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MORPHOLOGICAL PARAMETERS OF RAT KIDNEYS WITH ALIMENTARY MAGNESIUM DEFICIENCY

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Abstract. The study investigates morphological and morphometric changes in the kidneys of rats under conditions of dietary magnesium deficiency. The experiment was performed on male outbred white rats aged 3–6 months using standard and magnesium-deficient diets. Histological and morphometric methods with subsequent statistical analysis were applied. It was established that magnesium deficiency does not cause gross distortion of renal architecture but induces a complex of moderately expressed structural alterations, including vasoconstriction of afferent and efferent arterioles, reduced glomerular density, glomerular hypertrophy, and pronounced dystrophic changes of the tubular apparatus, predominantly in proximal tubules. A shift in the corticomedullary ratio toward the medulla was observed, reflecting chronic functional overload of nephrons and adaptive structural remodeling of renal tissue. The obtained results expand current understanding of the pathogenesis of magnesium-deficiency-associated nephropathies.

Keywords: magnesium deficiency, kidneys, morphology, morphometry, rats, nephron, tubular dystrophy, glomerular hypertrophy.

Relevance. Magnesium is the fourth most abundant macronutrient in the body and the second most abundant intracellular divalent cation. This mineral serves as a cofactor in over 300 biochemical reactions, including protein synthesis, energy production and storage, cell division, DNA and RNA synthesis, and the stabilization of mitochondrial membranes. Magnesium is involved in nerve impulse conduction, cardiac excitability, neuromuscular transmission, the regulation of vascular tone and blood pressure, and glucose and insulin metabolism. Due to this diversity of functions, magnesium is essential for the prevention of many diseases and the maintenance of overall health. Magnesium deficiency is associated with an increased risk of migraines, Alzheimer's disease, vascular accidents, arterial hypertension, cardiovascular disease, and type 2 diabetes. Foods richest in magnesium include whole grains, green leafy vegetables (especially spinach), nuts, legumes, and potatoes. This element is essential for every cell in the body, as it participates in enzymatic reactions related to energy metabolism, protein and DNA synthesis, and regulates the function of many ion channels. Magnesium balance is maintained through a combination of intestinal absorption, metabolism by bone tissue, and renal excretion. Despite magnesium's widespread use in foods, research shows that a significant portion of the population receives insufficient amounts. Deficiencies can be caused by genetic factors, inadequate intake, gastrointestinal diseases, renal losses, endocrine disorders, and the effects of certain medications [1-6].

Materials and Methods. The animals used in the experimental study were male albino rats weighing from 220 ± 20 grams (at the beginning of the experiment) to 300 ± 20 grams (at the end of the experiment); aged 12-24 weeks (3 to 6 months), n = 45.

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For the experiment, a special diet for laboratory animals developed by ALTROMIN Spezialfutter GmbH & Co., Germany, was used. The diet holds official certificate No. 36/2024. Each experimental group, including the standard diet, received 10 mm pellets in 5 kg buckets.

Types of diets used in the experiment

№	Name	Purpose
1	C1000	Standard diet
2	C1035	Magnesium-deficient diet

Following macroscopic assessment of the removed organ, histological kidney preparations were prepared. Deparaffinized sections were stained with hematoxylin and eosin (H&E). After staining, the sections were dehydrated in ethanol series, cleared in xylene, and coverslipped.

The following kidney parameters were studied: - Afferent and efferent arteriole diameters (μm); - Glomerular diameter (μm); - Total glomeruli number (in the cortex, in the entire kidney per 1 mm^3); - Proximal and distal convoluted tubule diameter; - Renal corpuscle diameter; - Capsular cavity volume in the renal corpuscles; - Capsule thickness; - Epithelial cell height; - Cortical width; - Medulla width.

The study materials were statistically processed using parametric and nonparametric analysis. The accumulation, adjustment, systematization of initial information and visualization of the obtained results were carried out in Microsoft Office Excel 2010 spreadsheets. Statistical analysis was carried out using the IBM SPSS Statistics v.23 program (developer - IBM Corporation).

Study results. In 6-month-old magnesium-deficient rats, the kidneys retain a typical lobar organization and the usual division into cortex and medulla. However, detailed analysis reveals a range of moderate structural deviations from the age-appropriate norm. Visually, a somewhat denser and thicker medullary zone is noted with a relatively stable cortex volume, which, at the macroscopic level, reflects a shift in stromal-parenchymal relationships in favor of the medullary component.

