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ROLE OF KIM-1, CYSTATIN C, AND MICROALBUMINURIA AS EARLY MARKERS OF GOUTY NEPHROPATHY IN PATIENTS WITH GOUT

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Abstract: Early detection of renal injury in patients with gout remains an important clinical problem, since conventional indicators do not always reflect subclinical kidney damage. Biomarkers such as kidney injury molecule-1 (KIM-1), cystatin C, and microalbuminuria (MAU), together with glomerular filtration rate (GFR), may improve early diagnosis of gouty nephropathy.

To assess the diagnostic significance of KIM-1, cystatin C, microalbuminuria, and glomerular filtration rate in the early detection of gouty nephropathy in patients with gout.

A total of 186 individuals were included in the study. The control group consisted of 96 healthy subjects. The patient cohort comprised 90 patients with gout and was divided into two groups: Group 1 included 45 patients with gout without gouty nephropathy, and Group 2 included 45 patients with gouty nephropathy. Serum creatinine, cystatin C, urinary KIM-1, microalbuminuria, and GFR were assessed. GFR was estimated using creatinine-based, cystatin C-based, and combined creatinine/cystatin C approaches. Statistical significance was assessed relative to the control group and between the two patient groups.

KIM-1, cystatin C, and MAU levels progressively increased from the control group to Group 1 and were highest in Group 2. KIM-1 increased from 0.3 in controls to 1.0 in Group 1 and 2.6 in Group 2. Cystatin C increased from 0.6 to 0.95 and 1.35, respectively. MAU increased from 15 in controls to 28 in Group 1 and 130 in Group 2. Serum creatinine also rose progressively (70, 85, and 119, respectively). In contrast, GFR declined across the groups. Creatinine-based GFR decreased from 114 in controls to 96 in Group 1 and 58 in Group 2. Cystatin C-based GFR declined from 112 to 93 and 52, while combined creatinine/cystatin C GFR decreased from 112 to 93 and 56, respectively. The most pronounced abnormalities were observed in patients with gouty nephropathy.

KIM-1, cystatin C, and microalbuminuria are informative early markers of renal injury in patients with gout. Their increase, together with a progressive decline in GFR, reflects the development and progression of gouty nephropathy. Combined assessment of these parameters may improve early diagnosis and risk stratification in patients with gout.

Keywords: gout, gouty nephropathy, KIM-1, cystatin C, microalbuminuria, glomerular filtration rate, early diagnosis, renal injury

Introduction.

Gout is a chronic metabolic disorder characterized by hyperuricemia and urate crystal deposition in tissues. In addition to articular involvement, renal damage is one of the most important extra-articular manifestations of the disease. Gouty nephropathy develops gradually and may remain clinically silent for a long period, which complicates timely diagnosis and delays preventive and therapeutic interventions.

Traditional laboratory parameters such as serum creatinine are widely used to assess renal function, but they often rise only after substantial nephron loss has already occurred. Therefore, there is a clear need for earlier and more sensitive biomarkers capable of detecting renal injury at preclinical or minimally symptomatic stages.

Kidney injury molecule-1 (KIM-1) is considered a sensitive marker of tubular epithelial injury. Its elevation reflects early tubular damage and may precede conventional signs of renal dysfunction. Cystatin C is a low-molecular-weight protein that is freely filtered by the glomeruli and is regarded as a more sensitive indicator of changes in glomerular filtration than serum creatinine. Microalbuminuria reflects early glomerular permeability changes and endothelial dysfunction and is an important marker of subclinical renal damage.

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Assessment of these biomarkers together with glomerular filtration rate may provide a more comprehensive view of early renal injury in patients with gout. Such an approach may be especially useful for identifying patients at increased risk of developing gouty nephropathy.

The aim of this study was to evaluate the role of KIM-1, cystatin C, microalbuminuria, and glomerular filtration rate in the early detection of gouty nephropathy in patients with gout.

Methods.

Study design and participants. This study included 186 individuals. The control group comprised 96 apparently healthy subjects. The main study population included 90 patients with gout, who were divided into two groups:

Group 1: 45 patients with gout without gouty nephropathy

Group 2: 45 patients with gouty nephropathy

The grouping was performed according to the presence or absence of renal involvement.

Laboratory and functional assessment. The following parameters were assessed in all participants:

- urinary KIM-1
- serum cystatin C
- microalbuminuria
- serum creatinine
- glomerular filtration rate estimated by creatinine
- glomerular filtration rate estimated by cystatin C
- combined glomerular filtration rate based on creatinine and cystatin C

Statistical analysis. Comparisons were made between the control group and each clinical group, as well as between Group 1 and Group 2. In the figures, * indicates statistical significance compared with the control group, whereas ^ indicates statistical significance of Group 2 compared with Group 1.

