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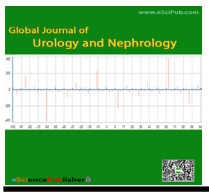

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NON-DIABETIC GLUCOSURIA AS ONE OF THE POSSIBLE FACTORS OF FORMATION OF CHRONIC KIDNEY DISEASE

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Relevance. The reason for this study was the discovery of patients with clinical and laboratory manifestations of chronic kidney disease (CKD) in the lumen of the tubules of red blood cells and their decay products during nephrobiopsy[1]. It is known that the appearance of glucose in urine makes an ideal nutrient base for microorganisms that provoke chronic microbial inflammatory damage to the kidneys. Glycated hemoglobin is included in 5% of red blood cells in healthy adults. Its increase is associated with episodes of glycemia exceeding 6.5 mmol/l. which occuts in diabetes decompensation. Moreover, glycated hemoglobin has toxic properties with respect to the viability of the surrounding cytomembranes. This explains the large number of multiple organ diabetic complications [2,3]. At the same time, it was found that the biochemical process of glycation of not only hemoglobin, but also other cellular glucoproteins is accompanied by oxidative damage to cytomembranes leading to their structural and functional failure [4,5]. However, the role of glycated hemoglobin and glucose released during the deteriation of erythrocytes in the renal tubules, as one of the possible pathogenetic factors in the occurrence of chronic kidney disease, no one has previously analyzed.

Goal. To conduct a comparative study of the results of nephrobiopsy and biochemical parameters of carbohydrate metabolism in 120 patients with the first clinically established diagnosis of chronic kidney disease.

Keywords:
non-diabetic glucosuria, chronic kidney disease.

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Materials and methods. Separately, the results of nephrobiopsy and biochemical studies of carbohydrate metabolism in 120 patients with newly diagnosed (CKD) were analyzed. The studies were carried out according to the standard method with generally accepted statistical processing of the obtained scientific data.

Results. Studies have shown that chronic damage to the renal parenchyma is independent of the nosological form, with normal blood sugar and glycated hemoglobin, in 26.7% it is accompanied by the appearance of degraded red blood cells in the urinary tract cavities in combination with glucosuria (Figure 1,2,3)

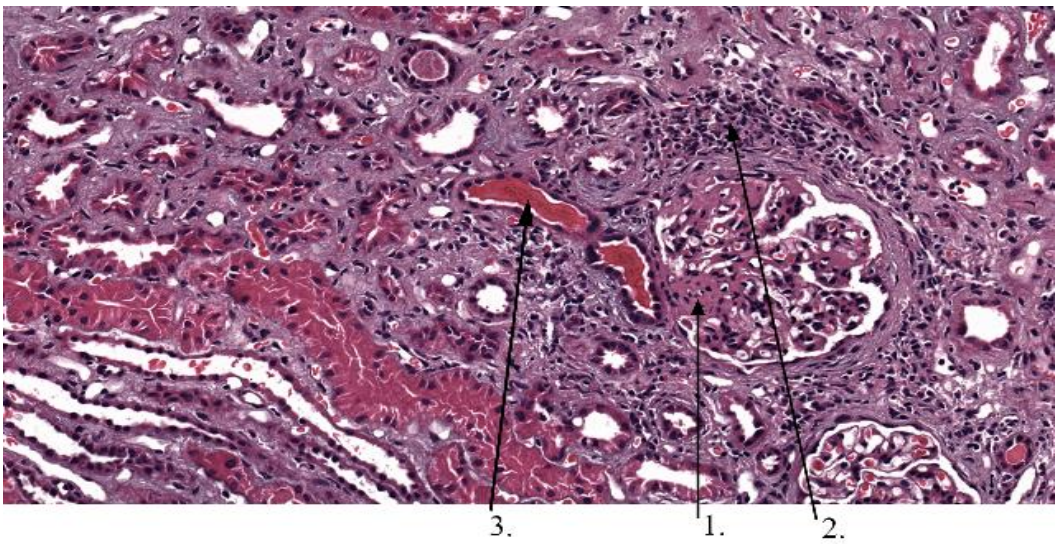

Figure 1. Chronic pyelonephritis. 1 - glomerulus. 2 - lymphocytic infiltration in the cortical layer. 3 - red blood cells and their decay products in the lumen of the distal tubules. [X25 magnification]

