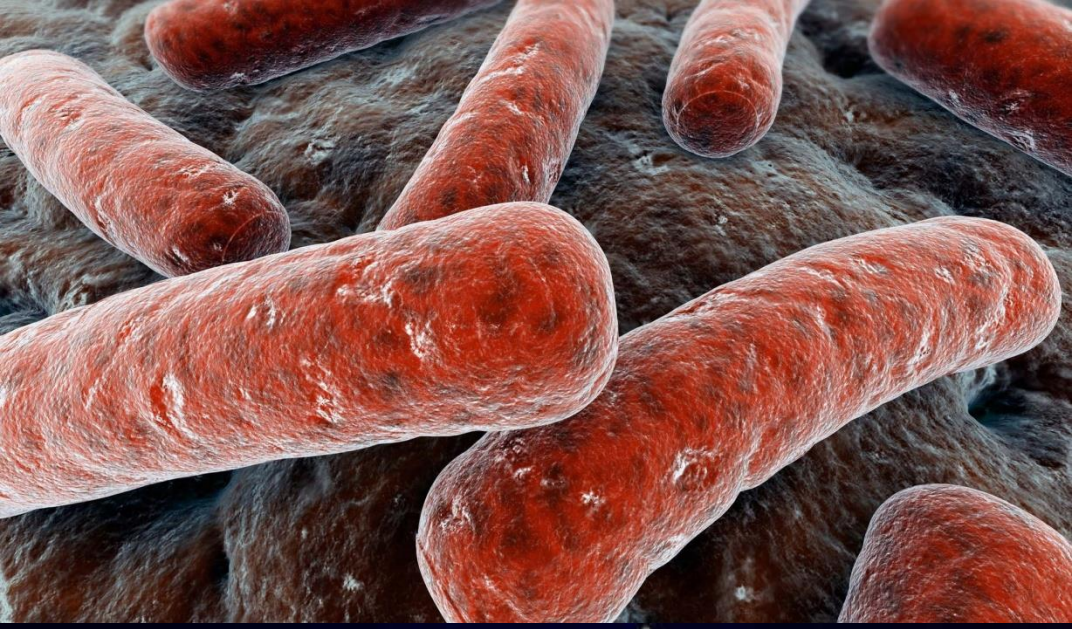
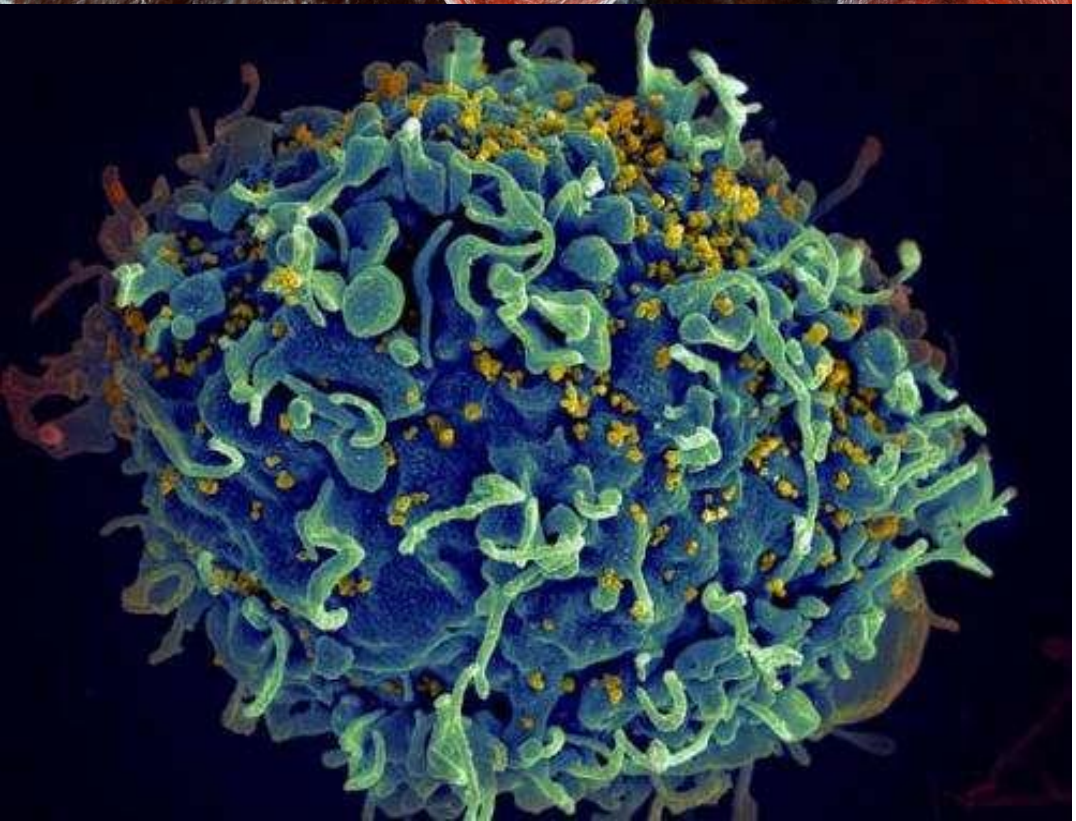




Tuberculosis. AIDS.

Tuberculosis. AIDS

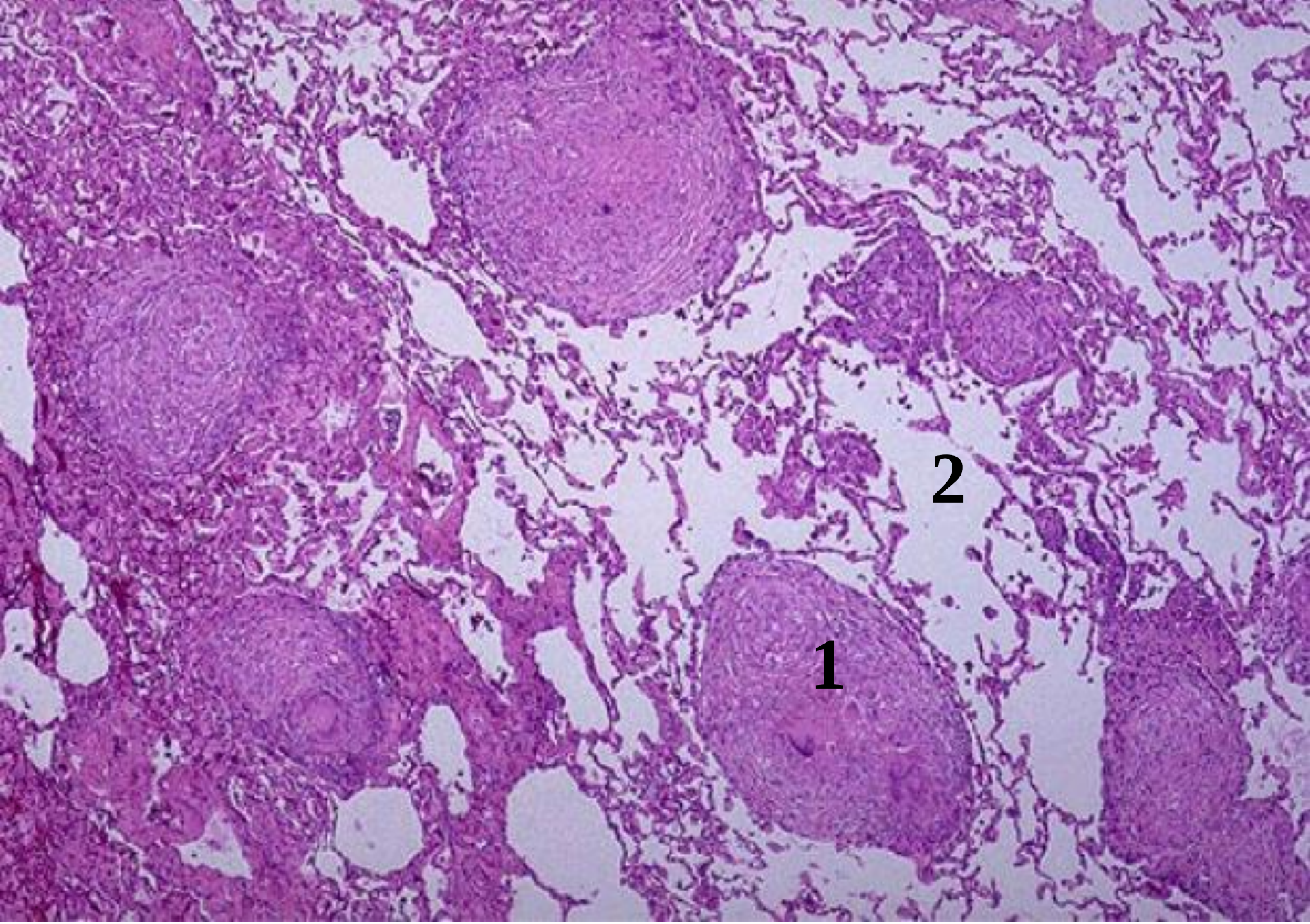
I. Microspecimens:

№ 79. Pulmonary miliary tuberculosis. *(H-E. stain).*

Indications:

1. Tuberculous granuloma:
   a. caseous necrosis in the center of granuloma;
   b. layer of epithelioid cells;
   c. giant cells Langhans;
   d. lymphoid cell layer.
2. Adjacent lung tissue.

In the lung tissue there are multiple tuberculous granulomas at different stages of development, some with caseous necrosis in the center, which is intensely colored eosinophilic, surrounded by a cell cord, consisting of epithelioid cells with elongated, pale nuclei, arranged radially, "in the palisade "; among them are giant polynuclear cells Langhans with eosinophilic cytoplasm and nuclei placed in the shape of a horseshoe, circular along the membrane or 2 poles of the cell, and at the periphery - a layer of small lymphocytes, compactly placed, with round nucleus, hyperchrome and poor cytoplasm, which may include macrophages and plasma cells; around some granulomas are collagen fibers; other granulomas are in the fibrosis stage (replacement with fibrous connective tissue); In the lung parenchyma between granulomas, foci of emphysema are observed, some interalveolar septa are thickened, sclerosed.



№ 79. Pulmonary miliary tuberculosis. *(H-E. stain).*

№ 80. Hepatic miliary tuberculosis. *(H-E. stain).*

Indications:

1. Miliary nodules in the liver tissue.
2. Adjacent liver tissue.

Tuberculous granulomas with the same structure as in the microspecimen № 79 are revealed in the liver tissue; adjacent hepatocytes with signs of proteic degeneration.

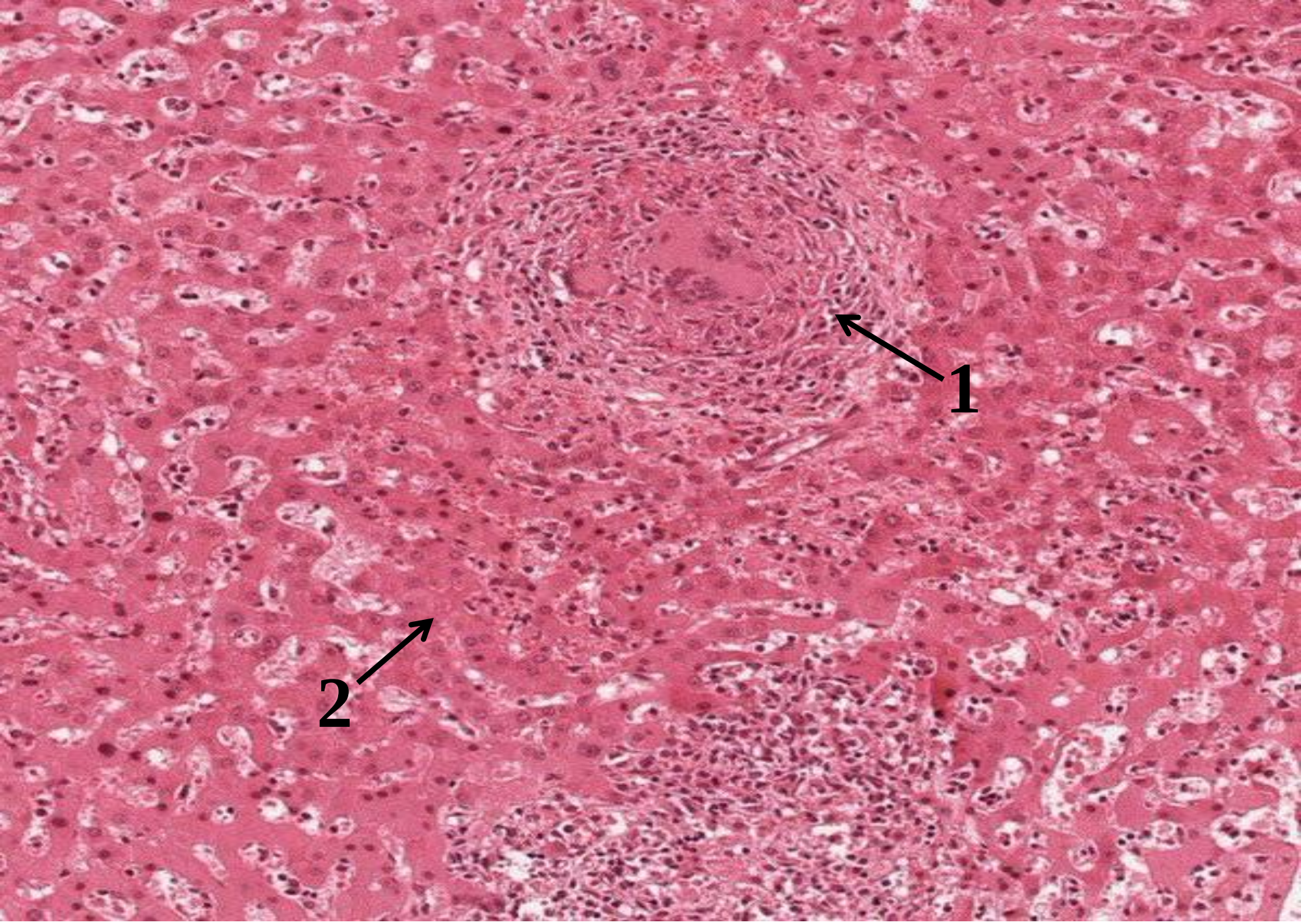
№ 82. Renal miliary tuberculosis. *(H-E. stain).*

Indications:

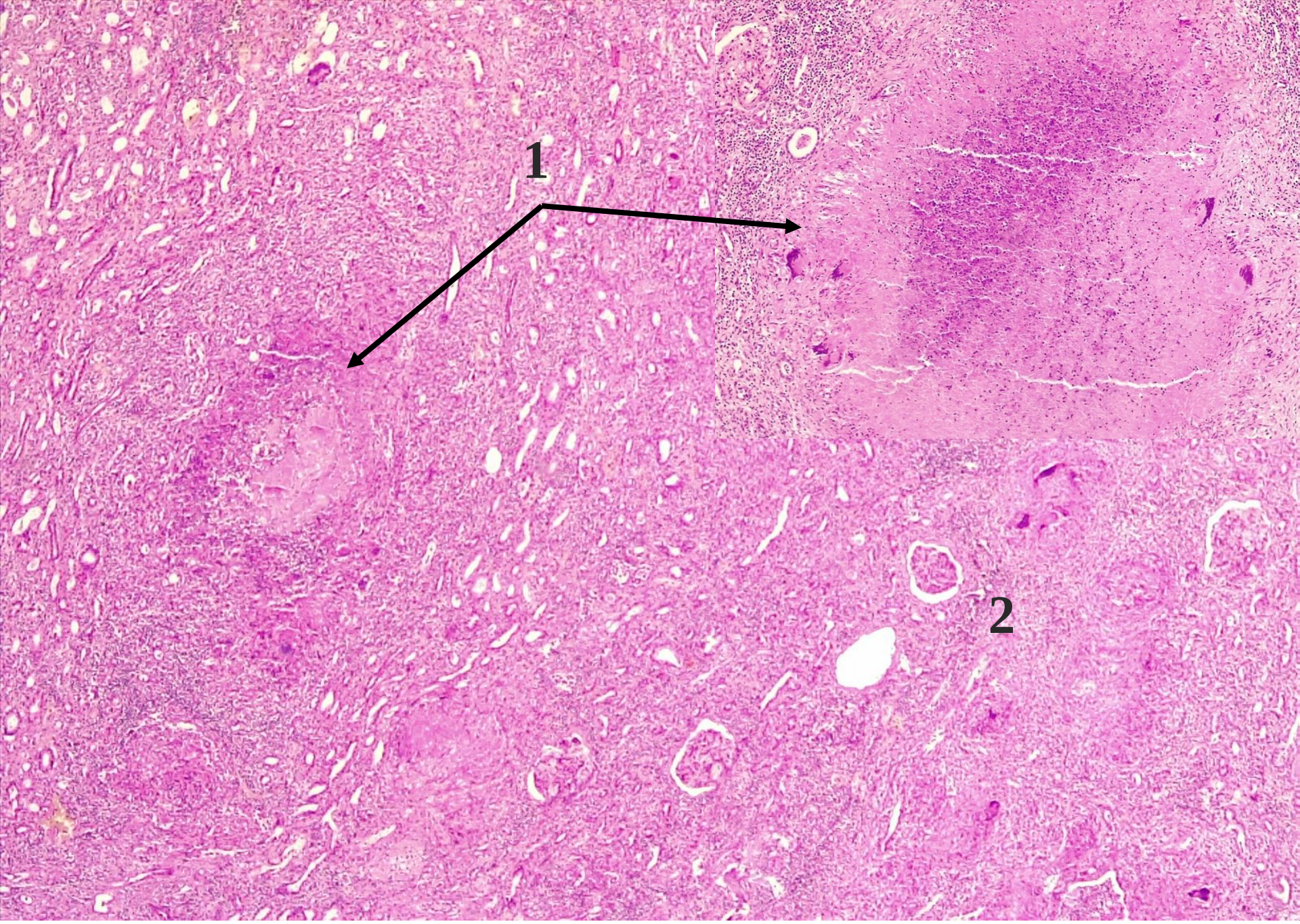
1. Tuberculous granuloma.
2. Adjacent renal tissue.

In the renal tissue are observed multiple tuberculous granulomas of different sizes, in some places confluent, with structure analogous to lung granulomas in microspecimen № 79, the necrotic center is well pronounced, intensely eosinophilic, with remnants of nuclei (cariorexis) colored basophilic, palisade disposition is clearly highlighted of epithelioid cells and numerous Langhans cells; moderate lymphohistiocytic infiltration (interstitial nephritis) is present in the stroma of renal tissue between granulomas.

Miliary tuberculosis (microspecimen № 79, 80, 82) occurs in the case of hematogenous spread of the infection. It is observed in both primary and secondary tuberculosis. In cases when bacilli enter the pulmonary vein, extrapulmonary miliary tuberculosis develops with damage to the liver, kidneys, spleen, brain, meninges, genitourinary system, bone marrow, adrenal glands or isolated organs, and when the infection enters the pulmonary artery, it develops. lung disease with damage to both lungs. In the affected organs appear foci of condensation of white-yellow color, with a diameter of 1-2 mm (the dimensions of a millet grain), evenly distributed throughout the organ, which microscopically have the structure of tuberculous granuloma. The most serious localization is tuberculous meningitis.



№ 80. Hepatic miliary tuberculosis. *(H-E. stain).*



№ 82. Renal miliary tuberculosis. *(H-E. stain).*

№ 85. Caseous pneumonia. *(H-E. stain).*

Indications:

1. Caseous necrosis area.
2. Interalveolar septa without nuclei (karyolysis).
3. Connective tissue infiltrated by lymphoid cells.
4. Adjacent emphysematous pulmonary tissue.

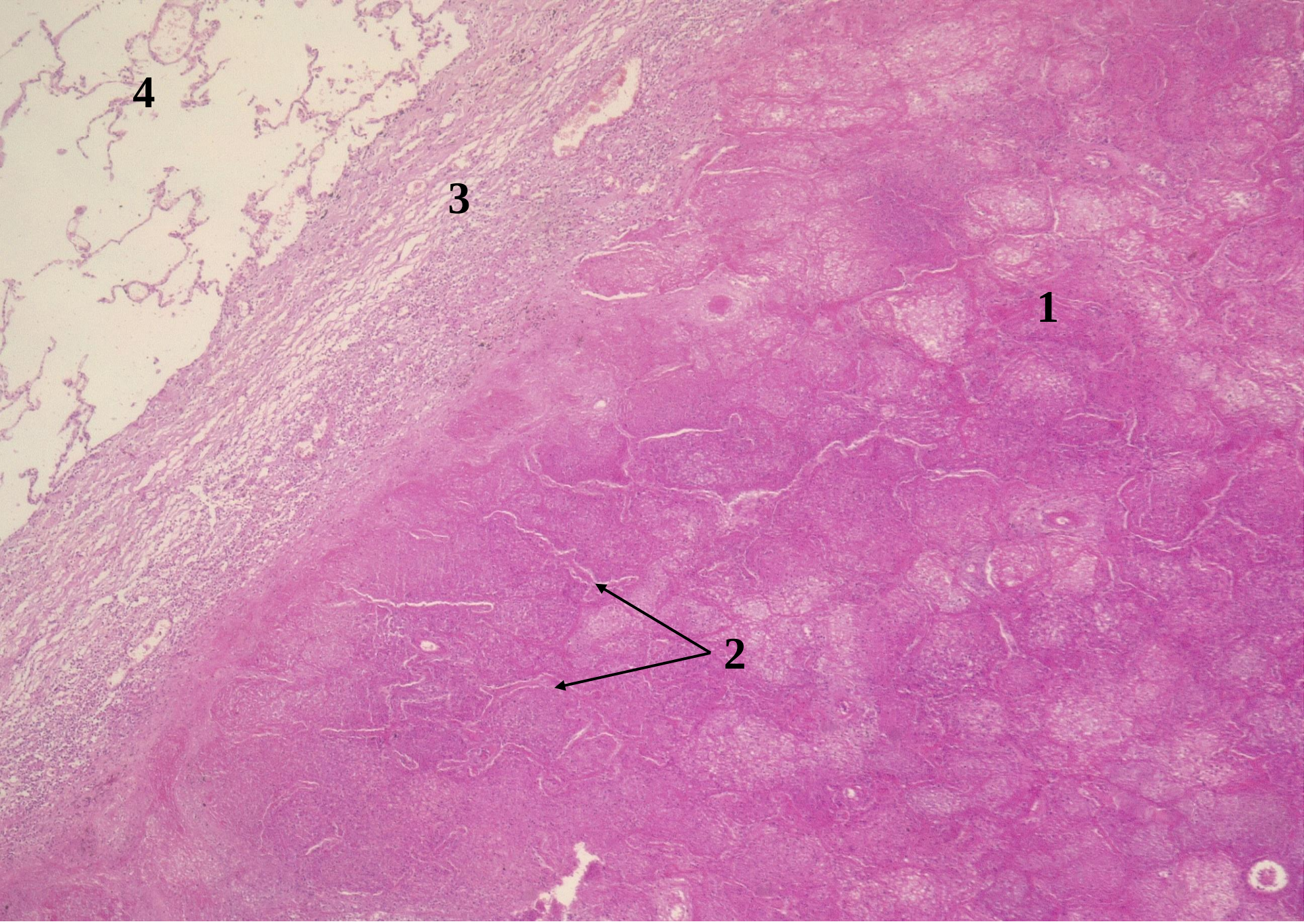
In the microspecimen there is an extensive area of necrosis of lung tissue, unventilated, the alveolar lumen contains intensely colored necrotic masses eosinophilic, fibrin, neutrophilic leukocytes, monocytes, disintegrated nucleus remains, necrotic interalveolar septa, devoid of nuclei with moderate lymphoid infiltration; in the adjacent lung tissue signs of emphysema.

II. Macrospecimens:

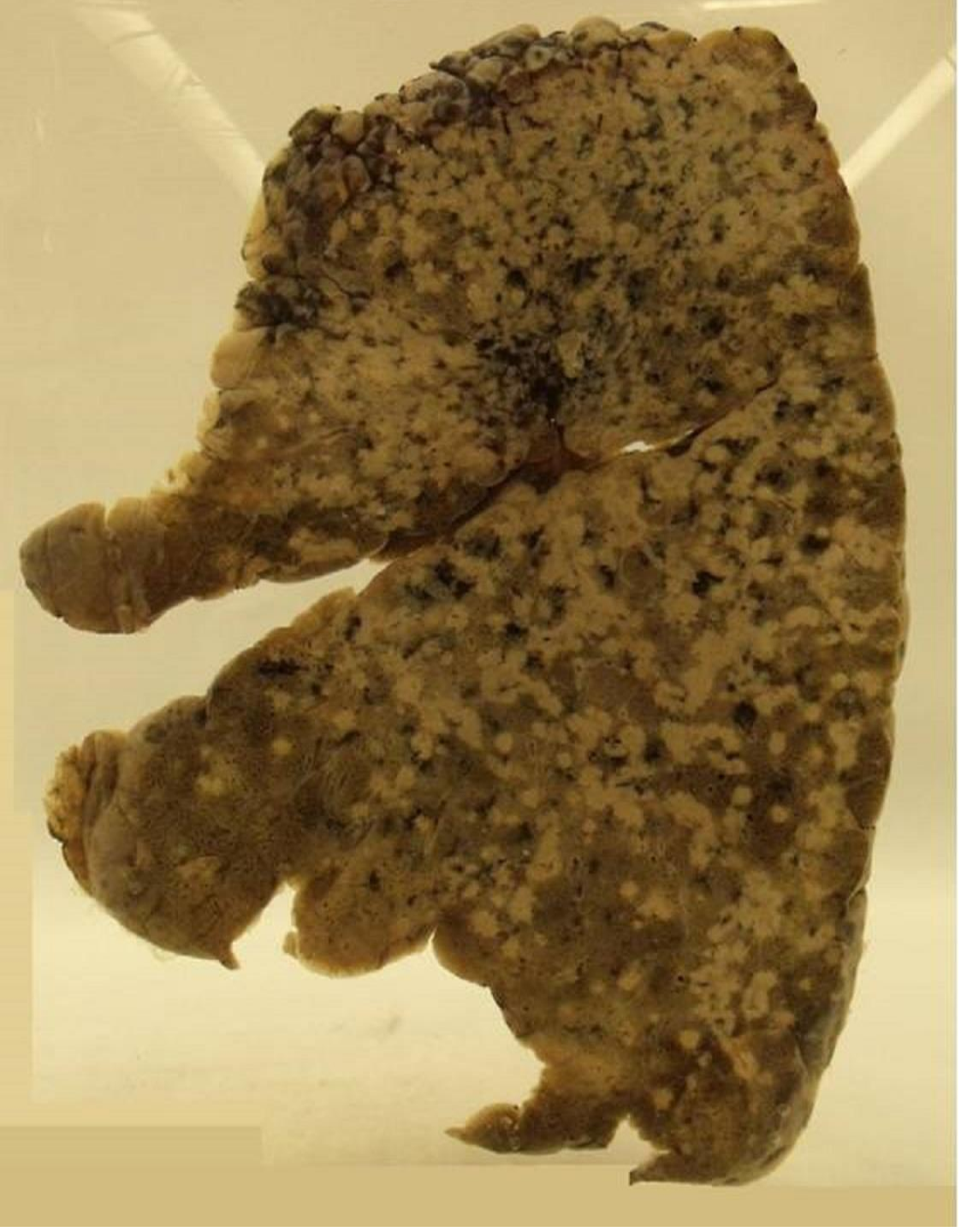
№ 43. Caseous pneumonia.

In the lung there are multiple foci of caseous necrosis, unventilated, of different sizes, white-yellow color, the necrotic masses have a friable, crumbly appearance, it resembles dry cow's cheese (lat. Caseum - cheese).

Caseous pneumonia is found in secondary tuberculosis, but can also be in primary tuberculosis. Initially, acinar, lobular caseous outbreaks appear, which can extend to the level of a segment or even of an entire lobe - lobar caseous pneumonia. It develops in patients with low immunity, malnourished. There are deposits of fibrin in the pleura. The curd masses can be subjected to purulent lysis and liquefaction with the appearance of decomposition cavities - caverns (cavernous tuberculosis).



№ 85. Caseous pneumonia. *(H-E. stain).*

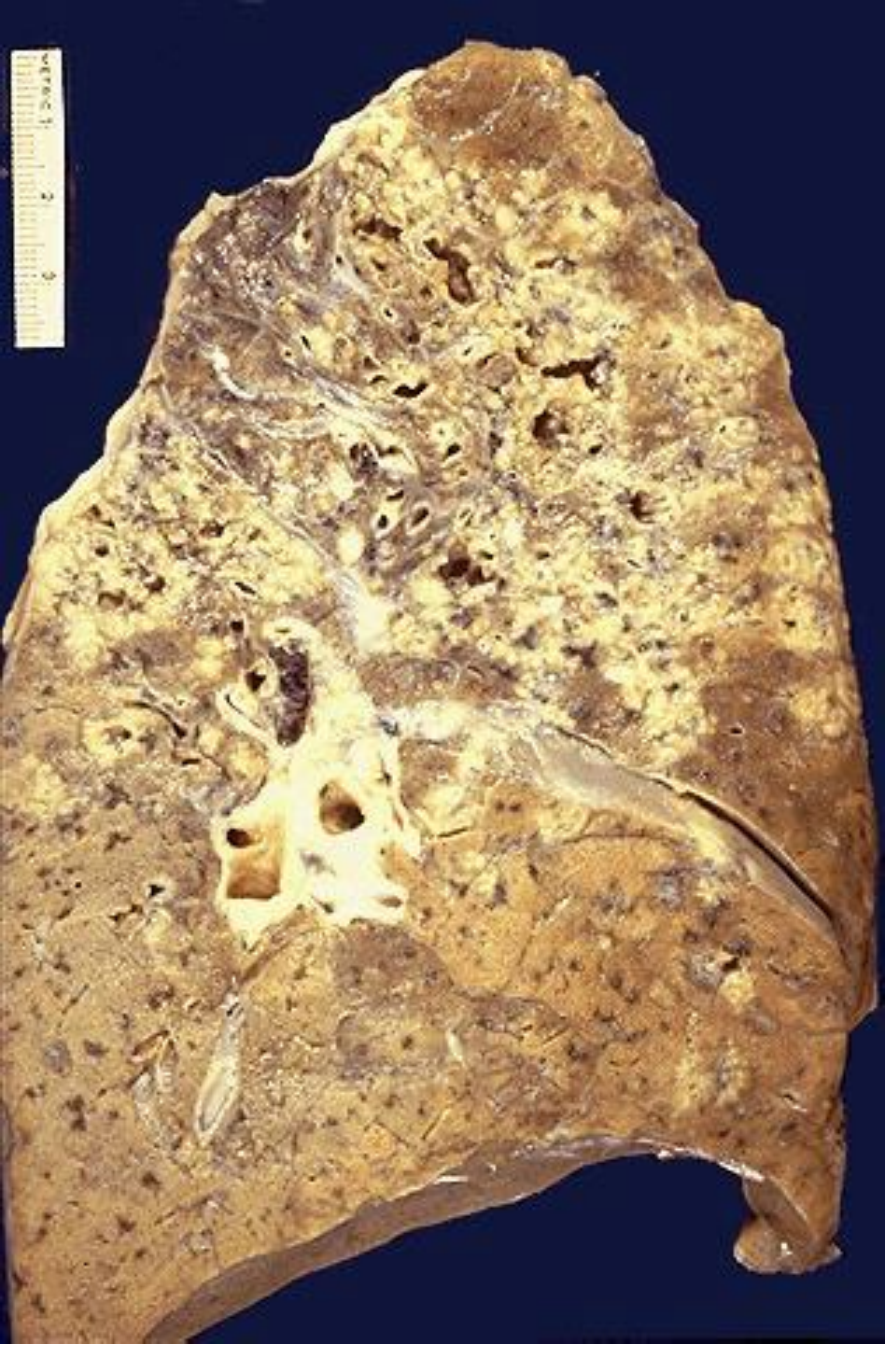


№ 43. Caseous pneumonia.

№ 44. Fibrocavitary tuberculosis.

The lung is deformed, on the section are observed multiple cavities of destruction - caverns of irregularly shaped, different sizes with thickened, sclerosed walls, rough internal surface, covered with necrotic masses; in the adjacent lung tissue unventilated white-yellow areas of caseous necrosis, pneumosclerosis, thickened bronchial walls may be seen.

Fibro-cavitary tuberculosis is a form of secondary pulmonary tuberculosis. In general, caverns are much more common in secondary tuberculosis than in primary tuberculosis. The formation of cavities for the destruction of lung tissue begins in the apical areas of the right lung and extends in the apico-caudal direction through direct contact and bronchogenic in the middle and lower lobes. The apical caverns are older than the distal ones. They have different sizes, irregular shape, walls consisting of 3 layers: caseous necrotic masses infiltrated with neutrophilic leukocytes, tuberculous granulation tissue, mature connective tissue. If the cavern is drained and communicates with the bronchi, the curd contents extend bronchially into the contralateral lung. At the same time, endobronchial, endotracheal, laryngeal and intestinal tuberculosis can develop by ingesting sputum containing tuberculous bacilli. In cases, when the contents of the cavern are evacuated bronchially, it collapses and heals. Possible complications: respiratory failure, pulmonary hemorrhage, pulmonary heart, secondary amyloidosis; in patients with compromised immunity, lymphatic and hematogenous dissemination may occur with the development of miliary tuberculosis.

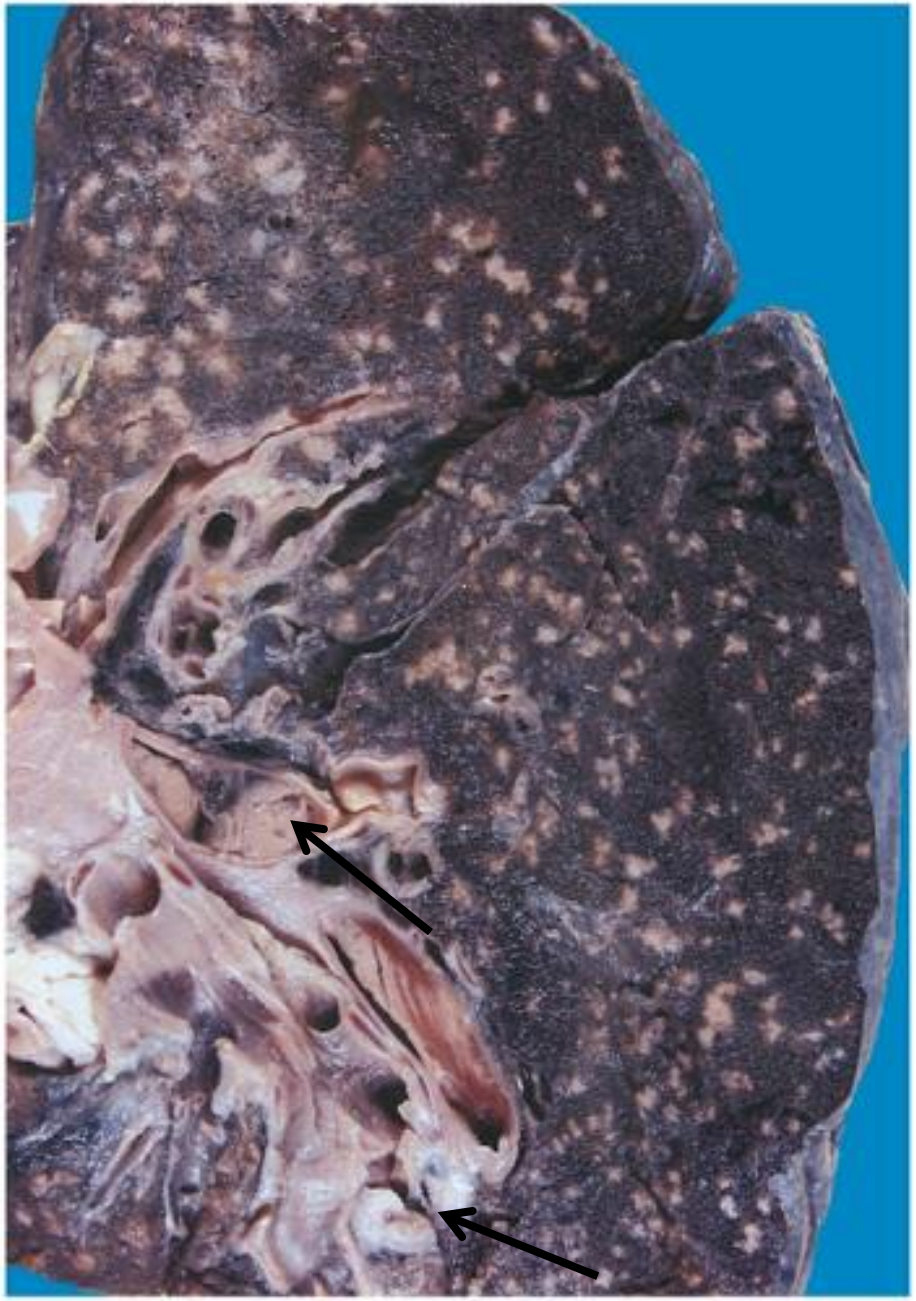


№ 44. Fibrocavitary tuberculosis.

№ 144. Tuberculosis of peribronchial lymph nodes.

The peribronchial lymph nodes are enlarged in size, dense, adhere closely to each other, forming bundles, conglomerates, on the section white-yellow color, dry cheese appearance.

Impaired lymph nodes are the most common manifestation of pulmonary tuberculosis. It is found primarily in primary tuberculosis as a component part of the primary tuberculous complex or the Gohn complex (primary affect, lymphangitis and lymphadenitis). In primary pulmonary tuberculosis, the hilar and bronchopulmonary nodules are affected, and in primary intestinal tuberculosis - mesenteric lymph nodes. In the initial period of secondary pulmonary tuberculosis, regional lymph nodes are much less affected due to the location of the tuberculous process in the apical areas of the lungs. Enlarged lymph nodes compress the nerves, blood vessels, neighboring organs, causing certain clinical manifestations. Viable tubercle bacilli may persist in the lymph nodes for several years, with the potential to reactivate the infection and develop secondary tuberculosis under conditions of decreased immunity.

