

Systematic Anatomic Pathology

Respiratory System

Systematic Anatomic Pathology

Malformations

■ Numeric – in defect:

⇒ Bilateral pulmonary agenesis

- Associated with failed laryngo-tracheal septimentation

⇒ Monolateral pulmonary agenesis

- Complete absence of lung
- Trachea devoid of keel (dd.: aplasia)
- Omolateral pulmonary artery ramus agenesis
- Vagus nerve agenesis
- Contralateral compensatory hypertrophy
- Often associated with malformations of:
 - Heart and aorta
 - Vertebro-costal skeleton
 - diaphragm and digestive apparatus

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Malformations

■ Numeric – in defect:

⇒ lobar agenesis/aplasia

- more frequent to the right (superior and median lobi)
 - Vasculo-nervous agenesis
 - Independent from fissures
 - Compensatory hypertrophy

⇒ lobular hypoplasia

- simple = lack of bronchial tree branches

reduced amount of alveolar parenchyma
pulmonary structure nearly unaltered

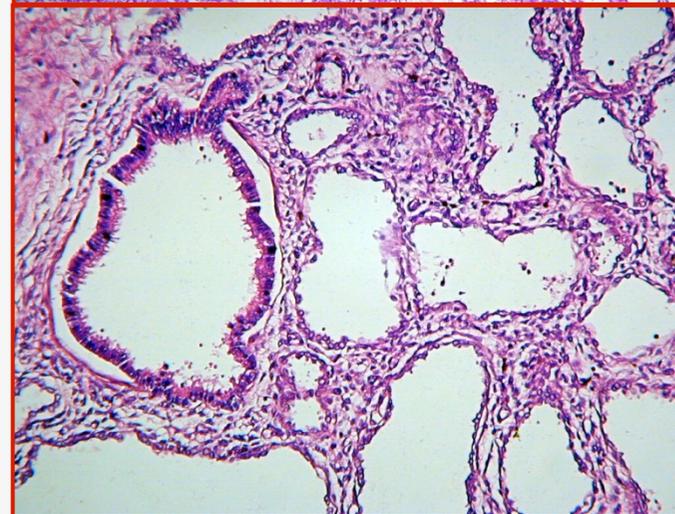
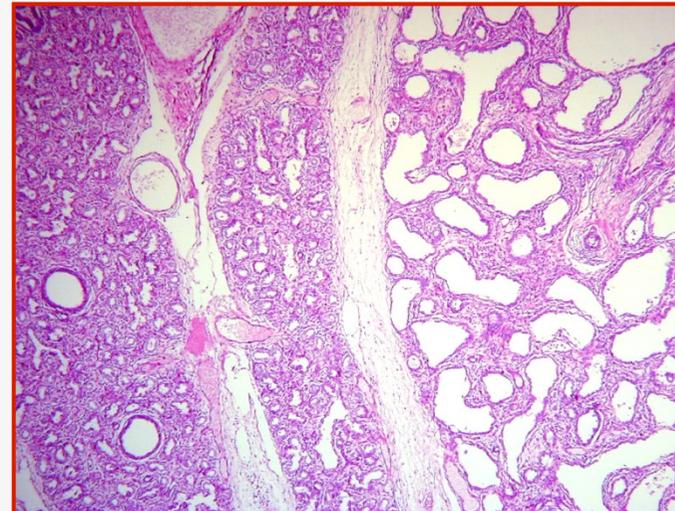
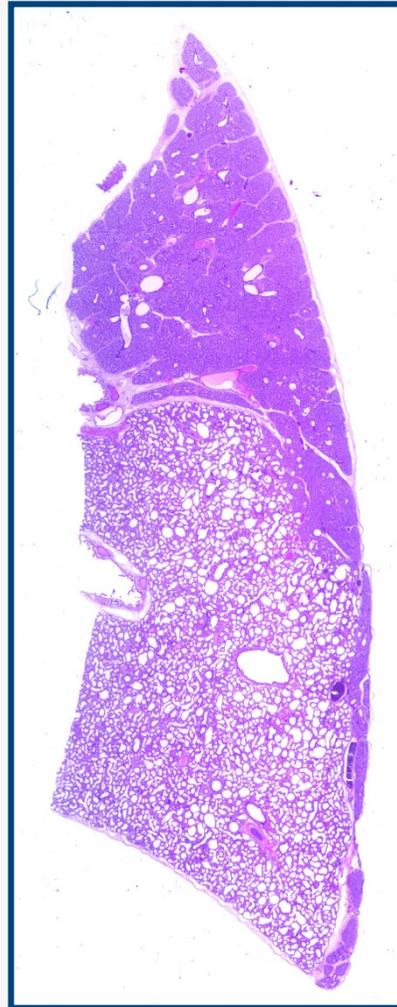
- cystic = block of respiratory tree development

