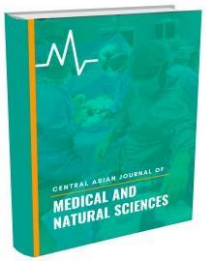

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Article

Morphological Features Cardiac Muscles in Hyperparathyroidism

Gulnoza Ozodovna Khidirova[1]

1. Assistant professor, Department of Anatomy, Pathological Anatomy, Tashkent Pediatric Medical Institute, Uzbekistan

* Correspondence: gulozo1980@mail.ru

Annotation: The goal of this research is to determine how variations in parathyroid gland functional activity affect the morphological intensity of the heart muscles. The dynamics of the genesis of cardiac muscles is disclosed, and changes in the structure and morphology of the heart's major vessels are researched, thanks to the findings of morphological research methods. The study's findings revealed a distinction between the hyperparathyroid patient's normal histological appearance of the heart's major arteries.

Keywords: calcium, hyperphosphatemia, hypocalcemia, parathyroid hormone, and hyperparathyroidism

1. Introduction

The parathyroid glands are special organs that are in charge of preserving calcium homeostasis, which is an essential function[1], [2], [3]. The parathyroid glands are normally found in the neck, posterior and inferior to the thyroid [4], [5]. They weigh around 40 grams apiece[6], [7], [8]. The hormone parathyroid hormone (PTH), which regulates calcium, is secreted by several organs [9], [10], [11], [12]. Through positive and negative feedback loops, serum calcium, phosphorus, and vitamin D all influence PTH secretion[13], [14]. The hormone parathyroid hormone (PTH), which regulates calcium, is secreted by several organs [15], [16].

When the body produces insufficient parathyroid hormone (PTH), the condition known as hyperparathyroidism results [17], [18]. PTH aids in controlling the body's calcium and phosphate levels. Individuals who suffer from hyperparathyroidism may be more susceptible to cardiac issues[19], [20]. Examples of patients with various complications associated with hyperparathyroidism can be found in medical literature [21], [22], [23]. These complications include dilated cardiomyopathy, which is characterized by a stretched, thin, and weak heart muscle, and heart arrhythmias, or irregular heartbeats [24], [25]. The heart may find it more difficult to pump blood to the body's tissues as a result of either of these problems.

2. Objective of the study

The rare disorder known as hyperparathyroidism is characterized by low or nonexistent PTH levels, hyperphosphatemia, and hypocalcemia. Hyperparathyroidism has a

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diverse etiology that can be either acquired or congenital. DiGeorge syndrome, for instance, is caused by a chromosomal deletion at 22q11.2 and is characterized, among other things, by parathyroid hypoplasia, cardiac anomalies, thymic hypoplasia, neurocognitive issues, and abnormalities of the kidneys and bones. The hypocalcemia that results from hyperparathyroidism causes cardiac consequences. For instance, QT prolongation brought on by hypocalcemia can put patients at risk for potentially fatal arrhythmias. Persistent hypocalcemia-related dilated cardiomyopathy is another well-known but rare consequence. Though the precise mechanism is unknown, hypocalcemia is thought to decrease cardiac contractility. PTH seems to affect cardiac cells in a different way as well. Remember that PTH has a positive chronotropic effect on cardiomyocytes, which is how it maintains appropriate cardiac contractility, as demonstrated by studies on molecules and animals. In fact, case studies have shown that treating hypocalcemia in people with isolated hyperparathyroidism can restore systolic dysfunction. Both the direct action of PTH on the heart and alterations of calcium homeostasis (e.g., hypercalcemia or hypocalcemia) comprise the two primary mechanisms by which diseases of the parathyroid affect the cardiovascular system. In recent years, clinical and molecular research has bolstered awareness of several cardiovascular complications that are associated with parathyroid disorders—namely, hypertension, arrhythmias, heart failure, and calcific disease of vessels and valves. Because parathyroid hormone (PTH) is produced by the parathyroid glands, it is essential for preserving calcium homeostasis. Through the downstream activities of G protein-coupled receptors in the myocardium and endothelial cells, PTH also affects the heart and vasculature, as demonstrated by recent clinical and molecular research. According to one study, individuals with hyperparathyroidism may also be suffering from cardiovascular autonomic neuropathy (CAN), a disorder that mainly affects diabetics. Autonomic nervous system dysfunction, or CAN, can cause weariness, arrhythmia, vertigo, irregular blood pressure, and unexpected heart attacks.

