THE ROLE OF NGAL AND CYSTATIN-C ESTIMATION IN PATIENTS WITH PRIMARY GLOMERULOPATHIES

Ian Proletov[1], Elena Saganova[1], Olga Galkina[1], Evdokiia Bogdanova[1], Irina Zubina[1], Vasilii Sipovskii[1] and Alexei Smirnov[1].

[1]Saint-Petersburg Pavlov State Medical University, Saint-Petersburg, Russian Federation

OBJECTIVES

NGAL and cystatin C are well known as biomarkers of acute kidney injury. Some studies during last years showed that using of this markers can reflect the course of chronic kidney disease. The aim of our investigation was assessment of relationship between level of biomarkers and clinical and pathomorphological signs of glomerular diseases.

METHODS

In cross-section study 71 patients with byopsy proven primary glomerulonephritis were included. Patients with acute kidney injury, infectious diseases, heart failure, respiratory insuffience and cancer pathology were excluded. According the results of light and electron microscopy 23 (32,4%) patients had IgA-nephropathy (mesangial proliferative glomerulonephritis), 14 (19,7%) – focal segmental glomerulosclerosis, 22 (31,0%) – membranous nephropathy, 12 (16,9%) – 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 using turbidometry. GFR was estimated using creatinine clearence rate, CKD-EPI equation and cystatin C formula by Hoek (–4.32 + 80.35 × 1/CysC in mg/L). Glomerulosclerosis, tubulointerstitial sclerosis and tubular atrophy were estimated quantitatively and semi quantitatively.

RESULTS

GFR estimated using cystatin-C most accurately reflects the degree of glomerular sclerosis than GFR CKD-EPI and creatinine clearance rate (r=0,63, r=0,49, r=0,036 respectively p<0,05). Serum NGAL correlated with GFR estimated using cystatin-C (r=0,037, p<0,05) while there was no correlation with CCr and GFR CKD-EPI. Urine cystatin C did not correlate with such morphological signs of chronic kidney injury as glomerulosclerosis, tubulointerstitial sclerosis and tubular atrophy while urine NGAL correlated with tubular atrophy as well. The rates of urine NGAL and cystatin-C excretion were the highest in patients with high range proteinuria (figure 1)

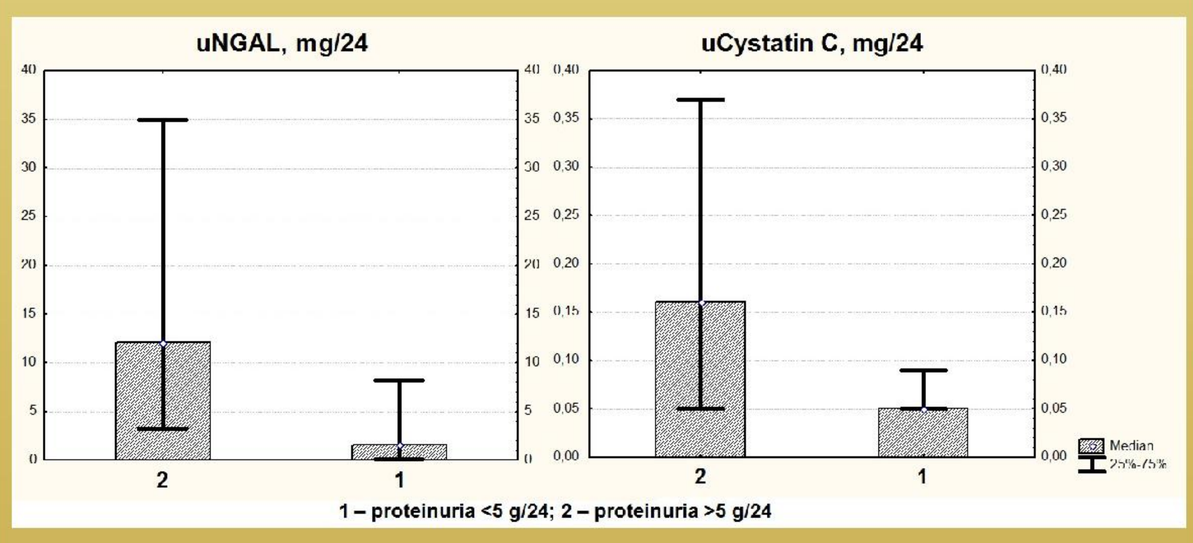

Figure 1.: Urine NGAL and Cystatin C excretion in patients with high and low ranges of proteinuria

CONCLUSIONS

The applying of GFR estimated using cystatin-C is preferable in assessment of glomerulosclerosis degree. Urine NGAL excretion most accurately shows the severity of tubular atrophy while serum NGAL reflects the earliest stages of glomerular cell injury. Low correlation between urine excretion of NGAL and cystatin-C and sclerotic changes can be explained by depending of urinary excretion of this biomarkers on proteinuria and this fact should limit their exploitation in acute kidney injury diagnostics in patients with primary glomerulopathies and high range of proteinuria.

50ERA-EDTA
335--MP
Clinical nephrology - miscellaneous
Ian Proletov
DOI: 10.3252/pso.eu.50era.2013
Poster presented at: ERA-EDTA 50th Congress Istanbul, Turkey May 18-21, 2013
PosterSessionOnline.com