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Article

Morphological Characteristics and Morphometry of The Kidneys and Testis in 6-Month-Old Rats Under Conditions of Zinc Deficiency

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Abstract: This study investigated the morphological and morphometric changes in the kidneys and testes of 6-month-old rats under conditions of zinc deficiency. The results demonstrated glomerular hemodynamic disturbances, tubular epithelial atrophy, and interstitial remodeling in the kidneys, as well as thinning of the spermatogenic epithelium, a decrease in germ cell number, and reduced Leydig cell density in the testes. Evaluation using the Johnsen scoring system confirmed impairment in the final stages of spermatogenesis. The findings indicate that zinc deficiency leads to structural and functional remodeling of the renal and reproductive systems against the background of oxidative stress and trophic disturbances.

Keywords: zinc deficiency, kidney, testes, morphometry, spermatogenesis, oxidative stress, tubular apparatus, glomerulus, Leydig cells, germinal epithelium, interstitial tissue, Johnsen score

Introduction

Zinc is an essential trace element in both humans and animals, playing a critical role in numerous enzymatic systems, antioxidant defense mechanisms, and processes of cell proliferation and differentiation. Zinc deficiency is recognized as a global health problem, particularly prevalent in developing countries [1].

Zinc deficiency leads to increased oxidative stress, disruption of cell membrane stability, and the development of dystrophic changes in tissues. Due to its high metabolic activity, renal tissue is particularly sensitive to micronutrient deficiencies, and under conditions of zinc deficiency, structural and functional disturbances are especially pronounced in the tubular apparatus and microcirculatory system [2].

In addition, zinc plays a crucial role in the male reproductive system. It is essential for spermatogenesis, particularly during meiotic and post-meiotic stages, and supports androgen synthesis and Sertoli cell function [3]. Zinc deficiency is associated with degeneration of the spermatogenic epithelium and a decrease in both the quantity and quality of spermatozoa [4].

However, the combined effects of zinc deficiency on renal and testicular tissues have not been sufficiently studied from a comprehensive morphological and morphometric perspective. Therefore, this study has significant scientific and practical relevance.[5]

Aim of the study. The aim of this study was to comprehensively investigate the morphological and morphometric changes in the kidneys and testes of 6-month-old rats under conditions of zinc deficiency, to determine the characteristics of oxidative-dystrophic processes, and to evaluate their functional significance.[6]

Materials and Methods

The study was conducted under experimental conditions using 6-month-old laboratory rats (Rattus norvegicus). The animals were divided into two groups: a control group consisting of healthy rats maintained on a standard diet, and an experimental group in which zinc deficiency was induced over a period of three months. Zinc deficiency was modeled by administering a diet with a significantly reduced zinc content. The duration of the experiment was 90 days.[7]

At the end of the experiment, the animals were euthanized in accordance with bioethical standards, and kidney and testicular tissues were collected for morphological examination. The specimens were fixed in 10% neutral formalin, processed using standard histological techniques, and embedded in paraffin. Sections of 5–7 μm thickness were prepared and stained with hematoxylin and eosin.[8]

Morphometric analysis was performed using an ocular micrometer and digital image analysis software. In the kidneys, the diameters of afferent and efferent arterioles, glomerular and renal corpuscle sizes, Bowman's space volume, tubular diameter, and epithelial height were measured. In the testes, the number of convoluted seminiferous tubules, their area and lumen size, the thickness of the spermatogenic epithelium, and the number of Sertoli cells, Leydig cells, and germinal elements were evaluated.[9]

Spermatogenesis was assessed using the Johnsen scoring system. Qualitative microscopic evaluation included epithelial dystrophy, interstitial edema, infiltration, and fibrotic changes. Statistical analysis was performed with results expressed as mean ± standard error (M ± m). Differences between groups were assessed using Student's t-test, with $p < 0.05$ considered statistically significant.

Results and Discussion

Under conditions of zinc deficiency, pronounced vascular–glomerular and tubular changes were observed in 6-month-old rats compared to the control group. The lumen of the afferent arteriole decreased to 15.9 ± 0.24 μm, indicating reduced blood flow into the glomerulus. The efferent arteriole lumen also decreased to 11.7 ± 0.18 μm, reflecting altered intraglomerular hemodynamics.[10]

The glomerular diameter increased to 117.4 ± 1.17 μm, while the renal corpuscle diameter reached 131.8 ± 1.90 μm. The volume of Bowman's space increased to 0.6 ± 0.02 $\times 10^6$ μm^3, indicating expansion of the filtration space. In addition, thickening of the basement membrane reflected structural remodeling of the filtration barrier.

In the tubular apparatus, the external diameter of the proximal convoluted tubules increased to 53.1 ± 0.46 μm, whereas no significant changes were observed in distal tubules (38.3 ± 0.53 μm). The most pronounced alterations were observed in the tubular epithelium: epithelial height decreased to 14.5 ± 0.31 μm in proximal tubules and to 8.5 ± 0.16 μm in distal tubules, indicating epithelial atrophy and reduced reabsorptive activity.[11]

The total number of glomeruli did not change significantly (30.2 ± 0.63 thousand vs. 30.5 ± 0.55 thousand in control); however, their density decreased both in the renal cortex (139.5 ± 1.91 cells/mm^3) and in the whole kidney (91.2 ± 1.39 cells/mm^3), reflecting a relative increase in stromal components. The cortical thickness remained unchanged (3.1 ± 0.03 mm), while the medullary thickness slightly decreased to 3.6 ± 0.03 mm.

