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PATHOANATOMICAL AND MORPHOLOGICAL FOUNDATIONS OF INFLAMMATORY PROCESSES

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Abstract.

Inflammation is a complex, multi-stage, and protective-adaptive response of the organism to various harmful factors. This process develops at the cellular, tissue, and organ levels and manifests itself through profound morphological changes associated with microcirculation, cellular migration, and proliferation[1,2]. The article extensively covers the pathoanatomical foundations of the ,inflammatory process, morphological features, cellular components, and forms of manifestation of acute and chronic inflammation in tissues on a scientific basis.

Keywords

Inflammation, pathoanatomical changes, morphology, exudation, alteration, proliferation, microcirculation, acute inflammation, chronic inflammation, cellular infiltration.

INTRODUCTION.

Inflammatory processes are considered a universal pathological process occurring in almost all areas of medicine. Inflammation plays a crucial role in the pathogenesis of infectious diseases, autoimmune conditions, traumas, ischemic injuries, and neoplasms[3,4,5]. An in-depth study of the mechanisms of inflammation from pathoanatomical and morphological perspectives allows for a correct understanding of the origin, course, and complications of diseases. Therefore, analyzing the morphological foundations of inflammation is of paramount importance in improving diagnostic and treatment strategies.

Inflammation is a biologically important adaptive reaction of the organism that develops in response to tissue damage and aims to eliminate the damaging factor and restore tissue integrity[6,7,8]. From a pathoanatomical perspective, the inflammatory process is characterized by a simultaneous combination of alteration, exudation, and proliferation phenomena. These processes do not occur in isolation but progress continuously and interconnectedly.

Inflammation is one of the oldest and most universal biological adaptive reactions of the organism, which forms in response to the disruption of tissue integrity under the influence of various physical, chemical, biological, and immunogenic factors. This process has evolved as a protective and adaptive mechanism, whose main task is to identify, contain, neutralize the damaging factor, and subsequently restore the structural and functional integrity of damaged tissues. Therefore, inflammation is regarded not only as a pathological condition but also as an important biological phenomenon that contributes to the survival of the organism.

From a pathoanatomical perspective, inflammation is a complex, multi-component, and dynamic process characterized by the simultaneous and interrelated occurrence of alteration, exudation[9,10], and proliferation phenomena. These three main components are not separate stages of inflammation, but rather a set of morphological processes that complement each other and develop continuously. The predominance of one of these components varies depending on the nature, intensity, and duration of the damaging factor, as well as the reactivity of the organism.

Alteration is considered the initial and central link in the inflammatory process. It proceeds with direct damage to cells and tissues, leading to the emergence of structural and metabolic disturbances. Morphologically, alteration manifests as increased permeability of cell membranes, disruption of ion exchange, and swelling of cytoplasmic organelles, especially mitochondria and the endoplasmic reticulum. As a result of energy metabolism disruption, ATP synthesis decreases, calcium ions accumulate inside the cell, and this condition leads to the activation of proteolytic enzymes[11,12]. Consequently, autolysis and destruction of intracellular structures occur.

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Under conditions of severe and prolonged alteration, cells undergo irreversible changes and die through necrosis or apoptosis. Pathoanatomically, signs of active inflammatory infiltration, vascular reactions, and exudation are detected around the foci of necrosis[13,14]. Necrotic tissue, in turn, serves as a secondary inflammatory stimulant and leads to the deepening of the process.

Exudation is the most pronounced and clinically noticeable component of inflammation, associated with complex vascular reactions occurring at the microcirculatory bed level[15,16]. In pathoanatomical examinations, a short-term spasm of arterioles and capillaries is initially observed, followed by their persistent dilation. As a result of increased permeability of the vessel walls, blood plasma proteins, including fibrinogen, immunoglobulins, and complement components, leak out of the vessels.

The specific composition of the exudate determines the type of inflammation. In serous exudate, fluid predominates, while in fibrinous inflammation, fibrin fibers settle on the surface or within the tissues. In purulent inflammation, neutrophil leukocytes predominate as the main cellular component. Enzymes and debris formed as a result of their breakdown further intensify tissue destruction. In hemorrhagic exudation, due to severe damage to the vessel walls, erythrocytes are also added to the exudate composition[17,18,19].

