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Popular Article

Inflammation-an introduction

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Abstract

Inflammation may be defined as protective and reactive response of the living tissue following to injury to eliminate the initial causative irritant. It includes complex progression of vascular and tissue changes developed in response to an irritant, in the living tissue which is not harsh to produce necrosis.
Key words- Inflammation, vascular, tissue, cardinal signs, microscopic, mediators etc.

Introduction

The inflammatory process is designed to inactivate and remove the injuries agent, exclude damaged tissue resulting from the injury, and help repair and healing. The inflammation carries the suffix “itis” which is quite specific, for example endocarditis and enteritis, exceptions as disease conditions such as pneumonia and pleurisy, are named pneumonitis and pleuritis respectively. Acute inflammation displays cardinal signs, chronic inflammation characterized by fibrosis. Chemical mediators of inflammation are biological molecules which originates from plasma, cell or damaged tissue which directs event of inflammation. Classification of inflammation is done on basis of the exudates present.

Definition

Inflammation is a local response (reaction) of living vascularized tissues to endogenous and exogenous stimuli. The term is derived from the Latin "inflammare" meaning to burn. Inflammation is fundamentally destined to localize

and eliminate the causative agent and to limit tissue injury. Thus, inflammation is a physiologic (protective) response to injury. Inflammation is itself not to be considered as a disease but as a salutary operation consequent either to some violence or to some diseases".

Causes: Causes of inflammation are apparently causes of diseases such as:

1. physical agents - mechanical injuries, alteration in temperatures and pressure, radiation injuries.
2. chemical agents- including the increasing lists of drugs and toxins.
3. biologic agents (infectious)- bacteria, viruses, fungi, parasites
4. immunologic disorders- hypersensitivity reactions, autoimmunity, immunodeficiency states etc
5. genetic/metabolic disorders- examples gout, diabetes mellitus etc

Cardinal signs of acute inflammation

Clinically, acute inflammation is characterized by 5 cardinal signs:

- Redness (Rubor)
- Hotness (Calor)
- Swelling (Tumor)
- Pain (Dolar)
- Loss of functions (Function laesa)

Redness and hotness results from the increased flow of blood carrying warmth from the higher interior temperature of the body to the periphery. Swelling results due to accumulation of fluid. Pain is due to release of chemicals that stimulate nerve ending. The loss of function is due to a combination of factors.

Microscopic picture of inflammation

Vascular and exudative changes are included in it. The inflammatory process, especially acute inflammation, is primarily circulatory phenomenon involving the blood vessels and flow. Cellular events are also countable.

Vascular changes and fluid exchange in inflammation

In the vascular phase, small blood vessels in periphery to the injury site dilate (vasodilatation) and blood flow to the area increases. Initially the endothelial cells swell, then show contraction to increase the space between them, thereby increasing the permeability of the vascular barrier.

A-Changes in blood flow in inflammation:

I) Changes in caliber and vascular flow:

a) Immediately after injury, smooth muscle of the arterioles contracts that results into transient constriction of the arterioles which is mediated by damaging stimulus. It disappears within 3 to 5 seconds.

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b) Long-standing vasodilatation which first involves the arterioles and then venules and results in opening of new microvascular beds in inflamed area, with increased blood flow and cause the tissue to become hyperemic, red and warm. Chemical mediators, mainly prostaglandin, are responsible for dilatation of arterioles. It lasts throughout the stage of acute inflammation to bring extra blood to the inflammatory area.

C) Slowing and stasis of blood flow due to increase in permeability of the microvasculature results in outpouring of protein rich fluid (exudate) into tissue leading to increment of viscosity.

ii) Changes in vascular permeability

The permeability of capillaries and venules is the function of inter cellular junction between the endothelium. Small molecules normally pass-through pores but pinocytosis permits passage of large one. Increased vascular permeability is one of the clearest changes that occur during inflammation leading to swelling or edema.

Cellular events

The cellular response has the following stages:

A. Migration, rolling, pavement & adhesion of leukocytes
B. Transmigration of leukocytes
C. Chemotaxis
D. Phagocytosis

Normally blood cells particularly erythrocytes in venules are confined to the central (axial) zone and plasma assumes the peripheral zone. As a result of increased vascular permeability, more and more neutrophils accumulate along the endothelial

surfaces (peripheral zone).

