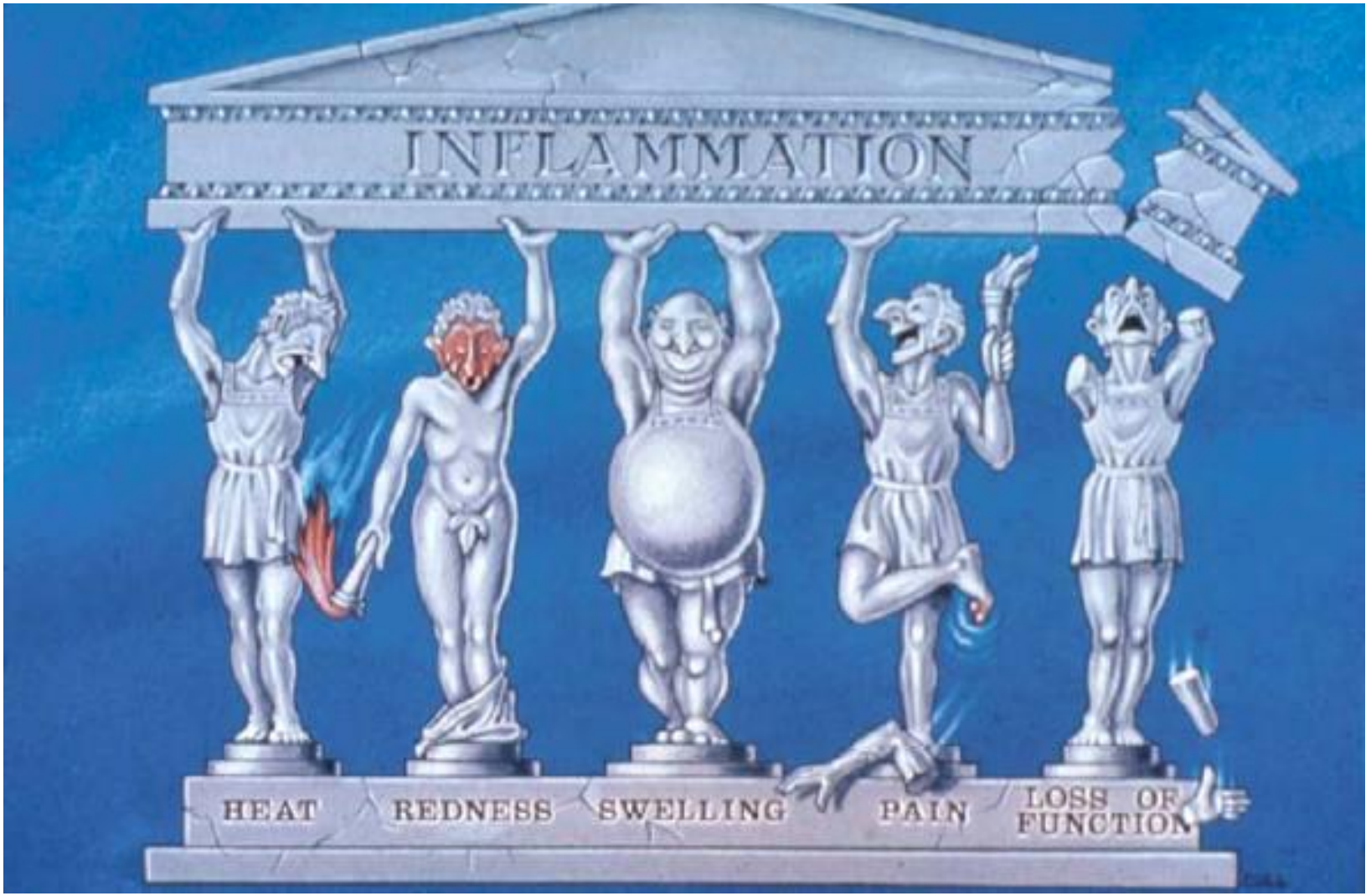
INFLAMMATION



Inflammation

- Protective response that is intended to eliminate the initial cause of injury
- Innate and acquired mechanisms
- Local or generalized (sepsis) processes
- Terminology: ~ itis ending

Etiology

- Physical effects:
  - Extreme temperature, electric shock, ionization, physical injury, etc.
- Chemical agents:
  - Metabolic substances, acids, alkalis drugs, tissue necrosis
- Microorganisms:
  - Bacteria, viruses, fungal infections, parasites, immune cells and immunocomplexes

Types of inflammation

- acute
  - Duration: few minutes to few days
  - Exsudate rich in fluid and plasma proteins and mainly neutrophil granulocytes
- subacute
  - Transition between acute and chronic inflammation
  - Mixed cellular infiltration
- chronic
  - Duration: few days to years
  - Lymphocytes, macrophages, vascular proliferation and fibrosis

Signs and symptoms

- **Rubor** (redness): arteriole dilatation + venous outflow impairment→ dilation of capillary loops, stasis
- **Calor** (warm)
- **Dolor** (painful): vasodilatative and permeability increasing substances
- **Tumor**: increased fluid outflow

  - exsudate (inflammation, protein > 3 mg/ml)

  - transsudate (stasis, increased hydrostatic pressure)
- **Functio laesa**

Outcome of acute inflammation

- Resolution – regeneration, reparation
- Secondary infections
- Hematogenous spread - sepsis
- Development of chronic inflammation
- Scarring - fibrosis

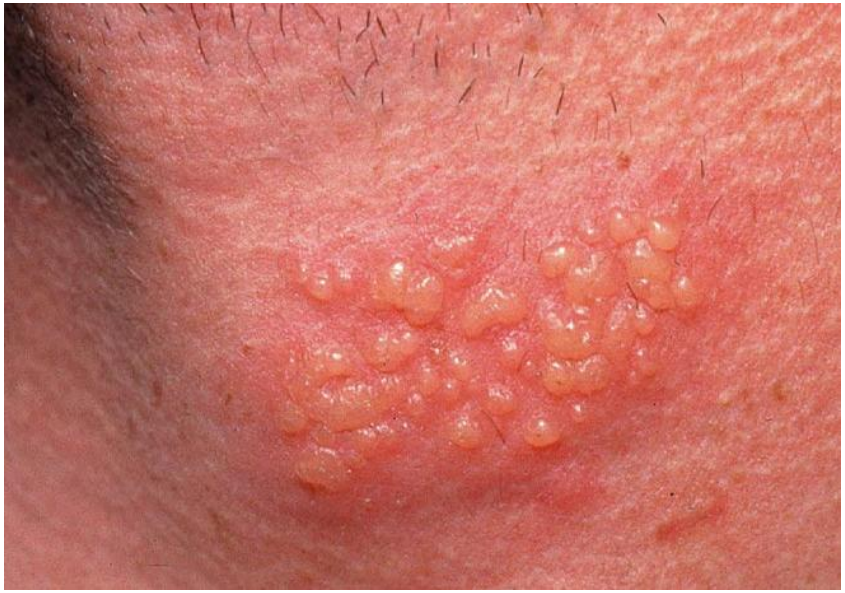
Morphologic patterns of acute inflammation

SEROUS

- mildest
- might be first stage of inflammation, mild increase in permeability, outpouring of a watery, relatively protein-poor fluid
- First stage of common cold, skin burns (bullae), erysipelas, herpes (first stage).
- Serous membranes:

exsudative

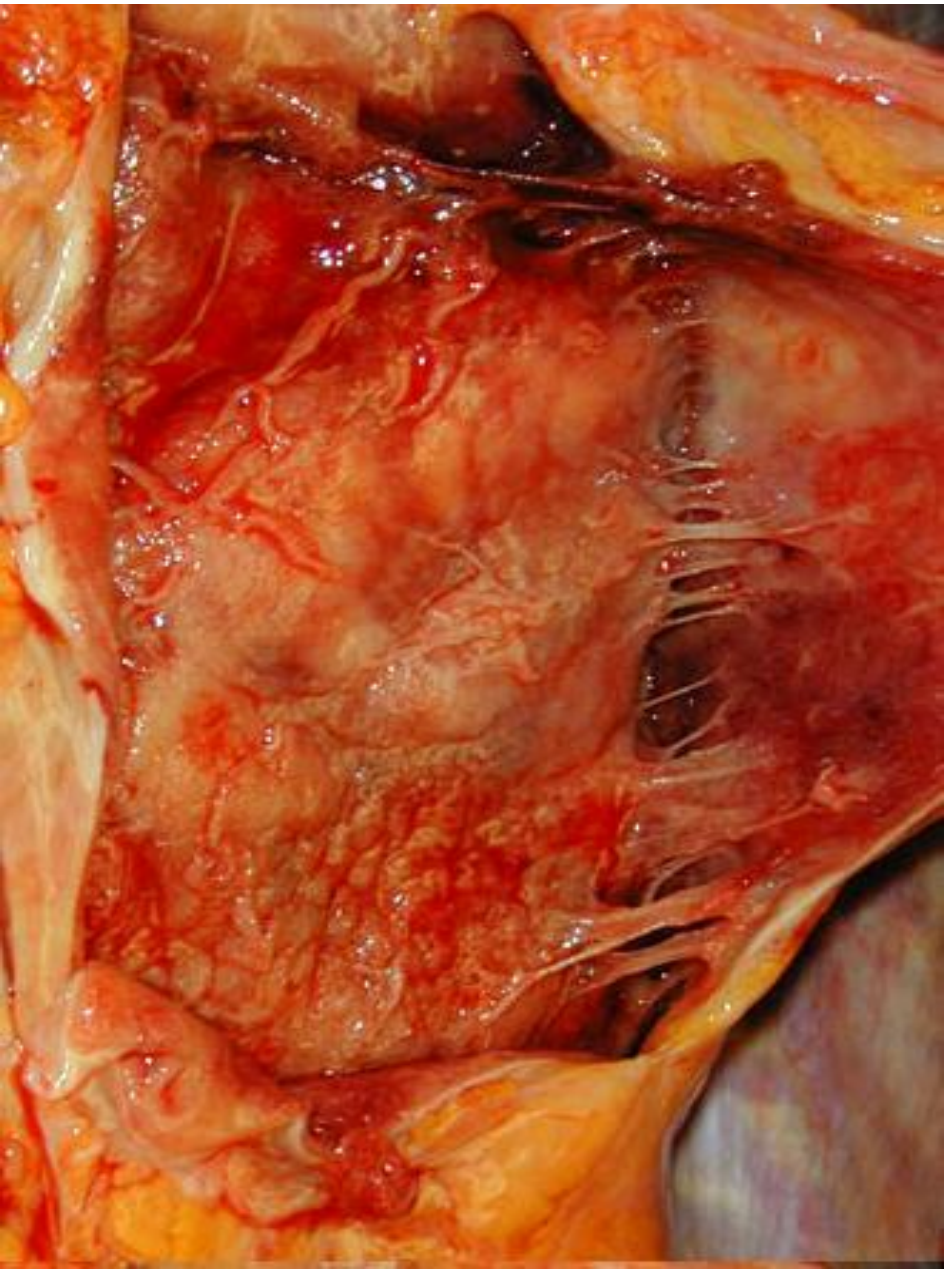
pleuritis/pericarditis/peritonitis

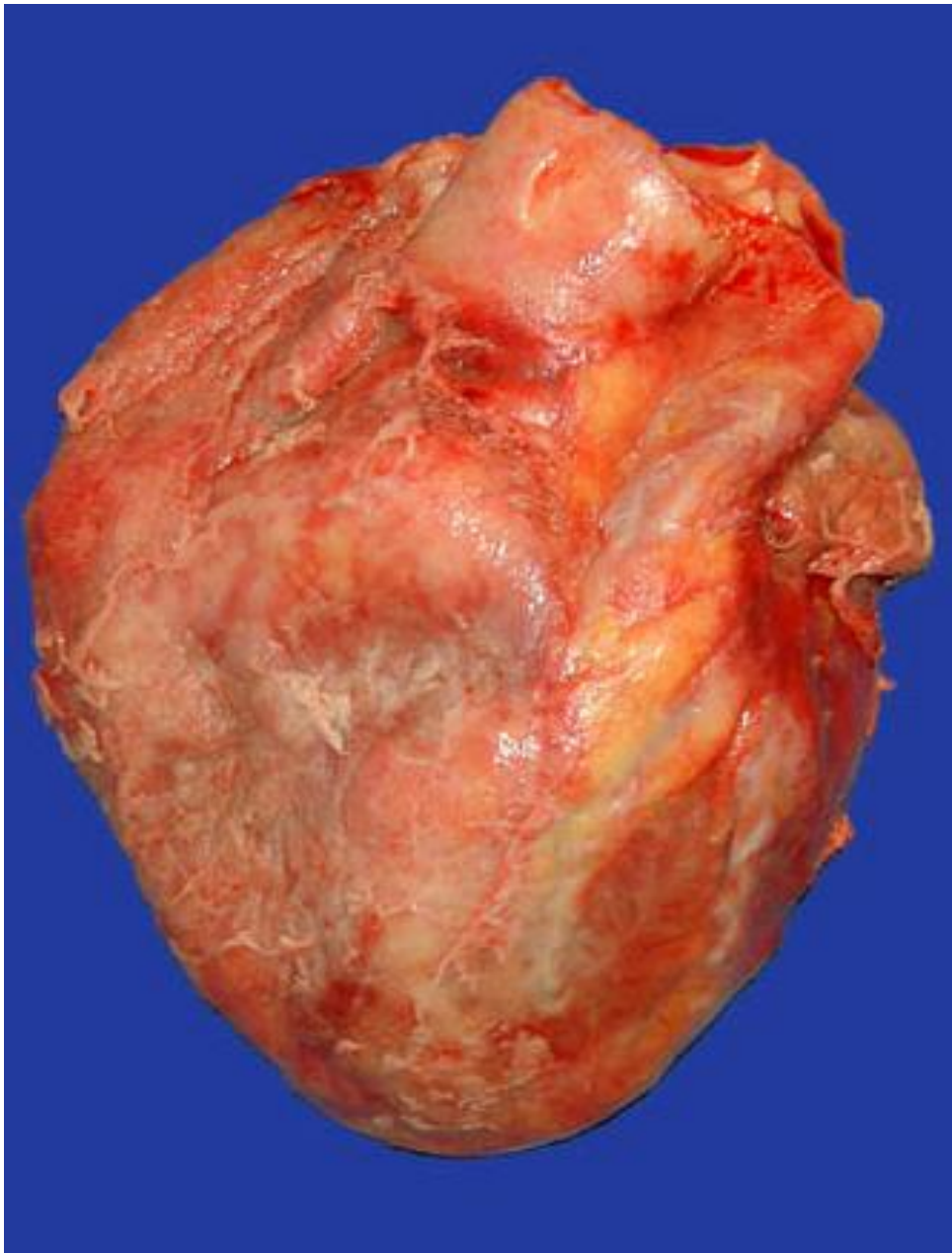


FIBRINOUS

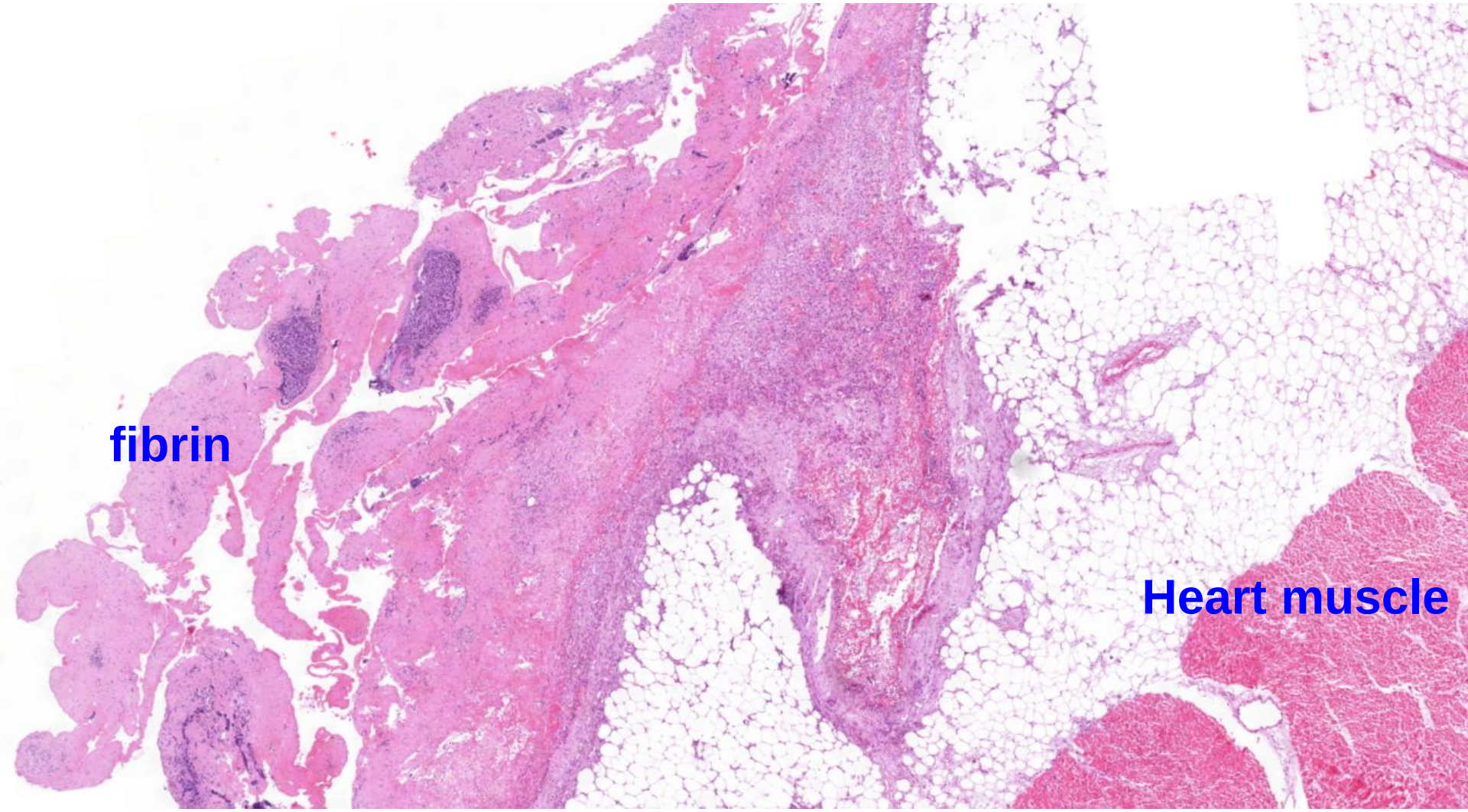
- greater vascular permeability that allows large molecules (such as fibrinogen) to pass the endothelial barrier.→ extravascular fibrin
- Greyish, sticky exsudate, which is removable
- **Pericarditis sicca**: friction rub; uremia (cor villosum/'bread and butter')
- **Fibrinous pleuritis**: painful, above lung infarction, diffusely as a complication of pneumonia
- **Fibrinous peritonitis**