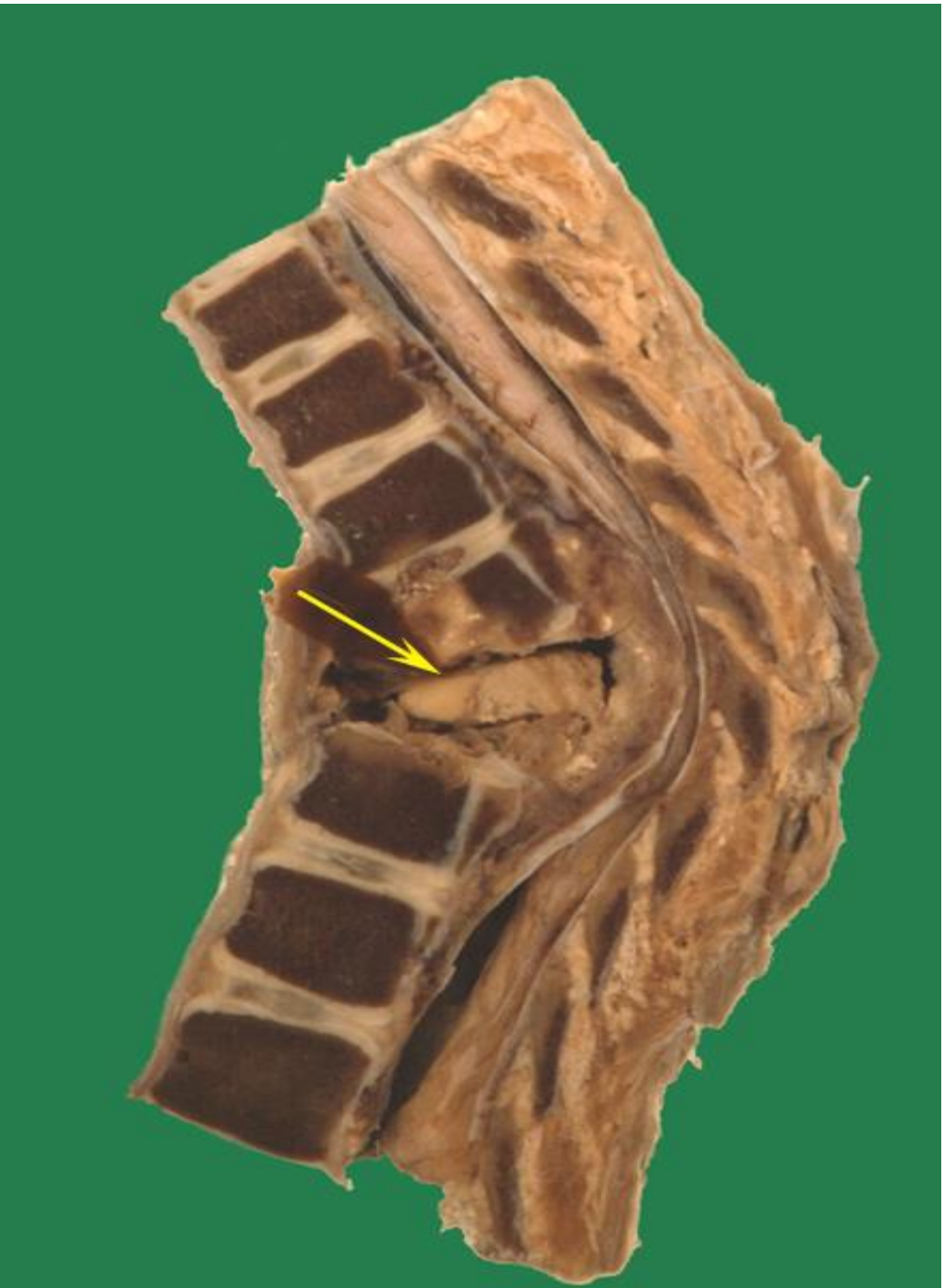


№ 144. Tuberculosis of peribronchial lymph nodes.

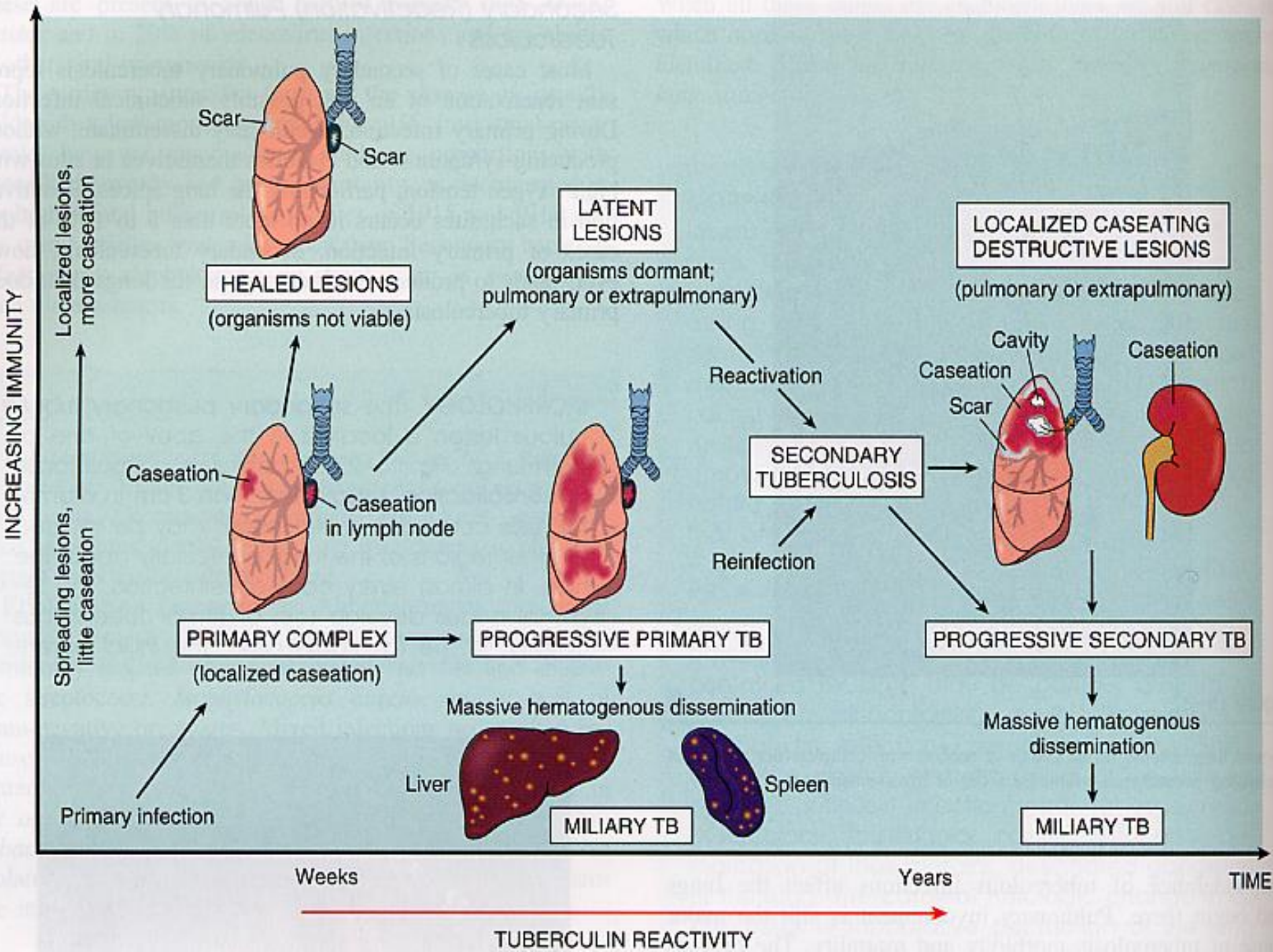
№ 153. Tuberculous spondylitis.

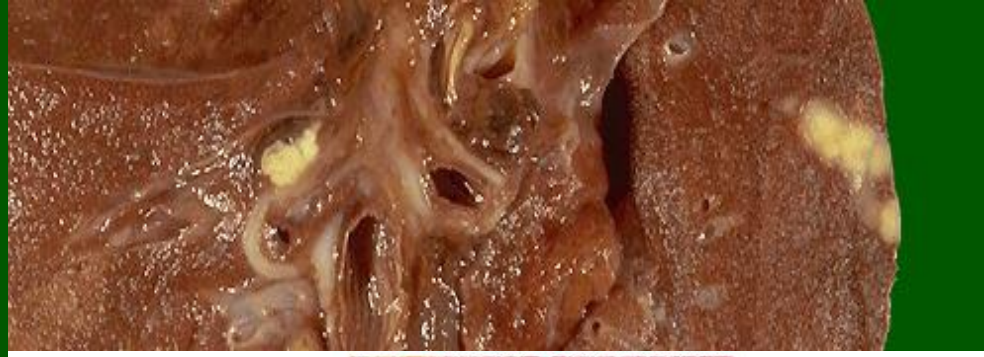
In the macrospecimen, there is a segment of the spine, the lumbar region, the deformation of the spine is observed, on the section the bodies of some vertebrae are destroyed, the apophyses are preserved, a cavity of destruction is outlined, the vertebrae are grown together.

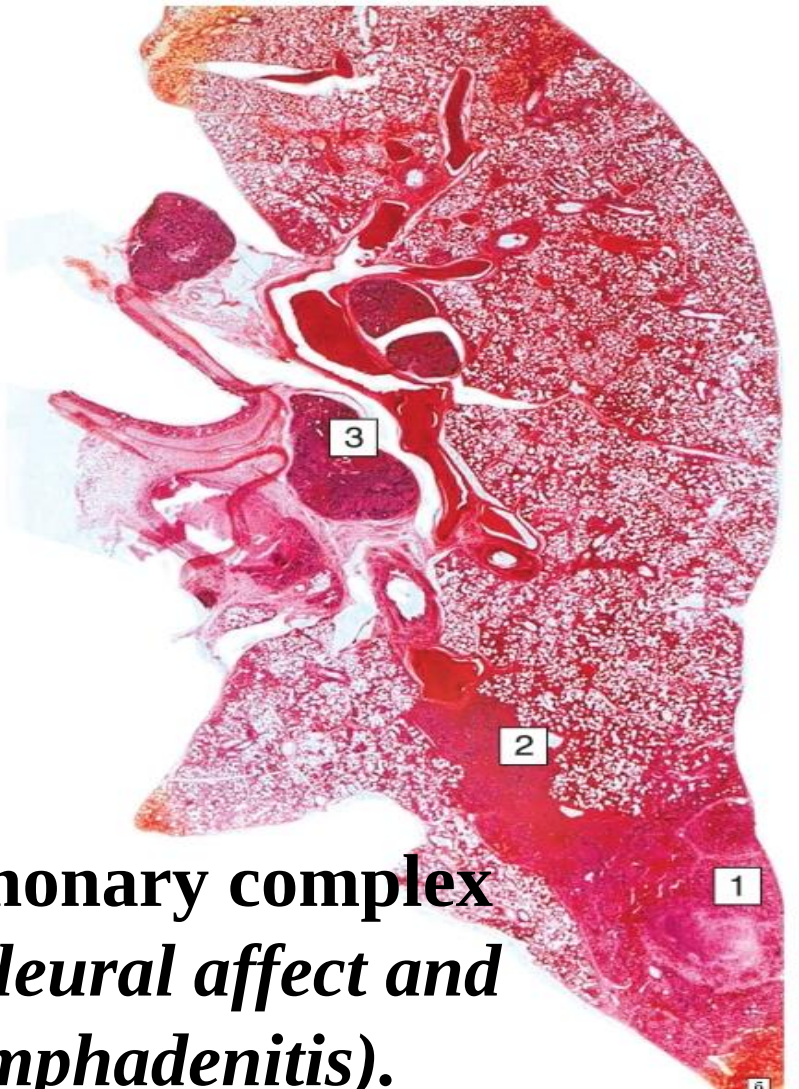
Spinal cord injury in tuberculosis (tuberculous spondylitis or Pott's disease) is found in miliary tuberculosis following the hematogenous spread of tuberculosis mycobacteria. It is more common in children and adolescents. It affects the bodies of the vertebrae, in which tuberculous osteomyelitis with caseous necrosis occurs, destruction of bone tissue and intervertebral discs, seizures are formed, filled with necrotic and purulent masses and consequently deformity of the spine occurs with the appearance of a convex curve in the region chest (kyphosis). Necro-purulent masses can spread to the soft paraspinal tissues forming "cold" abscesses, which can fistulate the skin by removing the contents of the abscesses. Chronic tuberculosis spondylitis can be complicated by secondary amyloidosis. At the same time, it can affect the coxo-femoral joint (tuberculous coxitis) and the knee (tuberculous gonitis).



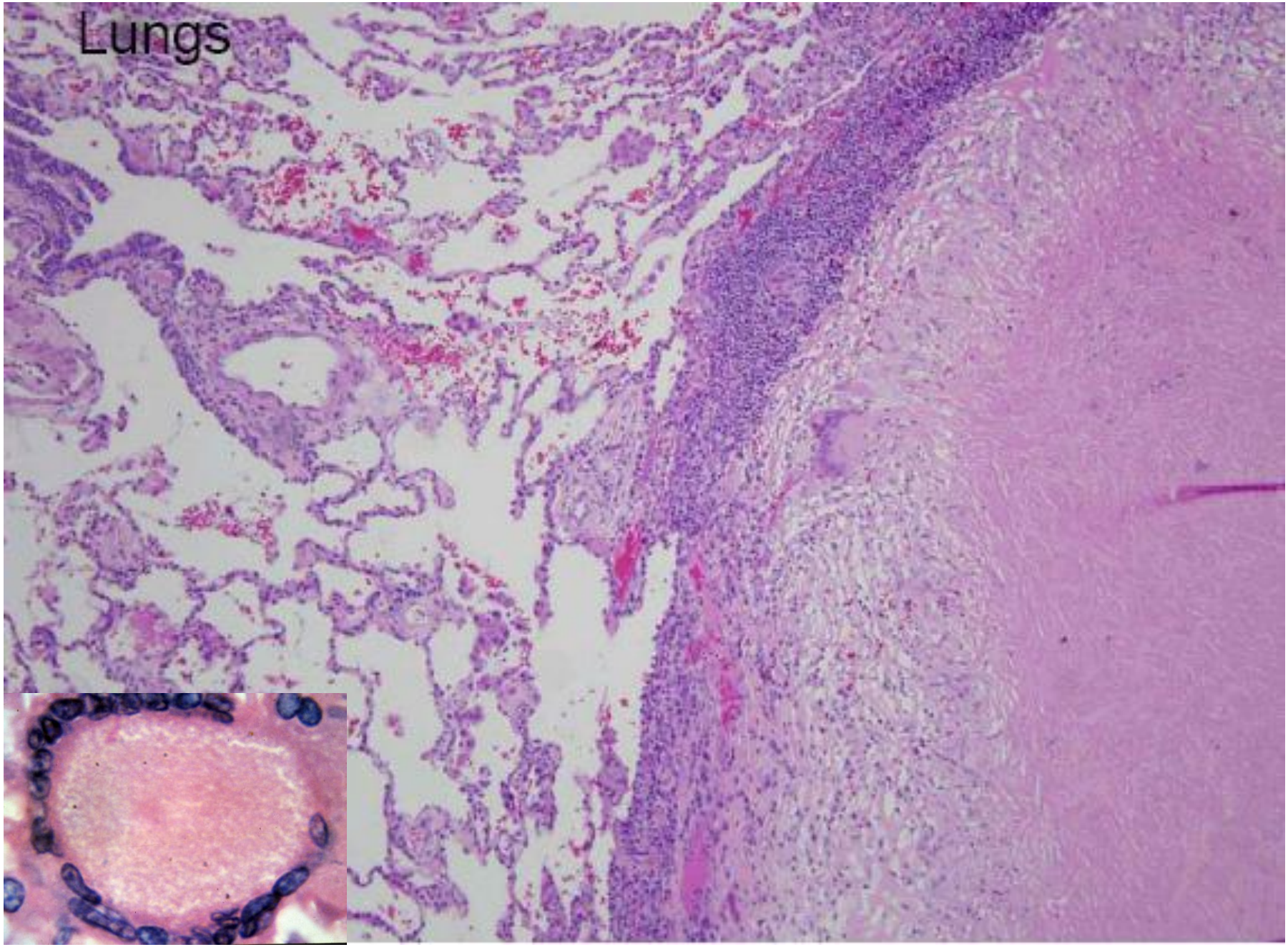
№ 153. Tuberculous spondylitis. *(Pott disease)*.


INCREASING IMMUNITY
Localized lesions, more caseation
Spreading lesions, little caseation
Scar
Scar
HEALED LESIONS
(organisms not viable)
LATENT LESIONS
(organisms dormant; pulmonary or extrapulmonary)
LOCALIZED CASEATING DESTRUCTIVE LESIONS
(pulmonary or extrapulmonary)
Reactivation
Cavity
Caseation
Caseation
Scar
Caseation
Caseation in lymph node
SECONDARY TUBERCULOSIS
Reinfection
PRIMARY COMPLEX
(localized caseation)
PROGRESSIVE PRIMARY TB
PROGRESSIVE SECONDARY TB
Massive hematogenous dissemination
Massive hematogenous dissemination
Liver
Spleen
MILIARY TB
MILIARY TB
Primary infection
Weeks
Years
TIME
TUBERCULIN REACTIVITY

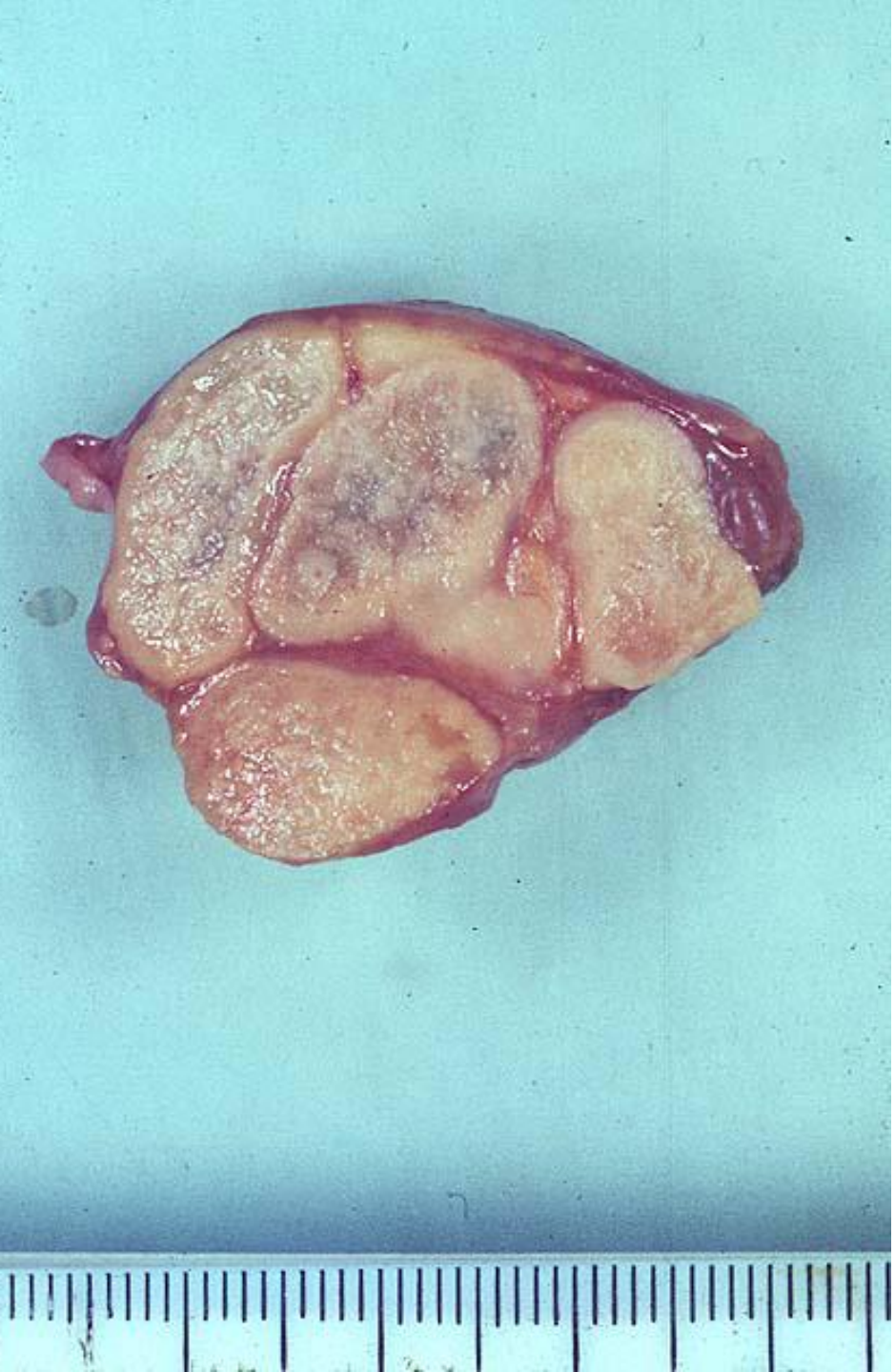


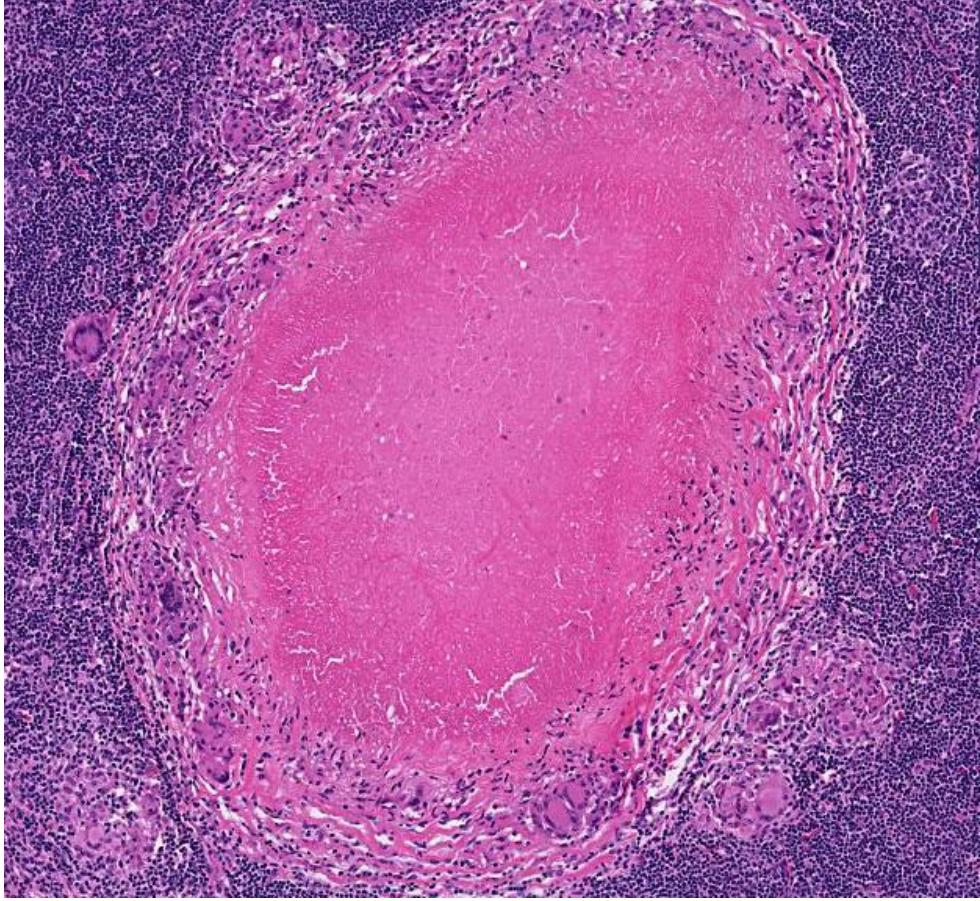


Primary pulmonary complex *(primary subpleural affect and caseous lymphadenitis).*



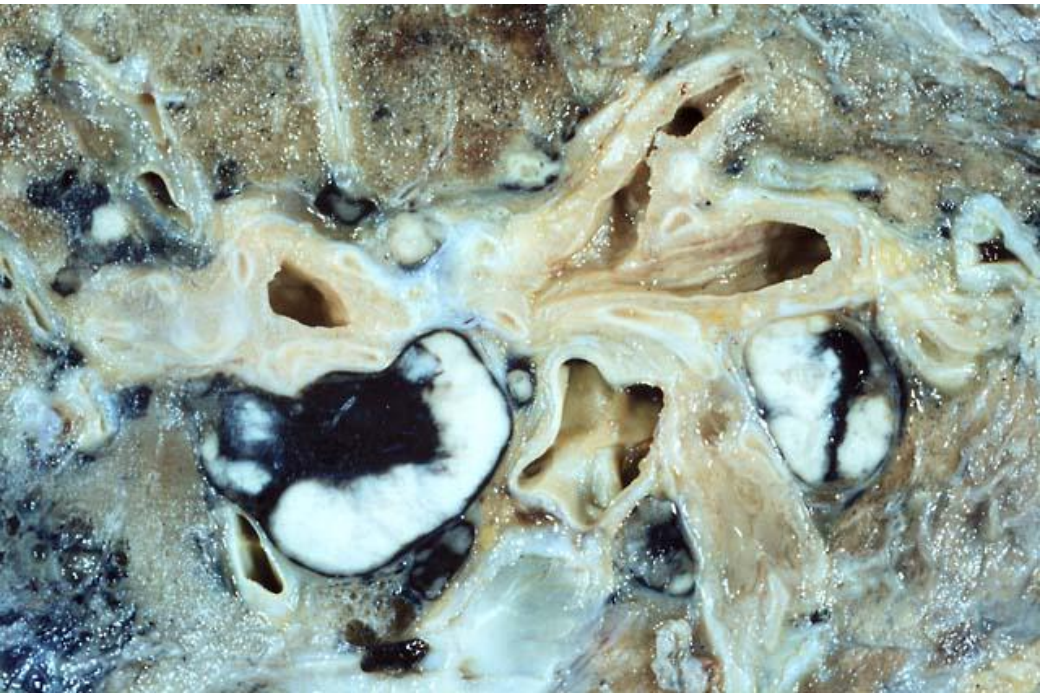
Primary pulmonary affect.



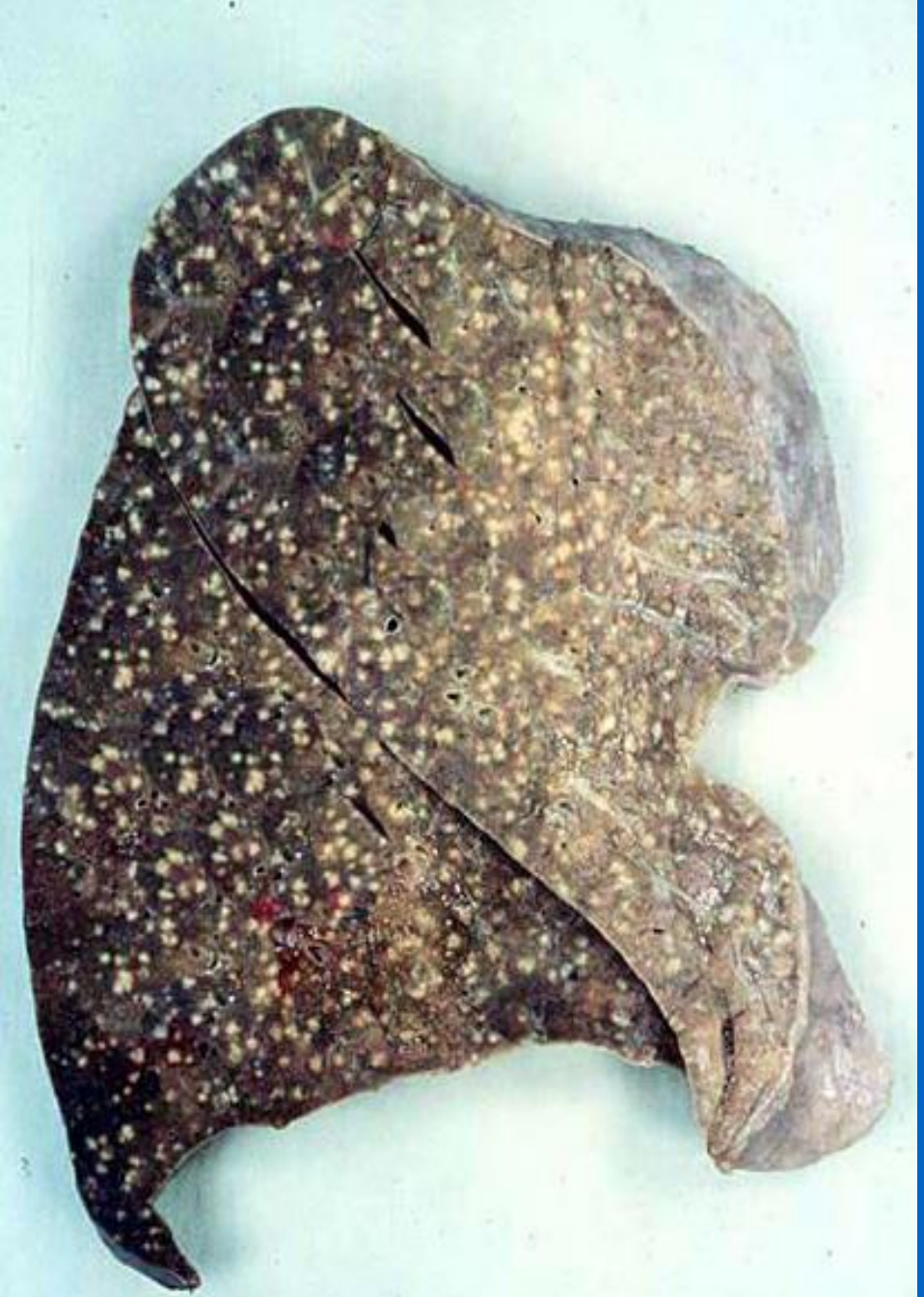


Tuberculous lymphadenitis.

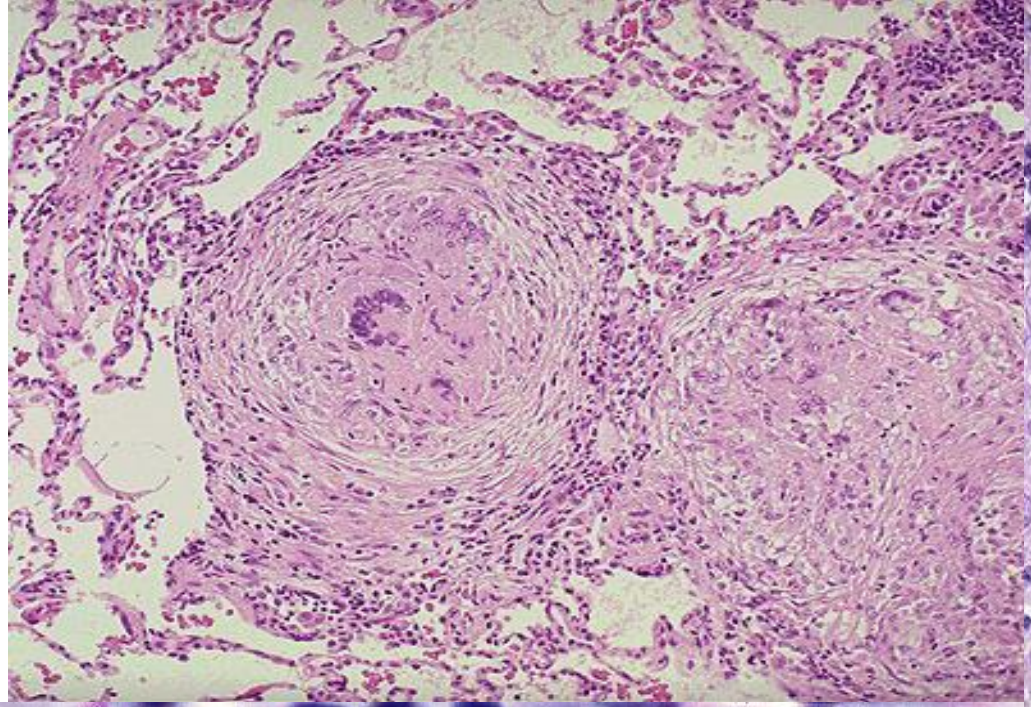
Primary intestinal complex.

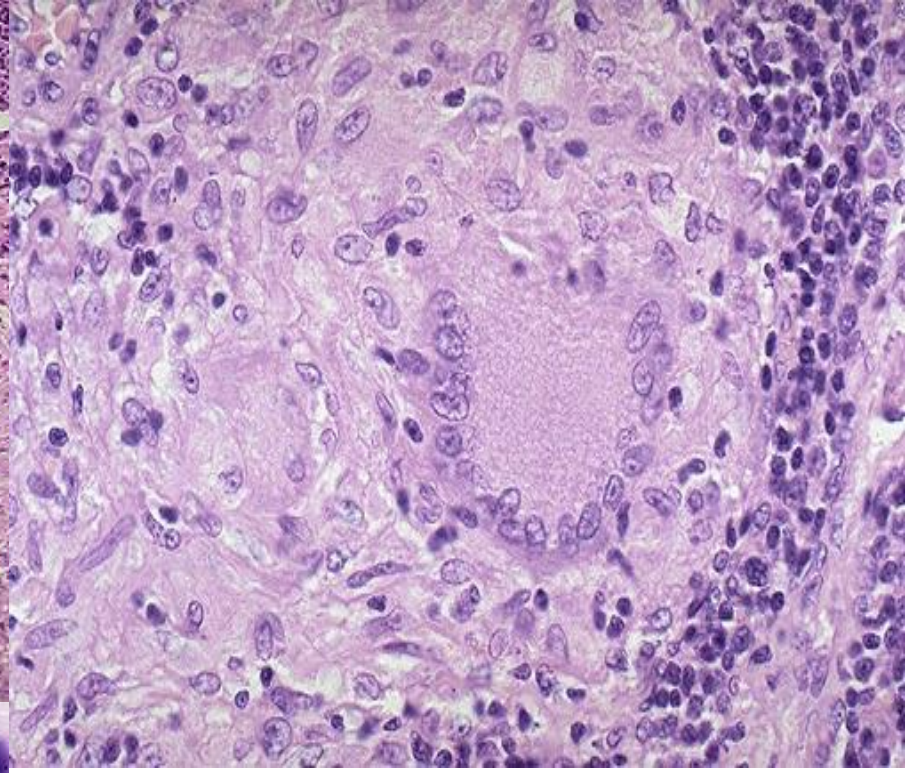


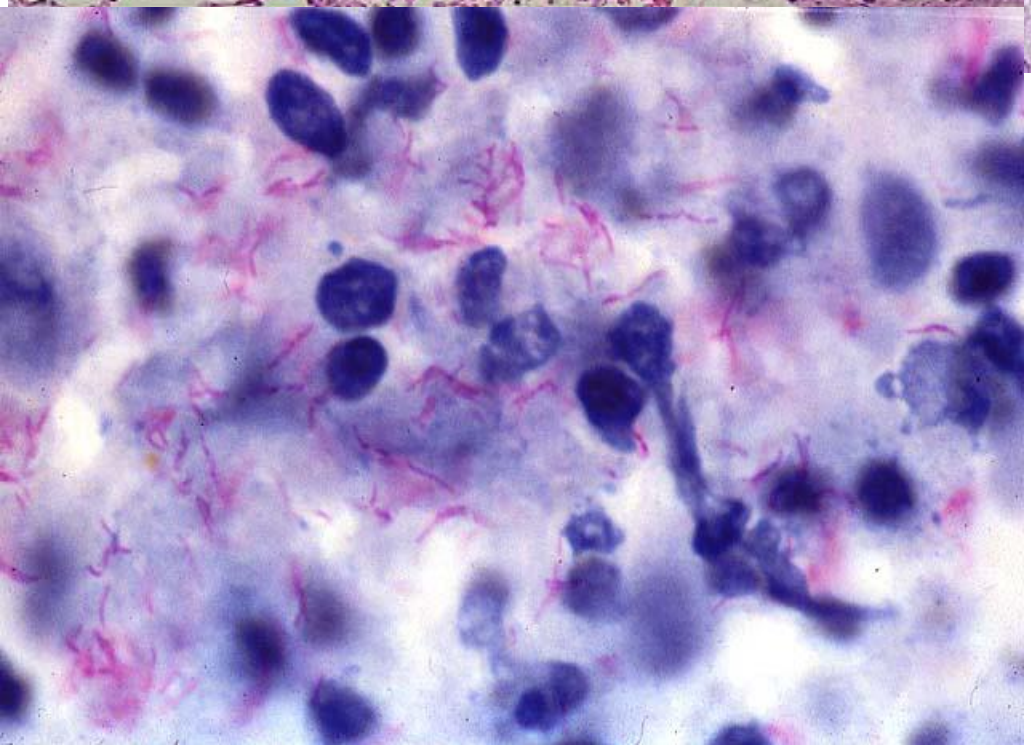
Healed primary complex *(scarring of the primary affect and calcified lymph nodes).*



Pulmonary miliary tuberculosis.

