GRANULOMATOUS PNEUMONIA

TUBERCULOSE

There are types of M. tuberculosis.

Human type affects humans, cattle; does not affect birds

M. tuberculosis bovis human bovine pig, sometimes affects the dog cat, horse sheep

M. tuberculosis avium sometimes affects cattle sheep at cattle.

M. Tuberculosis. Hominis sometimes affects monkeys, cattle dogs.

Mycolic acid provides acid resistance to the agent.

Lipid Increases the resistance of the agent. It increases the pathogenicity.

It keeps macrophages alive for 10 days. If the body is resistant or has already taken the causative agent, it is digested in macrophages.

If the body is resistant, they continue to reproduce and the disease is shaped.

Tuberculoprotein causes hypersensitivity.

Pathogenesis

Where it first enters, neutrophil is covered by leucocytes.

However, the lipid substance cannot be eliminated by neutrophil leucocytes.
Therefore, the agent is taken by macrophages in the region.
It maintains its viability for 10 days in macrophages.

In this period, the organism is sensitive and
how to develop a reaction.

Therefore, the organism presents lesions in different forms and patterns.

If the organism is strong and the condition is weak (dose, pathogenicity is low), the productive type of inflammation develops.

If the resistance of the organism is weak, if it is strong, the exudative type of inflammation develops.

Exudative tuberculose

Neutrophil begins with leukocyte and partly with fibrin exudation. With the Ziehl-Nelseen special paint method, small red rods are also found in these regions.

Subsequently, these areas undergo caseification necrosis. In the necrosis area, the basic structure of the tissue is selected in part. This is more evident in lung tissue rich in elastic yarns. The alveoli boundaries are further clarified by the application of the dyeing process, especially the dyeing of elastic yarns such as van Gieson.

This type of necrosis, in which the basic structure of the tissue can also be selected, is considered as the ild primary caseification necrosis .

Productive Tuberculose

Granulomatous inflammation from proliferative infections. Its appearance is typical and it is diagnosed by histological examination.

Caseification necrosis and post-calcification on the centrum; It is characterized by a capsule of connective tissue cells with histiocytes, epithelioid histiocytes and Langhas type giant cells in the outermost region (this is the last generation of granular tissue rich in capillary and connective tissue cells).

Produktif begins with macrophage (histiocyte) infiltration to the site of tuberculosis agent.

In these areas epitheloid histiocytes and Langhas type giant cells appear at the time. Among them, there are a large number of plasma cells and lymphocytes among them. These regions are surrounded by the aforementioned capsule. After a while, caecification necrosis is formed in the middle. In the area of necrosis, tissue structure is not chosen as before. This type of caseification necrosis in productive tuberculosis is a secondary caseification necrosis.

Milier tuberculose

It is prominent in the early and late generalization of tuberculosis. It is more common in the areas where the lung is not well aired.

It has a productive structure.
Transparent begins in small lesions; then fuzzy yellowish-brown milier turns into tubercles.

The microscopic cyst is rich in epithelioid cells. In this respect, it is called “epitheloid cell tubercle .

After entering the organism, the tuberculosis agent draws different tables according to the resistance of the organism, the factor and the time elapsed and is named according to these.

Primary infect or Ghon focus

where tuberculosis bacilli first enters, it emerges with the findings previously described.
(Ghon = Anton GHON, pathologist, 1866-1936)

Primer complex (Ghon complex)

It is formed by spreading to the region lymph nodes with primary infect.

Incomplete primer complex

recovery of primary infect; only the area is characterized by the presence of a lesion in the lymph nodes.

The first period of infection

Primer complex
and
Early generalization
Produktif tbc

Exudate TBC

Late generalization
Milier (productive)

Exudative, glopan tbc
lesions with
occur.

Postprimer infection period

Tuberculosis occurs after the first infection is overcome.

Tuberculosis of chronic organ,
open organ (lung) tuberculosis
as well as private names.
YOU HAVE SPECIAL FINDINGS!
Develops in two ways

Reinfection: Tuberculosis in the body of survivors of tuberculosis

Superinfection = Infected tuberculosis from outside with the TBc.