global: bronchial anarchic structure (early)

partial = cystic adenomatous malformation

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Adenomatoid Malformations



Systematic Anatomic Pathology

Malformations

- **Numeric – in defect:**

- ⇒ Partial polycystic lung

- ⇒ Aerial cysts

- ⇒ Enterogenous cysts (stomach, pancreas, ileum)

- Alterations compatible with life are often clinically silent.
- They cause clinical symptoms (>40 aa.) due to infections or haemorrhagic complications
- DD.: bronchiectasias

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Malformations

■ **Numeric - in excess:**

⇒ **Supernumerary lung / lobes**

- With autonomous broncho-vascular system
- apical

⇒ **Supernumerary fissures**

- Median lobe (lingular) to the left
- Basal medial fissure, right (paracardiac lobe)
- Inferior fissure, right apical (Deve-Fowler lobe)
- Apical right fissure

⇒ **Accessory pseudofissures**

- Vascular, pleuric and costal sulci
- Azigos vein lobe

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Malformations

Numeric - in excess:

- ⇒ **Sequestration**: derived from supernumerary drafts or lobes separated from the principal ones
 - separation of a tract of lung, whose extension is lobar or segmentary, due to the lack of a bronchus (no ventilation)
 - autonomous **aortic** vascularization
 - organoid configuration and cystic structure similar to fetal lung
- ***extralobar sequestration***: dense mass, ovoidal, extra-pulmonary, 2-8 cm, with mediastinic / aortic / gastro-oesophageal peduncles, clinically silent.
 - Associated with total/partial agenesis of the lung
 - Without pulmonary alterations (accessory gem)
 - Analogous to mediastinic, costal bronchogenic cysts
 - Azygous vein lobe

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Malformations

Numeric - in excess:

⇒ *intralobar sequestration* (intrapulmonary): postero-inferior region of the inferior lobes

- Pryce type: 1-2 segmental agenetic bronchi + hypoplasia of the omolateral pulmonary artery ramus (arises from the separation of a pulmonary draft)
- Le Brigand-Dupré type: interposed between branches of segmental bronchi (derived from accessorial draft)

Intralobar sequestrations cause late clinical manifestations, due to inflammatory and haemorrhagic complications

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Malformations: sequestrations



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malformations

- **Mirror-like lung**: symmetry of both lung
 - ⇒ Right / left isomerism, the left is often lethal because accompanied by additional severe congenital alterations
 - ⇒ The right inferior lobe is vascularized by the branches of thoracic aorta and drains blood into the inferior vena cava or in azygos vein. **Cianosis**, cough, dyspnoea
- **Bronchial stenosis**
 - ⇒ extrinsic (abnormal vessels)
 - ⇒ intrinsic (diaphragm, malacic cartilage)
 - Acute emphysema
- **Fistulas** broncho-oesophageal (traction diverticula)

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Pulmonary hypertension

- ⇒ Increased pressure regimen in pulmonary arterial circulation
- ⇒ (lung vascular sclerosis)
- ⇒ Right ventricular hypertrophy
- ⇒ Chronic pulmonary heart disease