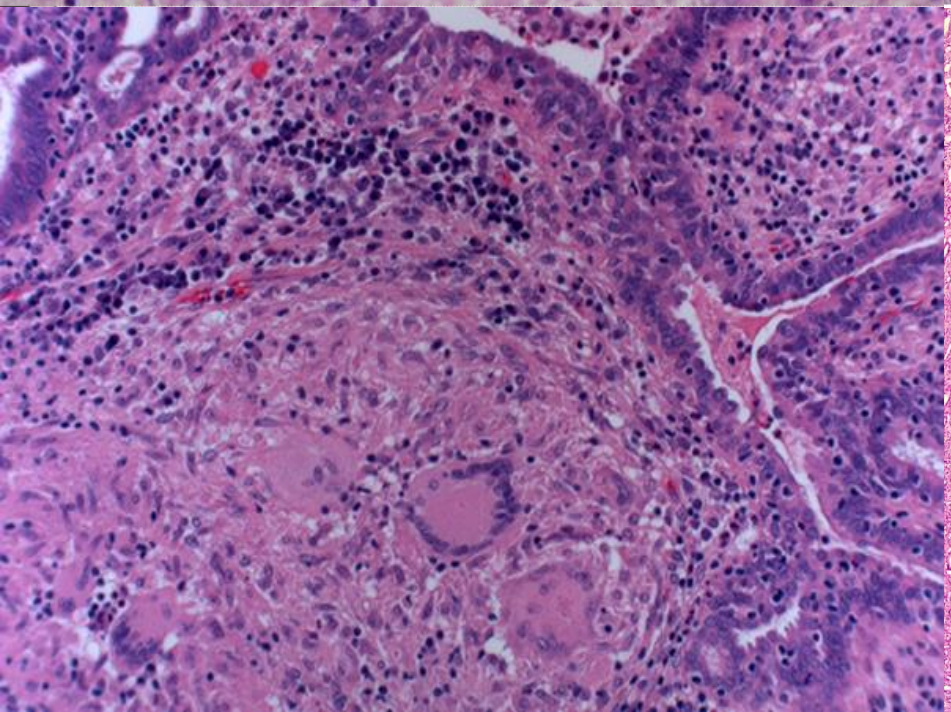


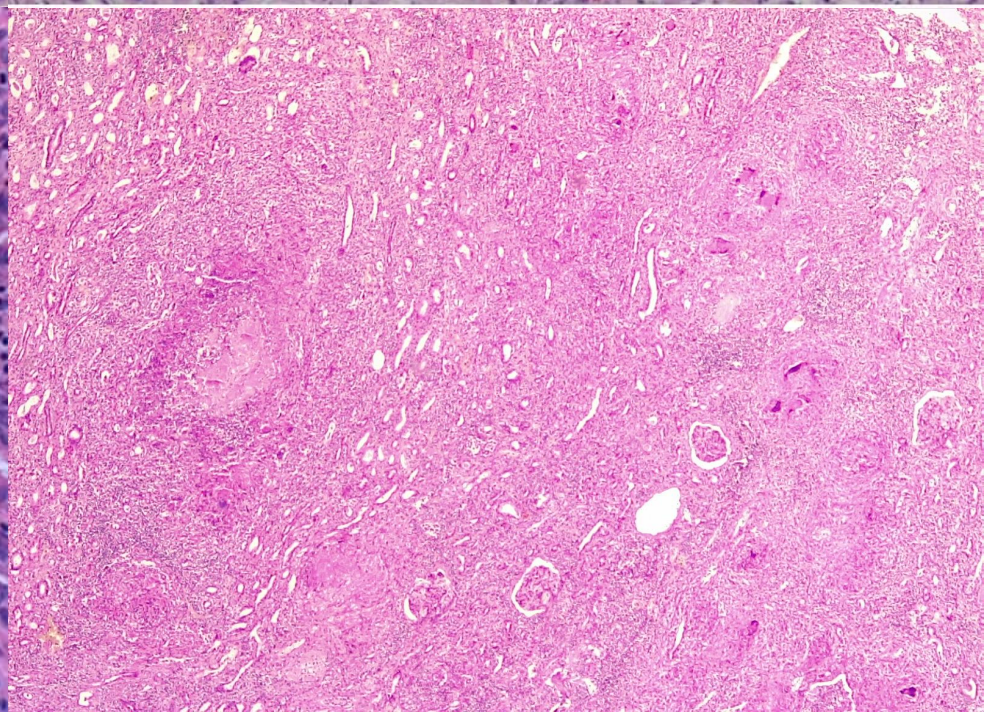


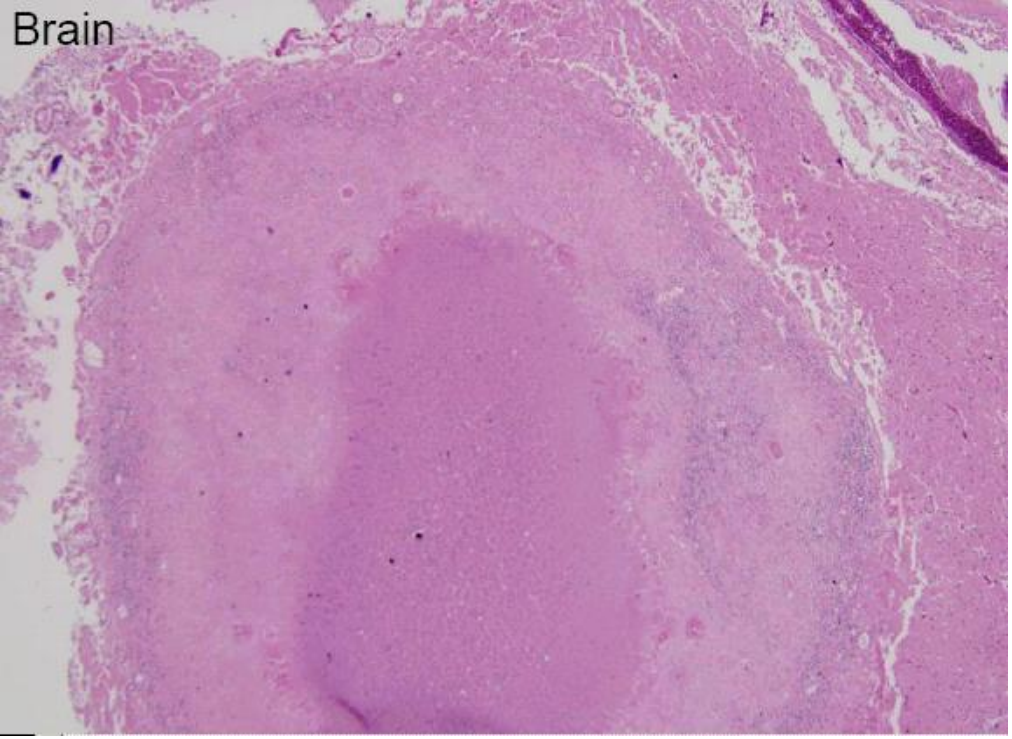


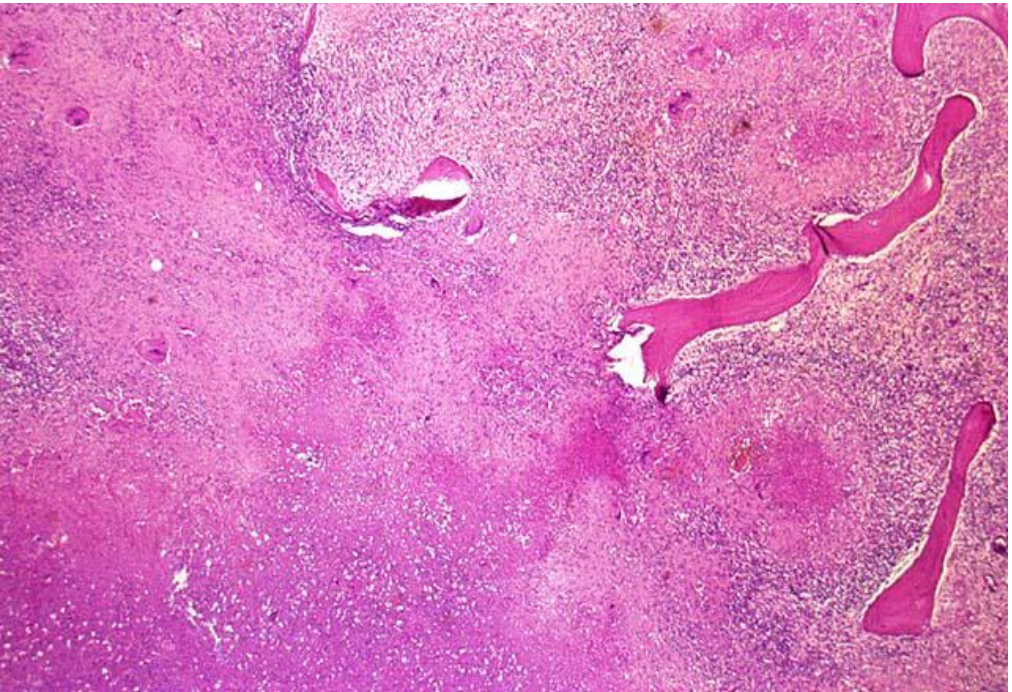
Tuberculous granulomas with giant Langhans cells, mycobacteria *(Ziehl-Nielsen staining)*.

Hematogenous tuberculosis, granulomas in the liver, adrenal glands, salpinges and kidneys.

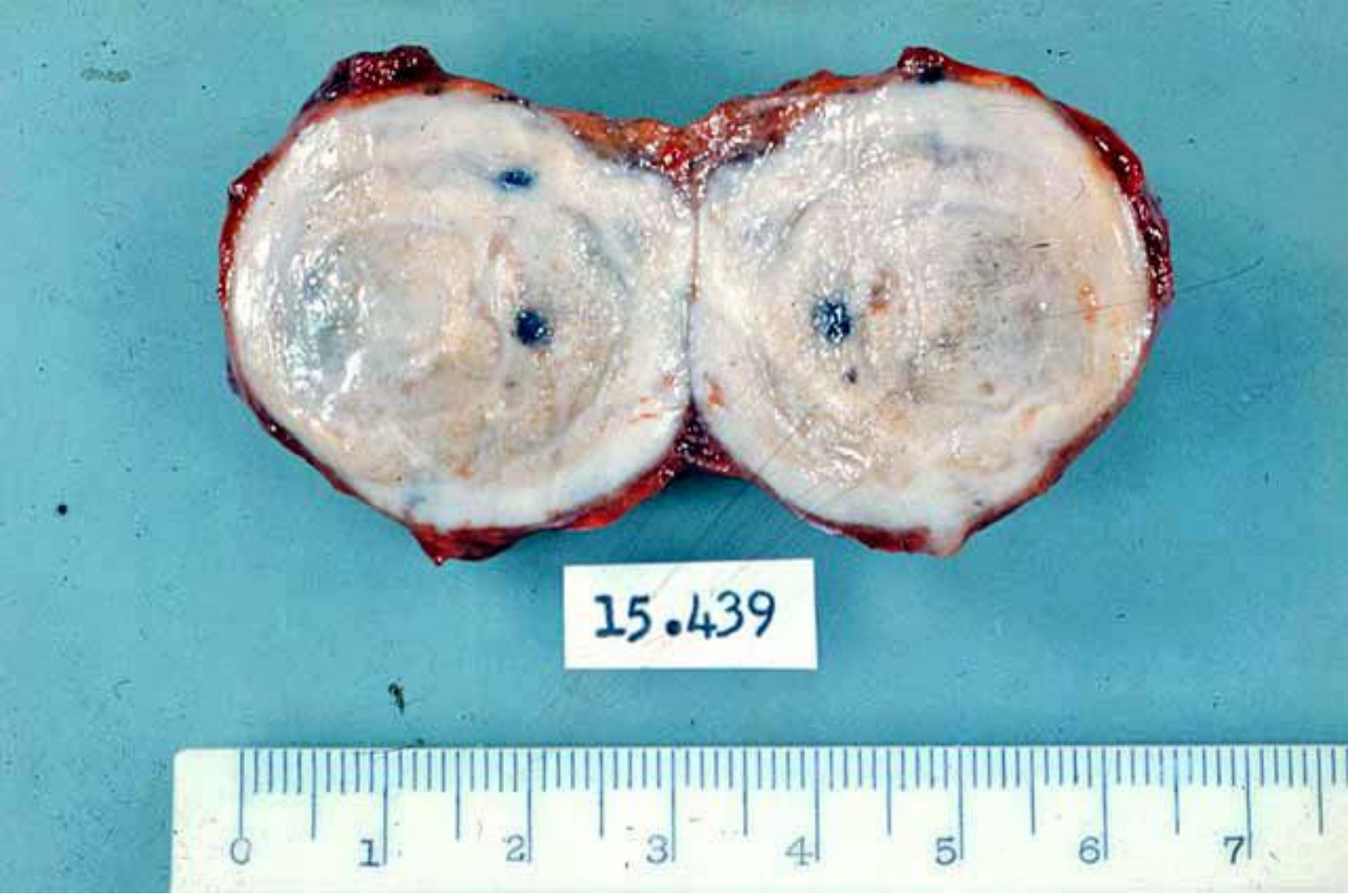




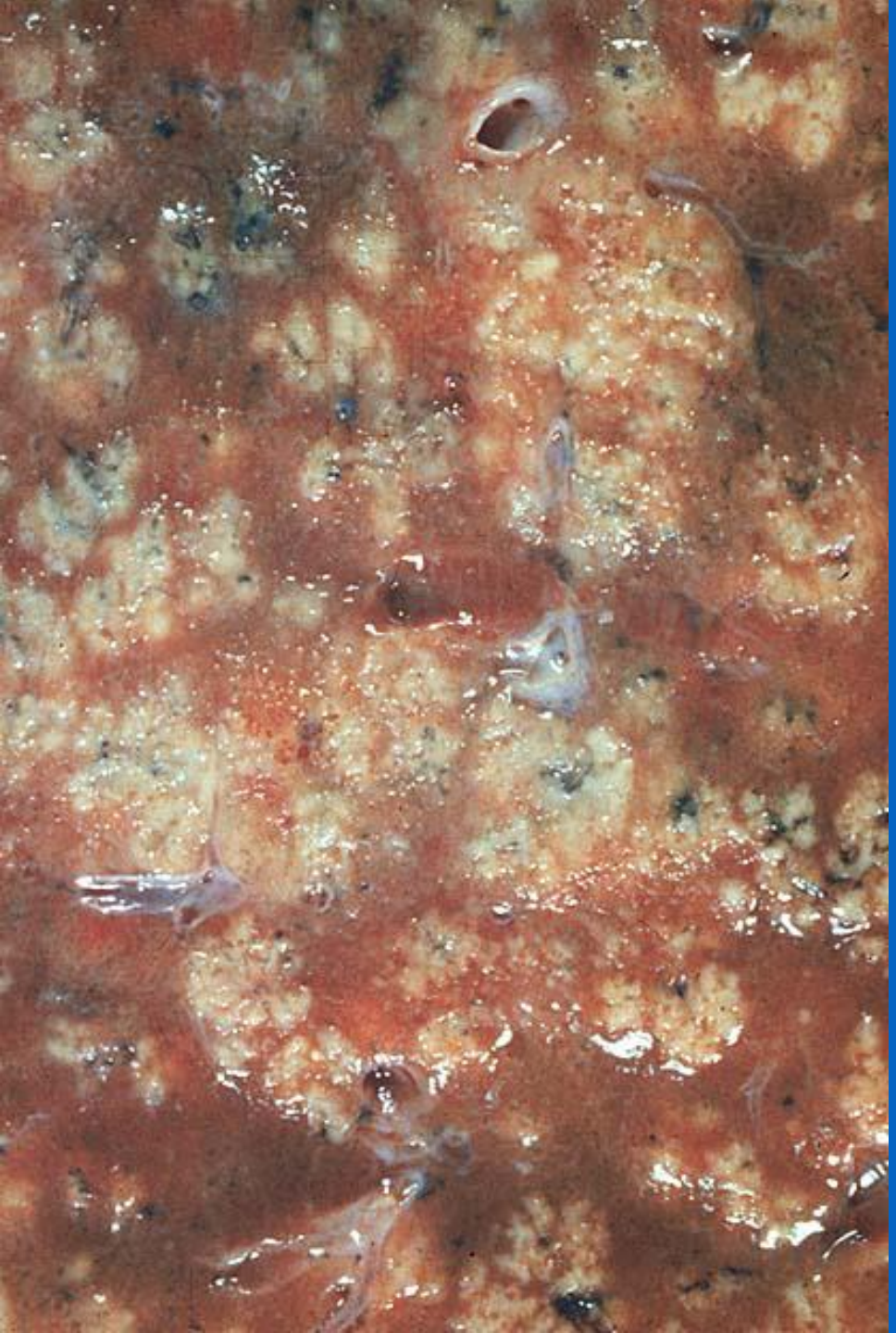


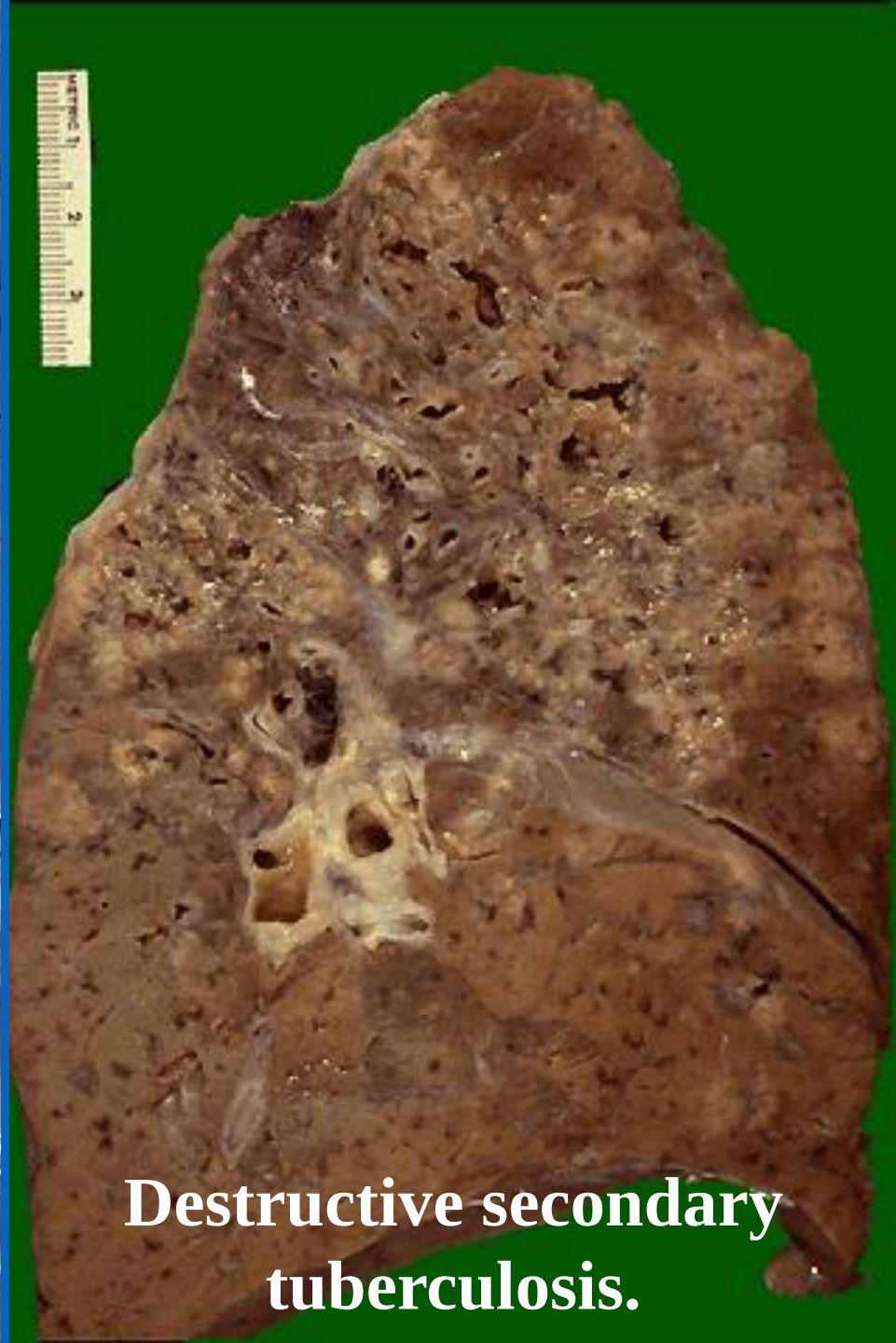


Hematogenous tuberculosis, granulomas in the brain and vertebra.

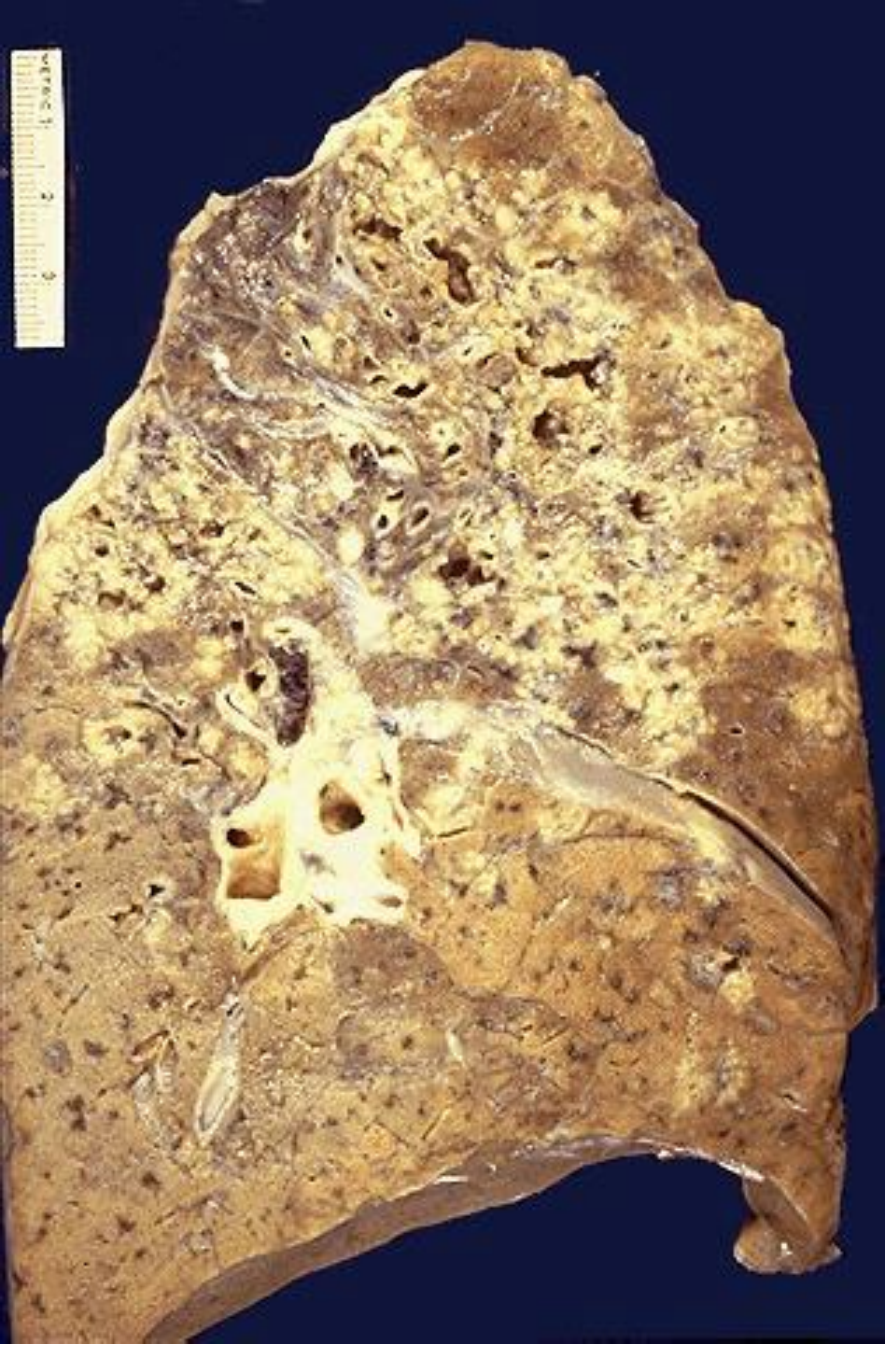


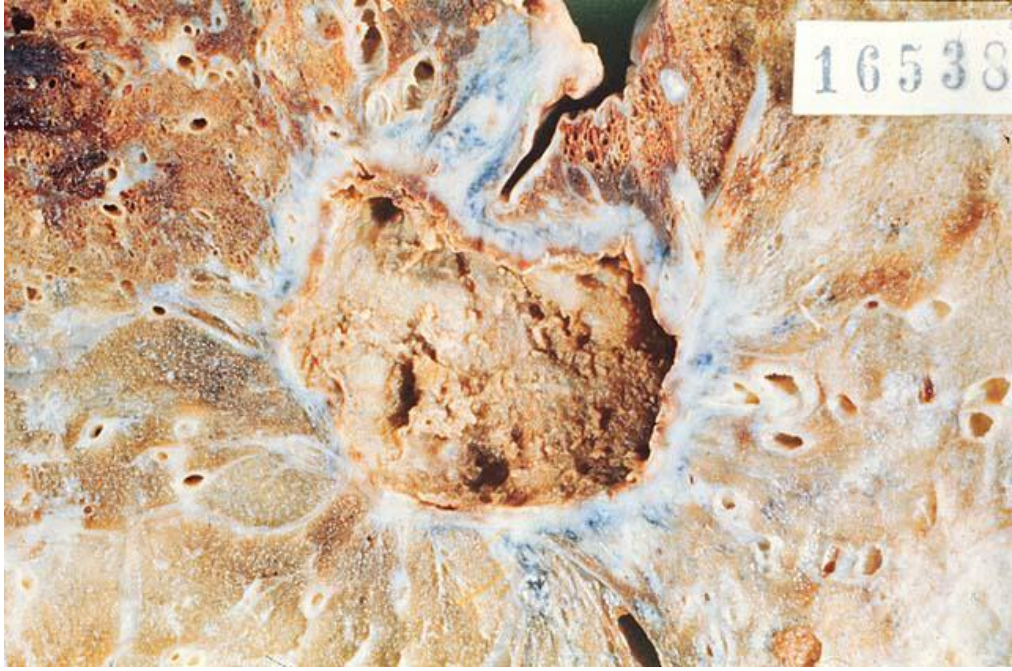
Encapsulated pulmonary tuberculoma.

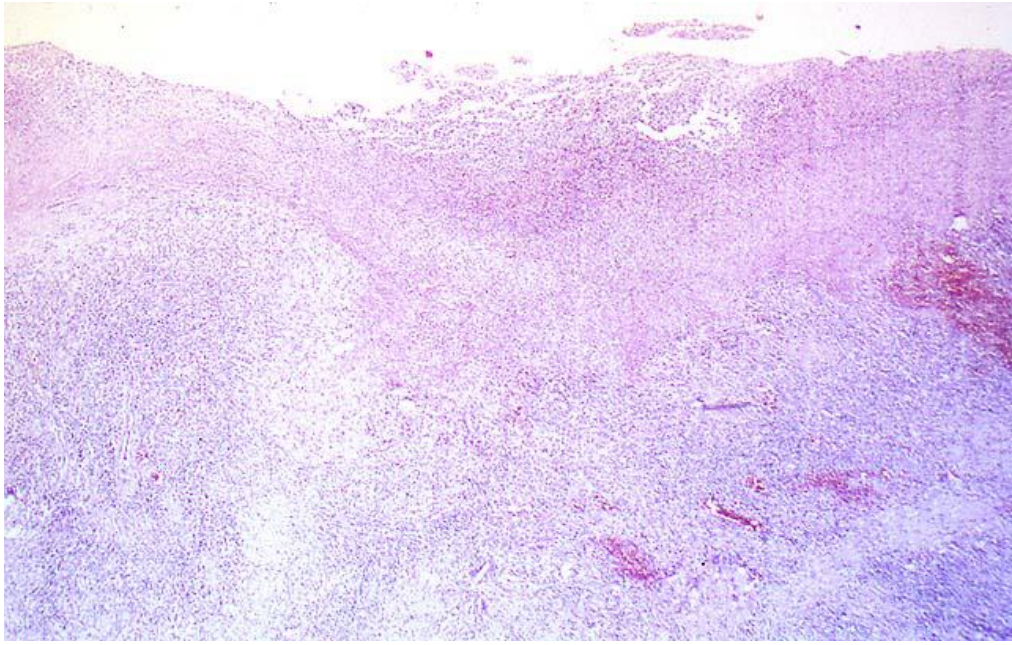




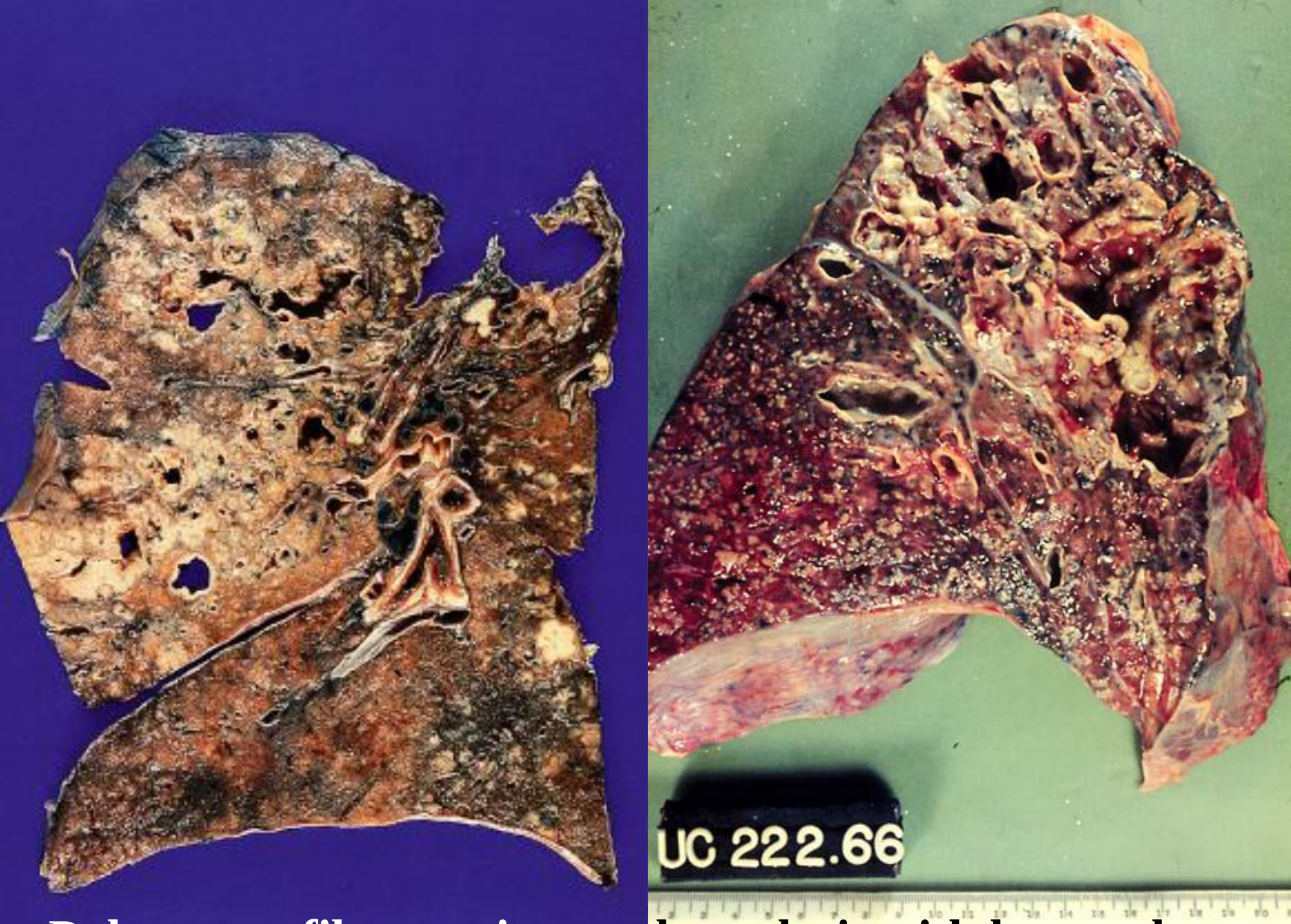
Destructive secondary tuberculosis.



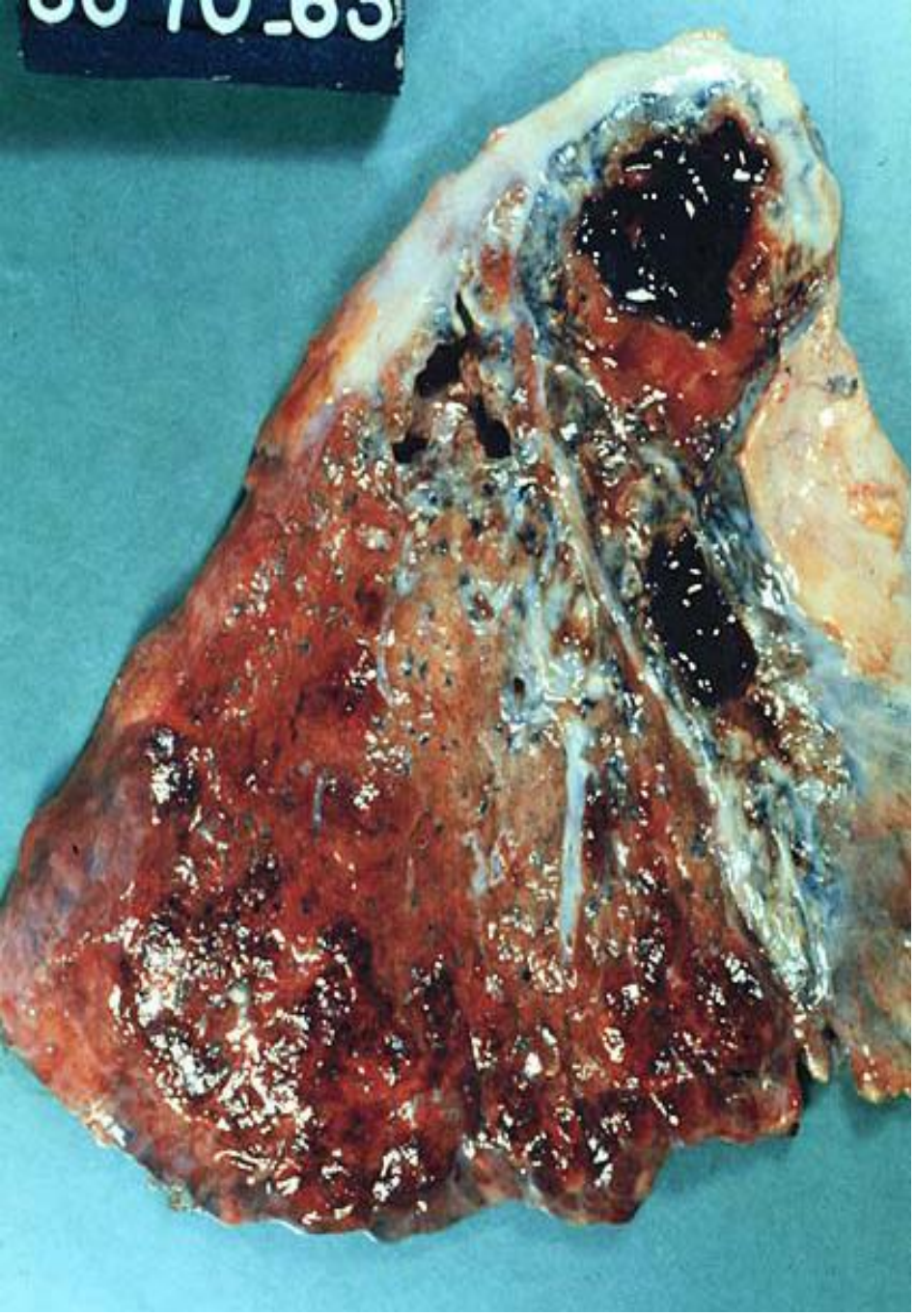


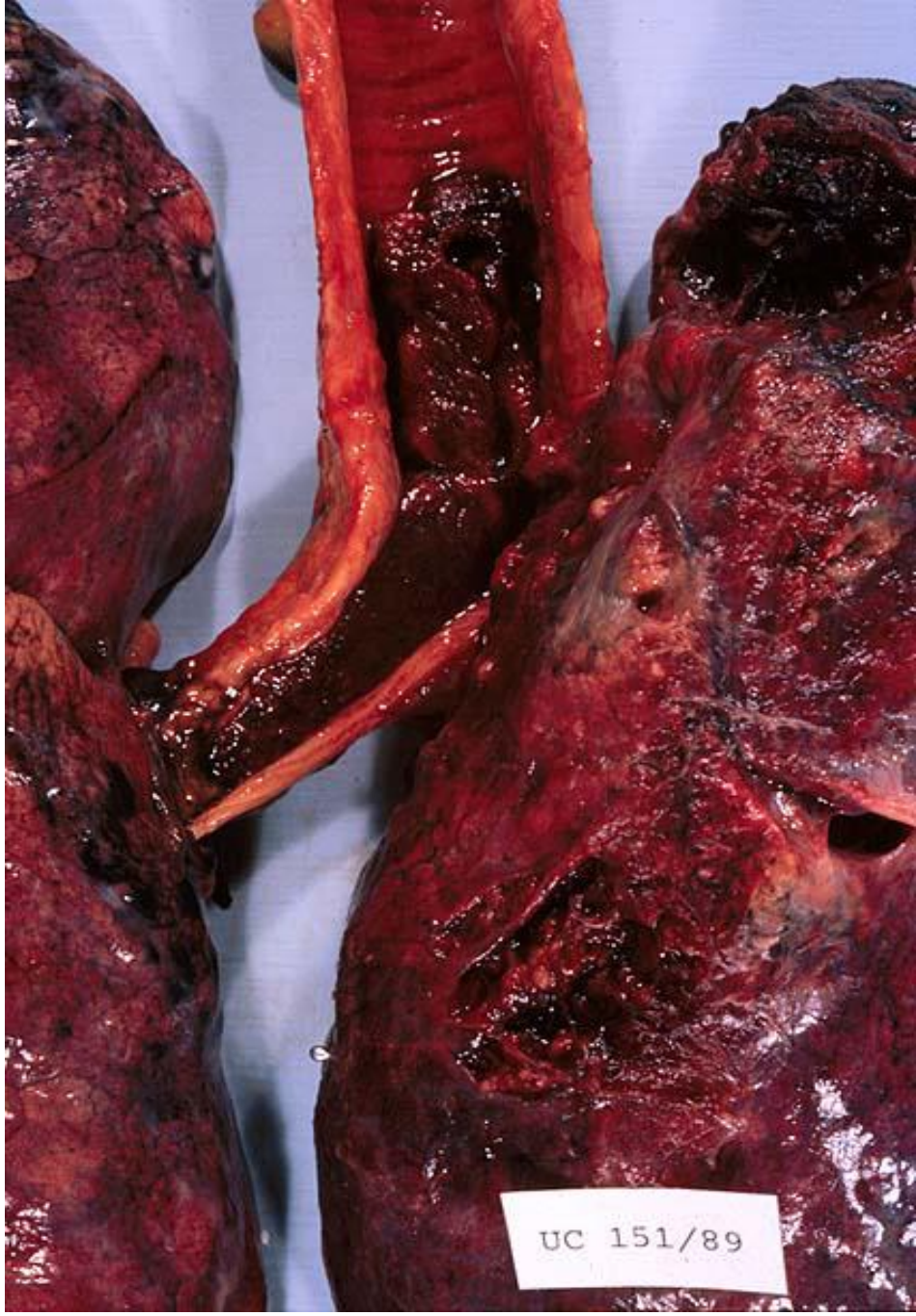


Secondary fibro-cavitary tuberculosis, cavity wall.