Transmission (Generalization)

Hematogen

This is the generalization of the body.

The result of intensive bacteraemia

larger and unified conglomerate tubercles are observed in the organs with the same age and size as the milier tubercles or their combination.

Occasional recurrent bacteremia

Different age and size (nodular militer) tubercles are encountered.

Lymphogenesis

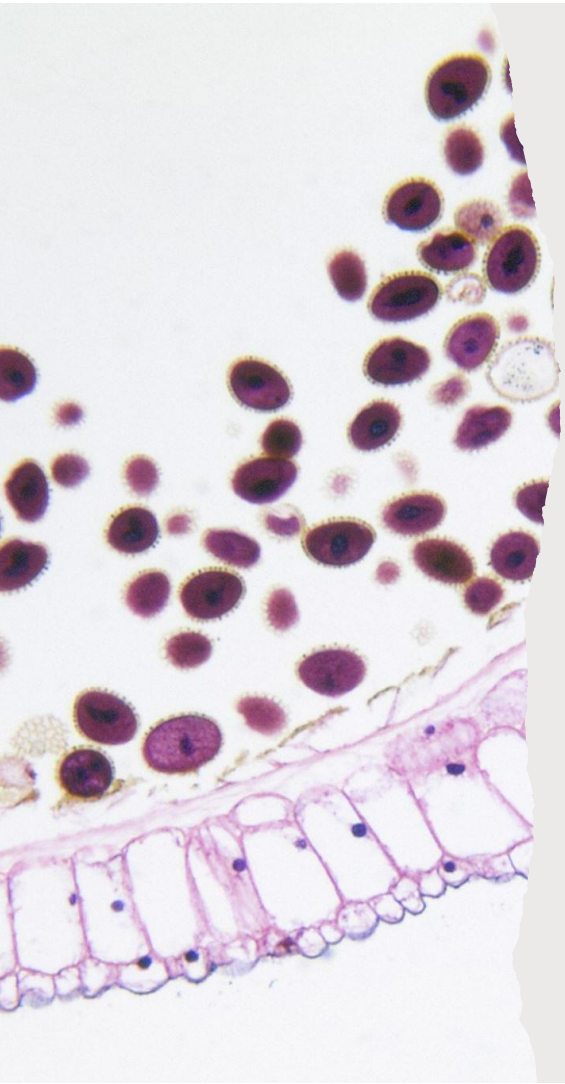
Propagated by lymph vessels. In particular, there is a retrograde lymphogen expansion. Infection of macrophages is transported from the lymph nodes to other regions in the opposite direction

Contact spread

One focus is transported to the environment by mocrophages. In this case, satellite lesions occur around the main lesion.

Canalicular spread

Chronic organ tuberculosis is characteristic. Moved through channels. For example, bronchus, bronchiol, trachea in the lung; Propagated by bile ducts in the liver



BOVINE TUBERCULOSIS

Lesions are generally on the lung and young animals.

1. FIRST INFECTION PERIOD

Primary focus

It is often located under the pleura in the diaphragmatic lobe.

From peas to fist size; caseification, later calcified and encapsulated in the form of tubercle.

It can also be found around milier, resorptive tubers.

- Microscopical Findings

- In the middle, the secondary caseicization surrounding the necrosis was surrounded by lymphocytes. Histiocytes, epitheloid histiocytes, Langhans giant cells are few. Caseification necrosis is subsequently calcified.
-
-
- All these regions are surrounded by capsules of connective tissue cells (fibrositis, fibroblasts).

- Regional Lymph Nodes

- It has increased by volume (swollen); In the radier style, it includes TB lesions.

- Or it is of normal size and contains small caseification necrosis and calcification areas.

a. Hematogenous acute (rapid) milier tuberculosis lesions

b. Chronic (slow, delayed) generalisation

- **EARLY GENERALISATION**

- Early generalization occurs shortly after the primary tuberculosis infection, when the immune response has not yet fully developed, allowing the tubercle bacilli to enter the bloodstream and disseminate throughout the body. This stage usually appears within a few weeks or months following the initial infection.

