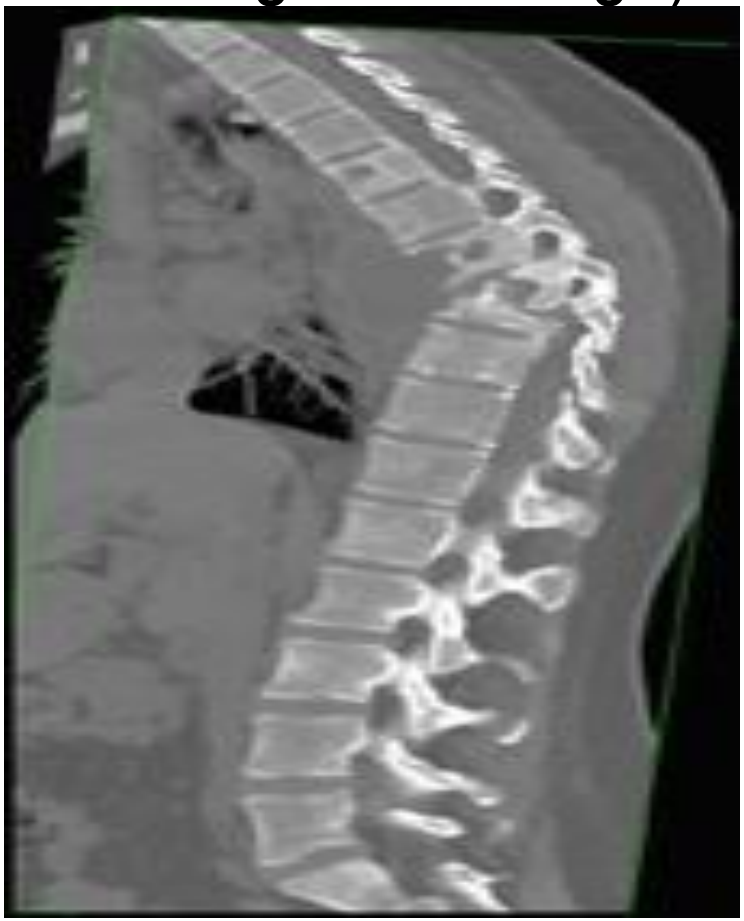


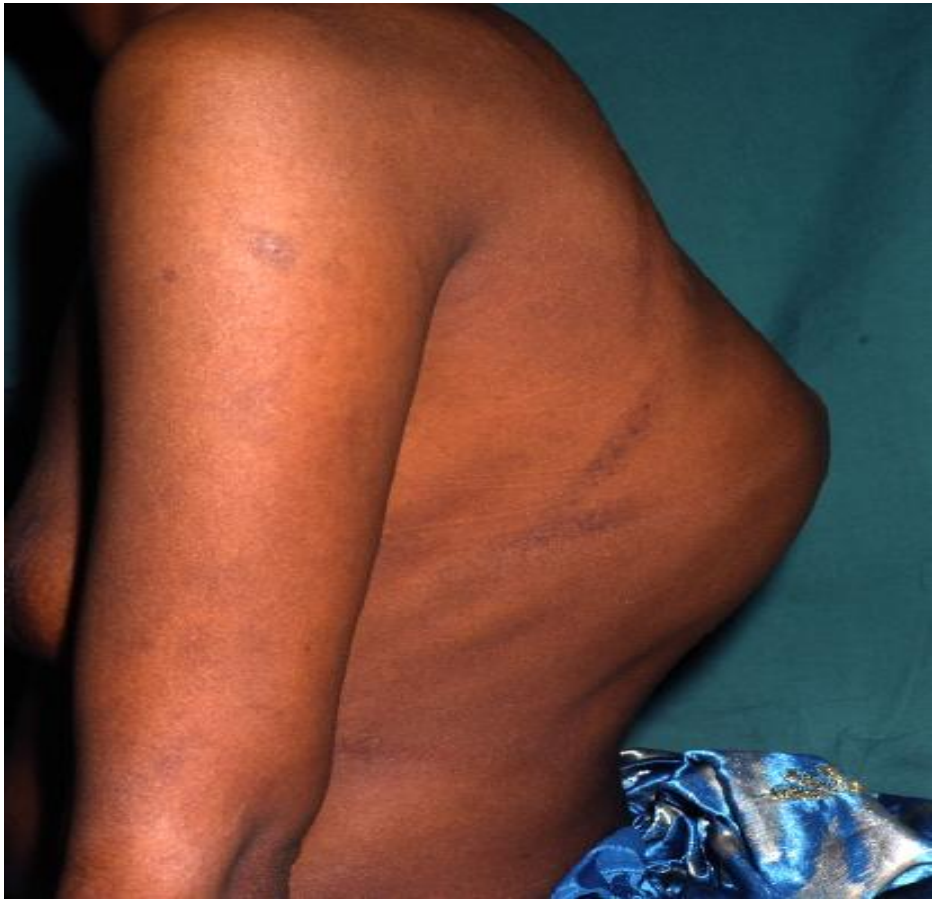


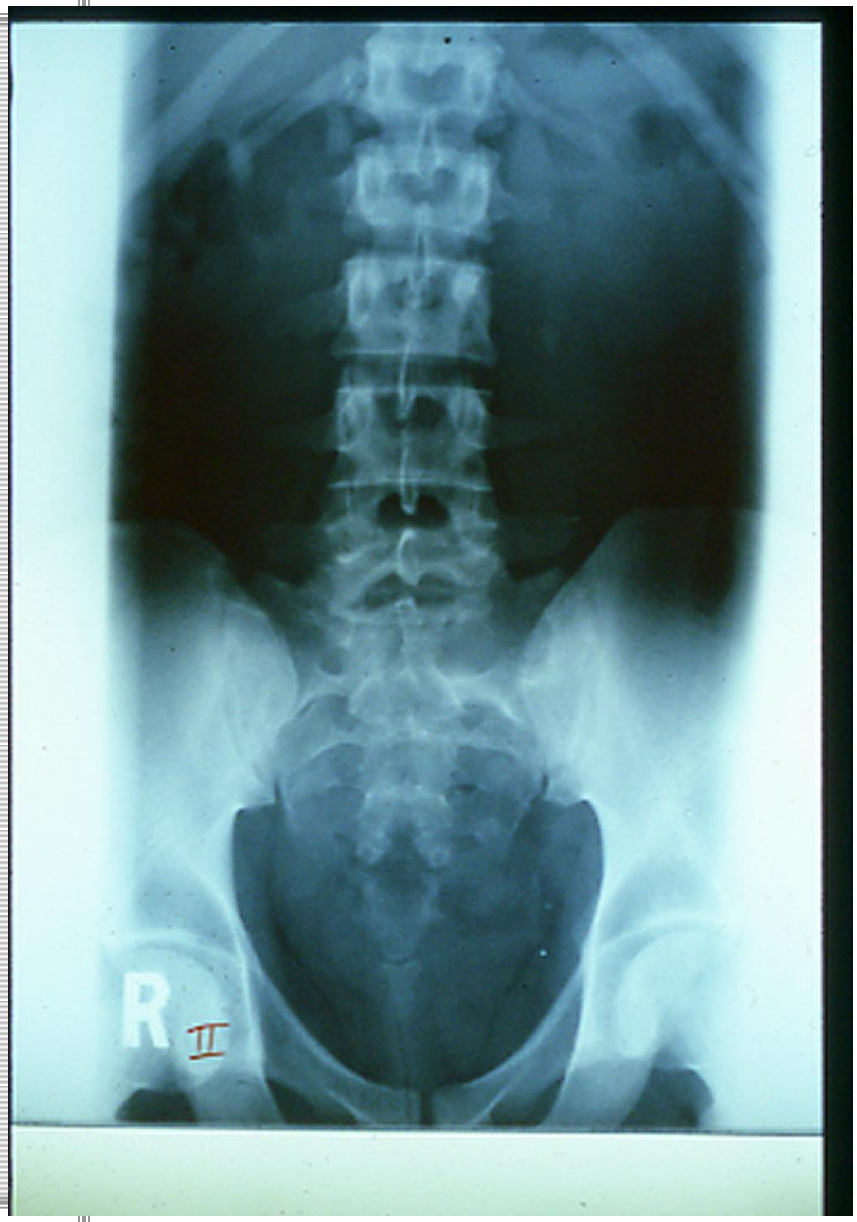


What will happen if treatment delayed? – gibbus formation (acute angulation of spine with or without neurological damage)



