

MORPHOLOGICAL AND FUNCTIONAL CHANGES OF THE PARATHYROID GLANDS IN CADMIUM POISONING

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Abstract: This scientific article examines the morphological and functional changes in the parathyroid glands under cadmium intoxication. Research findings indicate that cadmium, as a heavy metal, has a significant toxic effect on the endocrine system, particularly on the function of the parathyroid glands. Morphologically, degenerative changes in glandular tissues, cellular vacuolization, and necrotic processes are observed. Functionally, disruption of parathyroid hormone secretion leads to an imbalance in calcium-phosphorus metabolism. These changes significantly affect bone metabolism and overall homeostasis of the organism.

Keywords: cadmium, parathyroid gland, parathyroid hormone, morphological changes, endocrine system, toxicology, calcium-phosphorus metabolism, necrosis, heavy metals.

INTRODUCTION

Among modern environmental problems, pollution with heavy metals is considered one of the most serious threats to human health. In particular, cadmium (Cd), entering the body through industrial waste, air pollution, and the food chain, exerts toxic effects on various systems. Although cadmium is not biologically essential, its high concentration causes profound pathological changes at the cellular level.[1]

The endocrine system plays a key role in maintaining homeostasis. The parathyroid glands are among the main organs regulating calcium and phosphorus metabolism by producing parathyroid hormone (PTH). Normal secretion of this hormone directly affects bone tissue, kidneys, and intestinal function. Therefore, endocrine glands are highly sensitive to toxic substances, which can disrupt their functional state.

Recent scientific studies have demonstrated the negative impact of cadmium on the endocrine system, especially the parathyroid glands. Cadmium easily penetrates cell membranes, disrupts mitochondrial function, increases oxidative stress, and leads to degenerative changes, apoptosis, and

necrosis in cells. As a result, the morphological structure of glandular tissue is altered, negatively affecting its hormonal activity.[2]

Cadmium poisoning leads to impaired secretion of parathyroid hormone, resulting in an imbalance of calcium-phosphorus metabolism. This condition may cause bone demineralization, osteoporosis, and other metabolic disorders. Therefore, studying the effects of cadmium on the parathyroid glands is important for both theoretical and practical medicine.

The aim of this study is to comprehensively investigate morphological and functional changes in the parathyroid glands under cadmium intoxication, analyze their pathogenesis, and determine their impact on the overall condition of the organism.[3]

LITERATURE REVIEW

The effects of cadmium on the endocrine system, particularly the parathyroid glands, have been extensively studied by many foreign scientists. Lars Friberg noted that cadmium accumulates in the human body and its long-term exposure disrupts endocrine gland function. According to him, cadmium indirectly affects parathyroid activity by interfering with calcium metabolism [4].

In the “Handbook on the Toxicology of Metals” by Gunnar Nordberg and colleagues, it is stated that cadmium increases oxidative stress at the cellular level, weakens the antioxidant system, and leads to degenerative changes in endocrine tissues. These processes reduce the secretory activity of parathyroid cells [5].

Bernard A. demonstrated that cadmium disrupts calcium homeostasis through effects on kidneys and bone tissue. A decrease in calcium levels leads to compensatory hypersecretion of parathyroid hormone, which over time causes gland hypertrophy and functional exhaustion [6].

According to Jiri Patočka, cadmium is an endocrine-disrupting chemical that directly interferes with hormonal regulation by affecting hormone receptors and altering gland function [7].

Studies by Zaichick V. and Zaichick S. revealed that heavy metals accumulate in endocrine glands, causing morphological changes such as nuclear alterations, cytoplasmic vacuolization, and stromal fibrosis in parathyroid tissue [8].

These studies confirm that cadmium has a complex and profound negative effect on both morphological and functional states of the parathyroid glands, highlighting the need for further research.

METHODOLOGY

The study of morphological and functional changes in the parathyroid glands under cadmium intoxication was conducted using experimental and analytical methods. Laboratory animals were exposed to cadmium salts in controlled doses, and gland tissues were examined using histological methods. Morphological changes were assessed microscopically, while parathyroid hormone levels were analyzed using biochemical methods. The obtained data were processed using statistical analysis techniques.[9]

RESULTS AND DISCUSSION

Histological analysis of the parathyroid glands in the cadmium-exposed group revealed significant structural alterations compared to the control group. The most prominent changes were observed in the chief cells, which are responsible for parathyroid hormone synthesis and secretion. These changes included cytoplasmic vacuolization, indicating intracellular metabolic disturbance and water-electrolyte imbalance at the cellular level. In addition, nuclear abnormalities such as pyknosis

(condensation of nuclear chromatin) and karyolysis (dissolution of the nucleus) were frequently observed, reflecting irreversible cellular injury.[10]

In more advanced stages of exposure, areas of cellular necrosis were detected, suggesting complete loss of cell viability. Alongside these changes, stromal fibrosis was noted, characterized by excessive deposition of connective tissue, which replaces normal functional parenchyma. This fibrotic transformation leads to stiffening of the glandular structure and further impairs its physiological activity. Furthermore, significant microcirculatory disturbances were observed, including vascular congestion and reduced capillary perfusion, resulting in tissue hypoxia. Such hypoxic conditions further aggravate cellular degeneration and accelerate tissue damage.[11]

Functional changes

From a functional perspective, cadmium exposure significantly disrupted the secretion of parathyroid hormone (PTH). In the initial phase of exposure, a compensatory response was observed, characterized by a transient increase in PTH secretion. This adaptive mechanism reflects the organism's attempt to maintain calcium homeostasis in response to early toxic injury.