Under light microscopy, the cortex exhibits decreased glomerular density and areas of moderate tubular lumen dilation with epithelial thinning, while in the medullary zone, congested vessels and dilated lumens of the straight tubules are more clearly visible. These features indicate that magnesium deficiency does not lead to gross deformation of the organ architecture, but causes systemic microstructural changes in the nephrons and vascular bed. In the magnesium-deficient group, the lumen diameter of the afferent arteriole was, on average, approximately 10% smaller than in intact 6-month-old rats (decreasing from ≈17.2 to ≈15.4 μm). Similarly, the diameter of the efferent arteriole decreased by approximately 9% (from ≈12.4 to ≈11.3 μm). No significant overdevelopment or, conversely, collapse of the narrow segment of the efferent arteriole was observed, but its lumen was also somewhat smaller than control values.

This combined narrowing of the afferent and efferent branches indicates increased vascular tone and a tendency toward decreased intraglomerular blood flow. At the functional level, this can be considered a manifestation of magnesium-deficiency-induced vascular dysregulation, as magnesium is an important modifier of vascular reactivity and ion homeostasis. Despite the decreased arteriolar diameters, magnesium-deficient rats showed an increase in the size of the glomerular tuft: the glomerular diameter increased by an average of ≈5–6%, and the renal corpuscle diameter by ≈4% compared to age-appropriate norms. This indicates a tendency toward glomerular hypertrophy.

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The capsular space, however, does not change significantly in width; that is, no significant expansion of the Bowman's capsule (as a sign of severe hypofiltration) or narrowing (as with active inflammation) is observed. Against the background of narrowed arterioles and enlarged glomeruli, a pattern characteristic of chronic functional stress develops: some glomeruli operate in a mode of relative hyperfiltration, compensating for the functional losses of other nephrons.

Concomitant with the hypertrophy of individual glomeruli, a decrease in cortical and total glomerular density is observed. • Cortical glomerular density in magnesium-deficient animals was approximately 13% lower than in controls.

• Average "whole kidney" glomerular density also decreased by approximately 12–13%.

The total number of glomeruli per kidney (≈30,000–31,000) did not differ significantly from controls, on average. However, their distribution and density per unit volume of cortex changed. This can be interpreted as an early stage of structural reorganization, likely due to localized reduction of some nephrons and compensatory hypertrophy of the remaining glomeruli.

The most pronounced changes in magnesium deficiency were found in the proximal nephrons.

• The outer diameter of the proximal convoluted tubules increased by approximately 4.5% compared to controls.

• The diameter of their lumen increased even more significantly—by approximately 8%. • At the same time, the height of the epithelium of the proximal tubules decreased by almost 19%.

This triad (dilation of the lumen, moderate increase in the outer diameter, and marked thinning of the epithelium) clearly indicates the development of dystrophic-atrophic changes in the proximal segment: the tubules become "wider and thinner," and the epithelial cells lose volume and functional saturation. Functionally, this corresponds to a decrease in the reabsorption capacity of the nephrons and early tubulopathy.

In the distal sections, the changes are less pronounced but follow a similar direction.

• The outer diameter of the distal convoluted tubules is slightly increased (by approximately 3.5%),

• the lumen is slightly dilated (≈+1–1.5%),

• the epithelial height of the distal segment is reduced by approximately 18%. These data indicate a more moderate, but still noticeable, degeneration of the distal epithelium, which may affect the fine regulation of urine electrolyte composition and the involvement of the distal segment in tubuloglomerular feedback mechanisms.

The thickness of the cortex in 6-month-old magnesium-deficient rats was slightly less than control values (by approximately 2%). Although the absolute difference is small, combined with the decrease in glomerular density, this suggests a partial loss of parenchyma and a redistribution of volume in favor of the stroma and medullary structures.

The medulla, in contrast, demonstrated an increase in thickness of approximately 5–6%. This may reflect both a relative redistribution of volume (due to a decrease in the cortex) and possible changes in the system of straight tubules and vessels (plethora, mild edema, restructuring of the countercurrent system). Thus, the corticomedullary ratio is shifted in magnesium deficiency, which is a morphological reflection of chronic nephron overload and adaptive changes in the urine concentrating system.

The combination of these changes allows us to characterize the kidneys of 6-month-old magnesium-deficient rats as an organ in a state of chronic functional stress and structural reorganization:

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• The vascular component responds to magnesium deficiency with moderate generalized vasoconstriction of afferent and efferent arterioles.

• The glomeruli exhibit a combination of moderate hypertrophy with decreased density in the cortex, indicating a redistribution of the load between nephrons and possible loss of some glomeruli.

• The tubular apparatus, particularly the proximal tubules, is in a state of severe degeneration: dilation of the lumens with thinning of the epithelium indicates a decrease in reabsorption capacity and the formation of a tubulointerstitial component of the lesion (Fig. 3).