The physical appearance – Potts disease of spine - gibbus

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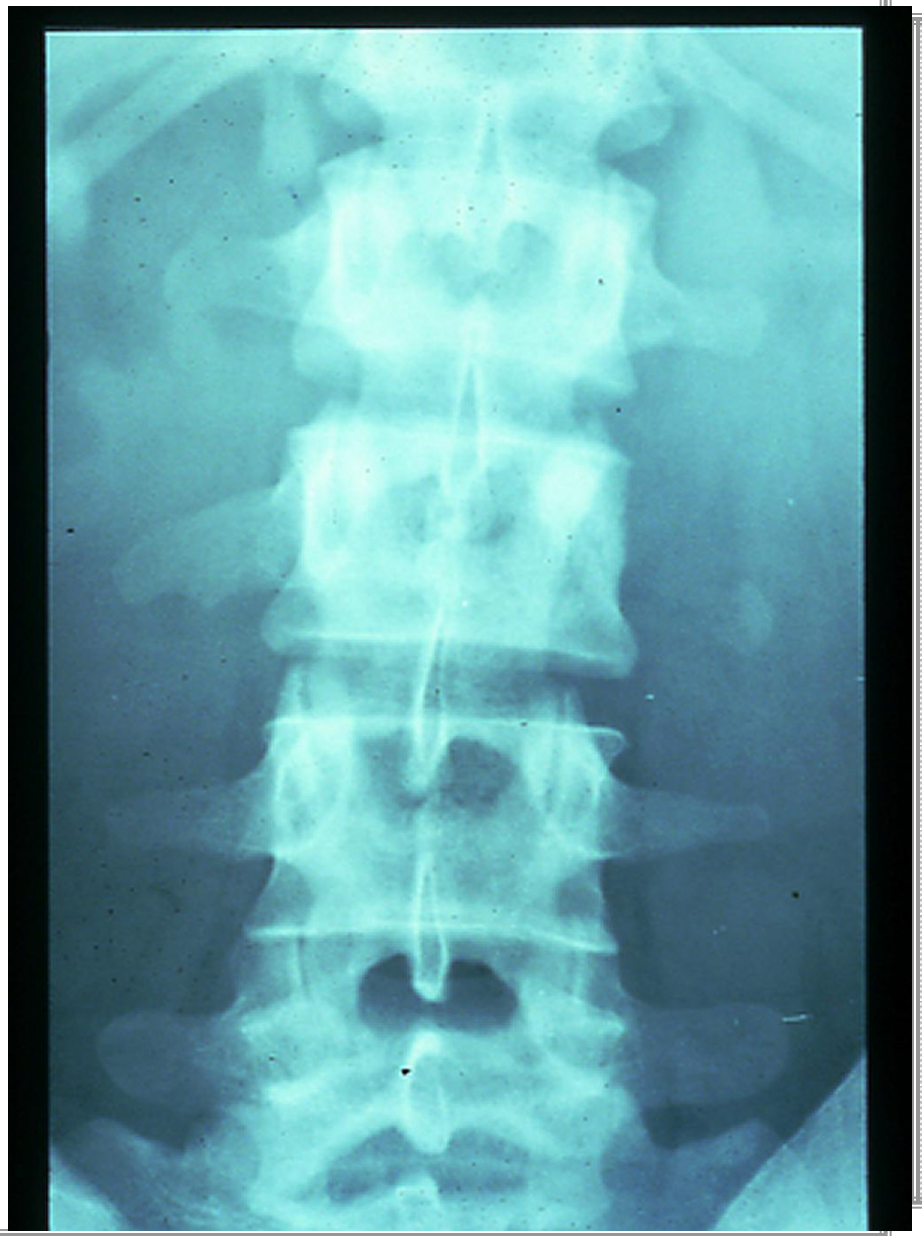
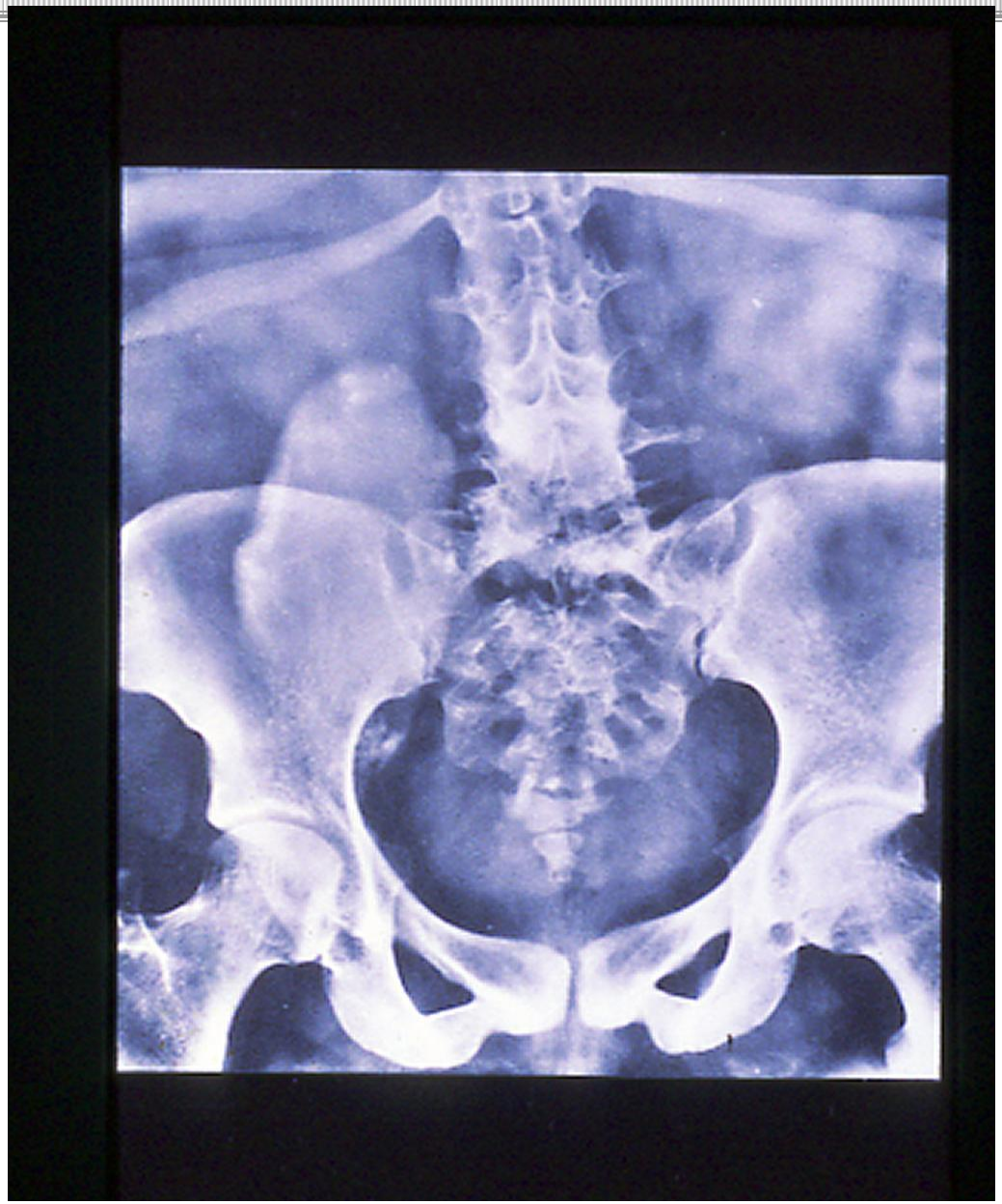




Types of specimens:

-Sputum.

- BAL.

-Pleural effusions

- Urine

- Stool

-CSF

-Aspiration (gastric – cold abscess)

- Blood in case of haematogenous TB

Diagnosis of TB

Clinical features are not confirmatory.

Zeil Nielson Stain - $1x10^4$/ml, 60% sensitivity

Release of acid-fast bacilli from cavities intermittent.

3 negative smears to assure low infectivity*

Culture most sensitive and specific test.

- Conventional Lowenstein Jensen media 3-6 wks.
- Automated techniques within 9-16 days

PCR is available, but should only be performed by experienced laboratories

PPD for clinical activity / exposure sometime in life.

PPD Tuberculin Testing

Sub cutaneous

Weal formation

Itching – no scratch.

Read after 72 hours.

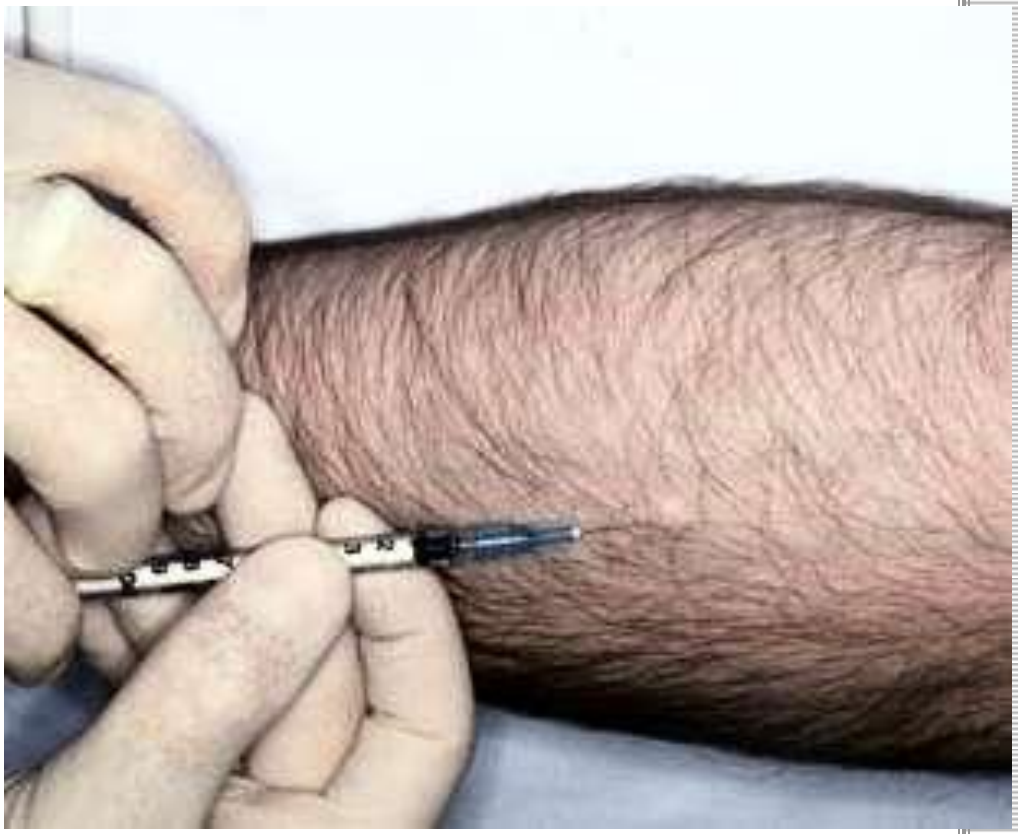
Induration size.

