DIAGNOSTIC VALUE OF SOME URINARY BIOMARKERS IN ESTIMATION OF TUBULOINTERSTITIAL INJURY IN PATIENTS WITH PRIMARY GLOMERULOPATHIES

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Objectives:

In recent years many studies were published dedicated to biomarkers of kidney injury in estimating course of different variants of renal disease. Markers of kidney damage can reflect pathological changes of different compartments of nephron. In this study our aim was to compare the level of excretion of urinary biomarkers of tubulointerstitial damage (such as neutrophil gelatinase-associated lipocalin (NGAL), cystatin C, alfa1-microglobulin, beta2-microglobulin) with clinical signs and morphological changes (glomeruloclerosis, tubulointerstitial sclerosis, tubular atrophy) in patients with different forms of primary glomerulonephritis.

Methods:

78 patients with byopsy proven primary glomerulonephritis were included in the study. Patients with acute kidney injury, infectious diseases, heart failure, respiratory insufficiency and cancer were excluded. According to the results of light and electron microscopy 28 (35,9%) patients had IgA-nephropathy (mesangial proliferative glomerulonephritis), 19 (24,4%) - focal segmental glomerulosclerosis, 22 (28,2%) - membranous nephropathy, 9 (11,5%) - minimal change disease. Besides standart laboratory and instrumental investigations samples of serum and daily urine were obtained in the day of byopsy. NGAL level was studied using ELISA-method, cystatin C, alfa1-microglobulin and beta2-microglobulin using turbidometry. Glomerulosclerosis, tubulointerstitial sclerosis and tubular atrophy were estimated quantitatively and semi quantitatively.

Results:

Correlation of daily proteinuria was positive with excretion of biomarkers ($r=0,43$, $r=0,44$, $r=44$, $r=0,50$ for NGAL, cystatin C, alfa1-microglobulin, beta2-microglobulin respectively, $p<0,05$). Alfa1-microglobulin and NGAL urinary excretion levels were higher if tubular atrophy existed in patients with daily proteinuria less then 3,5 g ($p<0,05$). Level of cystatin C and alfa1-microglobulin urinary excretion were significantly higher in patients with high proteinuria if focal tubulointerstitial fibrosis degree was more than 20% ($p<0,05$). Urinary NGAL level correlated with severity of tubular atrophy in patients with nephrotic range proteinuria ($r=0,48$, $p<0,05$).

Conclusions:

Urinary excretion of all studied biomarkers depended on level of proteinuria that can explain low correlation between their usage and sclerotic changes. This fact should limit their exploitation in acute kidney injury diagnostics in patients with primary glomerulopathies and high range of proteinuria. Alfa1-microglobulin excretion reflects the presence of tubular atrophy in patients with non-nephrotic range proteinuria while in its high range alfa1-microglobulin and cystatin C in urine represent the existence of severe tubulointerstitial sclerosis. Urinary NGAL reflects tubular atrophy in high range proteinuria group of patients.

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