Fibrinous pericarditis



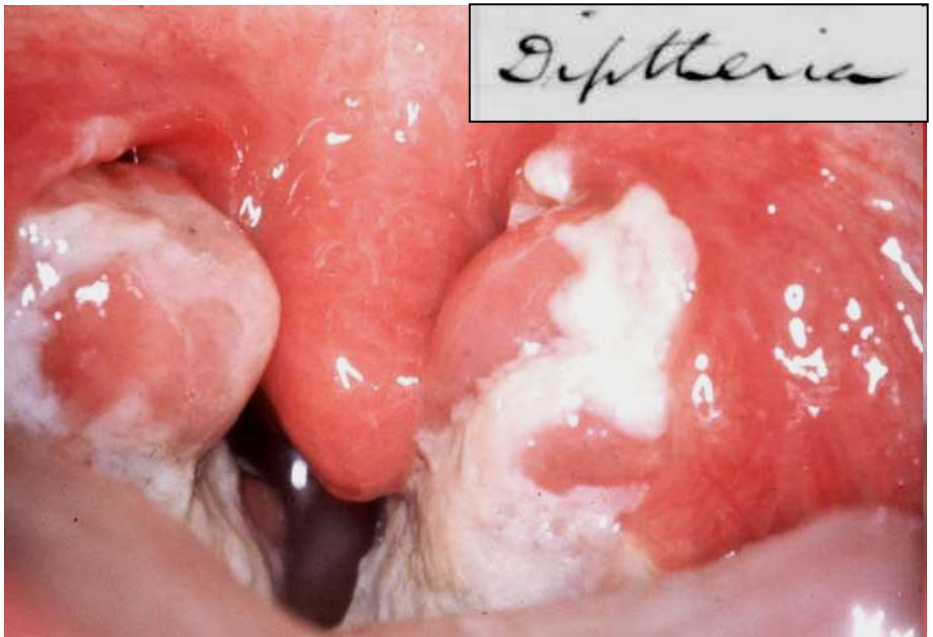


Fibrinous pericarditis

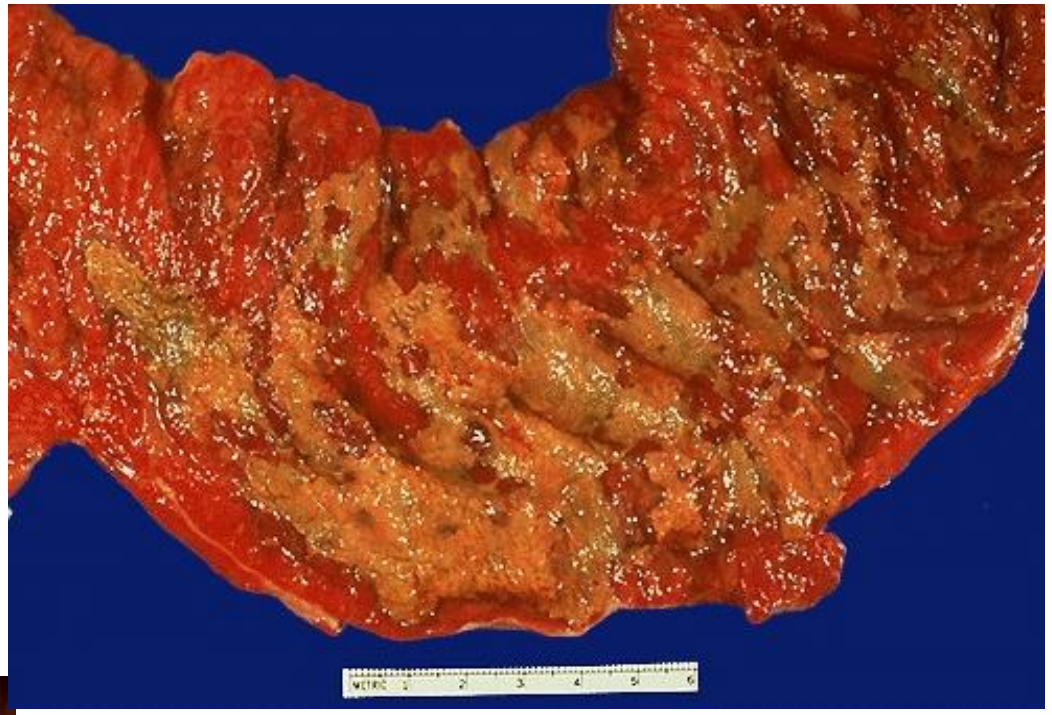


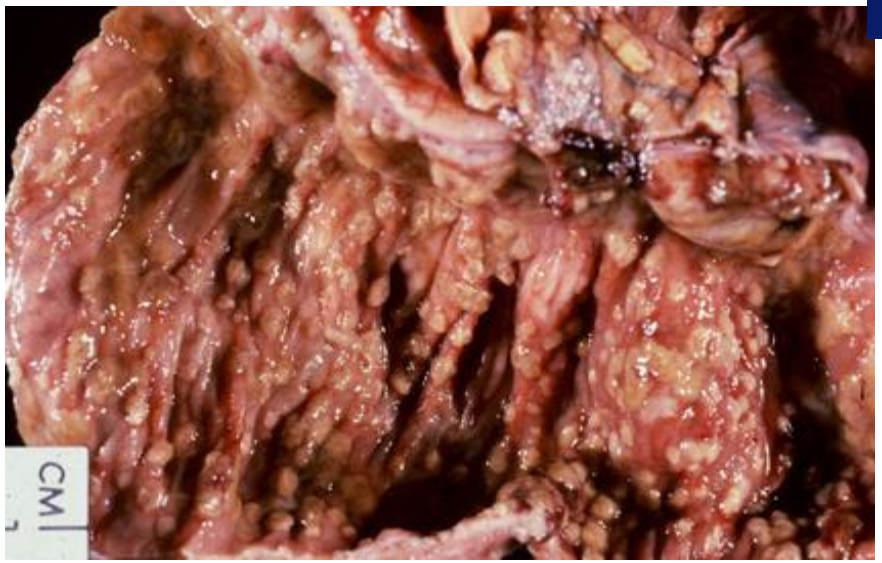
Mucosal membranes

- pseudomembranous inflammation;
- Fibrin is easily removable – diphteria
- Diptheritis (fibrin is not easily removable, the inflammation affects the submucosa as well, ulcer formation)
- typhus abdominalis, Shigella, pseudomembranous colitis (C. difficile).
- fibrin – resolution → healing/organization → chr. fibrous adhesions (filamentous or lamellar).



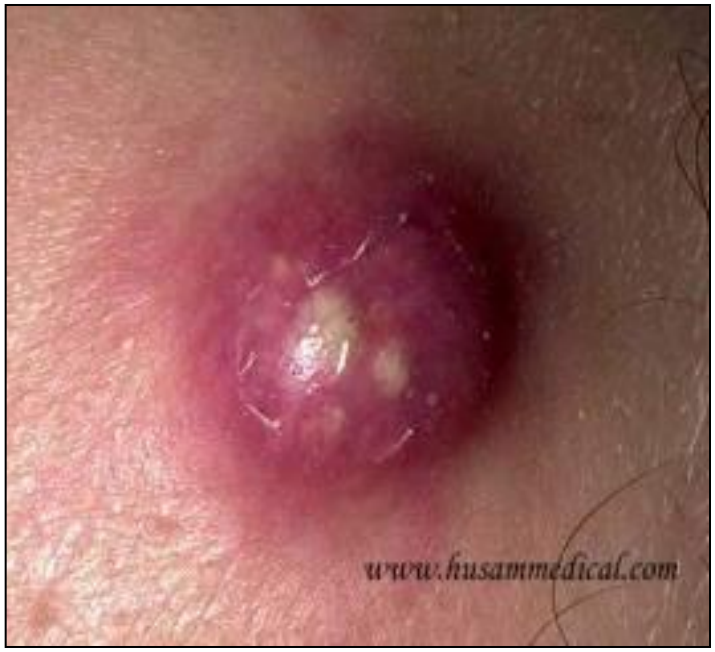
Pseudomembranous colitis





PURULENT

- Pus: neutrophyl granulocytes + necrotic cells and cell debris
- Folliculitis, furuncule, carbuncule, pustule (blisters filled with pus in the epidermis, purulent meningitis
- **Abscess:** Localised form of acute purulent inflammation forming a pus filled cavity
- **Empyema:** Pus in a preformed cavity (pyocephalus, pyometra, pyosalpinx, pyonephros…)
- **Phlegmon:** Diffuse form of acute purulent inflammation, spreading within the tissues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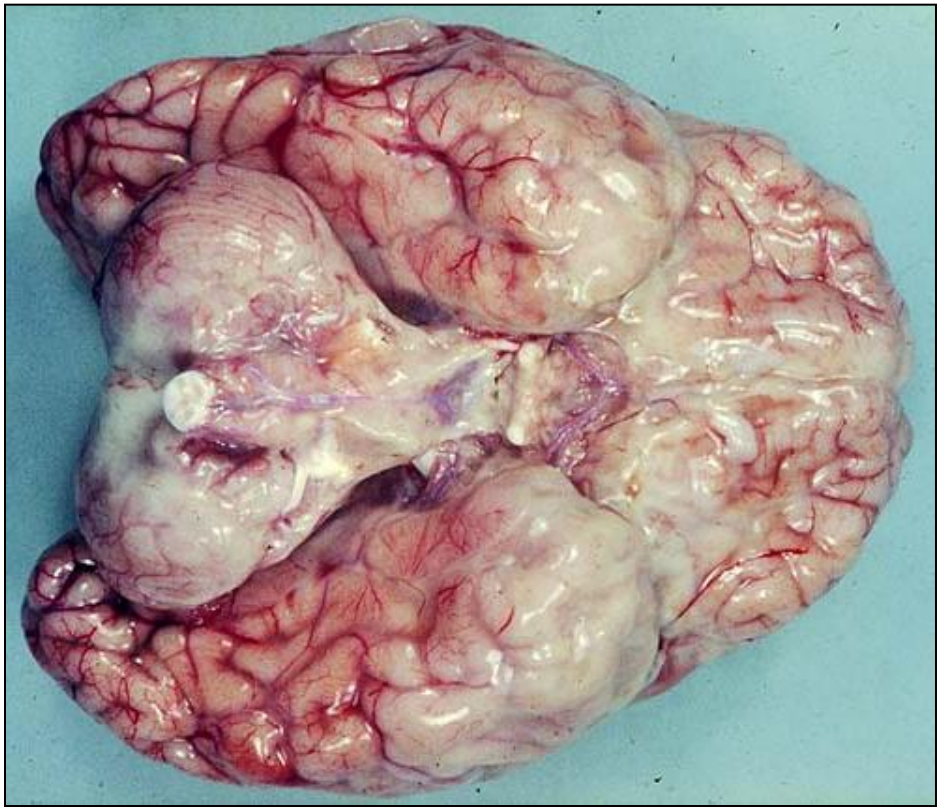
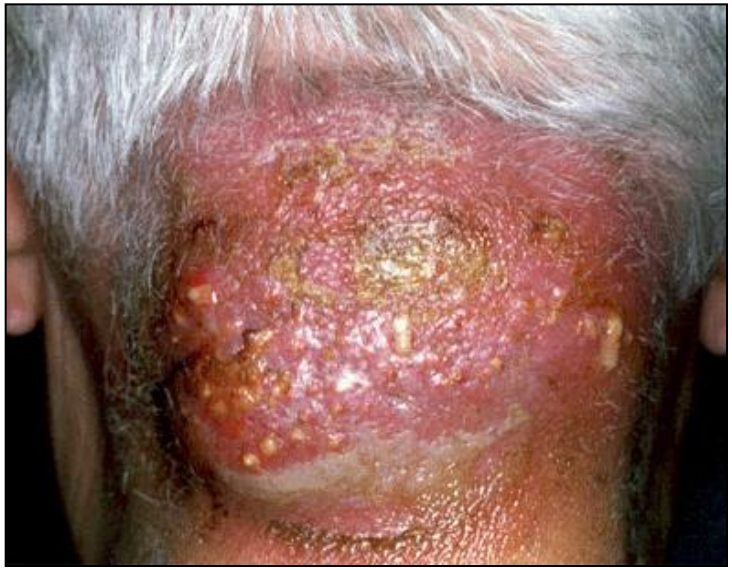
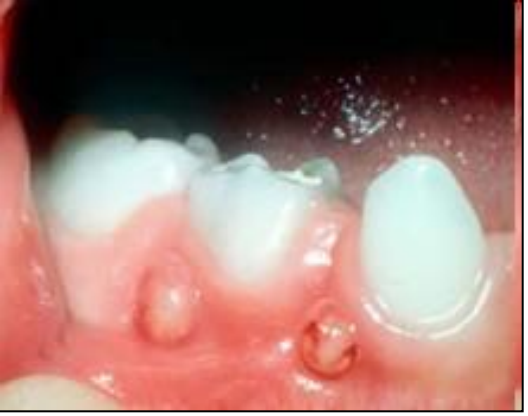
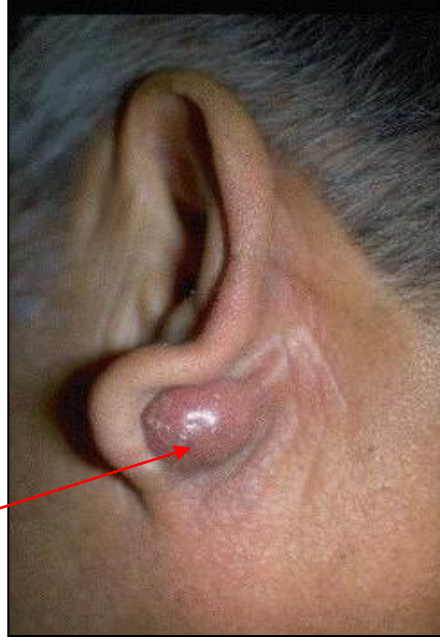