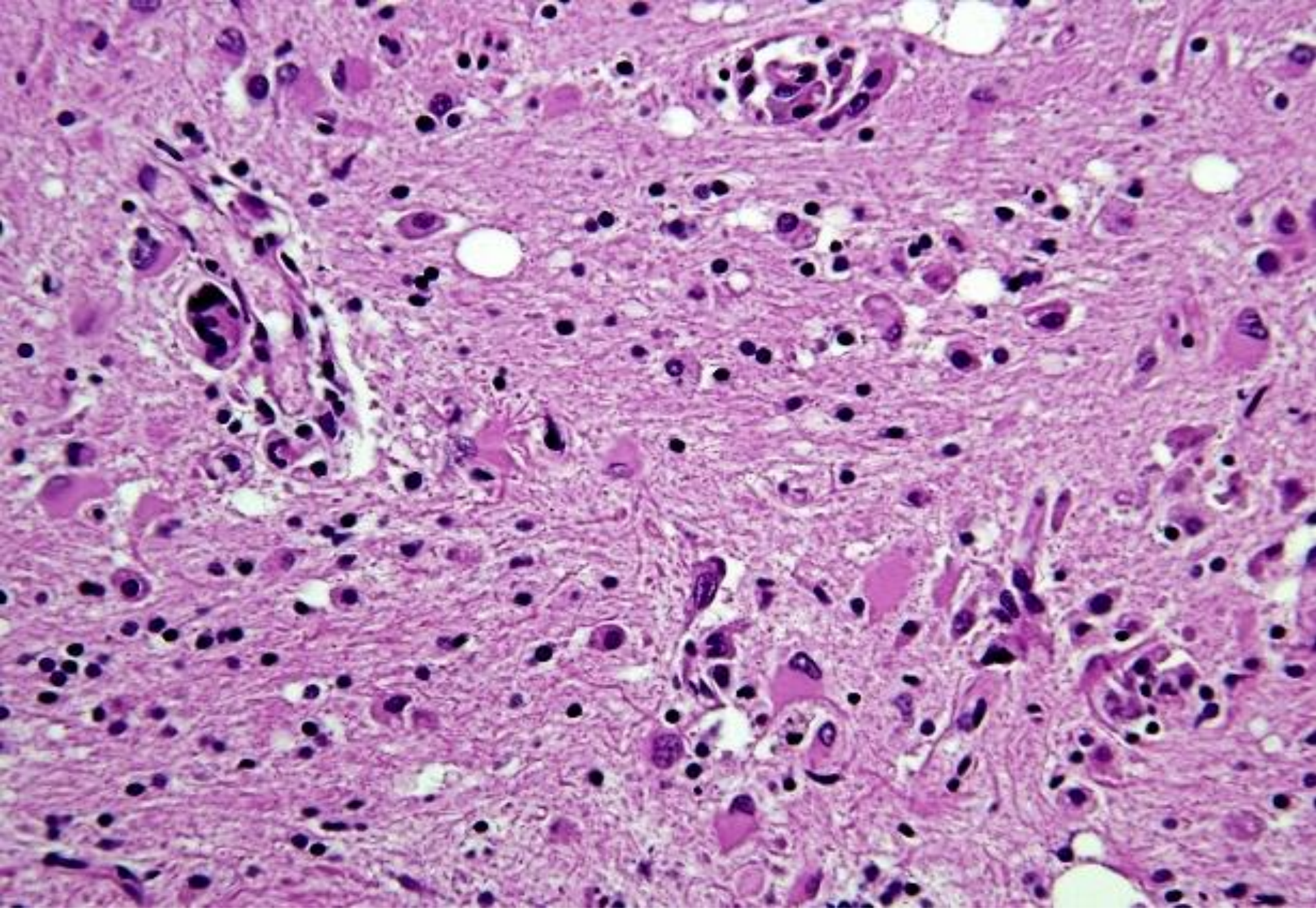


Pulmonary fibro-cavitary tuberculosis with hemorrhage.

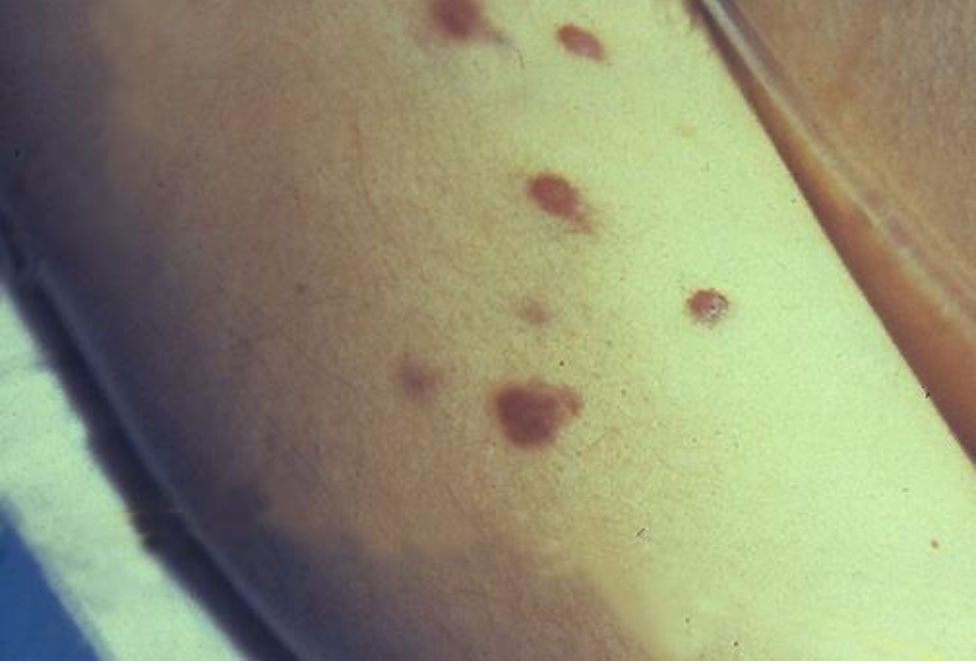


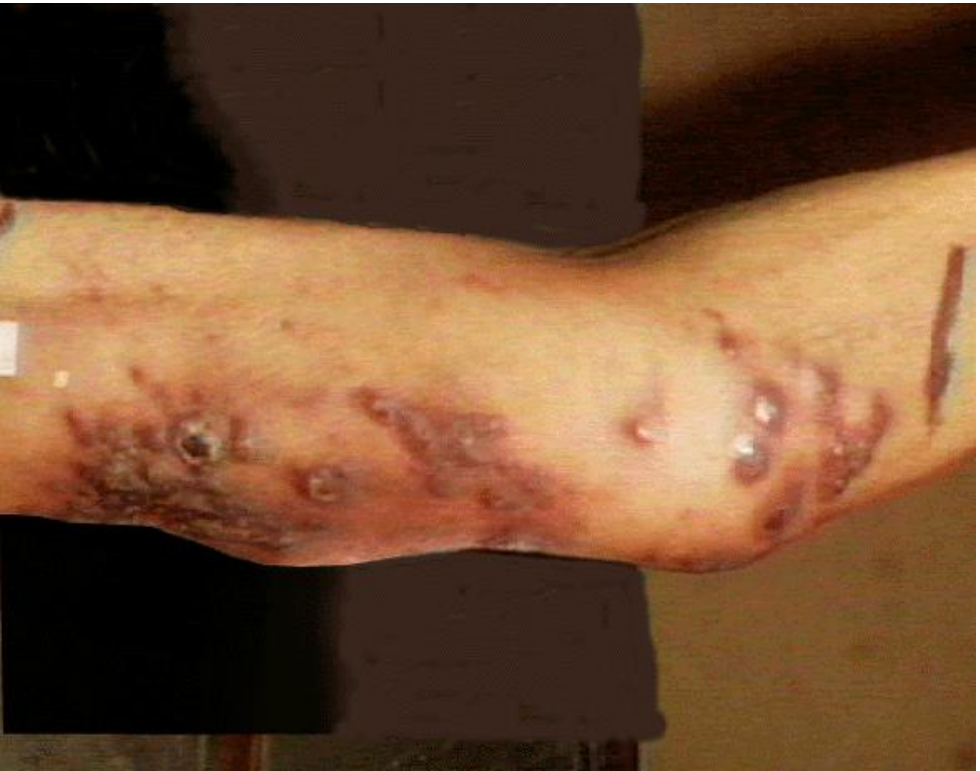


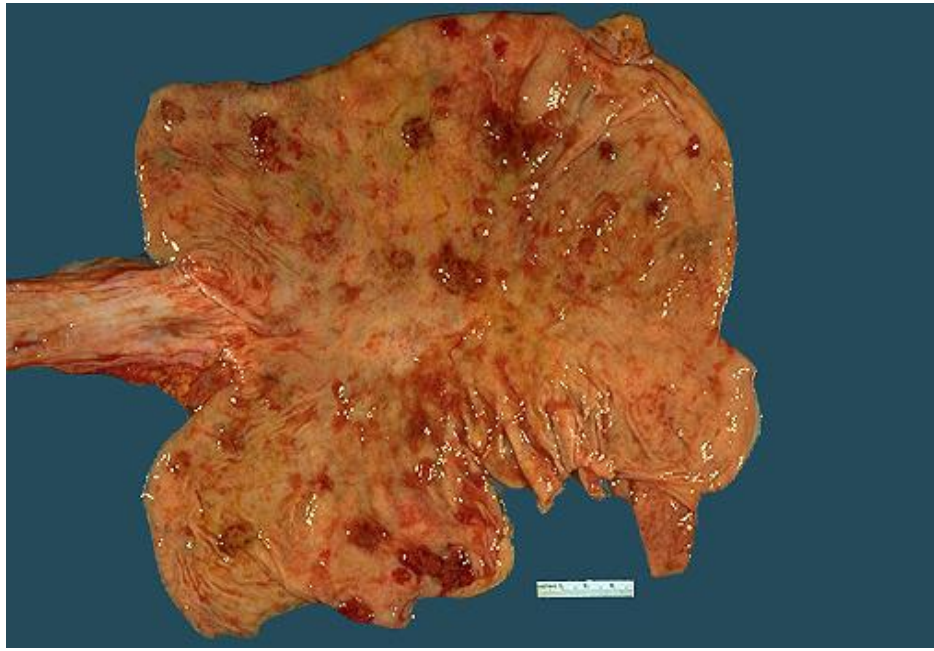
Secondary fibro-cavitary tuberculosis.



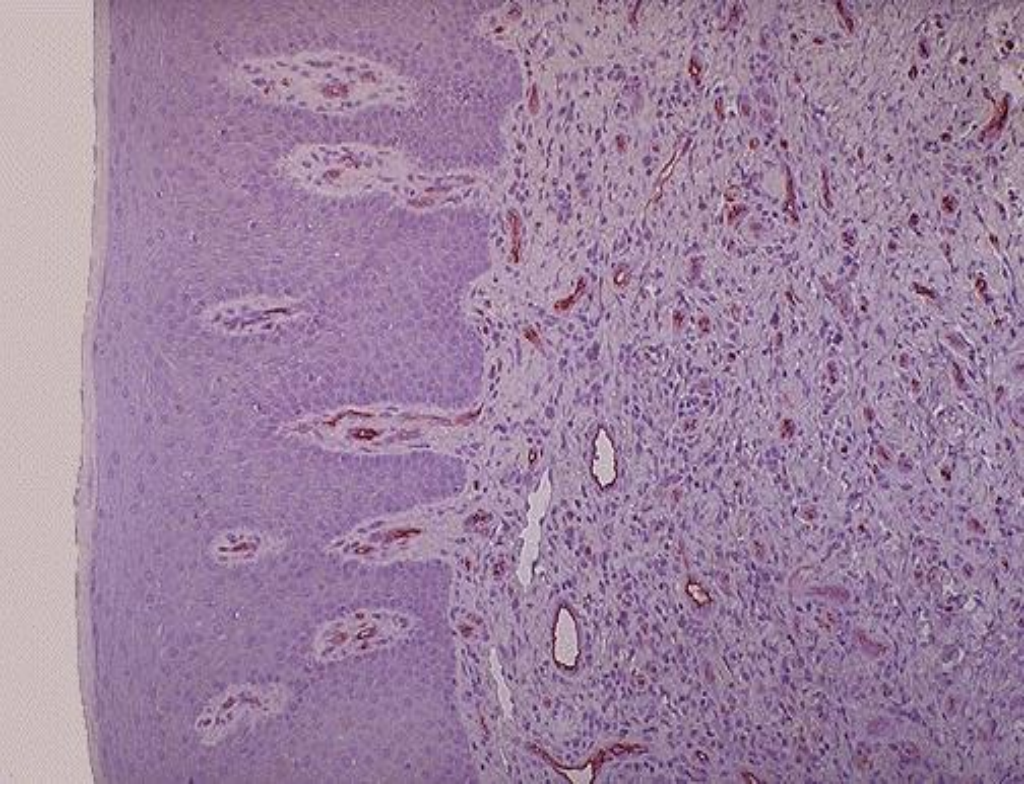
HIV encephalopathy with the presence of giant cells, resulting from fusion HIV-infected macrophages.

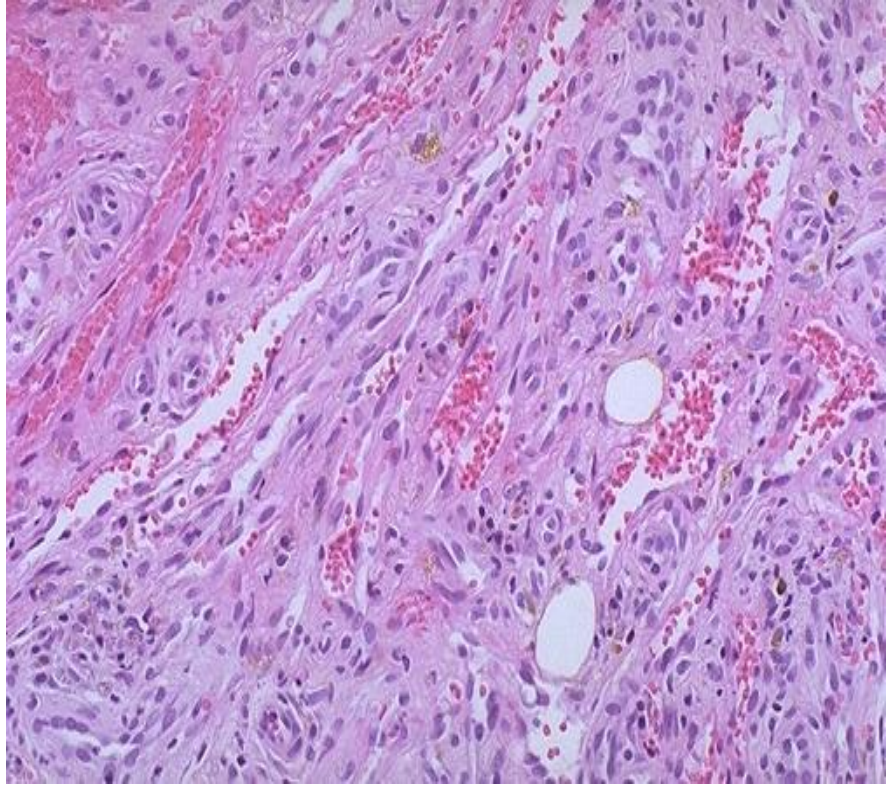


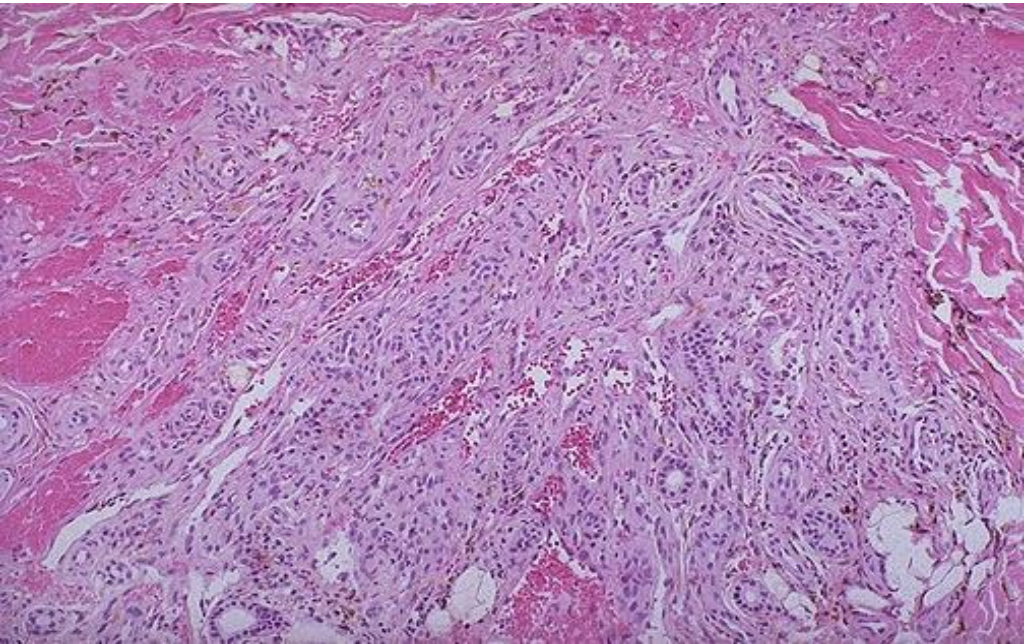




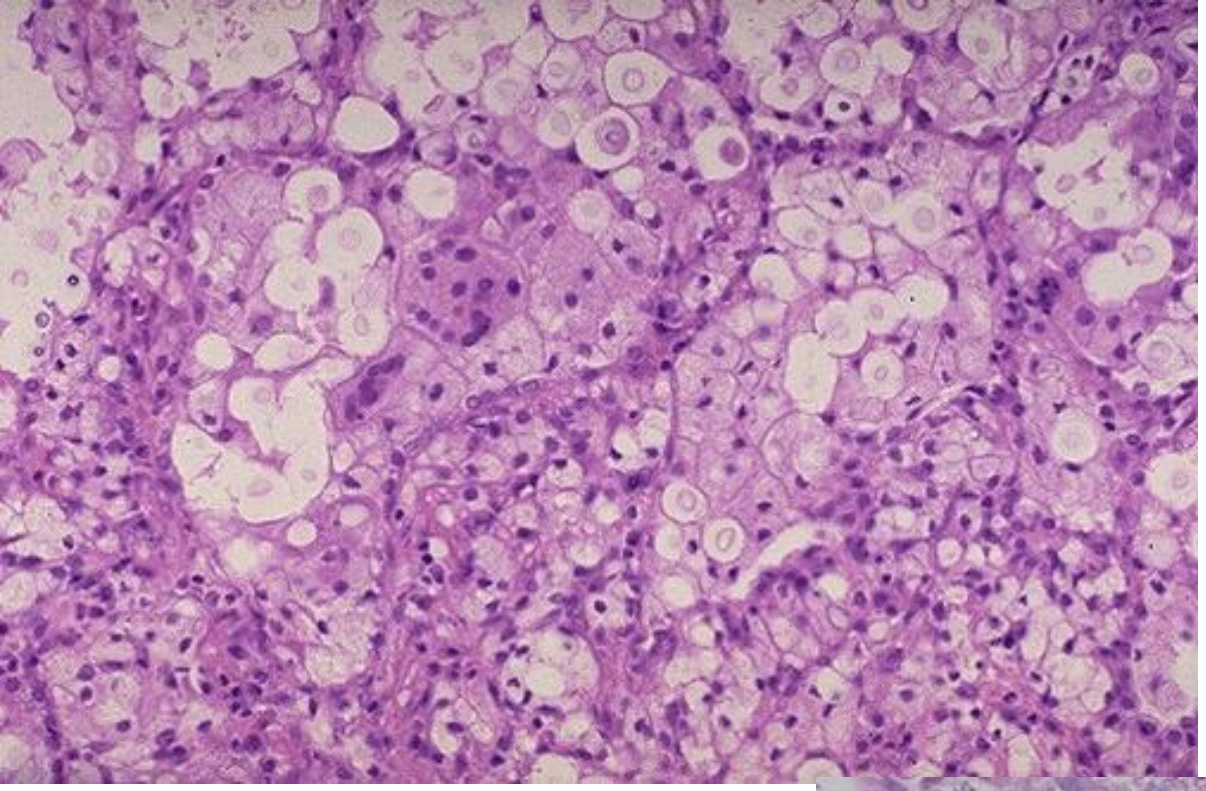
Kaposi's sarcoma (skin, liver, stomach).



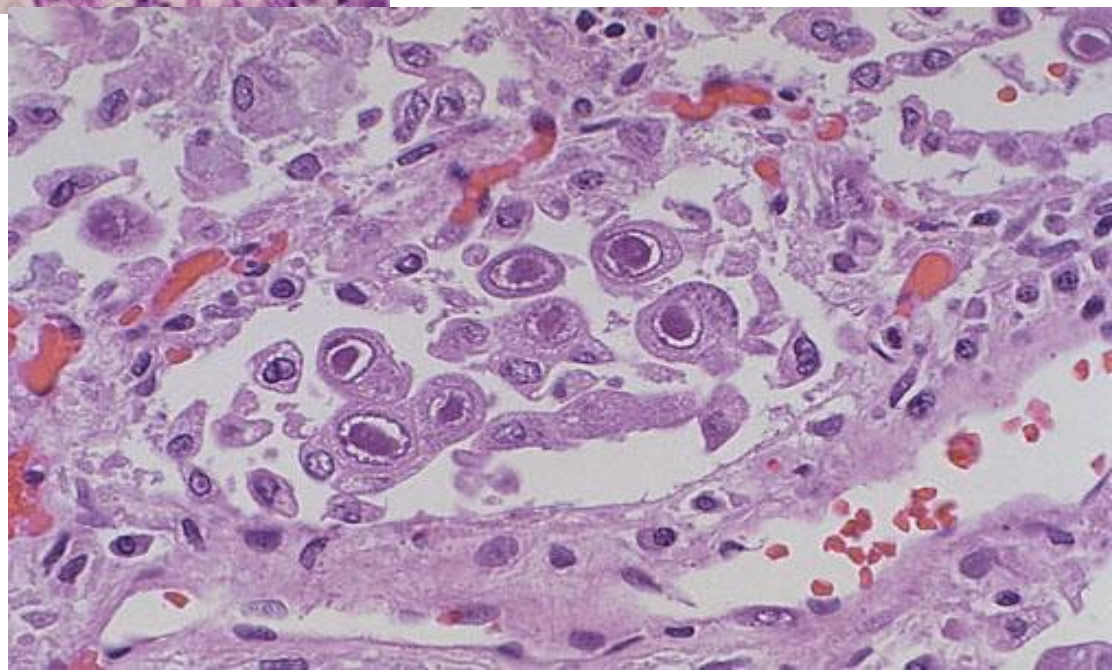




Kaposi's sarcoma
(vascular structures, hemorrhages, spindle-shaped stromal cells).



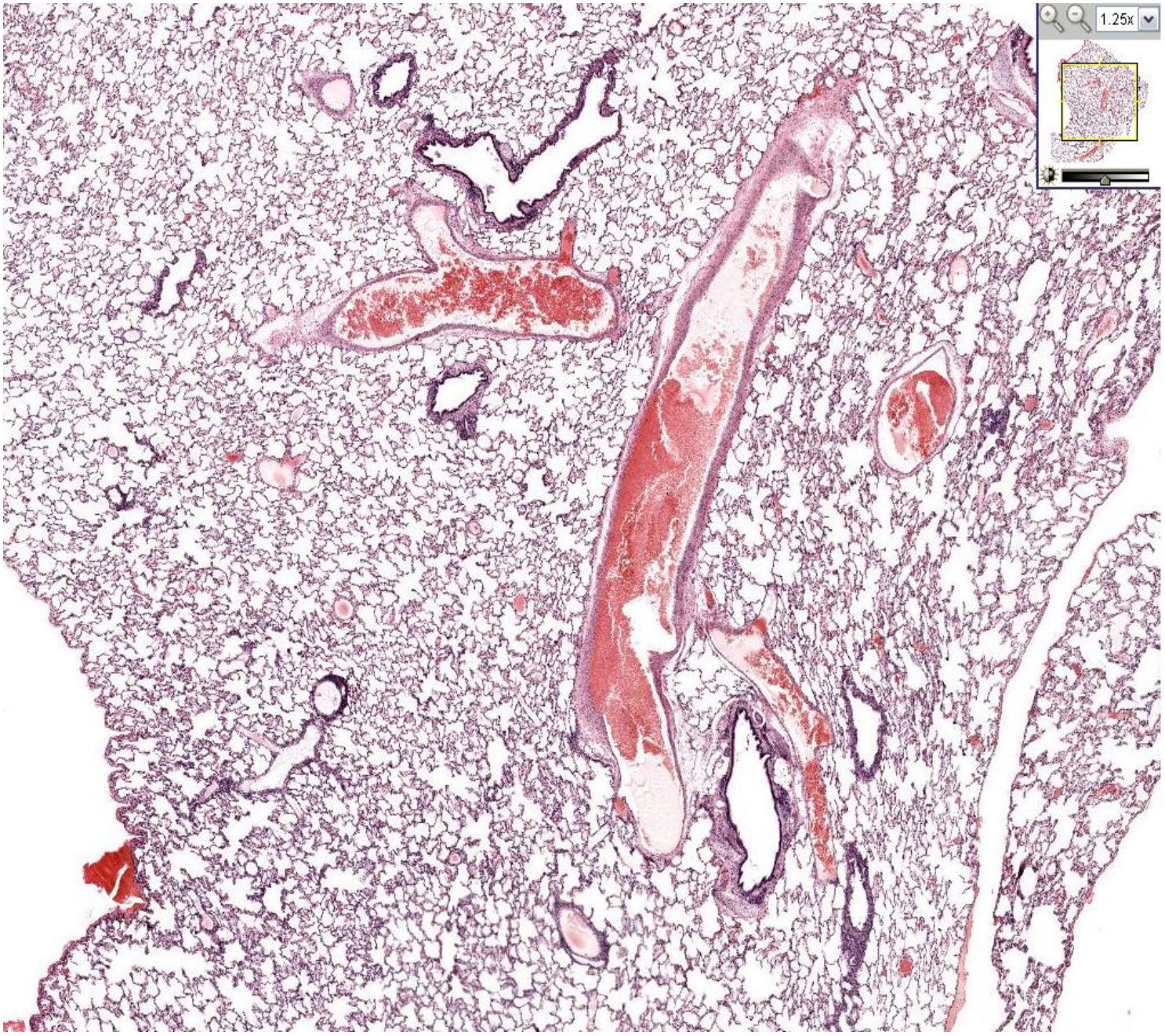
Pulmonary cytomegaloviral infection.



Pulmonary cryptococcosis.

Function of the Respiratory System

- Oversees gas exchanges (oxygen and carbon dioxide) between the blood and external environment
- Exchange of gasses takes place within the lungs in the alveoli(only site of gas exchange, other structures passageways
- Passageways to the lungs purify, warm, and humidify the incoming air
- Shares responsibility with cardiovascular system



Bronchi

Bronchioles

Terminal bronchioles

Alveolar ducts

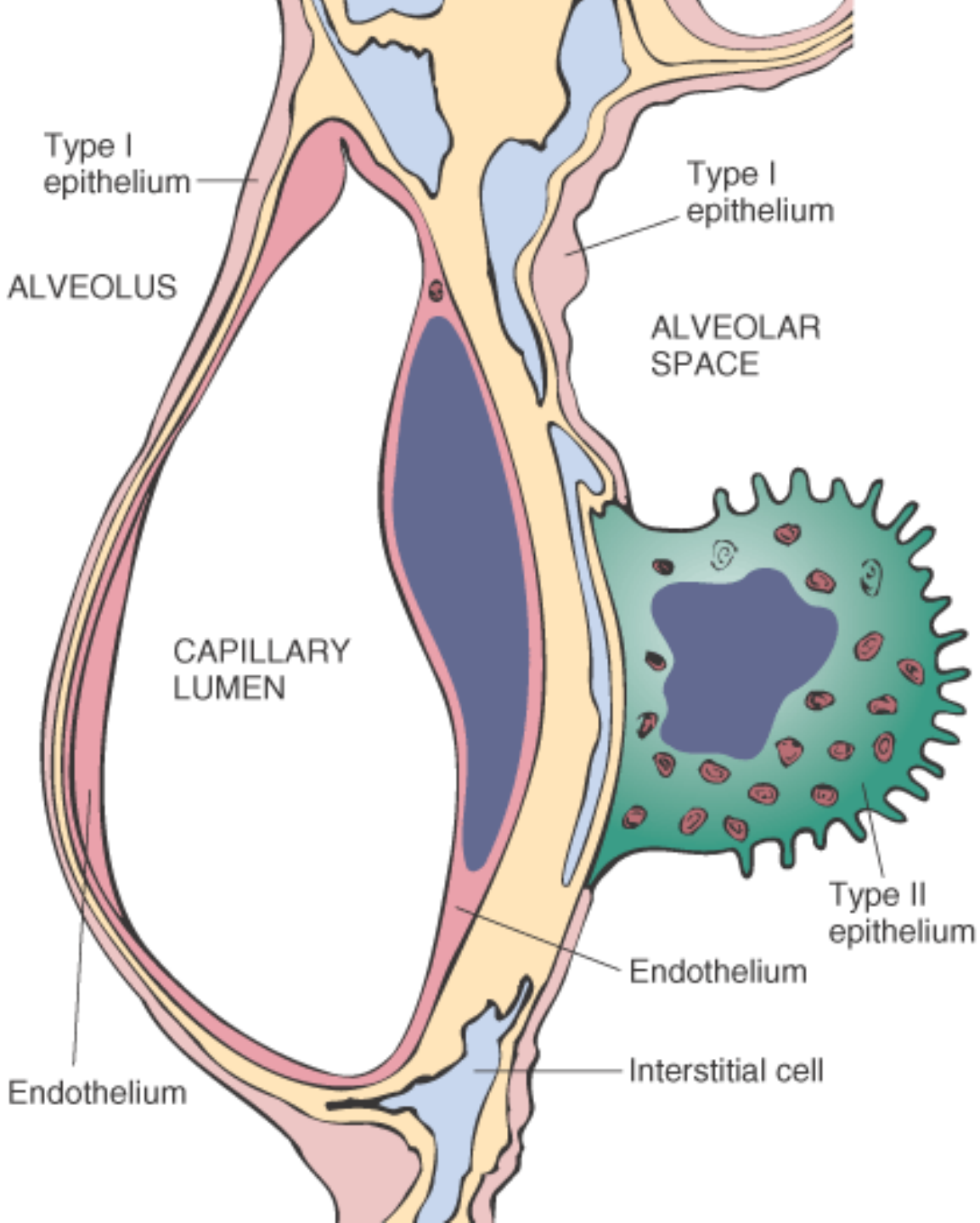
Alveoli

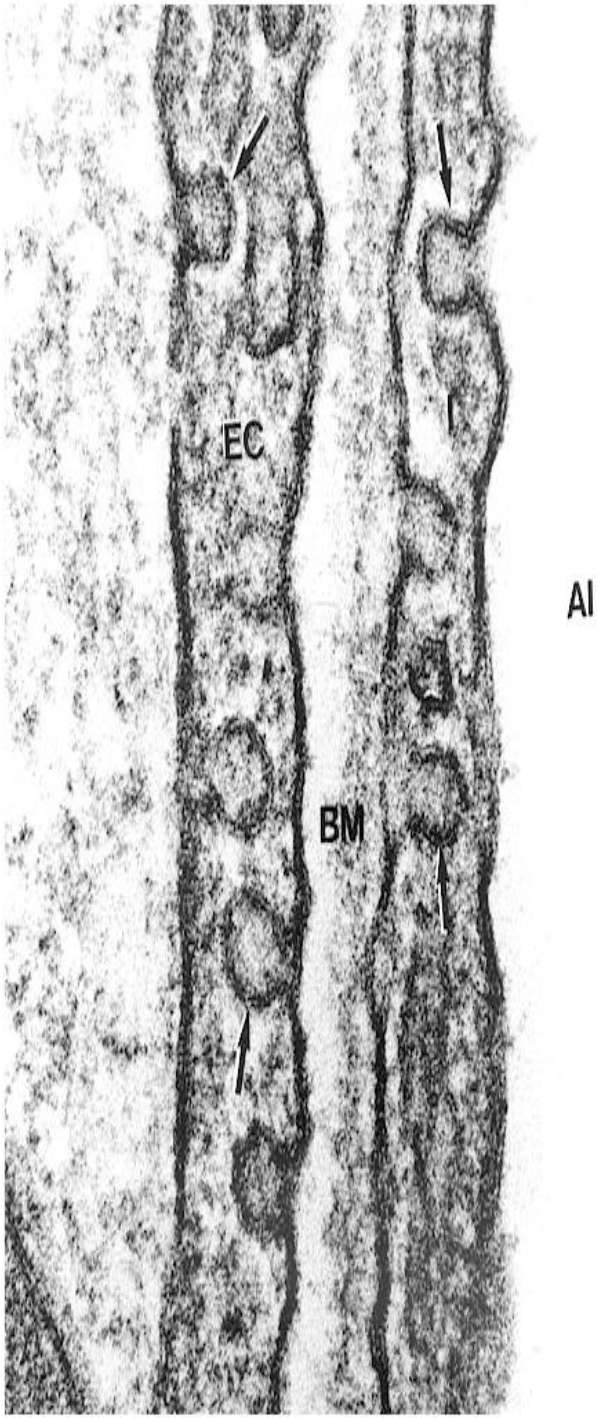
Type 1 pneumocytes

Type 2 pneumocytes

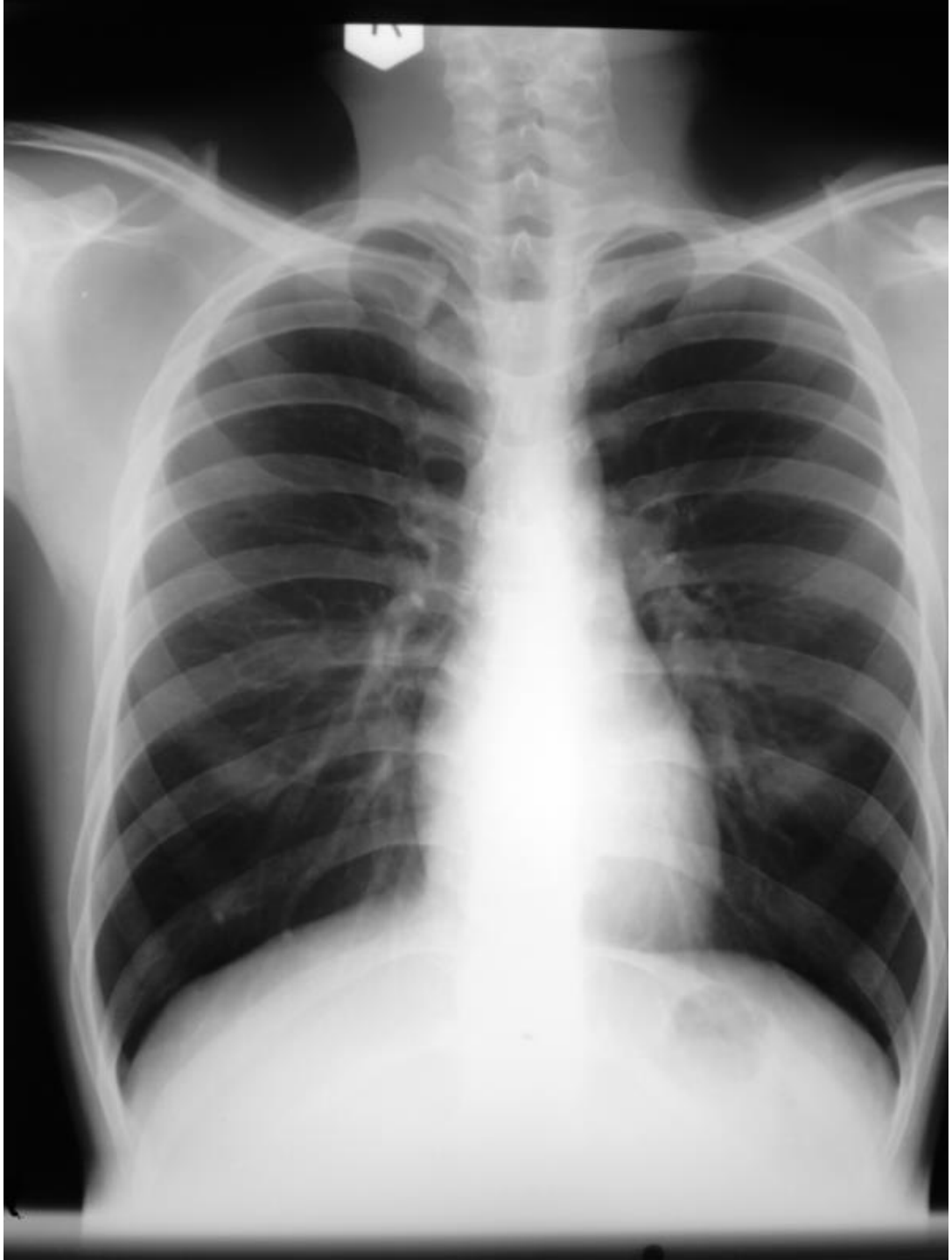
Macrophages

Capillaries


Type I epithelium
Type I epithelium
ALVEOLUS
ALVEOLAR SPACE
CAPILLARY LUMEN
Type II epithelium
Endothelium
Endothelium
Interstitial cell


