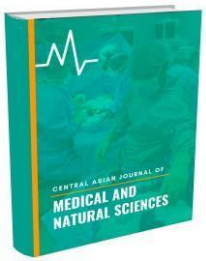

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Article

Comparison of Morphological and Morphometric Parameters of the Thyroid Gland in Zinc-Deficient White Rats

O.G. Aslanov[1], R.R. Baymuradov[2]

1. Independent Researcher, Department of Anatomy, Clinical Anatomy (OSTA), Bukhara State Medical Institute named after Abu Ali ibn Sino
2. Associate Professor, Department of Anatomy, clinical anatomy (OSTA), Bukhara State Medical Institute named after Abu Ali ibn Sino, PhD

Correspondence: [1]aslonov3171@gmail.com, [2]ravshan.baymuradov@bsmi.uz

Abstract: This study analyzes the morphological and morphometric alterations of the thyroid gland in zinc-deficient white rats compared with healthy controls. Zinc deficiency is known to impair thyroid hormone synthesis, transcriptional regulation, and antioxidant defense mechanisms through its essential role in the activity of thyroid peroxidase (TPO) and multiple zinc-dependent metalloenzymes. Experimental findings demonstrated significant changes in capillary parameters, follicular dimensions, epithelial height, stromal composition, and colloid structure under Zn deficiency. These results highlight the critical role of zinc in maintaining thyroid follicular integrity and endocrine homeostasis, confirming the close relationship between microelement balance and thyroid functional status.

Keywords: Zinc Deficiency, Thyroid Gland, Morphology, Morphometry, Microelements, TPO Activity, Follicular Structure, White Rats

1. Introduction

The thyroid gland is one of the most metabolically active endocrine organs, and its functional stability depends on a balanced supply of essential trace elements. Among these, zinc (Zn) plays a central biochemical and structural role in thyroid hormone synthesis, hormone receptor activation, redox homeostasis, and immune regulation. Numerous studies emphasize that zinc is required for the activity of several Zn-dependent enzymes and transcription factors, particularly thyroid peroxidase (TPO), which catalyzes iodination and coupling reactions essential for the production of T3 and T4 hormones. Maxwell and Volpe demonstrated that zinc deficiency reduces TPO activity and impairs the interaction between thyroid hormones and their nuclear receptors, leading to altered gene transcription and weakened hormonal signaling within thyroid cells [1].

Experimental investigations confirm that Zn deficiency produces significant structural changes in the thyroid gland. In a controlled model, de Angelis et al reported follicular shrinkage, epithelial atrophy, reduced colloid density, and disruption of microvascular architecture in Zn-deficient rats [2]. Similar outcomes were described by Baltaci and Mogulkoc who observed alterations in T3/T4 levels and morphological remodeling of follicular cells associated with reduced zinc availability [3].

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Zinc is also critical for the metabolic conversion of thyroid hormones. According to Severo et al. Zn participates in deiodinase function and modulates the peripheral metabolism of T4 into the biologically active T3 form. Clinical findings further demonstrate the physiological relevance of zinc [4]. Yildiz et al. reported that zinc supplementation in children with marginal Zn deficiency significantly improves T3 levels and enhances overall thyroid functional status. Additional human studies, including the work of Koumbi identify zinc as an important immunoregulatory factor that influences autoimmune thyroid disorders such as Hashimoto's thyroiditis [5].

Taken together, these findings indicate that zinc deficiency leads to a cascade of structural, functional, and immunometabolic disturbances in the thyroid gland. Despite a wide array of clinical and biochemical studies, detailed histomorphometric characterization of thyroid tissue alterations under Zn-deficient conditions in experimental models is still limited. Therefore, the present study aims to evaluate the morphological and morphometric parameters of the thyroid gland in zinc-deficient white rats compared with healthy controls, assess microcirculatory alterations, and clarify the pathogenetic mechanisms underlying Zn-related endocrine imbalance[6].

The aim of this study was to determine the histomorphometric characteristics of the thyroid gland in white outbred rats under conditions of zinc (Zn) deficiency, including alterations in follicular structure, colloid density, epithelial layer morphology, and elements of the microcirculatory system. In addition, the study sought to evaluate the impact of zinc deficiency on the functional activity and morphometric parameters of the thyroid gland and to provide a scientific rationale for the major pathogenetic mechanisms of endocrine imbalance associated with Zn deficiency[7].

2. Methodology

Experimental studies were conducted on male white outbred rats weighing 150–170 g, maintained under standard vivarium conditions. The animals were divided into two main groups:

Group I – Control: rats maintained on a standard diet.

Group II – Zinc-deficient (Zn-): rats fed a specialized diet with zinc content below physiological requirements[8].