EARLY GENERALISATION

- Dissemination takes place mainly via the hematogenous route, and occasionally through the lymphatic system. During this period, numerous small, millet seed–sized miliary tubercles form, particularly in poorly ventilated areas of the lungs and in the regional lymph nodes.

EARLY GENERALISATION

- The lesions are **productive in nature**, characterized by **epithelioid cells and Langhans-type giant cells** forming granulomatous structures. Initially translucent and glassy in appearance, these tubercles gradually become grayish-yellow and undergo caseation necrosis.

EARLY GENERALISATION

- Pathologically, although the immune system is not yet fully active, the host can partially limit bacterial proliferation, resulting in **small, organized, and often encapsulated lesions**. Consequently, early generalization tends to follow a **subacute or mildly chronic course**.

POSTPRIMARY INFECTION ! CHRONIC ORGAN TUBERCULOSIS !

- **Reinfection** develops as a result of hyperinfection, with the help of an allergic (hypergic) reaction.

- Tuberculosis of chronic organ is known as «open organ tuberculosis» in humans; It is the third period of tuberculosis in humans.

POSTPRIMARY INFECTION !
CHRONIC ORGAN TUBERCULOSIS !
Character of lesions

1. **Acinose (nodous tuberculosis lesions (foyers)**
2. **It spreads through canals.**
3. **Caverns are formed.**
4. **Bronch and trachea, pharynx, miler or fungous tubercles (nodes) in the mucosa of the larynx and ulcers are formed by opening them.**
5. **There are no changes in this type of lymph nodes in the lymph nodes. If any, belong to previous spread.**

POSTPRIMARY INFECTION !
CHRONIC ORGAN TUBERCULOSIS !

Character of lesions

1. **Acinose (nodous tuberculosis lesions (foyers)**

The lesion is located in the caudal upper part of the diaphragmatic region.
First, asinous foyers are formed.

Exudate accumulates in the alveoli where terminal bronches terminate and these areas are prominent in the appearance of yellowish cloverleaf.

The nodous foyers, on the other hand, are made up of large bulging nodules by spreading them. In addition, they are combined with lobar areas.

2. It spreads through canals.
The interior of the bronzes is full of bulky masses.

3.Caverns are formed.
There are two types of cavern formation.

a. Bronchiectasic cavern
Bronchlar expanses due to the excavation of the bronches, etk ectasie'ye suffers Bron. When the exudate is emptied, the cavern is formed in the ıyla hollow inde shape. It is noted that they were surrounded by a bronch wall. Such caverns are typical of cattle in particular.

b. Melting caverns
It is typical in humans. It forms in the tissue with the melting of Acinous-nodose foyers. Causes bleeding.

4. Bronch and trachea, pharynx, miler or fungous tubercles (nodes) in the mucosa of the larynx and **ulcers** are formed by opening them.

5. There are **no changes in this type of lymph nodes in the lymph nodes.** If any, belong to previous spread.

LATE GENERALIZATION

- Late generalization represents the stage in which the tuberculosis infection becomes chronic and a secondary wave of dissemination occurs due to renewed bacteremia, that is, the re-entry of bacilli into the bloodstream.
- This process usually develops during the **post-primary period**, when the immune system has already been sensitized but the disease reactivates. Dissemination may occur again **through the hematogenous route**, or **through the canalicular route** via the bronchi.

LATE GENERALIZATION

- The lesions at this stage are typically **caseous, exudative, and extensive** in character. Over time, these foci may **coalesce to form lobular areas**, showing **widespread necrosis, calcification, and fibrosis**.
- The lesions are not confined to certain parts of the lung but generally involve **all lobes** and may also spread to **other organs**, such as the kidneys and liver. The disease course may be **rapidly progressive (galloping form)** or **slowly evolving**, but in all cases, it remains **progressive and destructive**.

LATE GENERALIZATION

- Pathologically, the immune system is unable to completely control the infection at this stage; **previously formed tubercles become reactivated**, and **new necrotic foci** appear. Consequently, both **old fibrotic and calcified lesions** and **newly developed necrotic ones** coexist in late generalization.

OTHER SPECIES

EQUIDAE

It is rare because it is resistant. When it is usually characterized by productive lesions.