The process of exudation is closely linked to cellular migration. The adhesion of leukocytes to the vascular wall, diapedesis, and directed movement towards the damaged tissue are controlled by complex molecular mechanisms. From a pathoanatomical perspective, this process manifests as the formation of a cellular infiltrate at the site of inflammation. In acute inflammation, the infiltrate consists mainly of neutrophils, while in chronic inflammation, lymphocytes, plasma cells, and macrophages predominate.

Proliferation represents the restoration and reconstruction phase of the inflammatory process. During this process, fibroblasts are activated, collagen fiber synthesis increases, and new capillaries form. Morphologically, granulation tissue develops, which serves as a temporary structure filling the damaged area. If regenerative capabilities are sufficient, the tissue fully recovers; otherwise, fibrosis and scarring develop.

Thus, from a pathoanatomical standpoint, inflammation manifests as an integral unity of alteration, exudation, and proliferation, with each process being incomplete without the others. Their balance determines the course of inflammation, its outcome, and the degree to which organ function is preserved. It is precisely this complex morphological harmony that allows us to evaluate inflammation as a biologically important adaptive process rather than a simple pathological phenomenon.

Alteration is the initial stage of inflammation, representing the primary damage to cells and tissues. Morphologically, alteration manifests as disruption of cell membranes, swelling of cytoplasmic organelles, dysfunction of mitochondrial activity, and changes in nuclear structure. In severe cases, necrosis develops, leading to complete disintegration of the tissue structure. Pathoanatomical examination reveals active inflammatory infiltration around necrotic foci.

Exudation is one of the most pronounced morphological signs of inflammation, characterized by the release of plasma fluid and formed blood elements into the tissue as a result of increased permeability of blood vessel walls. Microscopic examination reveals dilation of capillaries and venules, stasis, diapedesis of erythrocytes, and migration of leukocytes through the vascular wall. Depending on the composition of the exudate, inflammation is classified into serous, fibrinous, purulent, hemorrhagic, and mixed forms. Each form is characterized by specific pathoanatomical features.

In acute inflammation, morphologically, a predominance of neutrophilic granulocytes is observed. They attempt to eliminate the damaging factor through phagocytosis and enzymatic breakdown. During this process, proteolytic enzymes are released, which can also cause secondary damage to surrounding tissues. Pathoanatomically, this condition is characterized by purulent exudate and tissue destruction.

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In chronic inflammation, the cellular composition changes fundamentally. Lymphocytes, plasma cells, macrophages, and fibroblasts predominate. This process is prolonged and leads to proliferative changes in tissues, as well as the development of fibrosis and sclerosis. Morphologically, an increase in collagen fibers, atrophy of parenchymal cells, and a decrease in organ function are observed.

Granulomatous inflammation is a special form of chronic inflammation characterized by the formation of granulomas involving epithelioid cells and giant cells. This type of inflammation is typical of tuberculosis, sarcoidosis, and certain fungal infections. Pathoanatomically, necrosis is detected in the center of the granuloma, surrounded by lymphoid infiltration.

Disruption of the microcirculatory system in the inflammatory process is of particular importance. Changes in vascular tone, slowing of blood flow, and stasis lead to the development of hypoxia. This results in disruption of cellular metabolism and intensification of the inflammatory process. From a pathoanatomical perspective, this condition manifests as edema, hemorrhages, and tissue necrosis.

The final stage of the inflammatory process is associated with reparation and regeneration, which concludes with complete healing or scarring, depending on the degree of restoration of the damaged tissues. If regeneration is insufficient, fibrosis and persistent organ dysfunction develop.

Thus, the inflammatory process is pathoanatomically complex and multifaceted, manifesting as an organic unity of alteration, exudation, and proliferation. A deep understanding of the morphological basis of this process is crucial for correctly assessing the pathogenesis of diseases and developing effective approaches in clinical practice.

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