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MORPHOLOGICAL CHANGES OBSERVED IN THE HEART MUSCLES AS A RESULT OF DISORDERS IN THE REGULATION OF HYPERKALEMIA, HYPOMAGNESEMIA, HYPOKALEMIA, AND HYPONATREMIA EXCHANGE

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Abstract
Mineral metabolism plays a crucial role in maintaining the structural and functional integrity of the cardiac muscle. Imbalance in macro- and microelements such as potassium, calcium, magnesium, and sodium disrupts ionic homeostasis, leading to morphological and structural changes in the heart tissue. This study focuses on examining the morphological and biochemical alterations occurring in the myocardium of rats under conditions of disturbed mineral metabolism, based on biochemical analyses.

Keywords: Myocardium, rat, mineral metabolism, biochemistry, cardiomyocytes, calcium, sodium, morphology.

Introduction

The cardiac muscle (myocardium) relies on precise regulation of mineral balance to maintain its contractile activity and excitability [1]. Under hypercalcemic conditions, calcinosis develops within the heart tissue [4]. This process leads to cellular degeneration and the formation of fibrotic foci. As a result of calcinosis, myocardial elasticity decreases, the conduction system is damaged, and calcific cardiomyopathy or valvular calcinosis may develop. Clinically, these conditions manifest as arrhythmia and bradycardia [2].

Hypomagnesemia is one of the major causes of oxidative stress and contractile dysfunction in the myocardium. A decrease in magnesium levels reduces Na^+/K^+-ATPase activity, resulting in decreased intracellular potassium and increased calcium [8]. Consequently, mitochondrial damage, reduced energy production, and myofibrillar disorganization occur in cardiomyocytes. These changes lead to metabolic cardiomyopathy, QT interval prolongation, arrhythmias, and heart failure [3].

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Hypokalemia represents the most significant ionic imbalance leading to electrical instability in the heart. Reduced serum potassium prolongs electrical impulses and causes arrhythmogenic alterations. Morphologically, myofibrillar fragmentation, cellular swelling, and even sudden cardiac arrest may occur. In hyponatremia, intracellular fluid volume increases, resulting in osmotic edema [7]. Cardiomyocyte enlargement and decreased myofibrillar tension contribute to reduced contractile activity and myocardial dystrophy. Consequently, cardiomyopathy and rhythm disturbances are observed [5].

Normal physiological ranges of mineral elements are as follows: calcium in human plasma 2.2–2.6 mmol/L (ionized form 1.1–1.3 mmol/L), in rats 2.2–2.8 mmol/L; potassium in humans 3.5–5.1 mmol/L, in rats 4.0–5.5 mmol/L; magnesium in humans 0.7–1.1 mmol/L, in rats 0.7–1.2 mmol/L; sodium 135–145 mmol/L in humans, 140–150 mmol/L in rats [9]. Deviations from these physiological norms can cause morphological and functional changes in cardiac tissue [6].

Aim of the Study

To investigate morphological and biochemical changes occurring in the myocardium of rats due to disturbances in mineral metabolism.

Materials and Methods

A biochemical method was used to assess changes in mineral metabolism by determining the plasma concentrations of key electrolytes — calcium (Ca^{2+}), potassium (K^{+}), magnesium (Mg^{2+}), and sodium (Na^{+}). The experimental work was conducted on laboratory rats, and the obtained results were compared with the normative indicators of human plasma.

Animals were divided into two groups:

1. Control group — fed with a standard diet.
2. Experimental group — maintained on a diet with excessive calcium and restricted potassium, sodium, and magnesium.

The experiment lasted for 30 days.

Biochemical analysis: The serum levels of K^{+}, Ca^{2+}, Mg^{2+}, and Na^{+} were measured using flame photometry and colorimetric methods. The colorimetric method was primarily used to quantitatively determine calcium and magnesium concentrations by measuring light absorption or transmittance at a specific wavelength. Flame photometry involved excitation of plasma at high temperature, producing characteristic spectral lines for potassium and sodium. The intensity of these spectral lines was directly proportional to the concentration of the measured element.

These methods do not require complex instruments and can be performed using standard laboratory equipment such as photometers, flame photometers, or titration devices. The obtained data were compared with physiological norms to evaluate the biochemical and morphological changes associated with mineral imbalance in cardiac tissue.