EC
BM
Al

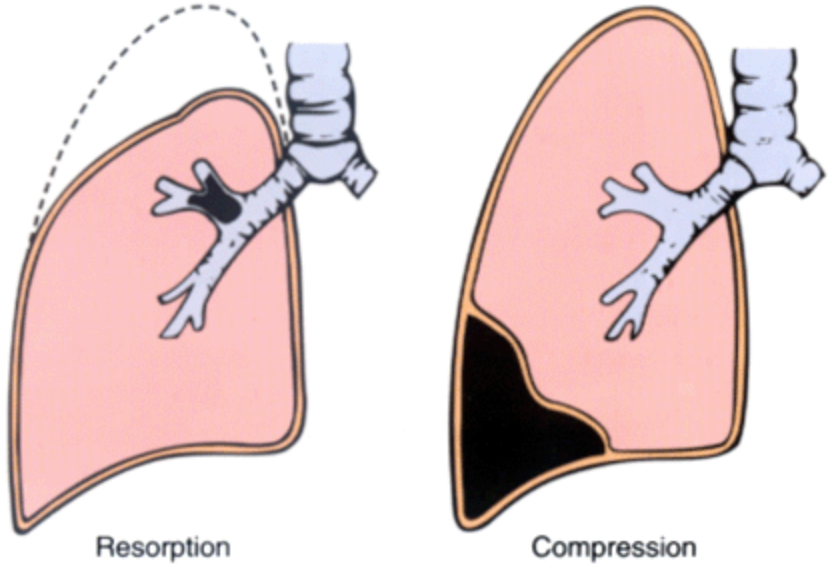
NORMAL

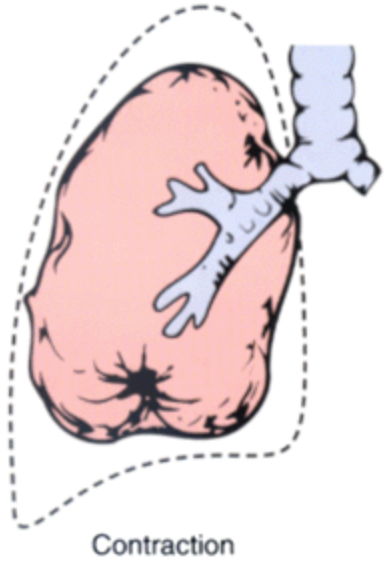


CXR

ATELECTASIS

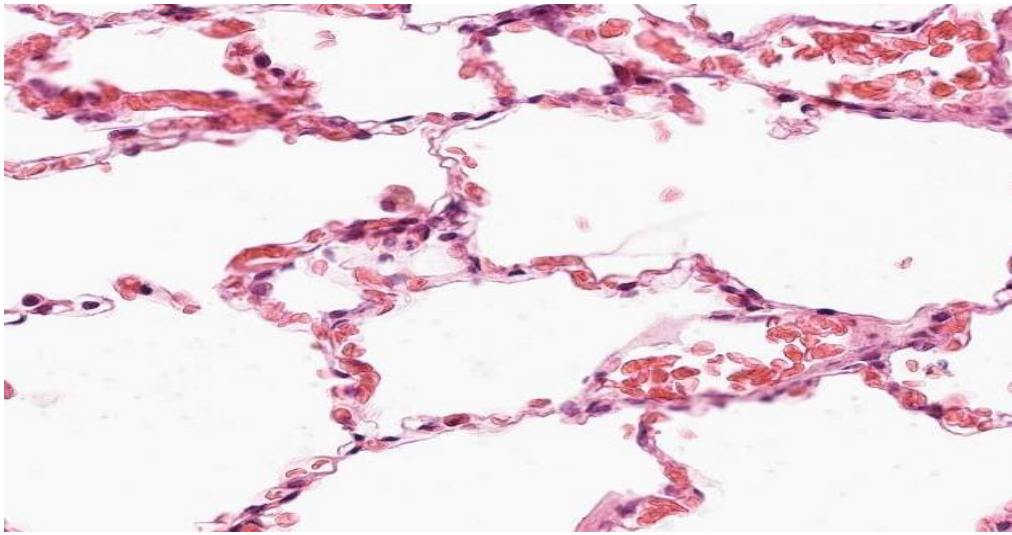
- **INCOMPLETE EXPANSION**
- **COLLAPSE**

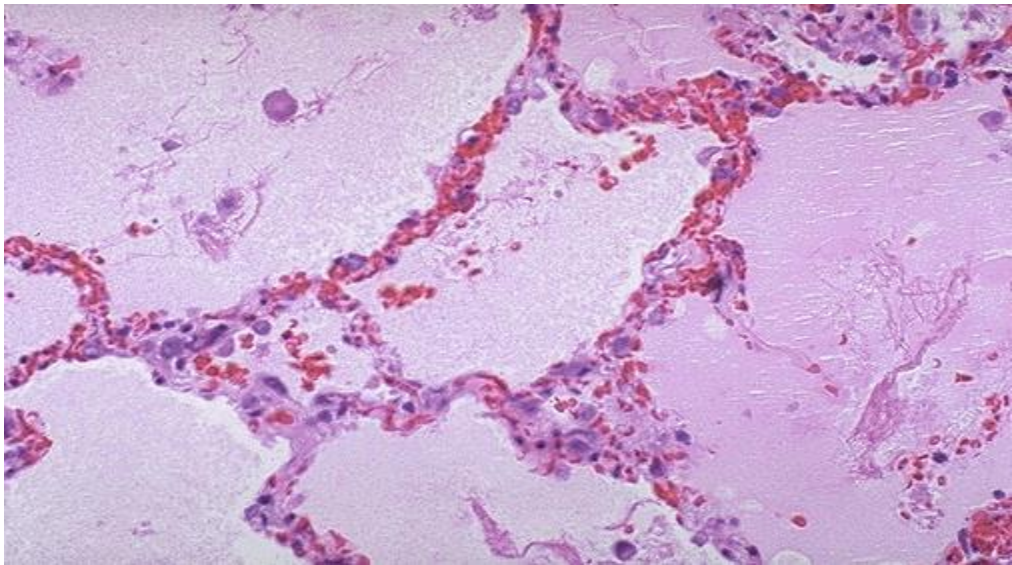


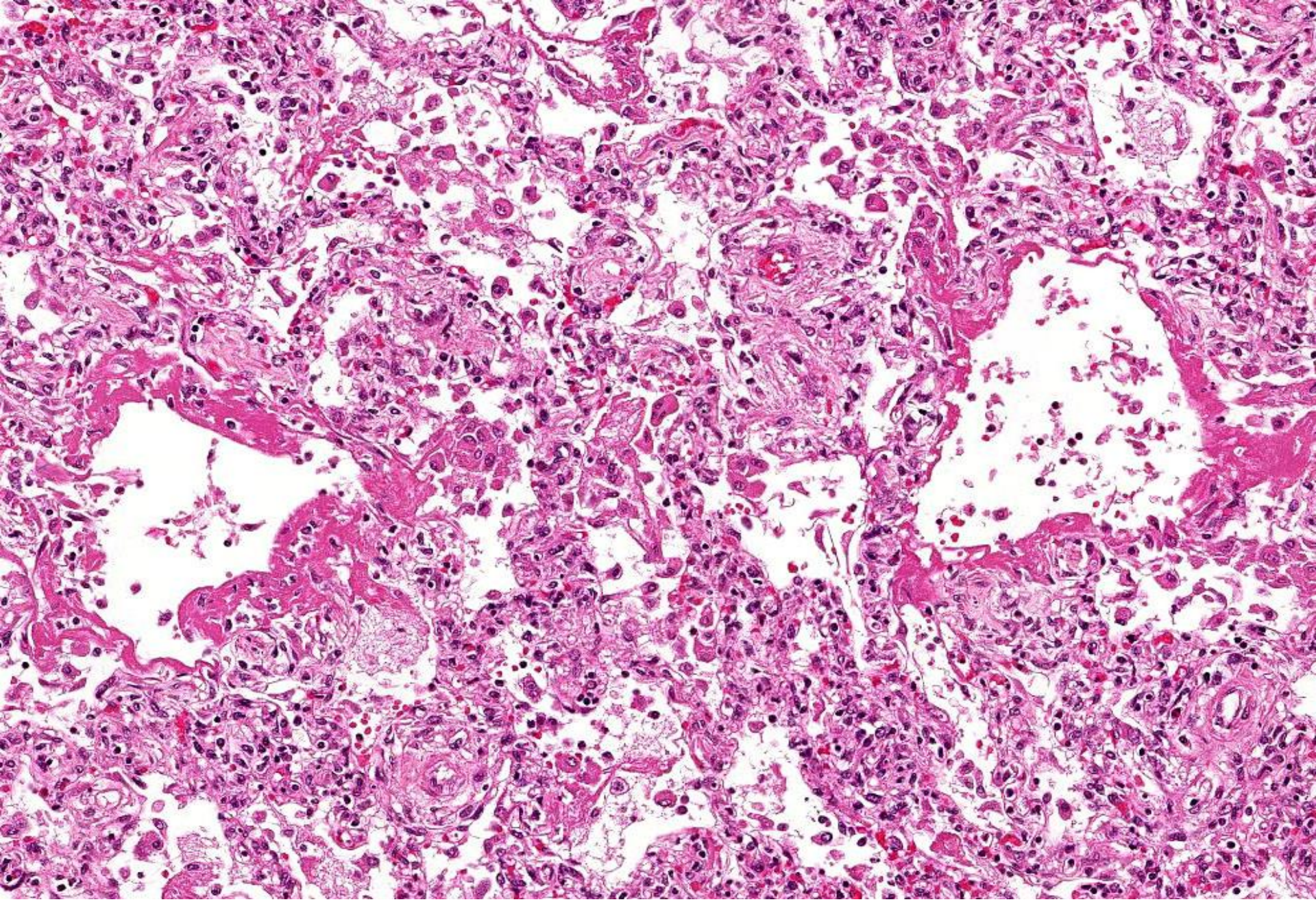


PULMONARY EDEMA

- **IN-creased venous pressure**
- **DE-creased oncotic pressure**
- **Lymphatic obstruction**
- **Alveolar injury**







ARDS

ACUTE INTERSTITIAL PNEUMONIA

- **Think of it as ARDS with NO known etiology!**

OBSTRUCTION v. RESTRICTION

- **OBSTRUCTION**

- **Air or blood?**
- **Large or small?**
- **Inspiration or Expiration?**

- **Obstruction is SMALL AIRWAY EXPIRATION obstruction, i.e., wheezing**
- **HYPEREXPANSION on CXR**

- **RESTRICTION**

"Compliance"
"Infiltrative"

REDUCED lung VOLUME, DYSPNEA, CYANOSIS
REDUCED GAS TRANSFER
"GROUND GLASS" on CXR

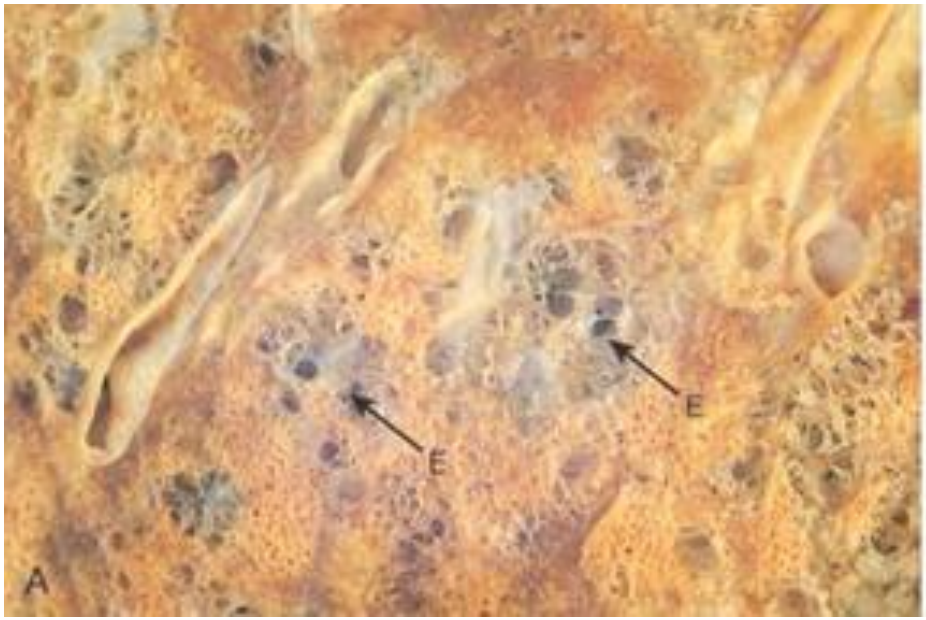
OBSTRUCTION (cOPD)

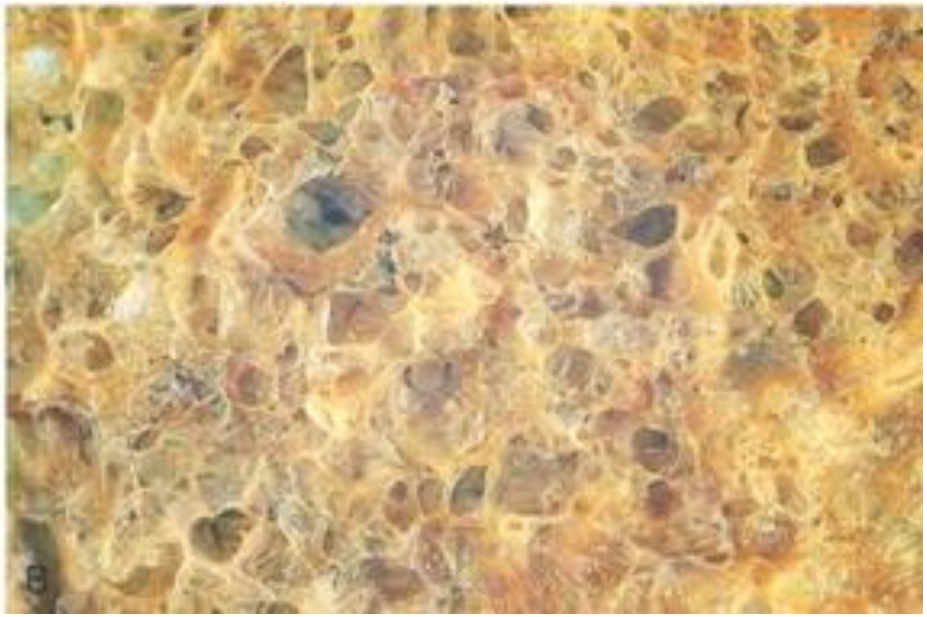
- **EMPHYSEMA (almost always chronic)**
- **CHRONIC BRONCHITIS→ emphysema**
- **ASTHMA**
- **BRONCHIECTASIS**

EMPHYSEMA

- **COPD, or "END-STAGE" lung disease**
- **Centri-acinar, Pan-acinar, Paraseptal, Irregular**
- **Like cirrhosis, thought of as END-STAGE of multiple chronic small airway obstructive etiologies**
- **NON-specific**
- **IN-creased crepitance, BULLAE (BLEBS)**
- **Clinically linked to recurrent pneumonias, and progressive failure**

EMPHYSEMA

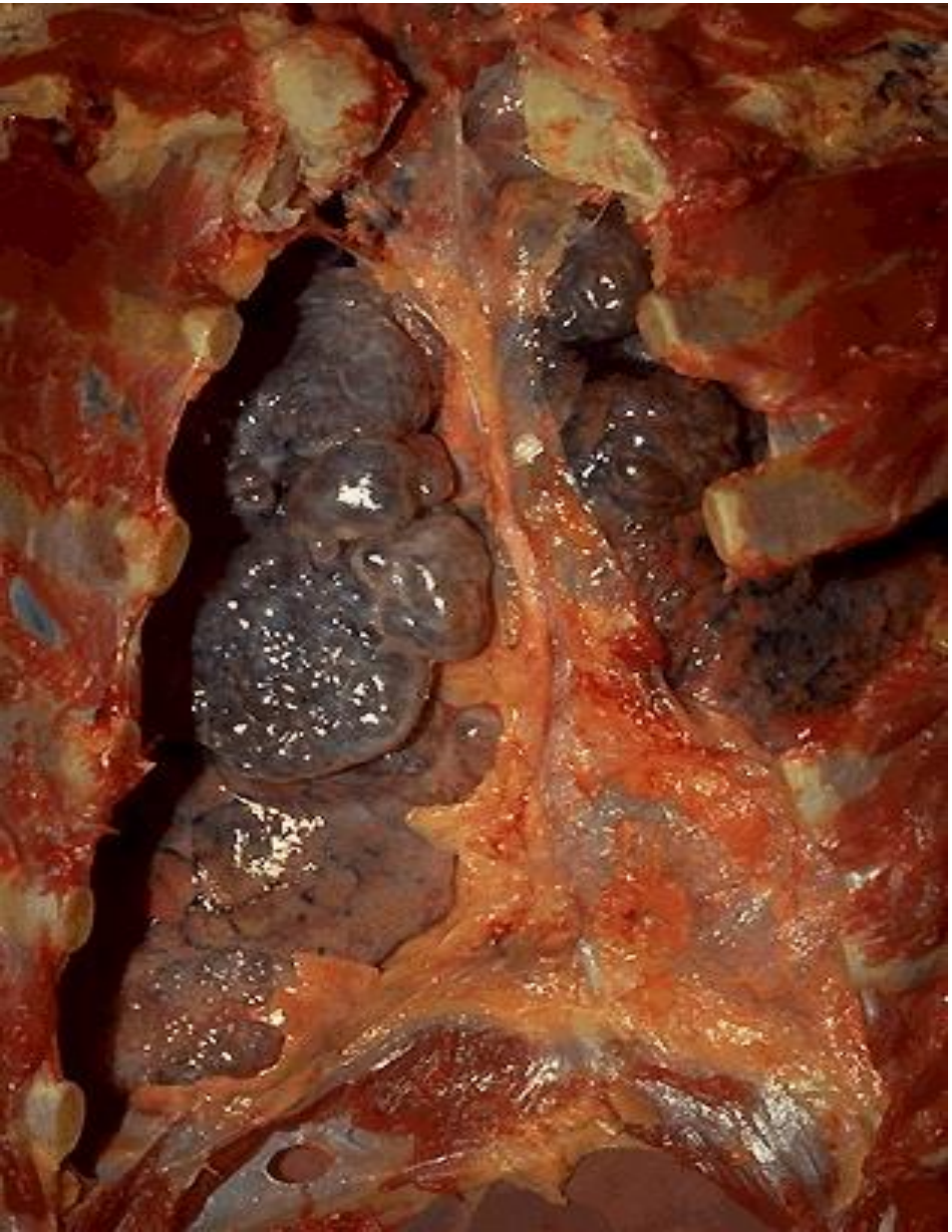


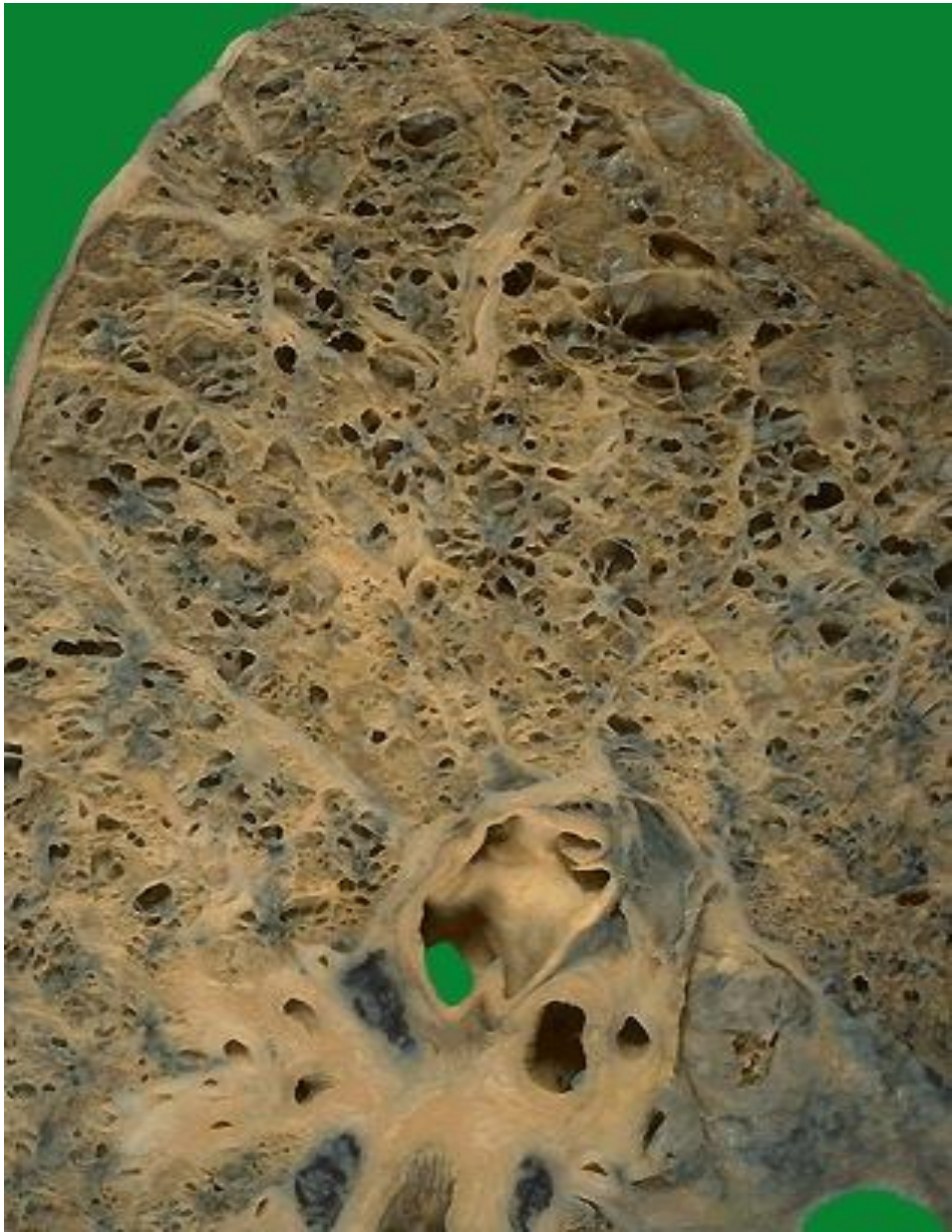


CENTRO-acinar

PAN-acinar

Bullae, or "peripheral blebs" are hallmarks of chronic obstructive lung disease, COPD.

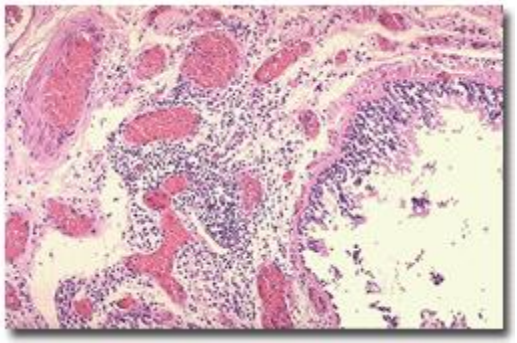




CHRONIC BRONCHITIS

- **INHALANTS, POLLUTION, CIGARETTES**
- **CHRONIC COUGH**
- **CAN OFTEN PROGRESS TO EMPHYSEMA**

- **MUCUS hypersecretion, early, i.e. goblet cell increase**
- **CHRONIC bronchial inflammatory infiltrate**



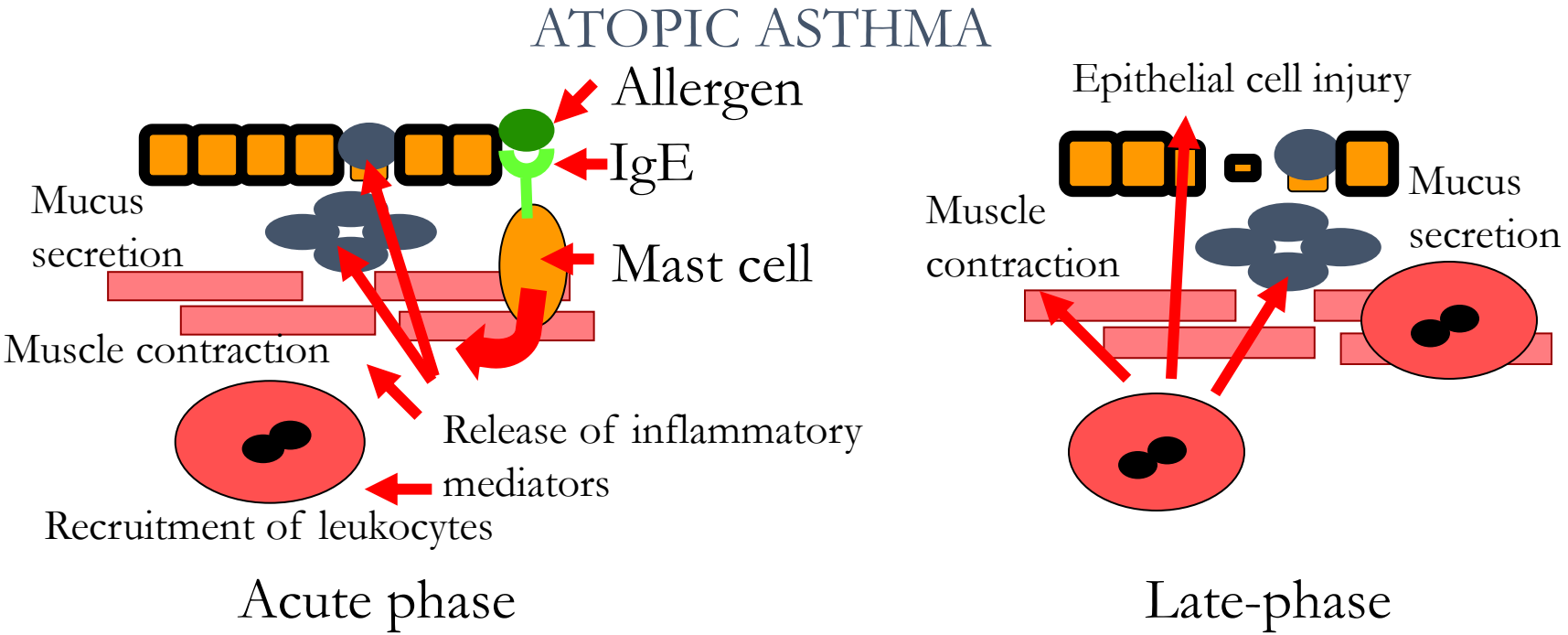
ASTHMA

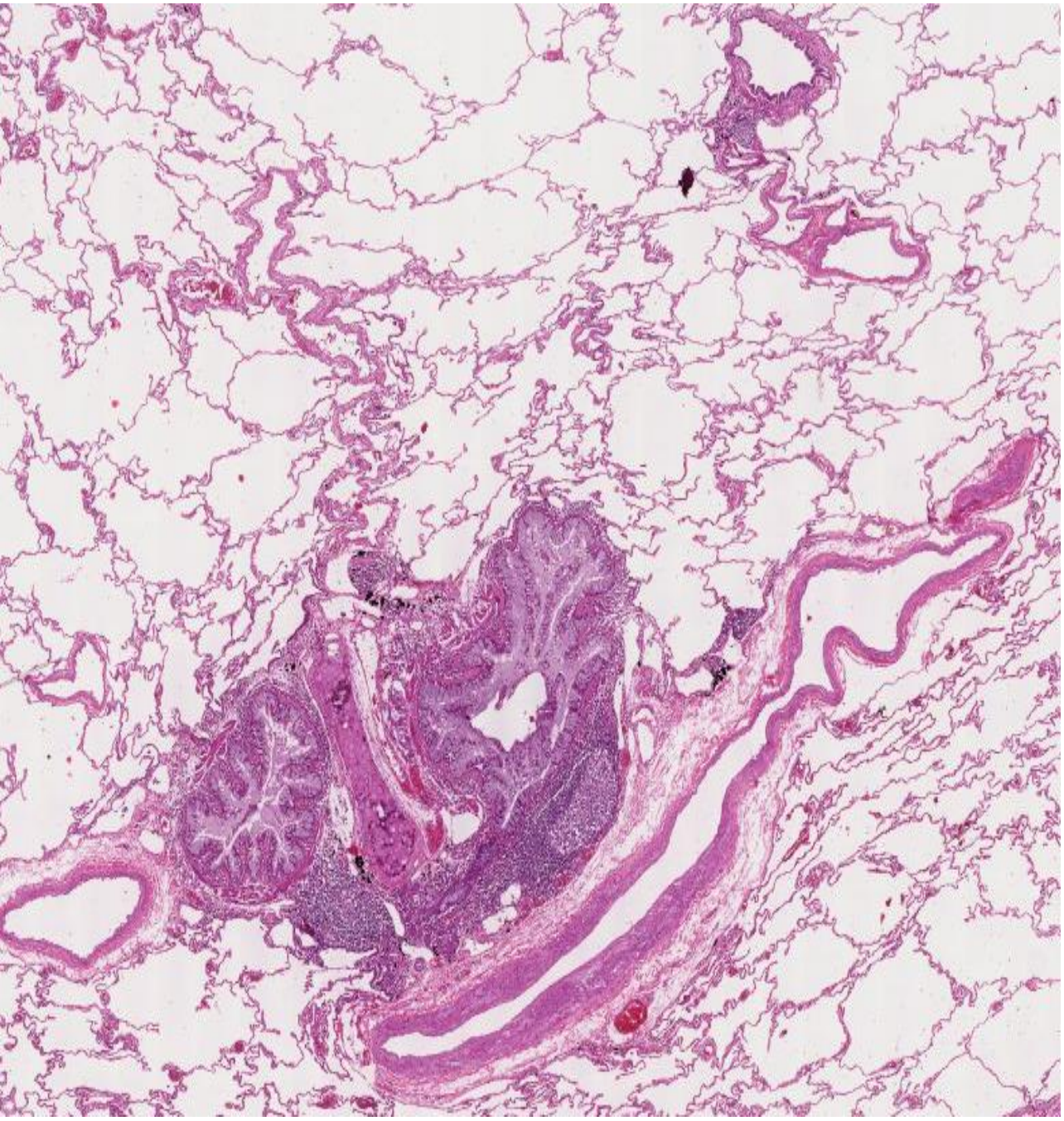
Similar to chronic bronchitis but:

- **Wheezing is hallmark (bronchospasm, i.e. "wheezing")**
- **STRONG allergic role, i.e., eosinophils, IgE, allergens**
- **Often starting in CHILDHOOD**
- **ATOPIC (allergic) or NON-ATOPIC (infection)**
- **Chronic small airway obstruction and infection**
- **1) Mucus hypersecretion with plugging, 2) lymphocytes/eosinophils, 3) lumen narrowing, 4) smooth muscle hypertrophy**

Bronchial Asthma

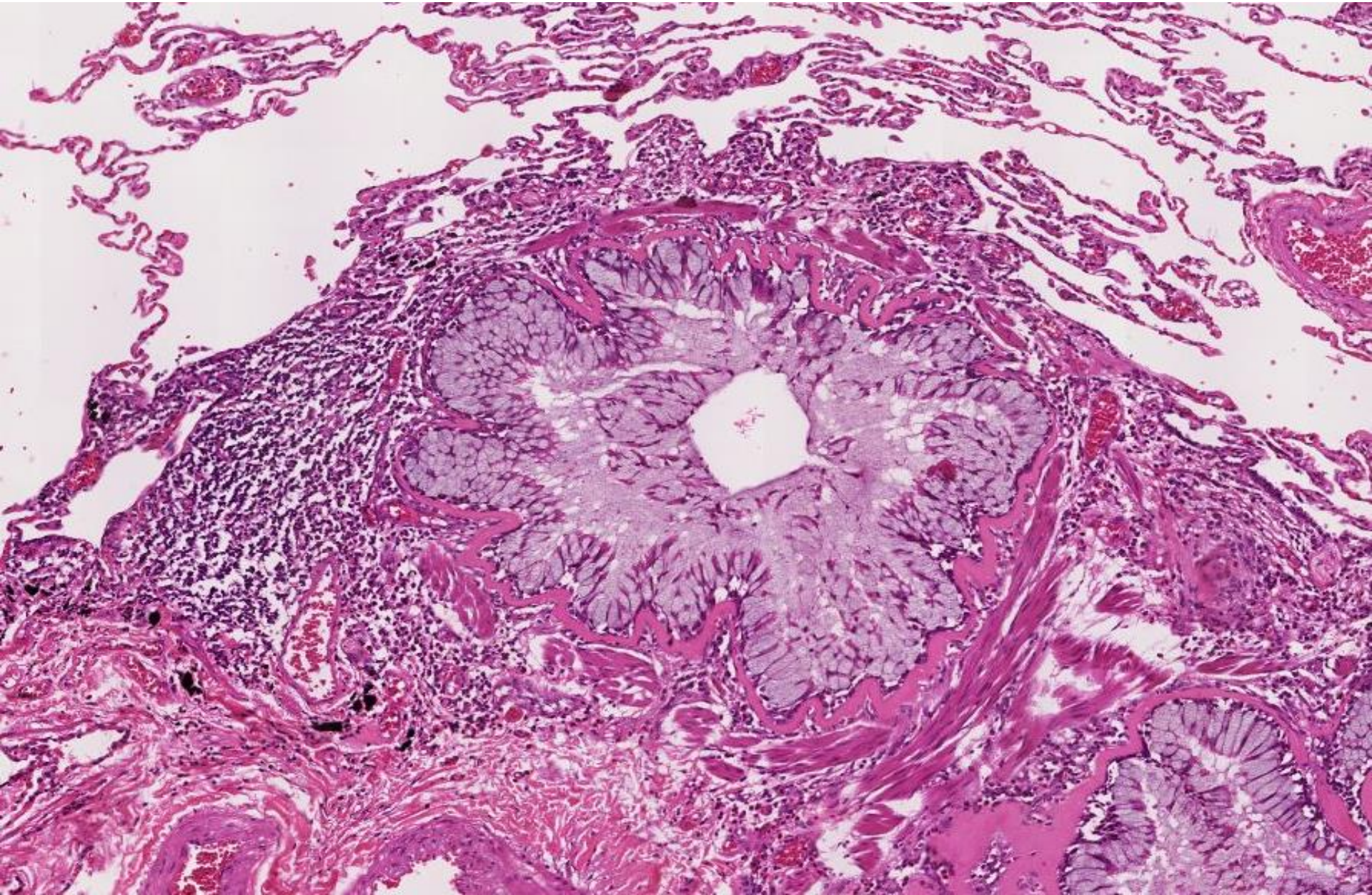
- **Chronic inflammatory disorder of the airways resulting in contraction of bronchial muscle**
- **Types**
  - **Extrinsic (atopic, allergic).**
    - **Allergens: food, pollen, dust, etc.**
  - **Intrinsic (non-atopic)**
    - **Initiated by infections, drugs, pollutants, chemical irritants**





Note the heavy inflammatory cell infiltrate around bronchioles and small bronchi.

What are the 4 classical histologic findings in bronchial asthma?