Thus Figure 1. in 6-month-old rats, zinc deficiency was characterized by narrowing of the arteriolar lumen, glomerular hypertrophy, expansion of Bowman's space, thickening of the basement membrane, marked tubular epithelial atrophy, and reduced glomerular density. The most sensitive

structures were the proximal and distal tubules, where a 17–23% reduction in epithelial height indicated impaired nephron trophism and reabsorptive function.**[12]**

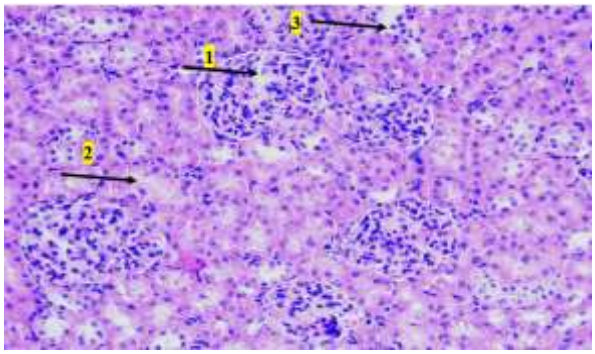

Figure 1. Microscopic image of the kidney of a 6-month-old rat under zinc deficiency conditions. Hematoxylin and eosin staining. Obj. 20×40. 1 — Moderate dilation of Bowman's space and thickening of the basement membrane; 2 — Dystrophic changes in the epithelium of proximal tubules (flattening, vacuolization, and rarefaction of the brush border); 3 — Interstitial changes: focal edema and early signs of fibrotic remodeling.

Morphometric analysis demonstrated that zinc deficiency induces pronounced remodeling in both parenchymal and interstitial components of the testes. The thickness of the tunica albuginea increased to 126.7 ± 3.53 μm in the zinc-deficient group, indicating intensified fibrotic processes.

The number of interstitial areas per microscopic field increased to 15.9 ± 0.39 (+10.7%), while the interstitial tissue area increased by 11.4% to 5.2 ± 0.12 ×10^5 μm². These changes reflect expansion of the stromal component and a relative reduction in the proportion of spermatogenic epithelium. The number of Leydig cells decreased from 27.70 ± 0.79 to 25.6 ± 0.67, indicating suppression of androgen-producing function.

The number of convoluted seminiferous tubules per field decreased to 9.4 ± 0.27, indirectly indicating an increase in their diameter. Although the cross-sectional area of the tubules increased to 6.6 ± 0.18 ×10^4 μm², this enlargement was mainly due to luminal expansion.[13]

The Figure 2. tubular lumen area increased by 34.3% to 11.2 ± 0.27 μm², which was accompanied by marked thinning of the spermatogenic epithelium. The thickness of the spermatogenic epithelium significantly decreased to 69.1 ± 1.69 μm, representing one of the most pronounced changes in zinc deficiency. The area of the spermatogenic epithelium remained relatively stable (4.6 ± 0.12), but its redistribution indicated disruption of the internal tubular architecture.

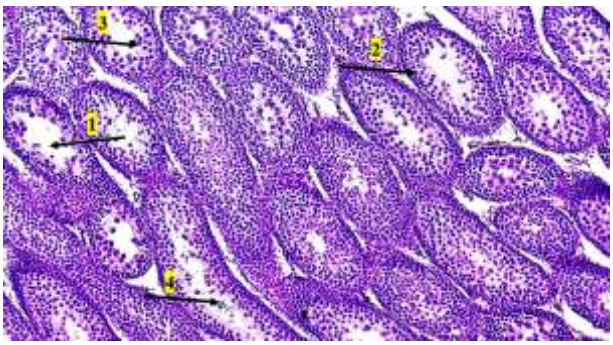

Figure 2. Microscopic image of the testis of a 6-month-old outbred white rat under zinc deficiency conditions. Hematoxylin and eosin staining. Obj. 20×4. 1 — Pronounced dilatation of convoluted seminiferous tubules accompanied by expansion of the tubular lumen; 2 — Thinning of the spermatogenic epithelium and reduction of cellular density; 3 — Decrease in the number of germinative elements and disruption of their orderly tratification; 4 — Marked reduction of mature spermatozoa within the tubular lumen.

The number of Sertoli cells showed a slight decrease (9.5 ± 0.21), indicating partial preservation of supporting function despite impaired spermatogenesis. The number of spermatogonia decreased (12.4 ± 0.34), accompanied by a reduction in their area, reflecting suppression of the proliferative compartment. Spermatocytes decreased by 14.6% (40.7 ± 0.84), indicating disruption of meiotic processes. Round spermatids decreased to 203.7 ± 3.21. The most pronounced reduction was observed in mature spermatozoa, which decreased to 295.5 ± 7.54, indicating a significant decline in the final efficiency of spermatogenesis.[14]

Table 1. Zinc deficiency was characterized by marked thinning of the spermatogenic epithelium, widening of the seminiferous tubule lumen, reduction in meiotic and post-meiotic cell populations, decreased number of Leydig cells, and increased interstitial components. These changes indicate progressive dystrophic processes and impaired spermatogenic function.[15]

Johnsen score for zinc deficiency:

ge	Experimental group	Morphological description of spermatogenesis	Score
6 months	Zn deficiency (3 months)	Spermatogenesis preserved up to the final stages; spermatozoa present in the lumen, however marked thinning of the spermatogenic epithelium, enlargement of the seminiferous tubule lumen, and reduction in the number of spermatocytes, round spermatids, and mature spermatozoa were observed	8.1–9.2 (8.5)

Conclusion

Thus, zinc deficiency induced structural remodeling in the testes, primarily characterized by impaired spermatogenesis at the meiotic and post-meiotic stages, thinning of the spermatogenic epithelium, and a relative increase in the stromal component. The obtained data confirm the essential role of zinc in maintaining the structural integrity and functional activity of the male reproductive system.

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