Acute Inflammation III

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Factors determining variation in inflammatory response

I Factors involving organisms

1.Type of injury & infection-
lung to pneumococci & Tb bacilli

2. Virulence-high & low

3. Dose- small & large

4. Portal of entry-

5. Product of organism- streptokinase, coagulase staphylokinase

II Factors involving host

1. General health/Systemic diseases- DM
2. Immune state
3. Leukopenia -spreading infections
4. Type of tissue involved- Lung vs Bone
5. Local host factors- Ischaemia, foreign bodies

III Type of exudation

1. Serous -- blister in burns,pleural effusion
2. Fibrinous --- Rheumatic pericarditis
3. Purulent – Abscess
4. Haemorrhagic – haemorrhagic pneumonia
5. Catarrhal – Common cold

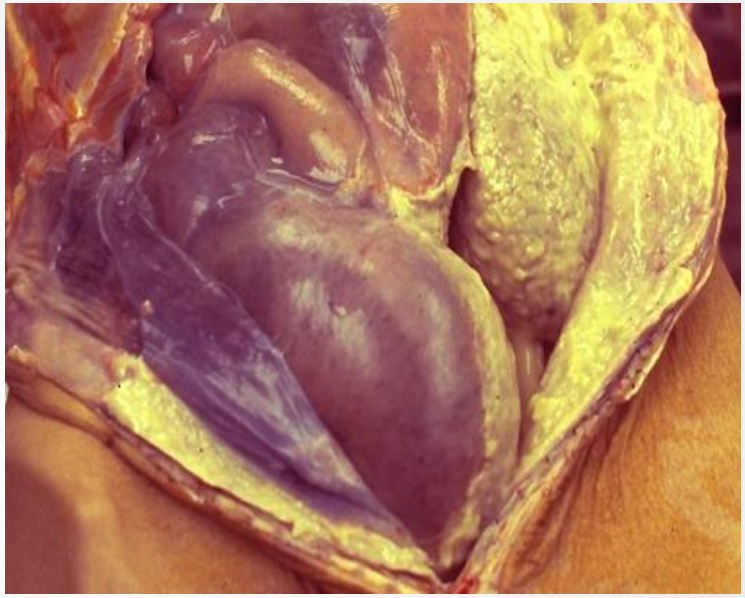
Morphological Types

1. **Acute Catarrhal inflammation – common cold**
2. **Serous inflammation** – pleura, peritonium

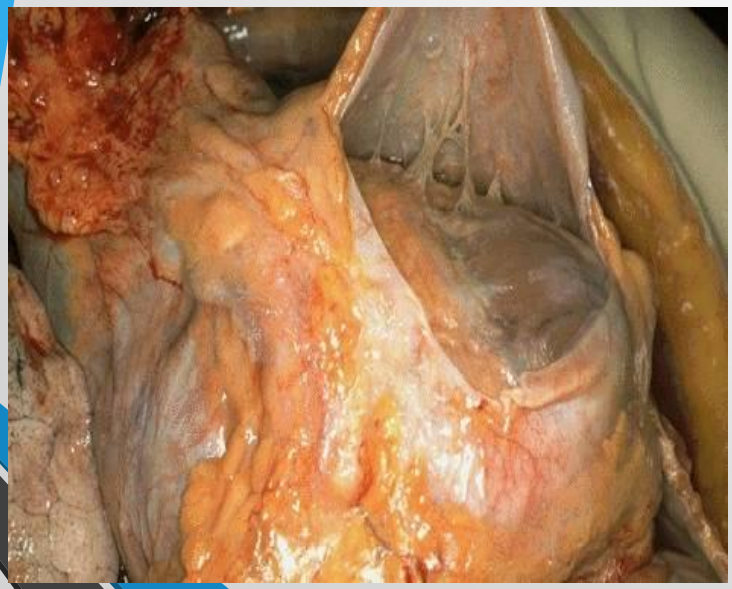
3. **Acute fibrinous inflammation** – Pleura, Peritonium
   bread & butter pericarditis

4. **Acute purulent inflammation** – pus formation
   - abscess & cellulitis
5. **Acute necrotising (pseudomembranous) Inflammation**
   -Diphtheria, Bacillary dysentery
6. **Allergic inflammation**

