# INFLAMMATION

The infalmmation is a universal and ancient form of host defence.

The inflammation is not a disease.

Inflammation is not equal with infection

Immunity: **Innate** (~inflammation): delaying the need for adaptive immunity, (mechanisms are coded in genome)

Recognizes infectious non self

Execution

Serves to alert the clonal, adaptive immune system (dendritic cells!!)

**Adaptive**: it recognizes fine details of pathogenic organisms,

It is highly specific and remembers (but not heritable), gene rearrangement is necessary

It is responsible for allergy, autoimmunity, rejection.

Other defence mechanisms: Haemostasis

Regeneration

Innate immunity recognizes Pathogen Associated Molecular Patterns (PAMP)

e.g. LPS, CpG DNA

PAMPs are recognized by pattern recognition receptors (PRP) (cell surface, intracellular, serum)

Principal functions of PRPs: activation of complement, coagulation, phagocytosis

pro inflammatory signaling cascades

induction of apoptosis

Terminology: +itis

Most common extraordinary names

| /tissue | |
|---|---|
| tongue | glossitis |
| oral mucose | stomatitis |
| cornea | keratitis |
| lung | pneumonia |
| stomach | gastritis |
| small intestine | enteritis |
| coecum | typhlitis |
| rectum | proctitis |
| testis | orchitis |
| vagine | colpitis |
| fallopian tube | salpingitis |
| belly bottom | omphalitis |
| | |
| breast | mastitis |
| adipose tissue | panniculitis |
| brain | encephalitis |

Characteristics of acute inflammation: rubor, tumor, calor, dolor

The purpose of the inflammatory vascular reactio is to deliver the humoral and cellular factors to the site of defence reaction

Mechanism of active hyperaemia : arteriole dilatation, reduced venous outflow, dilatation of capillary network

Tumor (swelling) exsudation early phase (histamin, bradykinin) postcapillary venule

Late phase (TNF, IL-1, IFN g) capillaries

Vascular damage

Exit of cellular elements

Chemotaxis bacteriale-, necro-, endogén chemotaxins

Margination integrins, ICAM, VCAM

Diapedesis (in venules)

Activation

Execution opsonisation, phagocytosis, killing (oxigén dependent and independent mechanisms)

***Cellular elements of inflammmaton:***

Neutrophil gr, Eosinophil gr, Basophil, Mastcell,
Macrophages, Endothel, Fibroblasts
Thrombocyte, Lymphocyte

***Humoral elements***

Vasoactive amins: histamine, serotonine (vasodilatation, permeability, pain)
Vasoactive peptids: bradykinin
complementr (MAC, vasodilatation, permeability, chemotaxis, opsonisation
Clotting, fibrinolitic cascade
Immunglobulins
Arahidonic acid derivatives
Cyclooxigenase prostaglandins
Lipoxigenase leukotriéns
Cytokins TNF, IL-6, IL-1
Exogén mediátors: fMLP, endotoxin, superantigens

**Classification of acute inflammation based on the exudate**

Serous common cold, pleuritis exs., burns

Fibrinous serous membranes peuritis sicca

Mucous memebranes Diphtery, typhoid fever, dysentery

Purulent folliculitis, furuncule, carbuncule

Abscess: circumscibed pus in tissue

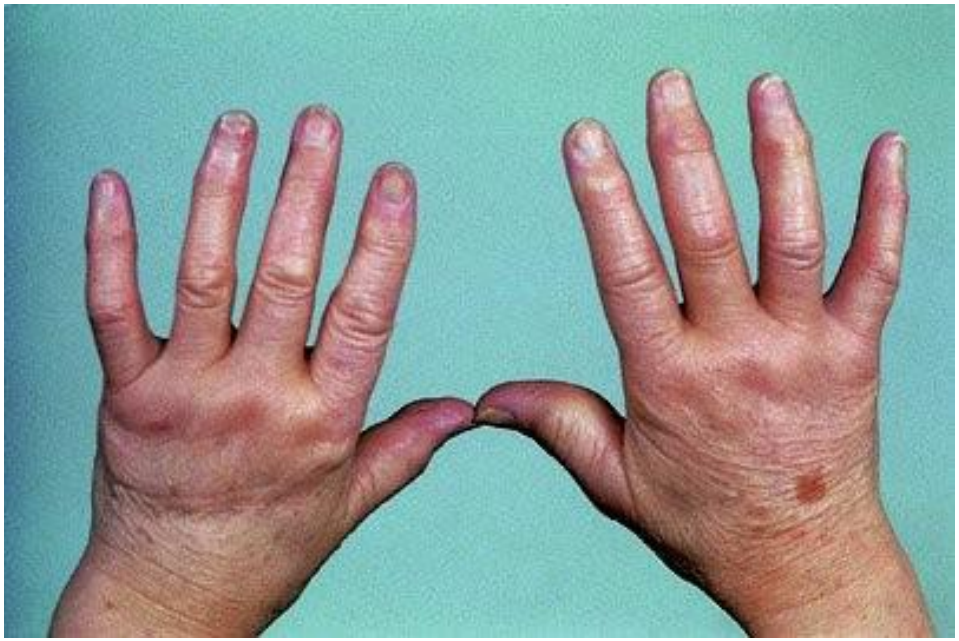
Empyema circumscribed pus in preformed body cavity

Phlegmone inflammation spreading in tissue spaces

Haemorrhagic: plague, smallpox, anthrax, flue

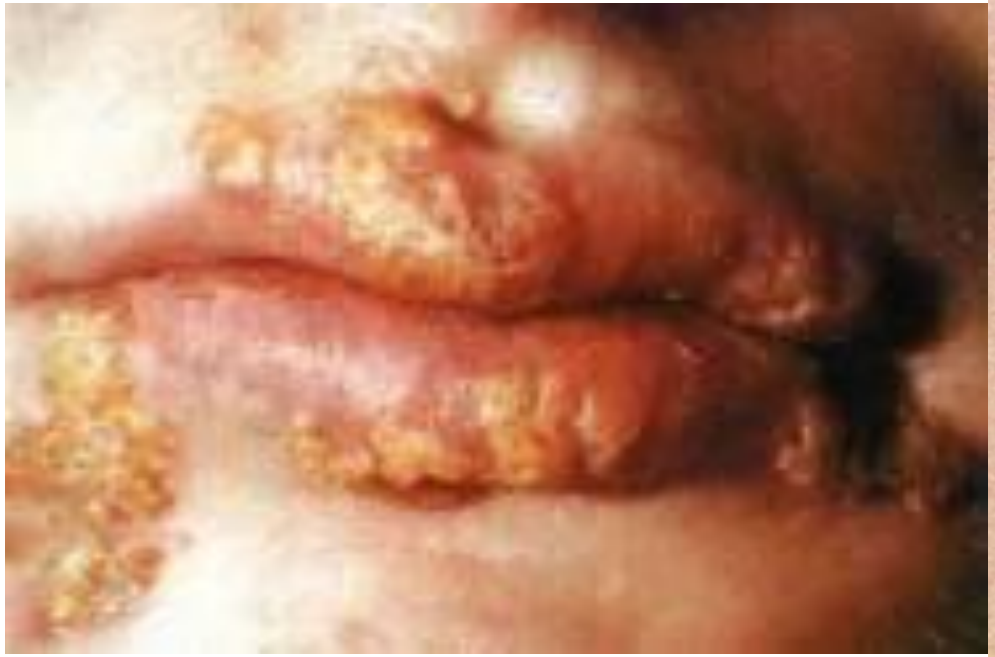
Gangraenous, - failure of inflammation

SEROUS INFLAMMATION
Serous mucous, serous membranes: rhinitis, serous memb. pleuritis exsudativa., skin: erysipelas, chicken pox (bulla vesicles)
Meningitis serosa