Created by M. bovis. The agent often reaches the lung through the intestines.

Acute milier tuberculosis

Lung is bulging and empirical.

Milier nodes are spread over the lung and the number is higher.

Regional lymph nodes

Hard, white-white

Chronic generalized tuberculosis

Milier is mostly nodular. Kazeification necrosis and calcification are rare. The lymph nodes of the region are similar to the previous ones.

GOAT AND SHEEP

Tuberculosis is usually exudate in goats.
It has a limited spreading caseous pneumonia type.
The generalization is usually with bronches.

TB is rarely seen in sheep.

During the early generalization period, the lung is included by the capsule from the connective tissue; caseification and calcified nodules.

Regional lymph nodes also develop related changes.

CAT AND DOG

The agent M. bovis, M. tuberculosis

It's dangerous to infect humans.
Cat cattle are more gentle on the human type of dog.

Contamination

More digestion to cat;

the dog is by means of digestion and respiration.

OTHER TISSUE TUBERCULOSIS

PLEURITIS TUBERCULOSA

It is prone to spread to serous membranes (peritoneal pleuria) in cattle.

Hematogen and lymphogen come from the surrounding tissues in relation to early and late generalization.

It is secondary because it comes with metastasis.

It is seen in different ways.

1. Tuberculosis with pearl: Pleura is a characteristic form of peritoneal tuberculosis. Handle without handle; from lentils to the size of chickpeas, or in the form of calcified nodules. They are combined with each other to form a larger pleuritis tuberculosa nodosa or granulomatosa.

2. Pleuritis tuberculosis caseosa

Mostly follows chronic pulmonary tuberculosis
Caseified areas are in the form.
Occasionally, fibrin is also present.

3. Pleuritis serofibrinosa - purulenta tuberculosis

It is the type of exudative TB which is seen in main carnivores.

4. Pleuritis tuberculosis infiltrativa

Non-Caseified is a plaque-like granulation tissue.

PERITONITIS TUBERCULOSA

Congenital (omphologen)
Liver TBC
Ulcerative bowel from Tbc barssi
Metritis tbc'sonu
From lung to hematogea to pleura

IT IS SIMILAR TO PLEURITIS TUBERCULOSA

HEPATITIS TUBERCULOSA

Omphologen
Intestine (V. Porta)
Hematogen transmission

Milier nodular in the form of tbc

MYCOTIC PNEUMONIA (PNEUMOMYCOSIS)

- Pneumomycosis refers to **fungal infections of the lungs**.
  These infections are usually **granulomatous** and **inflammatory** in nature.
  In some cases—especially when the infective form is **spores** or **hyphae**—lesions may appear more **exudative**, depending on the **host's immune resistance**.
- Fungal agents reach the lungs by two main routes:
- **Aerogenous** (inhalation of spores)
- **Hematogenous** (spread from other organs via the bloodstream)
- **Macroscopic appearance:** nonspecific; cannot be diagnosed by gross morphology alone.
  Diagnosis is based on **microscopic features** and **mycological (culture) examination**.
- Most fungi causing pneumonia are **opportunistic pathogens**.
  They become pathogenic when host defense mechanisms are impaired, such as during:
- Other concurrent infections
- Prolonged antibiotic treatment

ASPERGILLOSIS

Causative agents:

- *Aspergillus fumigatus* (most pathogenic and common)
- *A. flavus*
- *A. niger*
- *A. nidulans*
- Common in **poultry and mammals**.
  Infection occurs by **inhalation of spores** or **hematogenous spread** from other organs.
  In mammals, it can also infect the **placenta**.

