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MORPHOLOGICAL STRUCTURAL FEATURES HEARTS IN MINERAL METABOLISM DISORDERS

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Abstract

Disturbances in mineral metabolism (calcium, phosphorus, magnesium, potassium, and sodium) play a key role in the formation of structural and functional changes in the myocardium. Deficiency or excess of certain macro- and microelements leads to disruption of cardiomyocyte ion homeostasis, imbalance in calcium channel activity, and alterations in depolarization and repolarization processes. This is accompanied by morphological changes in cardiac tissue, including cardiomyocyte dystrophy and necrosis, interstitial edema, stromal sclerosis, and myocardial remodeling. Prolonged mineral metabolism disorders contribute to myocardial hypertrophy, fibrosis, microcirculatory disturbances, and accelerated development of heart failure. Of particular importance are changes in calcium-phosphorus metabolism, which are closely associated with endocrine regulation (parathyroid hormones, vitamin D), as well as potassium-magnesium imbalance, which directly affects the contractile function of the heart. Studying the morphological mechanisms of cardiac structural damage in mineral imbalance provides prospects for pathogenetically based prevention and therapy of cardiovascular diseases.

Keywords: Mineral metabolism, calcium, phosphorus, potassium, magnesium, myocardium, cardiomyocytes, morphological changes, dystrophy, necrosis, hypertrophy, fibrosis, heart failure, myocardial remodeling, microcirculation, vitamin D, parathyroid hormone.

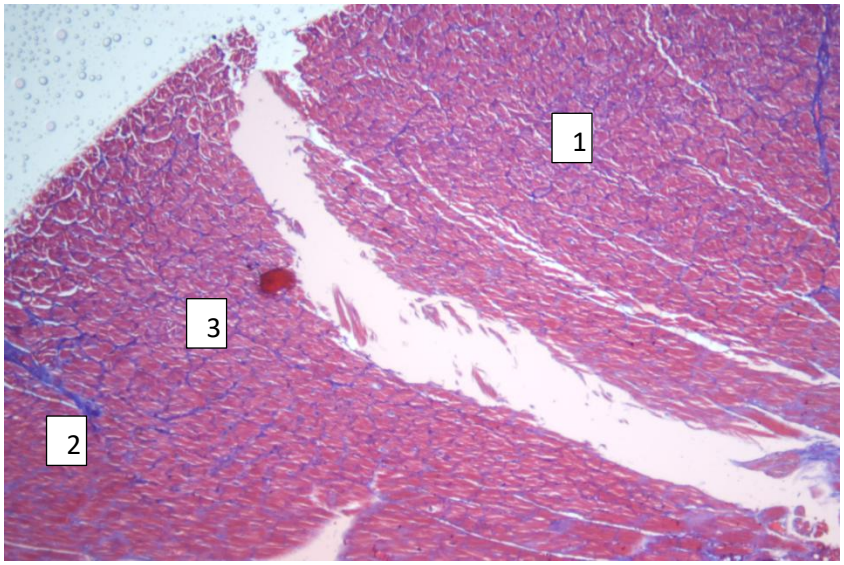
Introduction

Mineral metabolism is a complex, multifactorial process that ensures the stability of both the intracellular and extracellular environment of the body. Among all organs and tissues, the heart occupies a special place, as it functions under constant workload and has a high demand for energy. Cardiomyocytes are particularly sensitive to imbalances in macro- and microelements—primarily calcium, potassium, magnesium, and phosphorus—which determine both the structural integrity and the functional activity of the myocardium. Calcium plays a leading role in the processes of excitation and contraction of the cardiac muscle by regulating the contractile capacity of myofibrils. Disruption of calcium homeostasis leads to the development of calcification, mitochondrial damage, reduced ATP synthesis, and the initiation of apoptosis. Potassium is essential for maintaining the membrane potential and conducting electrical impulses; its deficiency or excess causes arrhythmias, destructive changes in cardiomyocytes, and damage to the vascular bed. Magnesium acts as a physiological antagonist of calcium, participating in the regulation of energy metabolism and stabilization of cell membranes. Phosphorus is vital for the synthesis of high-energy compounds and the construction of

phospholipid membranes; its deficiency or excess is accompanied by disturbances in cellular energetics and morphological changes in the myocardium. Disorders of mineral metabolism affect not only the cellular but also the tissue level. Morphologically, this is manifested by the development of dystrophy, fragmentation and necrosis of cardiomyocytes, interstitial edema, activation of fibroblasts, and the formation of interstitial fibrosis. These changes further contribute to myocardial remodeling, the development of chronic heart failure, and impairment of the pumping function of the heart. Endocrine factors play a particularly important role in regulating mineral metabolism. Parathyroid hormone, vitamin D, aldosterone, and other biologically active substances control the balance of calcium, phosphorus, sodium, and potassium. Their imbalance intensifies structural myocardial damage and aggravates metabolic disturbances. The relevance of studying the mechanisms of morphological changes in cardiac structure due to mineral metabolism disorders is underscored by the high prevalence of cardiovascular diseases worldwide and, in particular, in Uzbekistan, where cardiovascular mortality remains high. A deep understanding of the pathogenetic foundations of these processes provides a basis for developing new approaches to the prevention, diagnosis, and treatment of heart diseases associated with metabolic disorders.