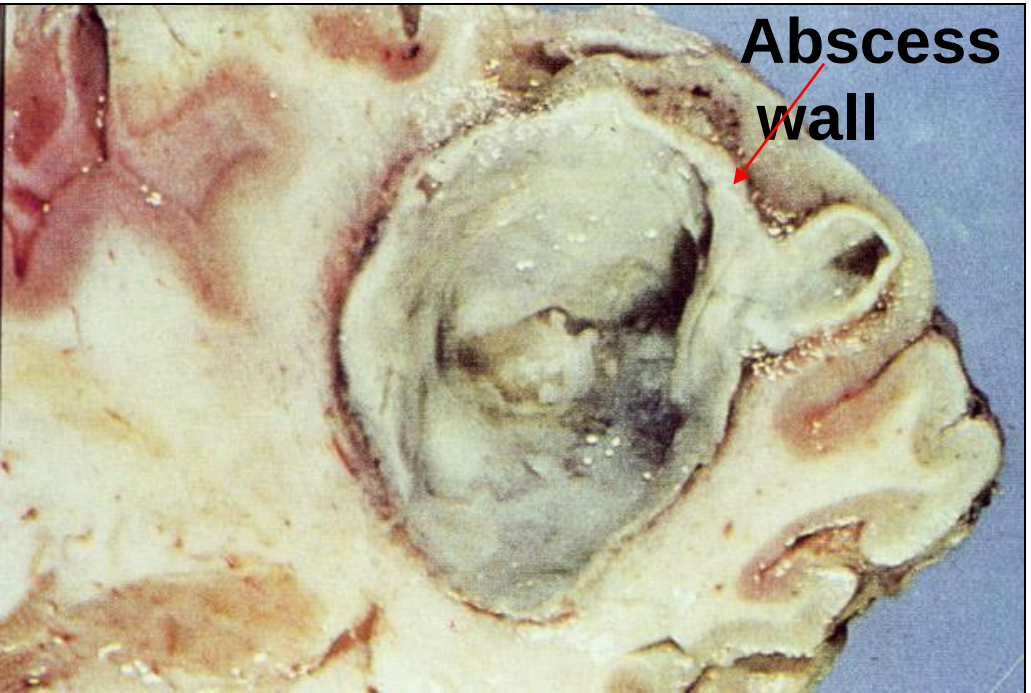


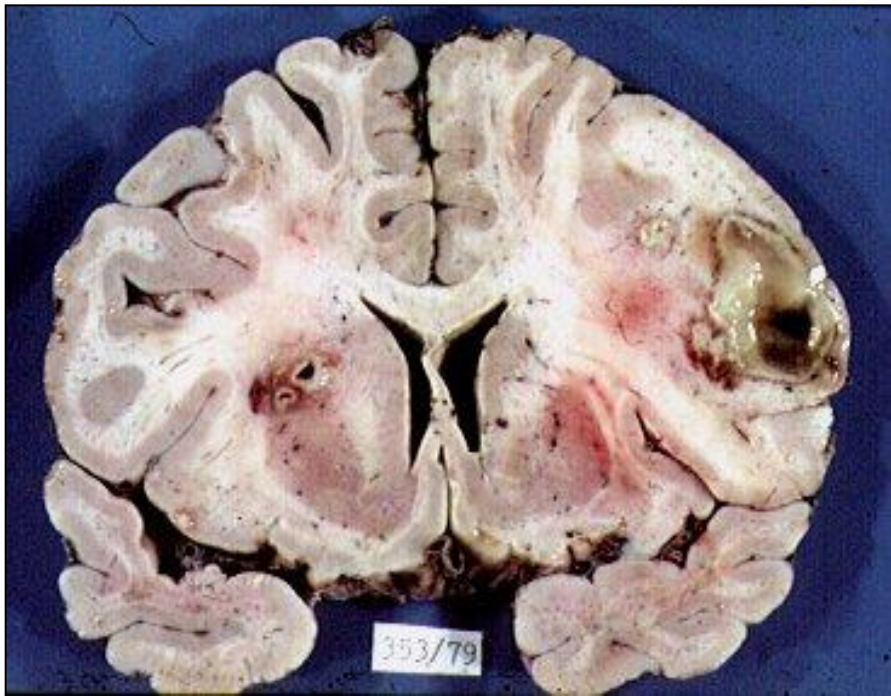


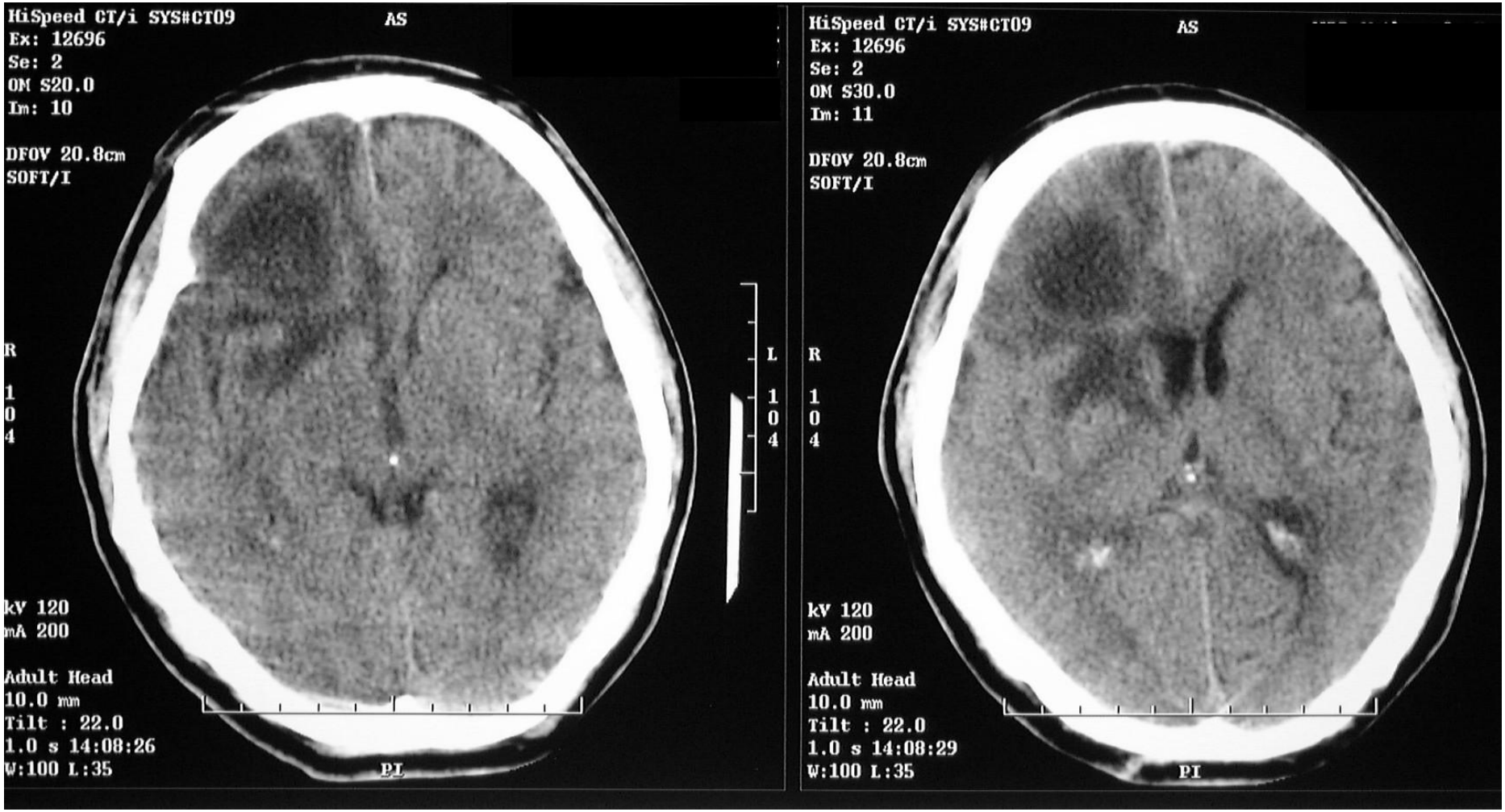


Retroauricular abscess

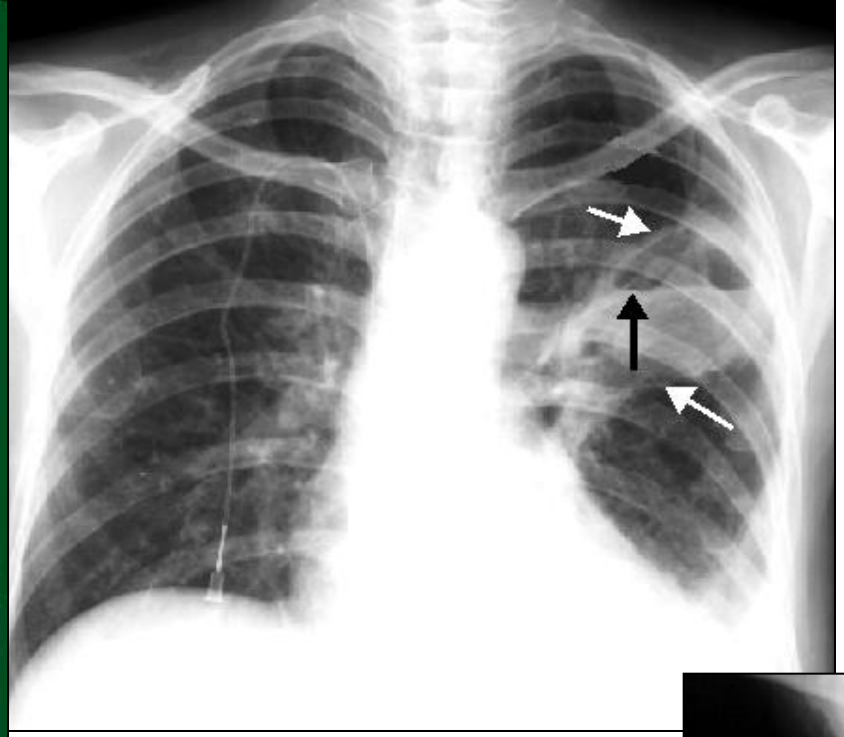
Brain abscess

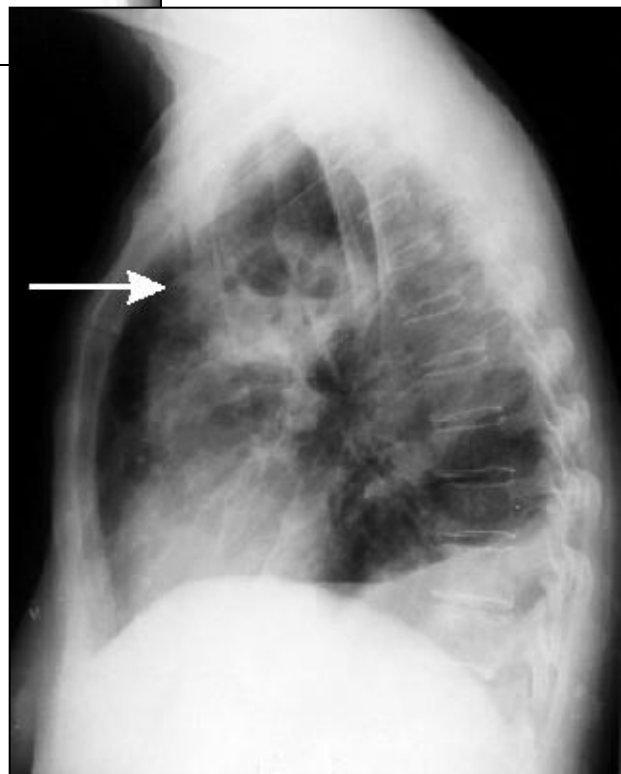




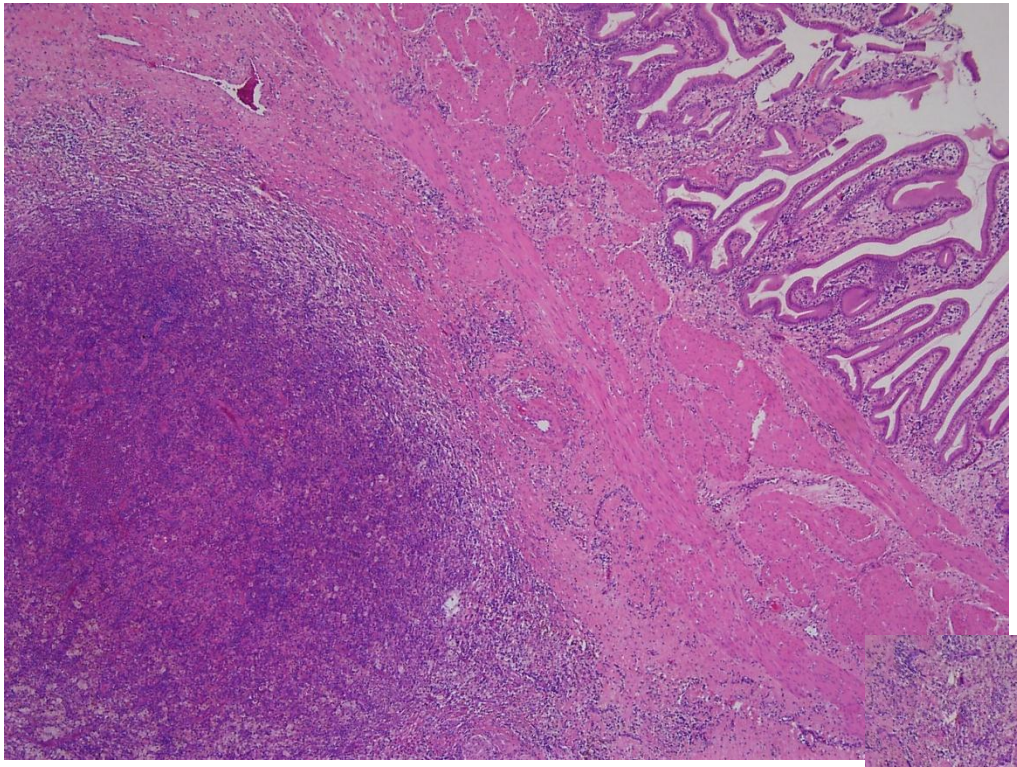


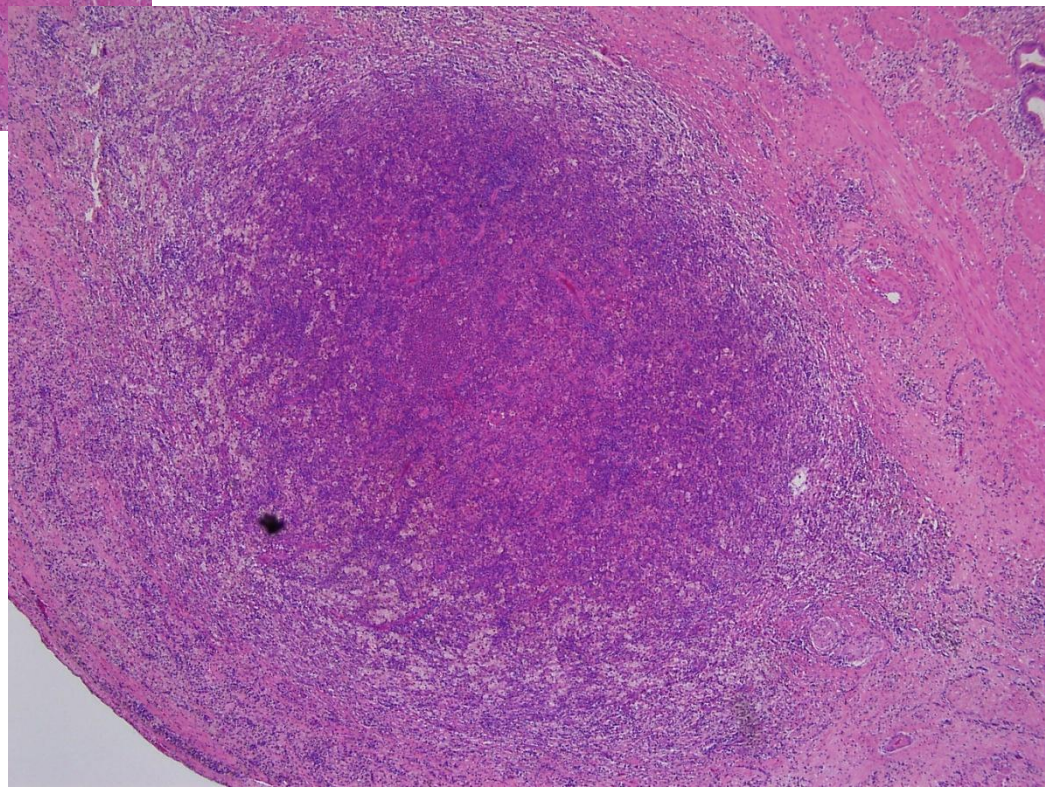
Brain abscess





Lung abscess

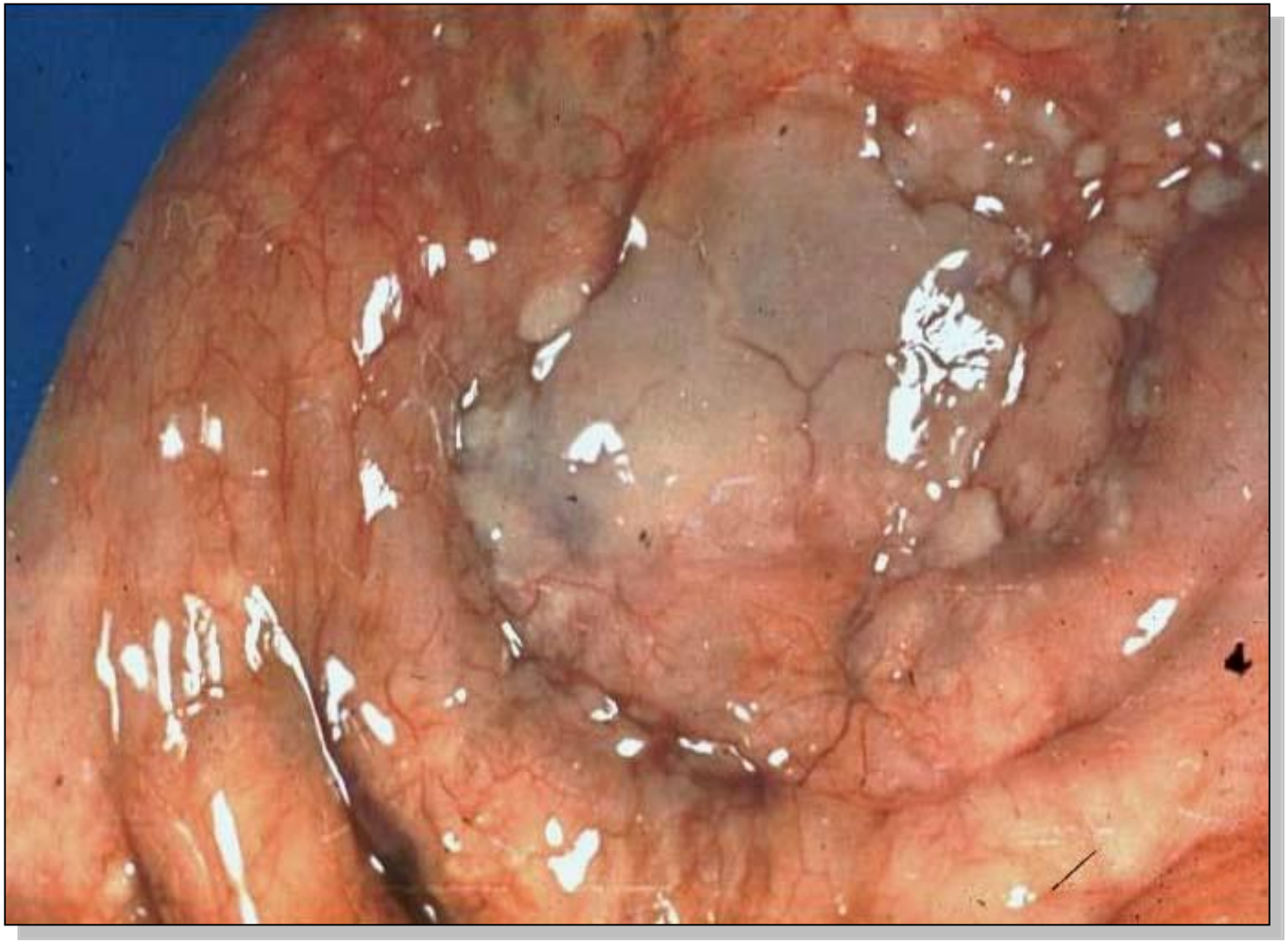




Abscess in the wall of the gallbladder

Phlegmon:

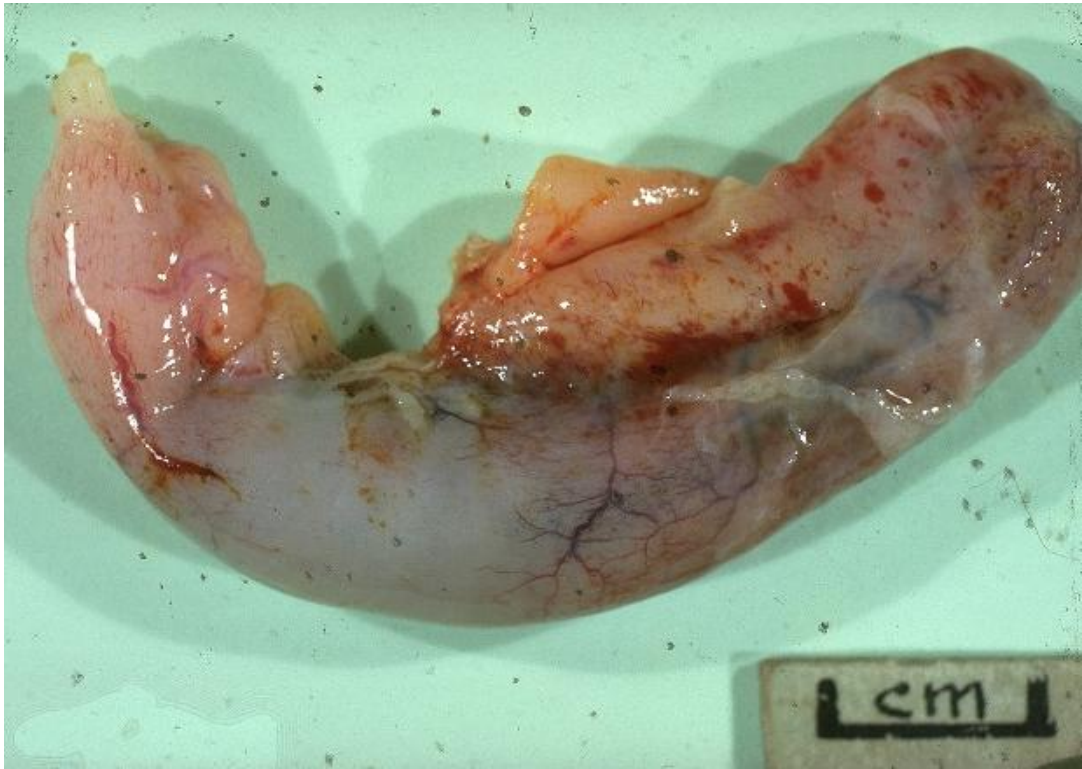
Diffuse form of acute purulent inflammation, spreading within the tissues (urinphlegmone)

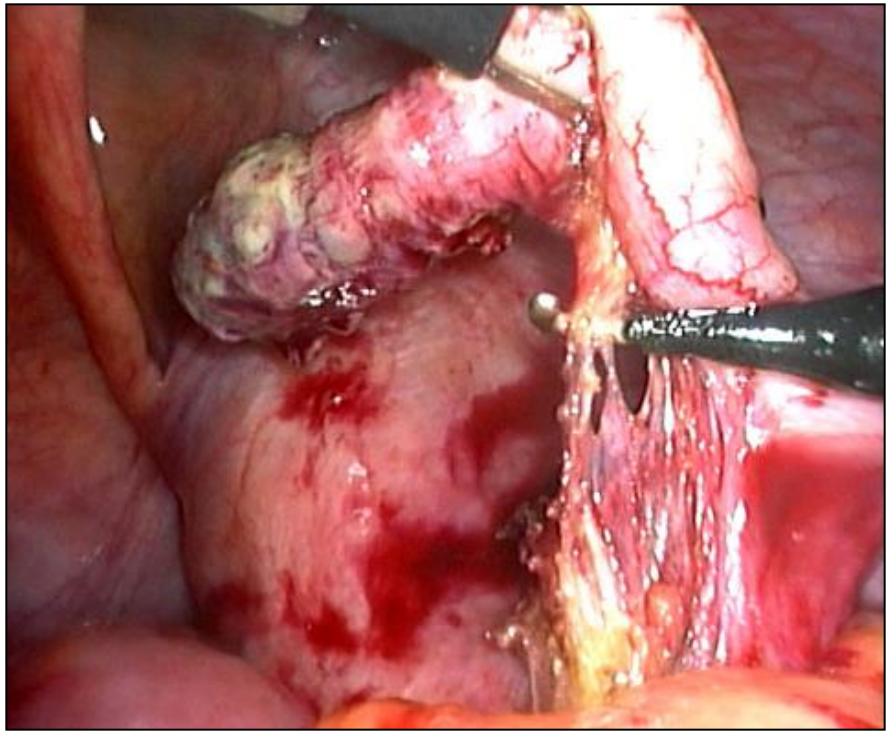


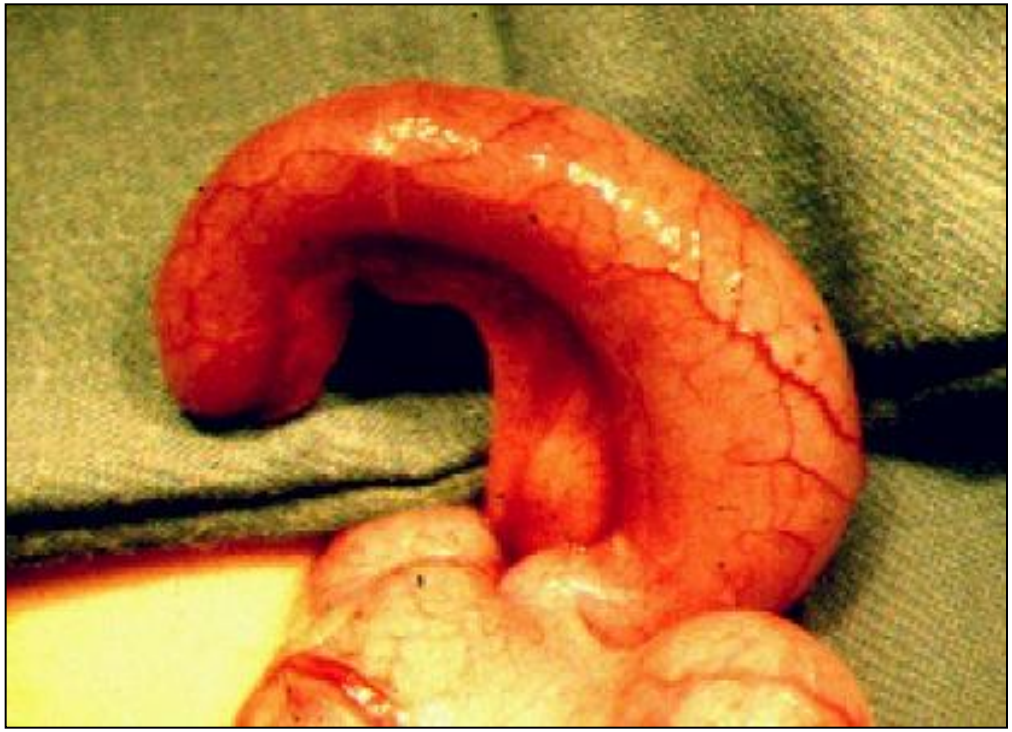
ACUTE APPENDICITIS

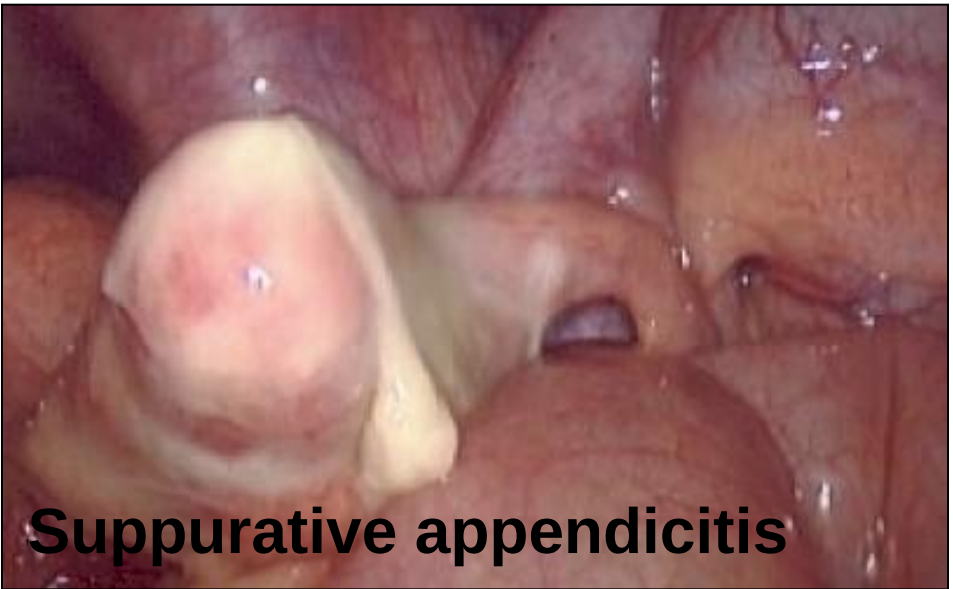
Differential diagnosis – acute abdomen:
- Meckel-diverticulitis
- Crohn’s disease
- salpingitis
- rupture of corpus hemorrhagicum
- mesenterial lymphadenitis
- extrauterine gravidity
- vulvulus, intussusception