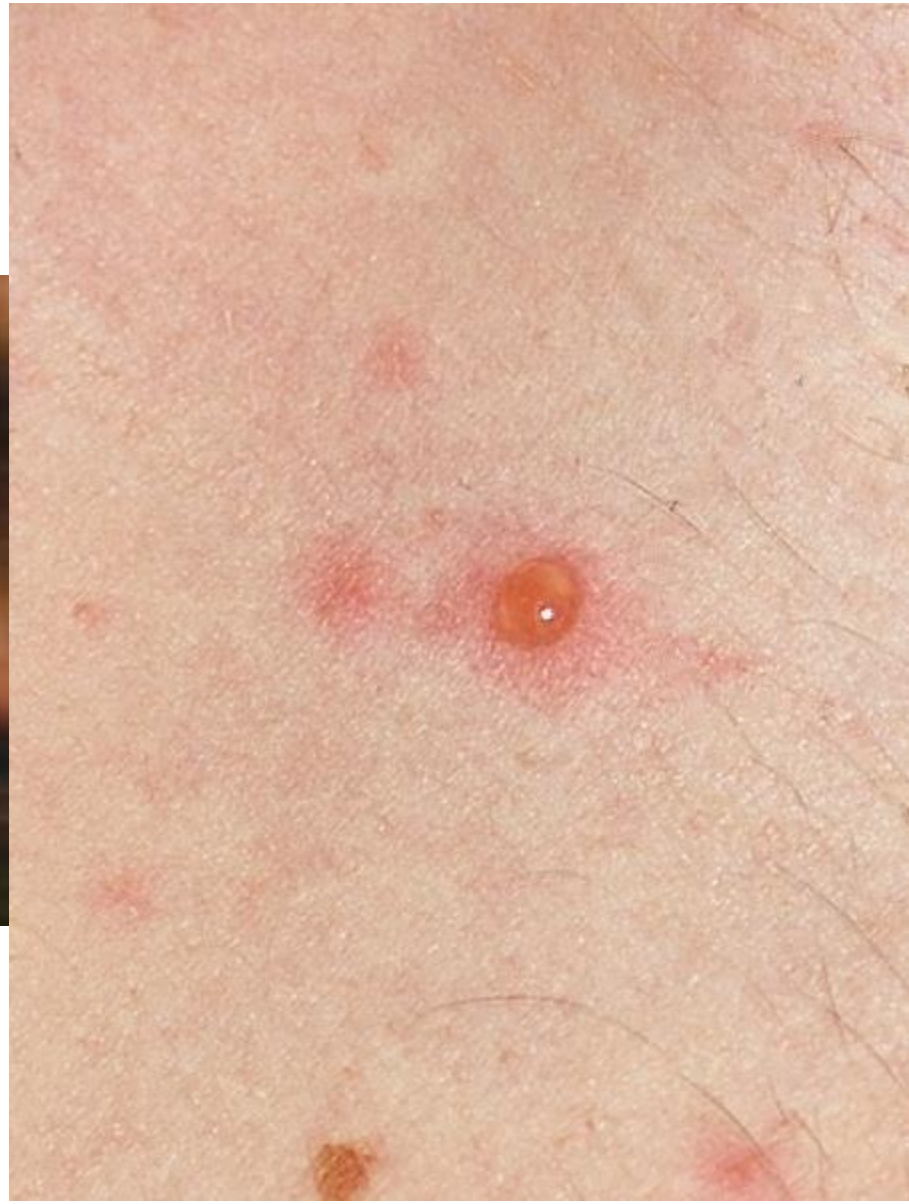
POLYARTHRITIS

SEROUS INFLAMMATION



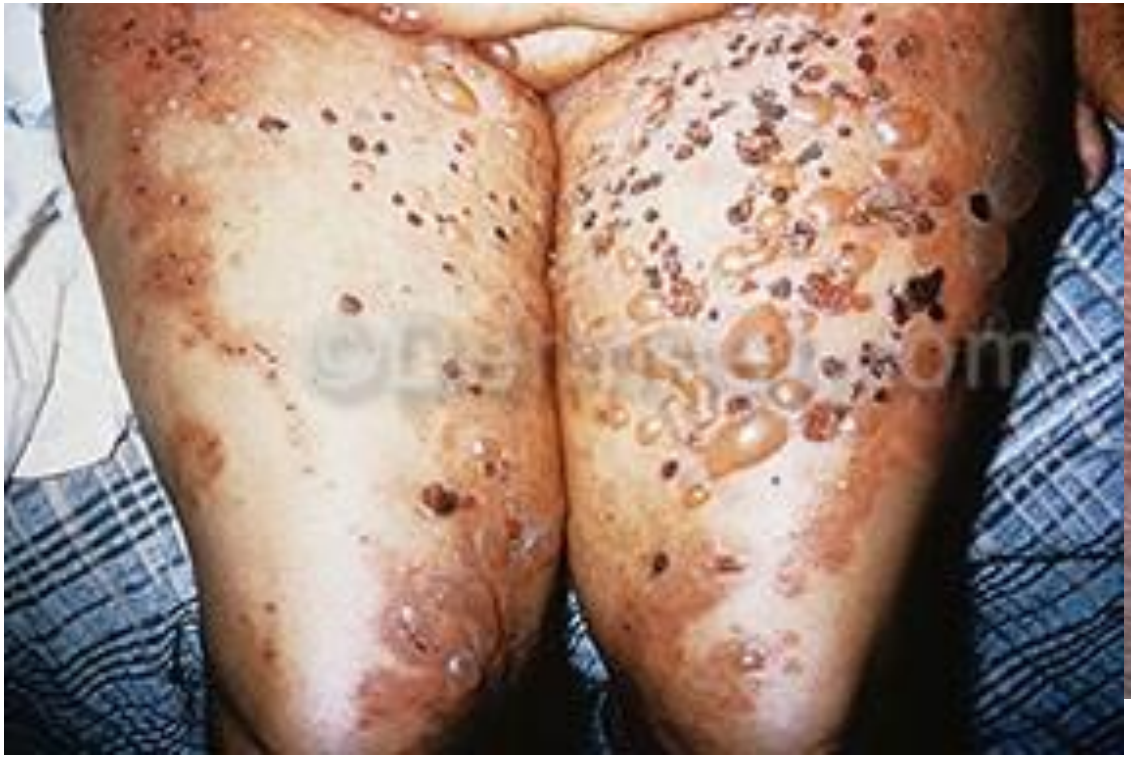
HERPES SIMPLEX,

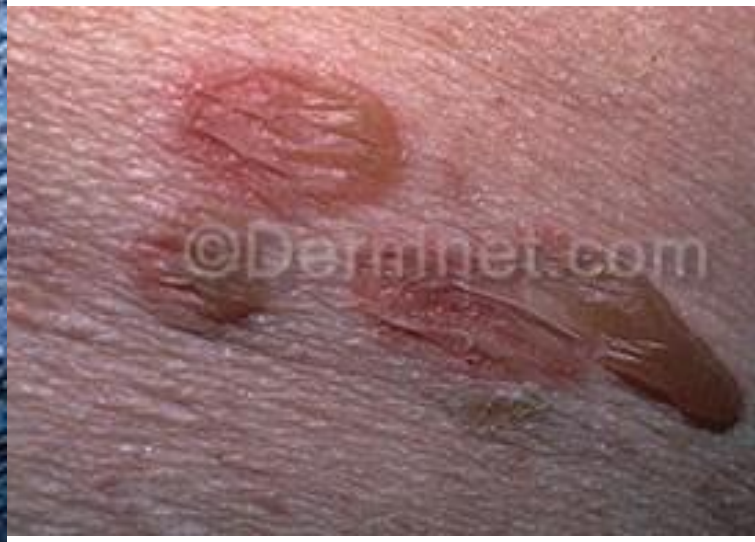
Herpes genitalis



VARICELLA (Chicken

SEROUS INFLAMMATION



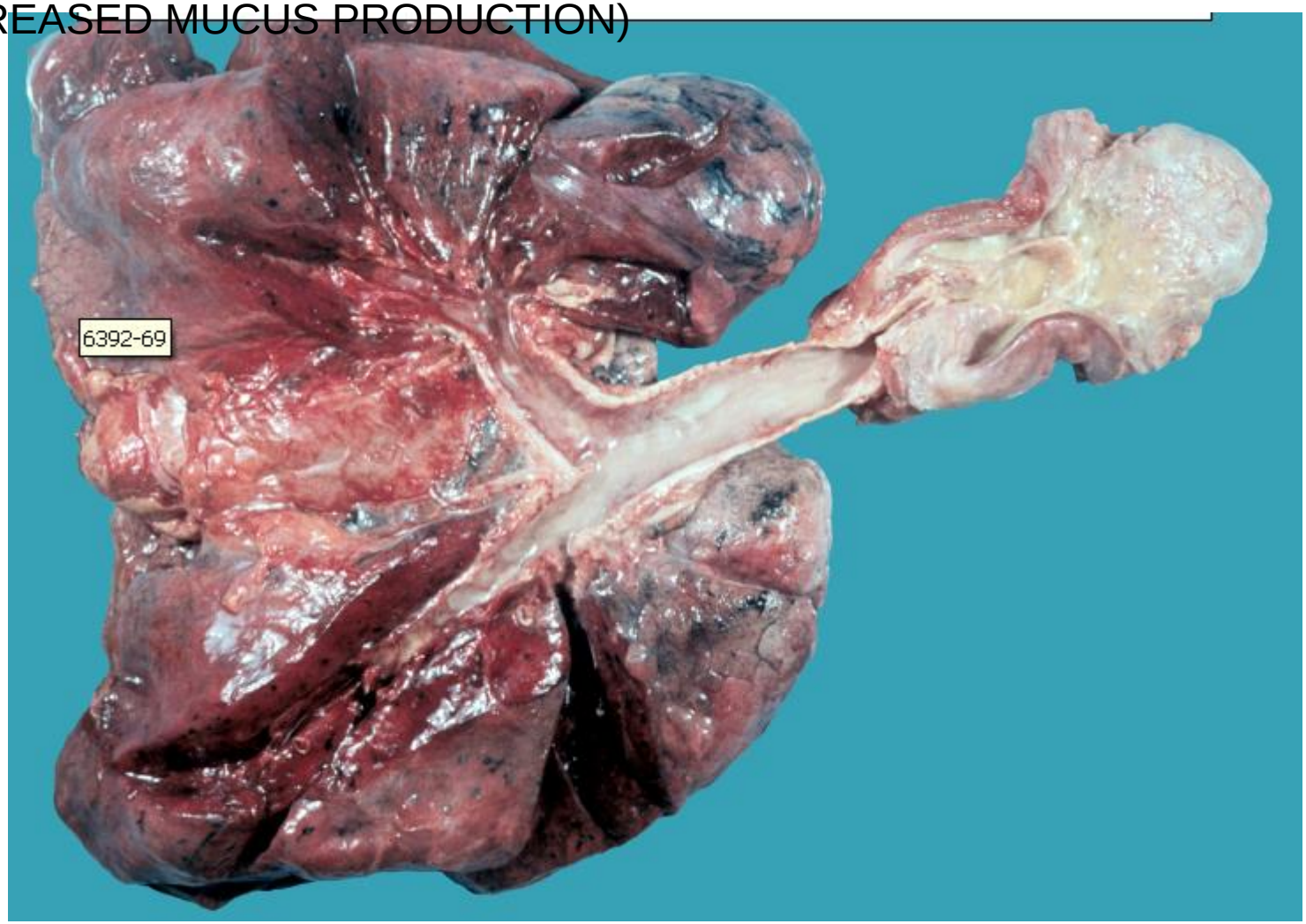



PEMPHIGUS VULGARIS

INFLAMMATIO MUCINOSA CATARRHALIS

(respiratory system GI)

(INCREASED MUCUS PRODUCTION)




TRACHEITIS CATARRHALIS

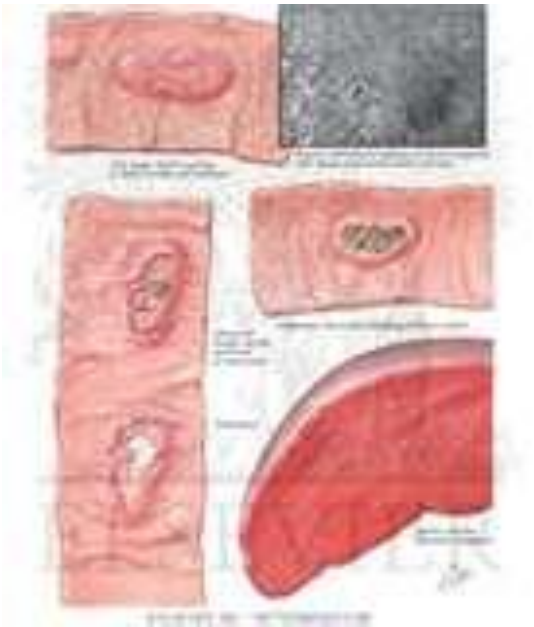
***Fibrinous inflammation***

pleuritis sicca, peritonitis

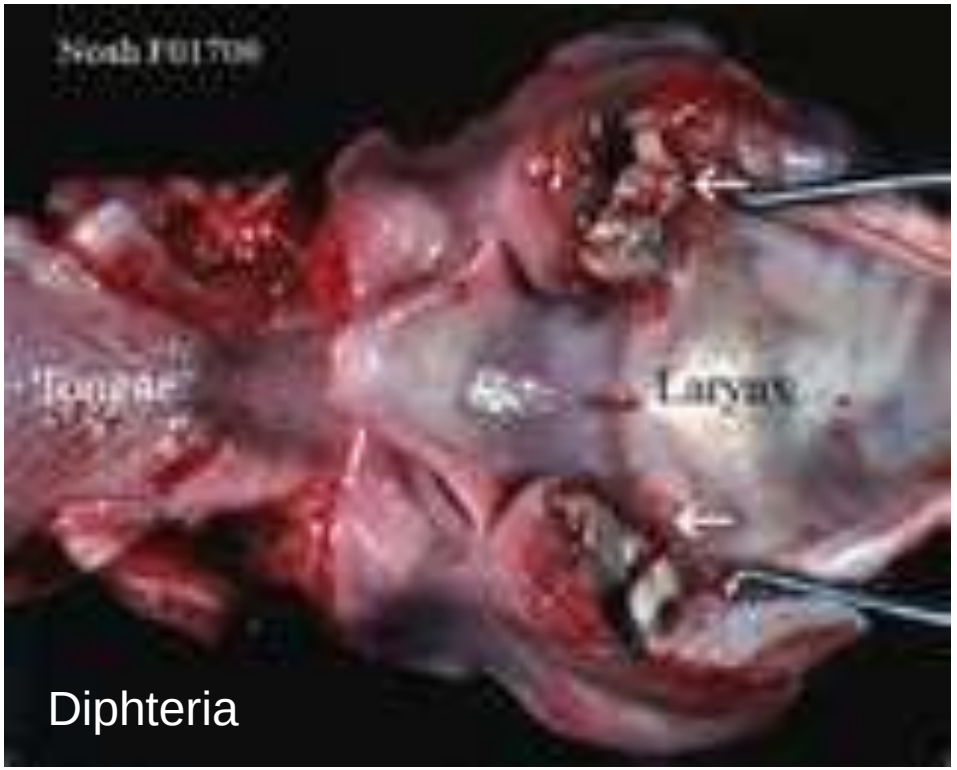
Mucous membranes:

Croupos diphteria

Diphteriás typhoid fever (ileum), dysenteria (colon)

