Pathology of the lungs 1

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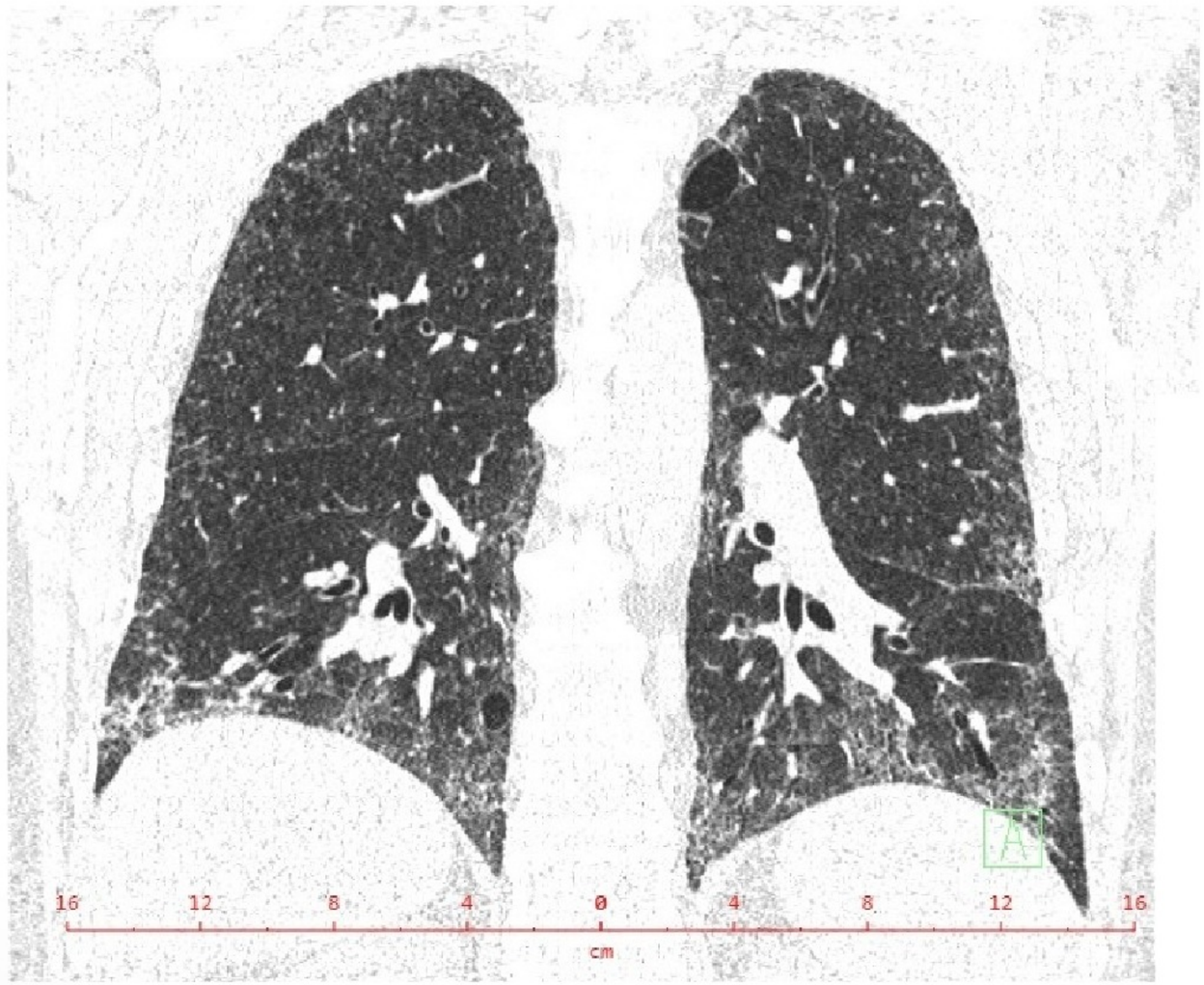