■ **Idiopathic**

- Young, females (5-10 aa.)
- Sympathetic hyperactivity
- Microembolism, arterial wall damage
- Autoimmune process involving the stroma of pulmonary vessels
 - Primitive damage of pulmonary endothelium
 - Chronic vasoconstriction
 - Pulmonary hypertension
 - Vascular sclerosis
 - Raynaud phenomenon

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Pulmonary hypertension

■ **Secondary** to a pre-existent pathological situation

⇒ **Parenchymal lesions**

- Emphysema (pulmonary circulation), pulmonary fibrosis
- Chronic bronchitis
- Bronchiectasia, abscess and pneumonia (fibrosis), fistulous **vascular lesions**
- Microembolisms
- Panarteritis Nodosa and Wegener disease

⇒ **Respiratory failure**

- (hypercapnia – respiratory acidosis – arterial stenosis)
- Compensatory polyglobulia (hyperviscosity - resistance)
- Kyphoscoliosis, Pott disease (Tb), Polio
- Muscular dystrophy

⇒ **Central respiratory deficit**

- Head trauma
- Major surgery

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Pulmonary hypertension

Morfology:

- Lesions of pulmonary arterial branches similar to those of systemic arteriosclerosis, of lesser severity.
- Anelastic lung, pale or brownish
 - Elastic arteries: uncomplicated intimal plaques
 - Muscular arteries: hypertrophy, intimal, eccentric thickening, delamination
 - Fibrinoid necrosis + inflammation (PMN)
 - Re-canalized platelets thrombi (plexiform lesions)

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Disorders of circulation

Stasis (congestion or passive hyperemia)

- ⇒ acute
- ⇒ Chronic

Caused by obstacles to venous outflow

- systemic (VS+VD)
 - Heart global failure
 - Dilatative/obstructive cardiomyopathy
- pulmonary (VS)
 - mitral stenosis
 - myocardial infarction
- local (local problems of venous drainage)
 - neoplasms
 - malformations

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Disorders of circulation

Lung with acute stasis:

- Tumid
- Congested and reddish
- Increased volume and weight
- Dampish
- Fluid blood, diffluent on cut section
- Massive congestion of interstitial capillaries

Systemic anatomic pathology

Circulatory disorders

■ **Chronic lung stasis** (*brown induration*)

■ Macro:

- Rigid
- Red-dark brown, dry
- Increased consistency, weight and volume
- Leakage of dense blood on section

■ Micro:

- Dilated and congested capillaries, aneurysmatic dilations
- Endoalveolar microhaemorrhages
- Erithrophagocytosis and iron rich macrophages (due to cardiac alteration)
- Thickening of septa + edema, interstitial fibrosis
- Vascular sclerosis (pulmonary hypertension)

Systemic anatomic pathology

Circulatory disorders

- **Oedema** = Accumulation of fluid in the interstitial spaces of the lung
 - ⇒ *Central causes (cardiogenic) – systemic edema*
 - Myocardial infarction, left valve disease, pericarditis etc.
 - ⇒ **Local venous obstruction** – localised oedema
 - ⇒ **Changes in osmotic pressure (renal, liver disease)**
 - >hydrostatic pressure in pulmonary capillaries
 - Capillary congestion
 - Transudation of fluid in the interstitial spaces
 - Lymphatic drainage
 - Accumulation into the alveoli (alveolar phase = acute edema)
 - Accumulation into the interstitial space (**interstitial phase** = chronic edema)

Systemic pathologic anatomy

Circulatory disorders

■ **Acute pulmonary edema**

(>>>cardiogenic, renal, hepatic)

- ⇒ Leakage of foamy fluid from main bronchi
- ⇒ Lungs are bulky and heavy, they don't retract (elastic recoil)
- ⇒ "fovea"
- ⇒ Moisty, pinkish-dark brown
- ⇒ Alveoli contain homogeneous eosinophilic fluid

Systemic pathologic anatomy

Circulatory disorders

ARDS adult respiratory distress syndrome

“Shock-like lung”