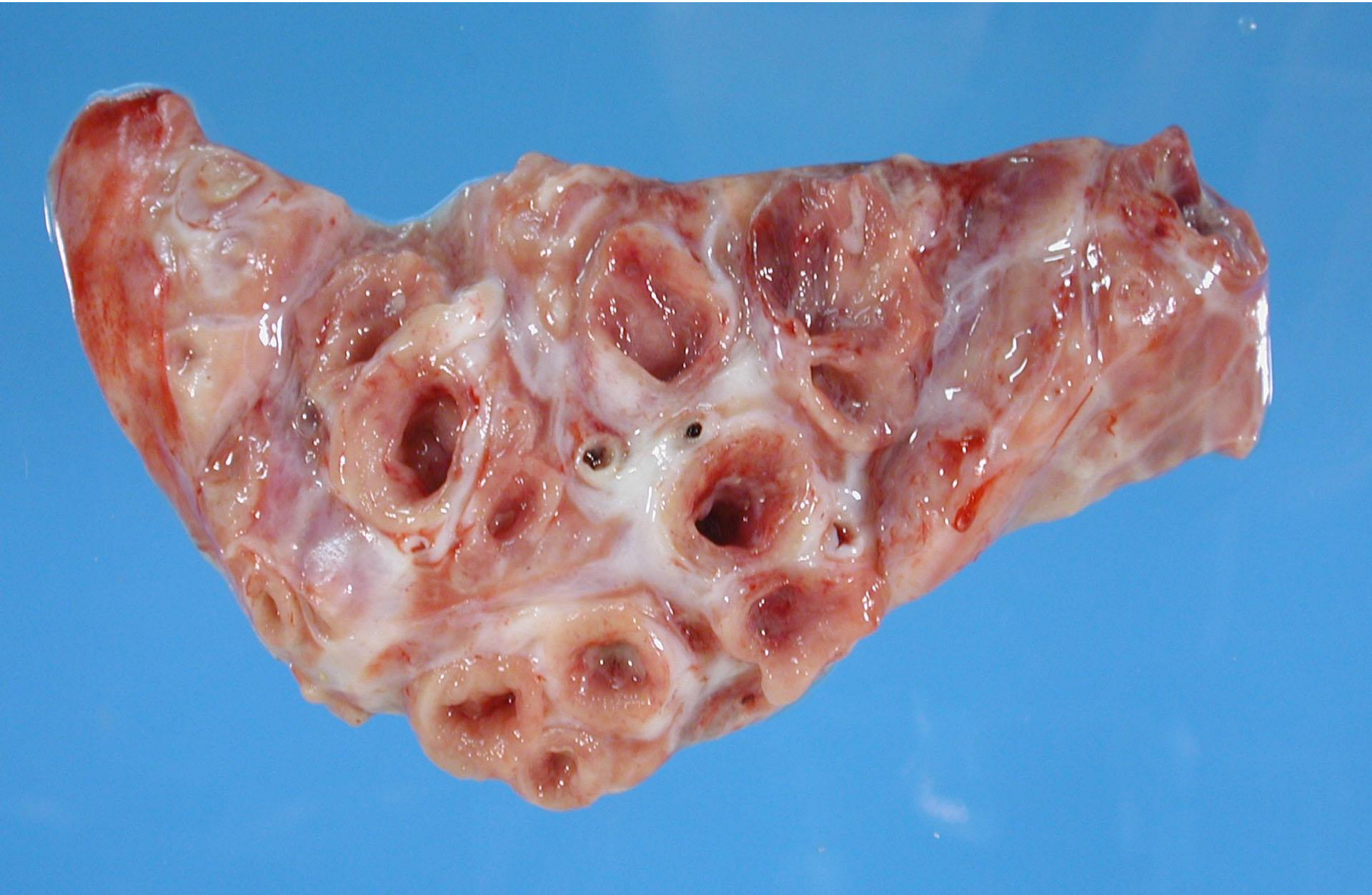


BRONCHIECTASIS

DILATATION of the BRONCHUS, associated with, often, necrotizing inflammation

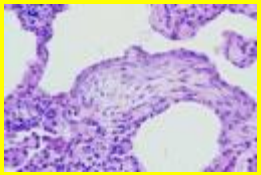
- **CONGENITAL**
- **TB, other bacteria, many viruses**
- **BRONCHIAL OBSTRUCTION (i.e., LARGE AIRWAY, NOT SMALL AIRWAY)**
- **Rheumatoid Arthritis, SLE, IBD (Inflammatory Bowel Disease)**

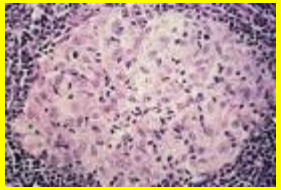
BRONCHIECTASIS



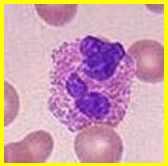
RESTRICTIVE (INFILTRATIVE)

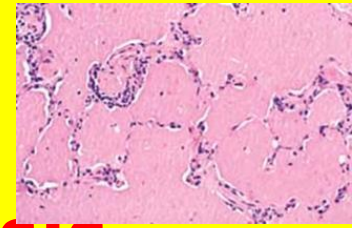
- **REDUCED COMPLIANCE, reduced gas exchange)**
- **Are also DIFFUSE**
- **HETEROGENEOUS**





- **FIBROSING**
- **GRANULOMATOUS**
- **EOSINOPHILIC**
- **SMOKING RELATED**
- **PAP (Pulmonary Alveolar Proteinosis**



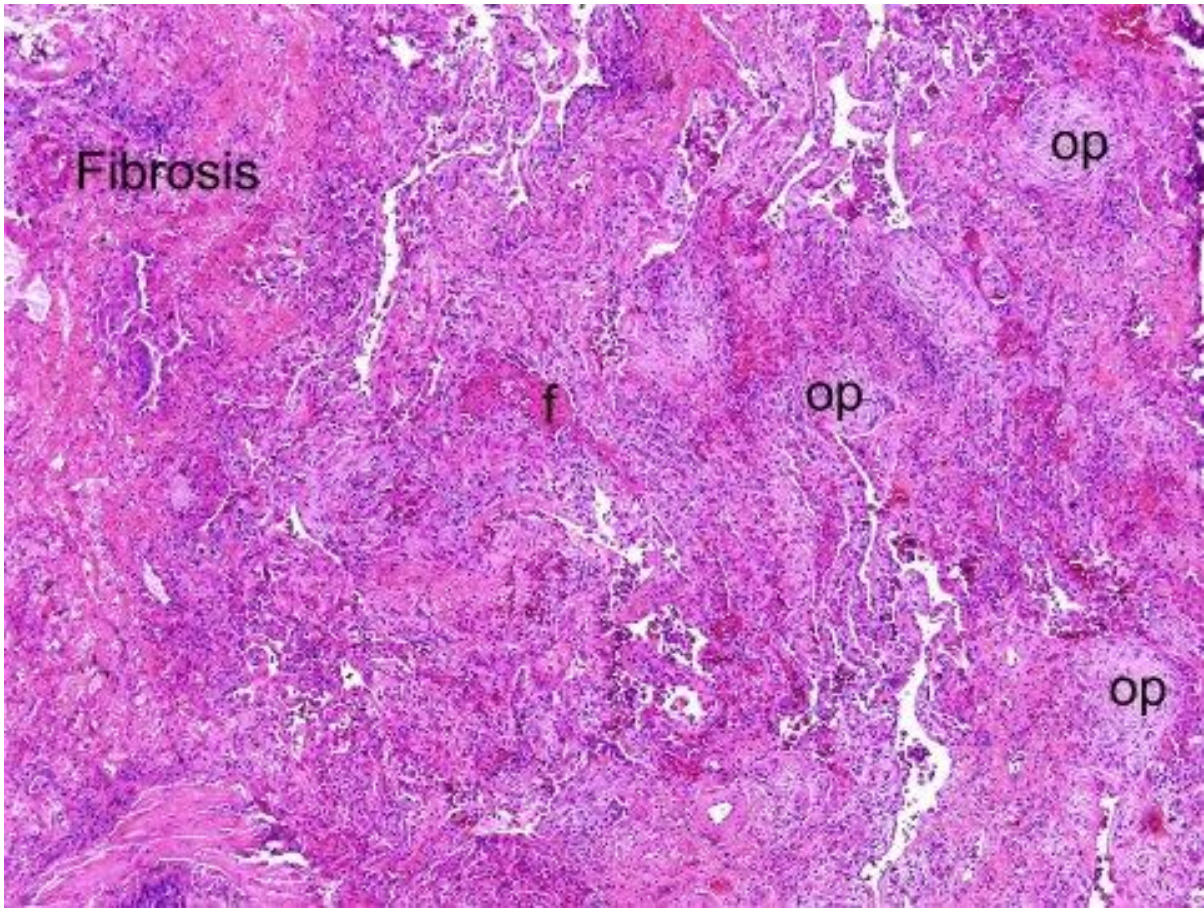


FIBROSING

- **“IDIOPATHIC” PULMONARY FIBROSIS (IPF)**
- **NONSPECIFIC INTERSTITIAL FIBROSIS**
- **“CRYPTOGENIC” ORGANIZING PNEUMONIA**
- **“COLLAGEN” VASCULAR DISEASES**
- **PNEUMOCONIOSES**
- **DRUG REACTIONS**
- **RADIATION CHANGES**

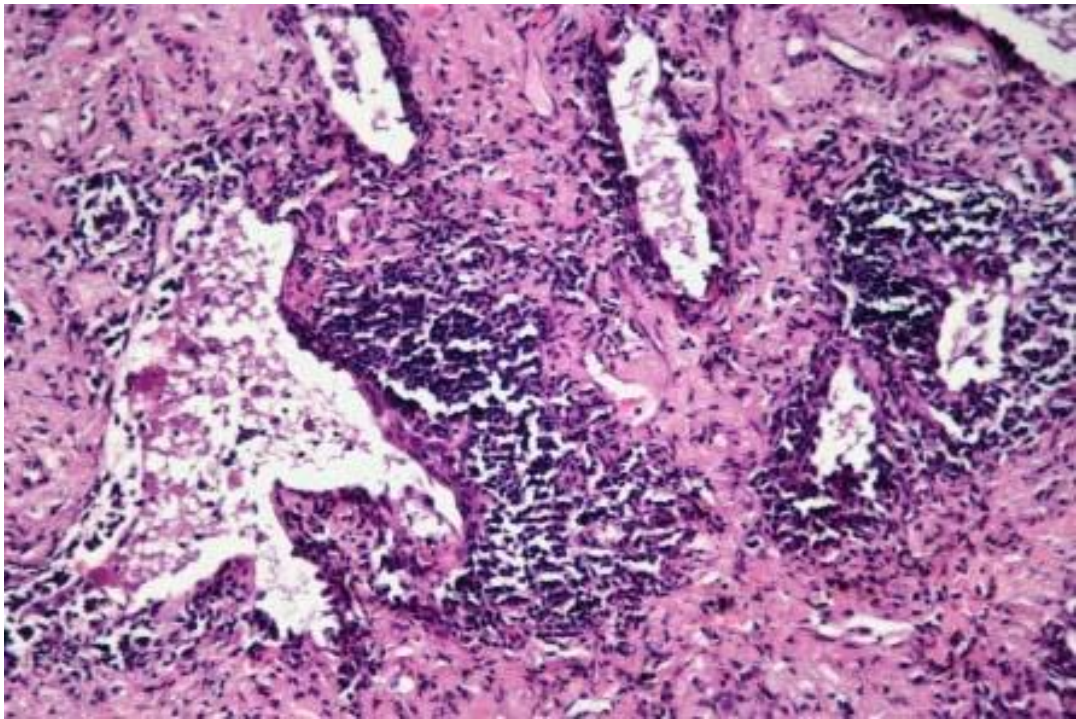
IPF (UIP)

- **IDIOPATHIC, i.e., not from any usual caused, like lupus, scleroderma**
- **FIBROSIS**



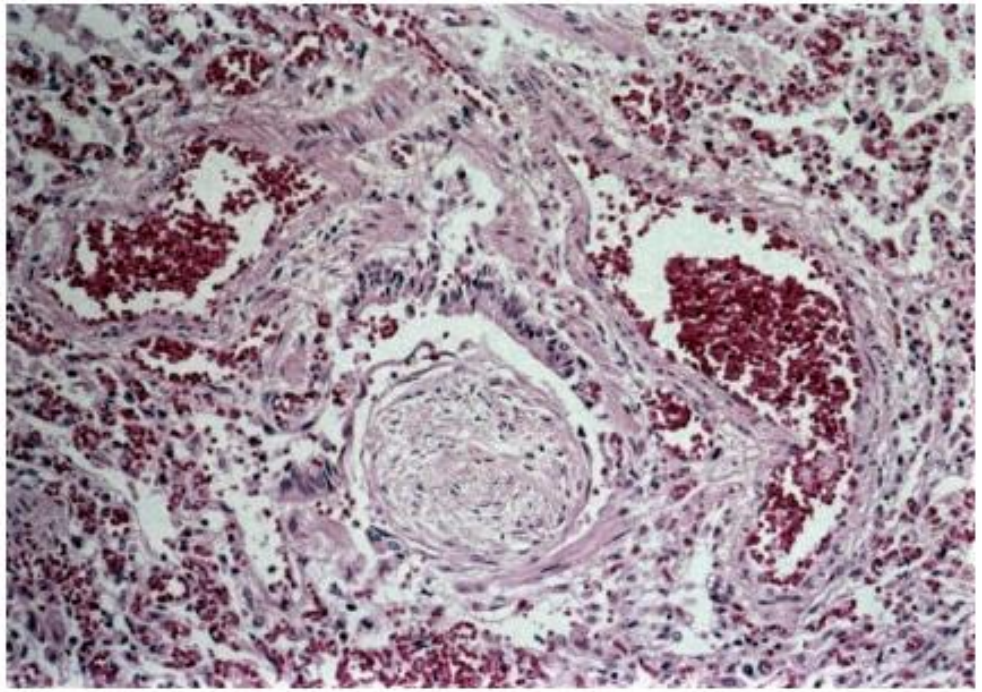
NON-SPECIFIC INTERSTITIAL PNEUMONIA

- **WASTEBASKET DIAGNOSIS, of ANY pneumonia (pneumonitis) of any known or unknown etiology**
  - **FIBROSIS**
  - **CELLULAR INFILTRATE (LYMPHS & PLASMA CELLS)**



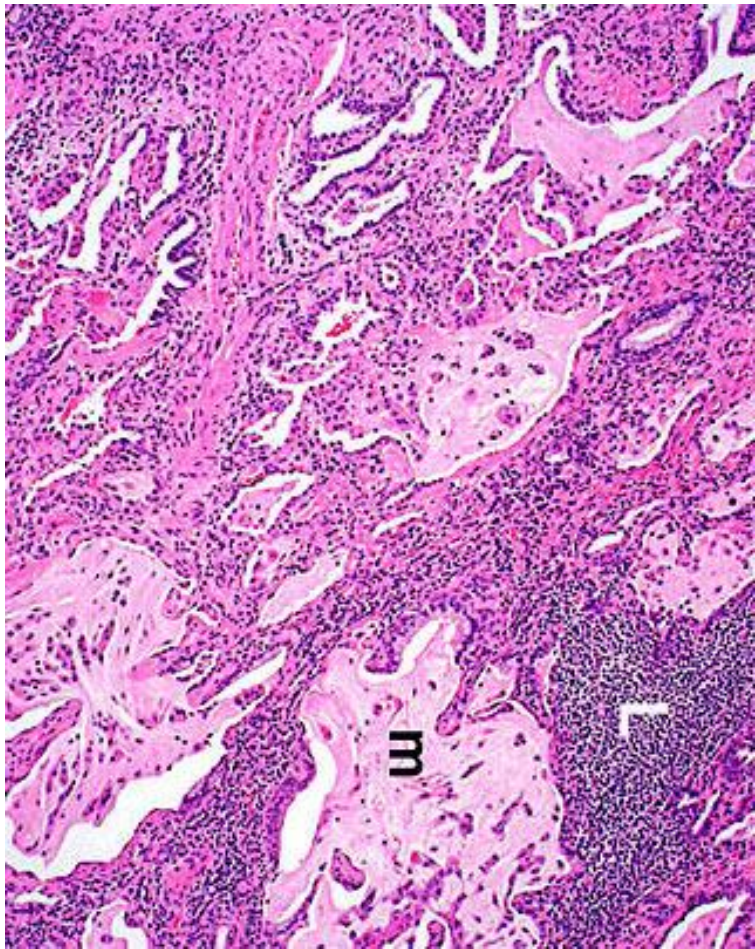
CRYPTOGENIC ORGANIZING PNEUMONIA (COP)

- **IDIOPATHIC**
- **“BRONCHIOLITIS OBLITERANS”**



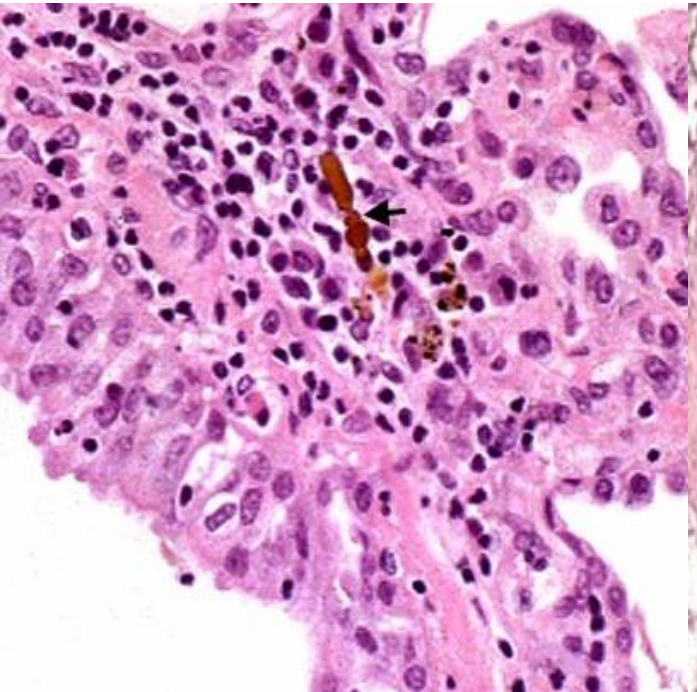
"COLLAGEN" VASCULAR DISEASES

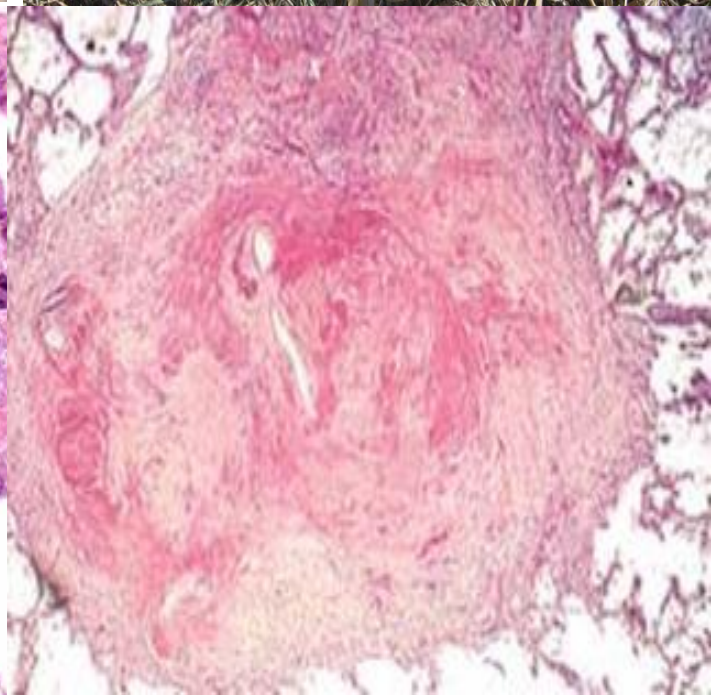
- **Rheumatoid Arthritis**
- **SLE ("Lupus")**
- **Progressive Systemic Sclerosis (Scleroderma)**



PNEUMOCONIOSES

- **“OCCUPATIONAL”**
- **“COAL MINERS LUNG”**
- **DUST OR CHEMICALS OR ORGANIC MATERIALS**
  - **Coal (anthracosis)**
  - **Silica**
  - **Asbestos**
  - **Be, FeO, $BaSO_4$, CHEMO**
  - **HAY, FLAX, BAGASSE, INSECTICIDES, etc.**





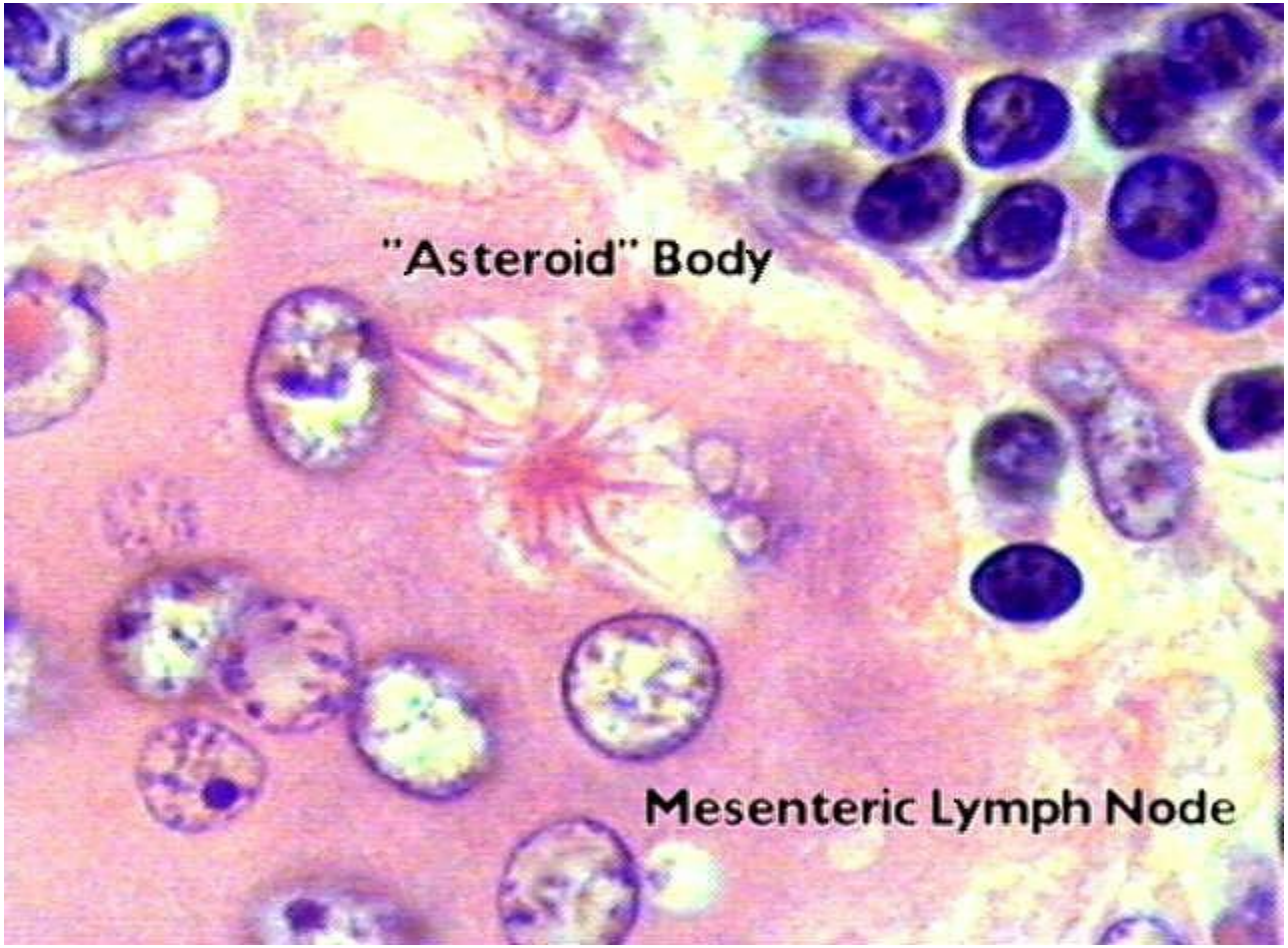
Coal, "bagasse", asbestos, silica nodules, and asbestos, going clockwise.

GRANULOMATOUS

- **SARCOIDOSIS, i.e., NON-caseating granulomas (IDIOPATHIC)**
- **HYPERSENSITIVITY (DUSTS, bacteria, fungi, Farmer's Lung, Pigeon Breeder's Lung)**

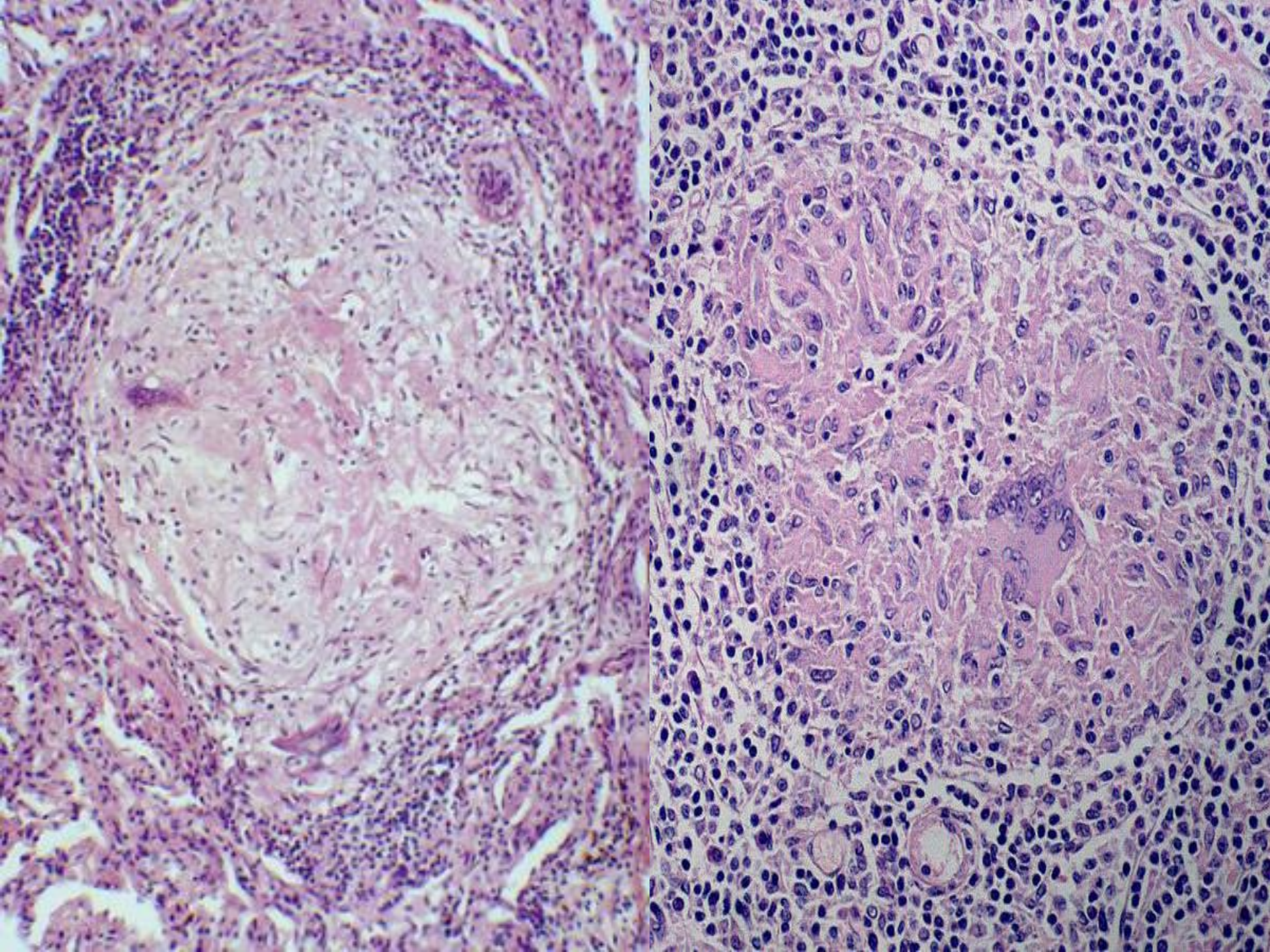
SARCOIDOSIS

- **Mainly LUNG, but eye, skin or ANYWHERE**
- **UNKNOWN ETIOLOGY**
- **IMMUNE, GENETIC factors**
- **F>>M**
- **B>>W**
- **YOUNG ADULT BLACK WOMEN**



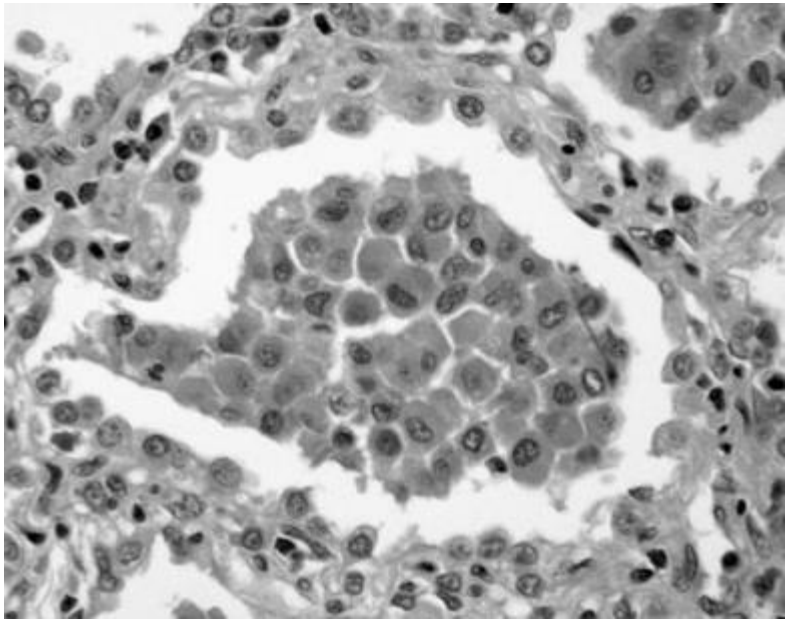
NON-Caseating Granulomas are the RULE

“Asteroid” bodies within these granulomas are virtually diagnostic



SMOKING RELATED

- **DIP (Desquamative Interstitial Pneumonia)**
  - **M>>F**
  - **CIGARETTES**
  - **100% Survival**



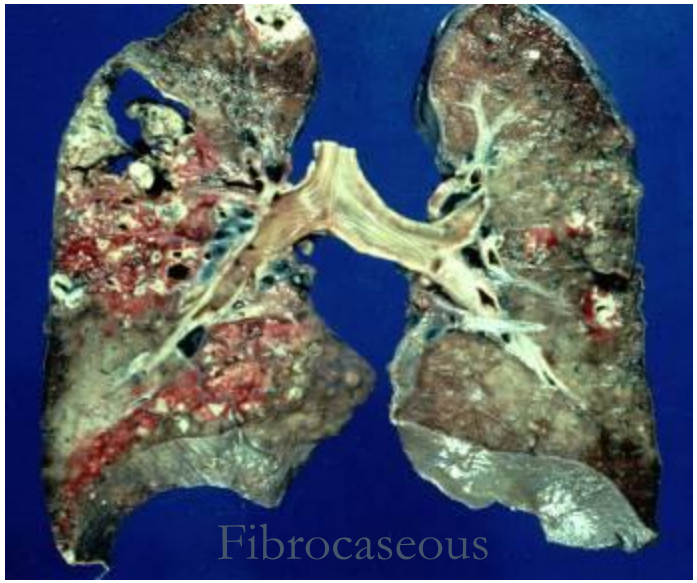
Alveolar Macrophages

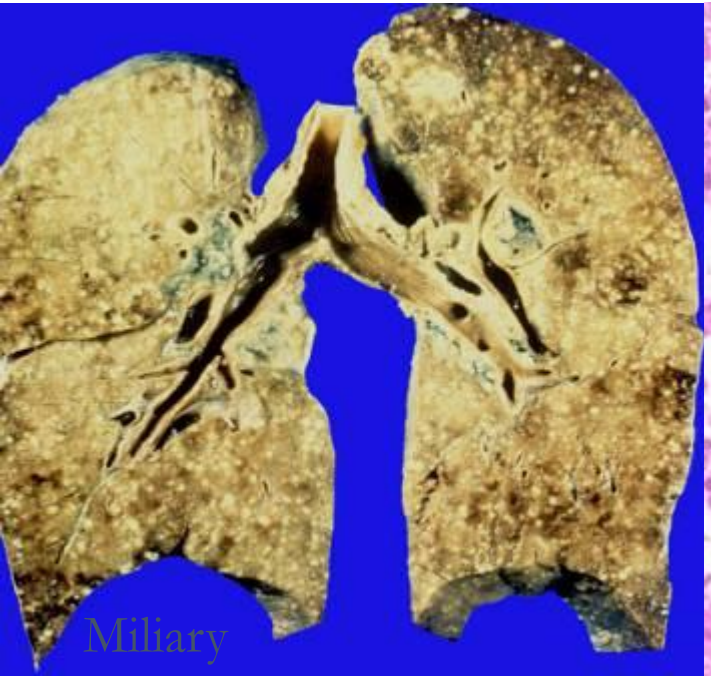
Pulmonary tuberculosis

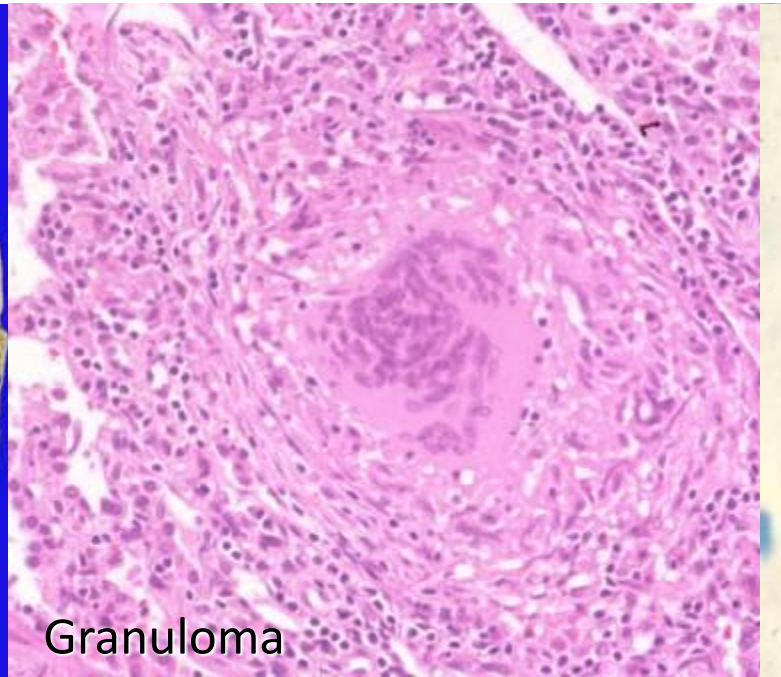
- **Caused by *Mycobacterium tuberculosis*.**
- **Transmitted through inhalation of infected droplets**
- **Primary**
  - **Single granuloma within parenchyma and hilar lymph nodes (Ghon complex).**
    - **Infection does not progress (most common).**
    - **Progressive primary pneumonia**
    - **Miliary dissemination (blood stream).**

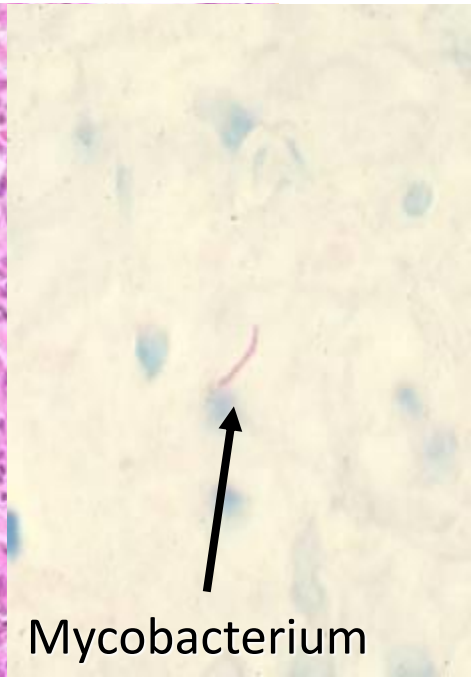
Pulmonary tuberculosis

- **Secondary**
  - **Infection (mostly through reactivation) in a previously sensitized individual.**
  - **Pathology**
    - **Cavitary fibrocaseous lesions**
    - **Bronchopneumonia**
    - **Miliary TB**







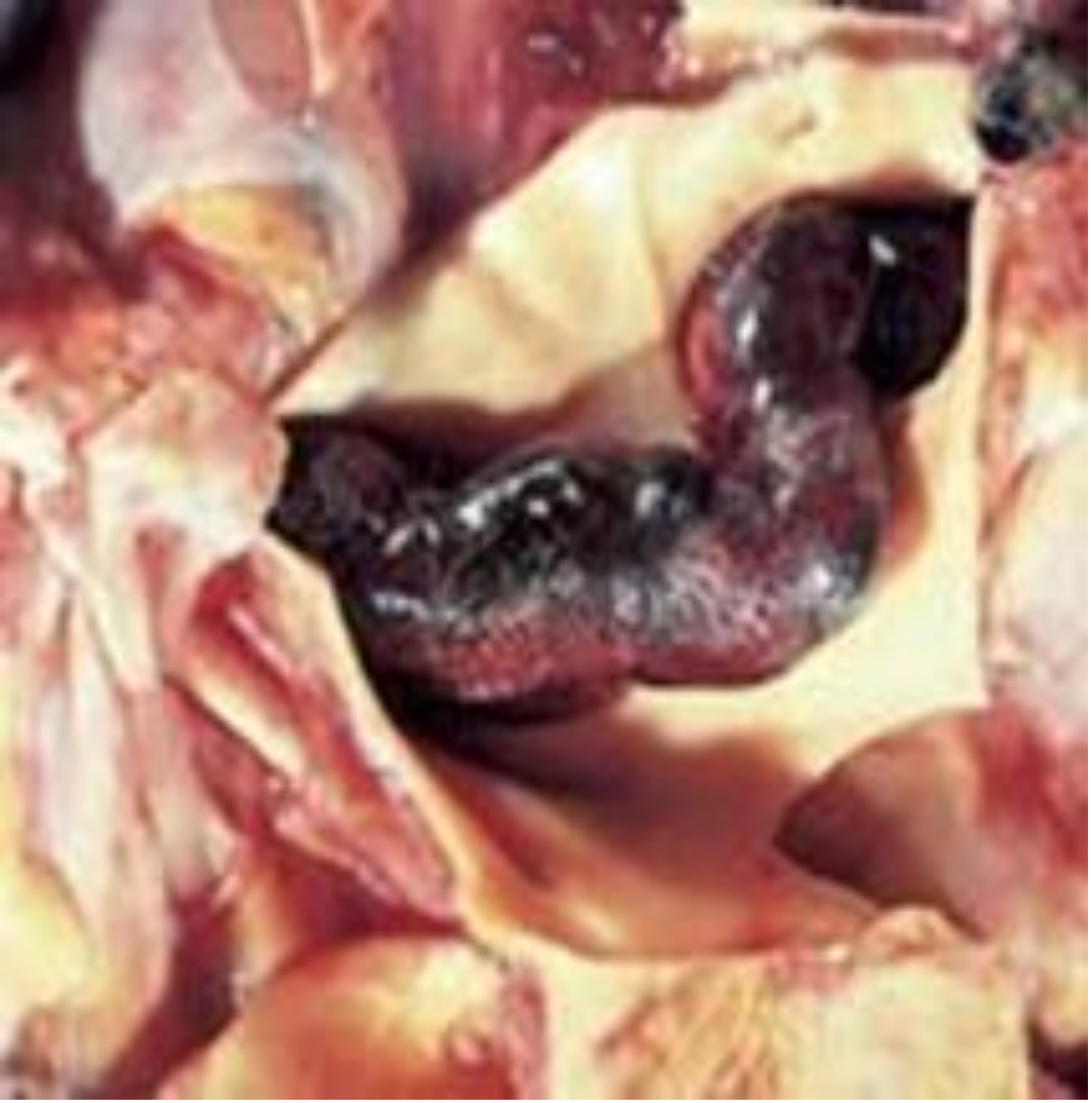


VASCULAR PULMONARY DISEASES

- **PULMONARY EMBOLISM** (with or usually WITHOUT infarction)
- **PULMONARY HYPERTENSION**, leading to cor pulmonale
- **HEMORRHAGIC SYNDROMES**
  - **GOODPASTURE SYNDROME**
  - **HEMOSIDEROSIS**, idiopathic
  - **WEGENER GRANULOMATOSIS**

P.E.

