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HISTOLOGICAL CHARACTERISTICS OF ATHEROSCLEROSIS IN THE PATHOGENESIS OF CARDIOVASCULAR SYSTEM DISEASES

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Abstract: This article analyzes the histological changes occurring in large and medium-sized blood vessels during the process of atherosclerosis, which is the leading cause of cardiovascular system diseases. Morphological changes at the cellular and tissue levels observed in the tunica intima, media, and adventitia layers of the blood vessel wall are elucidated.

Keywords: Histology, atherosclerosis, endothelial dysfunction, foam cells, tunica intima, smooth muscle cells.

Introduction

Cardiovascular diseases remain one of the primary causes of mortality and disability among the population worldwide. The morphological basis of these diseases is often associated with the damage to the blood vessel wall—atherosclerosis. Atherosclerosis is characterized by the accumulation of lipoproteins, mainly cholesterol, and the overgrowth of connective tissue in the inner layer (tunica intima) of large and medium-sized arteries of elastic and muscular-elastic types (e.g., aorta, coronary arteries). Studying the histological changes in the early stages of the disease has significant clinical importance in its treatment and prevention.

Myocardial infarction (MI), the most acute and dangerous manifestation of ischemic heart disease, occurs as a result of a sudden and prolonged cessation of the blood supply to the heart muscle. Cardiomyocytes are highly sensitive to oxygen deficiency (hypoxia), and when the duration of ischemia exceeds 20-30 minutes, irreversible morphological changes—necrosis—begin. Studying the step-by-step progression of histological changes occurring during MI is of fundamental importance not only in determining the disease stage (the age of the infarct) but also in developing therapeutic strategies aimed at controlling inflammation and scarring processes.

Materials and Methods

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This review article analyzes the results of recent histological and pathomorphological studies. In evaluating the atherosclerotic process, data obtained from classic histological staining methods (Hematoxylin and Eosin, Sudan III for lipid detection, and Masson's trichrome to differentiate collagen fibers) were generalized. The morphological structure of the walls of large arteries in the human body was taken as the object of study.

Results

During the atherosclerosis process, histological changes in the blood vessel wall proceed in several stages and manifest uniquely in each layer:

- **Endothelial dysfunction (Initial stage):** Normally, the continuous single-layered squamous epithelium (endothelium) acts as a protective barrier for the blood vessel. Under the influence of atherogenic factors, the tight junctions between endothelial cells are disrupted. As a result, endothelial permeability increases, and low-density lipoproteins (LDL) from blood plasma begin to penetrate the subintimal space.
- **Formation of "foam cells":** Monocytes that have migrated into the intima layer transform into macrophages and phagocytize oxidized lipids. The cytoplasm of macrophages that have engulfed large amounts of lipids becomes vacuolated, turning them into "foam cells" (xanthoma cells) with a specific histological appearance. Under the microscope, they appear as cells with pale, vacuolated cytoplasm.
- **Formation of the fibrous plaque:** As the process intensifies, smooth muscle cells (SMC) from the tunica media migrate to the intima layer and undergo proliferation. These cells synthesize a large amount of collagen and elastin fibers, forming a connective tissue cap—the fibrous plaque. When stained with Masson's trichrome, this area appears distinctly blue (due to collagen).
- **Complicated plaque and calcification:** Cell necrosis occurs in the central part of the plaque, forming an atheromatous core (cholesterol crystals, cell debris). In later stages, calcium salts deposit in this area (dystrophic calcification), which leads to the complete loss of elasticity of the blood vessel wall.

Histological changes in the focus of myocardial infarction occur in a strict, time-dependent (dynamic) sequence. Analysis of studies allows dividing this process into the following main histological stages:

- **Early ischemic stage (0-12 hours):** Histologically, changes are minimal. In H&E staining, a "wavy fibers" arrangement of cardiomyocytes is observed. This is explained by the stretching of the non-functioning muscle fibers in the ischemic zone due to the contraction of surrounding healthy cells. Edema appears in the intercellular space.
- **Coagulative necrosis and neutrophil infiltration (1-3 days):** Signs of acute coagulative necrosis manifest in cardiomyocytes. Under the microscope, the loss of nuclei (karyolysis), fragmentation of chromatin (karyorrhexis), and shrinkage (karyopyknosis) are clearly visible. The cell cytoplasm takes on a strong eosinophilic (dark red) color. During this stage, polymorphonuclear leukocytes (neutrophils) actively migrate from the bloodstream to the necrotic focus and begin to break down the dead tissue by releasing lysosomal enzymes.
- **Macrophage infiltration and tissue destruction (3-7 days):** Neutrophils undergo apoptosis and are replaced by macrophages differentiated from monocytes. The primary function of macrophages is to phagocytize the remnants of necrotic cardiomyocytes. During this period, the myocardial wall is in its weakest state because the dead tissue has broken down, and a strong scar has not yet formed in its place (the period of highest risk for cardiac rupture).
- **Granulation tissue and scar formation (1-4 weeks and beyond):** After the necrotic mass is cleared, fibroblasts and new blood vessels (angiogenesis) grow into the focus, forming