- ⇒ Infections, septic shock
- ⇒ Pancreatitis (degradation of surfactant by phospholipases)
- ⇒ Burns, trauma, toxins (fosgene, hyprite, narcotics, NO₂), iper-O₂

Damage to respiratory barrier by superoxides, activation of C5a, proteases

- Macro: similar to acute edema
- Micro: endoalveolar transudate
 hyaline membranes (4^a day)
 interstitial inflammation
 repair by type II pneumocytes (gland-like)
 fibrotic organisation

Systemic pathologic anatomy

Circulatory disorders

Other pulmonary oedemas

- Neurogenic oedema (systemic arterial vasoconstriction)
- *Post-transfusional oedema (with renal failure or cardiomyopathy)*
- *High altitude pulmonary oedema (peripheral vasoconstriction)*
- *Silo workers oedema (NO₂)*
- *Uremic oedema, perihilar (butterfly shadow), chronic, toxic*

Systemic pathologic anatomy

Circulatory disorders

Embolism

Presence of material insoluble with blood, carried away from its origin, reaching a smaller blood vessel in occlusive position

- solid (thrombi, clots, neoplasms, chorionic villi, bone, Ca⁺⁺)
 - liquid (amniotic fluid, lipophilic solutions)
 - gaseous (trauma, pneumothorax, N₂)
- Outcome depends on:
- Type of circulation (terminal, collateral)
 - Quickness
 - Size of obstructed vessel

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Blood flow disturbances

Embolism

■ Sources:

- Lower limbs
- Pelvic venous plexes (IVC tributaries)
- Right heart (5%) (fibrillation)

■ Pathogenesis of embolism:

- Phlebothrombosis
- Stasis of blood
- Scarce inflammatory component
- Fibrinolytic therapy

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Blood flow disturbances

Embolism

■ Location:

⇒ Pulmonary artery or principal trunks:

- Sudden pulmonary hypertension
- Acute hypoxia
- Right heart failure

⇒ Lobar branches:

- Collapse and stabbing pain
- Pulmonary hypertension (serotonin)
- Pulmonary heart disease
- Pulmonary infarction

⇒ Intralobar branches:

- +/- asymptomatic
- Downstream haemorrhage due to endothelial damage

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Blood flow disturbances

Embolism

- DD: Postmortem embolus / thrombus / clot
 - Shape with respect to the vessel (smaller / = / variable)
 - Adherence to the wall (adherent / strong / weak)
 - Color (red / white-reddish / gelatinous)
 - Consistency (friable / rigid / elastic)
 - Surface (wrinkled / irregular / smooth)

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Blood flow disturbances

Pulmonary infarction

- The most frequent complication of embolism
- Pulmonary infarction occurs in presence of the following predisposing conditions (**double circulation**):
 - Respiratory insufficiency and/or anemia
 - Pulmonary hypertension (emphysema, COPD)
 - Atherosclerosis of the bronchial aa.
 - Left ventricle insufficiency

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Blood flow disturbances

Pulmonary infarction

- location: inferior lobes
- shape: conical or pyramidal (consensual fibrinous pleuritis)
- color: red (haemorrhagic)
- consistency: hard
- surface: dry and retracted (in advanced stage)
- evolution: scarring sclerosis
- hystology: haemorrhagic infarction
 coagulative necrosis
 septal and bronchial disruption
 (dd.: haemorrhage with intact septa)

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Blood flow disturbances

Pulmonary infarction

- Complications:
 - Septic infarct
 - Abscess
 - Haemothorax
 - Pulmonary fibrosis

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Inflammations of the lung

- Sites of exudate accumulation:
 - Alveoli (pneumonias)
 - Bronchi (bronchitis) o bronco-alveolar (broncho-pneumonia)
 - Interstitium (interstitial pneumonia)
- **Lobar pneumonia** (frank or genuine)
 - *Diplococcus Pneumoniae* (rare: *Klebsiella Pneumoniae*)
 - Predisposing factors:
 - ⇒ *Cooling*
 - ⇒ *Metabolic diseases*
 - ⇒ *Alcoholism*
 - ⇒ *Thoracic traumas*
 - ⇒ “*Pneumococcal sensitization*”