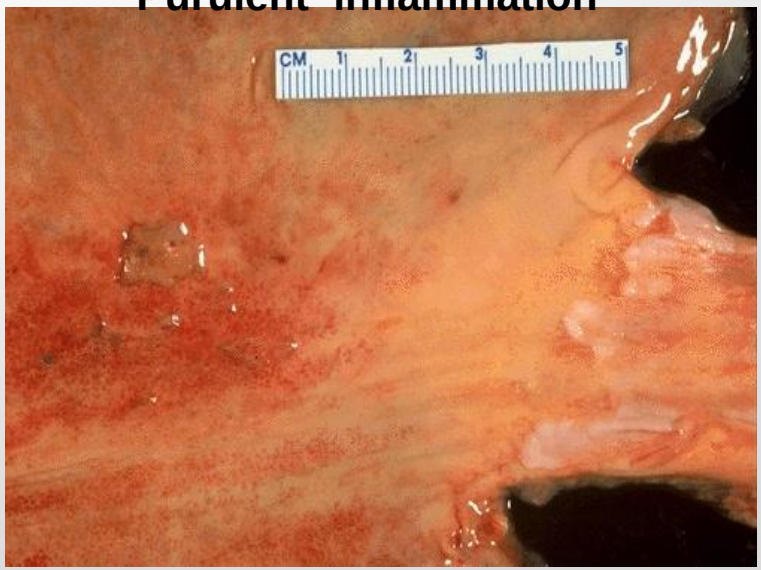
Serous inflammation



Purulent inflammation

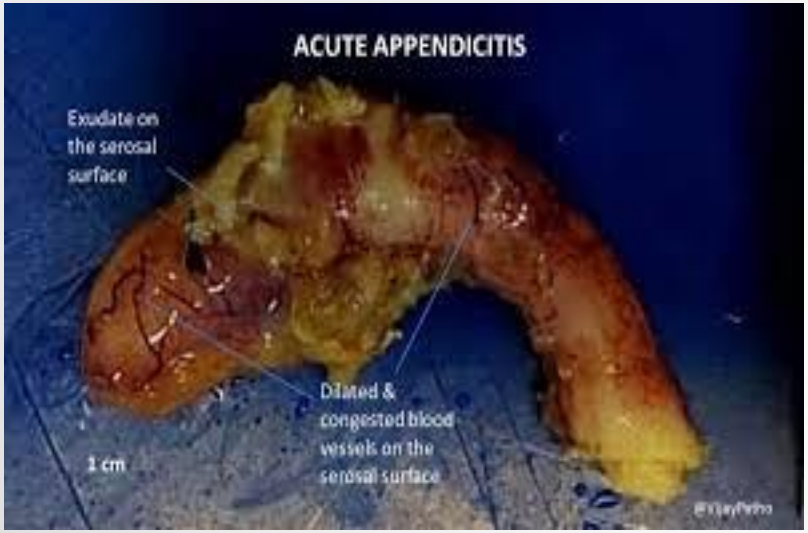


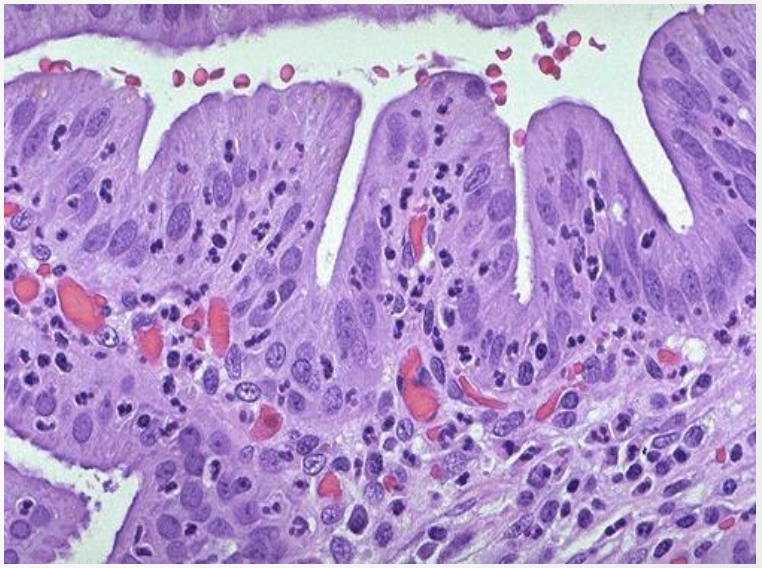
Fibrinous inflammation

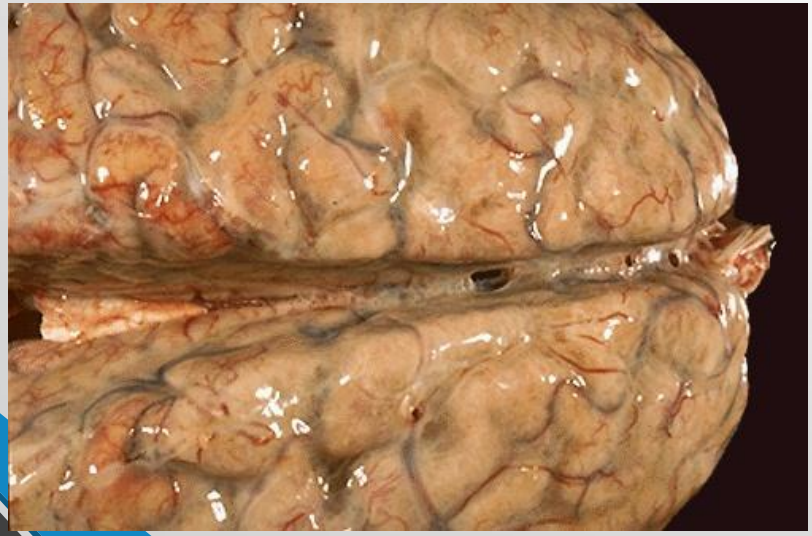


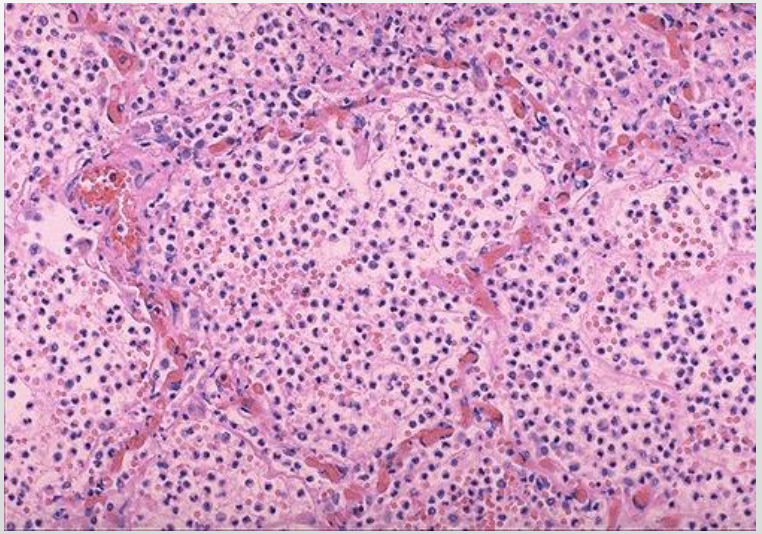
ulcers

Acute Inflammation

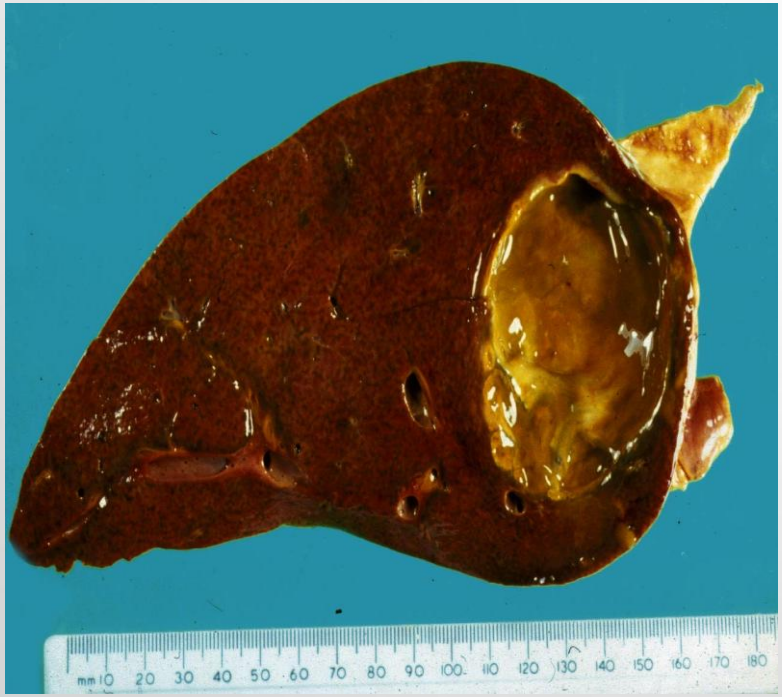








Hepatic Abscess



Cellulitis

7. **Ulcer** – local defect of the surface of an organ that is produced by sloughing of inflammatory necrotic tissue.

8. **Cellulitis** – diffuse inflammation of soft tissues.

9. **Bacterial infection of blood**

   a. Bacteremia – presence of bacteria in blood

   b. Septicaemia -- presence of rapidly multiplying bacteria in blood

   c. Pyaemia -- septicaemia + multiple pyaemic abscesses

   d. Toxaemia – presence of circulating toxin blood

Systemic effects of acute inflammation
Acute Phase Response

- **Fever (temperature > 37.8°C or >100 F)**
- **Leukocytosis**
  - **Neutrophilia** and left shift of neutrophils points to bacterial infection
  - **Lymphocytosis** points to viral infection
  - **Eosinophilia** point to allergy or parasitic infection
- **Acute phase protein production in liver**
  - fibrinogen, CRP,SAA leads to increased ESR

- **Lymphangitis-Lymphadenitis**
- lymphatics & LNs draining the inflamed tissue show reactive changes
- **Septic shock-** in severe & fulminant cases

  massive release of TNFά-profuse systemic vasodilatation ,increased vascular permeability→ hypotension & shock

  High levels of cytokines cause widespread clinical manifestations such as disseminated intravascular coagulation, hypotensive shock, and metabolic disturbances including insulin resistance and hyperglycemia. This clinical triad is known as septic shock.