5-10-15mm (non-ende)

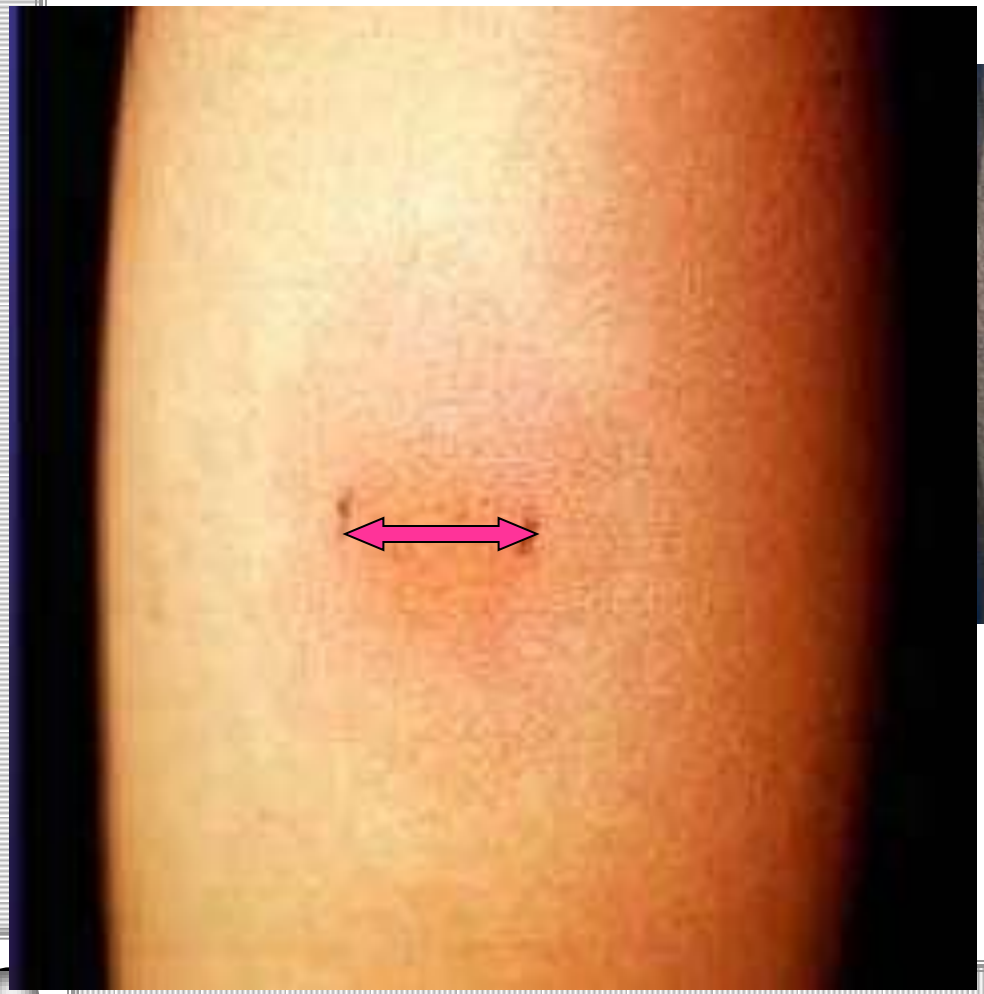
< 72 hour is not diag*

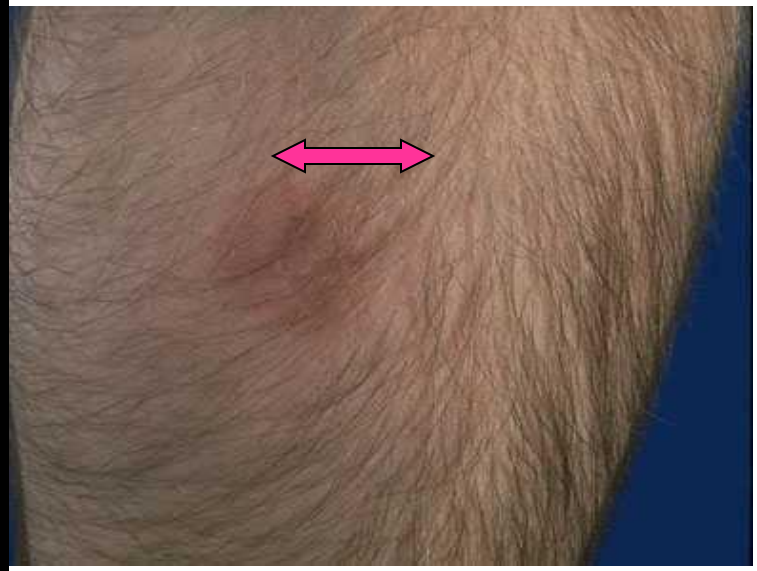
+ve after 2-4 weeks.

BCG gives + result.



PPD result after – 72 hours.





Granuloma or LH giant cell is not pathagnomonic of TB…!

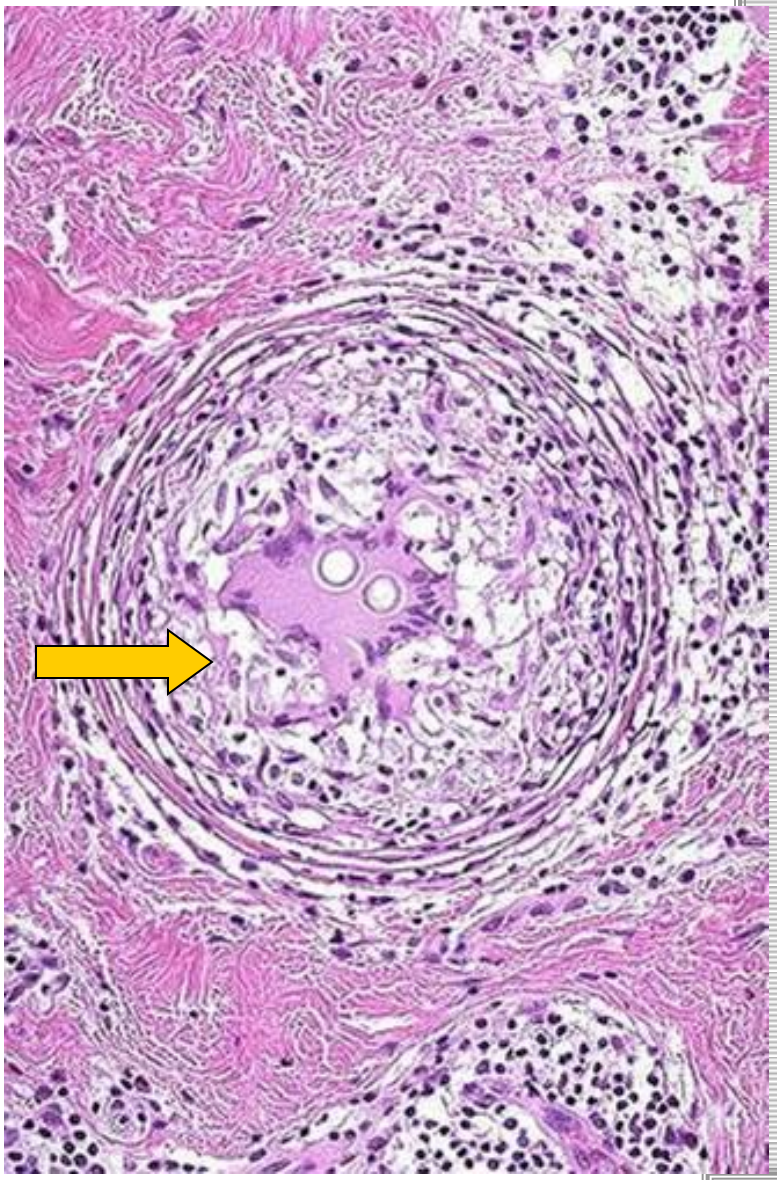
Foreign body granuloma.