Results

Biochemical analyses revealed significant alterations in serum ion concentrations under conditions of disturbed mineral metabolism. In experimental rats, such disturbances resulted in a series of

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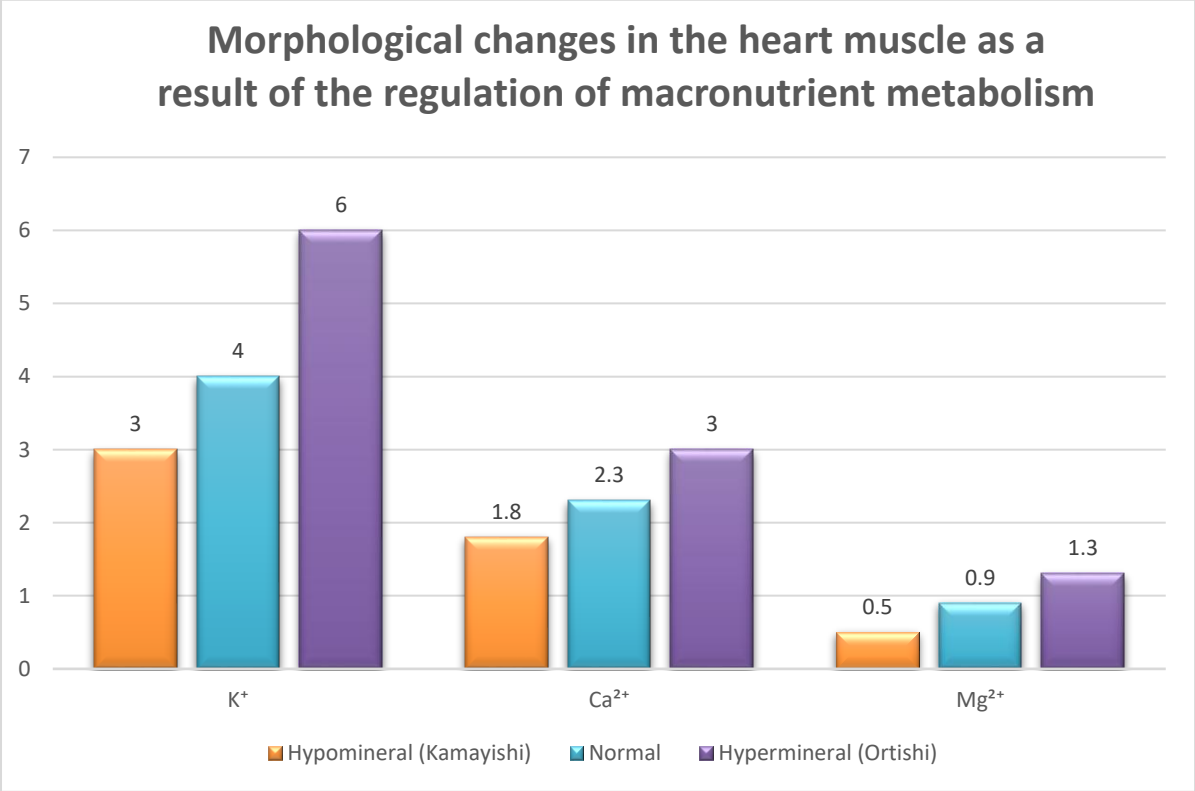
morphological and biochemical changes in the myocardium. Under hypercalcemia, calcinosis, reduced cardiomyocyte elasticity, and interstitial fibrosis were observed.

Electrolyte	Hypomineral (Decrease)	Normal	Hypermineral (Increase)
K^+	3.0	4.0	6.0
Ca^{2+}	1.8	2.3	3.0
Mg^{2+}	0.5	0.9	1.3
Na^+	120	145	160



The results confirm that mineral metabolism plays a decisive role in maintaining myocardial structural integrity and functional stability. Changes in calcium and potassium levels directly affect rhythmic contractility, while magnesium regulates energy metabolism and Na^+/K^+ balance. Sodium and chloride ensure osmotic equilibrium and stabilize cardiomyocyte volume. Thus, morphological findings in rats — including calcinosis, fibrosis, dystrophic alterations, and cellular swelling — form the structural basis of cardiomyopathy, arrhythmias, and heart failure.

Conclusion

The results of biochemical and morphological investigations demonstrated that disturbances in mineral metabolism significantly affect the structural and functional integrity of the myocardium. Experimentally induced hypo- and hypermineral states in rats led to notable changes in serum concentrations of calcium, potassium, magnesium, and sodium.

Fluctuations in potassium and calcium levels directly influenced electrical stability and contractility of the heart muscle. Hypercalcemia caused calcinosis and fibrosis, reducing myocardial elasticity.

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Hypokalemia resulted in repolarization disturbances, electrical instability, myofibrillar fragmentation, and cellular edema. Hypomagnesemia led to mitochondrial dysfunction, decreased energy synthesis, and oxidative stress, causing contractile dysfunction. Variations in sodium concentration caused osmotic imbalance, cell swelling, and cardiomyocyte hypertrophy.

Morphologically, these changes were observed as sarcoplasmic clarification, vacuolar degeneration, and interstitial fibrosis. These findings confirm that maintaining the physiological balance of mineral elements is essential for normal cardiac activity. Both deficiency and excess of electrolytes can directly impair myocardial metabolism, morphology, and electrical conductivity. Therefore, monitoring calcium, potassium, magnesium, and sodium levels is of critical diagnostic and preventive importance in conditions such as cardiomyopathy, arrhythmia, and heart failure.

Experimental findings in rats clearly demonstrate a direct relationship between mineral imbalance and morphological alterations in cardiac tissue, scientifically confirming that maintaining electrolyte homeostasis is vital for cardiac physiological stability in humans.

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