FN MOTOL

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Diseases of the lungs

1) maformations

2) inflammations

3) tumours

4) obstructive lung diseases

5) restrictive lung diseases

6) vascular lung diseases

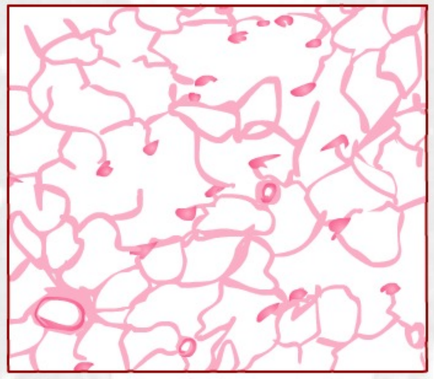
Inflammation of the lungs

Inflammation of the lungs

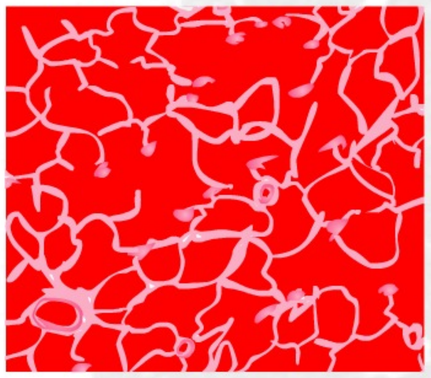
- very **common** (mainly infectious ones)
- high morbidity and **mortality** among risk groups

- **terminology** and **classification** has changed:

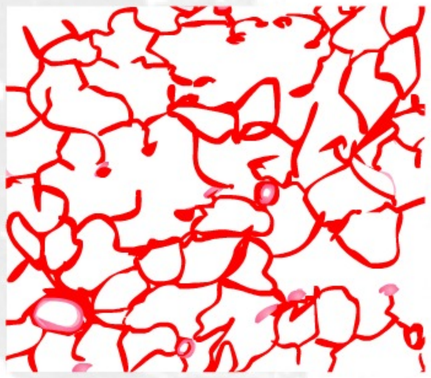
Hlava classification (pathological-radiological):



superficial



interstitial



acute

pneumonia

broncho-pneumonia

destructive pneumonia

pneumonitis

chronic

carnification

interstitial fibrosis

Clinical classification

- everything is labeled **pneumonia** now
(influence of ATB therapy changing virulence + morphology)

community-acquired pneumonia = immunocompetent p.
nosocomial pneumonia = hospitalized p.
opportunistic pneumonia = immunodeficient p.

Pneumonia

Definition

- *pneumonia fibrinoso-purulenta acuta*
- **pathology** = croupous
  - fibrinous-purulent inflmmation
- **radiology** = lobar / alar
  - affects pulmonary lobe / whole lung diffusely

- rare nowadays
  - thanks to ATB therapy

- commonly led to death in the past
  - nowadays it represents **community-acquired pneumonia** (healthy p.)

Pneumonia

Causes (etiology)

- **bacterial** infection
  - *Streptococcus pneumoniae* ("pneumococcus", α-hemolytic)
  - *Klebsiella pneumoniae* (hemorragic Friedländer's pneumonia)

Pneumonia

Developement (pathogenesis)

- severe purulent or **fibrinous** inflammation occurs
  - **croupous type** = superficial "mucosal", replacing alveolar epithelium
  - **lobar / alar** = affects pulmonary lobe / whole lung diffusely

- leads to the **fibrin organisation (OP)** and RI
  - **carnification** = production of intraalveolar fibrotic plugs (*caro* = lat. meat)
  - several stages of morphological changes precede (**hepatisations**):

Pneumonia

Morphology

- **macroscopically** a lobe / whole lobe is congested
  - bronchial mucosa is hyperemic with mucus on surface
  - **red hepatisation** = red colour + liver consistency
  - **grey hepatisation** = grey to brown colour + liver consistency
  - **carnification** = firm meat-like areas