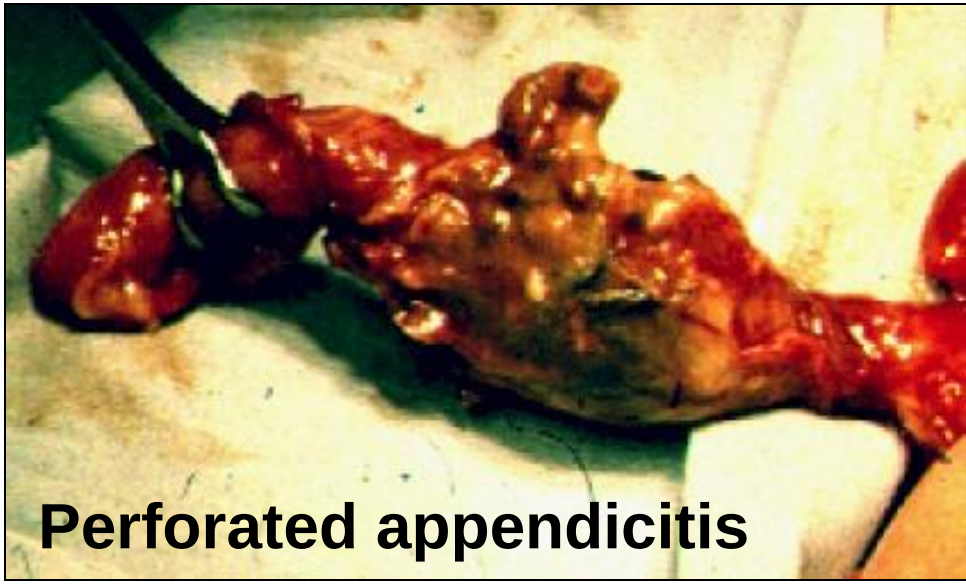
in children

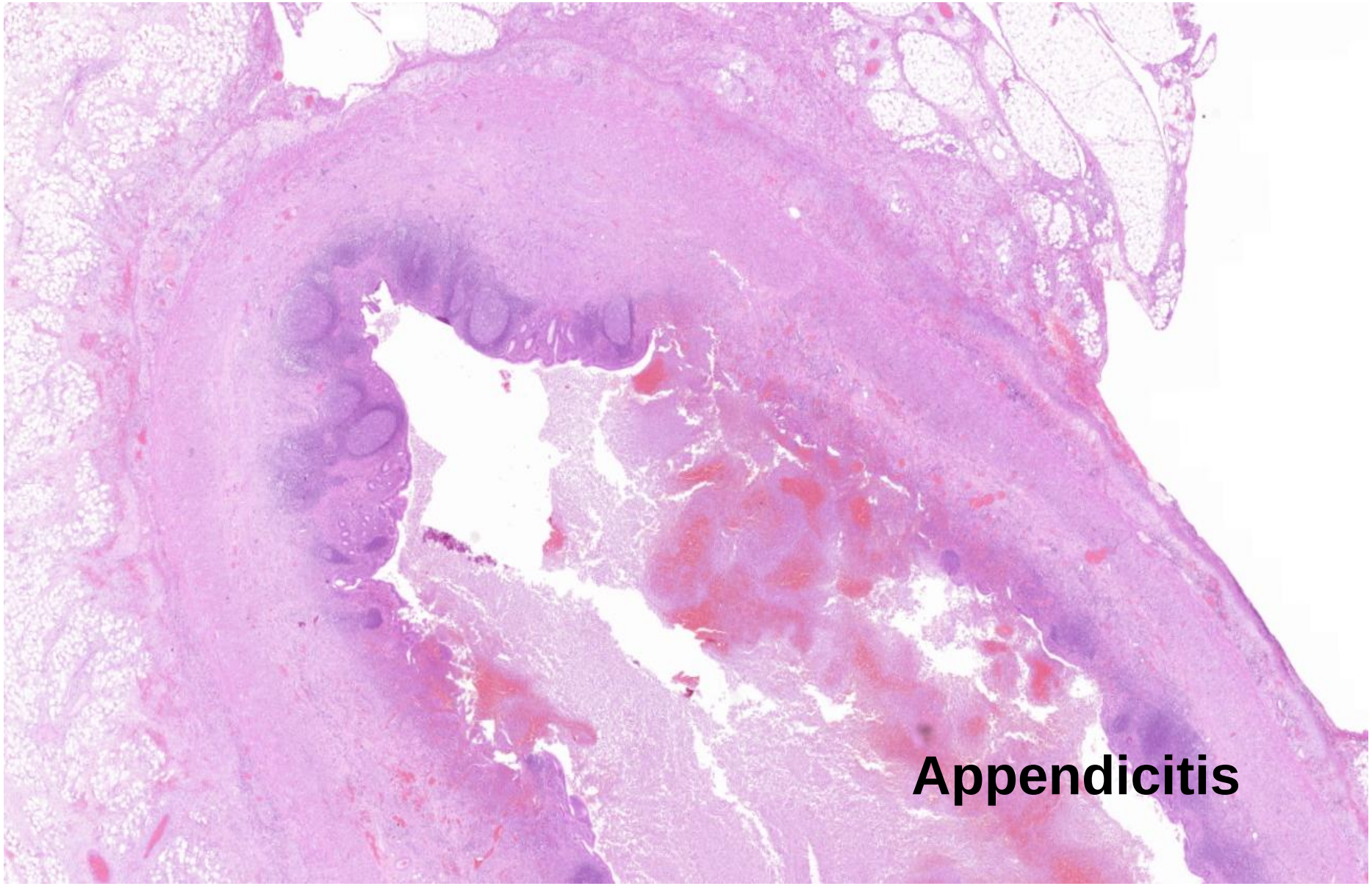


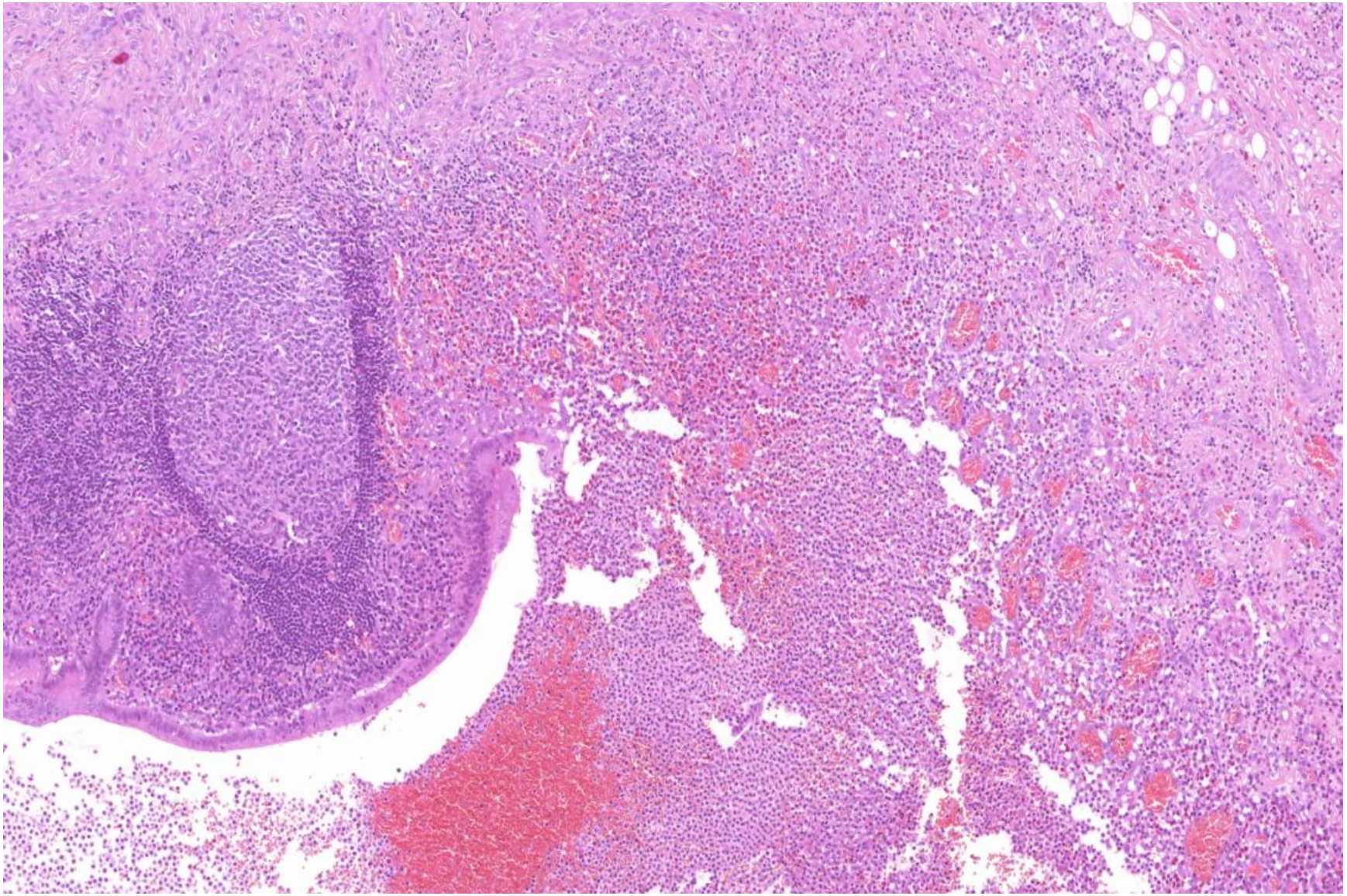


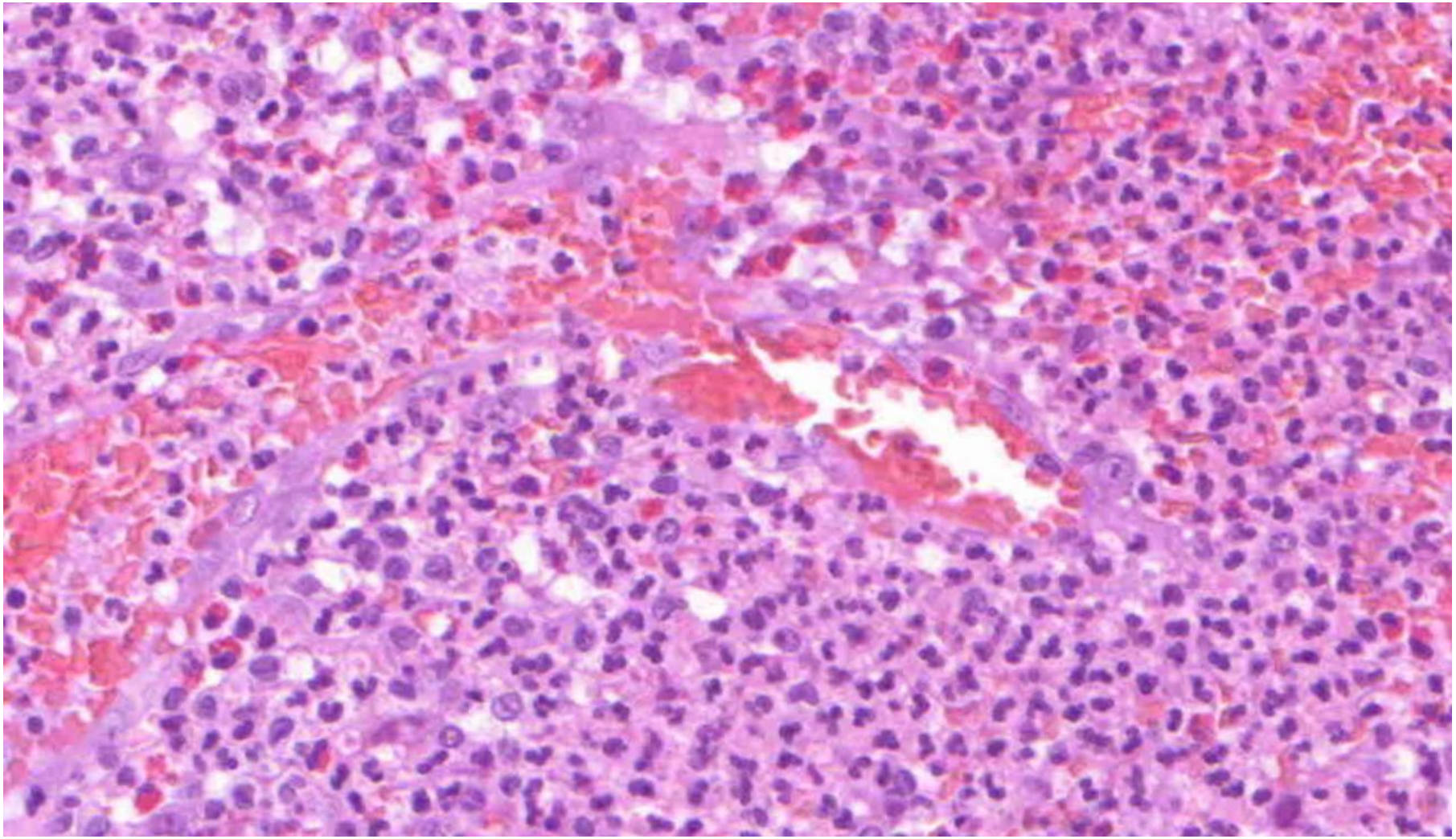



Suppurative appendicitis


Perforated appendicitis

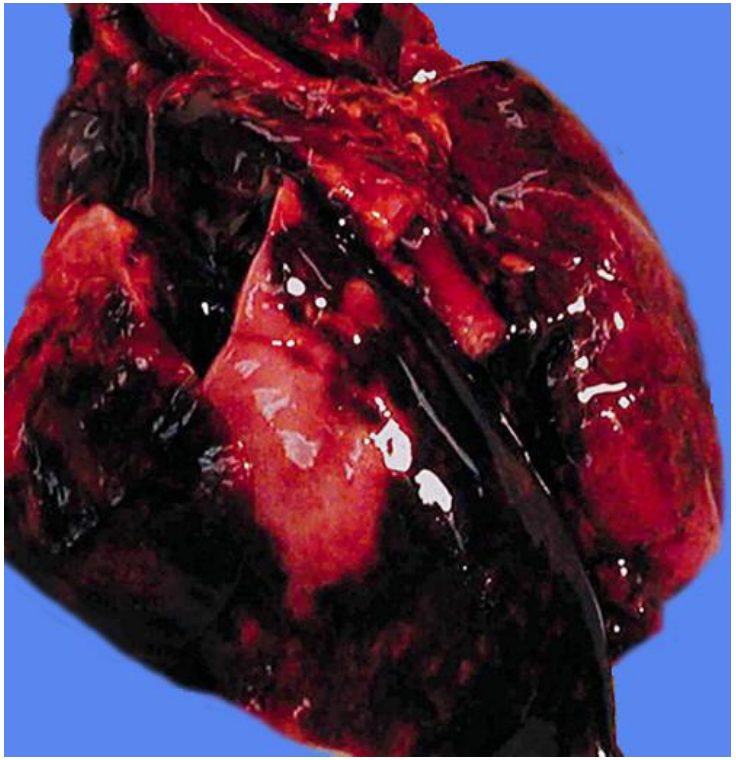

Appendicitis





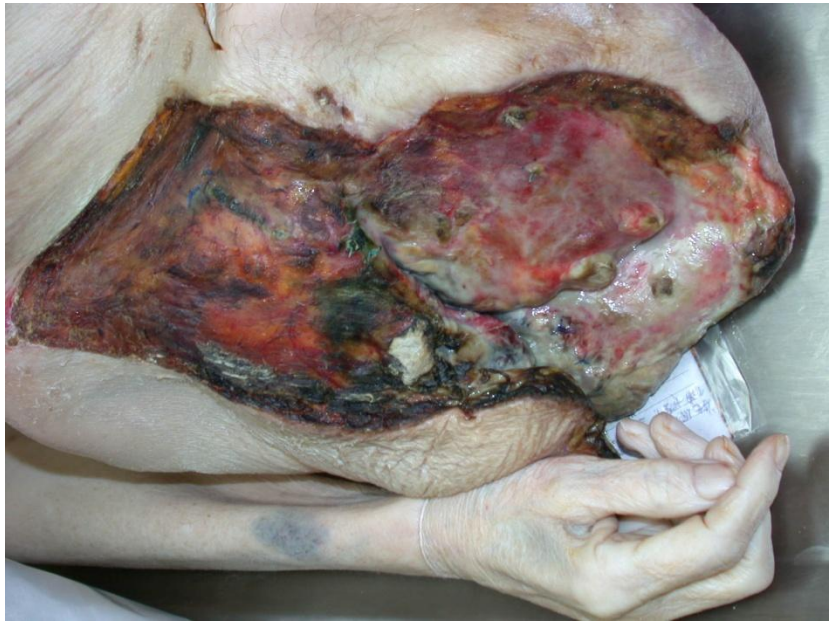
Hemorrhagic:

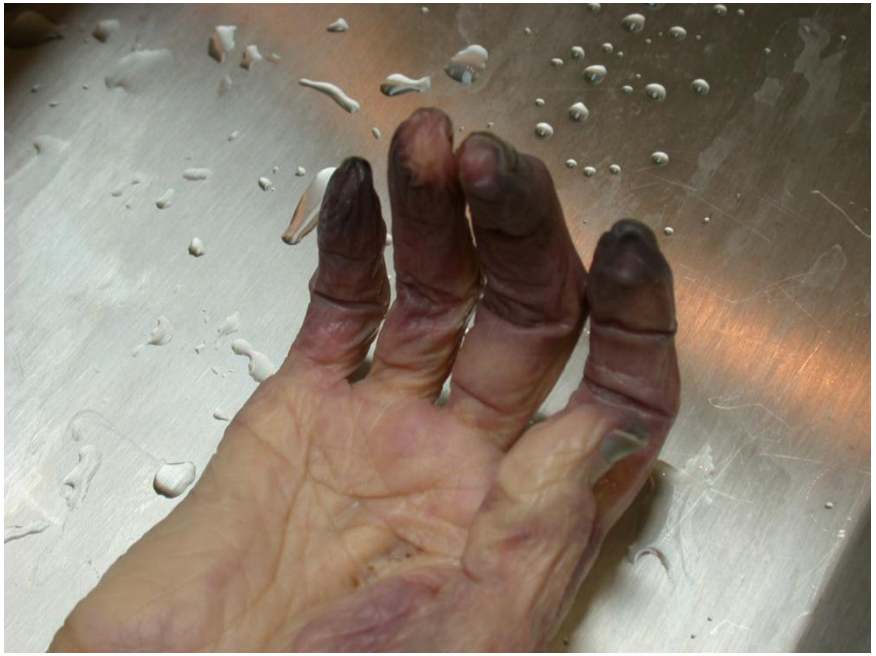
- Extravasation of red blood cells due to vascular damage
- plague, pox, anthrax, influenza, pancreatitis (vessel wall erosion), meningococcus-sepsis (DIC), hemorrhagic urocystitis

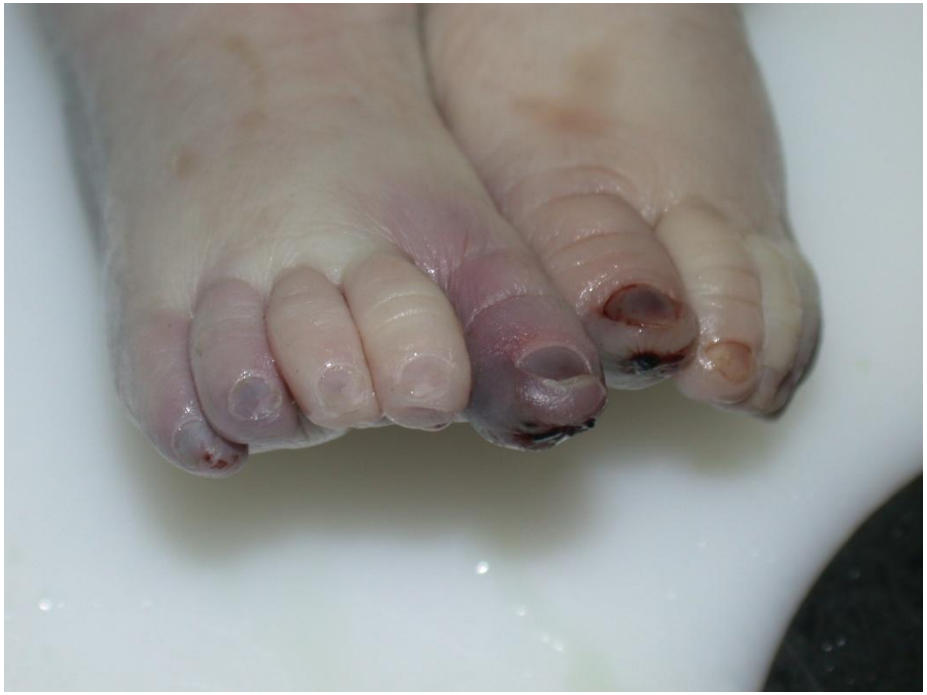


Gangrenous:

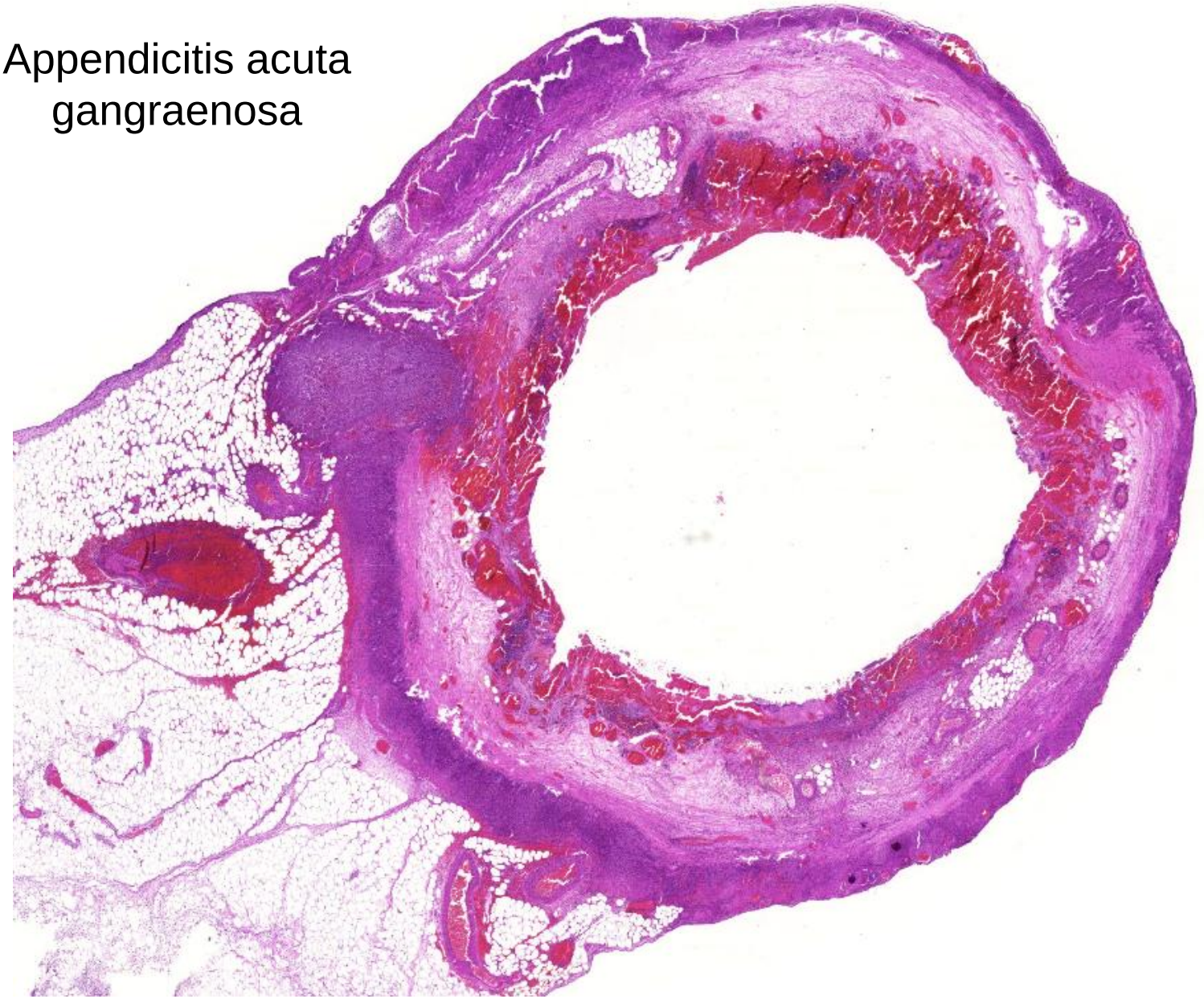
- Immune system is not effective/local hemodynamic disorder
- Extensive tissue damage
- arteriosclerosis, diabetes (arterial occlusion): dry gangrene → superimposed bacterial infection → wet gangrene
- in the lungs after aspiration
- as a complication of tumors
- In healthy individuals: gangrenous appendicitis, gangrenous cholecystitis
- gasgangrene: emphysematous gangrene caused by C. perfringens







Appendicitis acuta gangraenosa



Chronic inflammation

- Mononuclear cell infiltration (lymphocyte, macrophage, plasmacell)
- Tissue damage
- Reparation – angiogenesis and fibrosis

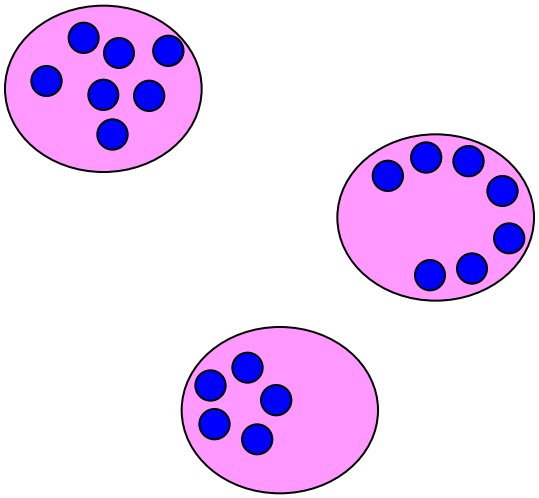
  are seen together

Causes:

- Persistant infection (TB, syphilis)
- Hypersensitivity reactions
- Continuous exposure to toxic agents

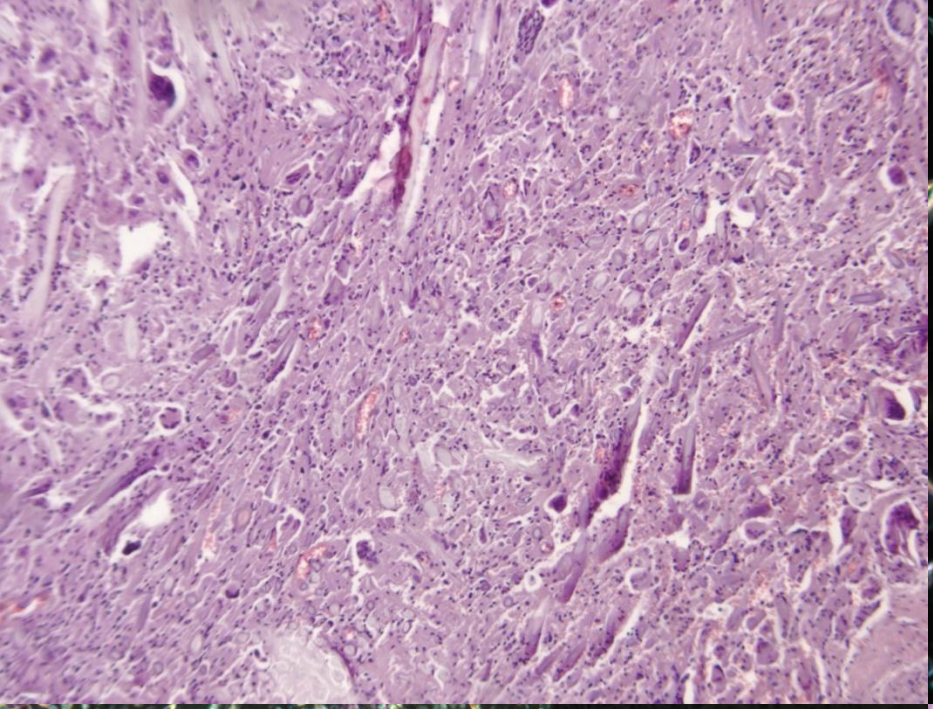
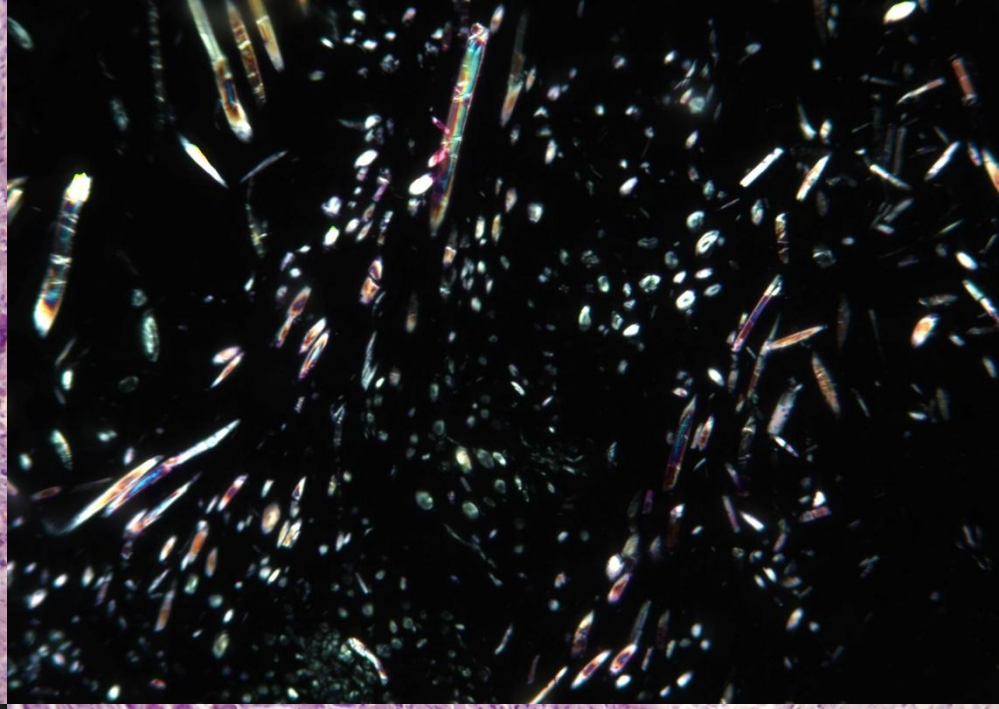
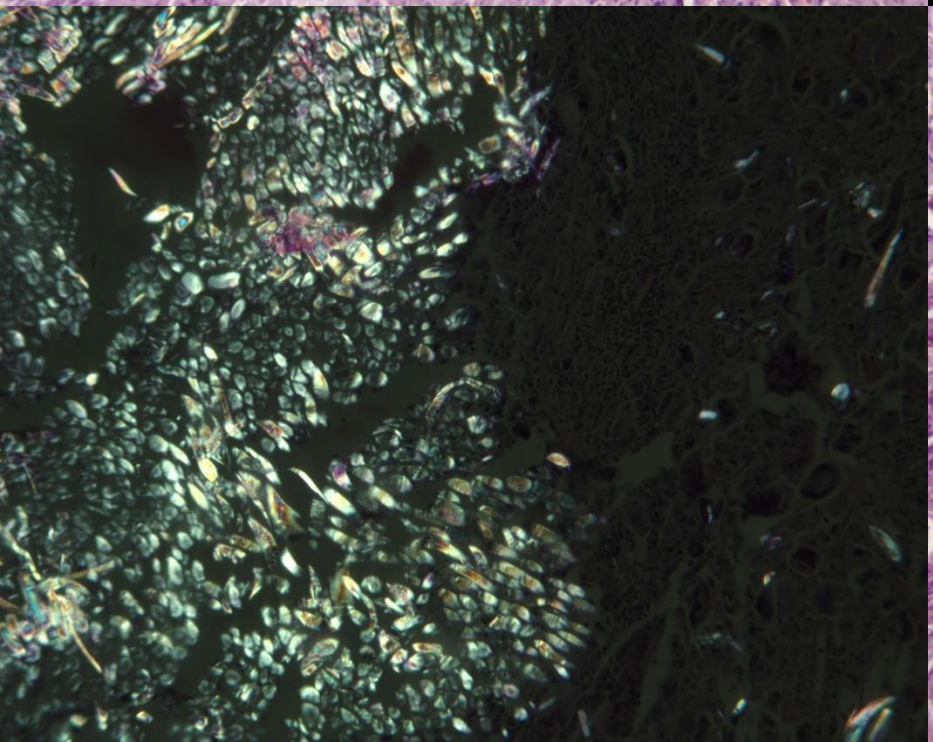
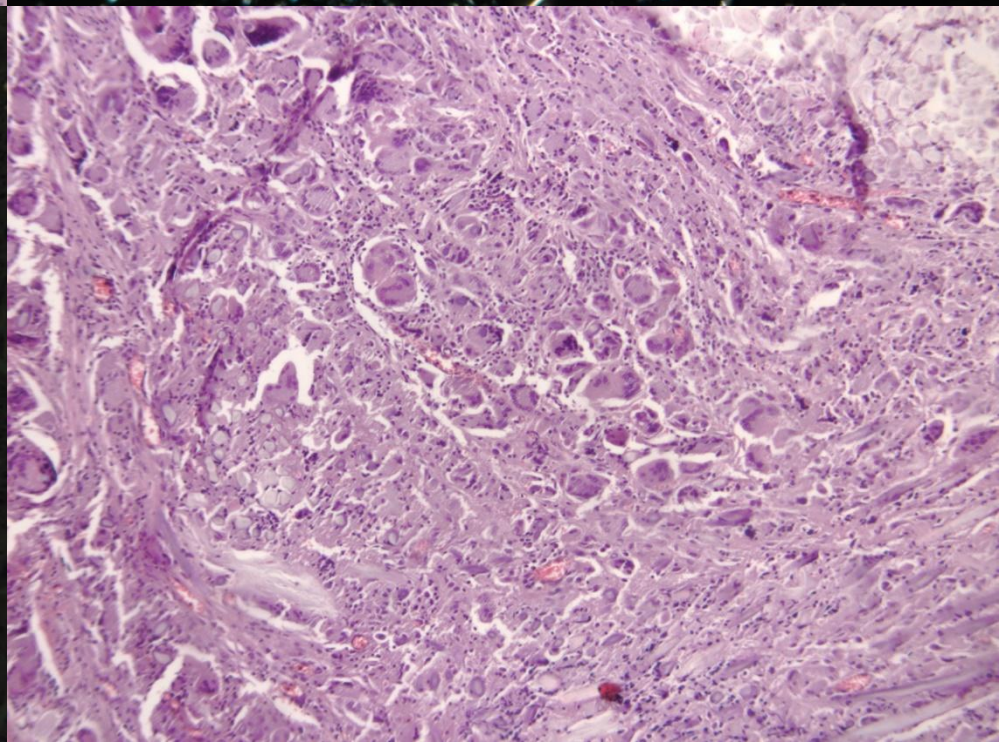
Granulomatous (specific) inflammation:

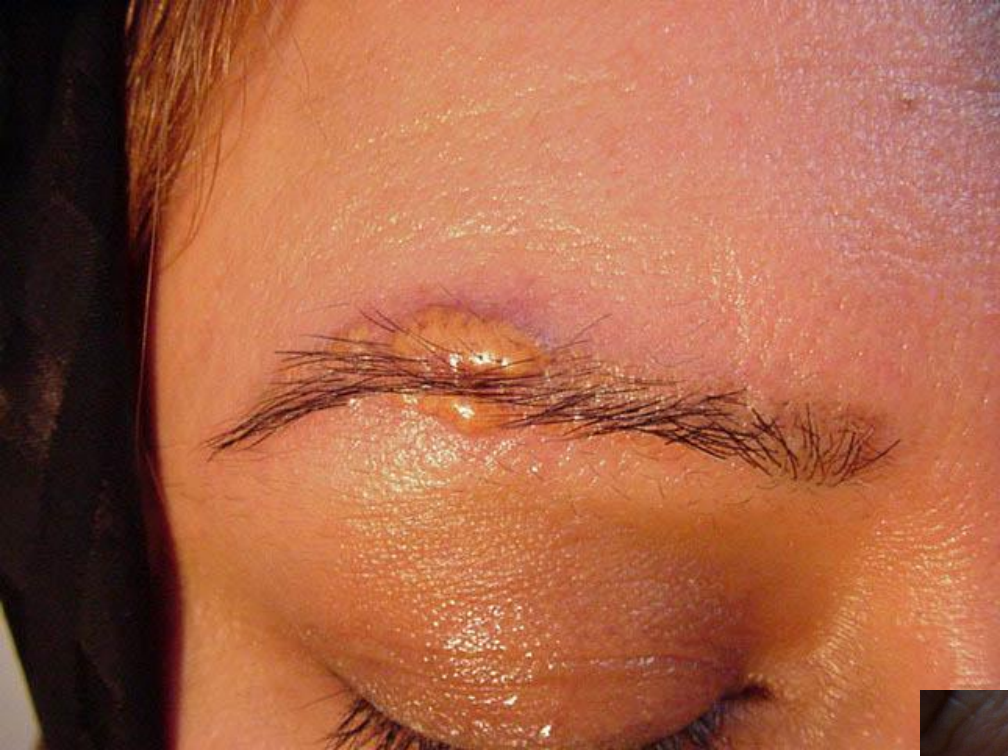
- Macrophages concentrated in the granuloma
- Granuloma: small roundish aggregations of epitheloid macrophages and giant cells
- Multinucleated giant cells (formed by fusion of epitheloid macrophages)
  - foreign body type
  - Langhans-type
  - Touton-type