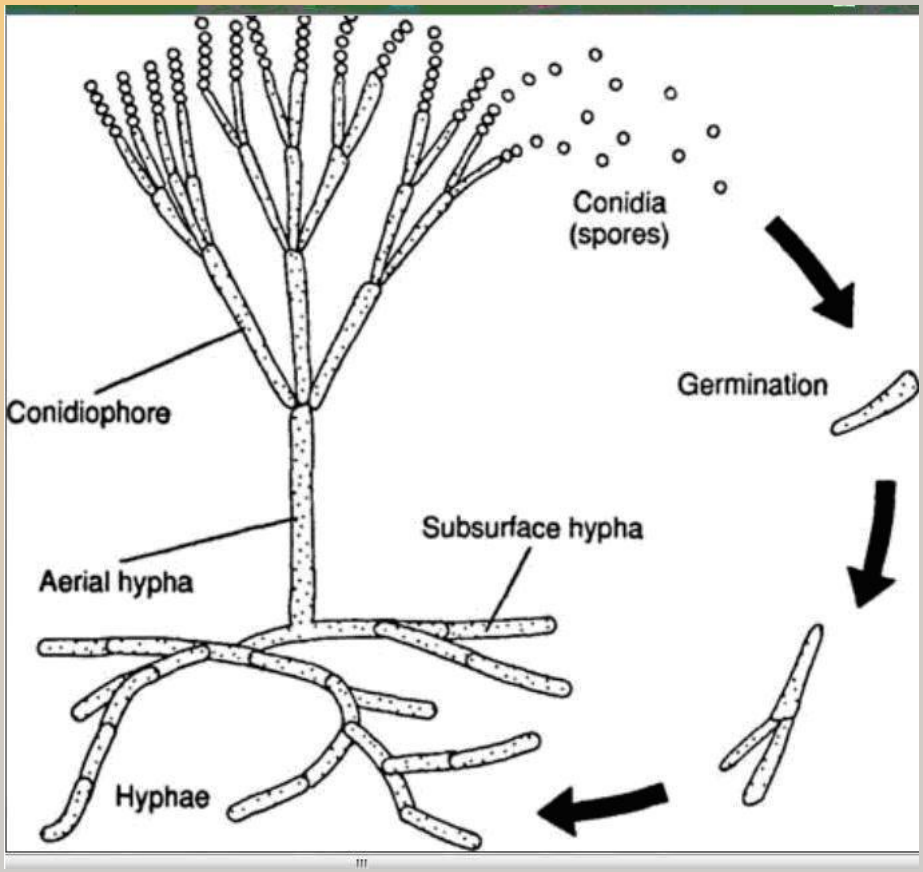
Pathogenesis and Lesions

- The fungus usually enters through the **bronchi** and **alveoli**, forming **focal lesions** which then spread throughout the lungs.

Acute Aspergillosis

- Characterized by **necrotic changes**.
- Lesions appear as **yellowish nodules** ranging from **lentil to walnut size**.
- Microscopically:
  - Central **necrosis** containing **conidiospores** and **hyphae**.
  - Surrounding **edema**, **leukocytic infiltration**, and **hemorrhage** in adjacent alveoli.
  - **Thrombosis** and **vascular invasion** are common.
  - **Bronchiectatic caverns** may form due to tissue destruction.

- **Chronic Aspergillosis**
- Characterized by **granulomatous inflammation**.
- Lesions develop into **capsulated nodules** (hazelnut to walnut size).
- Microscopically:
  - The fungus is surrounded by **macrophages**, **giant cells**, and **fibrous tissue**.
  - **Conidiospores** are diagnostic—commonly seen in **aerated regions** (bronchi, branch points).
  - In poorly ventilated areas, only **non-distinct hyphae** are observed.


Conidia
(spores)
Germination
Conidiophore
Subsurface hypha
Aerial hypha
Hyphae

HISTOPLASMOSIS

- **Causative agent:** *Histoplasma capsulatum.*
  It is **non-contagious** and spreads mainly by **inhalation** of spores from soil contaminated with bird or bat droppings.
  The infection may remain **localized in the lungs** or become **systemic**, spreading to other organs.
  It mainly affects **humans, dogs, cattle, horses, wild animals** (e.g., foxes), and **laboratory rodents**.

Forms of the Disease

- **Localized Histoplasmosis**
- The infection forms **capsulated nodules** that later undergo **calcification (liming)**.
- Histologically:
  - **Granulomatous inflammation** with **yeast-like organisms** inside macrophages and epithelioid cells.
  - Surrounded by **fibrous tissue** and **lymphocytic infiltration**.
- **Generalized Histoplasmosis**
- The **interstitial tissue** shows **plasma cells**, **lymphocytes**, and **epithelioid macrophages**.
- Numerous yeast-like organisms within macrophages.
- May spread to **liver, spleen, and bone marrow**.