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Inflammations of the lung

Endoalveolar

- Fibrinous (crupal)
- Cyclic
- Adults (hyper-ergic)

- Pathomorphosis induced by antibiotic therapy
- Uni/pluri-lobar, sincrhonous/metachronous
- Right lung, inferior lobes
- Massive parenchimal extension

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Lobar Pneumonia

- 1st stage of the **haemorrhagic blockade** (max. 24h)
 - *crepitatio indur*
 - > volume and consistency
 - dark-red colour
 - sero-haemorrhagic liquid at squeezing out
 - Dokimasia weakly +
 - capillary dilation, endo-alveolar sero-haemorrhagic exudate

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Lobar Pneumonia

- 2nd stage of **red hepatization** (48-72h)
 - *Hepatized sound*
 - >> volume and consistency
 - Dark-red, dry, granular surface
 - Dokimasia +++
 - Reddish, opaque and fibrinous pleura
 - Coagulation of the endoalveolar exudate
 - Haemorrhagic alveolitis + PMN
 - Residual vessel blockade
 - Pneumococcal phagocytosis

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Lobar Pneumonia

- 3rd stage of **grey hepatization** (24-48h)
 - fibrinolysis, RBC haemolysis, PMN chemiotaxis
 - Reduction of the haemorrhagic blockade
 - Max. > volume and consistency
 - Greysh and granular surface
 - Fibrinous pleuritis
 - Fibrinous-suppurative alveolitis with pale septi

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Lobar Pneumonia

- 4th stage of **resolution** (24-48h)
 - “critical” fluidification of the exudate
 - *crepitatio redux*
 - Volume & consistency +/-
 - Pinkish colour
 - Abundant pinkish fluid at squeezing out
 - Dokimasia +/-
 - Resolution of the fibrinous reticulum
 - Colliquation of PMN
 - Elimination of the exudate
 - Reactive lymphadenitis

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Lobar Pneumonia

Pulmonary complications :

⇒ Carnification

- Lack of reabsorption of the exudate (fibrinolytic deficiency)
- Fibrous organization
- Compact greyish, fleshy areas
- Pleuric adhesions
- *Young* or *old* granulation tissue

⇒ Abscess and Gangrene

- Destruction of intra-alveolar septa
- Necrosis and suppurative fusion (para/metapneumonic)

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Lobar Pneumonia

■ **Extra-pulmonary complications :**

- ⇒ Pleuric Empyema (para/meta-pneumonic)
- ⇒ Suppurative inflammation of mediastinum
- ⇒ Suppurative pericarditis
- ⇒ Toxic Myocarditis
- ⇒ Hepato-toxic jaundice
- ⇒ Glomerulonephritis
- ⇒ Paralyzed ileum
- ⇒ Reactive Splenic Hyperplasia
- ⇒ Suppurative 'metastases' (meninges, endocardium, peritoneum)

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Broncho -Pneumonia

- *Klebsiella Pneumoniae, Strepto-Staphylococci, Proteus V, Pseudomonas Aeruginosa, E. Coli*
 - Localised (multiple) lesions (lobular)
 - Mainly affects kids and elderly people (anergic)
 - *It is often a complication of another infectious disease*
- ⇒ Predisposing factors :
 - Local circulatory disturbance
 - Deficit of ventilation
 - Aspiration of infectious material (comatose)
 - Perfrigerations
 - Inhalation of irritating substances

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Broncho-Pneumonia

- Lesions : **bilateral** , **asymmetric** and **asynchronous**
- Lobular greyish centers
 - > consistency
 - disseminated / confluent / pseudolobar
 - Interposition of airy zones
 - Endobronchial suppurative exudate
 - Fibrino-suppurative consensual pleuritis
 - Scarce fibrinous endo-alveolar component
 - Peri/endo-bronchial PMN exudate
- Slow and asynchronous Resolution