Fat necrosis.

Fungal infections.

Sarcoidosis.

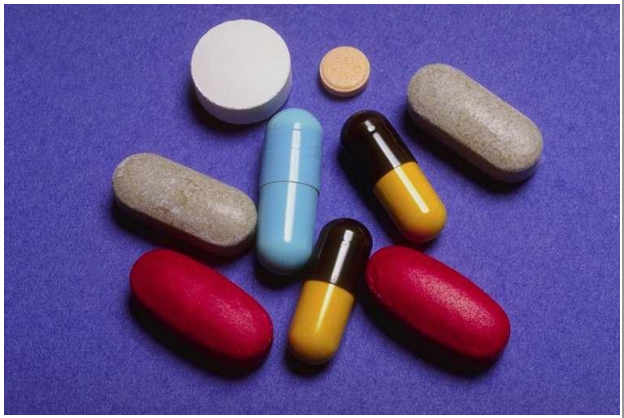
Crohns disease.



Treatment

Antibacterial chemotherapy:

- Combination of **first** and **second line** drugs for the first 2 months which could include:
  - **Isoniazid**
  - **Rifampicin**
- **Pyrazinamide**
  - **Streptomycin** or **Ethambutol**
- Next 4 months, combination of:
  - **Isoniazid**
  - **Rifampicin**



such as **ethambutol**, **streptomycin**, **pyrazinamide** and **fluoroquinolones** can be added to drug arsenal (treatment period also extended).

- These drugs are relatively effective in killing the bacteria, however, they also produce a wide variety of side effects.

First line drugs:

- **Bactericidal agents**: kill active bacteria, important in the early stages of infection.

Second line drugs:

- **Bacteriostatic**: hinder bacterial growth.
  - Strengthen treatment in the case of resistant bacteria.
  - Less efficient and generally more toxic than first line drugs.

Inappropriate chemotherapy:

- Monotherapy (single drug treatment)
- Decreased treatment period
- Low absorption of drugs

Conclusions:

Chronic, Mycobacterial, infection - Weight loss, fever, night sweats, lung damage.

Commonest fatal infection in the world.

CXR - apical lesions (CXR atypical AIDS)

AIDS, Diabetes, malnutrition & crowding.

Two forms Primary, Secondary

Pulmonary, extrapulmonary, miliary.

AFB positivity - infectiousness - isolation

Multi drug to prevent selection of resistance

Prevention depends on PPD & INH prophylaxis

What is New…?

14-30% of TB patients also HIV infected.

New drugs - Rifapentine, Interferons, Thalidomide.

Immune therapy : Killed M. vaccine stimulates CD8 cells (increased INF and IL-12).

The genome of TB has been identified (~4000 genes) potential to develop new vaccines and tests.

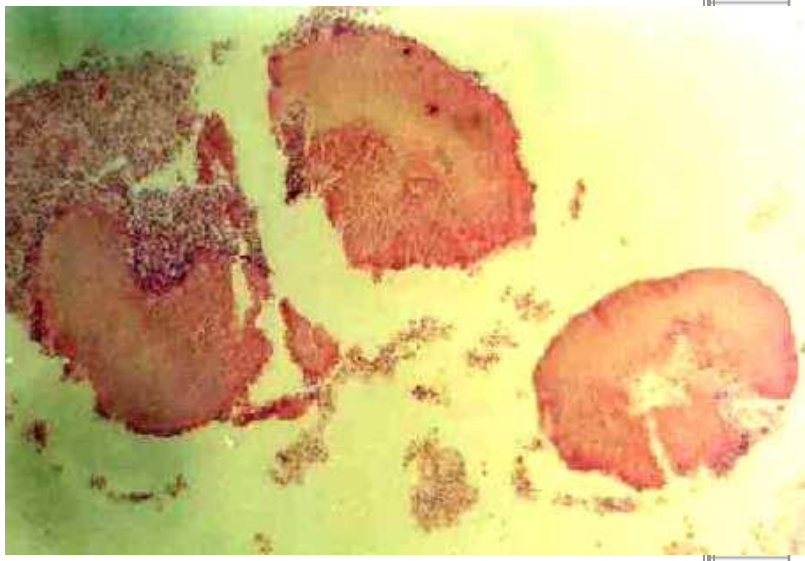
Actinomyces

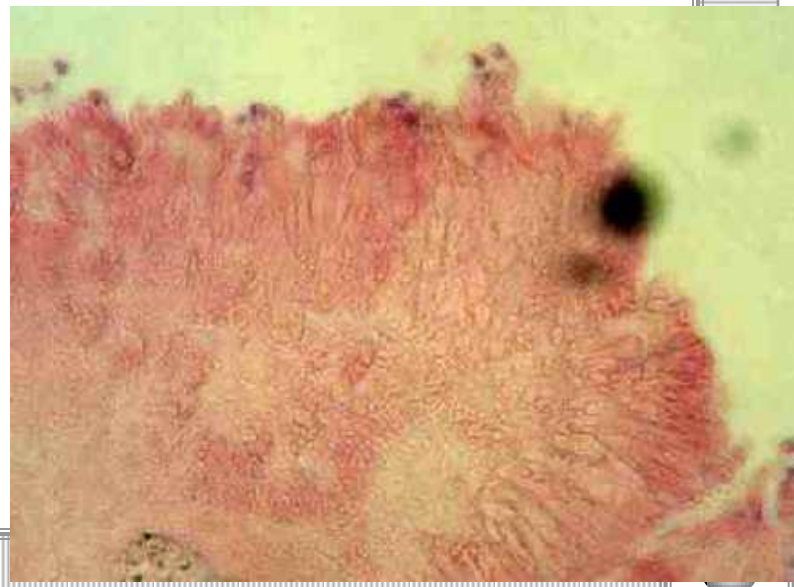
Actinomyces

A.israelii, A.naeslundii;

Gram positive; sulfur granule;

Related to decay tooth and peridentitis





ACTINOMYCES

Anaerobic, filamentous, gram positive bacillus

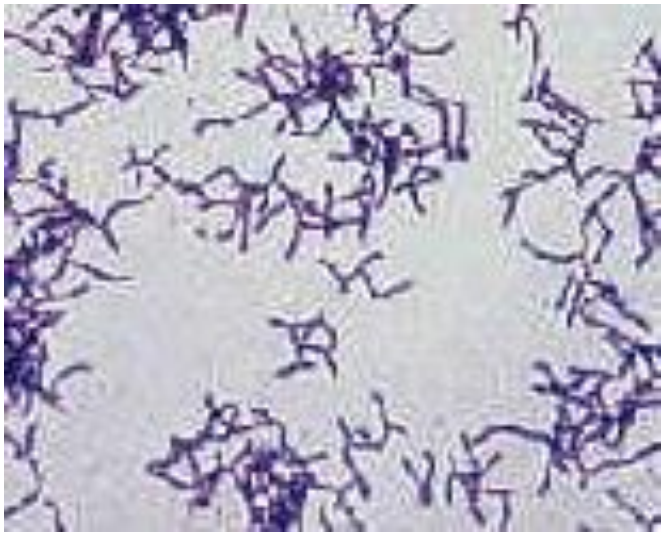
– *Exhibit true branching*

"Mykes" – Greek for "fungus"

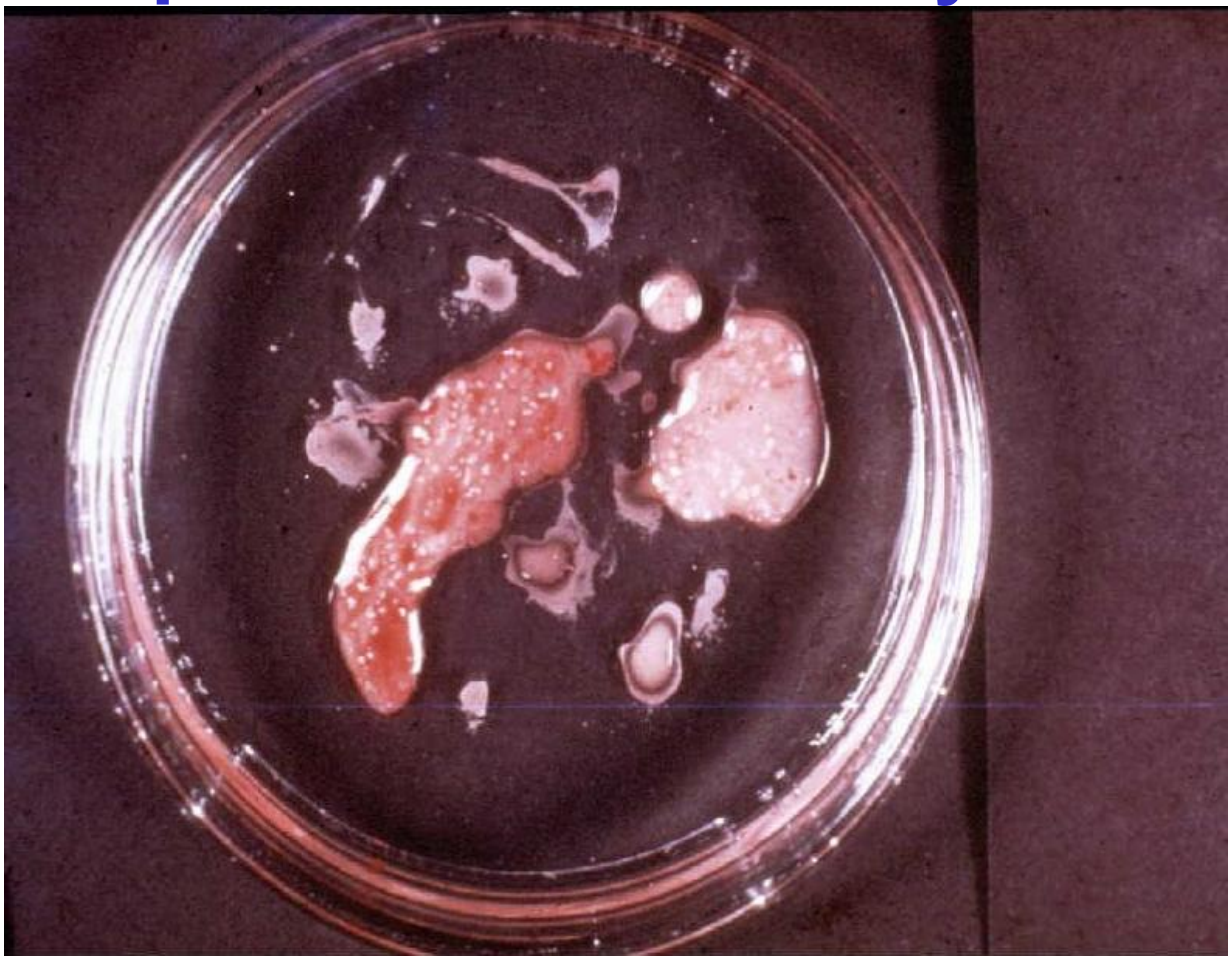
Thought by early microbiologist to be fungi because of:

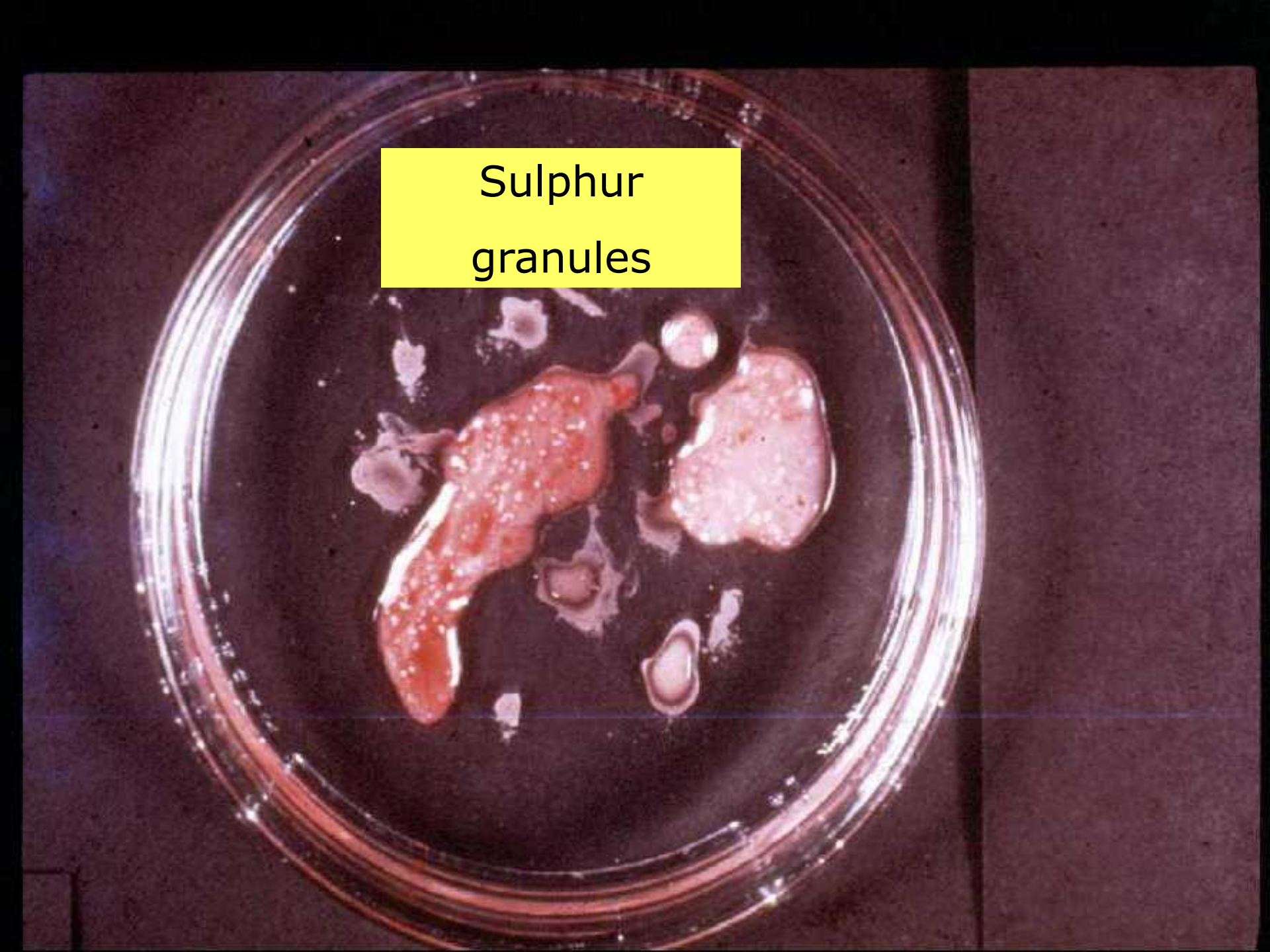
– *Morphology*

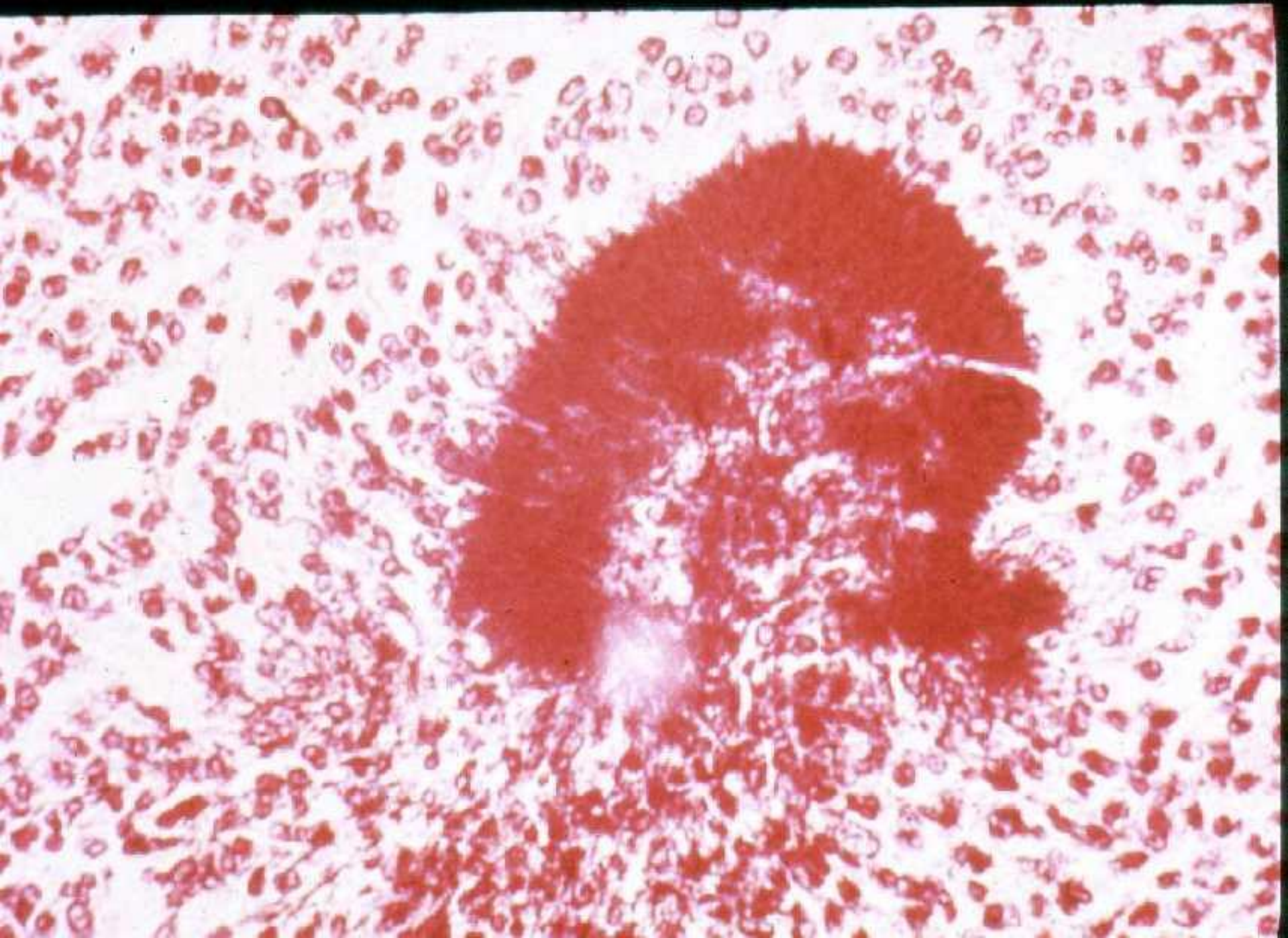
– *Disease they cause*



Clinical specimens:Actinomycosis-pus



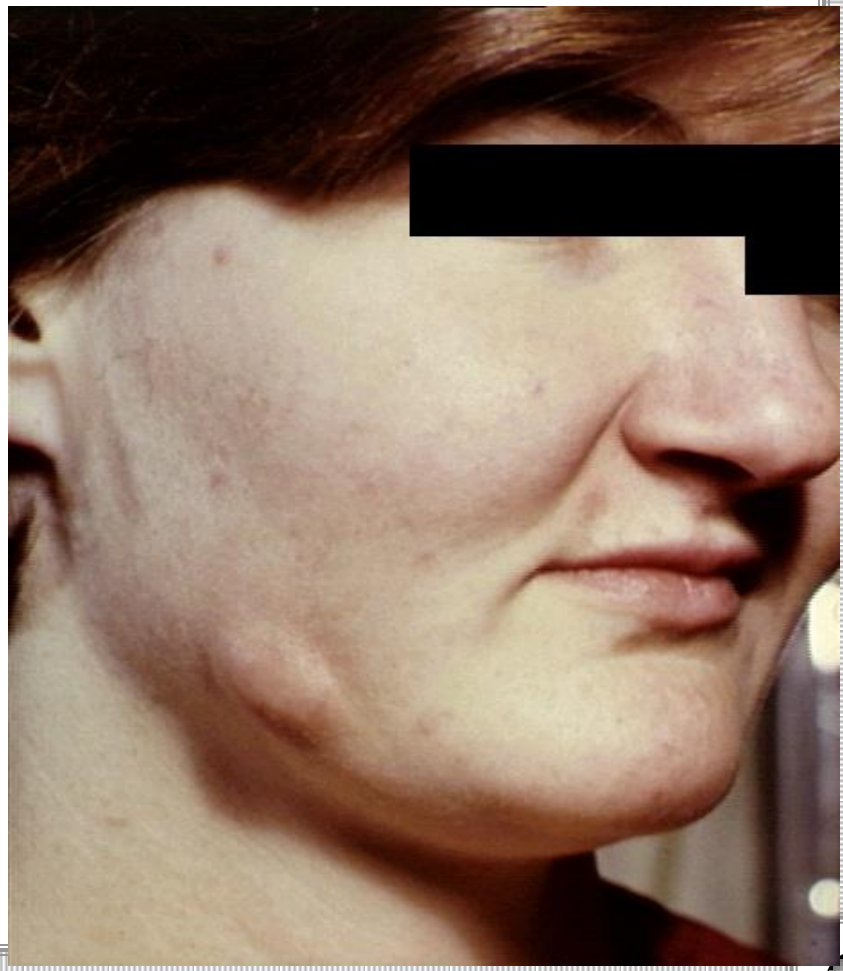

Sulphur
granules



ACTINOMYCOSIS

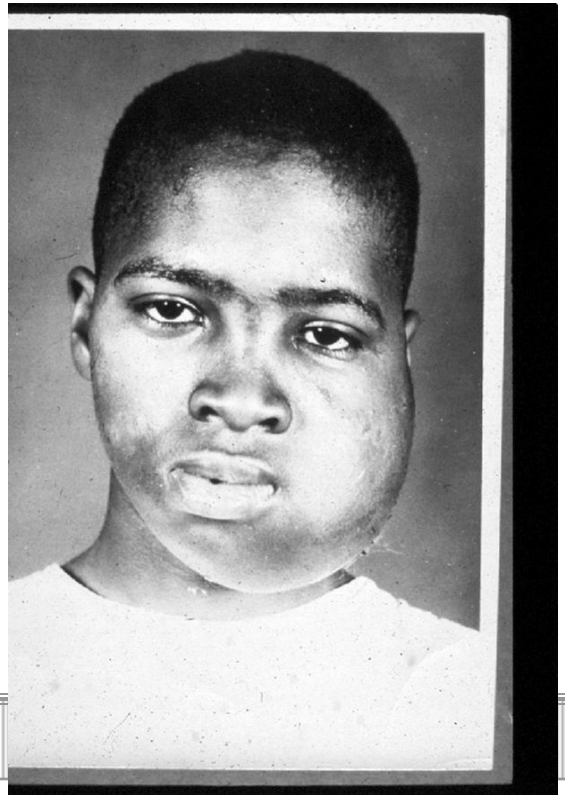
Not highly virulent (Opportunist)

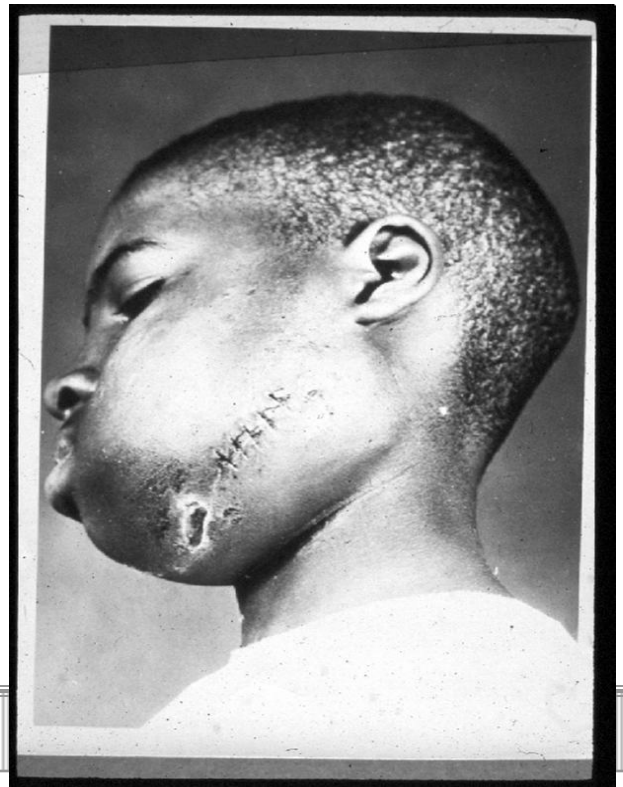
- Component of Oral Flora
  - Periodontal pockets
  - Dental plaque
  - Tonsilar crypts
- Take advantage of injury to penetrate mucosal barriers
