SPECIFIC FEATURES OF MORPHOLOGICAL CHANGES IN THE MAMMARY GLAND UNDER THE INFLUENCE OF VARIOUS RISK FACTORS IN EXPERIMENTAL ATHEROSCLEROSIS

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Abstract

This article analyzes the mechanisms of development of atherosclerosis under experimental conditions, the dynamics of morphological changes in the mammary glands, and the role of various risk factors such as dyslipidemia, hormonal imbalance, hypodynamia, and stress. Experimental studies revealed that atherosclerotic processes significantly affect the microcirculatory system, alveolar structure, and secretory function of the mammary gland. The findings deepen the understanding of the pathogenesis of atherosclerosis and its influence on hormone-dependent organs, contributing to the development of preventive and therapeutic approaches.

Keywords: atherosclerosis, mammary gland, morphological changes, experimental pathology, risk factors, lipid metabolism, vascular endothelium, hormonal imbalance, dystrophic processes, sclerosis.

Introduction

Atherosclerosis is one of the most widespread chronic diseases that pose a serious threat to human health. It is characterized by the accumulation of cholesterol and other lipids in the arterial walls, leading to their thickening, loss of elasticity, and impaired blood flow. Although atherosclerosis primarily affects the cardiovascular system, its impact on other organs—particularly hormone-dependent ones such as the mammary glands—remains insufficiently studied.

The mammary gland is a hormonally sensitive organ with a rich blood supply, making it highly responsive to changes in metabolic and circulatory homeostasis. Even minor disturbances in lipid metabolism or vascular tone can lead to morphological and functional alterations. Therefore, studying the structural changes in the mammary gland during the progression of atherosclerosis is important for understanding systemic manifestations of this disease.

Materials and Methods

The study was conducted on six-month-old white laboratory rats divided into three groups:

1. **Control group:** animals maintained on a standard diet.
2. **Atherosclerosis group:** animals fed a high-fat diet containing 1% cholesterol.

3. **Atherosclerosis + risk factors group:** animals exposed to a high-fat diet combined with hypodynamia (restricted movement), stress (noise and light exposure), and induced hormonal imbalance (decreased thyroxine secretion).

The experiment lasted 90 days. Blood samples were taken every 30 days to analyze lipid profiles (cholesterol, triglycerides, HDL, LDL). At the end of the experiment, the animals were euthanized, and mammary gland samples were collected for histological examination. Parafin sections were stained with hematoxylin-eosin, van Gieson, and Sudan III for structural and lipid visualization.

Results and Discussion

General Morphological Characteristics

In the control group, the mammary glands exhibited a normal histological structure: single-layered cuboidal alveolar epithelium with centrally located nuclei, intact connective tissue stroma, and clear vascular profiles.

In the atherosclerosis group, the alveoli showed irregular shapes, degenerative changes in epithelial cells, and thickening of the interstitial connective tissue with increased collagen fiber accumulation. Capillaries were constricted, and stromal sclerosis was evident.

The most severe changes were found in the group with additional risk factors (atherosclerosis + hypodynamia + stress + hormonal imbalance). Histological analysis revealed:

- Alveolar destruction and vacuolization;
- Pycnotic nuclei and cytoplasmic fragmentation in epithelial cells;
- Proliferation of fibrous connective tissue in the stroma;
- Endothelial cell swelling and thickened vascular walls;
- Accumulation of lipid-laden macrophages (foam cells).

Vascular Changes

Microscopic examination showed that vascular endothelium in atherosclerotic animals was uneven, sometimes necrotic, and contained subendothelial lipid deposits. The vascular lumen was narrowed, causing impaired microcirculation and tissue hypoxia, leading to reduced secretory activity.

In hypodynamic animals, perivascular edema, interstitial fluid accumulation, microthrombosis, and hemorrhagic foci were observed. These alterations further aggravated the trophic deficiency in mammary tissues.

Cellular-Level Alterations

At the cellular level, alveolar epithelial cells demonstrated vacuolization, fragmentation of mitochondria and the endoplasmic reticulum, and chromatin condensation, indicating apoptosis initiation. Electron microscopy confirmed increased membrane permeability, excessive lipid and calcium ion influx, and structural disorganization of organelles.

Pathogenesis

Atherosclerosis is not only a vascular pathology but a systemic metabolic disorder. In hormonally active organs such as the mammary glands, dyslipidemia plays a central role in triggering structural damage.

The accumulation of oxidized low-density lipoproteins (LDL) beneath the vascular endothelium provokes an inflammatory response. Activated macrophages transform into foam cells, initiating fibrotic plaque formation. Simultaneously, reduced estrogen and prolactin levels weaken trophic maintenance and delay regenerative processes in glandular tissue.

Hypodynamia slows blood flow and oxygen delivery, while chronic stress elevates catecholamine and corticosteroid secretion, further disrupting lipid metabolism. The combination of these factors intensifies morphological destruction in mammary gland tissue under atherosclerotic conditions.

Prevention and Recommendations

Preventing atherosclerosis-induced changes in mammary glands requires a comprehensive approach, including:

1. **Dietary regulation:** limit cholesterol- and saturated fat–rich foods; increase consumption of fish, vegetables, and plant-based oils.
2. **Physical activity:** daily exercise enhances circulation and stabilizes lipid balance.
3. **Stress management:** maintaining psychological stability reduces catecholamine overproduction.
4. **Hormonal monitoring:** especially in menopause, to maintain estrogen balance.
5. **Regular check-ups:** histological and ultrasound monitoring of mammary gland condition.

Conclusion

Experimental studies demonstrate that atherosclerosis significantly affects the morphological integrity and functional activity of the mammary glands. Dyslipidemia, hypodynamia, stress, and hormonal imbalance accelerate degenerative and sclerotic processes in the glandular tissue, resulting in reduced secretory capacity and impaired regeneration.

Therefore, preventing atherosclerosis requires an integrated strategy combining a healthy lifestyle, balanced nutrition, hormonal regulation, and early detection of vascular and tissue changes.

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