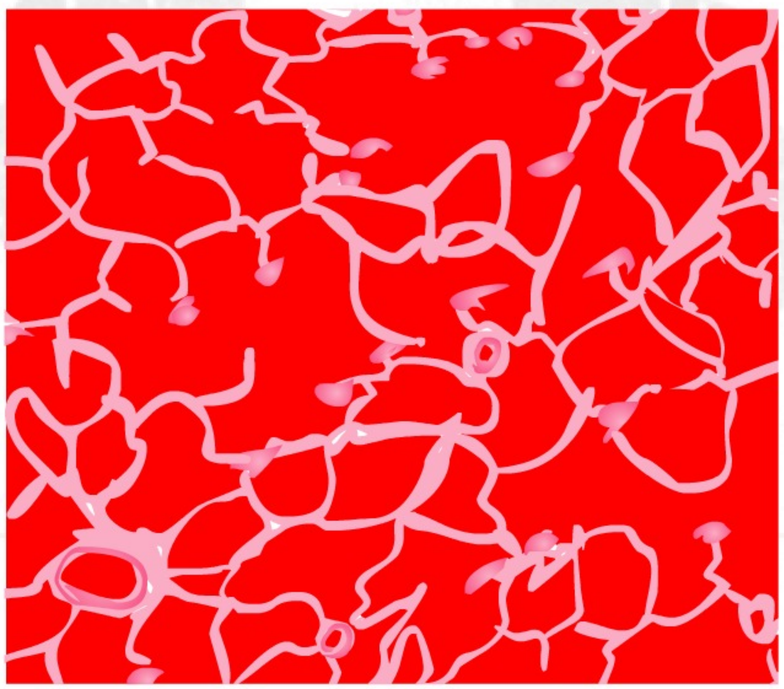
lobar

alar

Pneumonia

Morphology

- **microscopically** croupous inflammation in alveoli is detected
  - **red hepatisation** = exudation of oedema, extravasation of erythrocytes
  - **grey hepatisation** = exudation of fibrin, vessel compression, macrophages
  - **carnifications** = ns. gr. tissue to fibrotic plugs within alveolar tree



Pneumonia

Clinical manifestations

- **children** (infants) or **adults** (elderly)
  - attenuation of immunity
- suddenly "paralyses" the respiration = **severe symptoms**
  - dyspnoea, fever (rigor), exhaustion, dry to productive cough
  - auscultatory phenomena (dulled percusion, tubular breathing, crackles)
- **fulminant course** and fatal **complications**
  - sepsis, *cor pulmonale chronicum*, suffocation
  - **pleura** = hydrothorax or fibrinous-purulent pleuritis / pyothorax (empyema)
  - nowadays rare thanks to the ATB therapy and pneumococcus vaccine

Bronchopneumonia

Definition

- *bronchopneumonia catrrhalis acuta*
- **patology** = catarrhal-purulent
  - catarrhal to purulent exsudate (without abscess formation)
- **radiology** = lobular
  - affects pulmonary lobules (bronchus and connected alveoli)
- common disease
  - usually imobile patients (**nosocomial pneumonia**)
- may have fatal outcome
  - mainly among elderly and immunodeficient p. (**opportunistic pneumonia**)

Bronchopneumonia

Causes (etiology)

- **bacterial** infection / superinfection of viruses = flu etc.
  - mainly **streptococci** (β-hemolytic), **staphylococci**, **G- rods, haemophilus**
    + **nosocomial** (resistant) = MRSA, pseudomonas, burkholderia...
    + **opportunistic** (saprophytic) = *E. coli,* legionela...

- several **predispositions** exist:
  - **hypostatic** = within terrain of oedema / mucostasis
  - **bronchostenotic** = beneath obstruction (foreign bodies, tumours, aspiration)
  - **concomitant** = secondary / associated to other diseases

Bronchopneumonia

Developement (pathogenesis)

- milder **catarrhal** to **purulent** inflammation occurs
  - **catarrhal** = superficial mucosal (bronchi) serous exudate + mucus
  - **lobular** = exudate fills the lobuli (small bronchi and their alveoli)

- prompt **resorption**
  - usually is not as severe as pneumonia + rare carnifications

Bronchopneumonia

Morphology

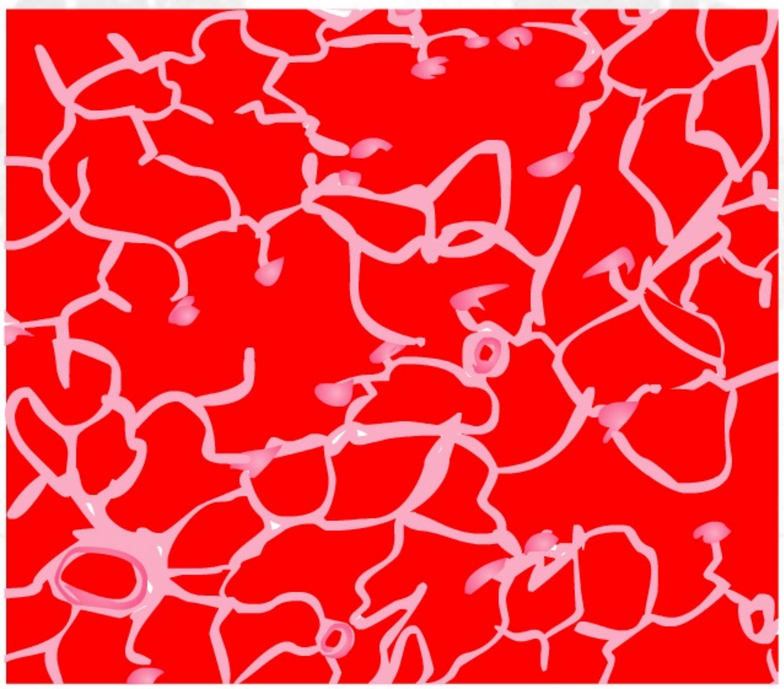
- **macroscopically** lobuli are consolidated and congested
  - **foci** shaped as "cat's footsteps" (may fuse giving "pseudolobar" appearence)
  - **hypostasis** mainly in D lobes / **aspiration** R inferior lobe / beneath **stenosis**
  - mucous pus is leaking from **bronch(iol)i** after compression