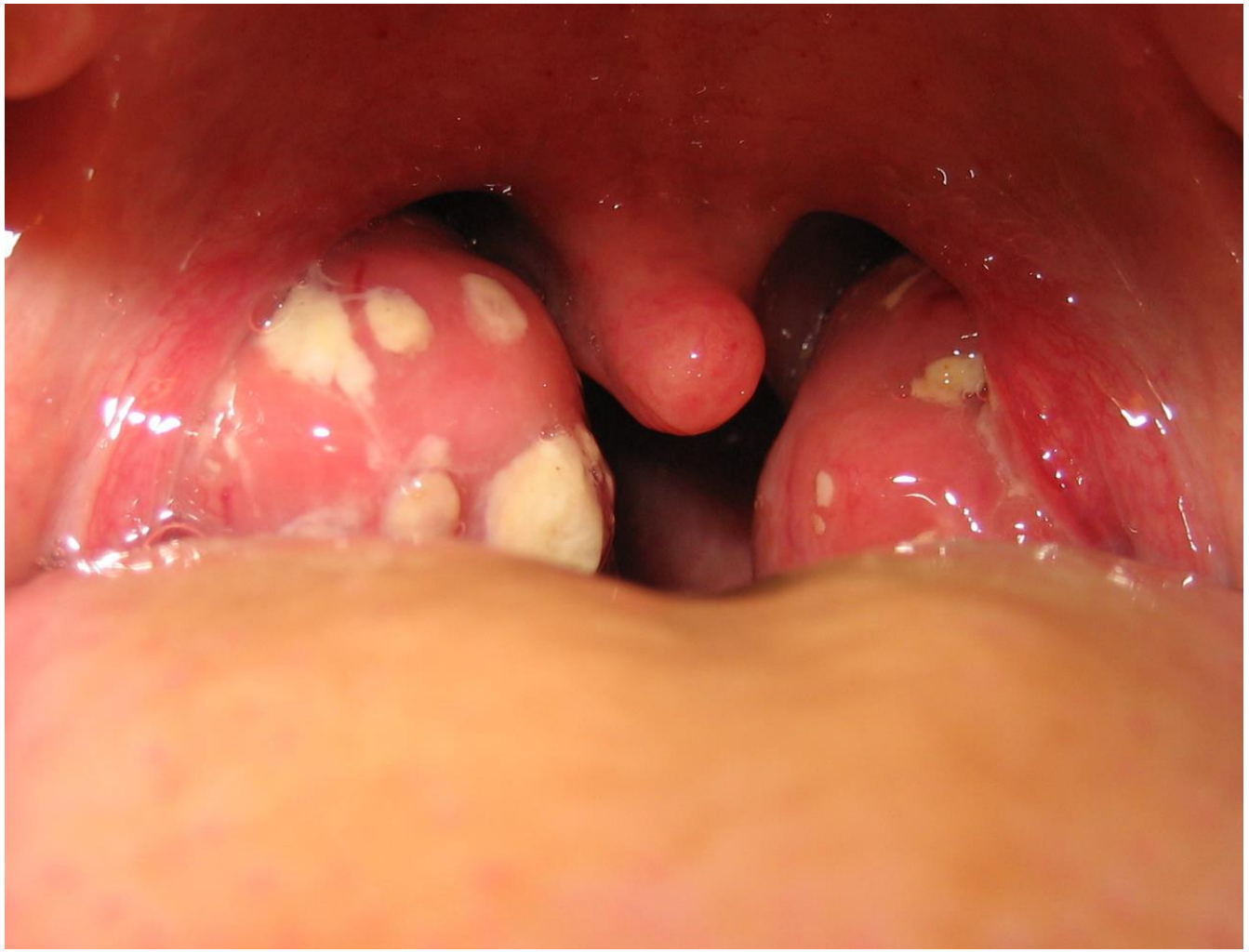


Typhoid fever



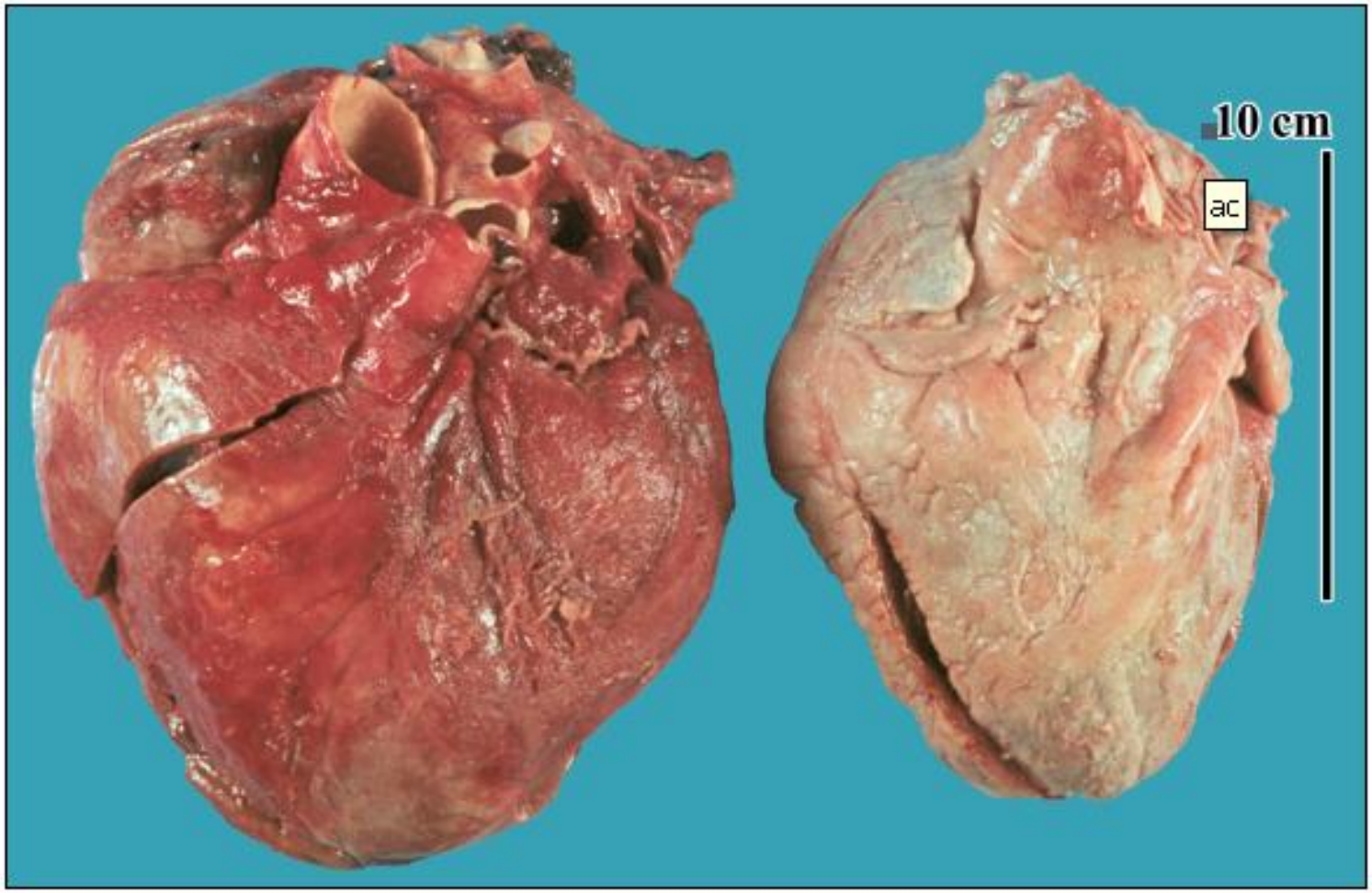

Diphteria

Peritonitis fibrinosa

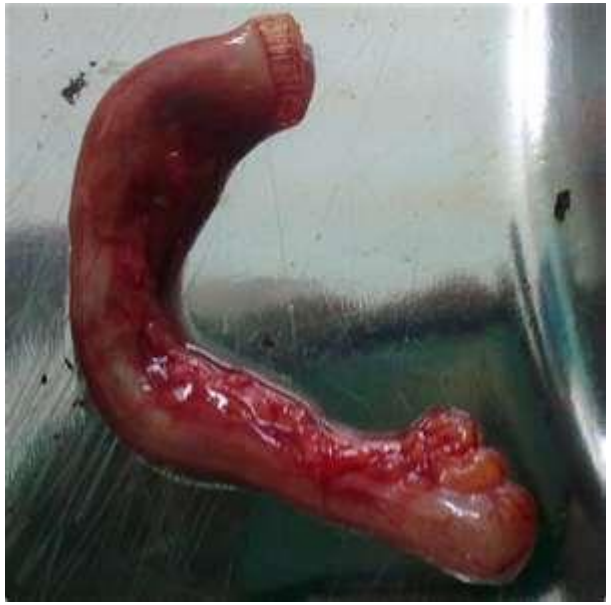


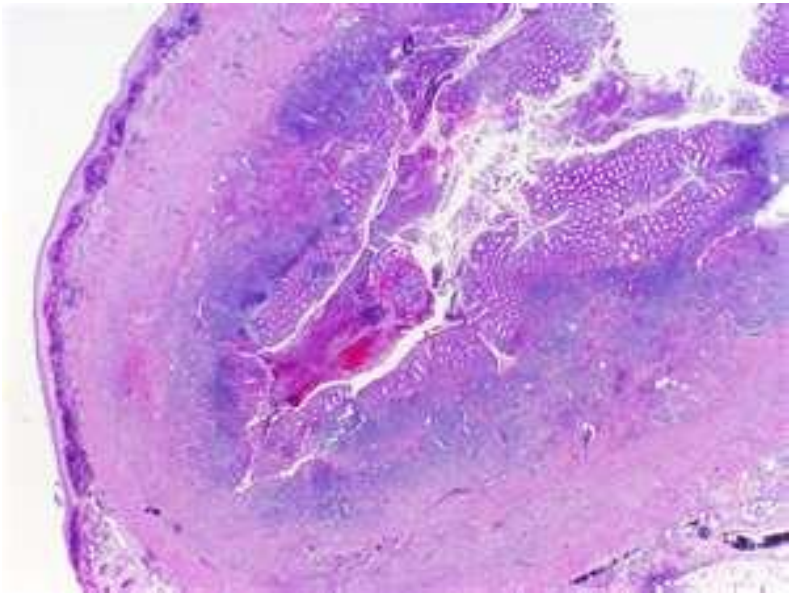
Tonsillitis follicularis

# Pericarditis acuta fibrinosa



# Inflammatio fibrinoso-purulenta





Appendicitis acuta

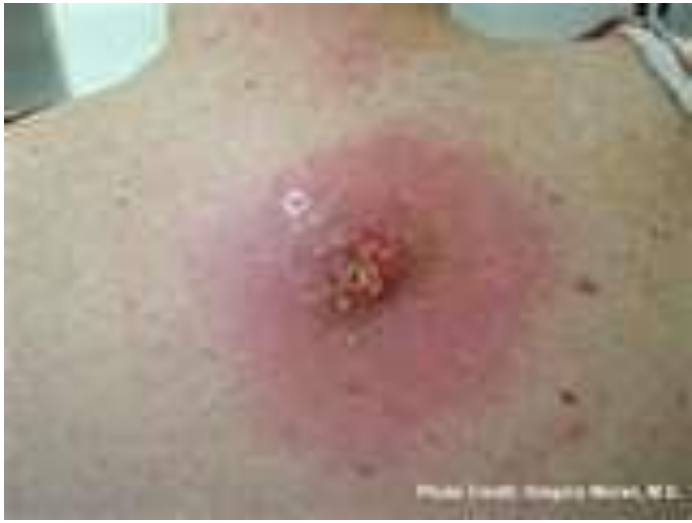
# Purulent inflammation

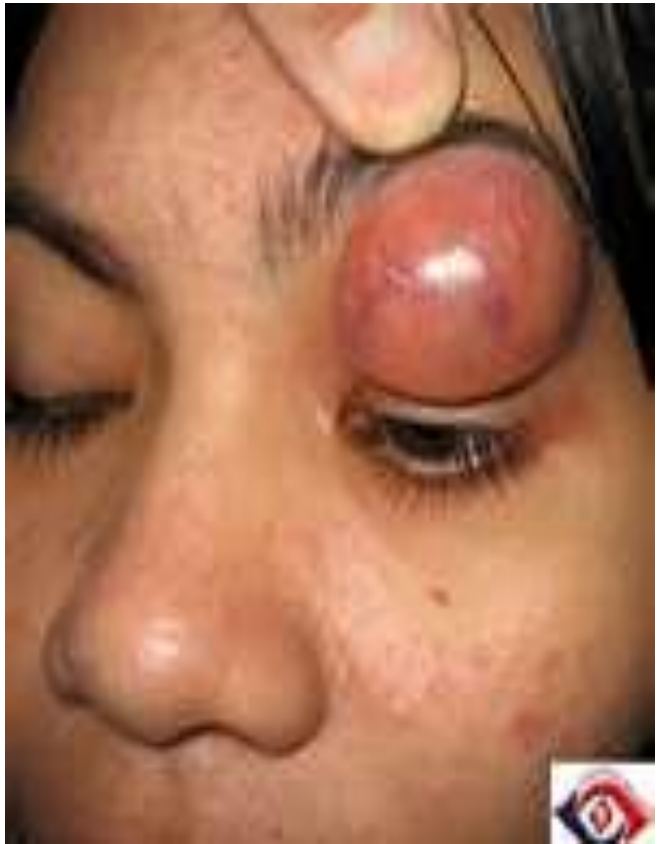
folliculitis, furuncle, carbuncle

**Abscess collection of pus**

**Empyema** collection of pus in preformed cavities

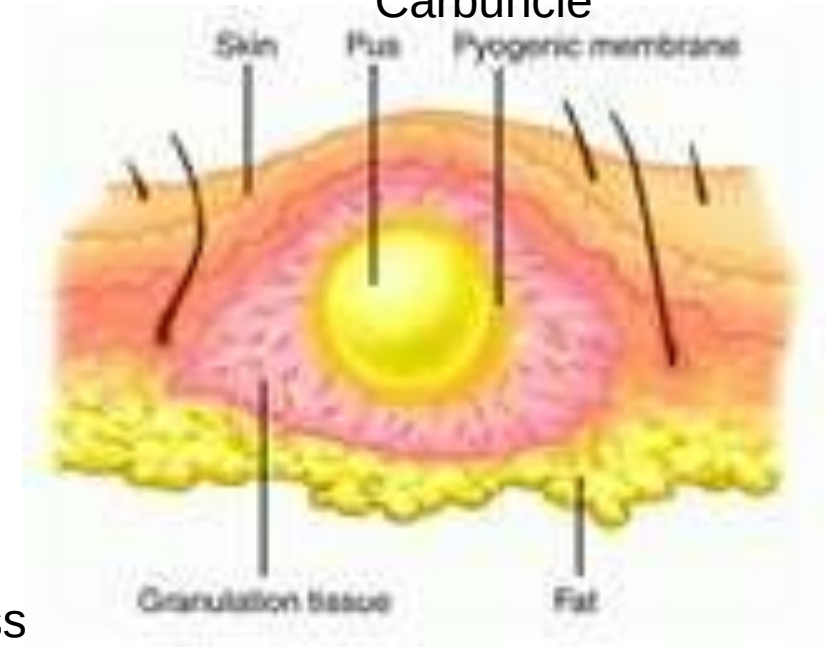
**Phlegmon** diffuse inflammation of soft or connective tissue



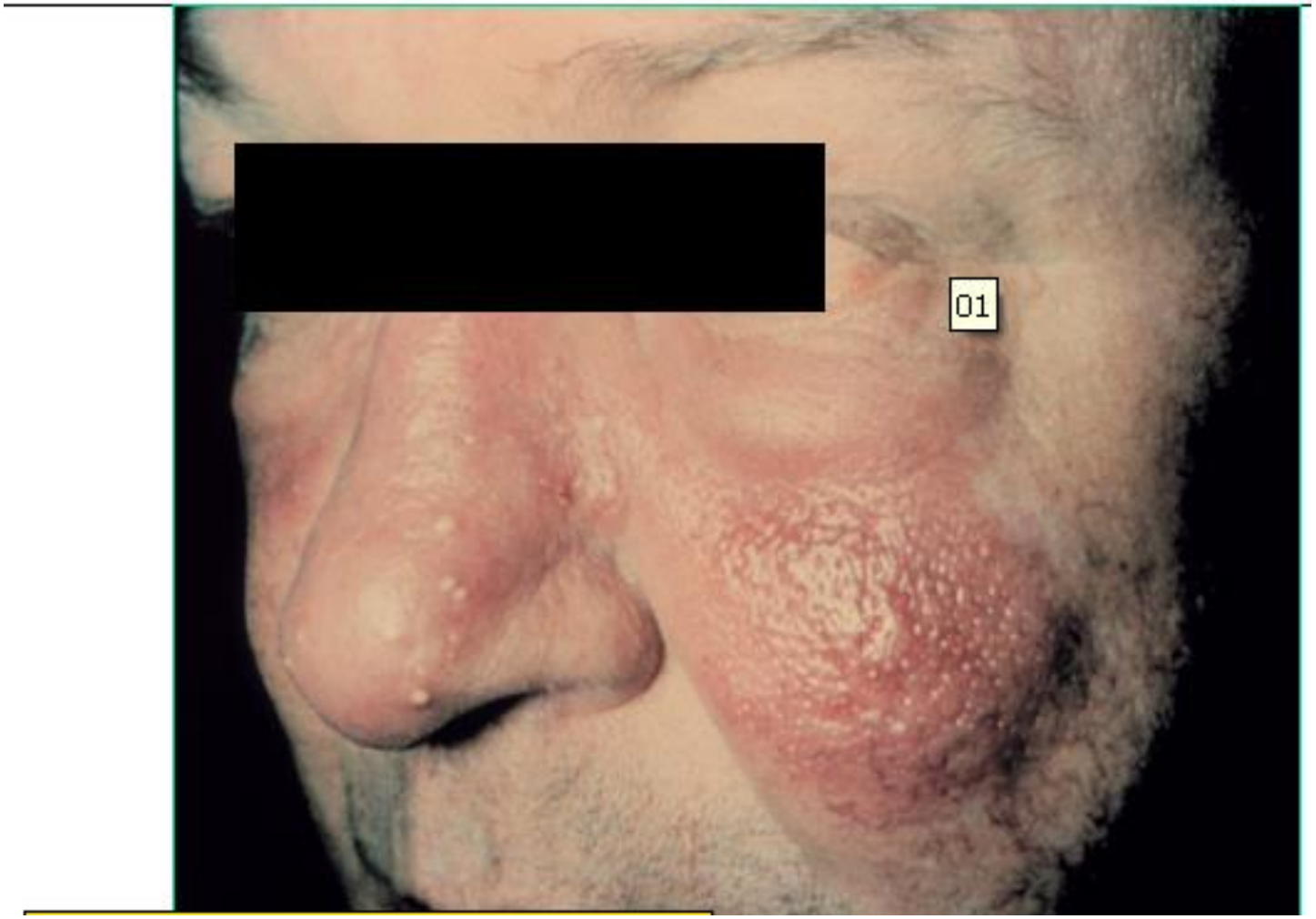


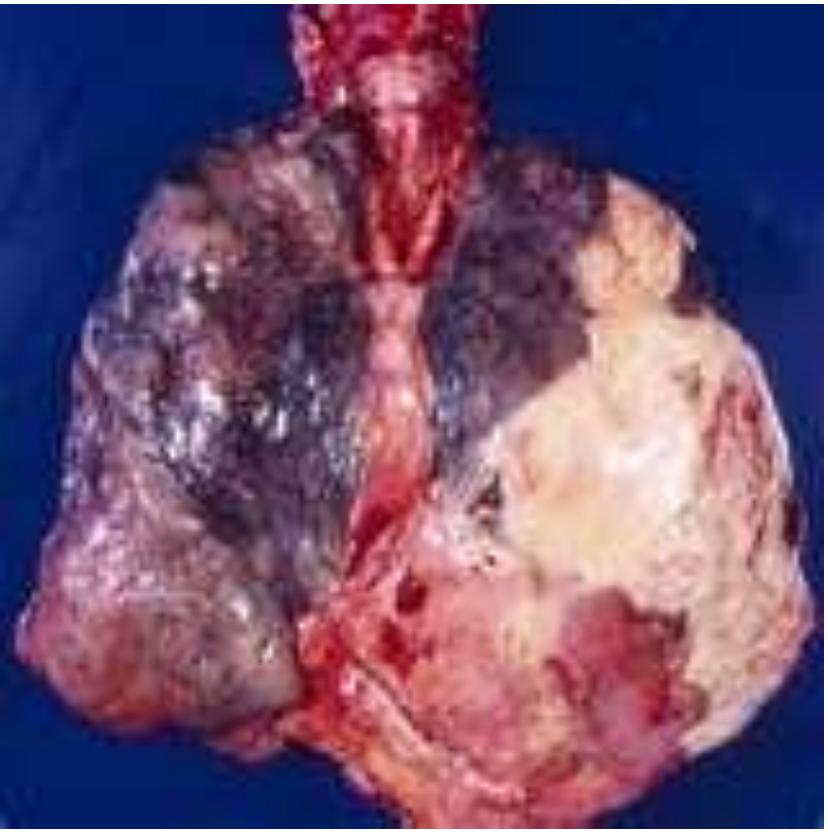
Abscess

Carbuncle

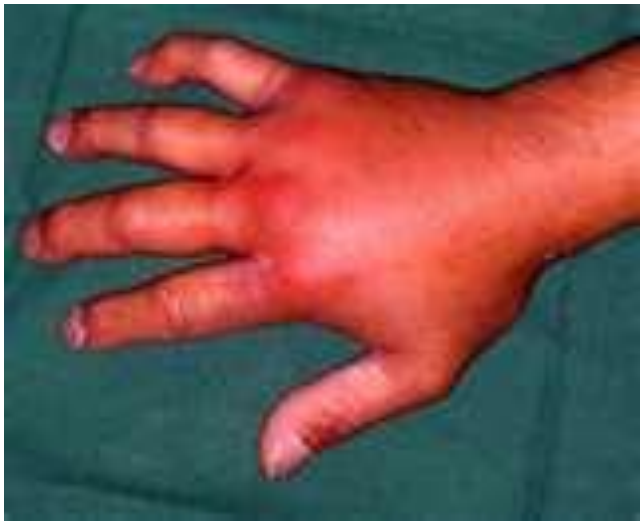


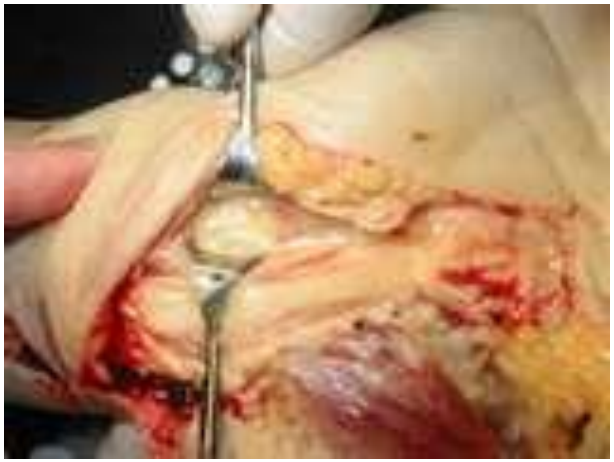
Folliculitis (boil) on the nose  carbuncle on cheek