PNEUMOCYSTOSIS

- **Causative agent:** *Pneumocystis carinii* (now *P. jirovecii*). Historically controversial — once thought to be a protozoan, but molecular studies show it is a **fungus**.
- **Characteristics**
- Causes **interstitial pneumonia**, not granulomatous.
- **Cysts** (5–7 μm) contain **1–4 μm trophozoites**.
- Stained by **PAS** or **GMS** stains (cysts appear black in GMS).
- **Trophozoites** attach to **type I alveolar epithelial cells**, causing damage.
- Microscopically:
  - **Pink fibrillar material** (excess surfactant) fills alveoli.
  - Alveoli contain **foamy exudate** with **cysts** visible under special stains.

ACTINOBACILLOSIS (Pneumonic Form)

- Although typically a disease of **soft tissues**, *Actinobacillus lignieresii* can cause **pulmonary pyogranulomatous inflammation**, especially in cattle and pigs.
- **Pathogenesis**
- May enter via **aerogenous** or **hematogenous** route.
- **Aerogenous infection:** affects apical and intermediate lobes; small abscesses may form.
- **Hematogenous infection:** affects all lobes, producing **walnut-sized abscesses**.
- **Microscopic Findings**
- **Pyogranulomatous inflammation**:
  - Center contains **necrosis and bacterial colonies**.
  - Surrounding **epithelioid macrophages**, **giant cells**, **lymphocytes**, and **fibroblasts**.
- Often resembles **granulomatous pneumonia** or **pleuropneumonia**.

BLASTOMYCOSIS

- **Causative agent:** *Blastomyces dermatitidis*.
  Occurs in **dogs** and **humans**.
- **Clinical Features**
- Causes **localized skin lesions** or **generalized systemic mycosis**.
- In dogs: starts in **lungs**, may disseminate to other organs.
- In humans: affects **skin and lungs**.

- **Pathological Findings**
- **Macroscopic:**
- **White nodules** scattered in all lung lobes and on pleura.
- Nodules may coalesce into larger masses.
- Liquefaction may occur, draining into bronchi or pleura (without pleuritis).
- **Granulomas and abscesses** may also form in regional lymph nodes.
- **Microscopic:**
- **Granulomatous and pyogranulomatous inflammation** with **Blastomyces yeast cells**.
- Two tendencies:
  - **Exudative type:** dominated by neutrophils, macrophages, and pyogranulomas.
  - **Proliferative type:** composed of epithelioid and histiocytic granulomas.
- **Systemic form:** similar lesions in **liver, kidney, spleen**, and lymph nodes.
  Cutaneous form: papules and abscesses forming draining lesions.

COCCIDIOIDOMYCOSIS

- **Causative agent:** *Coccidioides immitis*.
  Affects **humans**, **dogs**, **cattle**, and **sheep**.
  Primarily infects the **respiratory tract** (nose and lungs).
- **Diagnosis and Pathology**
- Organism forms **double-walled spherules** containing **endospores**, diagnostic of the disease.
- The **number of endospores** determines the **type and severity of inflammation**.
- Lesions can be focal or diffuse, with **necrosis and granulomatous response**.

CRYPTOCOCCOSIS

- **Causative agent:** *Cryptococcus neoformans*.
  Found in soil contaminated with **pigeon droppings**.
  Hosts include **dogs, pigs, cattle, horses, monkeys, and humans**.
- **Morphology**
- **Round or oval yeast cells** with a **thick mucinous capsule** made of mucopolysaccharides.
- In **H&E sections**, the capsule appears as a **clear halo** (unstained).
- **PAS** or **GMS stains** highlight the organism.
- **Pathogenesis**
- Acquired by **inhalation of spores** from the environment.
- Not transmitted directly between hosts.
- Causes **granulomatous inflammation** in **lungs** and **tracheobronchial lymph nodes**.
- May disseminate to the **central nervous system** and **other organs**.