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Broncho-Pneumonia

■ Complications:

- Empyema
- Abscess / Gangrene
- Meningo-endocarditis
- Mycosis (post-antibiotic treatment)

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Special types of Pneumonia

Lipidic Pneumonia (*foreign bodies*)

- ⇒ Caused by penetration of oily substances in the airways, mostly by aspiration
- Dystrophic infants
 - Elderly
 - Chronic Bronchopathic patients (aerosol)
 - Intubation

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Special forms of pneumonia

Lipidic pneumonia (foreign bodies)

- Origin:
 - ⇒ vegetal (olive oil)
 - emulsified, unhydrolyzed
 - eliminated by sputum, modest inflammation
 - ⇒ animal (milk, codfish)
 - Hydrolyzed = irritant fatty acids
 - Intense exudation
 - ⇒ mineral (paraffin, vaseline)
 - emulsified and phagocysed
 - foreign body granulomas

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Special forms of pneumonia

- ***Lipidic pneumonia*** (*foreign bodies*)
 - ⇒ Right lung, inferior lobes
 - ⇒ Compact appearance, grey-yellowish
 - ⇒ Bronchopneumonia with confluent outbreaks in 4 stages:
 - haemorrhagic stage
 - foamy macrophage stage
 - giant cells + PMN interstitial exudate
 - granulomas and fibrosis
 - healing
 - ⇒ Possible superinfection = purulent bronchopneumonia

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Special forms of pneumonia

■ *Loeffler's eosinophilic pneumonia*

- Fever, cough, asthmatic dyspnoea, eosinophilia
- Rx: isolated and fugitive pulmonary infiltrates (24-48h)
- Bronchopneumonia or pneumonia (sero-fibrinous) with eosinophilia
- Rare fibrotic evolution

⇒ Ling allergic reaction to airborne antigens (drugs, pollen). Pulmonary migration of *Ascaris L.*

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Special forms of pneumonia

■ *Uremic pneumonia*

- ⇒ peri-hilar pulmonary edema, “butterfly” - shaped
- ⇒ >>> consistency (solid oedema), scarcely diffluent
- ⇒ “fovea” +++
- ⇒ Increased endoalveolar sero-albuminous fluid, PAS+
- ⇒ Fibrin deposition of “hyaline membrane” - type
- ⇒ Hyperemia and focal haemorrhages, septal oedema
- ⇒ Scarse and rare tendency to fibrosis

■ Genesis: altered permeability of the capillary barrier

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Special forms of pneumonia

■ ***Pneumonia due to ionizing radiation***

⇒ Accidental or therapeutic exposure (breast, lung, mediastinum, thyroid cancer)

Lesions depend on:

- Overall dose
- Duration of exposure

⇒ Septal capillary congestion

⇒ Desquamative alveolitis

⇒ Edema and hyaline membrane due to damaged basal membranes and elastic fibers

⇒ Epithelial hyperplasia with distinct nuclear pleomorphism

⇒ Reparative fibrosis

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Interstitial pneumonia

- Newborns and infants (>>> interstitial connective tissue)
- *Etiology: viral, rickettsial, protozoal*
- Histopathological features dominated by *lymphomonocytic infiltration and/or interstitial plasma cells*
- Sometimes, secondary infection with alveolar involvement.

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Interstitial pneumonia

- Primary forms (*primarily interstitial*)
- Secondary forms (from bronchi/alveoli to interstitium)
 - acute / subacute / chronic
 - septal, peribronchial, perivascular, subpleural, interlobular, intralobular, perilymphatic
 - exudative (rare), sero-suppurative (pulmonary lymphangitis)
 - infiltrative - productive (lymphocytes, plasma cells, eosinophils)
 - granulomatous (tbc, sarcoidosis)
 - productive-sclerosing (pulmonary fibrosis)

Systematic anatomic pathology

Interstitial pneumonia

■ **Whooping cough** (Haemophilus Pertussis)