As the experimental model, diets produced by ALTROMIN Spezialfutter GmbH & Co. KG (Germany) were used, including C 1000, C 1035, C 1038, C 1040, C 1045, as well as a modified C 1035 diet adapted for zinc deficiency. The duration of the experiment was 10 weeks. All procedures were carried out in accordance with ethical approval certificate No. 36/2024.

Histological preparations of thyroid gland tissues were obtained. Standard hematoxylin–eosin (H&E) staining was used, and in selected samples, PAS reaction and additional staining methods were applied to assess colloid homogeneity. Morphometric measurements were performed using a calibrated micrometric scale and included such parameters as follicle diameter, epithelial height, colloid area, arteriolar diameter, and capillary density[9].

Both parametric and nonparametric statistical methods were applied to experimental data. Primary data collection, cleaning, and systematization were performed using Microsoft Excel 2016. Statistical analysis was carried out in IBM SPSS Statistics v.26[10].

The distribution of quantitative variables was assessed using the Shapiro–Wilk test ($n < 50$) or the Kolmogorov–Smirnov test ($n > 50$). For non-normally distributed data, the Mann–Whitney U-test was applied; for normally distributed data, the Student's t-test was used. Graphical visualization of results was performed using STATGRAPHICS 5.1. During statistical processing, the mean value (M), standard error of the mean (m), variance, and

coefficient of variation were calculated. Differences relative to the control group were evaluated in percentage terms. Statistical significance was accepted at $p < 0,05$[11].

3. Results and Discussion

Given the increasing evidence that zinc deficiency contributes to epithelial atrophy, impaired folliculogenesis, altered colloid synthesis, and microvascular changes in the thyroid gland, experimental evaluation using white rat models remains essential. Therefore, assessing structural and morphometric changes under Zn deficiency provides valuable insights into endocrine regulation and the pathophysiology of thyroid dysfunction related to micronutrient imbalance[12].

Zinc (Zn) is an essential trace element that serves as a structural and functional component of numerous transcription factors and enzymes, thereby participating in the regulation of gene expression, cell proliferation, and differentiation. Its role in metabolic processes associated with thyroid gland function is particularly important. Zinc acts as a cofactor for various metalloenzymes directly involved in thyroid hormone synthesis and peripheral metabolism[13].

The Figure 1 shows that, activity of the thyroid peroxidase (TPO) enzyme critically depends on zinc, as adequate Zn availability supports the iodination of thyroglobulin and the coupling of iodotyrosine residues, ensuring the synthesis of T4 and T3 hormones. In addition, zinc contributes to antioxidant defense by limiting the excessive generation of reactive oxygen species (ROS), thereby mitigating inflammatory responses.