Materials and Methods

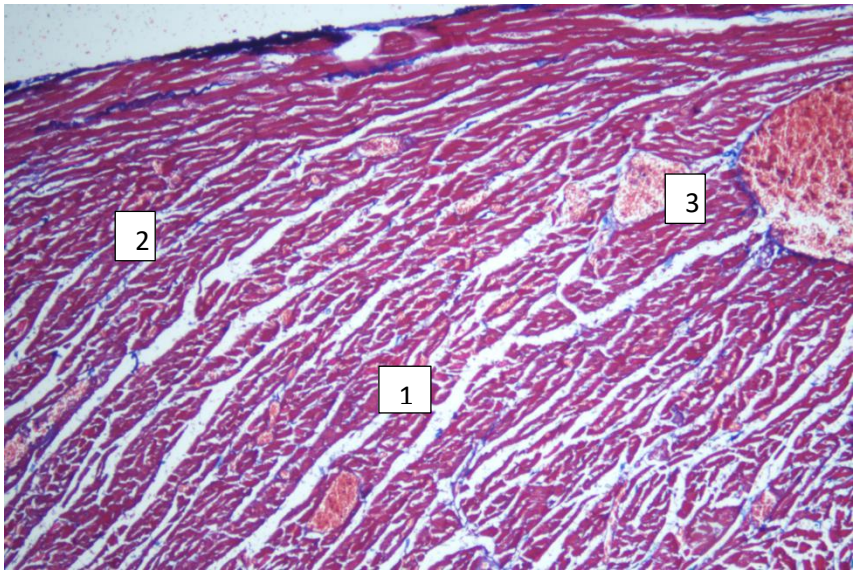
To study the mechanisms of morphological changes in cardiac structure under mineral metabolism disorders, experimental research was conducted on laboratory animals (Wistar rats), as this model is considered the most suitable for reproducing metabolic and morphological processes comparable to those in humans. The animals were divided into several groups: a control group and experimental groups, in which various types of mineral metabolism disturbances were modeled (hypocalcemia, hypercalcemia, hypokalemia, hypomagnesemia, and combined forms). To induce imbalance, special diets with a deficiency or excess of specific microelements were used. At the end of the experimental period, heart tissue was harvested and fixed in 10% neutral formalin. The material was processed using standard histological techniques and embedded in paraffin. Sections of 4–5 μm thickness were stained with hematoxylin-eosin for general morphology, Van Gieson's stain for connective tissue detection, and the PAS reaction for evaluation of carbohydrate components and membrane integrity. The animals were kept under standard vivarium conditions (temperature 22–24 °C, relative humidity 55–60%, light/dark cycle 12:12 h). Microscopy was performed using a Leica DM500 light microscope with digital photodocumentation. Morphometric analysis included measurements of myocardial fiber thickness, nuclear area, the degree of interstitial fibrosis, and capillary network density. Myocardial fibers were stained red, with well-defined cross-striated structures of cardiomyocytes. Connective tissue (collagen fibers) was stained blue, showing interlayers of connective tissue between muscle fibers, as well as areas of interstitial thickening, indicating interstitial fibrosis. The vascular bed and intermuscular spaces appeared as light, empty lumina, some of which were dilated. Local focal hemorrhage (hemorrhagia) was observed within the myocardium (brownish-red rounded formation to the left of the center). Overall, the tissue demonstrated signs of structural remodeling: alternating bundles of myocardial fibers with areas of connective tissue, which indicates fibrous-dystrophic changes.



Cardiac muscle stained with Masson's trichrome, ×400.

1 — cardiomyocytes (red muscle fibers with cross-striated structure).
2 — connective tissue (blue areas indicating interstitial fibrosis).
3 — focal hemorrhage (dark-red rounded inclusion).