■ 4^o/6^o years of life

⇒ Catarrhal stage (angina-like) (3-14 days)

⇒ Convulsive stage (3-7 weeks)

⇒ Resolution

- Laryngo-bronchial inflammation
- Intracytoplasmic inclusions into cylindrical epithelium
- Lympho-monocytic peri-bronchitis/bronchiolitis
- Suppurative bronchial pneumonia due to superinfection

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Interstitial pneumonia

- Pneumonia due to **Mycoplasma Pneumoniae** (primary, atypical)
 - Poor clinical symptoms (cough)
 - Benign course (2-3 weeks)
 - Complicated by sero-fibrinous pleuritis, interstitial myocarditis, meningoencephalitis and haemolytic anemia
 - Lymphangitic pleural striae
 - Wet and congested lungs with soft areas of thickening
 - Necrotizing peri- bronchitis and bronchiolitis (lymphocytes and plasma cells)

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Interstitial pneumonia

■ Psittacosis (C. Psittaci)

- Transmitted by parrots
- Aggressive evolution
- Areas of demarcated reddish hepatization
- Sero-mucous bronchial sputum

- Lymphocytic/monocytic infiltration
- Pneumocytes hyperplasia and fibrous endoalveolar exudate
- Levinthal bodies, cytoplasmic coccoids
- Capillary thrombosis and haemorrhagic foci

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Interstitial pneumonia

■ *Viral interstitial pneumonia*

⇒ Pneumonia and broncho-pneumonia (**Mixovirus**)

- Often due to superinfections (Haemophilus I.)
- Associated with interstitial oedema
- Septal thickening and lympho-monocytic infiltration
- Associated with myocardial, renal and hepatic degeneration (murky)

⇒ **Measles** broncho-pneumonia

- Appearance in the pre-exanthematous phase
- Bronchitis , bronchiolitis and lymphocytic peri-bronchitis
- Pneumocyte hyperplasia
- Endoalveolar multinucleated giant cells with inclusion bodies

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Interstitial pneumonia

■ Cytomegalovirus pneumonia

- Newborns and infants
 - - circumscribed form (salivary, pulmonary, renal, hepatic, pancreatic, intestinal)
 - - diffuse form, lethal
- Diffuse lung involvement
- Abundant interstitial exudate (lympho-monocytes)
- Giant epithelial cells with alveolar cytomegalic inclusions (nuclear with paranuclear corpuscles)
- Desquamation of alveolar epithelium

■ Pneumonia due to **Polio, Varicella, Mononucleosis**

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Interstitial pneumonia

■ Rickettsiosic pneumonia

- Q Fever (Queensland) (R. Burneti)
- Septicaemic
- Transmitted by pets (sheep, cattle)
- Elimination with fluids and childbirth
- Penetration by inhalation
- Silent lung lesions
- Ground-glass appearance, sublobar
- **Fissural pleural reaction**
- Isolated or multiple outbreaks of parenchymal condensations
- Mononuclear infiltration of alveolar septa
- High number of macrophages containing Rickettsiae

Systematic Anatomic Pathology

Protozoal Pneumonia

■ *Toxoplasma Pneumonia*

- Congenital (more frequent)
- Acquired (rare)
 - Intracellular protozoa; 4-7 μ , Giemsa +
 - Colonizes histiocytes, muscle cells and neural cells
 - Transmucosal penetration

Systematic Anatomic Pathology

Protozoal Pneumonia

- 1st Stage: visceral (8-10 days)
 - Lymphohaematic spread (liver, spleen, lungs)
 - Interstitial histiocytic granulomas
 - Piringer-Kuchinka lymphadenitis
 - Mono-histiocytic interstitial pneumonia, septal
- 2nd Stage: brain-retinal
 - Antibody reaction
 - Hosting in segregated organs (eye, CNS)
- 3rd Stage: chronicity
 - Intracellular pneumocysts for missed rupture of colonized cells
 - Microglial reaction

