**Mediators of inflammation**

There are two types of mediators of inflammation.

1. Cell derived mediators

2. Plasma derived mediators

**1. Cell derived Mediators:**

Cell derived mediators are stored inside the granules of cells and released by a process called “granular exocytosis”

For example: Histamine is stored inside the granules of mast cells and released when there is degranulation of mast cells.

All cell derived mediators are not store inside the granules but they are synthesized when they are needed.

Two types of synthesis are

**Pre-form mediators**:

Histamine is stored in the granules of mast cells and released by granular exocytosis.

**De-novo synthesis**:

Prostaglandins and cytokines are synthesized when they are needed.

**2. Plasma derived mediators**

These mediators are synthesized in liver and present in our circulation. They are inactive in circulation and become activated by

* Proteolysis
* Microbial product
* Necrotic substances
* Complement components

E.g. Bradykinins

**Properties of mediators:**

There are some properties of inflammatory mediators.

1. One mediator can stimulate the release of other mediator.

2. TNF-alpha stimulate the release of IL-

3. These mediators act on some specific target.

E.g. one mediator act on specific type of cell or selective for one cell.

4. These mediators can be rapidly destroyed.

5. These mediators have short life span and can be inhibited by some proteins

E.g. complement components can be inhibited by complement regulatory proteins.

6. There are some enzyme participate in destruction of inflammatory mediators

E.g. kinases enzyme are responsible for destruction of bradykinins

7. They are destroyed by some scavenger pathway

E.g. by use of anti-oxidants.

Examples

**Cell-derived mediators:-**

**Mediators Release Function**

Histamine Mast cells Vasodilation

 Basophils Increased vascular

 Platelets permeability

Serotonin Platelets vasoconstriction

Prostaglandins Leukocytes Increased vascular

 Mast cells permeability, Chemotaxis

 Leukocytes adhesion

Platelet activating Leukocytes Leukocytes adhesion

 Factors Mast cells

Reactive oxygen Leukocytes Killing process

 species

Nitric oxide Endothelial cells Relaxation

 Macrophages Killing process

 Cytokines Endothelial mast cells Endothelial activation

(TNF-X, IL-1 IL-6) mast cells Tissue damage

Chemokines leukocytes chemotaxis

 Activated

 Macrophages

**Plasma derived mediators:-**

**Mediators release participation**

Kinins From liver vasodilation

 Pain

Complement From liver chemotaxis

Components phagocytosis

 Leukocytes

 Activation

**Morphology of acute inflammation**

**1-Serous inflammation**

It is characterized by accumulation of protein poor fluid (transudate)

The fluid is derived either from the plasma of from the secretions of mesothelial cells lining the abdominal region cavities.

* Pleural cavity
* Peritoneum cavity
* Pericardial cavity

**2-Fibrinous inflammation**

Fibrinous inflammation results in case of severe injury and result in alteration of vascular permeability and cause leakage of fibrinogen. This fibrinogen appears as meshwork of cell known as “coagulum”

These fibrinogen rich exudate are killed by a process fibrinolysis and result in restoration of normal structure. This is called “Resolation process”

If fibrinogen rich exudates are not degraded, then it is replaced by engrowth of blood vessels and fibroblasts and result in organization or scar formation.

**3. Suppurative Inflammation:**

It is characterized by collection of prulent exudate. This prulent exudate exudate consists of neutrophils and necrotic cell.

This type of inflammation is due to pus forming micro-organisms staphylococci is responsible for this suppurative inflammation. This is also called pyogenic micro-organisms means pus forming organisms.

This local collection of pus is called “abcess formation”