**Inflammation**

It is a response of vascularized tissue against infection and tissue damage, that brings cells and molecules of host defense from circulation to the site where they are needed to eliminate the offending agent.

If no blood vessels are present as in avascularized tissue no inflammatory process will occur.

Inflammation is a protective, pathological and vascularized response.

**Components of inflammation:**

There are two major components of inflammation:-

1. Blood cells
2. Blood vessels

**Outcomes of inflammation:**

* To eliminate the causative agent of infection or tissue destruction.
* To eliminate the consequences of necrosis.

**Signs of inflammation**

There are five signs of inflammation:-

1. Caller(heat)
2. Rubor(redness)
3. Dollar(pain)
4. Tumor(swelling)
5. Functio laessa(loss of function)

**Causative agents of inflammation:**

Following are the causative agents of inflammation:-

1. Infection
2. Tissue injury
3. Foreign bodies
4. Immune system

**Infection:**

In case of infection, either bacterial or viral, tissue residual cells contain cytoplasmic receptors called tall-like receptors.

When these infectious agents are detected by tall-like receptors. They initiate signaling pathway that release chemical mediators to release inflammation.

**Tissue destruction:** Whatever the cause of tissue injury, hypoxia or ischemia, the necrotic agent released by that tissue or detected by another cytoplasmic receptors called ‘’inflammasomes’’. These release capases-1 that convert Pro-IL-1 into IL-1 which is active form and causes inflammation.

When tissue destruction is due to hypoxia, hypoxia induced factors 1-α that causes transcription of genes involved in inflammation.

**Foreign bodies:** Sutures or ligatures can also cause inflammation by acting as foreign bodies.

**Immune system:** Immune response e.g hypersensitivity or auto-immune responses may cause inflammation by releasing inflammatory mediators.

**Types of inflammation:**

There are two types of inflammation:

1. Acute inflammation
2. Chronic inflammation

|  |  |
| --- | --- |
| **Acute inflammation** | **Chronic inflammation** |
| * It has rapid onset nearly from minutes to few hours.
 | * In chronic inflammation, onset is very slow nearly i-e for few days.
 |
| * It has short duration. It occurs for short duration.
 | * It has long duration of action from days to weeks or months.
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| * Neutrophils are involved.
 | * Lymphocytes and macrophages are involved.
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| * Tissue injury in the case of acute inflammation, is minute and subsides itself.
 | * Tissue injury is severe and progressive.
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| * Signs of inflammation are prominent.
 | * Signs are less prominent in chronic inflammation.
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**Inflammatory process:**

There are two types of changings occurs:-

1. Vascular changes
2. Cellular changes

**Vascular changes:**

In vascular changes, there is vasodilation and alteration in vascular permeability.

**Vasodilation:** Vasodilation in inflammation is induced by chemical mediators e.g histamine and nitric oxide.

This vasodilation is followed by arteriolar vasodilation.

**Vascular permeability:** This leads to opening of capillary bed in that area that results in increased blood flow in that area. Due to increased blood flow there is development of heat and redness. Vasodilation will later leads to the alteration in vascular permeability. Due to alteration in vascular permeability there is loss of fluid and plasma proteins from blood vessels and cause slow down of blood flow called stasis.

Vasodilation and alteration in vascular permeability is the hallmark of inflammation. Loss of proteins-riched fluid from the microvasculature leads to edema formation. This protein riched fluid called exudate that has specific gravity > 1.020. The process of exudate formation is called exudation. If protein contents are less then it will be called transudate having specific gravity < 1.020. Transudate formation occurs when no gap occurs between interendothelial cells.

Vasodilation will be followed by increased vascular permeability. Vascular permeability is changed by:-

1. Endothelial cells contraction and retraction
2. Direct damage to the endothelium
3. Increased trancytosis

**Endothelial cells contraction and retraction:**

Contraction of endothelial cells result in increase in endothelial cells that cause leakage of plasma proteins. This contraction and retraction induced by chemical mediators like substance-p, leukotrienes, bradykinins, histamines. These mediators are released by tissue resident molecules. This type of response is called immediate transient response that usually occurs within 30 minutes after exposure. If there is immediate response, there may be some delayed response which usually occurs after some delay and occurs after 2-12 hours. It is delayed prolonged response. e.g sunburn.

**Direct damage to endothelium:**

Direct damage to endothelium may alter vascular permeability. E.g atherosclerosis.

Another example is leukocytes or white blood cells adhere to the endothelium and cause damage to the endothelium.

**Increased transcytosis:**

Endothelial cells contain organelles called vasiculovacular organelles responsible for transport process. Various growth factors release that increase size of these organelles and increase transcytosis that alter vascular permeability. Increase in diameter of blood vessels occurs due to release of histamine and nitric oxide that causes loss of blood and loss of proteins which results into the thickening of bllod and redness due to increased hydrostatic pressure and decreased colloidal pressure.