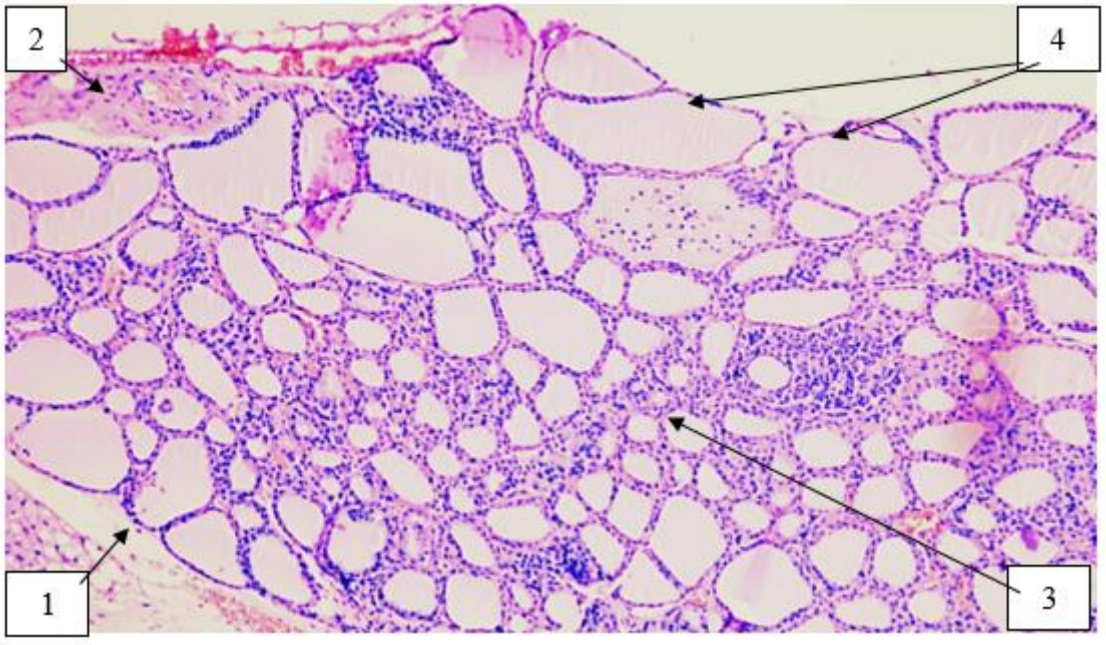

Figure 1. Microscopic changes in the thyroid gland of 6-month-old zinc-deficient white rats. Hematoxylin–eosin staining, ×200 magnification (10× ocular, 20× objective). 1 — the capsule of the gland is unevenly thickened. 2 — edema and proliferation of connective tissue in the vascular wall and perivascular region. 3 — centrally located follicles are reduced in size, contain a small amount of colloid, and exhibit a markedly thickened follicular wall. 4 — follicles in the peripheral region are of medium size, with decreased colloid content and thickened follicular epithelium.

Zinc is also essential for immune regulation, including the modulation of innate and adaptive immunity, activation of T-lymphocytes, and their differentiation into various subpopulations. A reduction in Zn levels can impair immune responses, alter signaling

pathways, and predispose individuals to autoimmune diseases. Disturbances in zinc homeostasis are particularly common in Hashimoto's thyroiditis and other autoimmune thyroid disorders[14].

Zinc deficiency affects not only thyroid hormone synthesis but also the functional activity of thyroid hormone receptors. This compromises the effective binding of thyroid hormones to nuclear receptors, disrupts normal gene regulation, and weakens thyroid hormone signaling. Consequently, hormone synthesis declines and the risk of developing hypothyroidism increases.

The Figure 2 shows that, zinc deficiency in patients with thyroid disorders manifests through various symptoms such as hair loss, impaired pigmentation, brittle hair, and dry skin. These manifestations are linked to the role of zinc as a component of multiple metalloenzymes required for keratin and melanin synthesis.

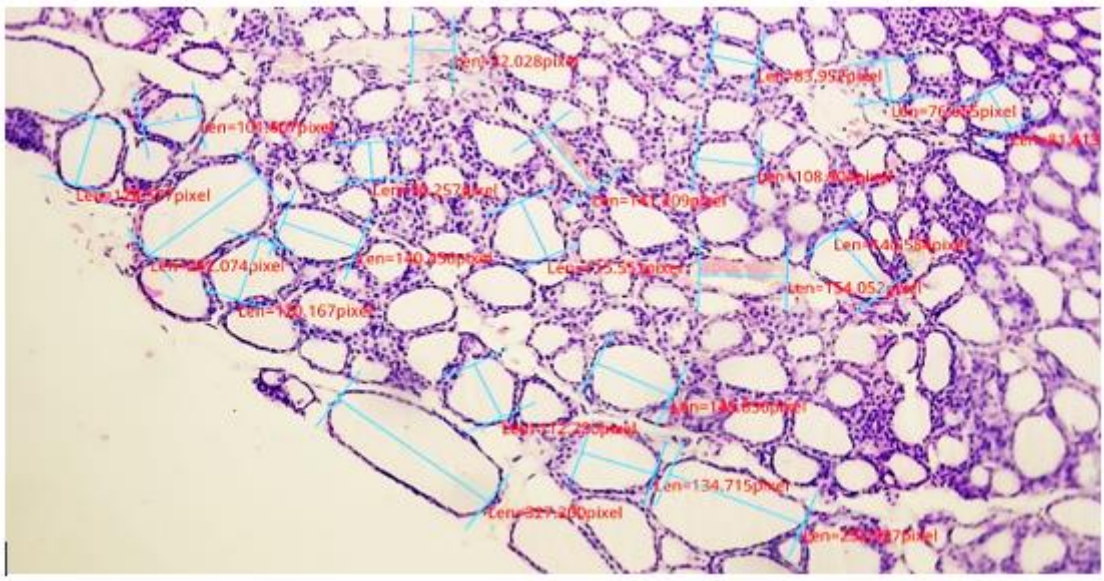

Figure 2. Morphometric characteristics of the thyroid gland in 6-month-old zinc-deficient white rats. Hematoxylin–eosin staining, ×200 magnification.

Major dietary sources of zinc include fish, red meat, whole grains, dairy products, nuts, and legumes. Adequate zinc intake is essential for maintaining thyroid gland function, supporting immune competence, and ensuring stable cellular metabolism.

In 6-month-old white rats with zinc deficiency, a marked decrease in the colloid-producing activity of thyrocytes was observed. Quantitative assessment revealed a reduction in the height and width of thyrocytes, indicating varying degrees of cellular atrophy[15].