A) Migration, rolling, pavement and adhesion of leukocytes

Margination is a peripheral positioning of white cells along the endothelial cells. Following this event, the endothelium virtually lined by white cells. This appearance is named as pavement.

B) Transmigration of leukocytes

Leukocytes escape from venules and small veins but occasionally these phenomena take place in capillaries. The movement of leukocytes by extending of pseudopodia from the vascular wall occurs by a process called diapedesis.

C). Chemotaxis

Chemotaxis is a unidirectional movement of leukocytes from vascular channels towards the site of inflammation within the tissue space guided by chemical gradients (including bacteria and cellular debris).

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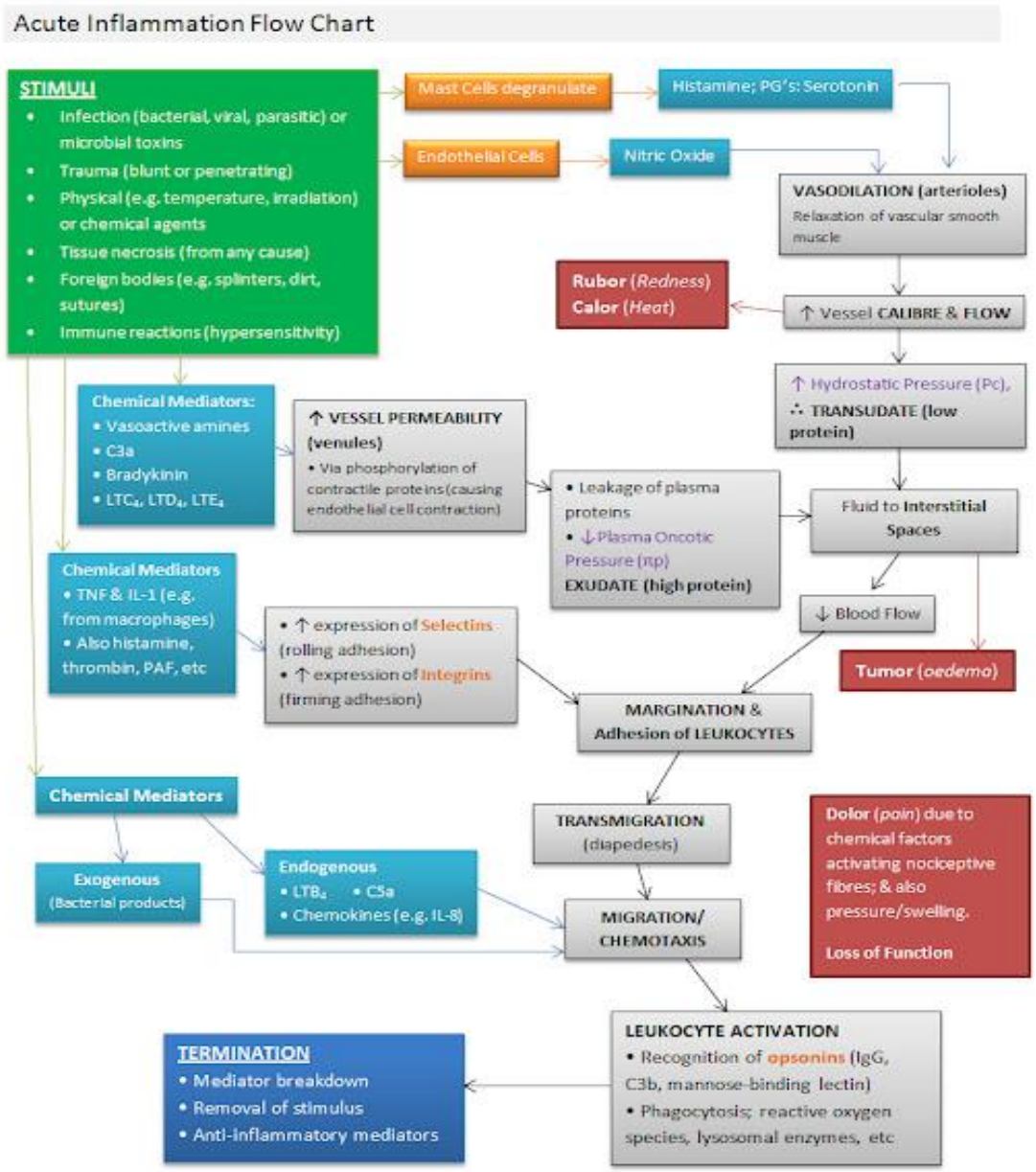
Chemical mediators

Mediators are substances that direct the vascular and cellular event in inflammation. They can originate from plasma, cells or damaged tissue.