Empyema

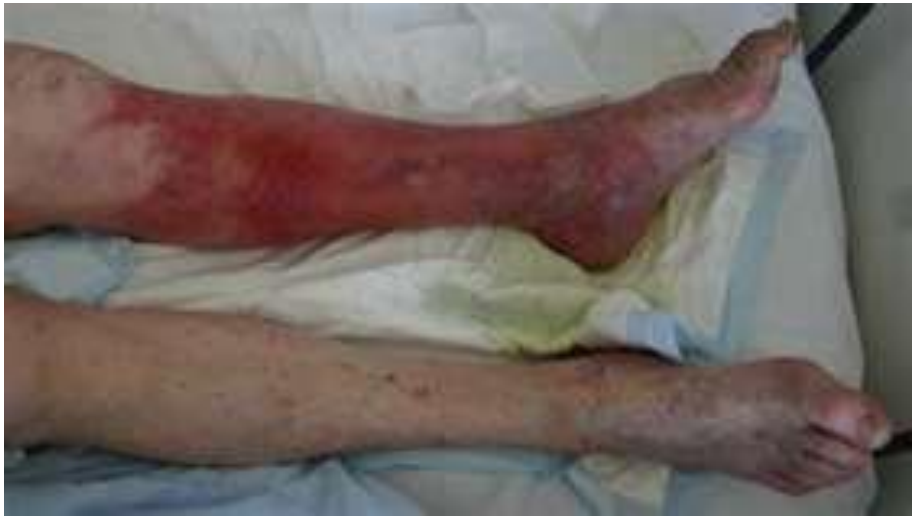




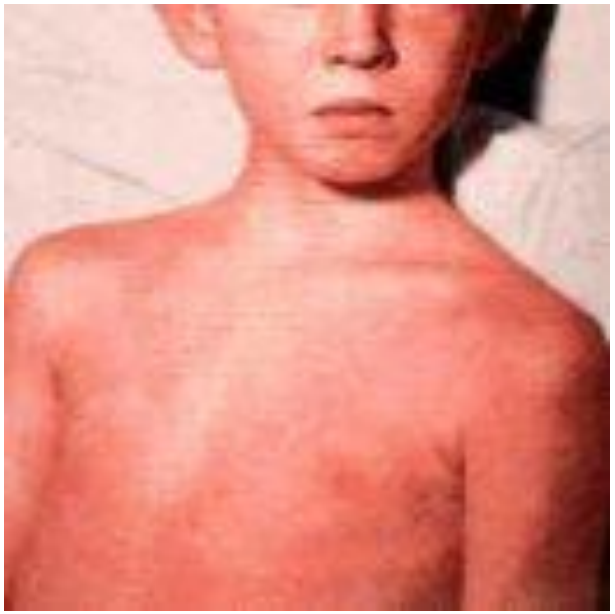
Phlegmon

# Sreptococcal related diseases

Purulent skin infections  (impetigo)
Tonsillitis, scarlat fever, bronchopneumonia
Necrotizing fasciitis



Erysipelas



Scarlat fever

Poststreptococcal diseases:

(rheumatic fever, proliferative glomerulonephritis)

# Staphylococcal infections

Purulent skin infections (furuncle, carbuncle)
Osteomyelitis
Mastitis,
Toxic shock synd.
Resp. infections
Food poisoning

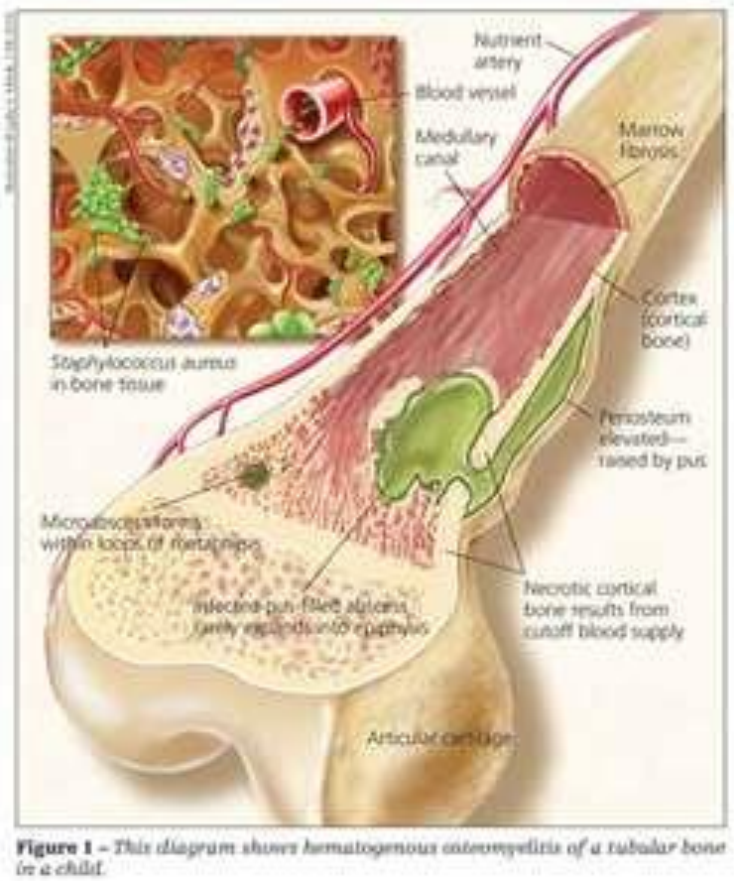
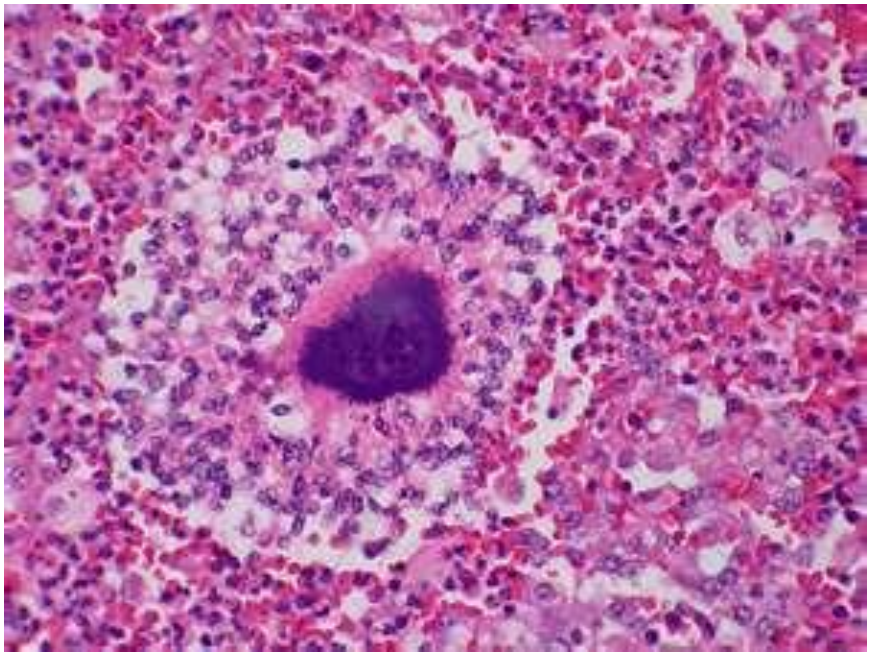


**Figure 1** – *This diagram shows hematogenous osteomyelitis of a tubular bone in a child.*

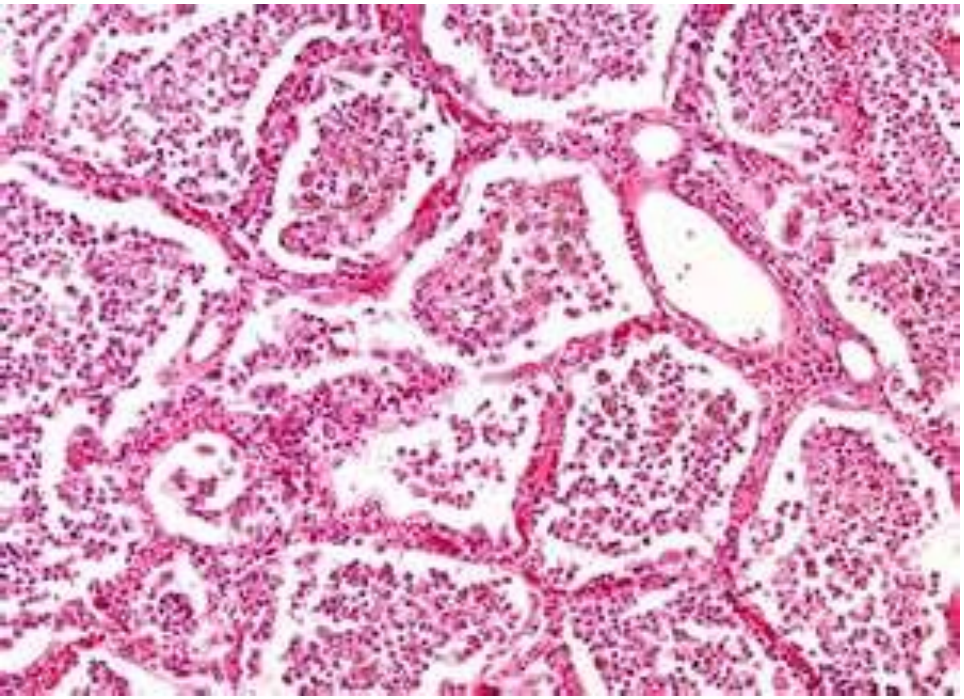
Osteomyelitis

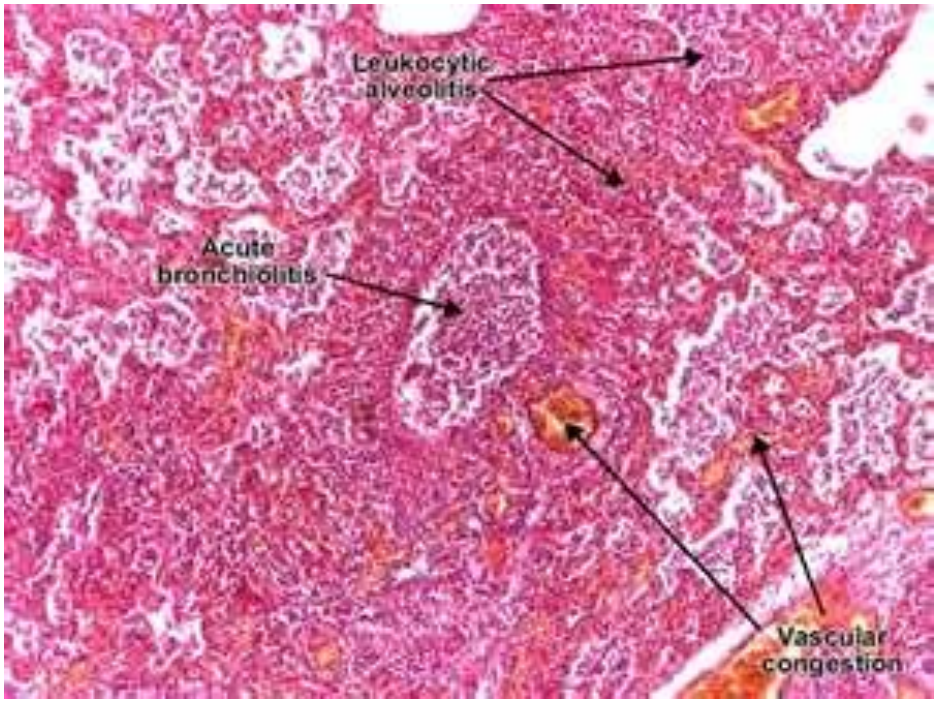


Actinomycosis (Actinomyces israelii)

Forms: facial, pulmonary, abdominal

# Pneumonias





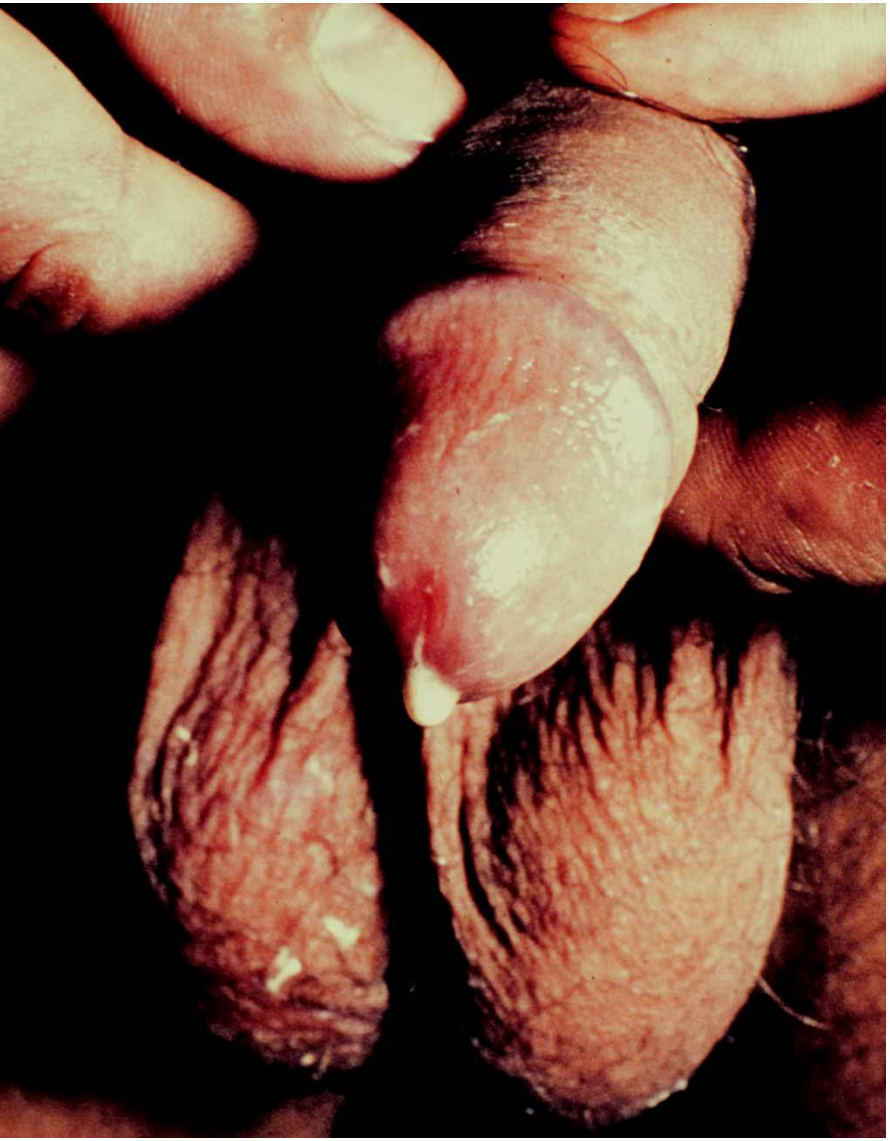

Lobar pneumonia

Hepatisation rubra, grisea, flava)

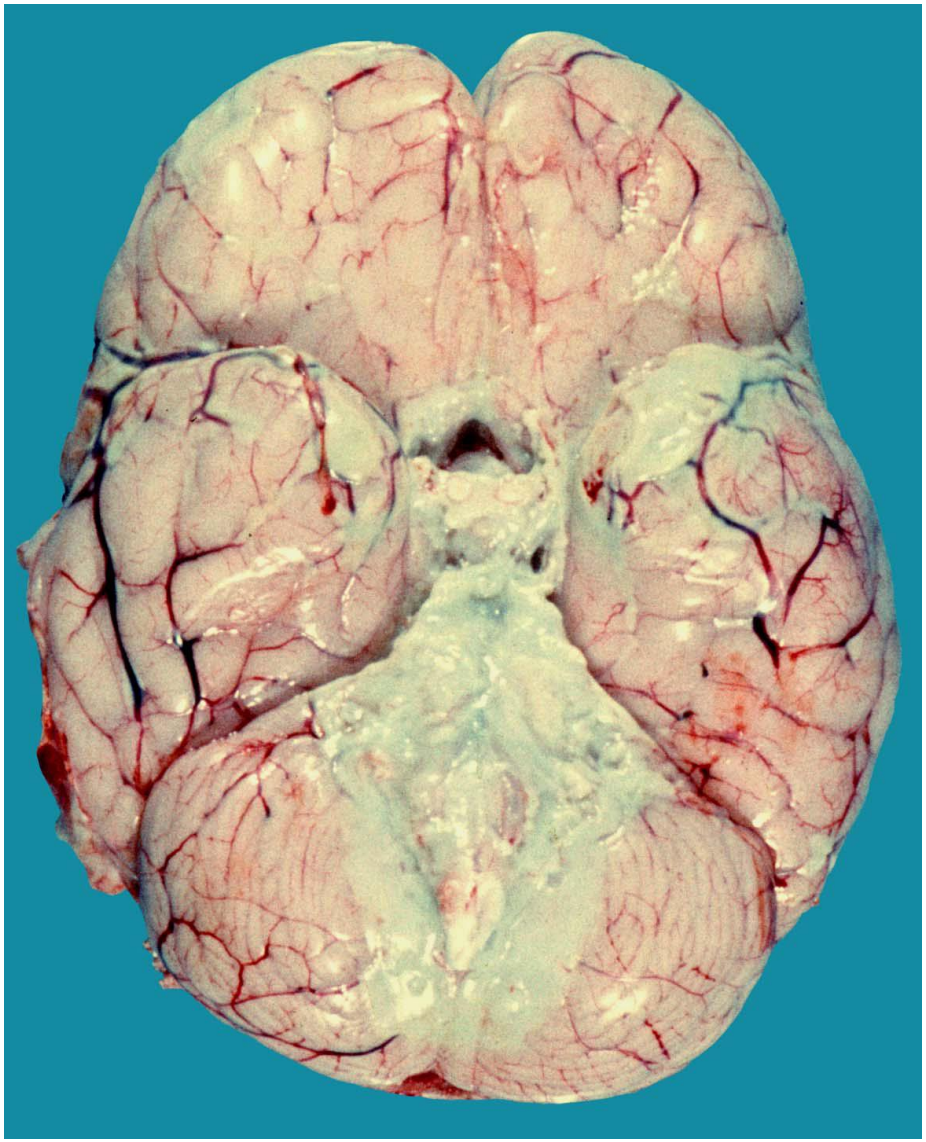
Bronchopneumonia

(Hypostatic, Aspiration)