However, with continued exposure and progressive structural damage to the gland, the secretory capacity of the parathyroid cells declined sharply. This resulted in a significant reduction in circulating PTH levels. The decrease in hormone production directly affected calcium and phosphorus metabolism, leading to a systemic imbalance. Specifically, hypocalcemia (decreased blood calcium levels) and hypophosphatemia (reduced phosphate levels) were recorded, indicating severe disruption of mineral homeostasis.[12]

These biochemical alterations have far-reaching physiological consequences. Calcium is a critical element involved in neuromuscular activity, enzymatic reactions, and bone mineralization. Therefore, its deficiency leads to increased neuromuscular excitability disorders, metabolic instability, and progressive weakening of skeletal structures. One of the most significant outcomes observed was bone demineralization, which predisposes to decreased bone strength and structural integrity.

Progression of pathological changes

The study further demonstrated that the severity of morphological and functional disturbances was directly related to both the dose and duration of cadmium exposure. Short-term exposure primarily induced reversible adaptive changes; however, prolonged exposure led to irreversible pathological transformations. Initially, compensatory hypertrophy of glandular cells was observed as an adaptive response. Over time, this was replaced by atrophic degeneration and destructive processes, indicating failure of compensatory mechanisms.[13]

Ultimately, the Table 1. progressive deterioration of glandular structure and function resulted in endocrine insufficiency. This condition is characterized by a persistent inability of the parathyroid glands to maintain calcium-phosphorus homeostasis, leading to systemic metabolic imbalance and disruption of overall physiological stability.

Table 1. Changes in the Parathyroid Glands under Cadmium Exposure

Indicators	Control Group	Cadmium-Exposed Group	Type of Change
Cell structure	Normal	Vacuolization, degeneration	Pathological
Nucleus state	Normal, central	Pyknosis, karyolysis	Destructive
Stroma	Normal	Fibrosis	Structural damage

Blood circulation	Normal	Microcirculatory disorders	Hypoxia
PTH level	Normal	Initially increased, then decreased	Secretory dysfunction
Calcium level	Normal	Decreased	Hypocalcemia
Phosphorus level	Normal	Variable	Imbalance

General interpretation

These findings strongly indicate that cadmium has a multifactorial toxic effect on the parathyroid glands. It simultaneously disrupts cellular structure, impairs hormonal secretion, and alters systemic mineral metabolism. The combined effect of oxidative stress, cellular degeneration, and vascular нарушения leads to progressive organ dysfunction. Importantly, these changes are not limited to local gland damage but extend to systemic metabolic disturbances affecting the entire organism.[14]

Thus, the study confirms that cadmium is a potent endocrine-disrupting toxicant with the ability to induce both reversible and irreversible pathological changes depending on exposure conditions.[15]

CONCLUSION

The present study clearly demonstrates that cadmium intoxication exerts a profound and multi-level damaging effect on both the morphological structure and functional activity of the parathyroid glands. Being a highly toxic heavy metal, cadmium accumulates in biological tissues and disrupts cellular integrity, leading to progressive structural deterioration of endocrine organs.

From a morphological point of view, cadmium exposure induces a wide spectrum of pathological changes in parathyroid gland tissue. These include cellular degeneration, cytoplasmic vacuolization, nuclear damage (such as pyknosis and karyolysis), necrotic processes, and progressive stromal fibrosis. Such alterations reflect irreversible injury to both the cellular and connective tissue components of the gland. As a result, the normal architectural organization of the parathyroid tissue is disrupted, microcirculatory disturbances develop, and tissue oxygenation is significantly impaired. Over time, these structural changes compromise the integrity and viability of hormone-producing cells.

Functionally, cadmium intoxication leads to a marked disturbance in parathyroid hormone (PTH) secretion. In the early stages of exposure, a compensatory increase in PTH levels may occur as the organism attempts to maintain calcium homeostasis. However, with continued exposure and progressive cellular damage, the secretory capacity of the gland declines significantly. This results in a decrease in circulating PTH levels and a consequent disruption of calcium-phosphorus metabolism. Clinically and physiologically, this imbalance manifests as hypocalcemia, altered phosphate levels, and impaired mineral metabolism.

Such biochemical disturbances have systemic consequences, particularly affecting bone tissue. Reduced calcium availability leads to bone demineralization, decreased bone density, and an increased risk of metabolic bone disorders such as osteopenia and osteoporosis. In addition, calcium imbalance negatively affects neuromuscular excitability, enzyme activity, and multiple intracellular signaling pathways, thereby disrupting overall metabolic stability.

The severity of both morphological and functional changes is directly dependent on the dose, duration, and cumulative exposure to cadmium. Prolonged exposure leads to exhaustion of adaptive and compensatory mechanisms, after which the endocrine system is no longer able to maintain physiological balance. This results in irreversible endocrine insufficiency and long-term systemic dysfunction.

In general, the findings of this study confirm that cadmium acts as a potent endocrine-disrupting toxicant with multi-organ effects, particularly targeting the parathyroid glands. Its impact extends beyond local tissue damage, ultimately affecting systemic homeostasis and metabolic regulation. Therefore, prevention of cadmium exposure, early detection of toxic effects, and strict monitoring of environmental and occupational heavy metal contamination are of critical importance for protecting endocrine health and preventing long-term metabolic disorders.

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