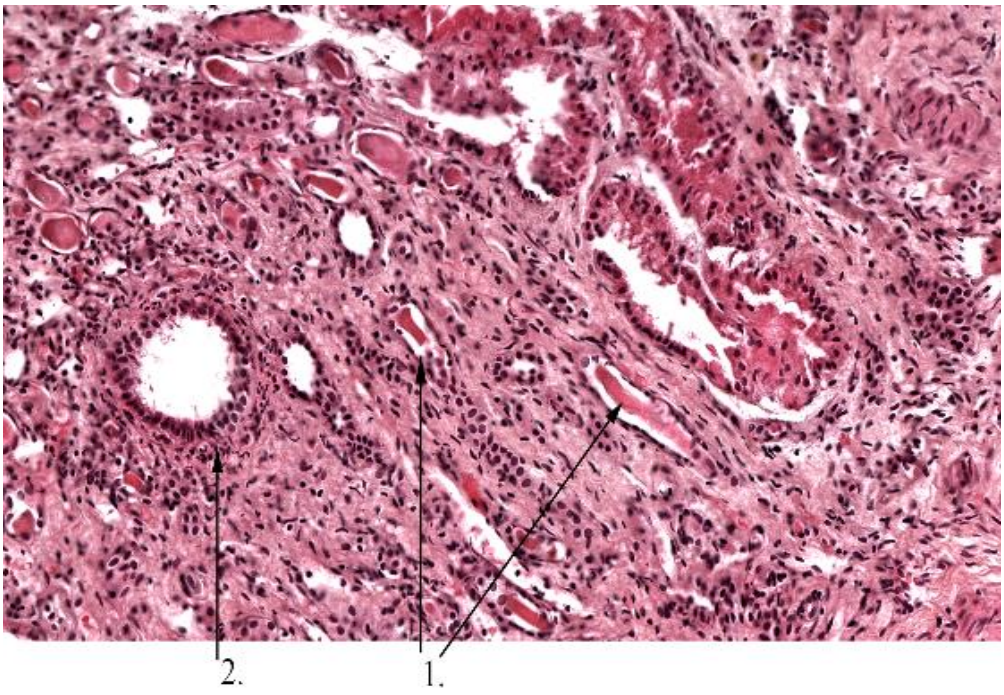

Figure 2. Chronic glomerulonephritis. 1 - sclerosed club-check. 2 - lymphocytic-histiocytic infiltration in the cortical layer. 3 - red blood cells and their decay products in the lumen of the distal tubules. [X25 magnification]

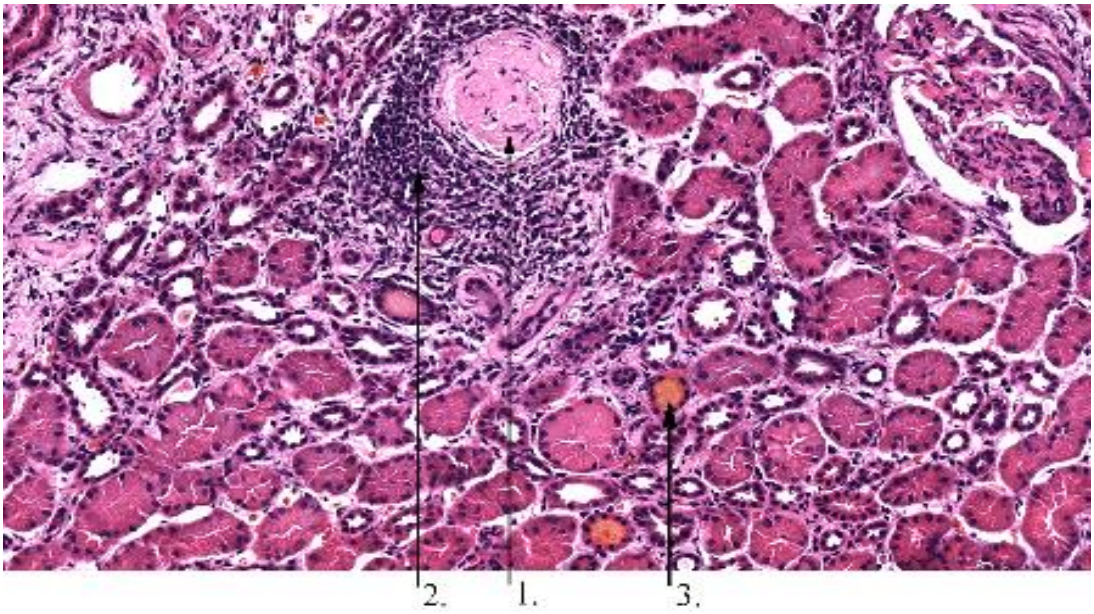

Figure 3. Chronic interstitial nephritis. 1 - erythrocytes and products of their decay in the lumen of the distal tubules. 2 - lymphocytic-plasmacytic infiltration in the cortical layer. [X25 magnification]

The relief of clinical and laboratory manifestations of chronic kidney disease on the background of etiotropic therapy coincided with a significant decrease in the manifestations of glucosuria (Table 1.)

Table 1. The effect of etiotropic therapy of chronic kidney disease on indicators of carbohydrate metabolism in patients with CKD (M± m)

Indicator	Before treatment (n-32)	After treatment (n-32)
Glycemia (mmol \L)	4,8± 0,5	4,5±0,2
Glycated HB (%)	5,2± 0,4	5,2±0,2
Glucosuria (mmol \ L)	0,21± 0,02*	0,09±0,02*

Note: p <0.05 * differences are statistically significant (t-Student confidence criterion)

Conclusion Morphofunctional manifestations of chronic kidney disease in every fourth patient without a diabetic history are accompanied by of degraded red bloodcells and glucosuria in the tubules. Complex etiotropic therapy with the relief of clinical and laboratory manifestations of the disease has proven to lead to a significant decrease in the level of glucosuria. The appearance of degraded erythrocytes in the tubules as a source of non-diabetic glucosuria and contact of glycated hemoglobin with epithelial cells may be one of the factors causing CKD

Authors declare no conflict of interest

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