Special forms: influenza-pneumonia, Pneumocystis carinii



Gonorrhea



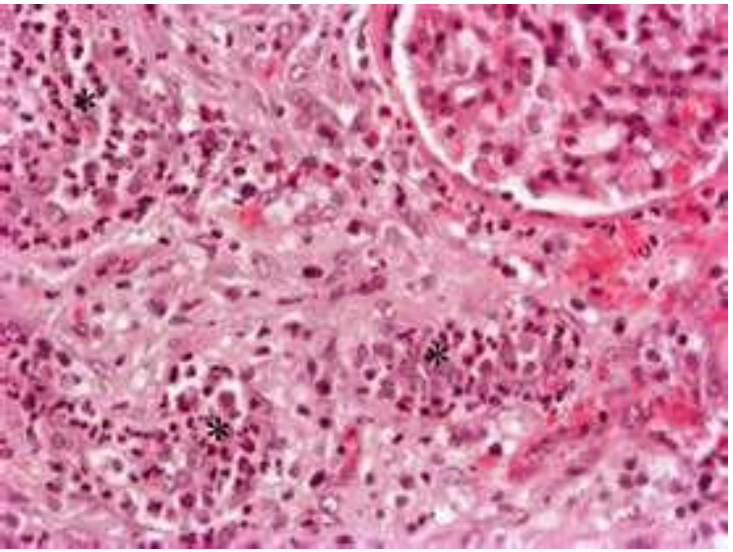
Meningitis purulenta

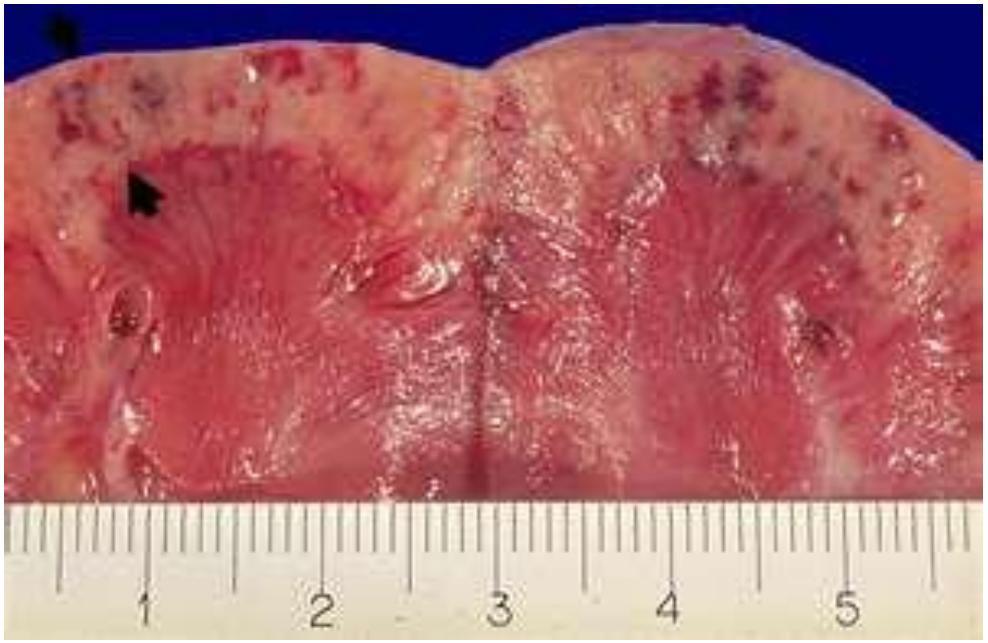
Route of inf: hematogenous, spreading from environment, direct

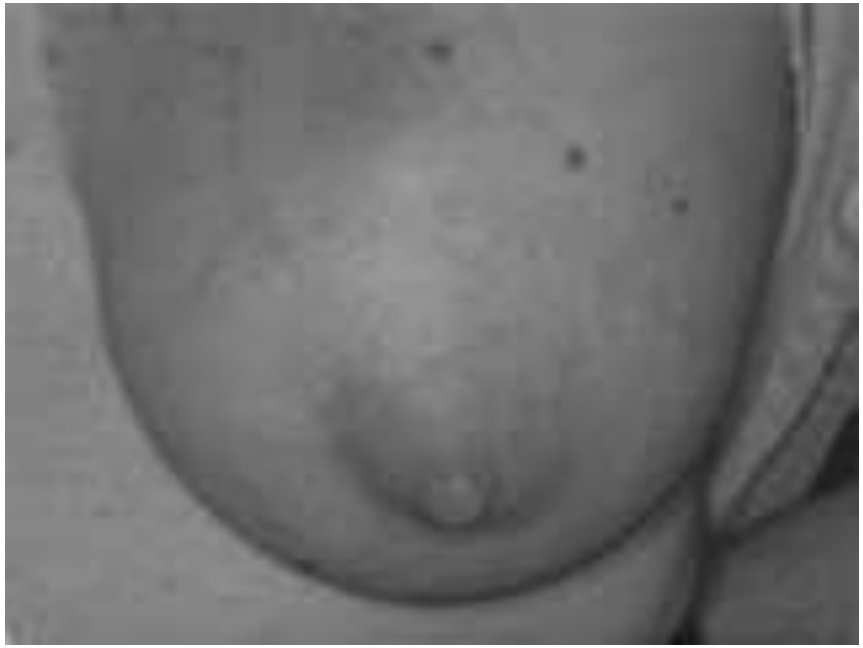
Acute Pyelonephritis (interstitial nephritis)

Spreading: Ascending, hematogenous

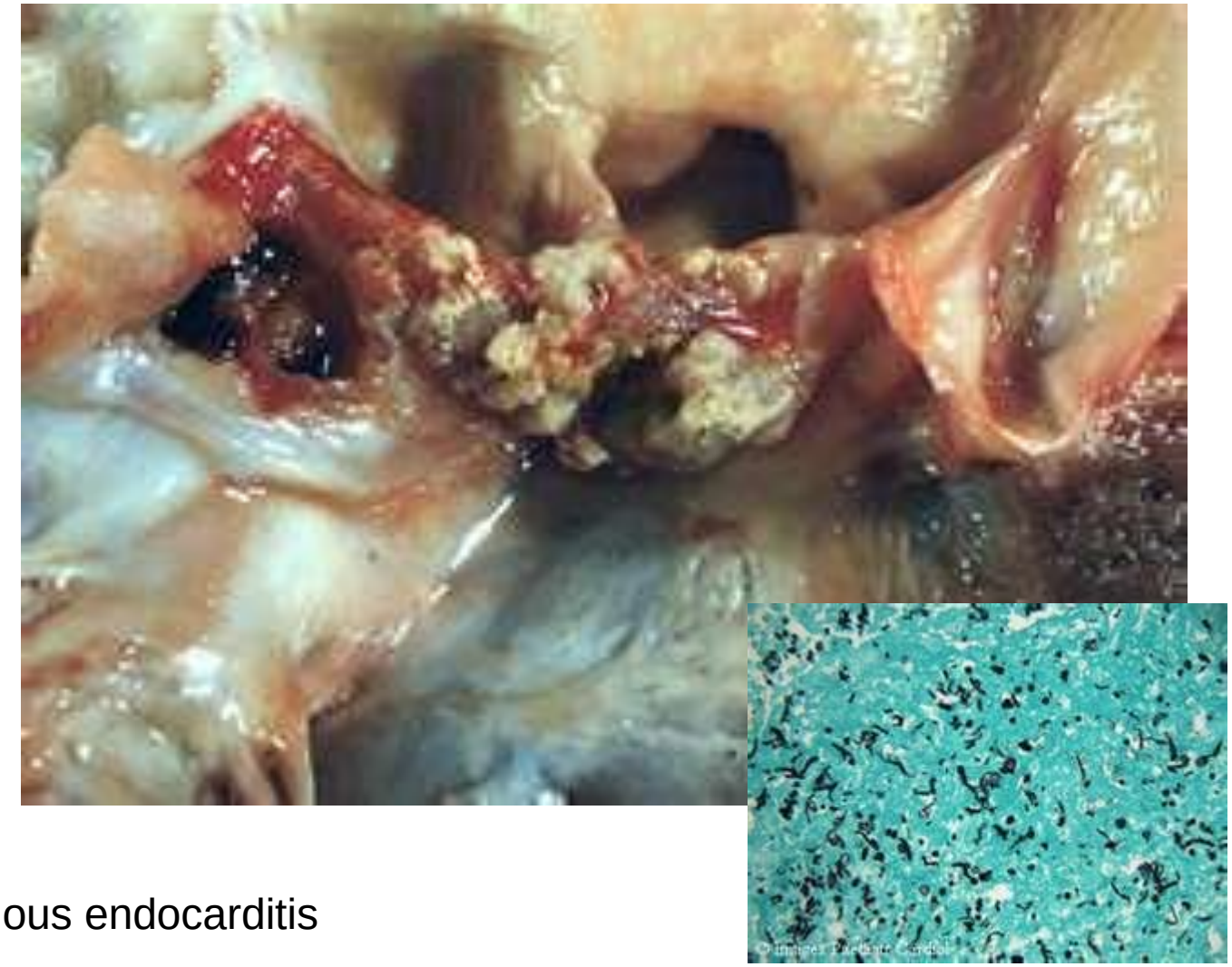
In diabetes, Aposthematous, papilla necrosis