  - Coincident infection
  - Trauma
  - Surgery



Actinomycosis

A chronic suppurative and granulomatous disease of the cervico-facial, thoracic or abdominal areas





ACTINOMYCOSIS

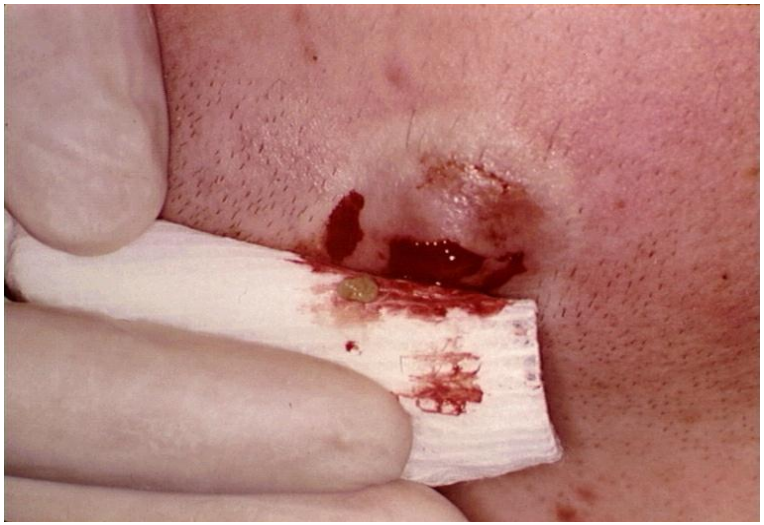
Form indurated masses with fibrous walls and central loculations with pus

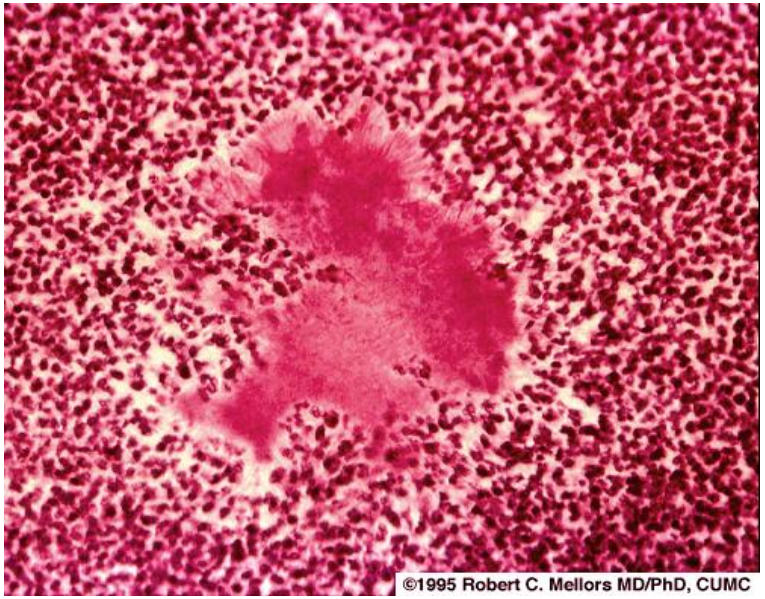
- Pus contains "Sulfur Granules"
  - Gritty, yellow white
  - Average diameter - 2mm
  - Composed of mineralized “mycelial” mass