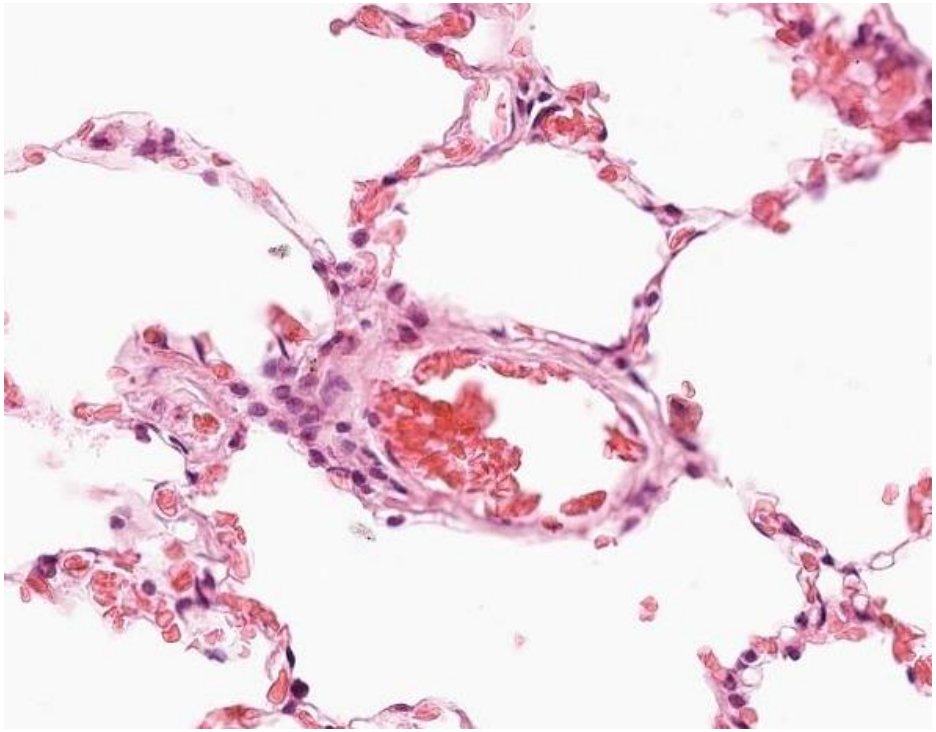
- **Usually secondary to debilitated states with immobilization, or following surgery**
- **Usually deep leg and deep pelvic veins (DVT), NOT superficial veins**
- **Follows Virchow's triad, i.e., 1) flow problems, 2) endothelial disruption, 3) hypercoagulabilty**
- **Usually do NOT infarct, usually ventilate**
- **When they DO infarct, the infarct is hemorrhagic**
- **Decreased PO2, acute chest pain, V/Q MIS-match**
- **DX: Chest CT, V/Q scan, angiogram**
- **RX: short term heparin, then long term coumadin**



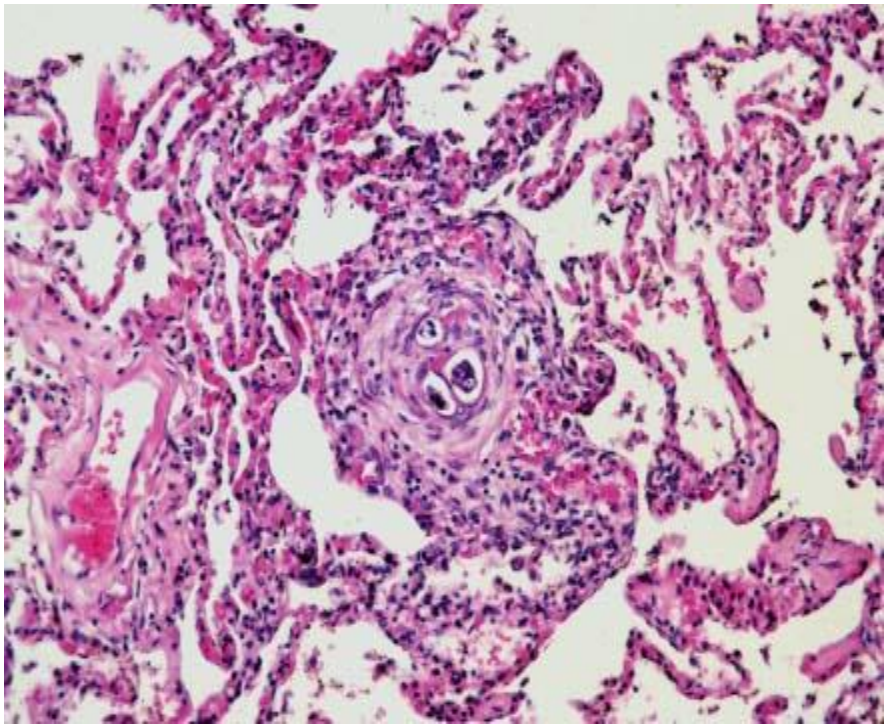
GROSS
“saddle”
embolism

PULMONARY HYPERTENSION

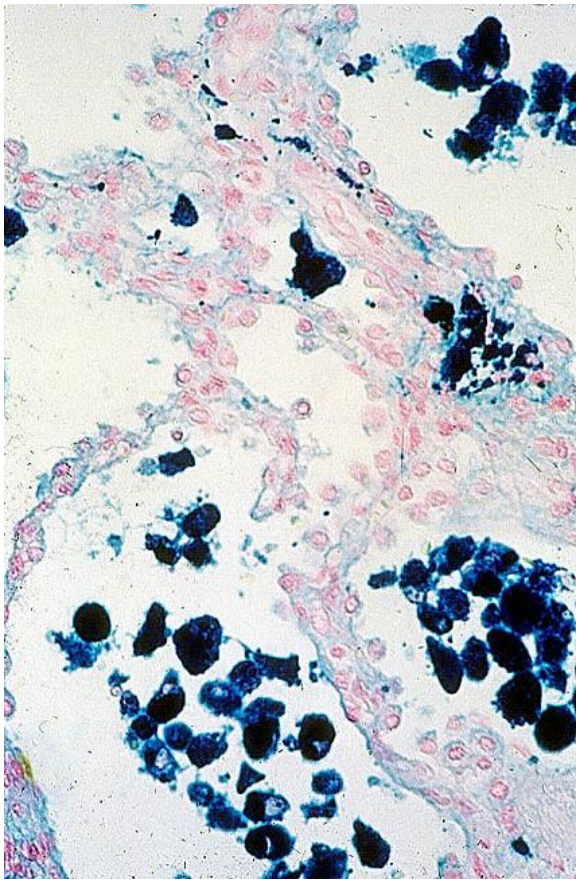
- **COPD, C"I"PD (vicious cycle)**
- **CHD (Congenital HD, increased left atrial pressure)**
- **Recurrent PEs**
- **Autoimmune, e.g., PSS (Scleroderma), i.e., fibrotic pulmonary vasculature**



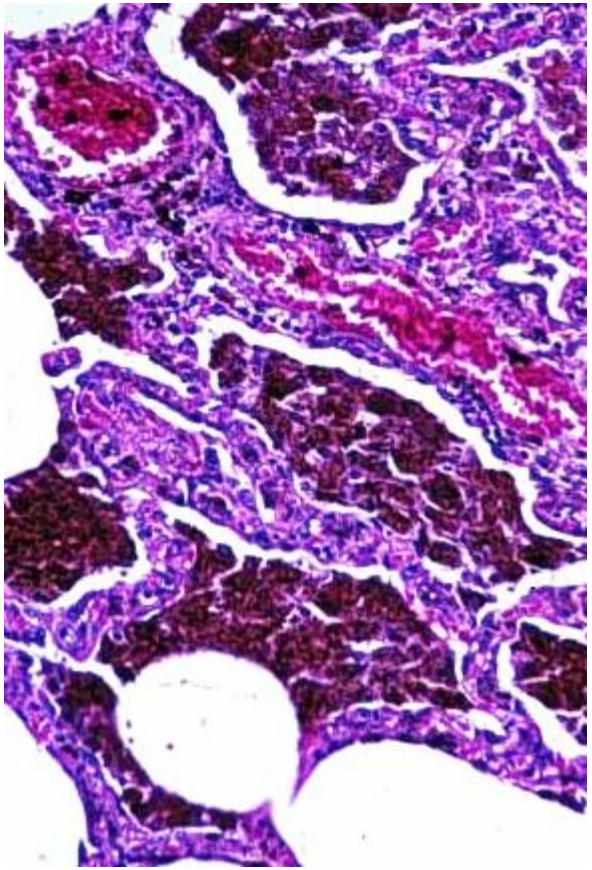
NORMAL pulmonary arteriole



VERY thickened arteriole in pulmonary hypertension



CHF, **CHRONIC**

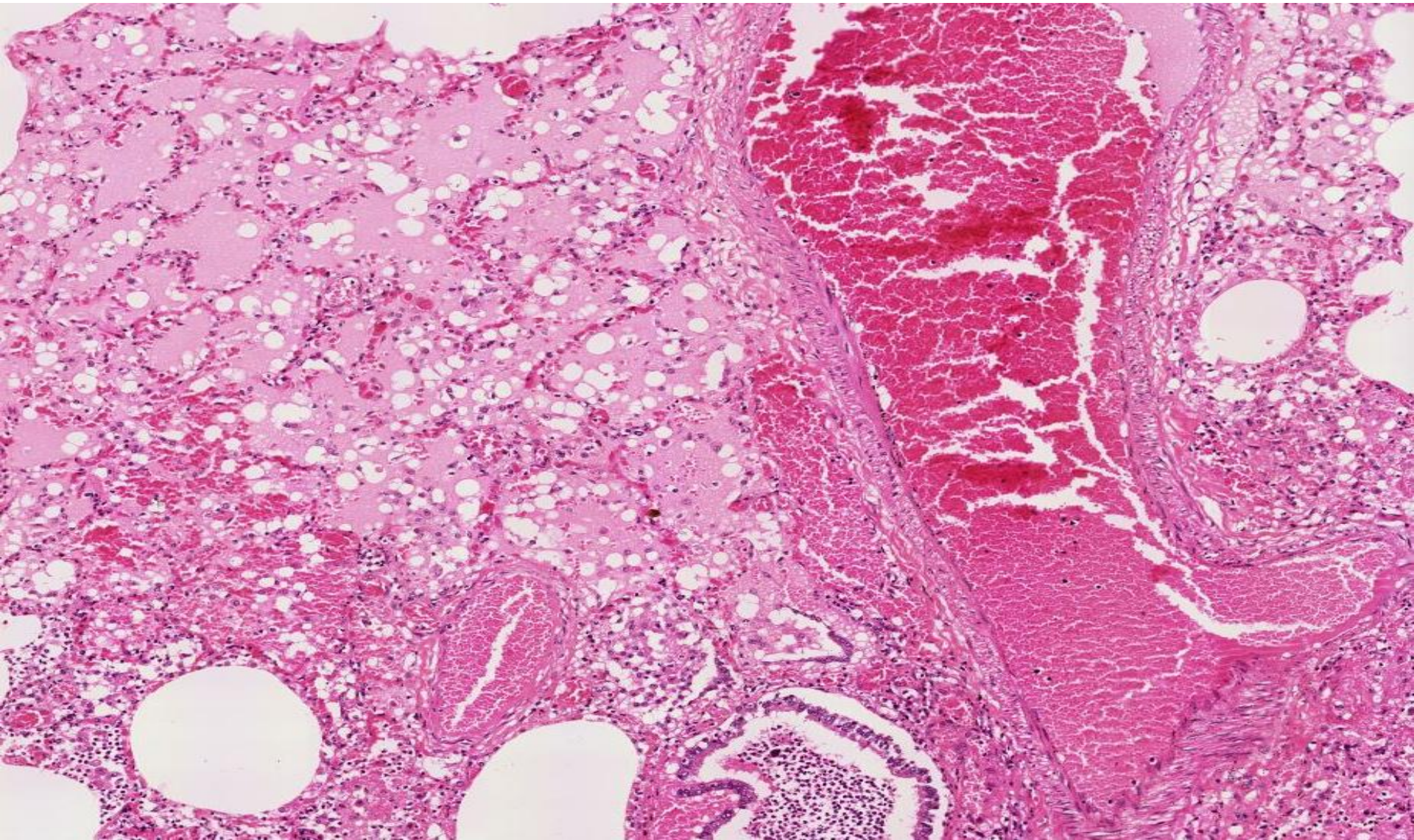


IDIOPATHIC

PULMONARY

HEMOSIDEROSIS

PNEUMONIA



PULMONARY INFECTIONS

COMMUNITY-ACQUIRED BACTERIAL ACUTE PNEUMONIAS

- Streptococcus Pneumoniae
- Haemophilus Influenzae
- Moraxella Catarrhalis
- Staphylococcus Aureus
- Klebsiella Pneumoniae
- Pseudomonas Aeruginosa
- Legionella Pneumophila

COMMUNITY-ACQUIRED ATYPICAL (VIRAL AND MYCOPLASMAL) PNEUMONIAS

- Morphology.
- Clinical Course.
- Influenza Infections
- Severe Acute Respiratory Syndrome (SARS)

NOSOCOMIAL PNEUMONIA

ASPIRATION PNEUMONIA

LUNG ABSCESS

- Etiology and Pathogenesis.

CHRONIC PNEUMONIA

- Histoplasmosis, Morphology
- Blastomycosis, Morphology
- Coccidioidomycosis, Morphology

PNEUMONIA IN THE IMMUNOCOMPROMISED HOST

PULMONARY DISEASE IN HUMAN IMMUNODEFICIENCY VIRUS INFECTION

BASIC CONSIDERATIONS

- **PNEUMONIA vs. PNEUMONITIS**
- **DIFFERENTIATION from INJURIES, OBSTRUCTIVE DISEASES, RESTRICTIVE DISEASES, VASCULAR DISEASES**
- **DIFFERENTIATION FROM NEOPLASMS**
- **CLASSICAL STAGES of INFLAMMATION**
- **LOBAR- vs. BRONCHO-**
- **INTERSTITIAL vs. ALVEOLAR**
- **COMMUNITY vs. NOSOCOMIAL**
- **ETIOLOGIC AGENTS vs. HOST IMMUNITY**
- **2 PRESENTING SYMPTOMS**
- **2 DIAGNOSTIC METHODS**
- **ANY ORGANISM CAN CAUSE PNEUMONIA!!!**

PREDISPOSING FACTORS

- **LOSS OF COUGH REFLEX**
- **DIMINISHED MUCIN or CILIA FUNCTION**
- **ALVEOLAR MACROPHAGE INTERFERENCE**
- **VASCULAR FLOW IMPAIRMENTS**
- **BRONCHIAL FLOW IMPAIRMENTS**

Although pneumonia is one of the most common causes of death, it usually does NOT occur in healthy people spontaneously

Classifications of PNEUMONIAS

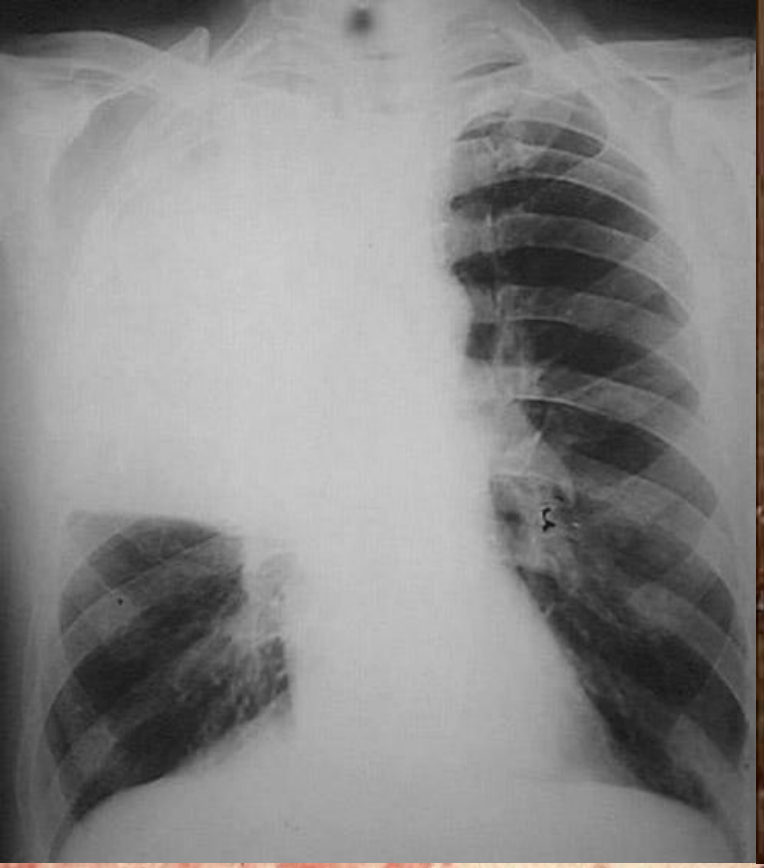
- **COMMUNITY ACQUIRED**
- **COMMUNITY ACQUIRED, ATYPICAL**
- **NOSOCOMIAL**
- **ASPIRATION**
- **CHRONIC**
- **NECROTIZING/ABSCESS FORMATION**
- **PNEUMONIAS in IMMUNOCOMPROMISED HOSTS**

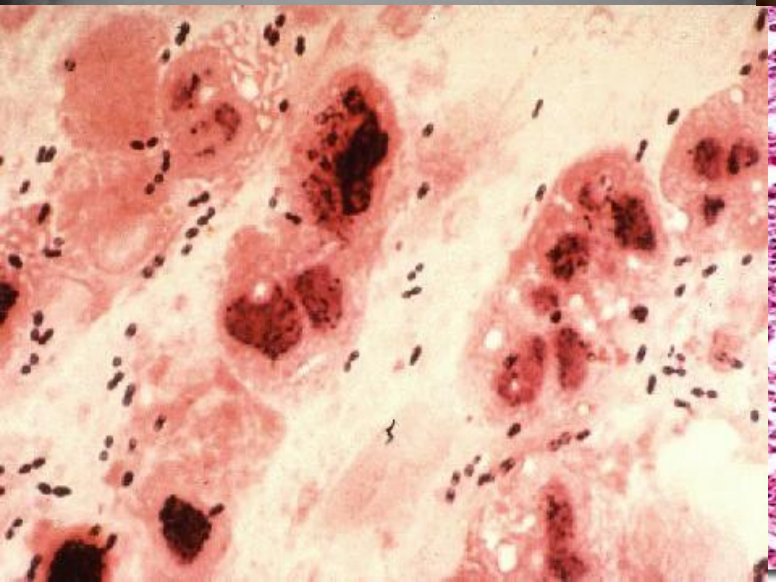
COMMUNITY ACQUIRED

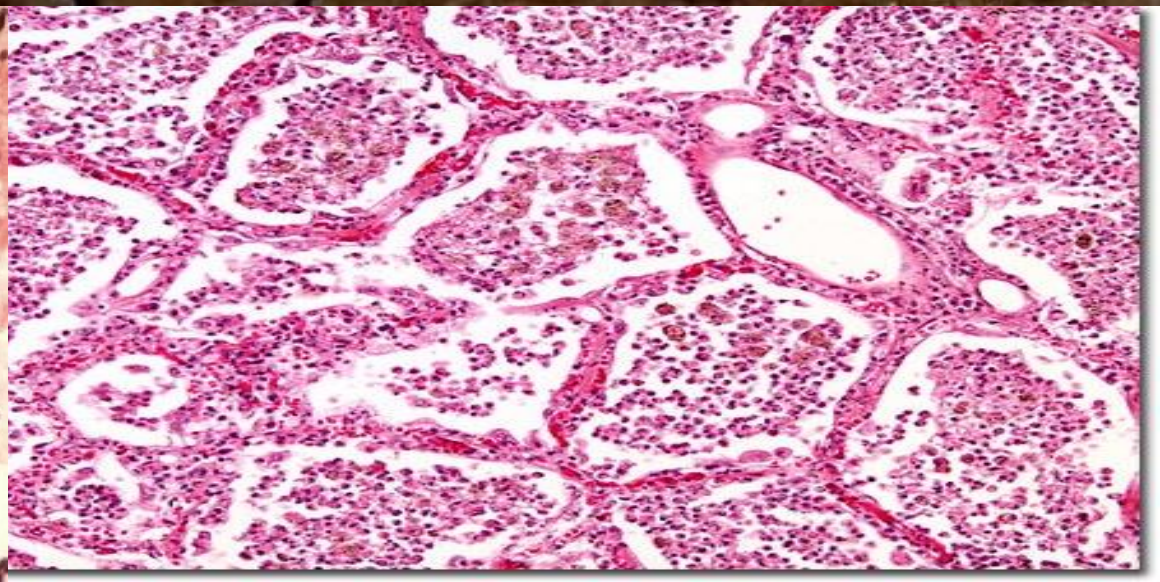
- **STREPTOCOCCUS PNEUMONIAE (i.e., “diplococcus”)**
- **HAEMOPHILUS INFLUENZAE (“H-Flu”)**
- **MORAXELLA**
- **STAPHYLOCOCCUS (STAPH)**
- **KLEBSIELLA PNEUMONIAE**
- **PSEUDOMONAS AERUGINOSA**
- **LEGIONELLA PNEUMOPHILIA**

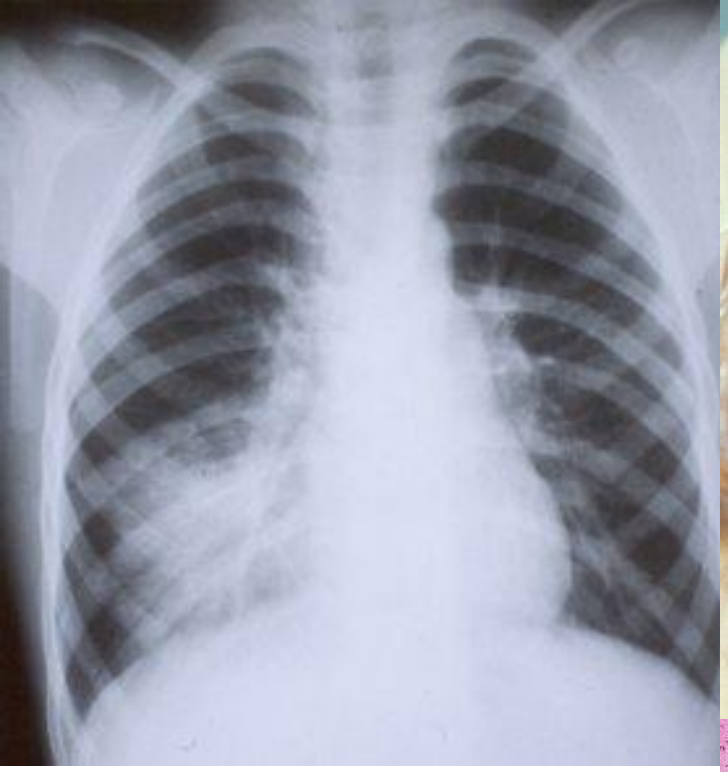
STREPTOCOCCUS

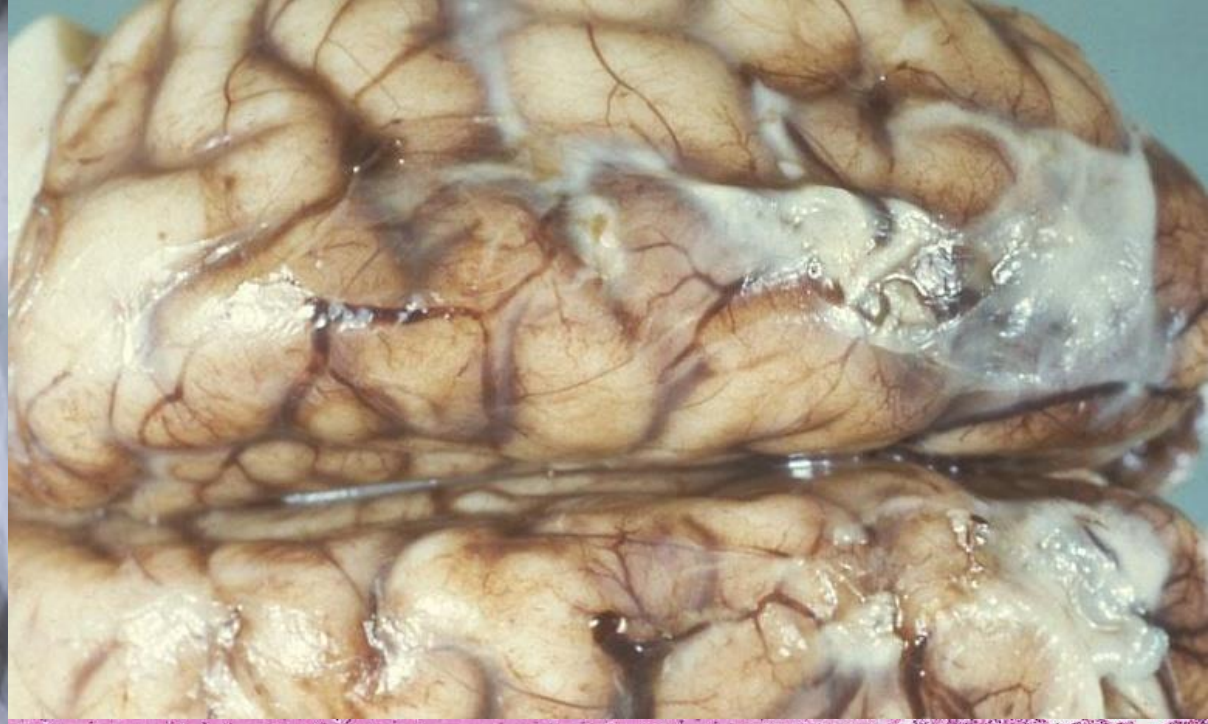
- **The classic LOBAR pneumonia**
- **Normal flora in 20% of adults**
- **Only 20% of victims have + blood cultures**
- **"Penicillins" are often 100% curative**
- **Vaccines are often 100% preventive**

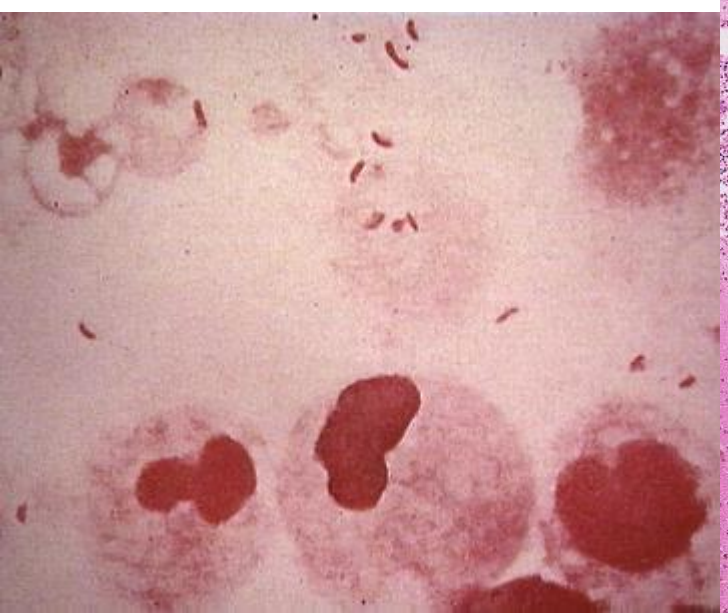


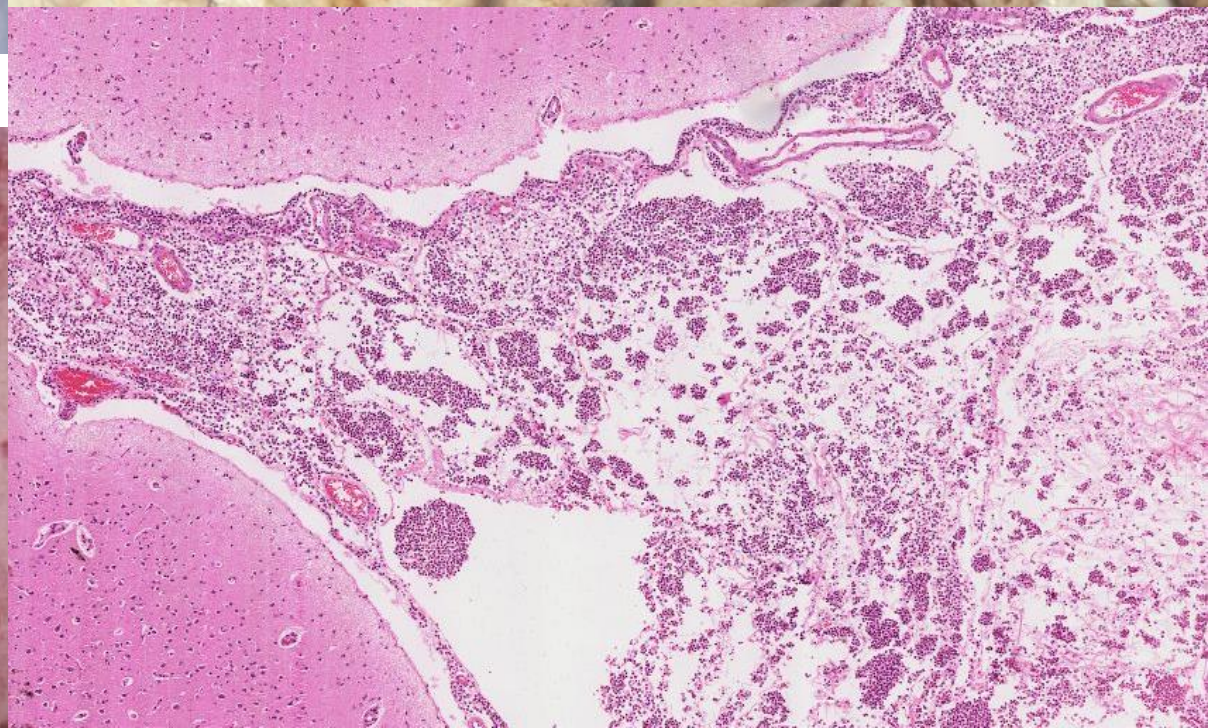












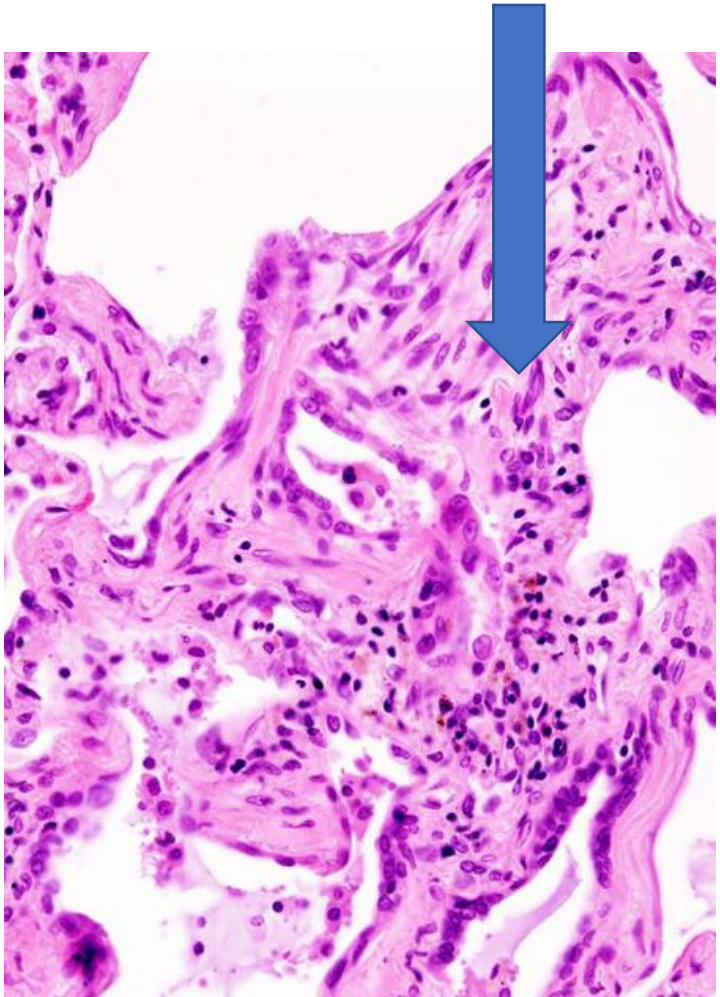
MORPHOLOGY

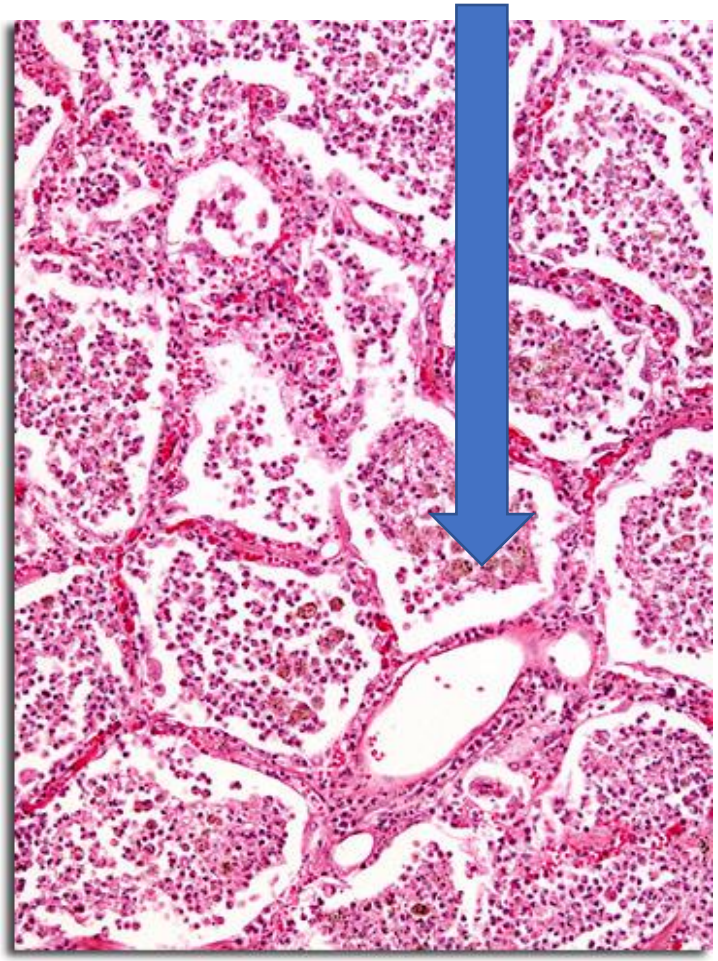
- **ACUTE**
- **ORGANIZING**
- **CHRONIC**
- **FIBROSIS vs. FULL RESOLUTION**

- **"HEPATIZATION", RED vs. GREY**
- **CONSOLIDATION**
- **"INFILTRATE", XRAY vs. HISTOPATH**
- **Loss of "CREPITANCE"**

VIRAL PNEUMONIAS

- **Frequently “interstitial”, NOT alveolar**





ASPIRATION PNEUMONIAS

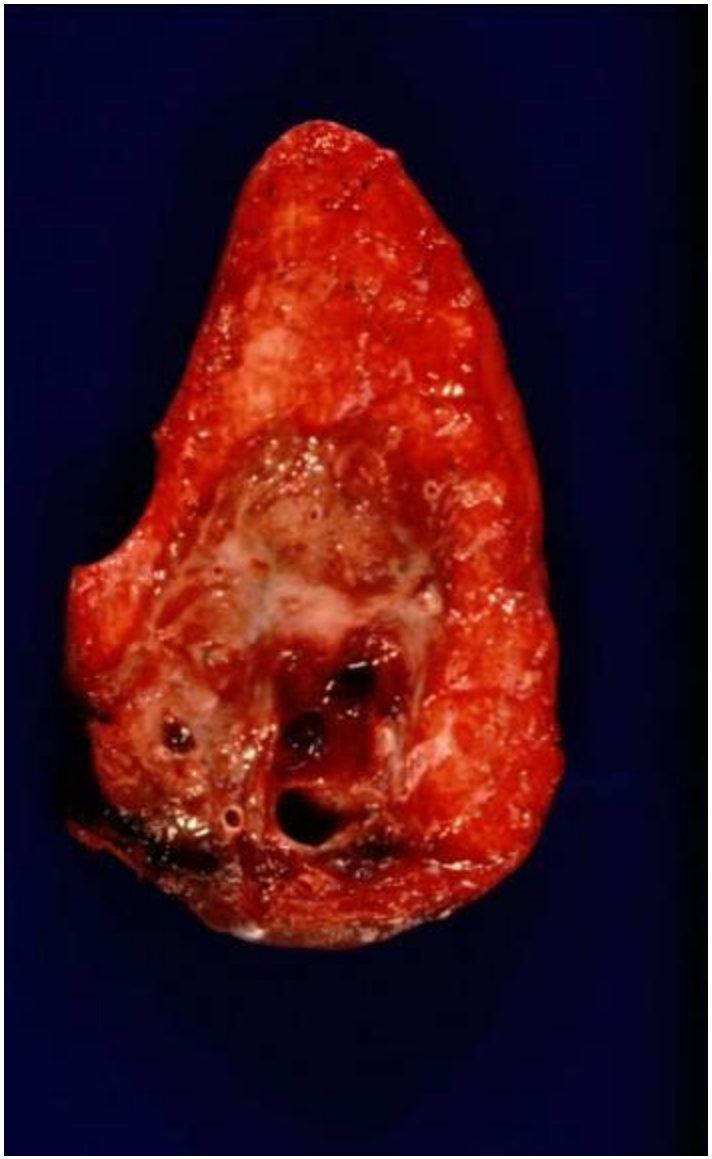
- **UNCONSCIOUS PATIENTS**
- **PATIENTS IN PROLONGED BEDREST**
- **LACK OF ABILITY TO SWALLOW OR GAG**
- **USUALLY CAUSED BY ASPIRATION OF GASTRIC CONTENTS**
- **POSTERIOR LOBES (gravity dependent) MOST COMMONLY INVOLVED, ESPECIALLY THE SUPERIOR SEGMENTS of the LOWER LOBES**
- **Often lead to ABSCESSES**