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Protozoal Pneumonia

■ *Leishmaniotic Pneumonia*

- ⇒ L. Donovanii
- ⇒ Transmucosal or hematic penetration
- ⇒ Perivascular pneumonia, monocyte-macrophage recruitment
- ⇒ Histiocytes with Leishman-Donovan bodies

Systematic Anatomic Pathology

Protozoal Pneumonia

■ *Pneumocystic pneumonia*

- Premature and dystrophic infants
 - Immunosuppressed patients
 - Haemopathy-associated (LNH, LAL)
- ⇒ Incubation time: 4-6 weeks
- ⇒ Cough, fever, increasing dyspnea, cyanosis
- ⇒ Mediastinal and/or subcutaneous emphysema, respiratory failure
- ⇒ Rx: ground-glass appearance
- ⇒ Round corpuscles, 4-5 m, with PAS+ capsule

Systematic Anatomic Pathology

Pneumocystic Pneumonia (P. Carinii)

- Lungs do not crouch
- Increased volume and consistency
- Dark-red color
- Thickening of the lobules (similar to pancreas)
 - Lesions of the alveolar septa:
 - thickening for lympho-monocytic and plasmacell infiltrate
 - hyperplasia of reticular fibers
 - Alveolo-bronchial lesions:
 - Amorphous substance, spongy
 - Beehive-like cells
 - Haematoxylinophilic corpuscles
 - Lack of endoalveolar exudate
 - Fibrotic evolution

LUNG ABSCESS

Suppurative inflammatory process, with necrotizing evolution, tending to demarcation, formation of a new cavity, communicating with the bronchial tree.

- Often consequent to aerobics Gram+ bacterial infection
 - ⇒ Abscesses subsequent to bronchopneumonic processes (50% of cases)
 - para/metapneumonic
 - para-bronchiectasias
 - paraneoplastic
 - ⇒ Aspiration abscesses
 - Oral mucosa, tonsils, adenoids surgery
 - General anesthesia and debilitated patients
 - Gastric contents
 - Right lung, inferior lobes

LUNG ABSCESS

⇒ Haematogenous abscesses

- Septic embolism
- Thromboembolism of inferior limbs and pelvis
- Bacterial endocarditis in drug addicted
- Endocarditis in malformative cardiopathies

⇒ Lymphogenic abscesses

- Purulent infections of pleura and mediastinum
- Abdominal surgery

⇒ Abscesses for contiguity (pleura, vertebrae, liver)

⇒ Post-traumatic abscesses

LUNG ABSCESS

- Localisation: dorsal segment of superior lobes
- Single/multiple
 - ⇒ 0,5 cm (haematogenous) / 5-10 cm (bronchogenic)
 - ⇒ Grey or grey/yellowish mass
 - ⇒ Tumour-like
 - ⇒ Centrally located, draining in a brochus
 - ⇒ Content: yellow pus
 - ⇒ Wall:
 - fibrino-suppurative / necrotic-suppurative alveolitis
 - granulation tissue
 - +/- fibrosis

LUNG ABSCESS

■ Evolution:

- scarring
- cleansing with persistent cavity
- suppurative pleuritis
- haemoptysis and bronchiectasias
- metastatic abscesses

Pulmonary gangrene

Putrefactive decomposition of lung parenchyma, with progressive tendency to extension into contiguous tissues, induced by anaerobes

- Elderly subjects and/or with heavily debilitating diseases
- Often secondary to chronic lung lesions
- Restricted / diffuse form

Pulmonary Gangrene

- 1st phase: gangrenous eschar
 - necrotic mass, dry, grey-green
 - haemorrhagic halo
 - shadows of residual septi
 - central suppurative alveolitis
 - catarrhal marginal alveolitis
- 2nd phase: necrotic break up
 - extensive parenchymal necrosis
 - softening
 - cavities with irregular walls
- 3^o phase: cavernous shaping
 - transbronchial elimination of necrotic material
 - bronco-alveolar necrotic residues
 - lack of demarcation
 - sero-fibrinous consensual pleuritis
- Sudden and lethal evolution in septicemia