lobular

Bronchopneumonia

Morphology

- **microscopically** catarrhal-purulent intraalveolar exsudate
  - **broncho-** = starts within small bronchi and spreads into connected alveoli
  - aspiration can also lead to the developement of granulomas
  - immunodeficient patients may show modified inflammatory response



Bronchopneumonia

Clinical manifestations

- **children** or **adults** (of any age)
  - previous attenduance / hypostasis, aspiration or stenosis (recurrence)
  - **nosocomial** = hospital / healthcare-acquired (ventilation support)
  - **opportunistic** = imunodeficient (AIDS, chemotherapy, Tx...)

- usually **mild symptoms** and good prognosis
  - subfebrilia or fever, productive cough (expectoration), fatique
  - auscultatory phenomena (dulled percusion, alveolar breathing, crackles)

- immunodeficient = **severe / chr. course** and **complications**
  - developement of **DAD** / **destructive pneumonia** (abscess) / bronchiectasis

Destructive pneumonia

Definition

- includes **3 different conditions**:
  - common feature is destruction of the lung **parenchyma** and **interstitium**

1) necrotizing pneumonia
2) lung abscess
3) lung gangrene

- relatively **rare**, but severe course and **fatal** outcome

Destructive pneumonia

Causes (etiology)

- **bacterial** infection (primary / progression of superficial inf.)
  - **primary** pneumonic plague (*Y. pestis*), anthrax (*Bacillus anth.*)
  - **secondary** mainly anaerobic saprophytic fusobacteria, *Bacteroides supp.* (alkoholics, poor dental hygiene, epilepsy, swallowing disorders...)

- **mycotic** infection (opportunistic pneumonia)
  - **invasive mycoses** = aspergilosis, histoplasmosis, coccidioidomycosis, cryptococcosis, blastomycosis, pneumocystosis
  - **non-invasive mycoses** = mycetoma (aspergiloma), progression of candida

Destructive pneumonia

Developement (pathogenesis)

1) necrotizing pneumonia

- developement of venous thrombosis and infarzation leading to **hemorrhagic necrosis** of the lung

2) lung abscess

- interstitial delineated **purulent** inflammation (+ bronchial fistula)
- inhalation / aspiration / hematogenous origin
- healing leads to **cavitation** / **pneumatoceles** (numerous small cysts)

3) lung gangrene

- necrosis mofied by bacterias (**wet gangrene**)
- tumour decay, cesspit aspiration...

Destructive pneumonia

Morphology

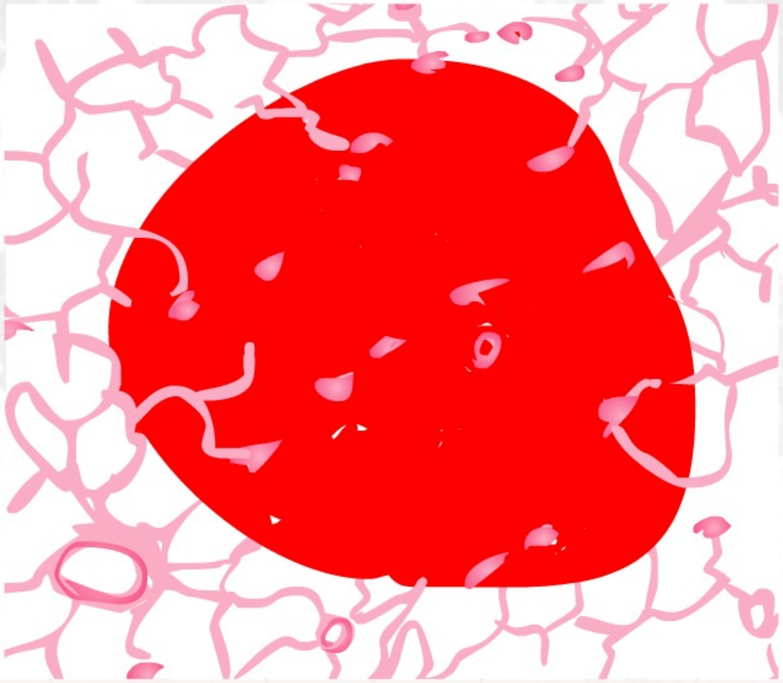
- **macroscopically** there is a focus of destruction
  - hemorrhagic necrosis / abscess / gangrene
  - may be multiple (*pneumonia abscendens*)

focus of destruction

Destructive pneumonia

Morphology

- **microscopically** destruction of the parenchyma + interstitium
  - replacement of the tissue with hemorrhagic necrosis / abscess / gangrene