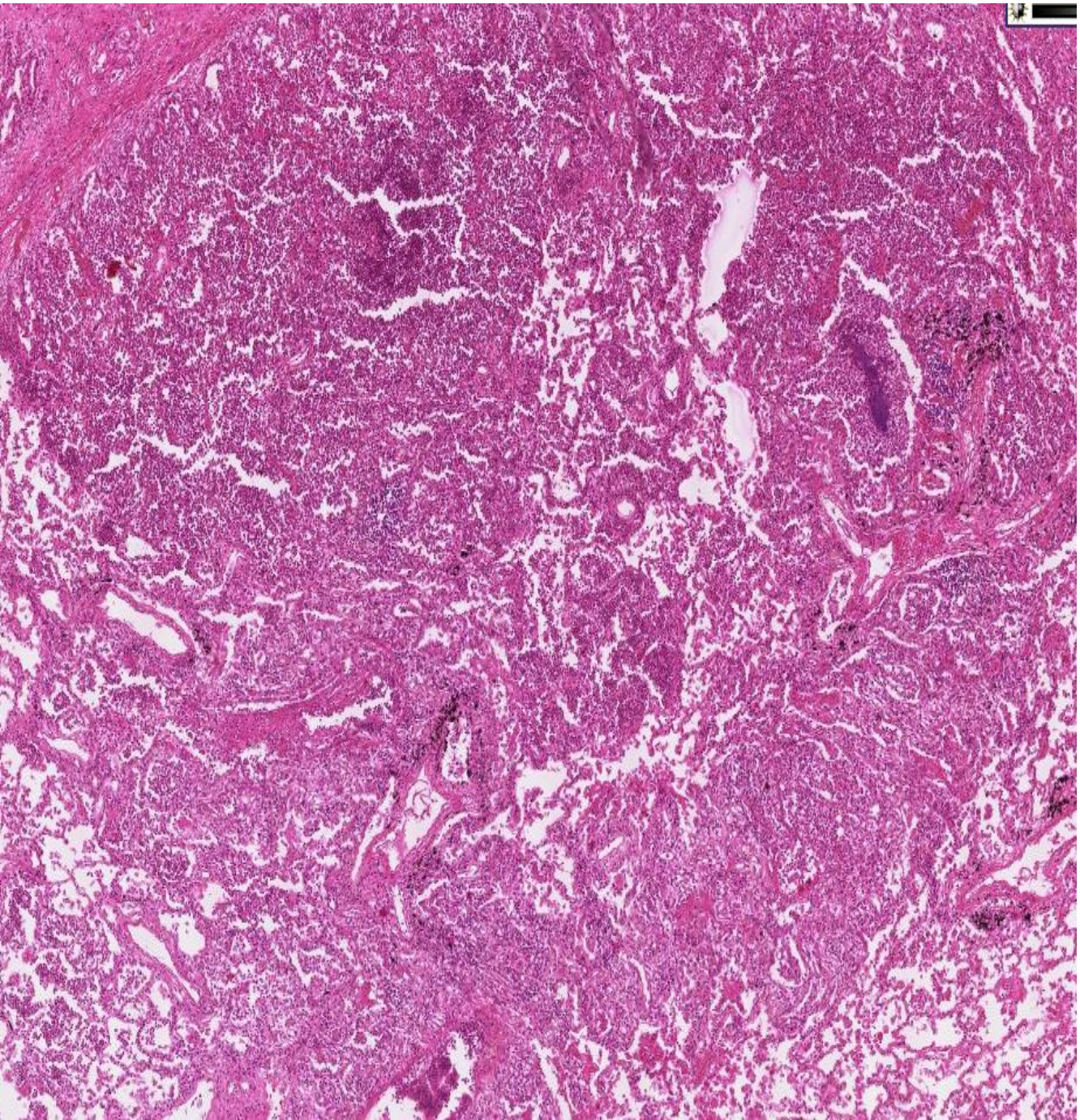
LUNG ABSCESSES

- **ASPIRATION**
- **SEPTIC EMBOLIZATION**
- **NEOPLASIA**
- **From NEIGHBORING structures:**
  - **ESOPHAGUS**
  - **SPINE**
  - **PLEURA**
  - **DIAPHRAGM**
- **ANY pneumonia which is severe and destructive, and UN-treated enough**

Lung abscess

- **Localized suppurative necrosis**
- **Organisms commonly cultured:**
  - **Staphylococci**
  - **Streptococci**
  - **Gram-negative**
  - **Anaerobes**
  - **Frequent mixed infections**
- **Pathogenesis:**
  - **Aspiration**
  - **Pneumonia**
  - **Septic emboli**
  - **Tumors**
  - **Direct infection**



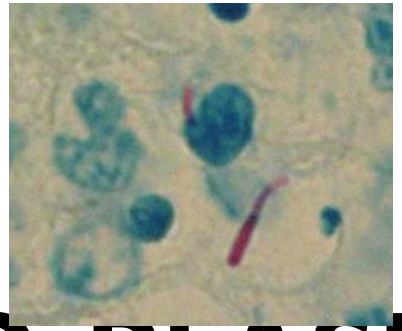


An abscess can be thought of as a pneumonia in which all of the normal lung outline can no longer be seen, and there is 100% pus.

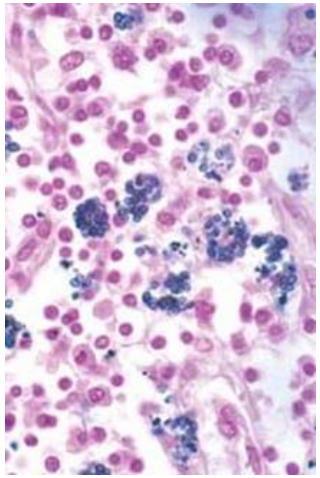
CHRONIC Pneumonias

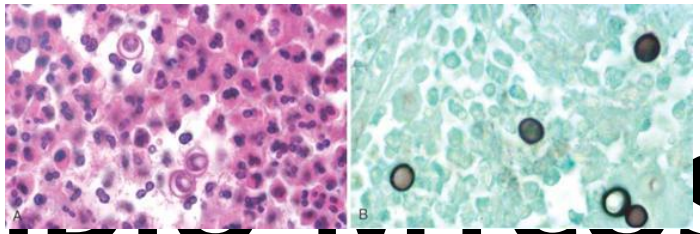
- **USUALLY NOT persistences of the community or nosocomial bacterial infections, but CAN BE, at least histologically**
- **Often SYNONYMOUS with the 4 classic fungal or granulomatous pulmonary infections infections, i.e., TB, Histo-, Blasto-, Coccidio-**
- **If you see pulmonary granulomas, think of a CHRONIC process, often years**

CHRONIC Pneumonias

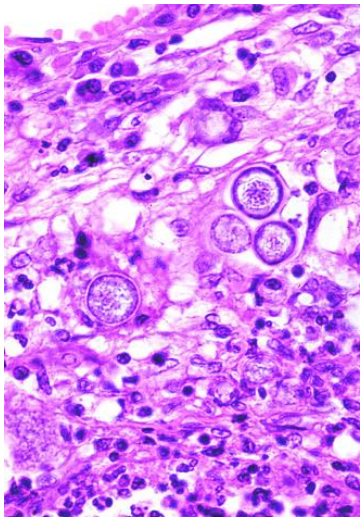


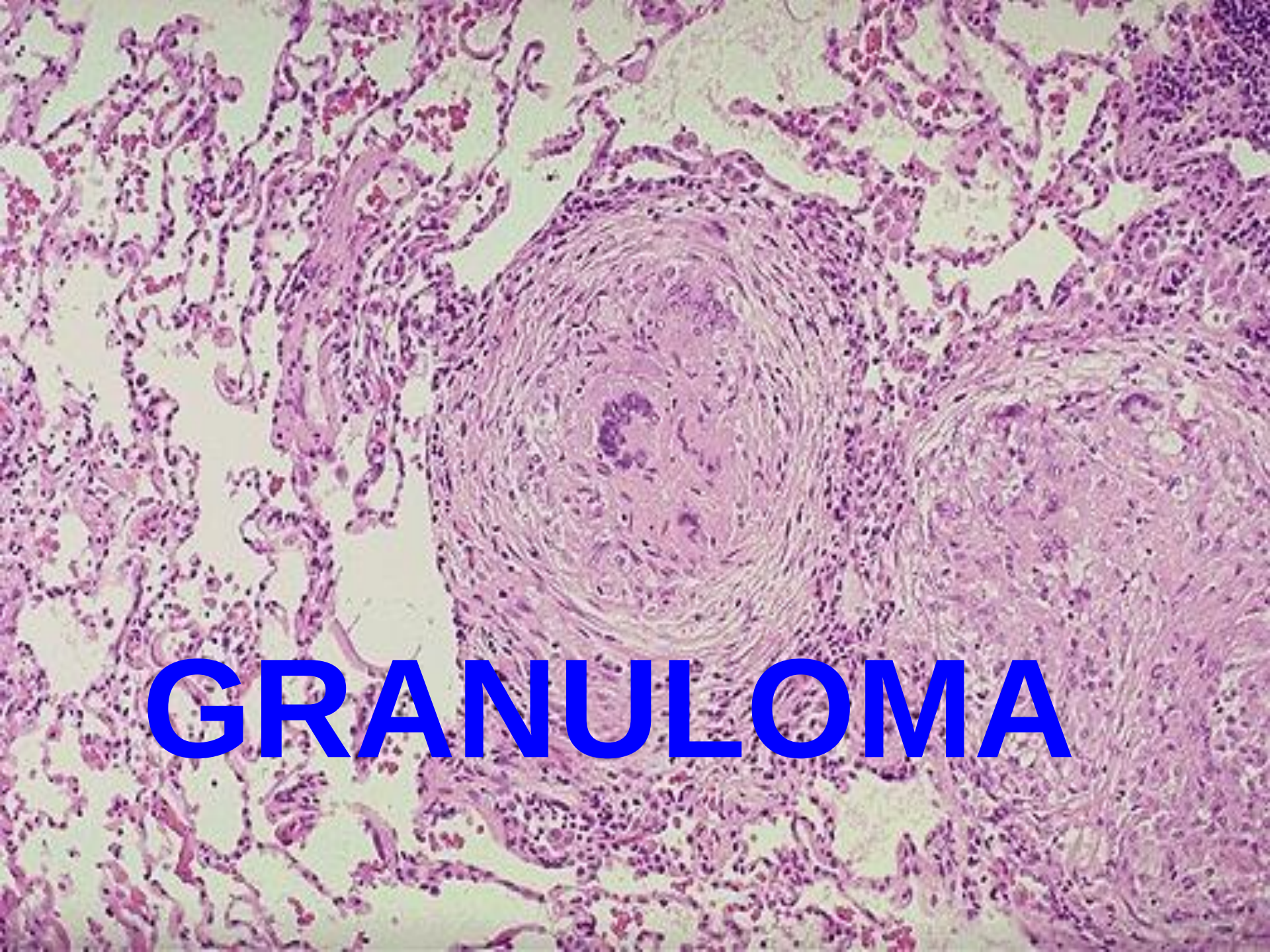
- **TB**
- **HISTO-PLASMOSIS**
- **BLASTO-MYCOSIS**





- **COCCIDIO-MYCOSIS**




GRANULOMA

LUNG TUMORS

- **Benign, malignant, epithelial, mesenchymal, but 90% are CARCINOMAS**
- **BIGGEST USA killer. Why? Ans: Prevalence not as high as prostate or breast but mortality higher. Only 15% 5 year survival.**
- **TOBACCO has polycyclic aromatic hydrocarbons, such as benzopyrene, anthracenes, radioactive isotopes**
- **Radiation, asbestos, radon**
- **C-MYC, K-RAS, EGFR, HER-2/neu**

PATHOGENESIS

- **NORMAL BRONCHIAL MUCOSA**
- **METAPLASTIC/DYSPLASTIC MUCOSA**
- **CARCINOMA-IN-SITU (squamous, adeno)**
- **INFILTRATING (i.e., “INVASIVE”) cancer**

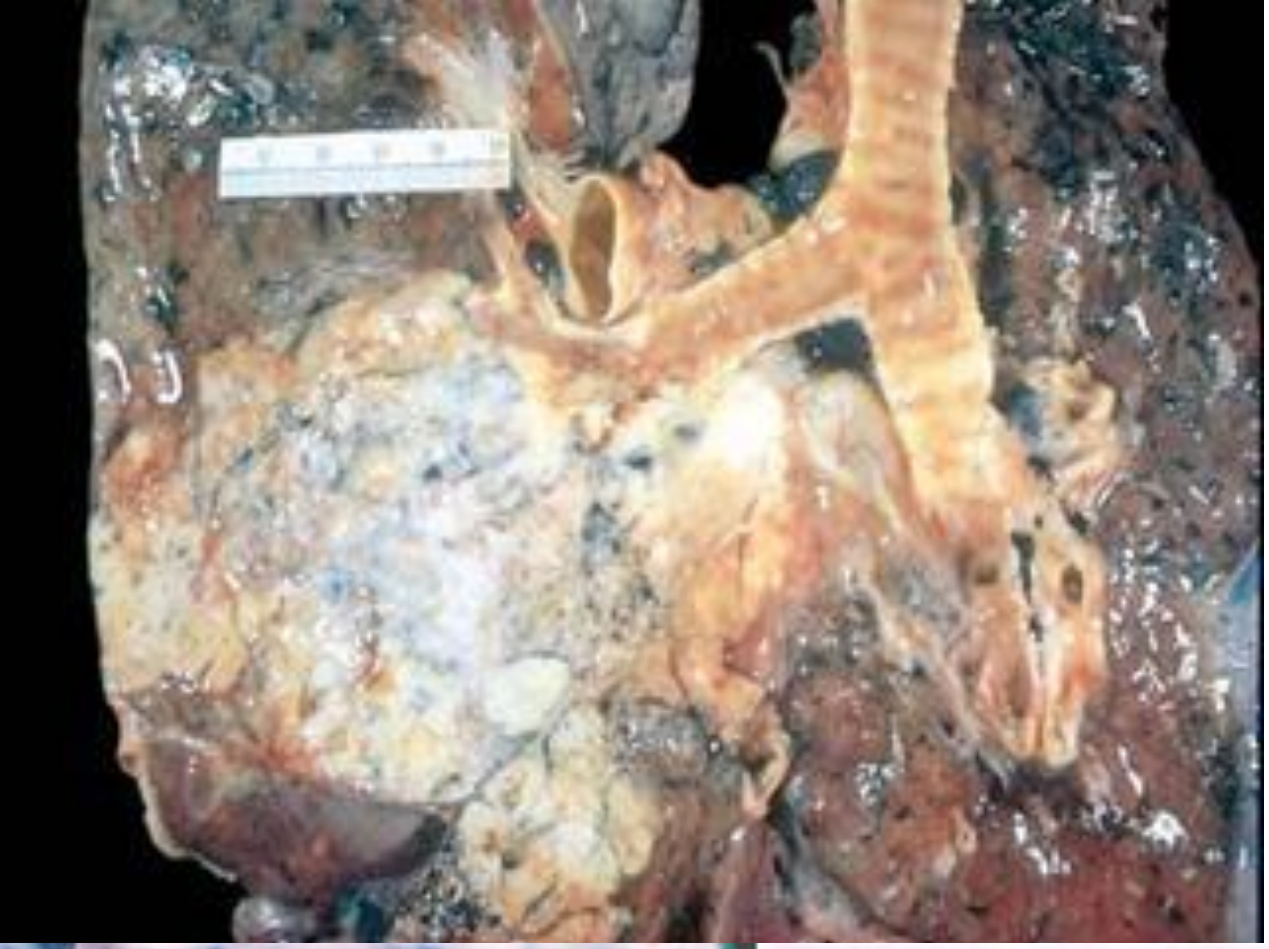
TWO TYPES

- **NON-SMALL CELL**
  - **SQUAMOUS CELL CARCINOMA**
  - **ADENOCARCINOMA**
  - **LARGE CELL CARCINOMA**

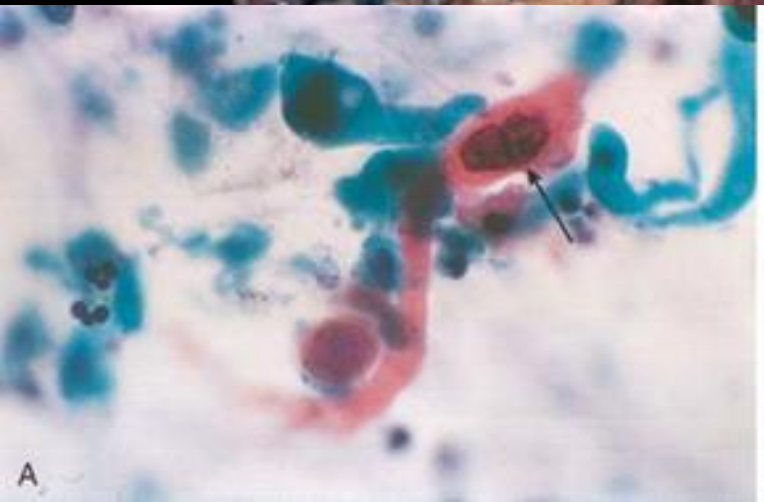
- **SMALL CELL CARCINOMA**

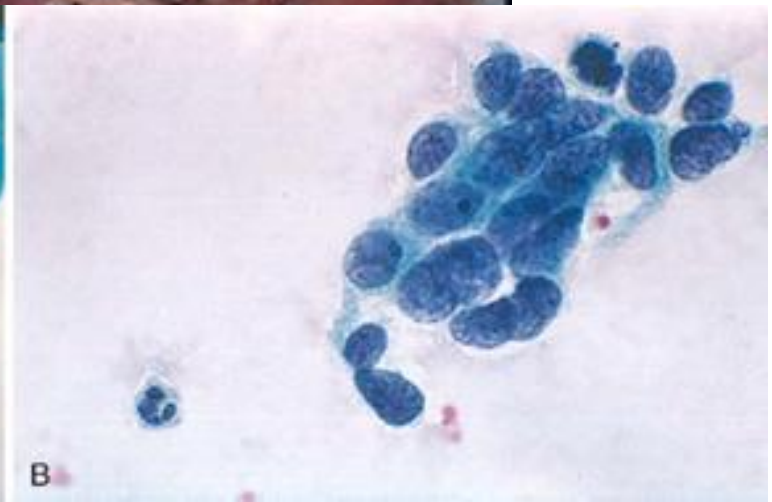
The BIG list

- **Squamous cell carcinoma**
- **Small cell carcinoma**
- **Combined small cell carcinoma**
- **Adenocarcinoma: Acinar, papillary, bronchioloalveolar, solid, mixed subtypes**
- **Large cell carcinoma**
- **Large cell neuroendocrine carcinoma**
- **Adenosquamous carcinoma**
- **Carcinomas with pleomorphic, sarcomatoid, or sarcomatous elements**
- **Carcinoid tumor: Typical, atypical**
- **Carcinomas of salivary gland type**
- **Unclassified carcinoma**

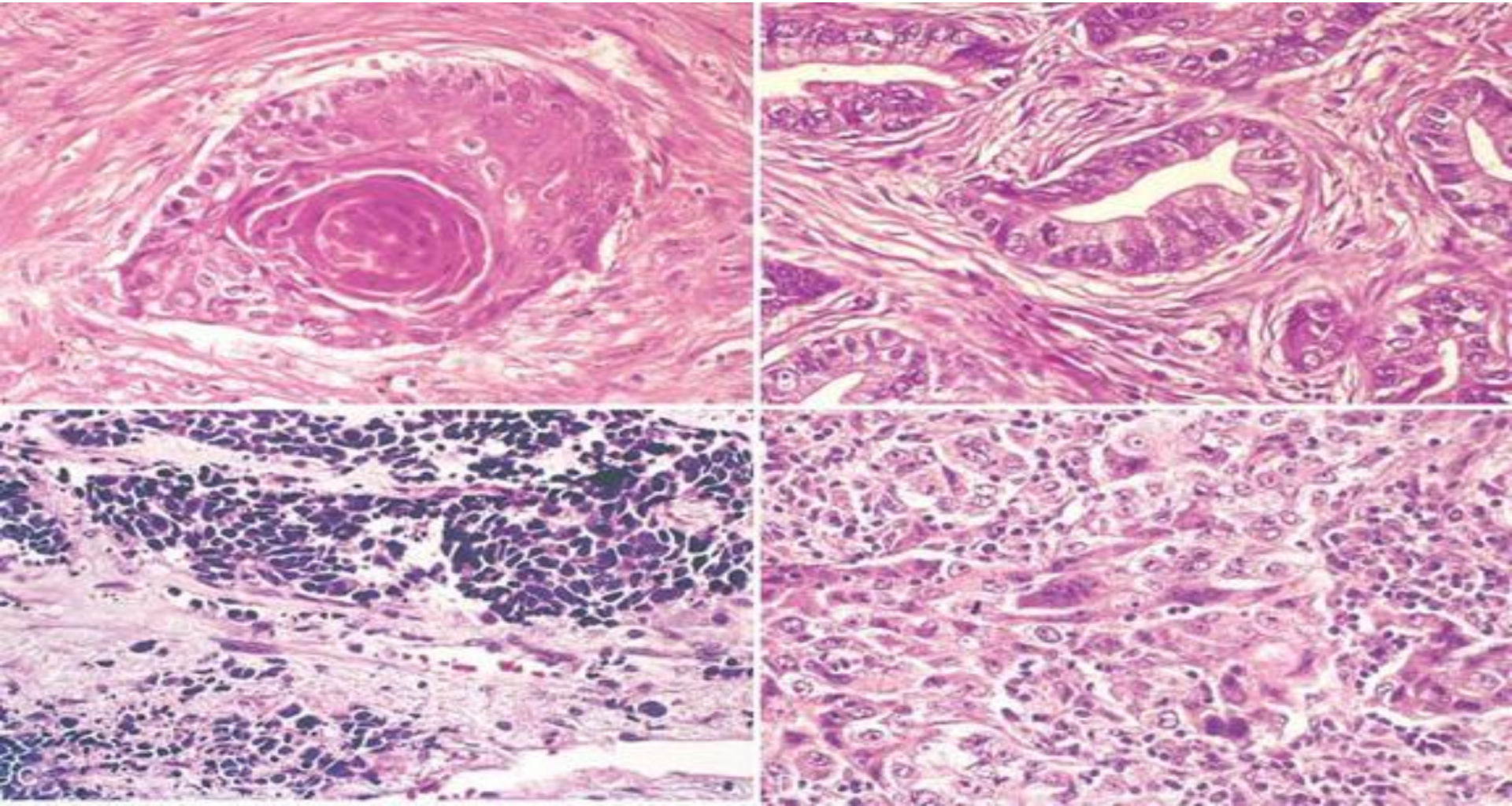


The classical squamous cell carcinoma starting in a large bronchus centrally, with bronchial obstruction. Adenocarcinomas tend to be more peripheral. Note the features of malignant cells on sputum cytology.





Name the four most common histologic patterns of lung carcinoma and explain why!
Squamous, adeno, large, small.



LOCAL effects of LUNG CANCER

Clinical Feature	Pathologic Basis
Pneumonia, abscess, lobar collapse	Tumor obstruction of airway
Lipid pneumonia	Tumor obstruction; accumulation of cellular lipid in foamy macrophages
Pleural effusion	Tumor spread into pleura
Hoarseness	Recurrent laryngeal nerve invasion
Dysphagia	Esophageal invasion
Diaphragm paralysis	Phrenic nerve invasion
Rib destruction	Chest wall invasion
SVC syndrome	SVC compression by tumor
Horner syndrome	Sympathetic ganglia invasion
Pericarditis, tamponade	Pericardial involvement

SVC, superior vena cava.

METASTATIC TUMORS

- **LUNG is the MOST COMMON site for all metastatic tumors, regardless of site of origin**
- **It is the site of FIRST CHOICE for metastatic sarcomas for purely anatomic reasons!**

PLEURA

- **PLEURITIS**
- **PNEUMOTHORAX**
- **EFFUSIONS**
  - **HYDROTHORAX**
  - **HEMOTHORAX**
  - **CHYLOTHORAX**
- **MESOTHELIOMAS**

PLEURITIS

- **Usual bacteria, viruses, etc.**
- **Infarcts**
- **Lung abscesses, empyema**
- **TB**
- **“Collagen” diseases, e.g., RA, SLE**
- **Uremia**
- **Metastatic**

PNEUMOTHORAX

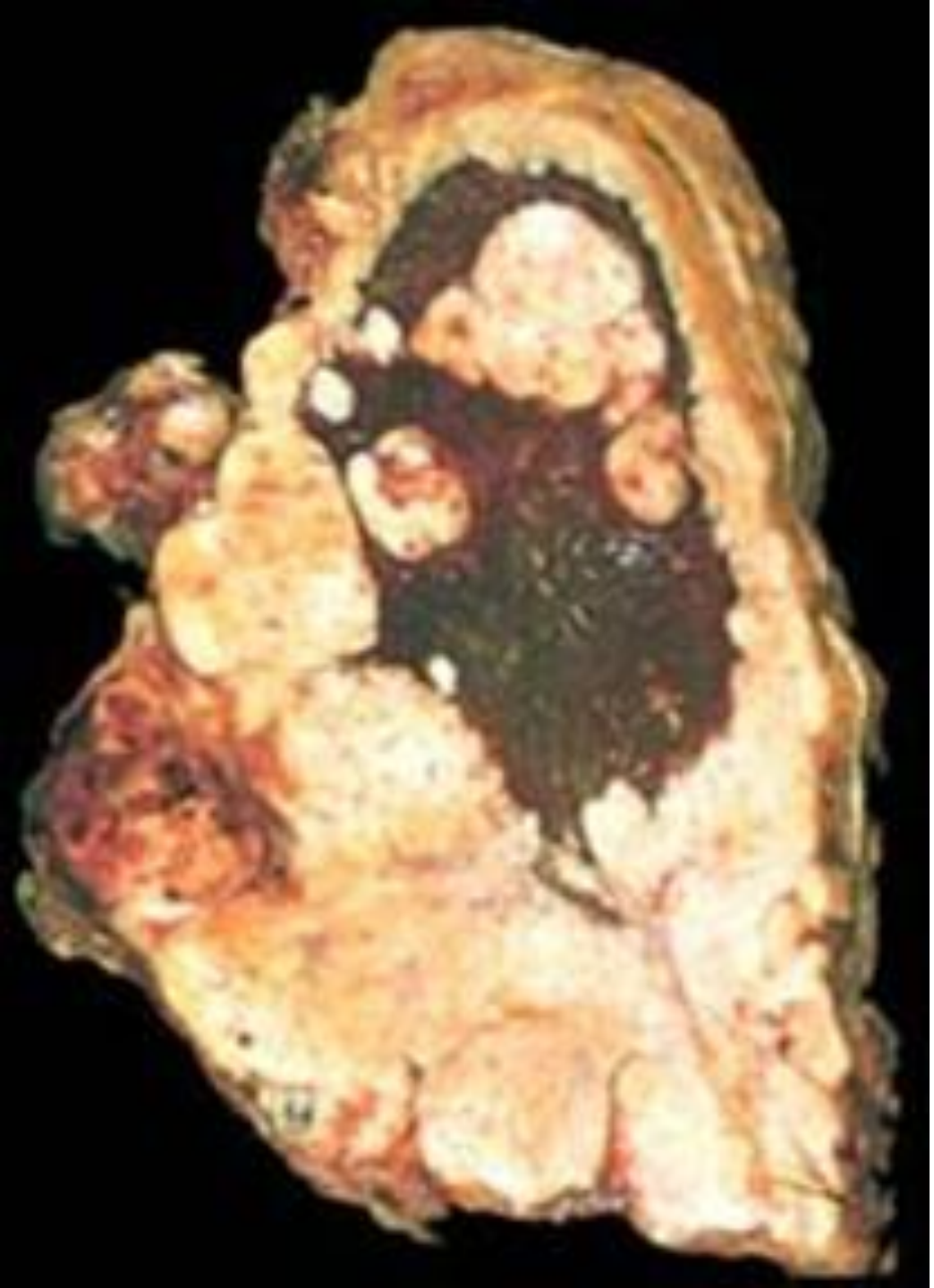
- **SPONTANEOUS, TRAUMATIC, THERAPEUTIC**
- **OPEN or CLOSED**
- **“TENSION” pneumothorax, “valvular” effect**
- **“Bleb” rupture**
- **Perforating injuries**
- **Post needle biopsy**

EFFUSIONS

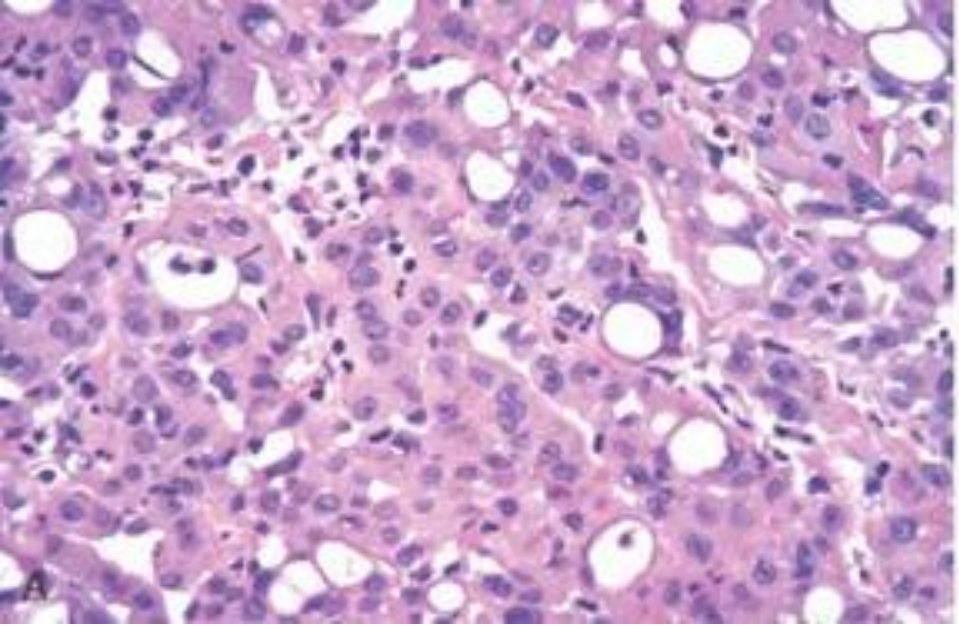
- **TRANSUDATE (HYDROTHORAX)**
- **EXUDATE (HYDROTHORAX)**
- **BLOOD (HEMOTHORAX)**
- **LYMPH (CHYLOTHORAX)**

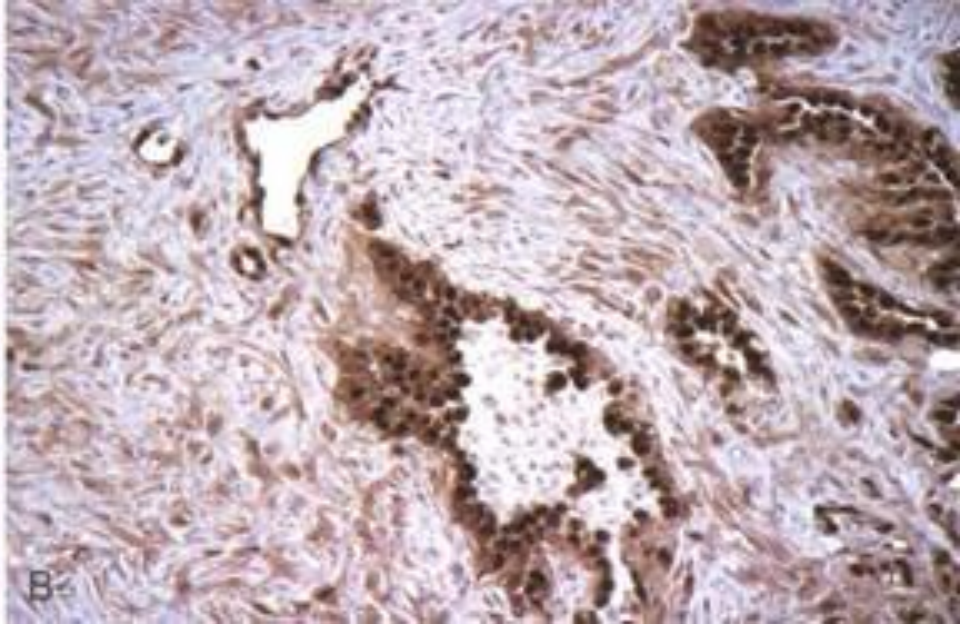
MESOTHELIOMAS

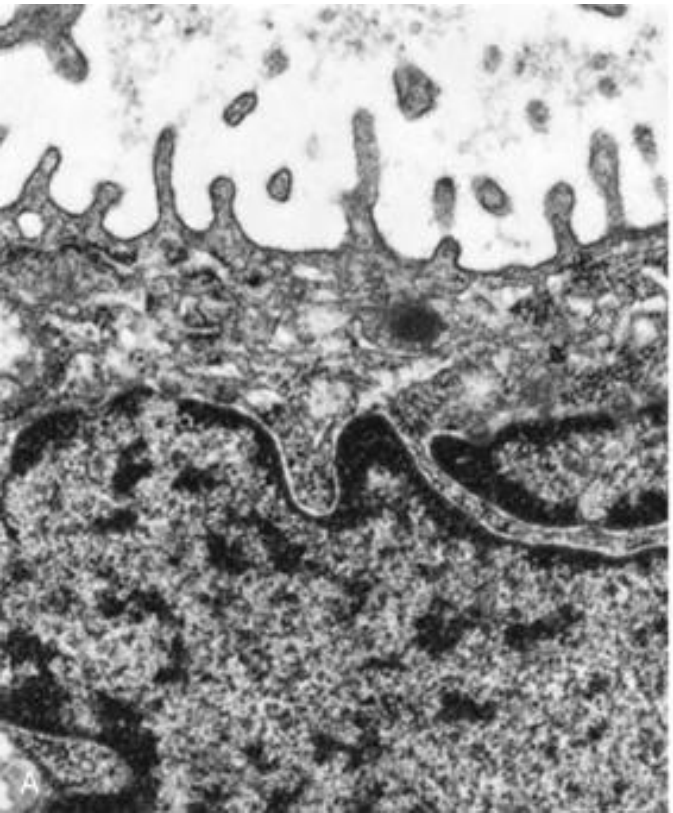
- **“Benign” vs. “Malignant” differentiation does not matter, but a self limited localized nodule can be regarded as benign, and a spreading tumor can be regarded as malignant**
- **Visceral or parietal pleura, pericardium, or peritoneum**
- **Most are regarded as asbestos caused or asbestos “related”**

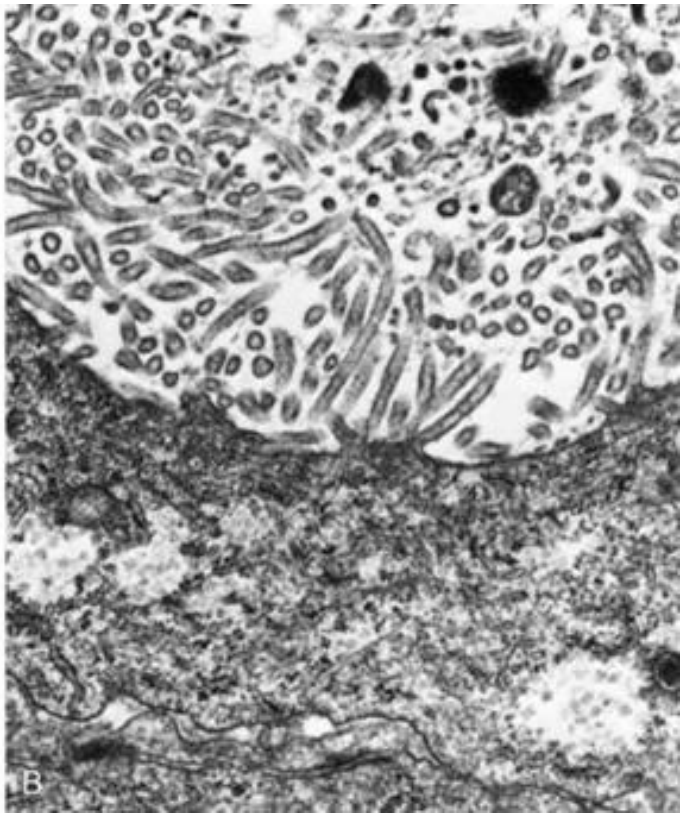


Typical growth appearance of a malignant mesothelioma, it compresses the lung from the OUTSIDE.









H&E,
IMMUNOCHEMISTRY

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