

PATHOPHYSIOLOGY – BP204T

UNIT : 1

CLASS: 5

TOPIC: Inflammation

Inflammation is defined as the local response of living tissues to injury due to any agent.

inflammation, a process by which the body's immune system malfunctions. (failure to function normally).

Eg. diseases, like [arthritis](#), diabetes, myasthenia gravis

ETIOLOGY

The causes of inflammation are many and varied:

- Exogenous causes:
 - Physical agents
 - Mechanic agents: fractures, foreign corps, sand, etc.
 - Thermal agents: burns, freezing
 - Chemical agents: toxic gases, acids, bases
 - Biological agents: bacteria, viruses, parasites
- Endogenous causes:
 - Circulation disorders: thrombosis, infarction, hemorrhage
 - Enzymes activation – e.g. acute pancreatitis
 - Metabolic products – uric acid, urea

SIGNS OF INFLAMMATION

CELSUS in 1st century A.D named the famous 4 *cardinal signs* of inflammation as:

- *rubor* (redness)
- *tumor* (swelling)
- *calor* (heat)
- *dolor* (pain)

To these, 5th sign *functio laesa*, or loss of function was later added by VIRCHOW

TYPES OF INFLAMMATION

ACUTE

Rapid onset Short duration

Fluid accumulation, plasma protein exudation Neutrophils

CHRONIC

Onset- insidious Longer duration

Lymphocytes, macrophages, plasma cells as inflammatory cells

Pathogenesis: Three main processes occur at the site of inflammation, due to the release of chemical mediators

- Increased blood flow (redness and warmth).
- Increased vascular permeability (swelling, pain & loss of function).
- Leukocytic Infiltration.
- Mechanism of Inflammation
- Vaso dilatation
- Exudation - Edema

- Emigration of cells
- Chemotaxis

ACUTE INFLAMMATION

VASCULAR EVENTS CELLULAR EVENTS

1. HAEMODYNAMIC CHANGES
2. ALTERED VASCULAR PERMEABILITY
 1. EXUDATION OF LEUKOCYTES
 2. PHAGOCYTOSIS

VASCULAR EVENTS

1. HAEMODYNAMIC CHANGES:
 1. TRANSIENT VASOCONSTRICTION
 2. VASODILATATION (arterioles, venules and capillaries)

obvious within half an hour of injury

Increase blood volume in microvascular bed

Elevation of HYDROSTATIC PRESSURE

Results in transudation of fluid in the extracellular space

Swelling

. Slowing or stasis

Increased vascular permeability

Increased concentration of RBCs Raised blood viscosity

Slower blood flow

slowing followed by

LEUCOYTE MIGRATION (neutrophils mainly) to the vascular endothelium

Leukocytes then move and migrate through gaps between the endothelial cells in the extravascular space.

EMIGRATION

CHRONIC INFLAMMATION

It is a prolonged process in which tissue destruction and inflammation occurs at the same time.

Time course:

- > 48 hours (weeks, months, years)
- Cell type
 - Mononuclear cells (Macrophages, Lymphocytes, Plasma cells)

Can be caused by 1 of the following 3 ways:

1. Chronic inflammation following acute inflammation
2. Recurrent attacks of acute inflammation
3. Chronic inflammation starting *de novo*

RCP