Chronic infection

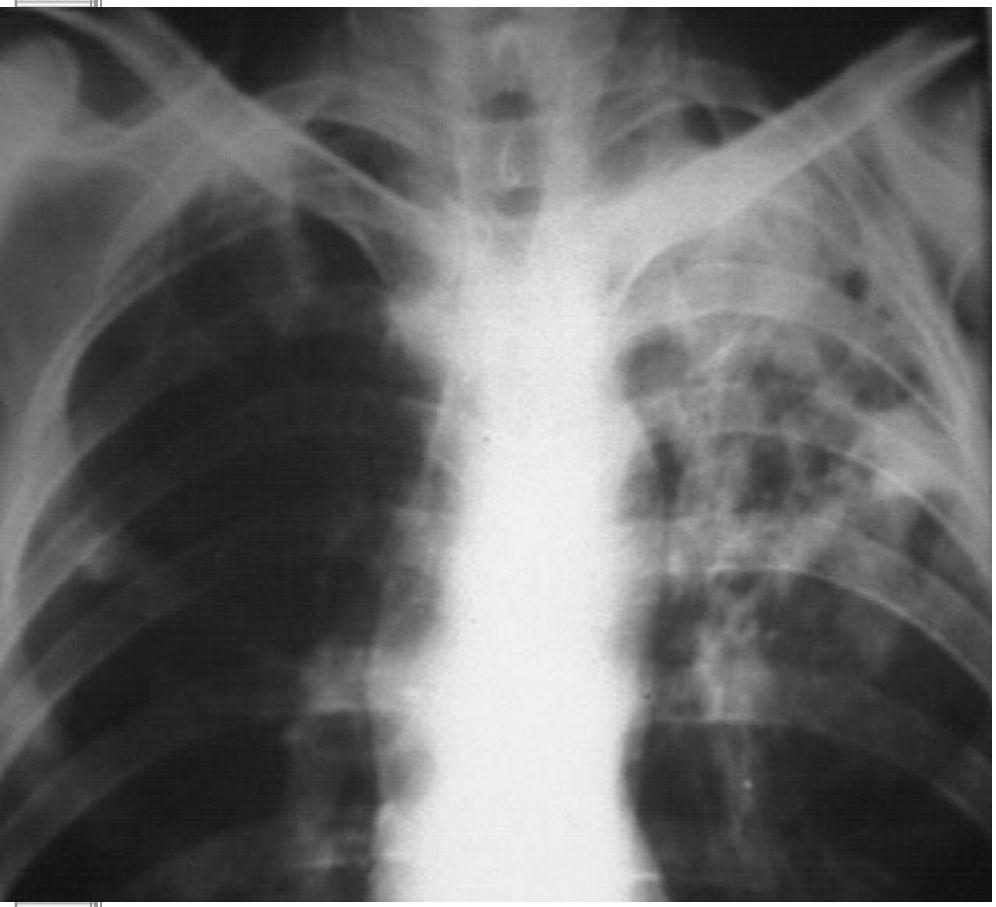
- Form burrowing sinus tracts to skin or mucus membranes
  - Discharge purulent material

Actinomycosis - sulfur granule



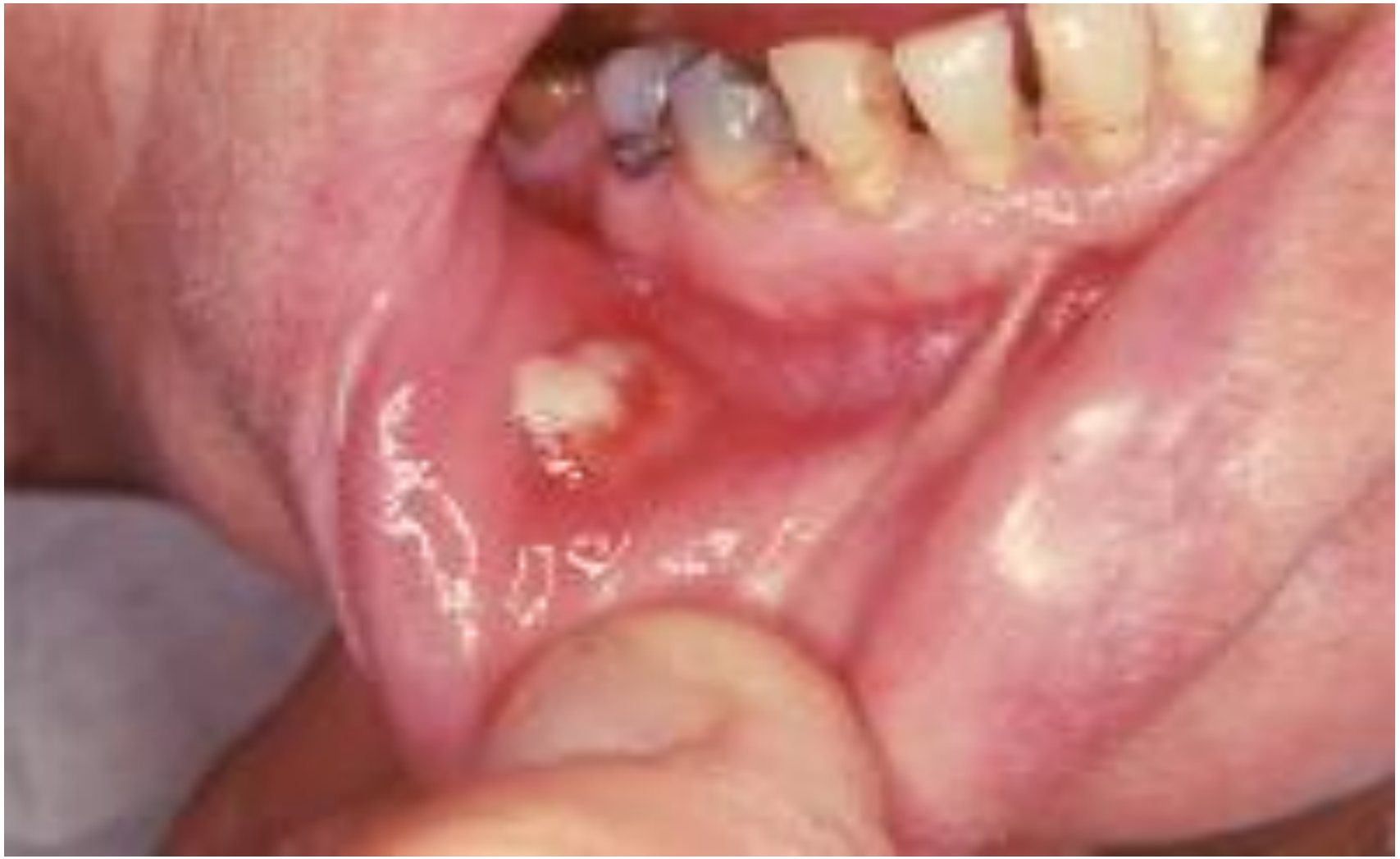


Pulmonary Actinomycosis



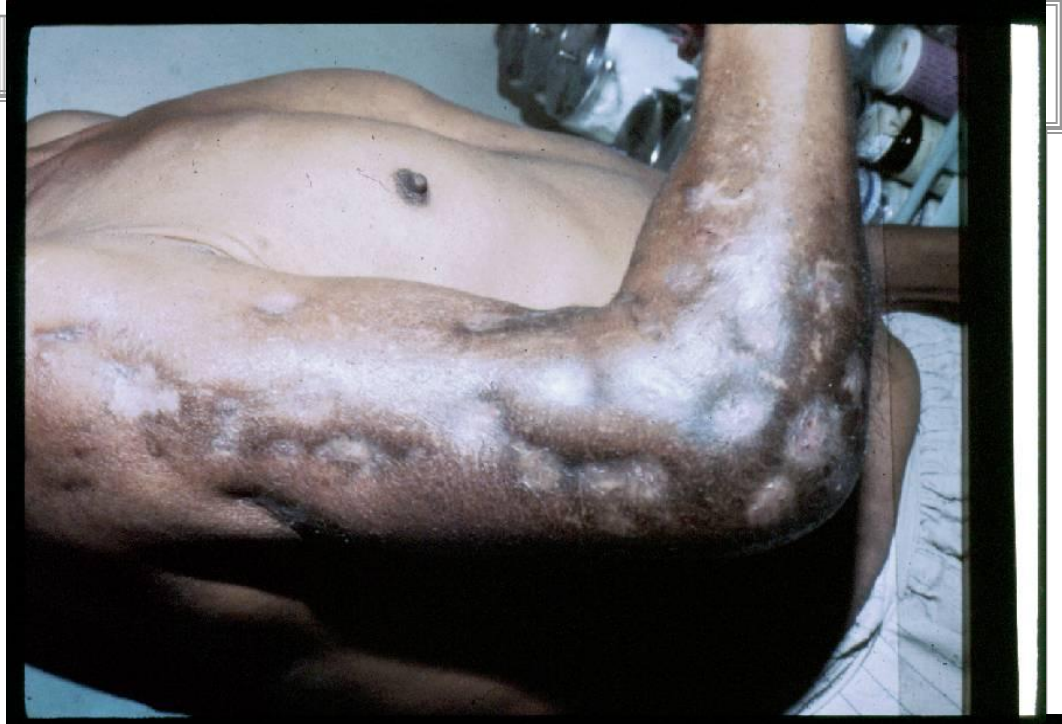
- 15% of cases
- Aspiration of organism from the oropaharynx
- Slowly progressive process involving lung and pleura
  - May be mistaken for malignancy
- Chest pain, fever, wgt loss and hemoptysis