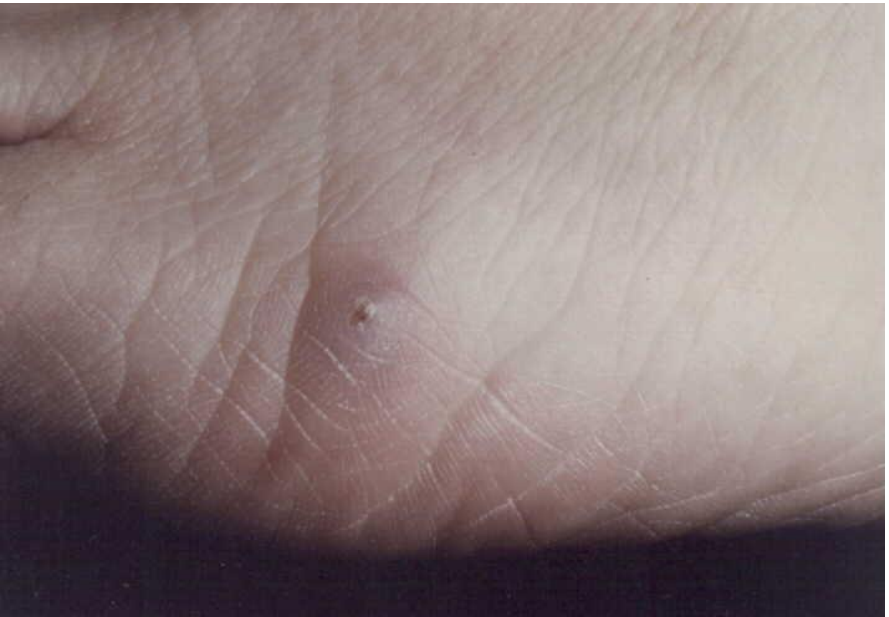
Types of granulomas

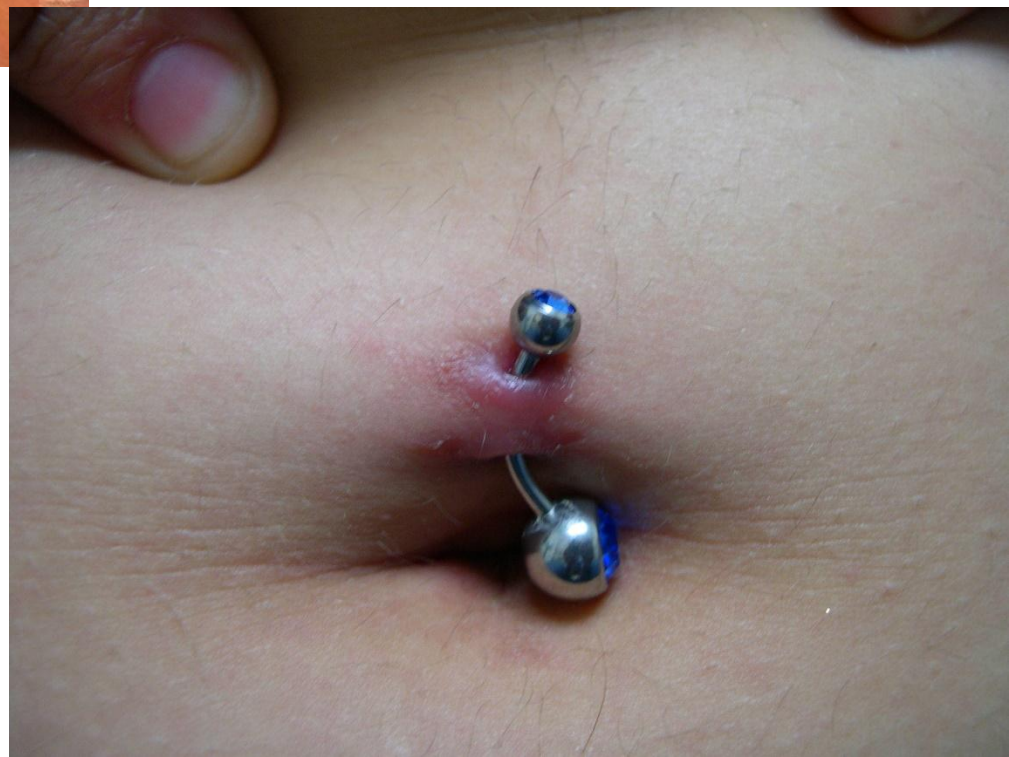
- Immune – infective (tb) - non-infective
- **Foreign body type:** the substance is recognized as foreign, but it does not result in immunoreaction
  - Suture granuloma around stitches, lipid granuloma around fat necrosis
  - Few lymphocytes
  - Hagemann-factor (fXIII) induces inflammation
- Sarcoid granuloma, tuberculosis,
  - Rheumatic granuloma – Aschoff's nodule
  - Rheumatoid granuloma – fibrinoid necrosis

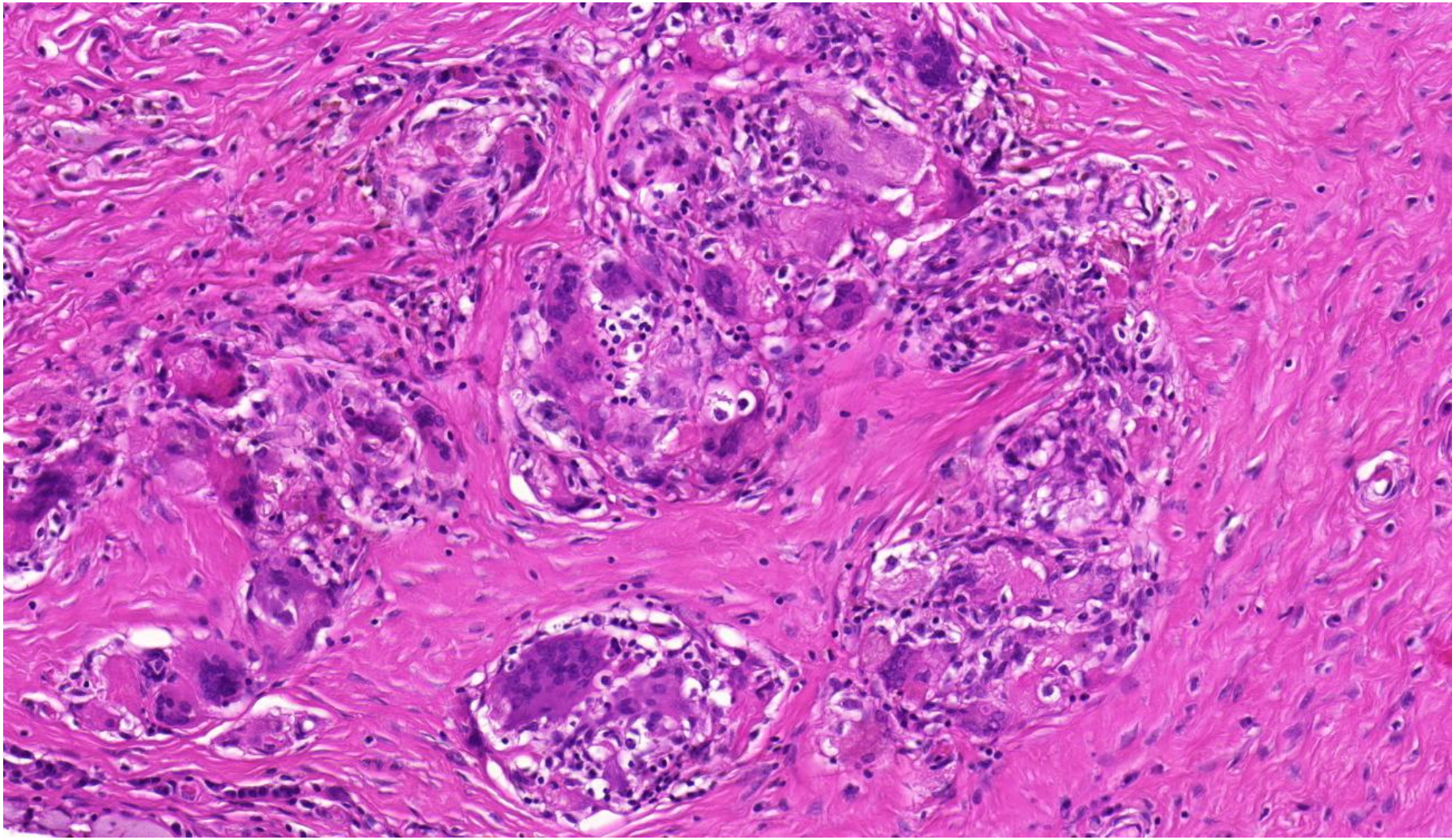







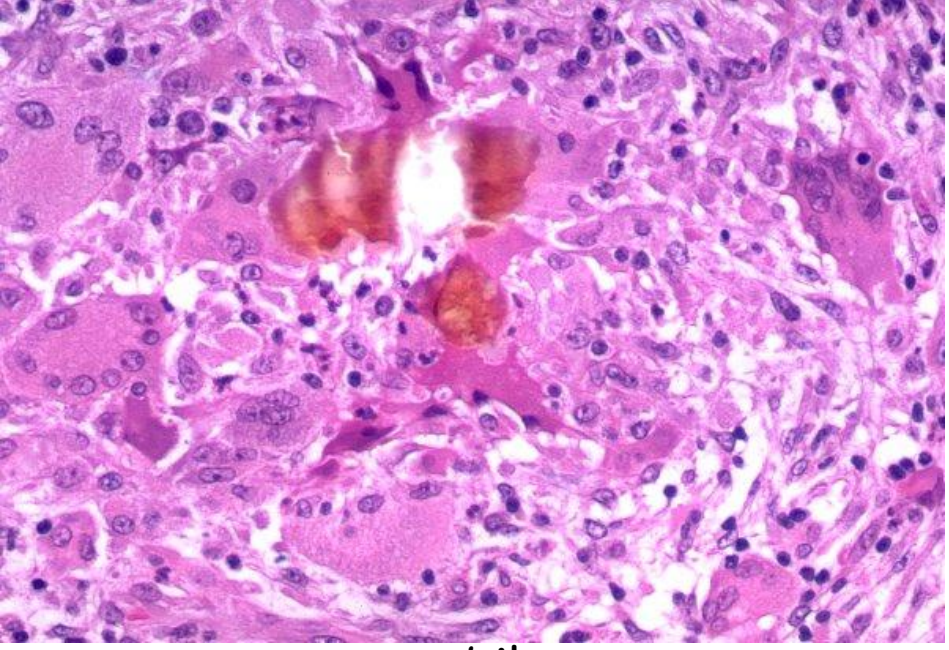
Foreign body granuloma



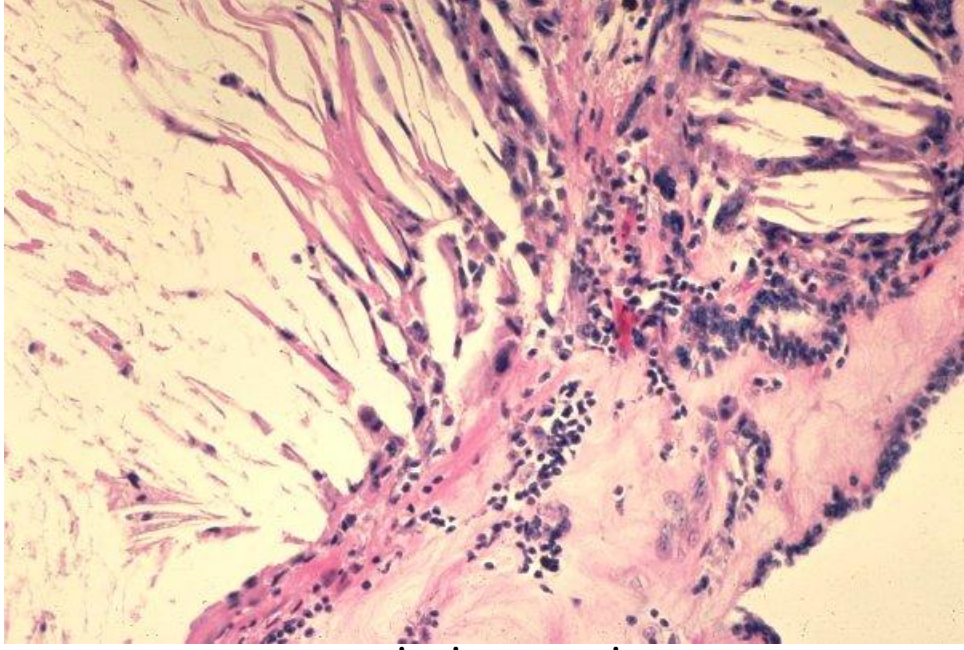




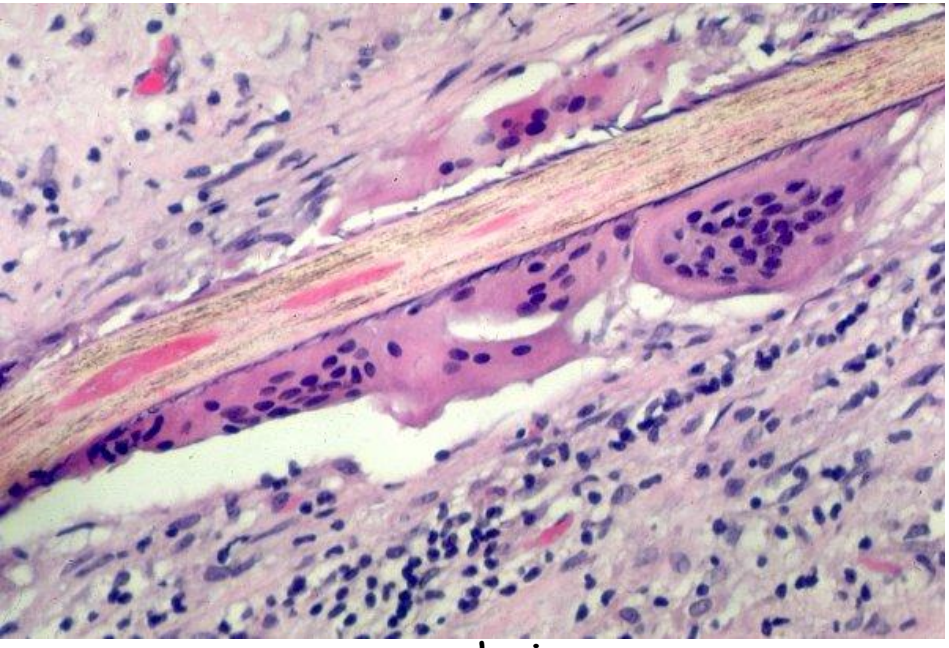
Foreign body type giant cells



bile



cholesterol



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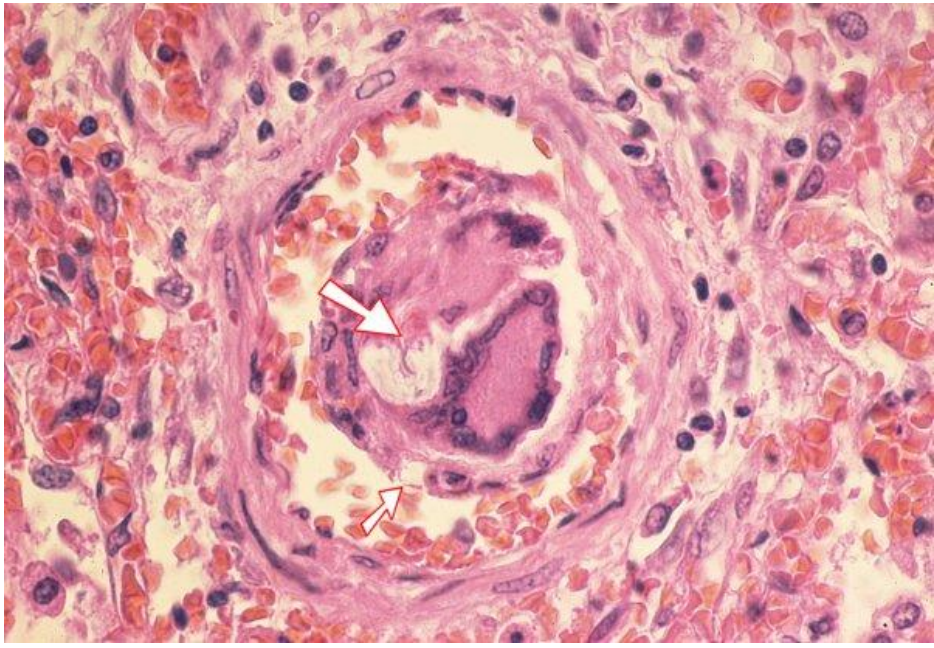


