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Perspective

Introduction to Inflammation of Human Body Status

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DESCRIPTION

The word inflammation comes from the Latin word "inflammare" which increases to burn. This is a complex, integrated host response found only in vertebrates. The inflammatory response has two facets: inflammation and repair. Inflammation serves to destroy, dilute, or wall off the injurious agent and the tissue cells that may have been destroyed. In turn, the inflammatory response sets into motion a complex series of events, which heal and reconstitute the damaged tissue. Repair begins during the active phase of inflammation, but reaches completion usually after the injurious influence has been neutralized. Destroyed cells and tissues are repaired thereby. Both inflammation and repair generally serve useful purpose. Without inflammation, bacterial infections would remain unencountered, wounds would never heal and injured tissues and organs might be permanently defected. But inflammation may be potentially harmful. Inflammatory reactions underlie the genesis of crippling rheumatoid arthritis, life threatening sensitivity reaction and some forms of fatal glomerular diseases.

Inflammation was known as 'phlogosis' to the Greeks and as inflammation to the Romans. The four main signs of inflammation are rubor, tumor, calor and dolor. Galen in third century A.D. defined the inflammation as a reaction of the body against injury. The views developed by the above persons, with the four signs of Celsius in the background constituted 'master leads' to understand the whole pattern developed by the body to fight against aggression. In late Nineteen Century, the science of immunology had been developed with the pioneering contributions from Jenner, Pasteur, Landsteiner, etc. Thereafter, conclusive evidences have accumulated that body fluids and blood serum have a protective effect against variety of invasive microorganisms.

Pathophysiology of inflammation

Physical agents, chemical agents, infections and immunological reaction may bring about the injury which causes inflammation.

Essentially there are two categories of inflammation, acute and chronic. The classical signs of acute inflammatory reaction are warmth, redness, pain, swelling and loss of function.

Zweifach reviewed the vascular changes and phenomena which occur in acute inflammation after injury. Immediately after the injury, the arterioles of the injured tissue contracts followed by relaxation. As a result of that, the capillary network and post capillary venules become engorged with rapidly flowing blood, which warms the normal skin and also causes redness.

In the area of engorged microcirculation, at first the blood flows rapidly, later it becomes progressively slower and there might be a temporary static of blood. After the onset of active hyperaemia the protein-rich fluid escapes from the blood vessels into the surrounding tissue and forms interstitial oedema. The inflammatory exudates are much richer in plasma proteins than normal extracellular fluids indicating the increased permeability of the vessels to macromolecules.

CONCLUSION

The increased leakage of fluid and electrolytes in acute inflammation is explained by the increased hydrostatic pressure of blood in small vessels in active hyperaemia. Exudation of plasma proteins from small vessels in acutely inflamed tissue requires an increase in permeability of endothelium and this is provided by the reversible opening up of relatively large gaps between endothelial cells. In inflammation, when slowing of flow ensues, the axial streaming disappears, the leukocytes in the venules passes into the peripheral stream, make contact with vascular endothelium and thereby get arrested on it, eventually forming a continuous layer.

The adhesion may be due to neutralization of negative charges or reduction of charge density of the leukocytes. Divalent ions also play an important role and the chemostatic factors increase the adherence of neutrophils to endothelial cells.

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