MORPHOLOGICAL CHARACTERISTICS AND MORPHOMETRY OF THE TESTES IN 6-MONTH-OLD RATS UNDER CONDITIONS OF SELENIUM DEFICIENCY

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Abstract: *This study investigated the morphological and morphometric changes in the testes of 6-month-old experimental rats maintained under conditions of selenium deficiency for three months. The results revealed oxidative-dystrophic alterations characterized by thinning of the spermatogenic epithelium, disorganization of cellular layers, and a decrease in the number of germ cells and mature spermatozoa. Enlargement of the lumens of convoluted seminiferous tubules and expansion of the interstitial tissue were observed, accompanied by a reduction in Leydig cell density. Evaluation using the Johnsen scoring system demonstrated impairment in the final stages of spermatogenesis. The findings confirm that selenium deficiency induces oxidative stress–mediated tubular-interstitial remodeling in the testes, leading to a decline in reproductive function.*

The aim of this study was to comprehensively investigate the morphological and morphometric changes in the testes of 6-month-old experimental rats subjected to selenium deficiency for three months, to determine the characteristics of oxidative-dystrophic processes in the spermatogenic epithelium and interstitial tissue, and to assess the degree of spermatogenic impairment.

Materials and Methods. The study was conducted under experimental conditions using 6-month-old laboratory rats. 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 selenium deficiency was induced over a period of three months. Selenium deficiency was modeled by administering a diet with a significantly reduced content of this trace element. The duration of the experiment was 90 days. At the end of the experiment, the animals were euthanized in accordance with bioethical standards, and testicular tissues were collected for morphological examination. The obtained 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.

Morphometric analysis was performed using an ocular micrometer and digital image analysis software. The number of convoluted seminiferous tubules, their cross-sectional area and lumen area, the thickness of the spermatogenic epithelium, and the number of Sertoli cells, spermatogonia, spermatocytes, spermatids, and spermatozoa were determined. In addition, the interstitial tissue area and Leydig cell density were evaluated.

The condition of spermatogenesis was assessed using the Johnsen scoring system. During microscopic evaluation, qualitative features such as epithelial disorganization, thinning of cell layers, interstitial edema, infiltration, and signs of fibrosis were analyzed.

Statistical analysis was performed using standard methods, with results expressed as mean ± standard error (M ± m). Differences between groups were assessed using Student's t-test, and a value of $p < 0.05$ was considered statistically significant.

Results. In 6-month-old rats maintained under selenium deficiency for three months, a complex of morphological changes developed in the testes, characterized by oxidative-dystrophic damage to the spermatogenic epithelium and early signs of interstitial remodeling.

Although the overall organ architecture was preserved, microscopic examination revealed impaired functional stability of spermatogenesis and deterioration of tissue trophism.

The tunica albuginea showed moderate thickening, reaching 134.1 ± 3.17 μm, reflecting early connective tissue remodeling. Fibrous structures appeared more densely arranged and irregularly oriented in some areas, although no pronounced sclerosis was observed.

The number of convoluted seminiferous tubules per field of view (×100) decreased to 9.5 ± 0.28, which was associated with dilation of their diameter and lumen. The cross-sectional area of the tubules increased to 6.6 ± 0.16 $\times 10^4$ μm², and the lumen area to 9.3 ± 0.29 $\times 10^3$ μm², exceeding control values by 10–15%, indicating tubular dilation against the background of epithelial weakening.

The thickness of the spermatogenic epithelium significantly decreased to 70.2 ± 2.09 μm, with areas of thinning, disorganization of cellular layers, and reduced density of germinal elements.

The cellular composition of the spermatogenic epithelium demonstrated pronounced quantitative changes. The number of Sertoli cells per tubular cross-section remained relatively stable at 9.5 ± 0.23; however, spermatogonia decreased to 11.5 ± 0.39, spermatocytes to 41.7 ± 1.08, and round spermatids to 168.7 ± 4.28.

The most pronounced changes were observed in mature forms: the number of spermatozoa in the tubular lumen decreased to 320.1 ± 7.25, representing a 25–30% reduction compared to control, confirming suppression of the final stages of spermatogenesis.

The interstitial tissue showed a tendency toward expansion, with its area increasing to 5.3 ± 0.19 $\times 10^5$ μm². Foci of edema, moderate perivascular infiltration, and initial fibrotic changes were observed. The number of Leydig cells decreased to 25.5 ± 0.49, with cytoplasmic pallor and focal vacuolization, indicating reduced steroidogenic activity.

Thus, selenium deficiency in 6-month-old rats resulted in a complex of morphological and morphometric changes, including dilation of seminiferous tubules, thinning and disorganization of the spermatogenic epithelium, a decrease in spermatogonia, spermatocytes, and spermatids, reduced numbers of mature spermatozoa, expansion of interstitial tissue, and a decrease in Leydig cell density.

A decrease in the Johnsen index to 9.0 after a 6-month exposure primarily reflected progressive impairment of spermatogenesis at the level of late differentiation stages.

Preservation of Sertoli cells and the absence of total tubular destruction suggest that the observed alterations are predominantly functional and associated with oxidative stress and reduced activity of selenium-dependent antioxidant systems.

In conclusion, selenium deficiency does not lead to severe necrotic damage but induces stable oxidative stress–mediated remodeling characterized by tubular weakness, progressive epithelial thinning, decreased reproductive potential, and the formation of a morphological basis for chronic testicular dysfunction.