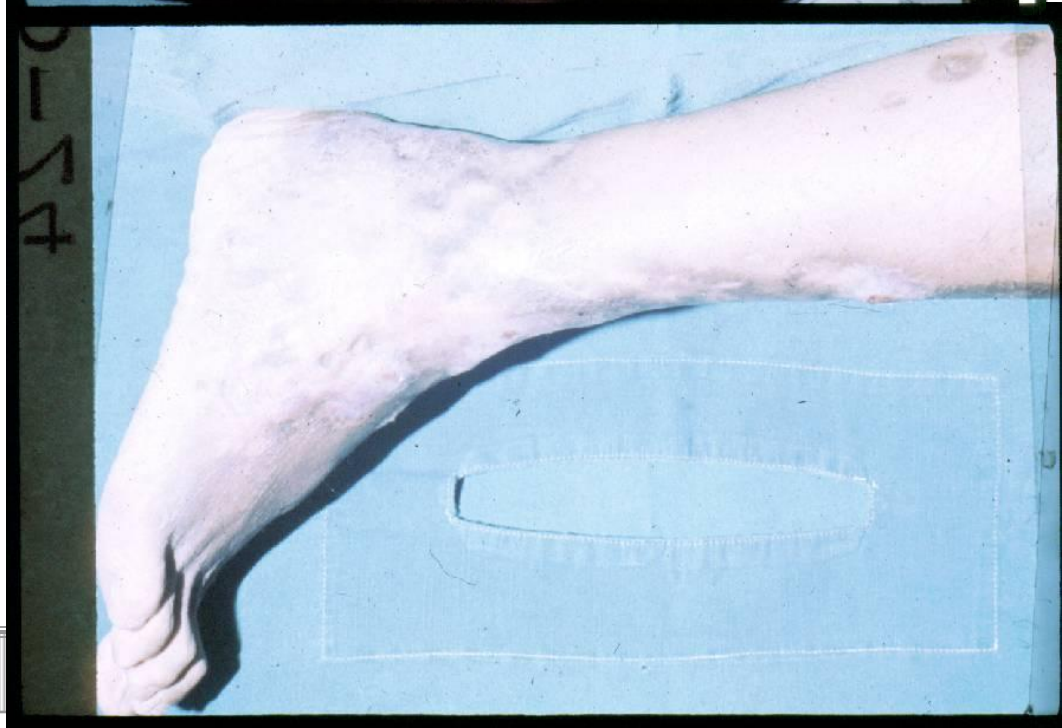
The Cervicofacial type



Mycetoma

Streptomyces species?

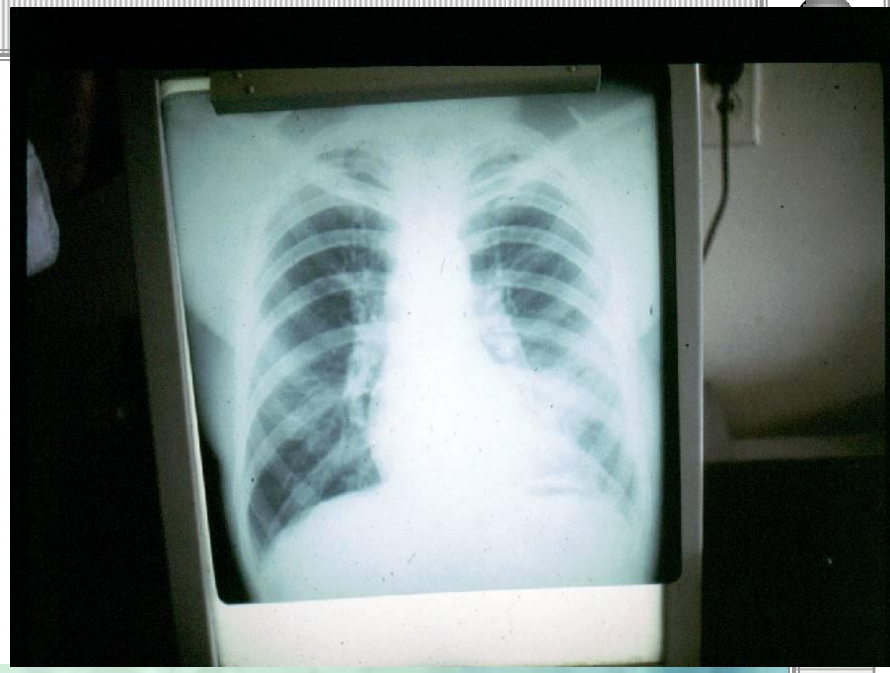


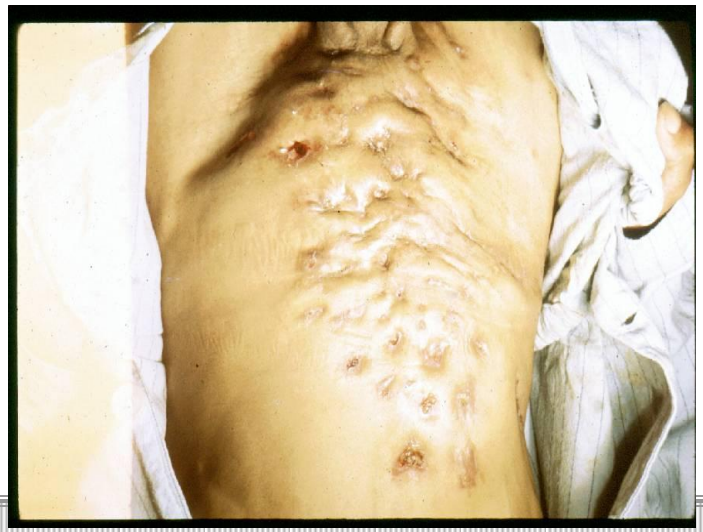


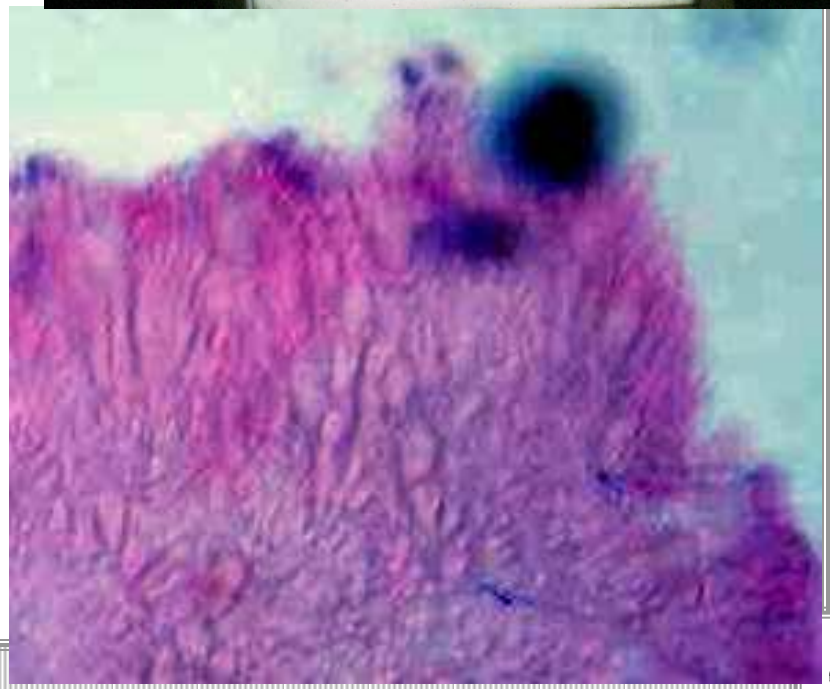
Nocardiosis

Subcutaneous infections, pulmonary infections, and brain abscesses

N. asteroides; N. brasiliensis







Complications:

Osteomyelitis(although not common).

Otitis media.

Meningitis.

Lung infections.

Laryngeal infections(rare0

"Troubles are often the tools by which God fashions us for better things." Exams…!

- Henry Ward Beecher

Thank you