The picture corresponds to morphological changes of the myocardium under mineral metabolism disorders: interstitial fibrosis, areas of vascular disturbances, and focal hemorrhage are revealed, reflecting processes of cardiac tissue remodeling. Cardiomyocytes partially preserve their typical cross-striated structure; however, signs of cytoplasmic staining heterogeneity are observed, indicating dystrophic changes. In some areas, loosening and deformation of muscle fibers are detected. The interstitial connective tissue is pronounced, with thickening of intermuscular septa and enhanced deposition of collagen fibers (stained blue), which indicates interstitial and perivascular fibrosis. Within the myocardium, focal hemorrhage is identified, reflecting vascular disturbances and increased capillary permeability. Intermuscular spaces are locally expanded and contain accumulations of collagen fibers, further confirming the processes of myocardial remodeling.



Cardiac muscle stained with Masson's trichrome, ×400.

1 – muscle fibers **2** – collagen fibers **3** – perivascular tissue

The overall picture corresponds to fibrous-dystrophic changes of the myocardium with signs of chronic damage, combining muscle atrophy, sclerosis, and vascular disorders. Dystrophic changes include granular, vacuolar, and fatty degeneration. Necrotic processes are identified, such as focal or massive myocardial necrosis. Interstitial alterations demonstrate edema, lymphocytic and macrophage infiltration, and fibrosis. Vascular disturbances include arteriolar calcification, sclerosis, and impaired microcirculation. Cardiosclerosis is observed as the outcome of chronic mineral imbalance. Histological slides were examined using a Leica DM500 light microscope equipped with a digital imaging system. Morphometric measurements included thickness of myocardial fibers, nuclear area of cardiomyocytes, percentage of interstitial fibrosis, density of capillary network. Quantitative morphometric data were collected from at least 10 randomly selected high-power fields per specimen.

Results of the Study

In the course of experimental morphological research, it was established that disturbances of mineral metabolism (deficiency and excess of calcium, phosphorus, magnesium, potassium, and trace elements) cause pronounced changes in the structure of the myocardium. In animals with mineral imbalance, the heart showed increased mass and chamber dilation. Histological examination revealed cardiomyocyte dystrophy (granular, vacuolar, fatty), focal and subtotal necrosis of myofibrils, interstitial edema, and infiltration by lymphocytes and macrophages. In calcium metabolism disorders: hypocalcemia was accompanied by sarcolemma destruction, myofibril fragmentation, and sarcoplasmic vacuolization. Hypercalcemia led to the formation of metastatic calcification foci, predominantly in subendocardial zones. Phosphate imbalance showed the following effects excess phosphates enhanced calcification, resulting in coarse calcium deposits in the myocardium and vessels. Phosphate deficiency caused severe energy deficiency, morphologically manifested by mitochondrial swelling and myofibrillar disorganization. Additionally, interstitial edema, small-focal necrosis, and pronounced fibrosis were observed. The number of connective tissue cells increased, and areas of cardiosclerosis were formed. Potassium metabolism disorders showed hypokalemia caused vacuolar dystrophy and focal myofibril breakdown. Hyperkalemia induced degenerative changes in the cardiac conduction system (nodes, bundles), confirmed by the development of arrhythmogenic foci. In cases of trace element imbalance iron excess (modeled hemochromatosis) produced hemosiderin granules in the myocardium and pronounced pigment dystrophy. Copper deficiency was accompanied by myocardial fiber thinning and the development of dilated cardiomyopathy. Quantitative analysis showed that the density of cardiomyocytes in affected zones decreased by 15–25% compared to the control. The connective tissue area increased by 30–40% in groups with pronounced hypocalcemia and hypomagnesemia. The incidence of focal calcinosis in hypercalcemia reached 60–70% of cases.

Conclusion

The conducted study demonstrated that disturbances in mineral metabolism exert a profound and multifaceted influence on the morphological structure of the heart. Imbalance of calcium, phosphorus, magnesium, potassium, and trace elements leads to disruption of ionic homeostasis, energy deficiency, and destructive changes in cardiomyocytes. Morphologically, this is manifested by dystrophy, necrosis, interstitial edema, calcification, and gradual replacement of functionally active fibers with connective tissue. The most severe myocardial damage was observed in combined calcium–magnesium and

phosphorus–calcium metabolism disorders, which resulted in the formation of calcification foci and diffuse cardiosclerosis. The outcome of these processes is structural and functional remodeling of the myocardium and the cardiac conduction system, creating a morphological substrate for the development of arrhythmias, chronic heart failure, and cardiomyopathies. Histological slides were examined using a Leica DM500 light microscope equipped with ×10, ×40, and ×100 oil immersion objectives, and a high-resolution digital camera for microphotography. Digital images were acquired under identical illumination and magnification settings to ensure comparability.Thus, morphological changes under mineral imbalance are regular in nature, reflecting a direct relationship between metabolic disturbances and myocardial structural organization. Their early detection and correction of mineral metabolism are of fundamental importance for the prevention of progressive cardiac pathology.

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