3. Materials and Methods

According to recent research, PTH affects smooth muscle and cardiomyocyte physiology. PTH is structurally a peptide hormone made up of 84 amino acids, of which the first 34 are physiologically active. Target organ PTH1R is a G-protein-coupled receptor with seven transmembrane domains that this moiety interacts to. The downstream activation of the phospholipase C/protein kinase C (PKC) pathway or the adenylyl cyclase and protein kinase A pathway is typically involved in the subsequent activation of this receptor, depending on the target organ. PTH binding in adult cardiomyocytes triggers G-protein signaling, which in turn causes an inflow of calcium into the heart cells. However, this calcium influx is expected to cause a number of indirect effects on the myocardium rather than directly causing contractile effects on cardiac myocytes. One of these is PKC activation, which can reduce contractility by obstructing the stimulation of B-adrenoceptors. Furthermore, it is believed that PKC's downstream effects on protein production, gene expression, and cell proliferation result in excessive growth.

4. Results

Heart failure and reversible dilated cardiomyopathy are major outcomes of hyperparathyroidism with severe hypocalcemia, which can be treated with calcium. Ignorance of this etiology may result in improper treatment of heart failure with loop diuretics, exacerbating hypocalcemia and posing potentially fatal risks. Blood calcium levels corrected for albumin had a correlation with the coronary calcium index. The chances of developing cardiovascular pathology persist even in the face of evidence of elevated markers of cardiovascular illnesses in people with chronic hyperparathyroidism. A big Danish study found no difference in the general population's risk of cardiovascular pathology (coronary heart disease, heart rhythm problems, etc.) principles. That being said, there

was a database of patients with surgical hyperparathyroidism that was retrospectively studied. Additional research examining the pathogenetic and risk variables associated with cardiovascular disease in chronic hyperparathyroidism.

5. Discussion

Through the downstream signaling of cardiac G protein-coupled receptors, parathyroid hormone affects smooth muscle cells and cardiomyocytes directly as well as indirectly. These effects alter the cardiac myocytes' contractility, proliferation, and hypertrophy. PTH is a vasorelaxant that acts directly on vascular smooth muscle cells within the vasculature. Excess PTH is known to have detrimental effects on the cardiovascular system, including an increased risk of heart failure, arrhythmias, hypertension, left ventricular hypertrophy, and calcific valve disease. However, it is still unknown how a persistent PTH deficiency affects the structure, operation, and conduction of the heart.

6. Conclusion

The parathyroid glands play a critical role in calcium homeostasis in relation to the cardiovascular system, in addition to being essential for preserving bone metabolism. It is evident that PTH, calcium, phosphorus, and vitamin D all have direct and indirect effects on the heart, even though the precise processes are still unclear. It is becoming more widely acknowledged that abnormalities in parathyroid function are linked to calcific disease, heart failure, LVH, and hypertension—all of which raise the risk of cardiac morbidity and mortality. Research is beginning to demonstrate that treating patients with parathyroid illness at an early stage of the condition may be able to prevent or even reverse negative consequences on the cardiovascular system. Meanwhile, more studies with bigger cohorts and longer follow-up periods of individuals with parathyroid problems are required to gain a deeper understanding of the long-term cardiac consequences of untreated disease. The two main ways that parathyroid disorders impact the cardiovascular system are by direct effect of PTH on the heart and changes in calcium homeostasis, such as hypo- or hypercalcemia. Individuals with parathyroid gland abnormalities are more likely to experience heart failure, hypertension, arrhythmias, left ventricular hypertrophy, and calcific disease. These conditions raise the risk of cardiac morbidity and mortality.

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