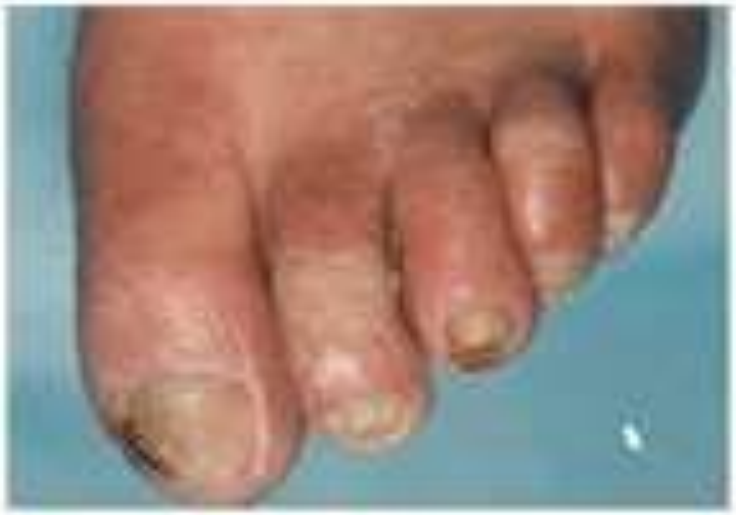


Mastitis



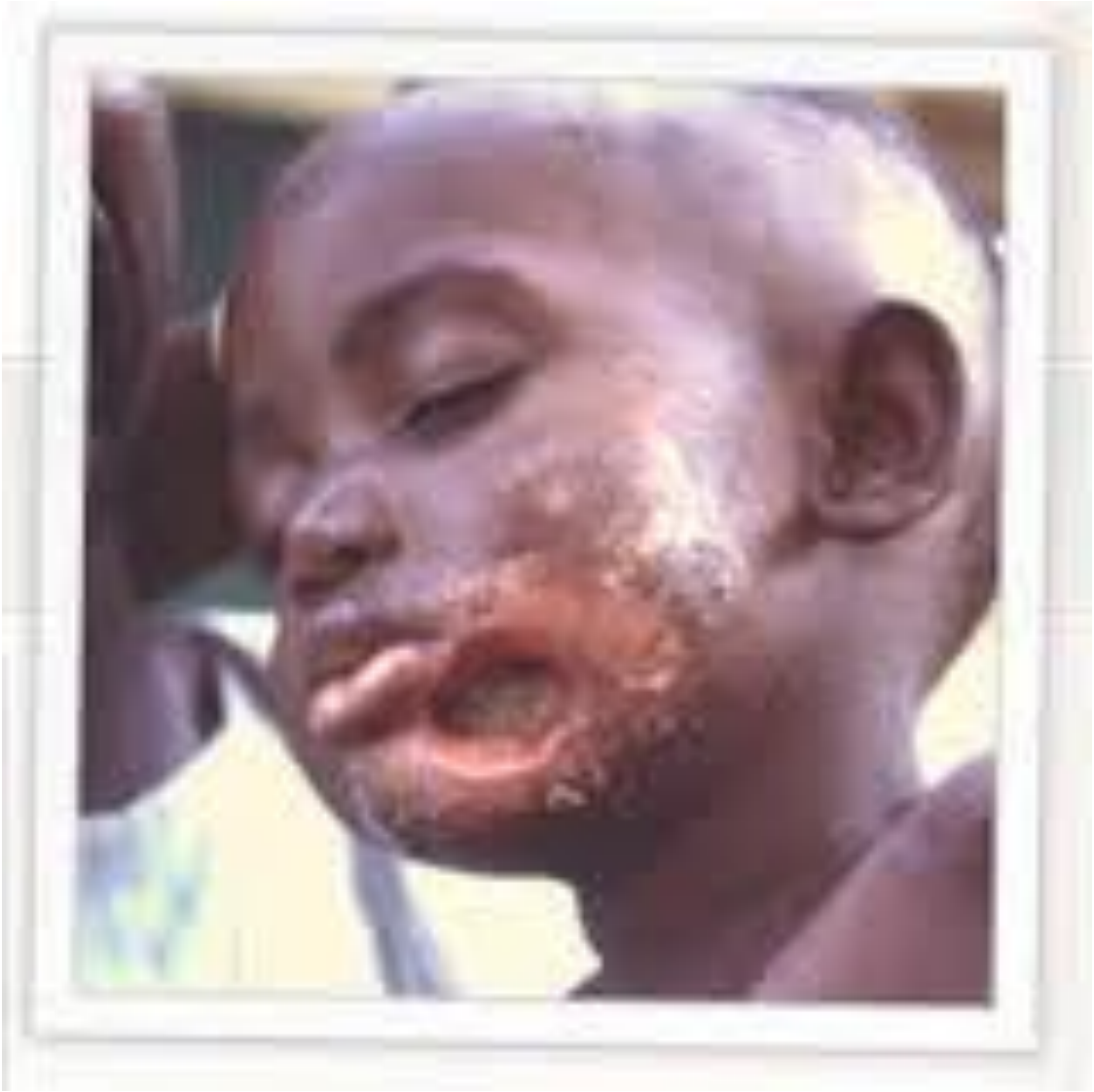
Infectious endocarditis

**Gangraenous, ichorous inflammation** failure of inflammation, heterolysis of tissues

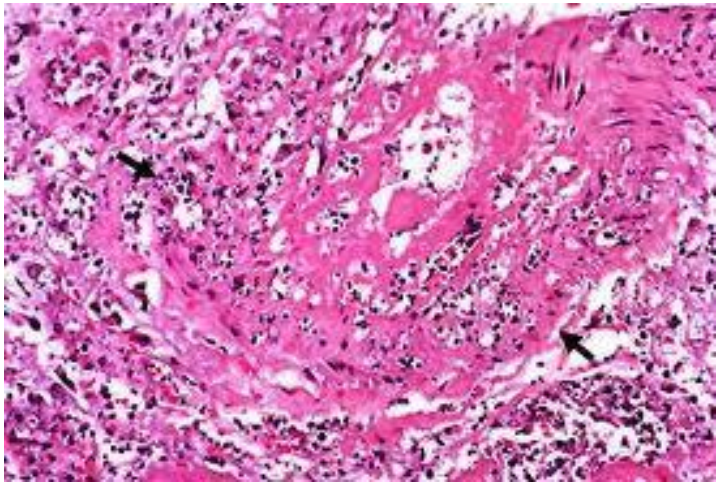


Gas gangrene, aspiration pneumonia, gangrenous pulpitis

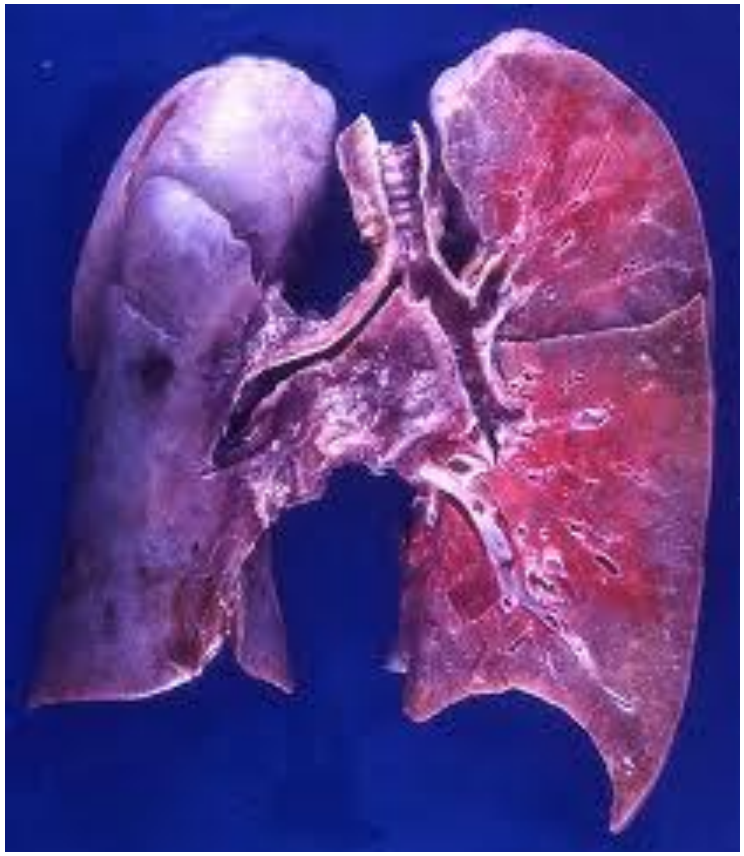
Stomatitis gangrenosa,



Aspiration (gangrenous) pneumonia

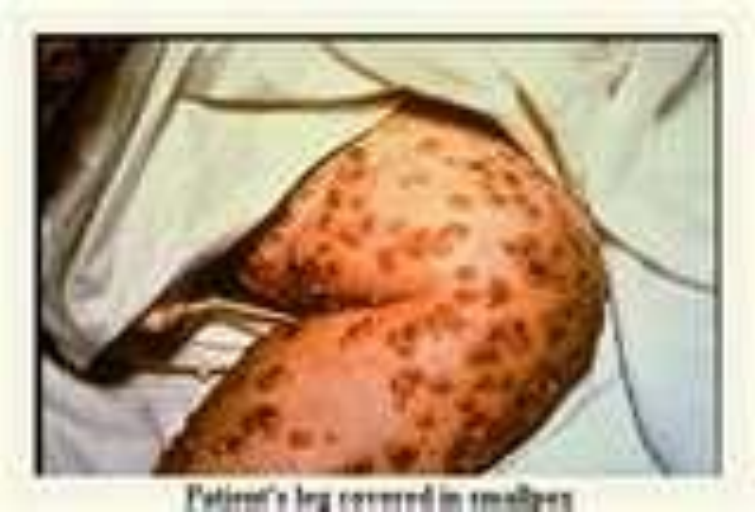


**Haemorrhagic inflammation** plague (black death), smallpox, athrax, influenza

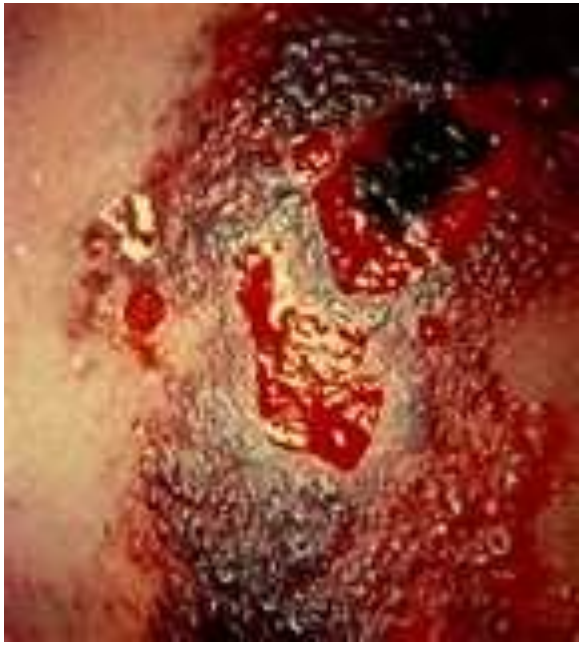


Hemorrhagic pneumonia in flu

# Haemorrhagic inflammation

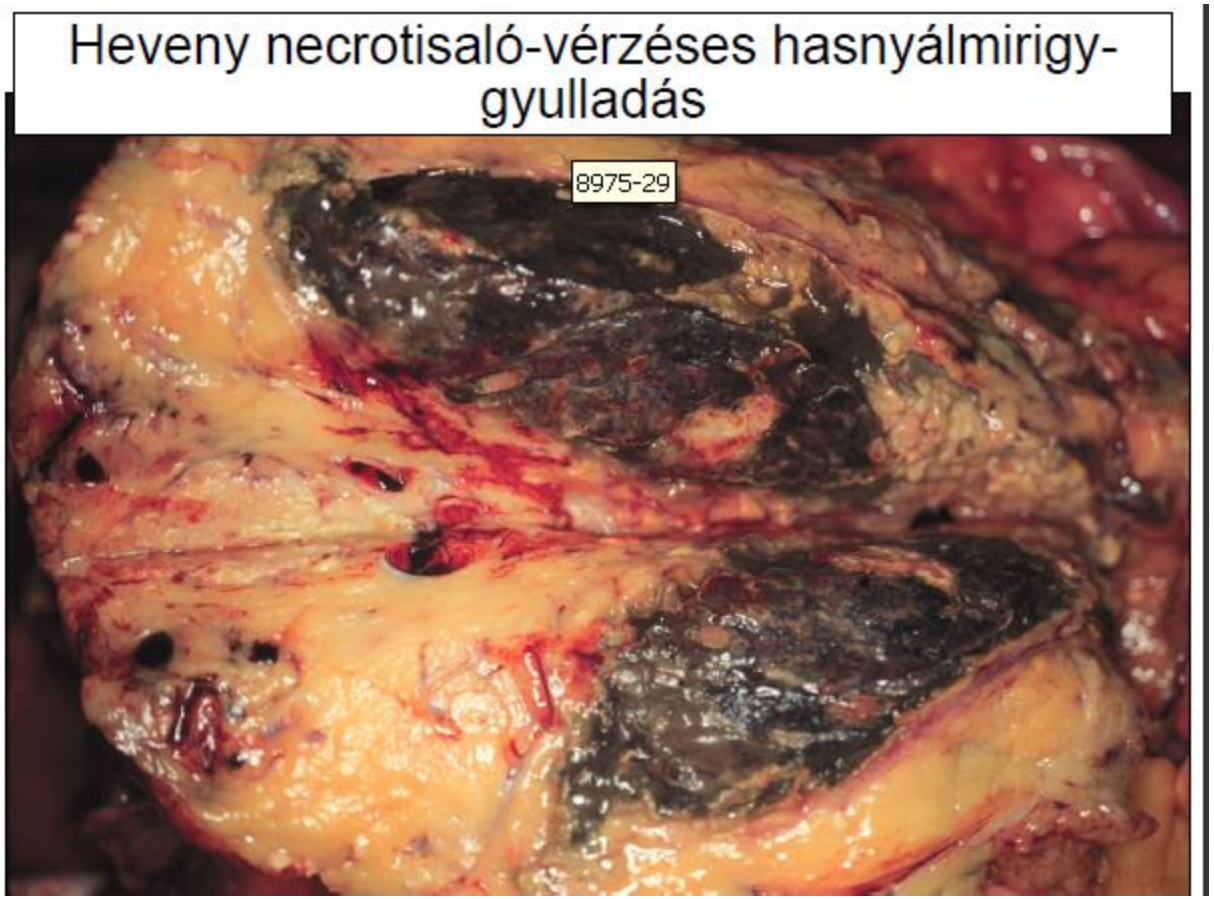


Variola vera
(smallpox)



Skin anthrax

Necrotizing, haemorrhagic pancreatitis



**Chronic inflammation**: acut inflammation is unable to eliminate the cause or it can be chronic from the beginning

Not exsudative proliferative

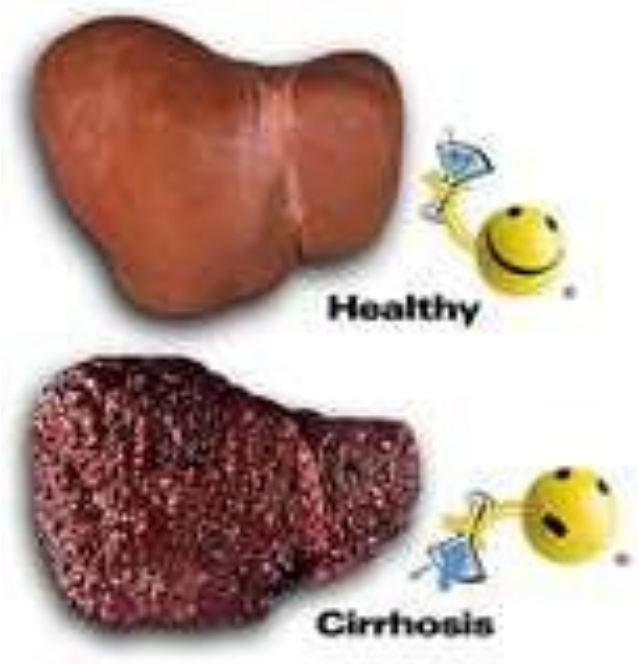
„small round cell „ infiltration

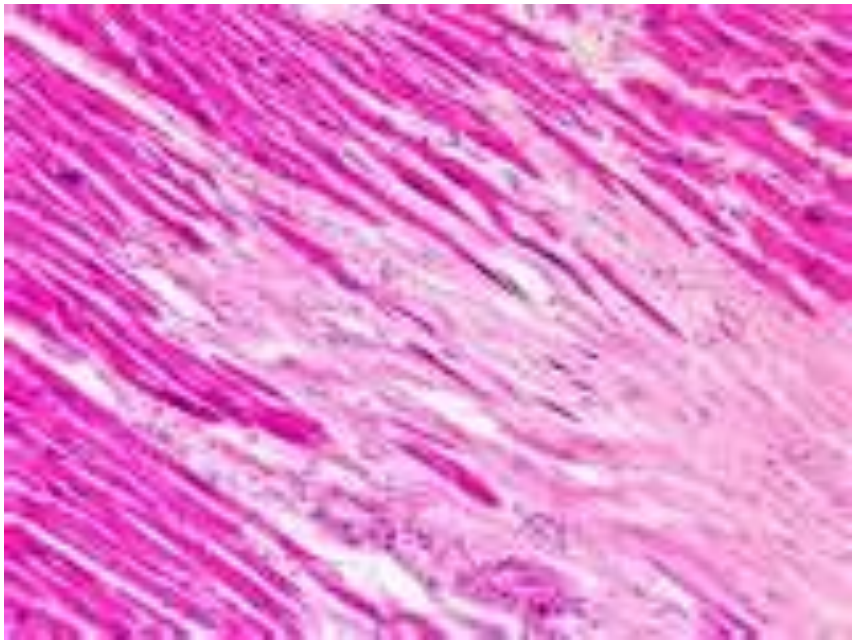
Fibrosis scaring- may destroy the parenchymal tissue

