

# INFLAMMATION

## L28- CELLS OF INFLAMMATION AND TYPES OF INFLAMMATION

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### 4. NATURAL KILLER CELLS (NK)

- These are related to CTL but most NK cells do not have TCRs
- They can recognize cells with non-self MHC molecules.

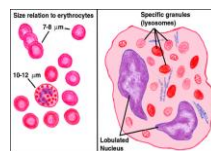
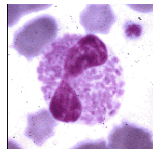
### 5. PLATELETS

- Coagulation of blood
- Platelets are activated by collagen
- Release the contents, e.g., ADP, serotonin, PAF, fibrinogen, hydrolytic enzymes and AA metabolites.
- The released ADP, TXA<sub>2</sub>, PGs leads to aggregation of platelets and eventually plug the damaged vessel.
- Platelets secrete permeability enhancing protein — push neutrophils out of vessel.

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### 6. EOSINOPHILS

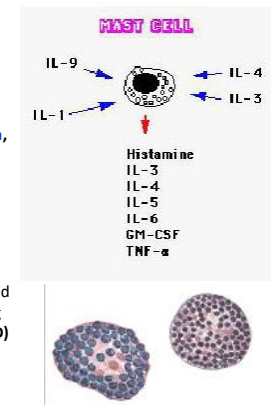
- Reside predominantly in sub-mucosal tissue
- Contains large ellipsoid granules
- They contain four distinct cationic proteins those exert a range of biological effects on host cells and microbial targets:
  - major basic protein (MBP) -- Parasite killing
  - eosinophil cationic protein (ECP)
  - eosinophil derived neurotoxin (EDN)
  - eosinophil peroxidase (EPO).
- These contents act mainly on extracellular target structures such as parasites
- Have limited phagocytic activity
- Contribute to chronic inflammation, including fibrosis
- Have a short life span.
- Have receptors for IgE



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### 7. BASOPHILS AND MAST CELLS

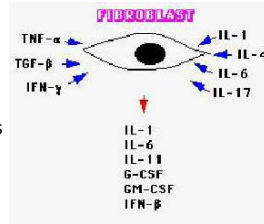
- Basophils are circulating cells (<1%),
- Mast cells are present in perivascular sites in skin, GIT and Respiratory tract.
- Both – similar in function and contents.
- The granules of the cells contain heparin, histamine, proteases and other mediators.
- Both cells have receptors for IgE
- Mast cells and basophils play a central role in inflammatory and immediate allergic reactions.
- Newly generated mediators are produced during IgE-mediated activation including AA metabolites (principally LTC and PGD) and other cytokines.



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**7. ENDOTHELIAL CELLS AND FIBROBLASTS**

- **Endothelial cells** interact with leukocytes
- Adherence and **emigration** process
- **Healing** and **repair** responses.
- **Fibroblasts** participate in the **healing** and **repair** process
- Products of fibroblasts **activate leukocytes** and induce **locomotion**.



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**TYPES OF INFLAMMATION**

- **Inflammatory Reaction/Response Depend On:**
  - inciting agent
  - the host factors

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**Inflammatory Reaction Varies On Basis Of :**

EXTENT	DURATION	DISTRIBUTION	EXUDATE
Mild	Peracute	Focal	Serous/Heamorrhagic
Moderate	Acute	Multifocal	Catarrhal
Severe	Subacute	Diffuse	Fibrinous
			Purulent
			Proliferative
			Granulomatous

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**1. DEGREE OF SEVERITY**

- inflammatory reaction can be
  - mild,
  - moderate and
  - severe.

**MILD**

- Little or no tissue destruction
- Have **slight evidence** of vascular involvement (hyperaemia and oedema)
- **Little exudation**, non-suppurative, e.g., mild bronchitis, mild catarrhal enteritis, mild stomatitis as in FMD.

**MODERATE**

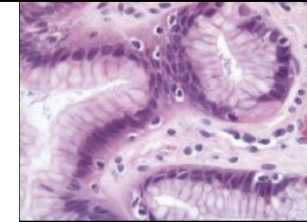
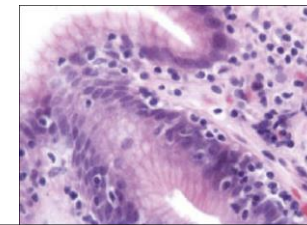
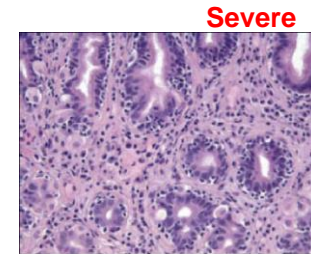
- **Some damage** to tissue
- **Visible host reaction** (hyperaemia, oedema and leukocyte accumulation), e.g., moderate enteritis, moderate bronchitis.

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**SEVERE**

- Considerable tissue destruction
- Abundant exudation of plasma and leukocytes, e.g., severe bronchopneumonia as occurs in *Pasturella* infection in sheep and buffalo, severe hepatitis in **canine distemper**.