Destructive pneumonia

Clinical manifestations

- **children** or **adults** (of any age)
  - **opportunistic pneumonia** = usually immunodeficient, *casus socialis*...

- severe **symptoms** similar to pneumonia
  - fatigue, fever, productive cough (expectoration) + **hemoptysis** to **hemoptoe**, vomiting if lung abscess is present

- always **severe course** and high **mortality**
  - **complications** = pleuritis, pyemia, sepsis, pyopneumothorax (empyema), mediastinitis, amyloidosis

Pneumonitis

Definition

- *pneumonitis interstitialis*
- **pathology** = interstitial
  - non-purulent (lymphoplasmocytic) interstitial inflammation
- **radiology** = atypical
  - rich RTG, but poor or non-specific clinical / physical finding in contrast

- common disease

- may have fatal outcome
  - in case of occurence of **ARDS** to **ILDs**

Pneumonitis

Causes (etiology)

- **bacterial** infection (usually intracellular pathogens)
  - *chlamydophila* (psittacosis / ornitosis), *mycoplasma*, *rickettsia*

- **viral** infection (the most common)
  - flu, parainfluenza, COVID-19, adenoviruses, RSV, CMV, HSV, measles, pox

- **non-infectious** (overlaps with secondary chronic ILDs)
  - **inhalation** (toxic gases), **aspiration** (vomit, lipoid substances), **drugs** (busulfan...), **immune disorders** (SLE, sclerodermia...)

Pneumonitis

Developement (pathogenesis)

- **non-purulent (lymphoplasmocytic) inflammation**
  - **interstitial** = fills the interalveolar septa and restricts **diffusion of gases**
- progression into **DAD** is possible
  - occurence of hyalinne membranes

Pneumonitis

Morphology

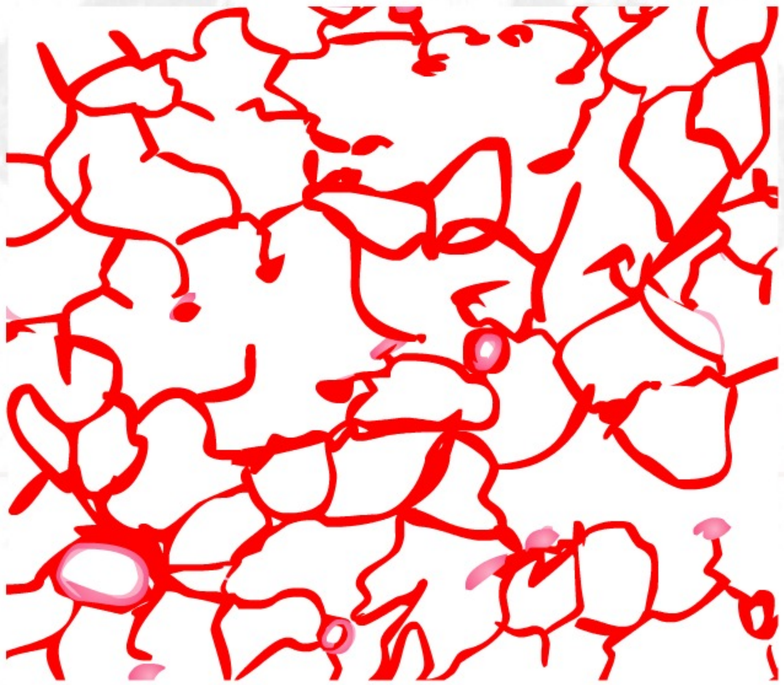
- **macroscopically** diffuse lung consolidation
  - **atypical** = rich RTG finding, but poor / non-specific clinical signs

atypical pneumonia

Pneumonitis

Morphology

- **microscopically** non-purulent interstitial inflammation
  - **interalveolar septa** widened, filled with lymphocytes, macrophages, plazma cells
  - **viruses** = virions can create inclusions (CMV...) and mucosal **necrosis** of airways (trachea, bronch(iol)i) with *bronchiolitis obliterans*