The Figure 3 shows that, number and size of follicles were diminished. Follicle diameter measured 33.62 ± 0.29 μm in the central region and 39.84 ± 0.18 μm in the peripheral region; follicular area was 1132.8 ± 17.52 μm² centrally and 1462.3 ± 15.84 μm² peripherally. Analysis of the proportional composition of follicular components showed that thyroid epithelium accounted for 36.4 ± 0.34% centrally and 31.8 ± 0.41% peripherally.

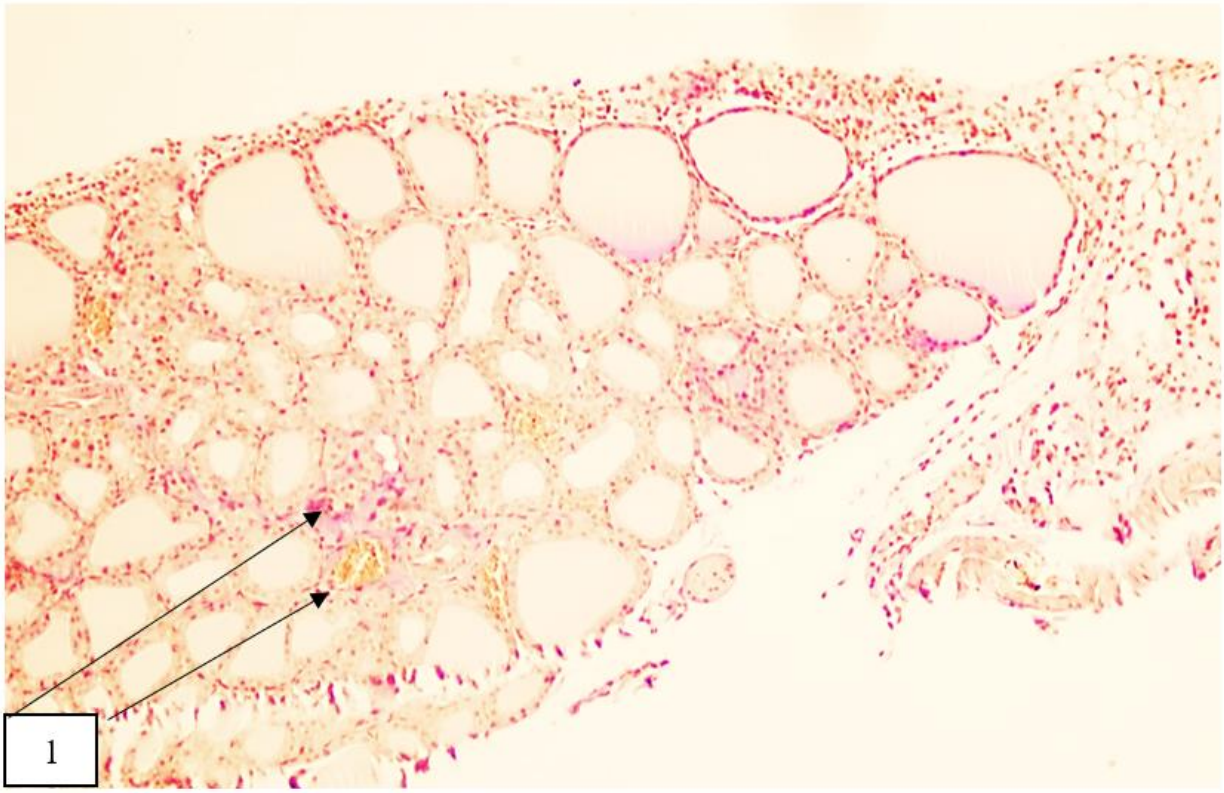

Figure 3. Microscopic appearance of the thyroid gland in 6-month-old zinc-deficient white rats. Van Gieson staining, ×200 magnification (10× ocular, 20× objective). 1 — collagen fibers located within the vascular wall and perivascular connective tissue.

4. Conclusion

In 6-month-old white rats with zinc deficiency, a marked decrease in the colloid-producing activity of thyrocytes was observed. Quantitative assessment revealed a reduction in the height and width of thyrocytes, indicating varying degrees of cellular atrophy. The number and size of follicles were diminished. Follicle diameter measured 33.62 ± 0.29 μm in the central region and 39.84 ± 0.18 μm in the peripheral region; follicular area was 1132.8 ± 17.52 μm² centrally and 1462.3 ± 15.84 μm² peripherally. Analysis of the proportional composition of follicular components showed that thyroid epithelium accounted for 36.4 ± 0.34% centrally and 31.8 ± 0.41% peripherally.

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