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**Mild****Moderate****Severe**

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**2. DURATION**

- On the basis of time factor,
- With increase in time the inflammatory reaction changes and can be:
  - Per-acute
  - Acute
  - Sub-acute
  - Chronic

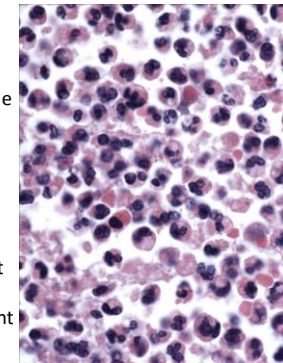
**PERACUTE**

- Manifestation developing in **short time**, i.e., in only few hours (~less than 4 hours)
- Usually caused by **potent stimuli**.
- Host response is minimal and **without much exudation**.
- The **tissue damage variable**
- Very **less vascular involvement** (slight oedema, hyperaemia, haemorrhage with few leukocytic infiltrates).
- Per-acute inflammatory response is **less frequently seen**.

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**ACUTE**

- Inflammation begins within few hours, i.e., **4-6 (hours)** and continues for several days.
- **Vascular response** is clearly visible (excessive vascular dilatation).
- **Oedema**
- **Haemorrhages** (may be)
- All the **cardinal signs** of inflammation are present.
- **Neutrophils** are the predominant cells although sometimes mononuclear cells may be present in large numbers.



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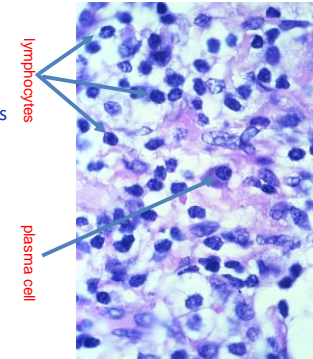
### SUBACUTE

- Decline of vascular response (hyperaemia and oedema)
  - Oedema fluid drained by lymphatics
- Change in type of leukocytic infiltrate (may be)
  - e.g., lymphocytes, macrophages and may be plasma cells.
- Occur in about few days -- few weeks.

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### CHRONIC

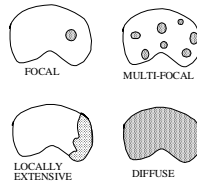
- Involvement of host tissue reparative process.
- Persistence of causative factor
- Predominant cells are lymphocytes and macrophages.
- The evidence of repair
  - regeneration
  - fibrosis
  - granulation tissue.
- May develop after an acute episode
- May develop as chronic from beginning, as in TB



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### 3. DISTRIBUTION OF INFLAMMATION

- Distribution in the tissue
  - focal
  - multifocal
  - locally extensive
  - diffuse



#### FOCAL

- Small single area in a tissue (radius of 1mm or less or extending up to a centimeter or slightly more)
- Most often no leukocytosis in the blood picture
- Mostly resolve quickly.

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#### LOCALLY EXTENSIVE

- Involve a considerable area of a tissue.
- It may arise from a single focal inflammation spreading now to a wider area or may arise from coalescence of multiple foci.
- This type of inflammation usually has bacterial aetiology.

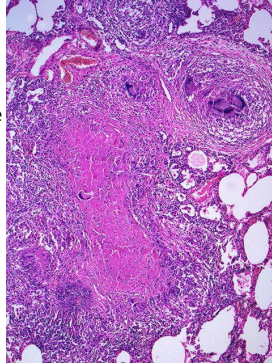
#### DIFFUSE

- Involve all the tissue or organ.
- At certain areas the inflammation is more severe, while at other is not so
- Such lesions are often of viral aetiology
- Some of the toxicant also produces similar lesions.

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**MULTIFOCAL**

- Multiple small scattered foci of inflammation in a tissue.
- Between those inflamed areas is normal tissue.
- Such lesions occur most often due to vascular basis, e.g., spread of septic emboli from an inflamed heart valve.
- Inflammatory reaction in **tuberculosis** is mostly of this type where multiple granuloma formation takes place although diffuse reaction can occur simultaneously.



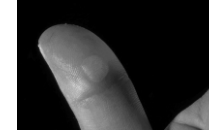
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**4. TYPE OF EXUDATE**

- Nature of the components of an inflammation
  - fluid, protein contents and cellular components

**SEROUS**

- Exudate largely contain **fluid rich in proteins**, e.g., blisters produced in burns.
- With the passage of time fibrinogen exudes, form fibrin, the exudate is sero-fibrinous.
- Later, neutrophils exudes, exudate is termed as fibrinopurulent.
- At the end, the blister is ruptured and fluid is drained and absorbed leaving fibrin and debris to form a scab.



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- The very early exudate in most inflammations is serous in nature
- The serous exudate dilutes the irritant in the tissue
- In flu, the running nose condition is serous exudate.
- **Albumin** is the first protein to leak from the vessel having the lowest molecular mass (69,000), next come the **IgG** (γ-globulin; 150,000) and being the bigger **fibrinogen** (340,000) leaks late.
- All the proteins may leak simultaneously but the speed to reach at the site of inflammation may vary according to their molecular weight.
- The serous exudate also acts as a good source for bacterial proliferation being good medium.
- However, the amount of antibodies present in the serous exudates are important,

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**Grossly**

–the exudate is fluid in nature and easily drools on opening of the cavity/blister.

**Microscopically**

–it stains pink and appears homogenous granular in sections.

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