Pneumonitis

Clinical manifestations

- **children** (infants) or **adults** (elderly)
  - **opportunistic pneumonia** = usually immunodeficient

- variabile **symptoms**
  - **pulmonary** (dyspnoe, dry cough), **others** ("flu-like" = fatique, muscle and joint pain, fever)
  - **atypical pneumonia** = mild pulmonary symptoms in contrast with RTG
  - **"walking pneumonia"** = mild form of atypical pneumonia

- may lead to fatal **complications** in immunodeficiency
  - **DAD** progression, bacterial **superinfection**, *bronchiolitis obliterans*

Pulmonary TBC

Pulmonary TBC

Definition

- *tuberculosis*
- infectious multisystemic disease
  - rich pathogenesis and morphological finding
- worldwide ↑ spread disease (but in CZ just 500 cases)
  - ↓ thanks to **antituberculotic drugs** and **vaccine** (5 cases/year)
  - thread to **immunodeficient** and **socially deprived** p. (role of migration)
- may have **fatal** outcome
  - historically important factor of mortality (Europe = developed resistence)

Pulmonary TBC

Causes (etiology)

- **bacterial** = "Koch's bacteria" (*Mycobacterium tuberculosis*)
  - acidoresistent aerobic non-sporulating rods (**intracellular** occurence)
  - **"waxy" coating** from lipids within membranes (Gram-; **Ziehl-Neelsen** st.)
  - **resistent** (pH, desinfection) and **hard to destroy**

- **spread** through air droplets / alimentary / inoculation / transplacental
  - ↓ **infectious dose** (10 bacteria)

- **other mycobacteria** = atypical mycobacteriosis
  - mimics of TBC

Pulmonary TBC

Developement (pathogenesis)

- hard to destroy = **IV. type of immunopathological reaction**
  - specific = **granulomatous** inflamation

TBC granuloma

- tuberculous nodule (**macroscopically** resembles millet = lat. *milium*)

1) central caseification = necrotizing granoluma (caseous necrosis)

2) epitheloid histiocytes

3) Langhans giant cells

4) reticular fibres

5) rim of lymphocytes at the priphery

- **avascular** = no vessels

Pulmonary TBC

Developement (pathogenesis)

- specific inflammation has **2 forms**:
  - determined by the level of host immunity

1) proliferative (granulomatous)

- ↑ **resistance** = mycobacteria isolated by **granulomas** (better option)
- **tubercles** = firm (< 2 mm, grey-white) / soft (> 2 mm, yellow, necrotic)

2) exudative (granulomatous-necrotizing, caseous)

- ↓ **resistance** = mycobacteria not isolated, **caesiefication** dominates (worse)
- **caseous necrosis** = caseification / cheese-like (macro) / typical microscopy (inhibition of proteases within debris with leftovers of nuclear chromatin)
- Orths cells within exudate

Pulmonary TBC

Developement (pathogenesis)

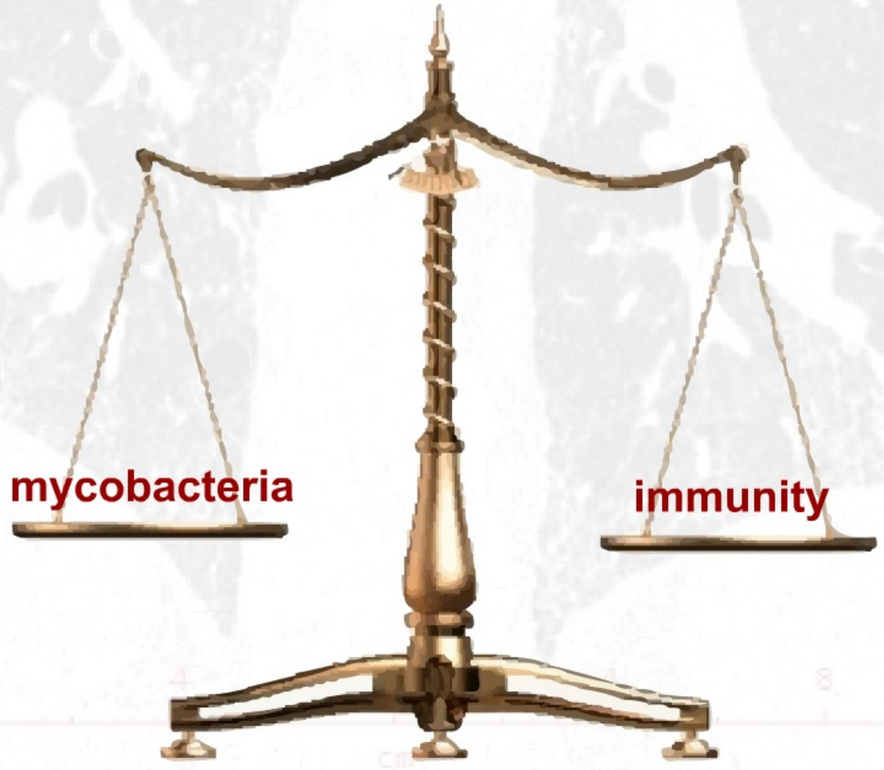
- **healing** by inspissation and encapsulation with scaring
  - **scar** tends to **calcify** (+ anthracosis)