Termination of acute inflammation

- Eradication of an offending agent should lead to discontinuation of the inflammatory response
- Neutrophils have only a short life span (few hours -1 day)
- Most mediators are very short lived and are degraded immediately

However, the exact mechanisms by which acute inflammation resolves remain still somewhat elusive

Outcome of acute inflammation

- Complete restitution
- Abscess formation (encapsulation and pus)
- Chronic inflammation
- Healing with scar formation

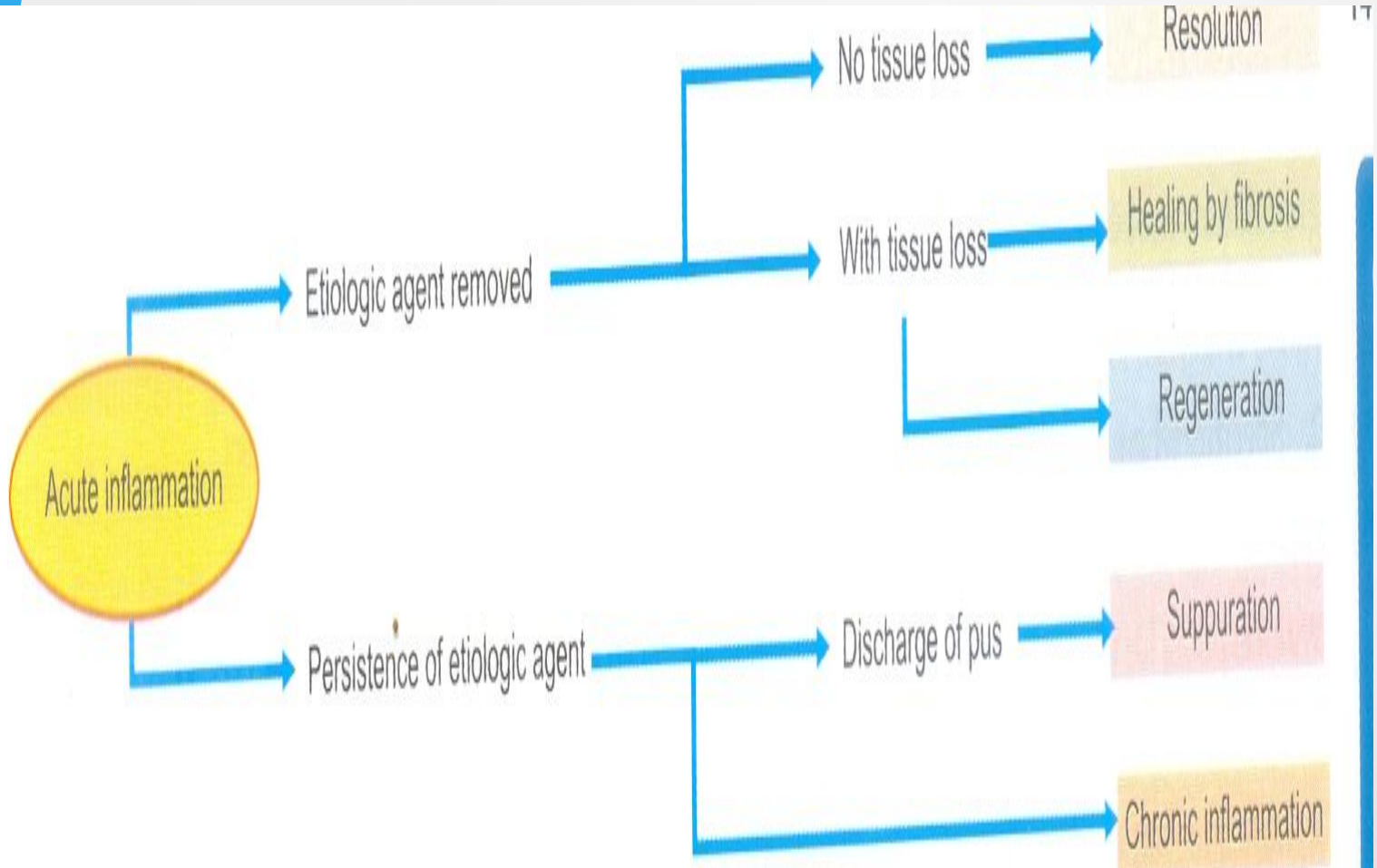


Figure 6.17 Fate of acute inflammation.