Chemical mediators of inflammation

Molecules which are capable of inducing edema upon injection, stimulating smooth muscle contraction, or altering blood pressure following administration. These substances can act via direct stimulation of cell surface receptors (e.g., histamine) or function as plasma derived factors that cause direct tissue damage (e.g., complement). Alternatively, they may function as chemotactic agents to

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attract specific cell phenotypes to areas of inflammatory response (e.g., chemokines; arachidonic acid metabolites.

Type of inflammation

This is Morphological classification based on exudates in acute inflammatory lesions

1. Serous inflammation- Serous inflammation is identify by tissue response in which accumulation of fluid (watery) with low no of plasma protein rather than leucocytes. This watery fluid is released by gap between endothelial cells and inflamed serous gland. Serous inflammation mainly seen in thermal injury (burn and hypersensitivity) and allergic condition (watery eyes and runny nose). Grossly lesion is affected tissue contain yellowish watery fluid on cut section. Fluid filled vesicles on surface of mucous membranes of nose or eyes. Microscopically, connective tissue fibre is separated, capillaries are dilating with erythrocytes.

2.Catarrhal inflammation or mucoid inflammation- It is identified by accumulation of thick gelatinous fluid (mucous) in affected tissue. This type of inflammation mainly seen in where abundant goblet cells are found. Catarrhal inflammation mainly seen in chronic conditions i.e., asthma, allergy and gastrointestinal diseases. Grossly, mucin is found on cut surface. Microscopically, hyperplastic epithelial cells of mucous gland and goblet gland.

3.Haemorrhagic inflammation- This type of inflammation is identified by accumulation of erythrocyte. Cause of this inflammation are violent injury in blood vessels, bacteria, virus and some parasitic infection. Grossly, the affected tissue covered with blood.

4.Fibrinous inflammation- In this inflammation accumulation of more no of plasma protein specially fibrinogen. Fibrinous inflammation mainly occurs due to severe endothelial injury which cause release of fibrinogens from capillaries. After leaking of fibrinogen polymerise with fibrin and form pink protein stained with H&E stain. Fibrinous inflammation mainly seen in Fibrinous pleuritis, Fibrinous pericarditis and IBR viral infection. A typical example of Fibrinous inflammation is Fibrinous pneumonia in which fibrin deposit in alveoli. Grossly, the affected tissue is thick and covered with fibrin. Microscopically, Capillaries and post capillaries are dilated with erythrocytes. When fibrin exudates contain neutrophils then term use a Fibrinous suppurative inflammation.

5.Suppurative inflammation- This is identified by accumulation of large quantity of plasma protein and large number of neutrophils. The commonly exudates in this inflammation are pus. If pus collects in a circumscribed cavity, tern us abscess. If pus can be distributed in tissue layer, then term use

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cellulitis. Suppurative inflammation is mostly caused by bacteria. Grossly, affected organ is hypermic and covered with thick yellow pus. Microscopically, large number of neutrophils are present. Saprophytic Bacterial may be found.

6. Lymphocytic inflammation- It is identified by accumulation of lymphocytes. Cardinal sign are absent in this inflammation. Cause of this inflammation is viral infection and intoxication. Microscopically, exudate contain high number of lymphocytes. In central nervous system lymphocytes are found in the Virchow Robin spaces.

7.Allergic inflammation- This type of inflammation is identified by accumulation of eosinophils. Allergic inflammation generally associated with serous, Fibrinous and haemorrhagic inflammation. Cause of this inflammation are hypersensitivity, metazoan parasites. Microscopically, eosinophils are found with other inflammatory cells.

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