Pleuritis chr. adhaesiva

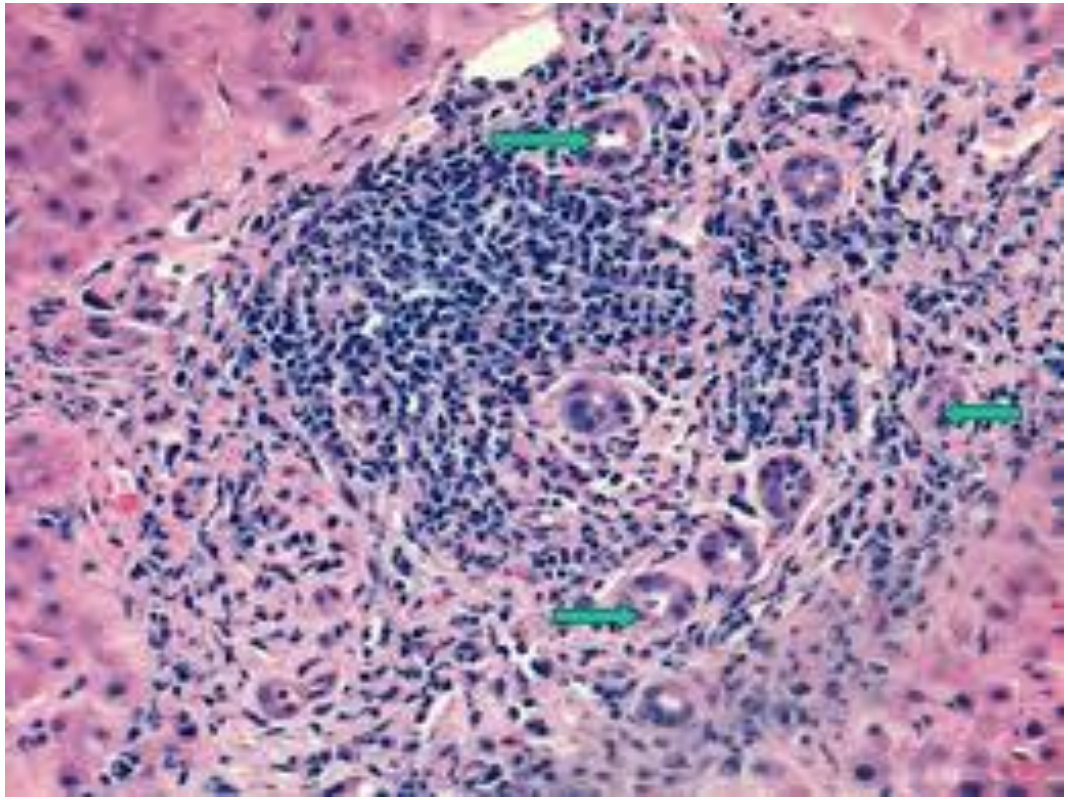
Cicatrix myocardii

Cirrhosis hepatis

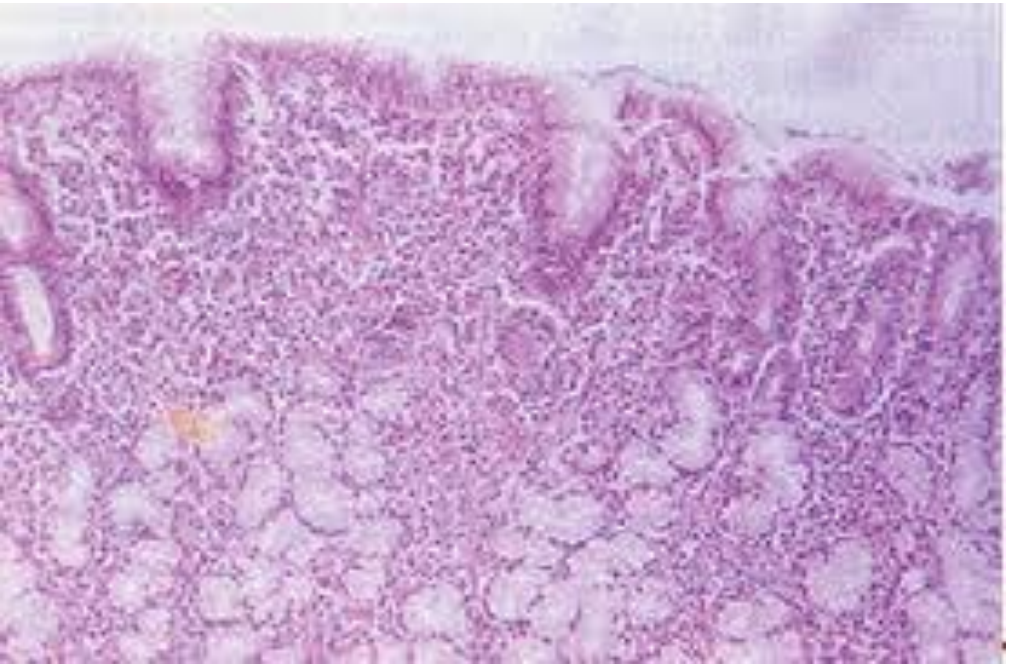


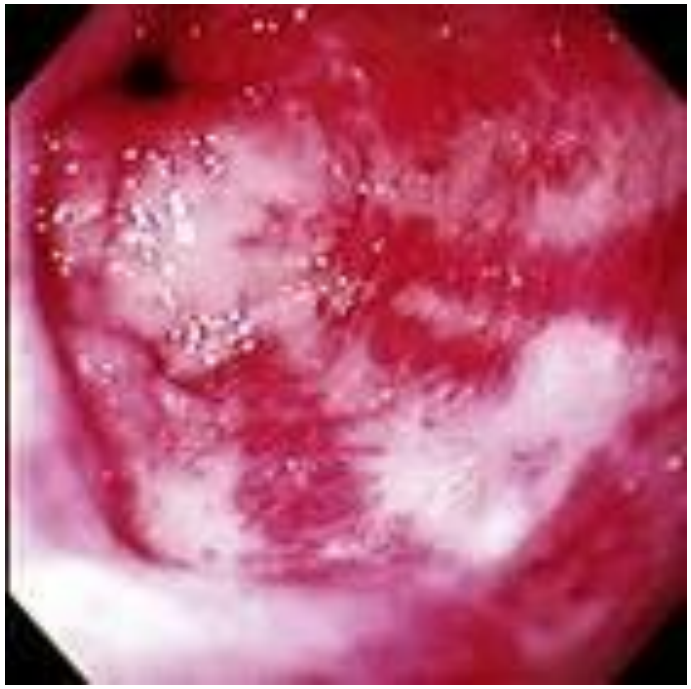



Forms of hepatitis:
Acute
Fulminant
Chronic



Chronicus hepatitis





Chronic gastritis

Forms: **A** utoimmune
**B**acterial (Helicobacter p.)
**C**hemically induced

Granulomatous (specific) inflammation

Charactesistic cellular elements are the epitheloid cells (macrophage origin)

Giant cells are frequent (Langhans , foreign body, Touton type)

Classification of granulomas:

Granulomas a/ necrotising (tbc, lues, rheumatic nodule)

b/ non necrotising

Non immunogenic granulomas: Foreign body- lipogranulomas

(non immunogenic materials)

**Immungranulomas**

Infectious granulomas

TBC most common infectious disease on the world

Cause: Mycobact. tuberculosis

Primary (Ranke-Ghon complex (tuberculoma,lyphangiitis, lymphadenitis

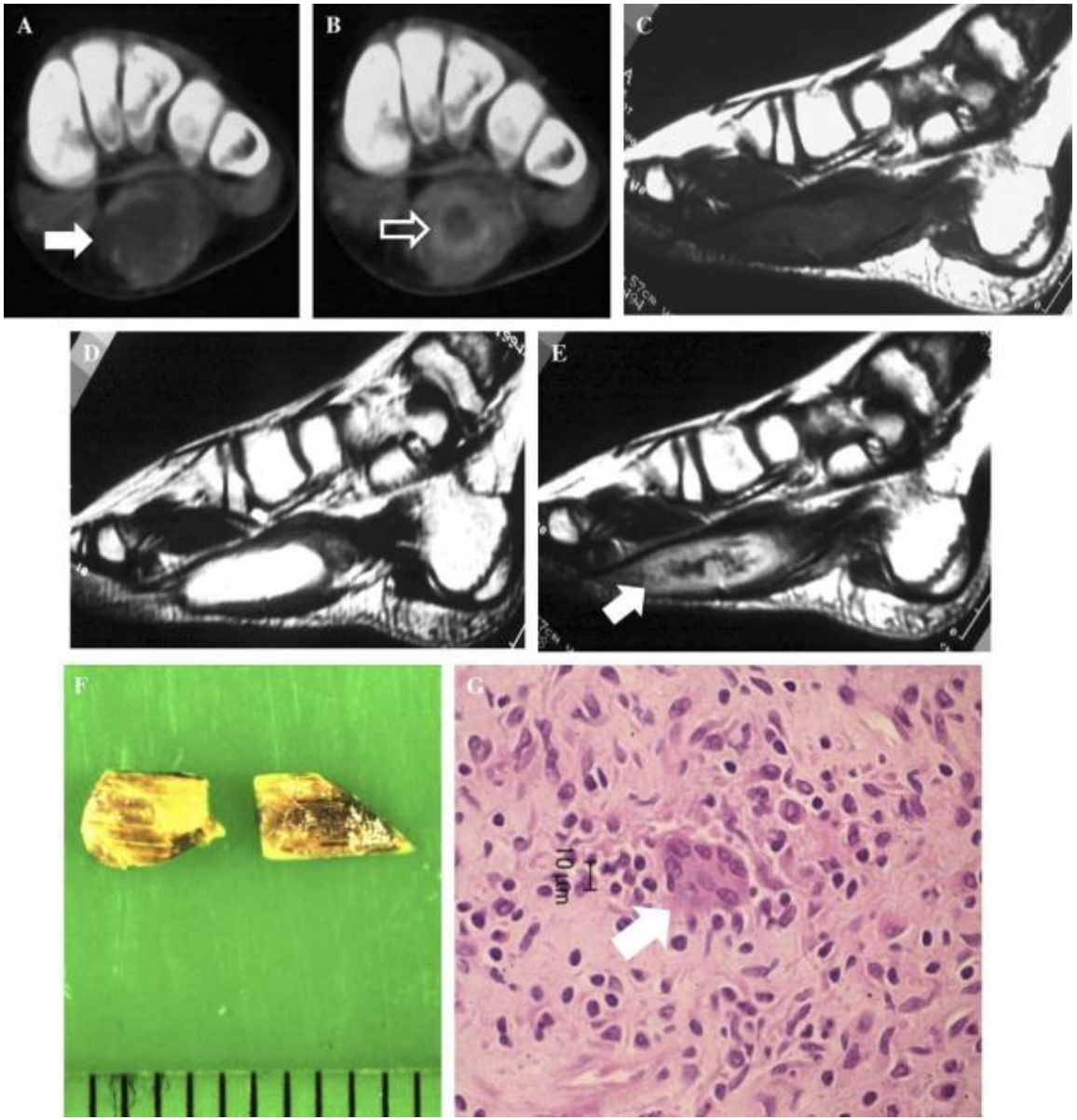
Secondary

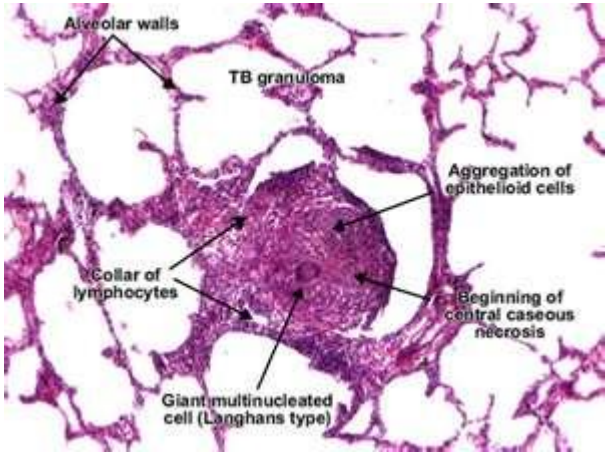
Components of tuberculoma : caseous necrosis, epitheloid-, Langhans type giant cells, lymphocytes

Demonstration of pathogen: Ziehl-Neelsen, auramine, PCR

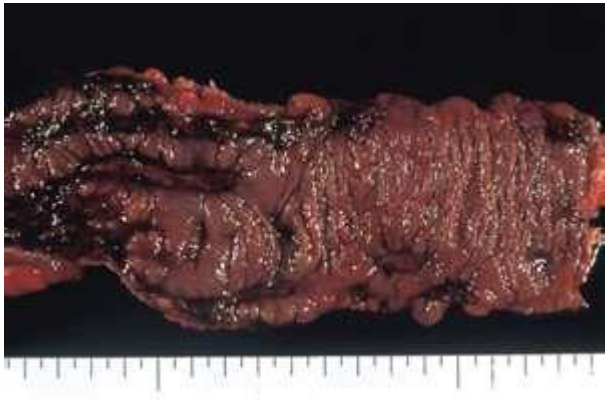
Spreading:local, lymphogenic, haematogenic, canalicular, serous membranes

