6. KIDNEY DAMAGE IN GOUT PATIENTS

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Introduction. In patients who have suffered from gouty arthritis for many years, renal cone disease is very common. After arthritis, the most common clinical manifestation of hyperuricemia is renal interest.

Aim of study. Study the clinical and paraclinical aspects of renal lesions in gout for early diagnosis of these lesions, followed by initiation of early treatment to prevent the development of comorbid diseases.

Methods and materials. It has been used the information resources of the Medical Scientific Library of the *Nicolae Testemitanu* State University of Medicine and Pharmacy, as well as the publications from the specialized journals in PubMed, Medline, MedScape, Google Scholar, Wikipedia.

Results. The only marker currently recognized as highly specific and sensitive for ischemic injury in the proximal renal tubules is KIM-1. This is a transmembrane protein with immunoglobulin and mucin domains, first described in 1998. In intact renal tissue it is found in small amounts, but after ischemia its level in regenerating proximal tubules is significantly increased. As experimental studies have shown, increased KIM-1 levels are associated with ischemic effect on the kidney and are not always accompanied by an increase in blood urea nitrogen and creatinine, indicating a high diagnostic value of KIM-1 as an early diagnostic marker of proximal tubule damage and has therefore been studied in recent years as a marker of renal damage in CKD of various etiologies. Studies in patients with non-diabetic proteinuric nephropathy have been performed and have shown that KIM-1 is significantly increased in patients with proteinuria compared to controls and has a direct correlation with urine protein levels - it decreases in response to ACE inhibitor treatment, but even when the target urine protein level (1 g/l) is reached, KIM-1 does not reach normal values, confirming continued deterioration of the renal tubular apparatus and, if increased against a background of decreased left ventricular function, is considered a predictor of all-cause mortality and repeated hospitalizations for heart failure. Based on the findings of Jungbauer CG et al, it has been suggested that tubular lesions in CKD may be present in patients with normal renal function. Despite the high sensitivity, specificity and prognostic value of KIM-1 in the diagnosis of tubular renal lesions in CKD of various etiologies, this protein has not been studied in patients with gout.

Conclusion. The assessment of early kidney damage in patients with gout will improve the prognosis of kidney damage, at the same time we will contribute to decrease sudden complications and disability of patients, so they will remain fit for work.