Crystal in lung artery (i.v. drug)

Wound healing

- Primary
  - Sterile, cut wounds, with sharp edge
- Secondary

  - damaged wounds with loss of tissue, infected wounds

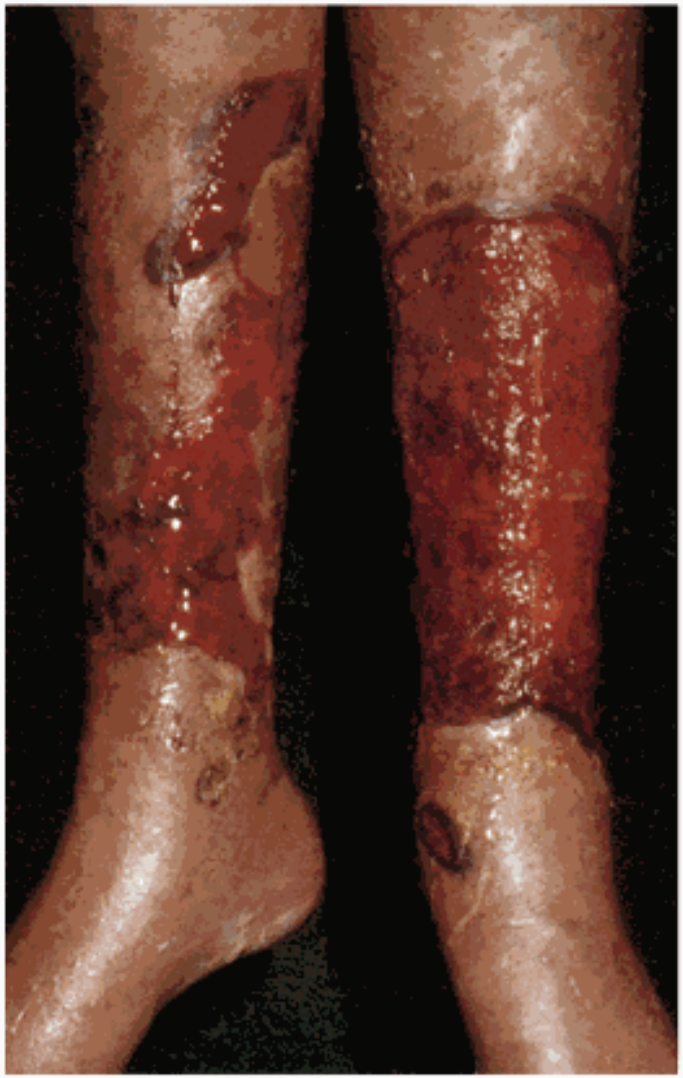
Phases:

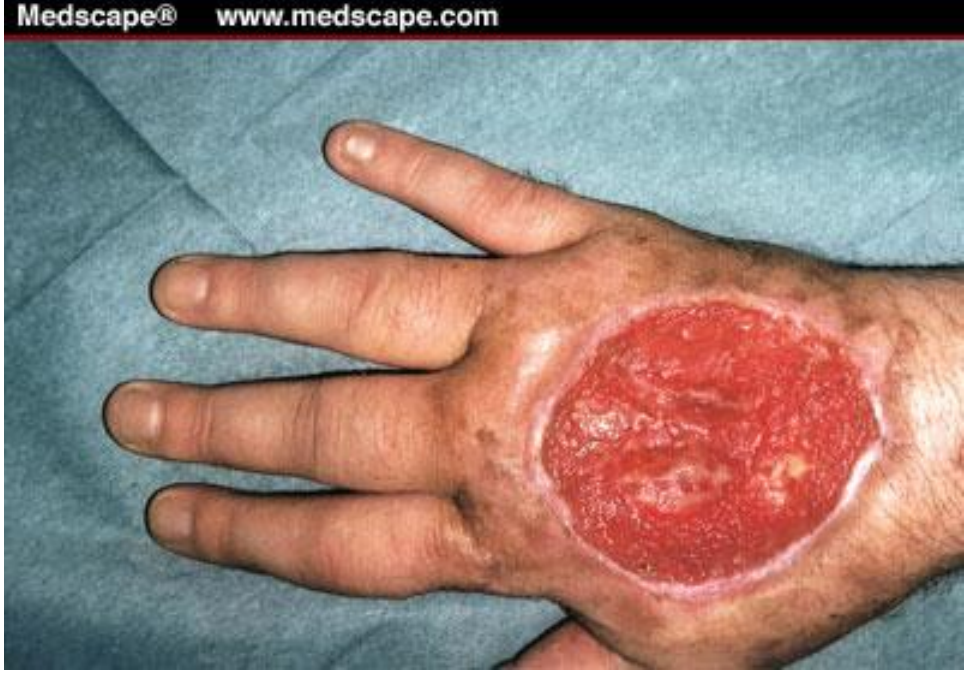
-Exsudative: wound exsudate, edema

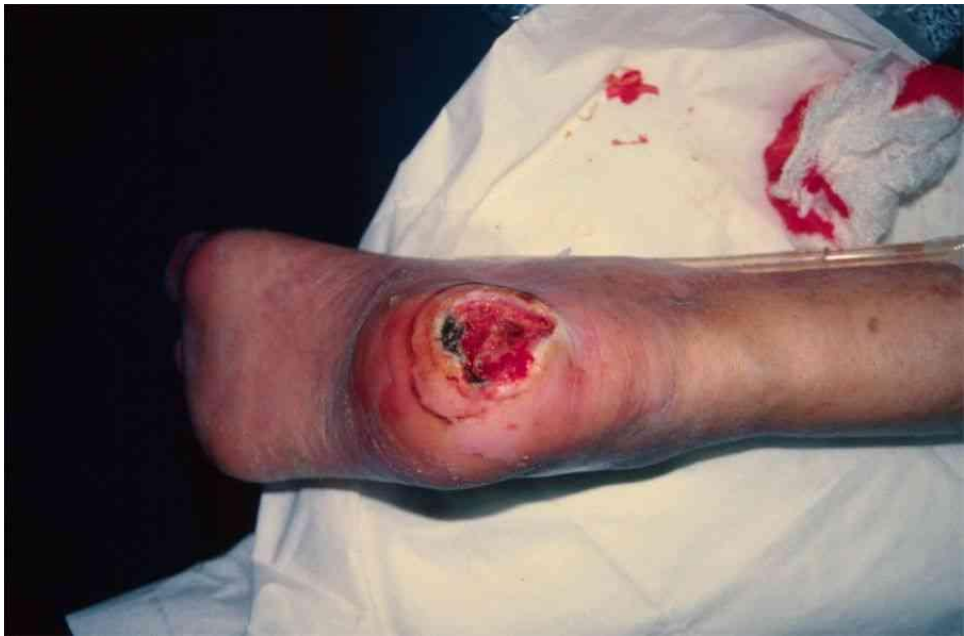
-Resorptive: complement system (6 hours),
cellular immunity (12 hours)

-Proliferative: 3rd day, granulation tissue

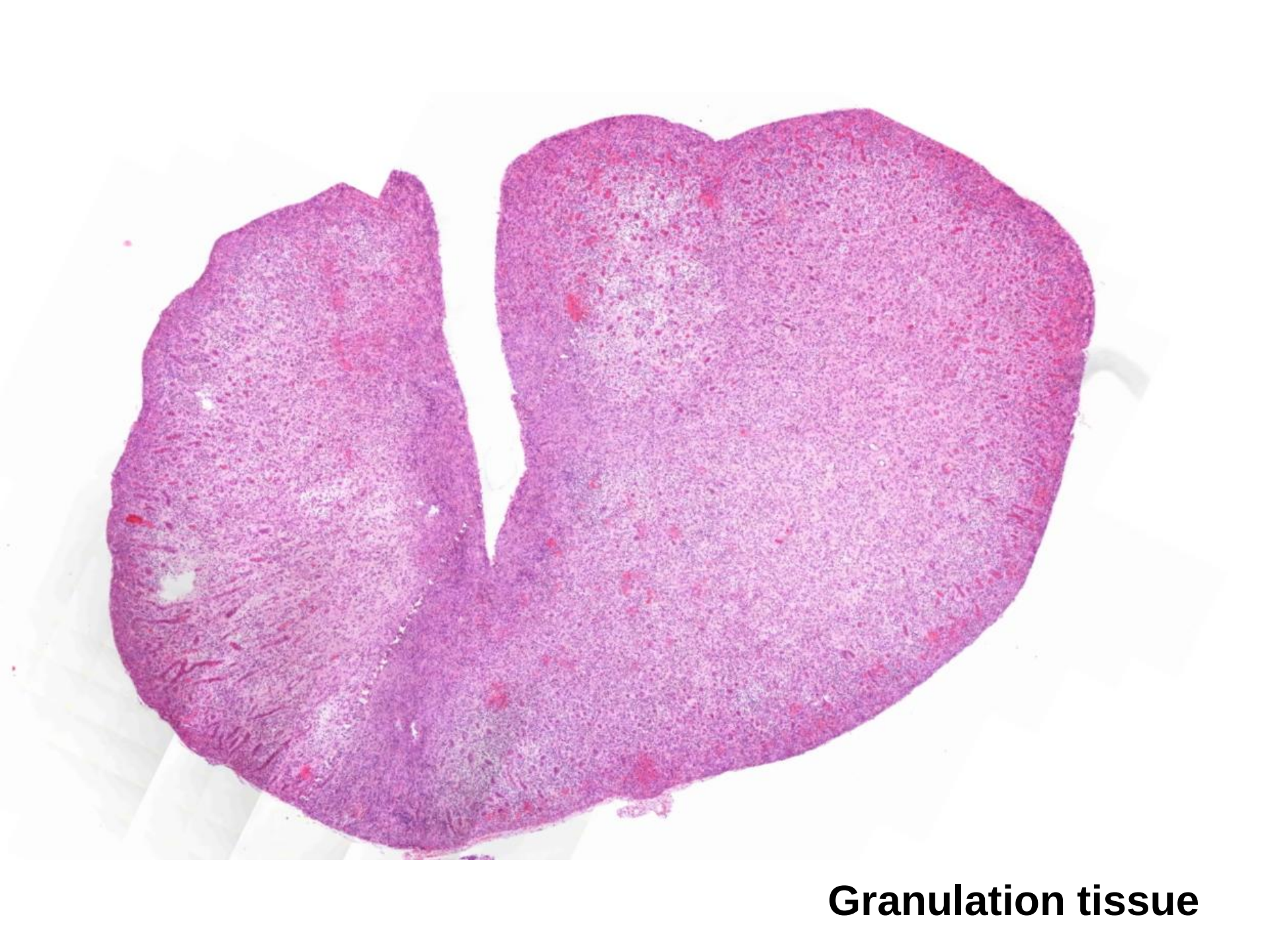
-Reparative: epithel migration, reepithelisation, macrophages, collagen, scar



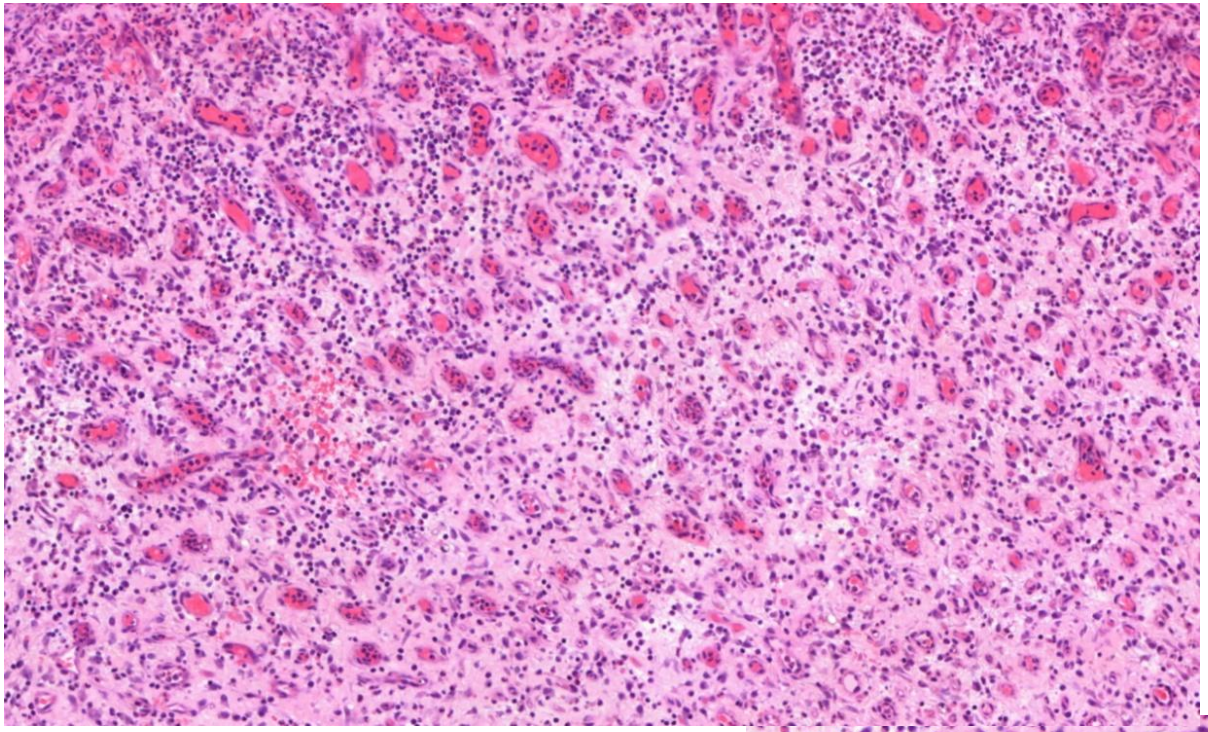


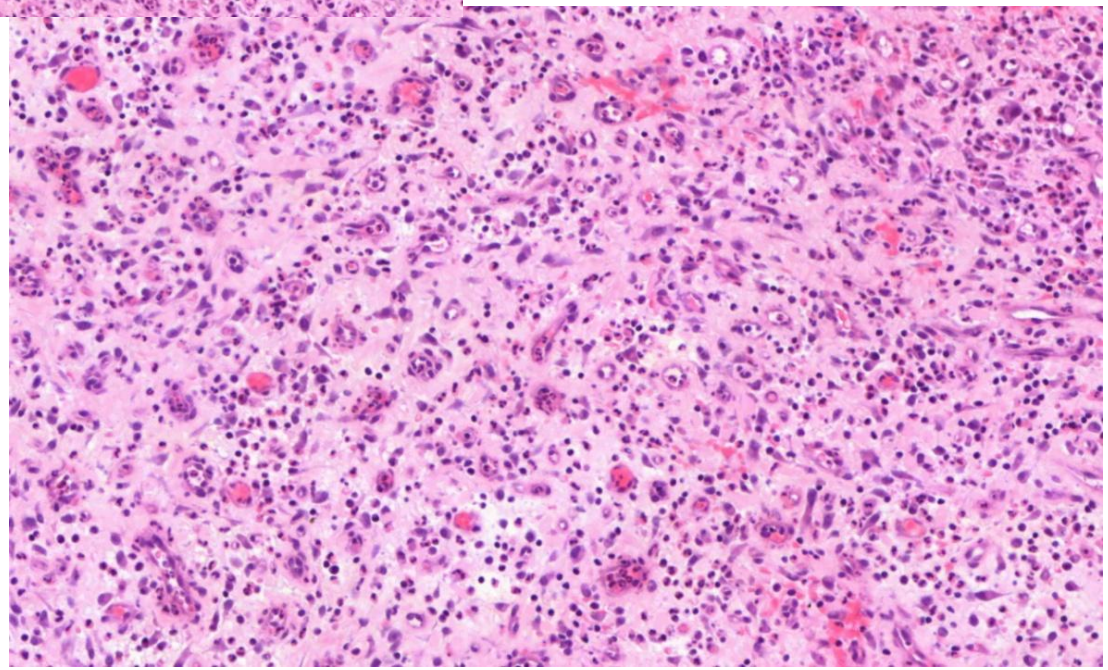


Granulation



Granulation tissue





Capillarization

Complications of wound healing:

- rupture
- infection
- granuloma formation
- traumatic epithelial cyst
- seroma
- keloid: too much scar tissue
- caro luxurians: too much granulation tissue