- or **progression** of TBC foci is pssible (by coliquation)
  - caseous necrosis is changed to **liquefactive** ("tuberculous pus")

Pulmonary TBC

Morphology

- organ manifestation determined by **portal of entry** + **host immunity**



Pulmonary TBC

Morphology

- portal of entry (**primoinfection**) + regional LN (**primocomplex**):

1) inhalation

- **primoinfection** = superior part of inferior lobe of the R lung
- **primocomplex** = hilar / mediastinal LN

2) ingestion

- **primoinfection** = ileocaecum / *tonsilla pharyngea*
- **primocomplex** = mesenterial / cervical LN

3) inoculation

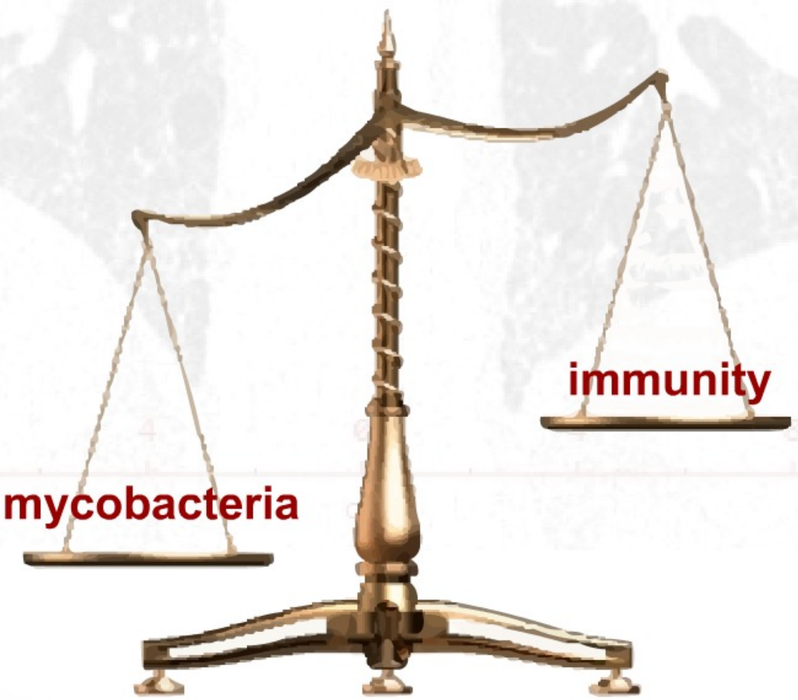
- **primoinfection** = skin defect
- **primocomplex** = regional LN

Pulmonary TBC

Morphology

1) Primary TBC (preimmune, "infantile")

- cases with **domination** of **mycobacteria** over immunity
  - naive "virgin" terrain (children, non-immunized adults = native Americans...)
- severe course with **dissemination** (generalisation)



Pulmonary TBC

Morphology

1) Primary TBC (preimmune, "infantile")

- rise of **primoinfection** (primary / Ghon focus)
  - inicial infectious focus (**granuloma** within portal of entry)

- followed by **primocomplex** (primary / Ghon complex)
  - regional **lymphangitis** and **lymphadenitis** by lymphogenic spreading

- **3 different options** can develope:
  1) recovery = calcified scar (cca 90%, so called Ranke complex)
  2) latency = asymptomatic form
  3) primary progressive TBC = variable **continuous spreading** (↓ immunity)

Pulmonary TBC

Morphology

1) Primary TBC (preimmune, "infantile")

- **porogenous** spreading:
  - **TBC / caseous (broncho)pneumonia** (enlargement of the primoinfection)

- **lymphogenous** spreading::
  - **scrofula** (from primocomplex into cervical LN; +/- fistula)