• The cortical-medullar ratio shifts in favor of the medulla, which may reflect both adaptive changes in hydrodynamics and early manifestations of structural reorganization of the renal stroma.

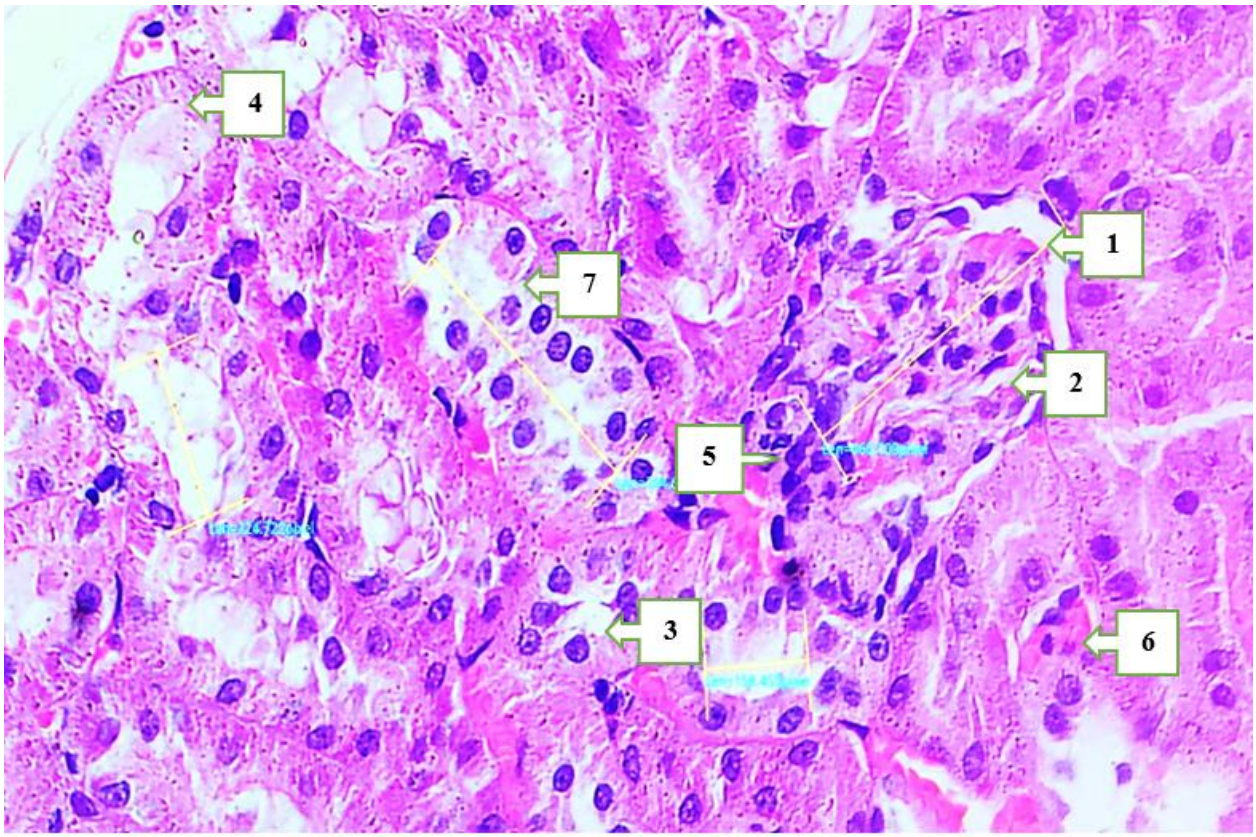

Fig. 3. Microscopic image of the kidney of 6-month-old rat with magnesium deficiency. G-E staining. Ok. 20×40. 1-Renal corpuscle: moderate segmental dilation of the capsular space, swelling and vacuolation of podocytes in places; 2-Glomerular capsule: thickened in places, looser due to impaired collagenogenesis and edematous; 3-Distal tubules: light cytoplasm, signs of osmotic swelling, slightly hyperchromatic nuclei; 4-Proximal tubules: pronounced granular cytoplasm in places - vacuolation and foci of epithelial desquamation; 5-Dense spot: cells are flattened, their compactness is impaired; 6-Peritubular capillaries: moderate plethora, edematous endothelium; 7-Collecting ducts: epithelium with signs of initial vacuolization in the cytoplasm.

Conclusion. Magnesium deficiency in 6-month-old rats is accompanied by a complex of structural and functional disturbances in the kidneys. The most pronounced changes include

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moderate vasoconstriction of the afferent and efferent arterioles, glomerular hypertrophy with decreased cortical and total glomerular density, and profound degenerative processes in the tubular component of the nephrons—primarily in the proximal tubules, where luminal dilation is observed with a significant reduction in epithelial height.

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