Volume 3, Issue 11, November 2025 ISSN (E): 2938-3765

CURRENT UNDERSTANDING OF THE MOLECULAR MECHANISMS OF THE INFLAMMATORY PROCESS

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Abstract
Inflammation is a complex local vascular-mesenchymal response to tissue damage caused by various types of agents. Particularly extensive inflammatory processes develop after extensive thermal burns [1,2].

Keywords: Inflammation, alteration, exudation, proliferation, cells, mediators.

Introduction

Inflammation develops in the histion area and consists of closely related and sequentially developing phases:

1) alteration; 2) exudation; 3) proliferation of haematogenous and histiogenous cells and, less commonly, organ parenchyma (epithelium) [3,5,6].

Alteration - tissue damage determines the initial phase of inflammation, during which biologically active substances (mediators) are formed and released, triggering the inflammatory response. As the number of cells increases and plasma proteins accumulate in the site of inflammation, mediators continue to be released in the subsequent phases of inflammation. The mediators originate from certain elements of blood plasma—kinins, kallikreins, factor XII of the blood coagulation system (Hageman factor), complement components, etc., as well as effector cells: labrocytes, basophils and thrombocytes, which release histamine, serotonin and other mediators; neutrophils, which release leukokines; macrophages that release monokines; lymphocytes that release lymphokines; other cells (mast cells release histamine, endothelial cells release arachidonic acid metabolites) [9,10,11,12].

Under the influence of mediators, the exudation phase begins, which consists of several stages: a) under the influence of histamine, a reflex spasm of the lumen of arterioles and precapillaries occurs, which is quickly replaced by the expansion of the entire vascular network of the inflammation zone, primarily postcapillaries and venules, followed by a slowdown in blood flow, the formation of areas of stasis, and the development of microthrombosis; b) under the influence of a wide range of other mediators, there is an increase in the vascular permeability of the microcirculatory bed, which leads to the formation of exudate and inflammatory cellular infiltrate [2,7].

As a result of the action of mediators, the final phase of inflammation occurs – proliferation [1,5,8].

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Inflammation is regulated by hormonal, nervous and immune factors. Somatotropic hormone of the pituitary gland, deoxycorticosterone and aldosterone enhance the inflammatory response (pro-inflammatory hormones), while glucocorticoids and ACTH reduce it. Cholinergic substances, by stimulating the release of inflammatory mediators, act similarly to pro-inflammatory hormones, while adrenergic substances, by suppressing mediator activity, behave like anti-inflammatory hormones [1,2,3].

Thus, mediators cause the development of local oedema. In addition, thermal tissue damage activates a large number of mediator cascades, such as complement activation, the release and cascade of arachidonic acid transformations, and cytokine production, among which interleukin-1 (IL-1) and tumour necrosis factor (TNFα), which promote the accumulation of neutrophils and macrophages in the affected area [4,5,6].

Many cells are involved in inflammation, and their interaction is mediated by four groups of factors: adhesive molecules, extracellular matrix, soluble mediators, and oncogenes. Interactions between cells, as well as between cells and intercellular substance, are facilitated by several families of adhesive molecules: integrins, the immunoglobulin superfamily, cadherins, selectins, homing receptors, etc. All these substances facilitate intercellular interactions under certain conditions [1,2,6].

Thus, integrins function as both cell-substrate and intercellular adhesive receptors. Integrins are integral membrane receptors that connect one cell to another or to the extracellular matrix via the cytoskeleton. The integrin receptor family determines many of the adhesive properties of cells. They act as a link between the extracellular matrix and the cytoskeleton by transmitting information generated during the interaction of the extracellular domain with extracellular matrix ligands into the cell, influencing the organisation of the cytoskeleton, cell shape and cell motility. Each integrin can transmit different information from the extracellular microenvironment, determining the morphology and physiology of the cell. Depending on the connections between the beta1, beta2, and beta3 subunits with various alpha subunits, a distinction is made between VLA proteins (slow-reacting antigen), leukocyte integrins, and cytoadhesins [7,8,9,10].

Endothelial cells play a leading role in the development of inflammation, as it is they that, after stimulation by cytokines and bacterial products, acquire the ability to ‘direct’ leukocytes to the site of damage. Mononuclear phagocytes play a central role in the repair process, synthesising mediators that cause fibroblast proliferation. One of the main proteins in blood serum, alpha2-MG, interacts with a large number of cytokines. In the first stage, the native molecule is converted into a ‘fast’ form under the influence of endogenous proteins. Then, the ‘fast’ form binds to the cytokine, and the resulting complex interacts with cytokine receptors on the surface of target cells or with receptors for the ‘fast’ form of alpha2-MG on the surface of macrophages, hepatocytes, or fibroblasts. Alpha2-MA molecules serve to rapidly eliminate excess cytokines that are not bound to cell receptors, performing a protective function together with natural antibodies [9,10]. They themselves can be removed from the blood as a result of binding to receptors on the surface of macrophages, fibroblasts, and hepatocytes [10,12,13].

In addition to cytokines, alpha2-MG molecules are capable of binding endopeptidases, mitogens, and various ions, including zinc and nickel cations. The interaction of the native alpha2-MG molecule with proteolytic enzymes causes important conformational changes in its structure,

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accompanied by changes in electrophoretic mobility: low in the native form and high in its complex with proteases. The resulting hydrophobic binding region with receptors on the surface of macrophages, fibroblasts, and hepatocytes promotes the removal of alpha2-MG molecules from the blood [16,17,18].

Simultaneously with the molecular transformations that cause inflammation, processes begin that are aimed at restoring damaged tissues and healing them, which are associated with the activation and regulation of growth factors [12,13,14,15].

The first visible stage of acute inflammation is vasodilation at the site of inflammation. There, blood flow slows down, stasis occurs, and leukocytes leave the bloodstream at the site of organ or tissue damage. Vasodilators cause vasodilation. However, it has been found that the injection of such vasodilators does not cause significant inflammation. Only the combination of chemoattractants and vasodilators significantly increased the permeability of the vessel walls and the accumulation of leukocytes [24].

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