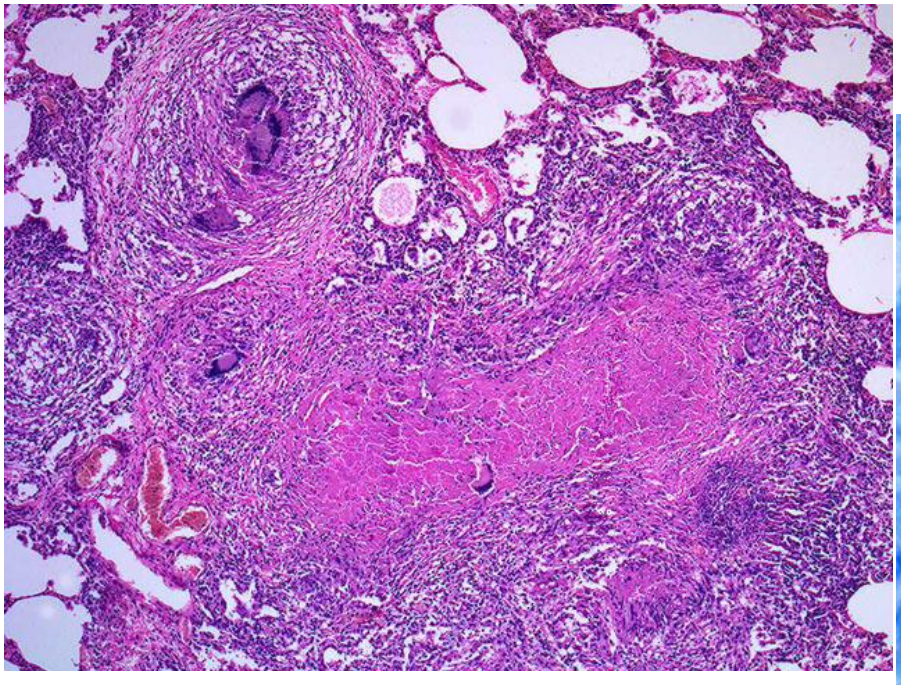
# Foreign body granuloma



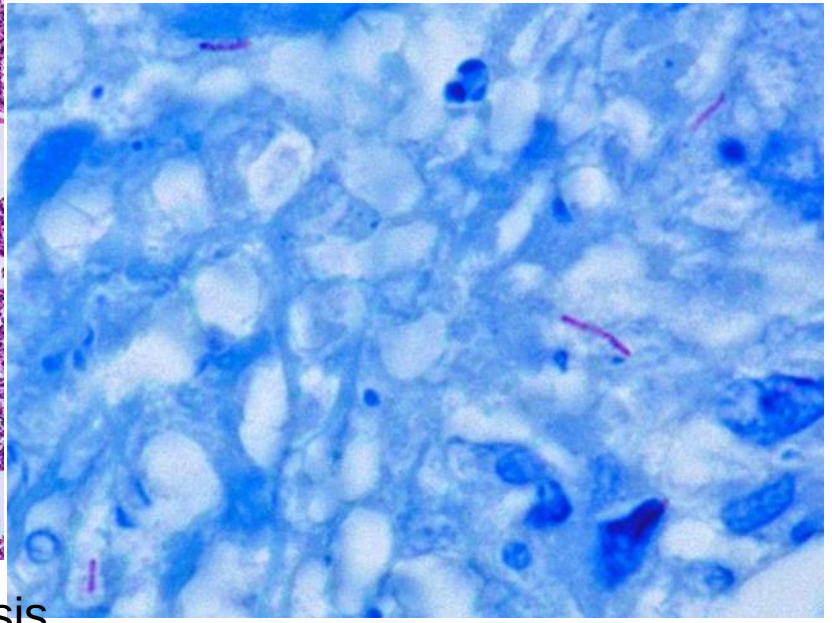



# Tuberculosis

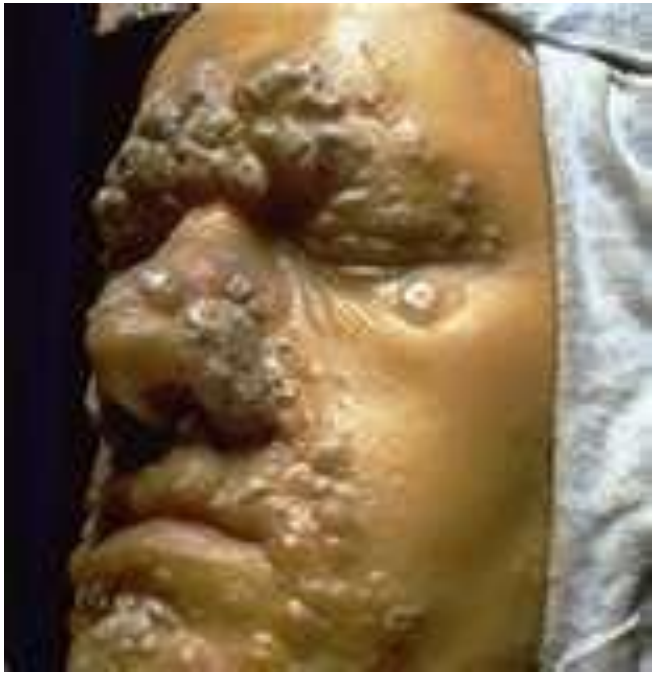




Components of tuberculoma :necrosis, epitheloid-, giantcells, lymphocytes

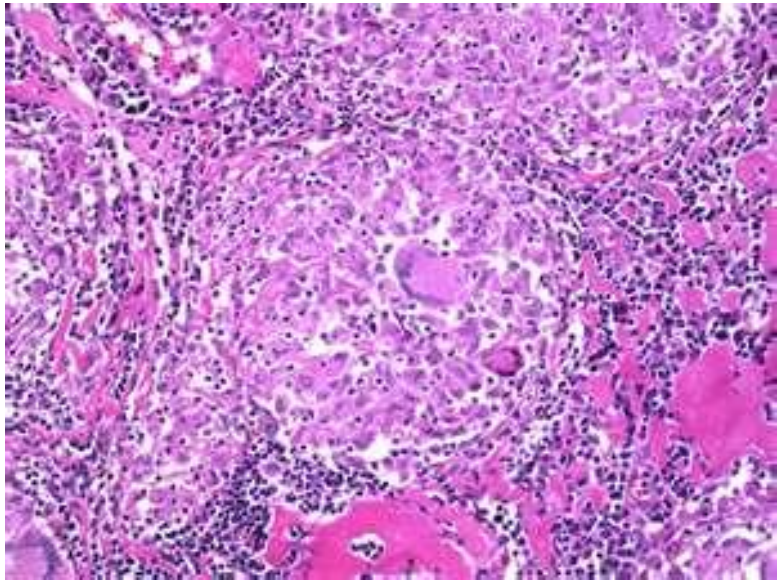


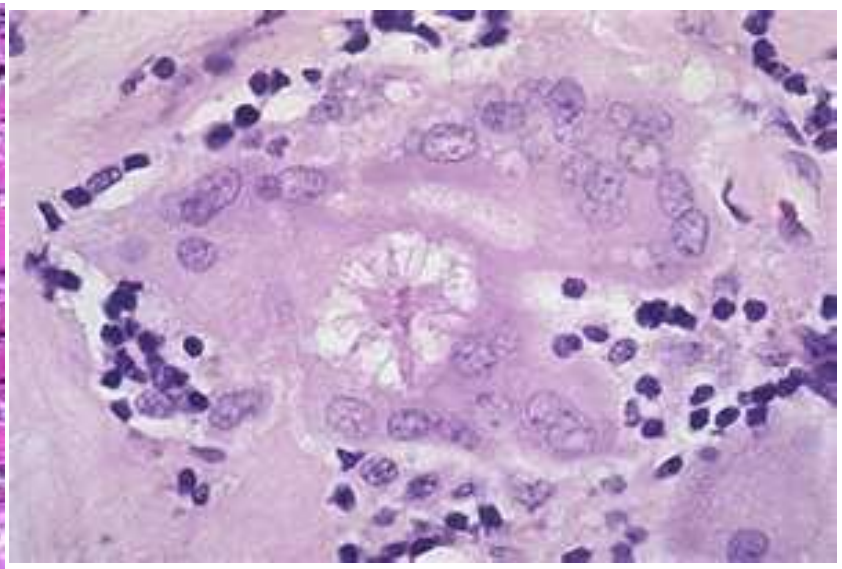
Ziehl-Neelsen (Acid fast) staining



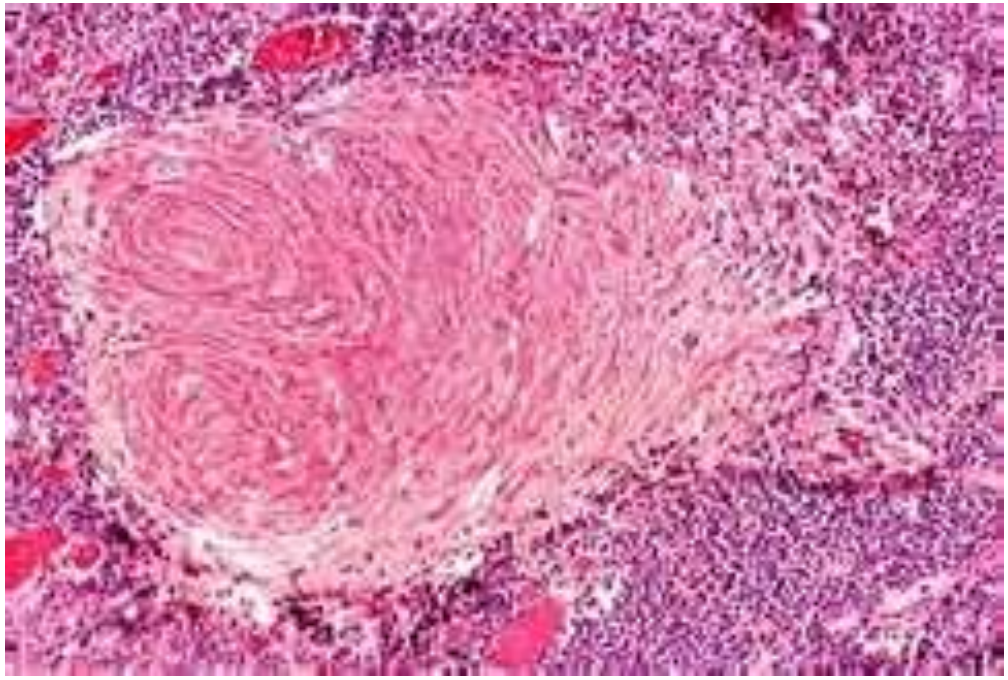
Syphilis, lues — cause Treponema pallidum

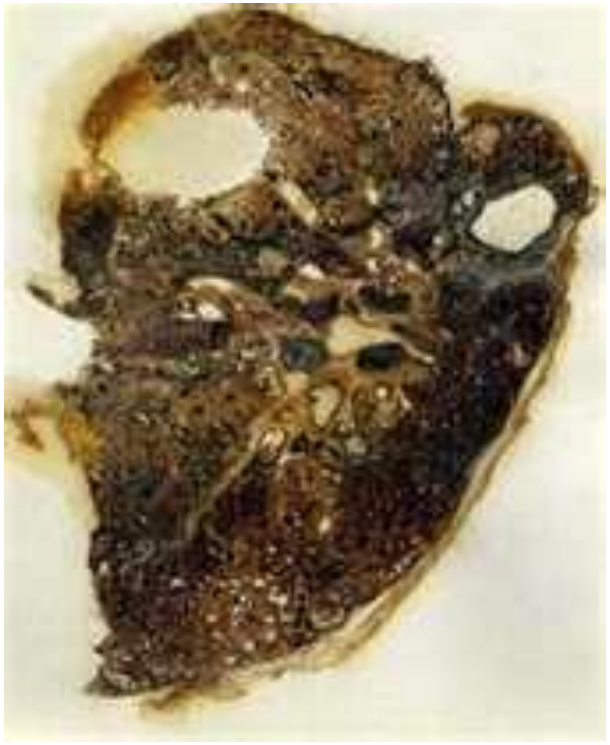
- Acquired
  - primery:ulcus durum, bubo indolens
  - Secundary: bacteriaemia, exanthaemas
  - Tertiaer: granulomatous – gumma
    - Lues
      - Cardiovasc: aneurysma
      - Neuoro sy: tabes dorsalis, paralysis prgr.
- Connatal



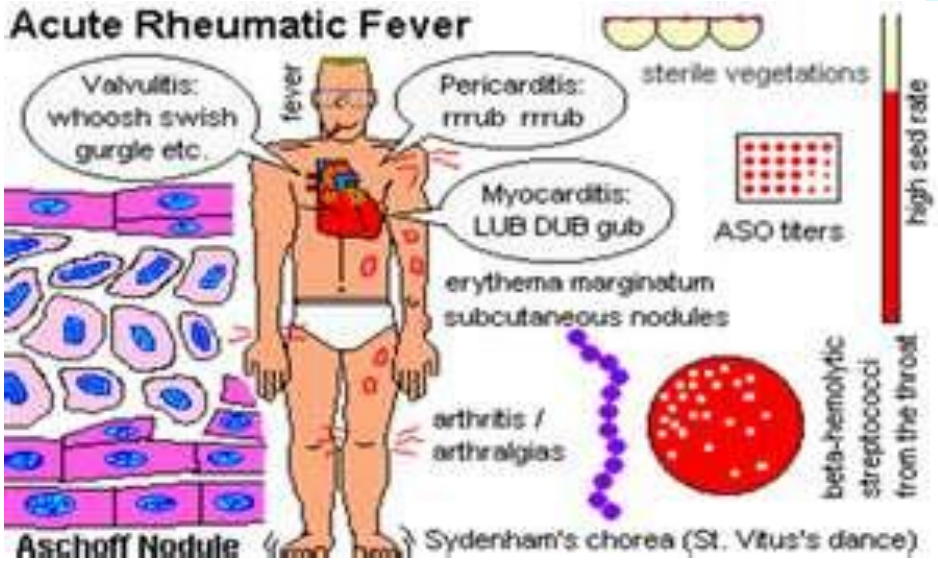


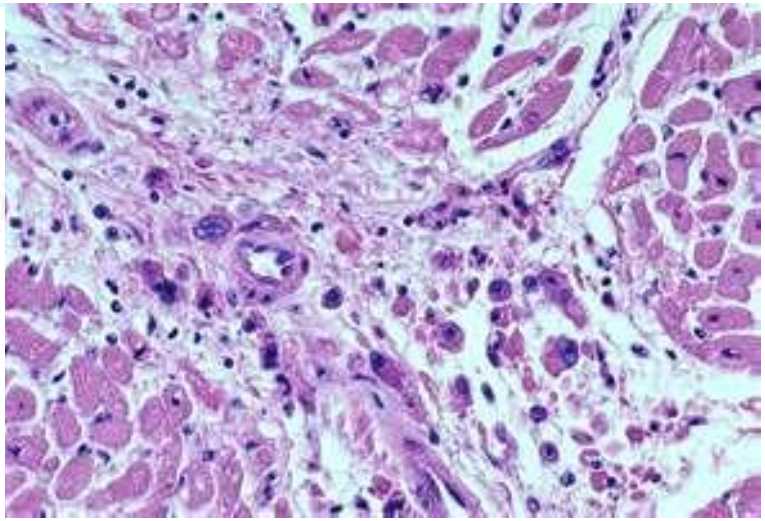
Sarcoidosis





Silicosis



<u>Rheumatic fever</u>

Rheumatic pancarditis

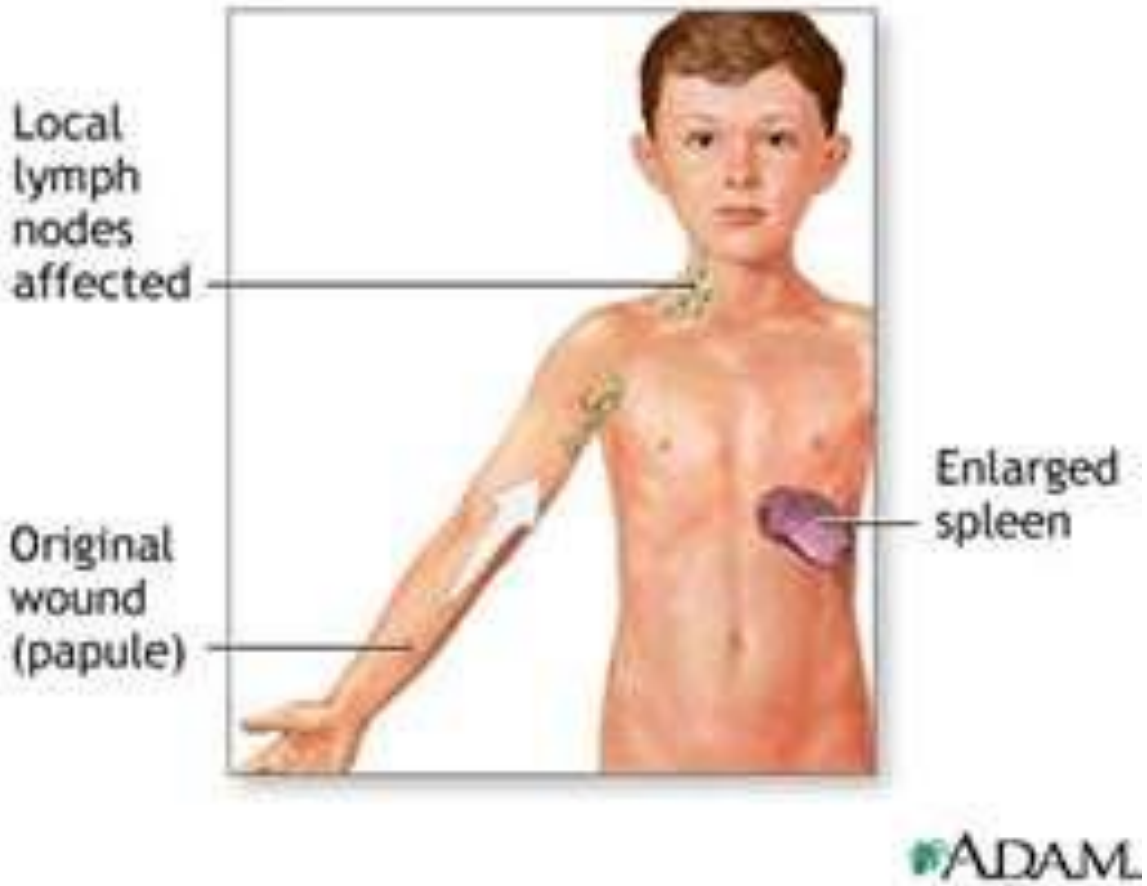
Polyarthritis

Rheumatic nodule

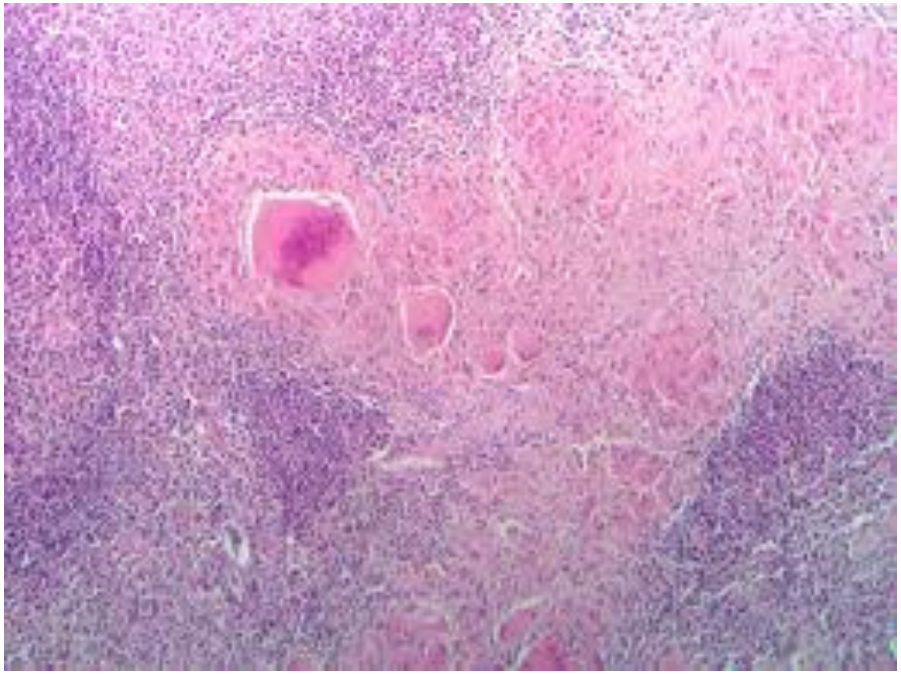
Erythema marginatum

Granulomatous myocarditis

(Aschoff –Talalajev nodule)




Cat scratch disease (Bartonella henselae)



Lymphadenitis mesenterialis (abscedasing histiocytic lymphadenitis)

Syphilis, lues — cause Treponema pallidum

Acquired form — primer:ulcus durum, bubo indolens
Secunder: bakteriaemia, exanthaemas
Tertiaer: granulomatous – gumma
Cardiovasc: aneurysma

Neuoro sy: tabes dorsalis, paralysis prgr.

Connatalis

Lepra tuberosus- lepromatosus

Cat scratch disease, Whipple disease, Brucellosis, Schistosomiasis

Non infectious immungranulomas

Sarcoidosis

Rheumatic fever: pancarditis, Aschoff granuloma, (Anichkov giant cell)

Primary bilary cirrhosis, Crohn, Wegener

## Harmfull consequences of inflammation

| | |
|---|---|
| Overshooting | A/inflammatory raction again harmless lesions: appendicitis, crystals<br>B/hypersensitive ,autoimmune diseases<br>C/Too intense reaction: endotoxin shock, ARDS sepsis (infectios SIRS) |
| Chronic inflammatory reactions caused fibrosis | |
| Failure of inflammation | a/congenital defects: neutropenia, LAD, chronic. granulomatous disease<br>b/acquired lesions: neutropenia, Splenectomy<br>Saturation of macrophages (hemolysis, immunocomplex diseases)<br>Leukocyte malfunction (alcohol, extensive burns)<br>failure of blood supply, diabetes<br>malignant tumours |

## General effects of inflammation

Fever,
Leukocytosis,
Acut phase reaction
(Negative: albumin)
Positive: fibrinogen-increased sedimentation rate,
Lactoferrin, coeruloplasmin
SAA – amyloidosis