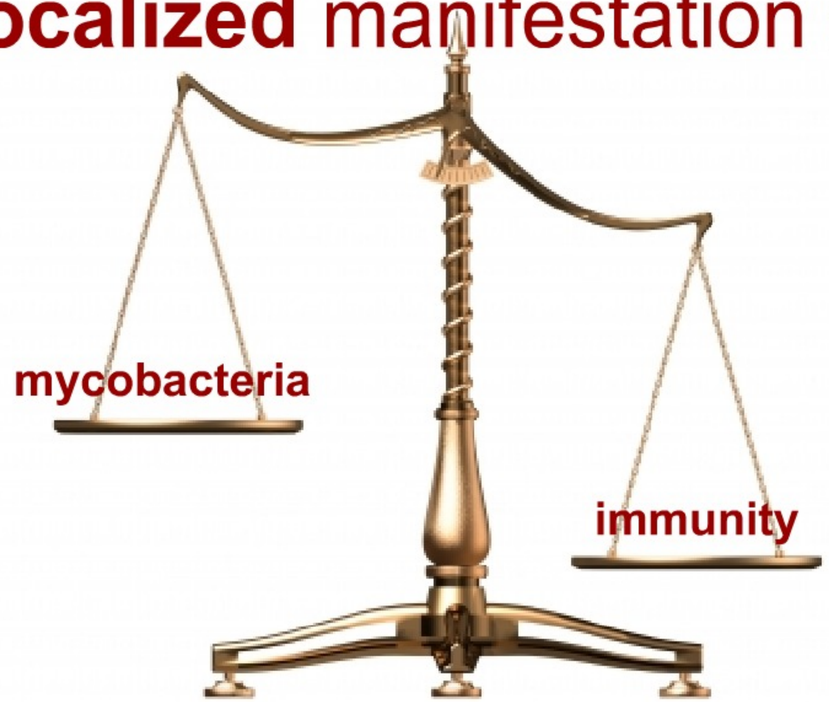
- **hematogenous** spreading (mycobacteremia / Landouzy sepsis):
  - **pneumonic** = mainly pulmonary manifestation (**miliary TBC** of the lungs)
  - **typhoid** = abdominal manifestation (miliary TBC of the spleen and liver)
  - **meningitis** = neurologic manifestation (basilar meningitis)

Pulmonary TBC

Morphology

2) Secondary TBC (postprimary, "adult")

- **immunity** dominates over mycobacteria (immunized p.)
  - **reactivation / reinfection** after vaccine / primary TBC (after latency)
  - **attenuation** ("misery disease" = elderly, alcoholics, malnutrition, stress...)
- usually milder **localized** manifestation (1 organ)



Pulmonary TBC

Morphology

2) Secondary TBC (postprimary, "adult")

- development of **apical TBC** (Simon focus / nodule)
  - secondary TBC tends to occur in **apex of superior lobe of the lungs**
  - **reinfection** / **reactivation** (hematogenous metastasis from primocomplex)
    = tropism caused by ↑ airiness of the apex and aerophilia of mycobacteria)
  - RTG term **Assmann infraclavicular infiltrate**
  - if reactivation appeared there is a primocomplex scar present too

- **isolated metastases** can occur
  - memory cells usually prevent dissemination (mainly **porogenous** spreading)

Pulmonary TBC

Morphology

2) Secondary TBC (postprimary, "adult")

- **porogenous** spreading:
  - **TBC / caseous (broncho)pneumonia** (spreads within lungs)
  - **cavern** = post-colliquative pseudocysts (fibrotic capsule; aspergilloma)
  - **"opened" TBC** (bronchial destruction and coughing / swallowing)
  - **bronchopleural fistula** (TBC pleuritis + pneumothorax + empyema)
  - **adenobronchial fistula** (reactivation of primocomplex within LN)
- **lymphogenous** and **hematogenous** spread (isolated):
  - **destruction of the vessels** = anywhere (lungs, adrenal glands, kidneys, LN, bones, skin...)
  - **dissemination** rare (miliary TBC)

Pulmonary TBC

Clinical manifestation

- **children** (primary TBC usually) or **adults** (secondary TBC)
  - ***habitus phtisicus*** = usually asthenia and narrow chest

1) primary
- **resistent terrain** = asympt. / weakness,fever, inappetence, fatique, erythema
- **naive terrain** = multioragn dissemination

2) secondary
- **latency** = asymptomatic
- **manifestation** = cachexia (phtisis), night sweats, fever, cough (variable), hemoptysis (anemia, fatal Rasmussen's arterial anerysm)

+ rich symptoms among other organs, AA amyloidosis...

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