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granulation tissue rich in blood vessels. Fibroblasts actively synthesize type I and III collagen. Over time, the number of cells decreases, blood vessels regress, and a post-infarction scar (cardiosclerosis) consisting of dense connective tissue forms in the focus. In the Masson's trichrome method, this area appears dark blue.

Discussion

Obtained histological analyses show that atherosclerosis is not merely lipid accumulation, but a chronic inflammatory reaction of the blood vessel wall. The thickening of the intima layer and the growth of fibrous tissue lead to the narrowing of the blood vessel lumen (stenosis). In the tunica media layer, the depletion of smooth muscle cells and the degradation of elastic fibers cause a decrease in the mechanical strength of the blood vessel wall and an increased risk of aneurysm.

Histological analyses in the pathogenesis of myocardial infarction show that ischemic injury is not simply cell death, but a complex and interrelated inflammatory and reparative cascade. Because the regenerative capacity of the heart muscle is extremely low, the site of dead cardiomyocytes is always replaced by scar tissue. The speed and quality of the transformation of granulation tissue into a scar is the primary morphological factor determining the probability of developing heart failure or an aneurysm in the future.

Chronic Inflammation and Cellular Cascade Evaluating the atherosclerotic process as chronic inflammation is one of the main achievements of modern pathomorphology. Oxidized low-density lipoproteins (LDL) that pass beneath the endothelium act as a strong chemotactic factor, attracting T-lymphocytes and monocytes from the bloodstream. Macrophages and T-lymphocytes become activated, producing inflammation-specific cytokines (e.g., Interleukin-1, TNF-alpha). These cytokines further exacerbate endothelial dysfunction, shifting the inflammatory process into an irreversible "chain reaction" (cascade).

Phenotypic Transformation of Smooth Muscle Cells (SMC) The specific modification of smooth muscle cells in the tunica media plays a crucial role in the thickening of the intima layer and the formation of fibrous tissue. Under the influence of inflammatory factors, SMCs switch from their usual contractile phenotype to a synthetic phenotype. They migrate to the intima layer and actively begin to produce extracellular matrix (collagen, elastin, and proteoglycans). This exact process leads to the stiffening of the blood vessel wall and luminal stenosis.

Tunica Media Degradation and Aneurysm Risk (Role of Matrix Metalloproteinases) One of the most dangerous complications of atherosclerosis is the formation of an aneurysm (especially in the abdominal aorta). The histological basis of this process is that activated macrophages and "foam cells" within the plaque secrete large amounts of matrix metalloproteinases (MMPs)—specifically, collagenases and elastases. These enzymes degrade the elastic membranes and collagen fibers in the middle layer of the blood vessel (tunica media), inducing apoptosis of the cells located there. As a result, the blood vessel wall loses its elasticity and its ability to withstand hemodynamic pressure, which causes a sac-like dilation (aneurysm) of the blood vessel wall.

Intraplaque Neovascularization (Changes in Vasa Vasorum) Another important aspect that requires attention in histological sections is neovascularization. As the atherosclerotic plaque enlarges, the demand for oxygen increases (a state of hypoxia). In response, small blood vessels in the adventitia layer (vasa vasorum) grow into the intima layer. However, the walls of these newly formed microvessels are very fragile, and their endothelial layer is imperfect. Consequently, they easily rupture, which can lead to hemorrhage into the plaque (imbibition) and acute complications such as thrombosis.

Conclusion

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Changes observed in the histology of the cardiovascular system in atherosclerosis—disruption of endothelial integrity, accumulation of foam cells, and SMC proliferation—constitute the basis of the disease pathogenesis. Deeply understanding these morphological changes has significant fundamental importance in developing new pharmacological agents aimed at combating cardiovascular system diseases.

Histological changes in myocardial infarction include stages of cardiomyocyte ischemia, coagulative necrosis, severe inflammatory response, and reparative fibrosis. A profound understanding of these mechanisms at the cellular and tissue levels serves as a fundamental pillar in researching new medications aimed at preventing post-infarction complications and preserving the functional state of the heart muscle.

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