Results

KIM-1, cystatin C, and microalbuminuria. Comparative analysis showed a gradual increase in early renal injury markers from the control group to patients with gout without nephropathy and then to patients with gouty nephropathy.

KIM-1 level was 0.3 in the control group, increased to 1.0 in Group 1, and reached 2.6 in Group 2. This indicates progressive tubular injury, with the highest value observed in patients with established gouty nephropathy.

Cystatin C demonstrated a similar pattern. Its level increased from 0.6 in the control group to 0.95 in Group 1 and further to 1.35 in Group 2, suggesting gradual deterioration of renal filtration function.

Microalbuminuria showed the most pronounced increase among the studied markers. Its value was 15 in the control group, 28 in Group 1, and 130 in Group 2, reflecting substantial worsening of glomerular permeability and renal damage in patients with nephropathy.

These findings indicate that KIM-1, cystatin C, and MAU are sensitive indicators of early renal injury in gout and are markedly elevated in the presence of gouty nephropathy.

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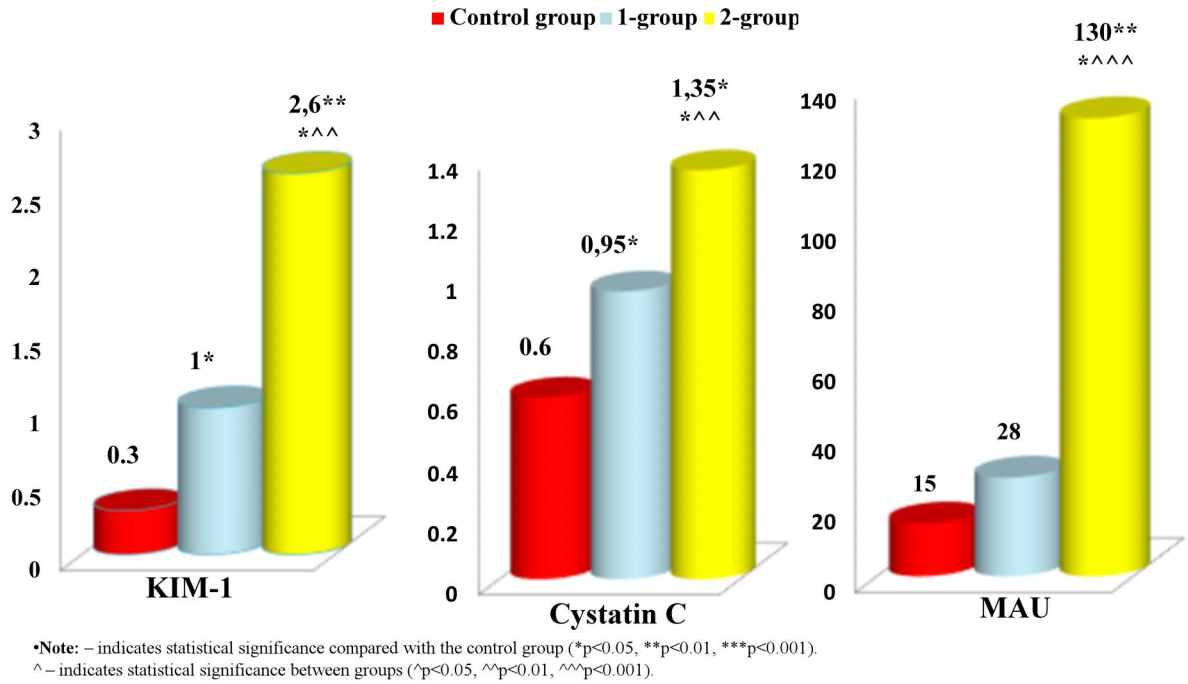

Figure 1. KIM-1, cystatin C, and microalbuminuria levels in the control group, patients with gout without nephropathy (Group 1), and patients with gouty nephropathy (Group 2).. *Note:* * — compared with the control group; ^ — Group 2 compared with Group 1.

Serum creatinine and glomerular filtration rate. Serum creatinine increased progressively across the studied groups. Its level was 70 in the control group, 85 in Group 1, and 119 in Group 2. This confirms worsening renal function in gout patients, particularly in those with nephropathy.

At the same time, glomerular filtration rate progressively declined. Creatinine-based GFR was 114 in the control group, 96 in Group 1, and 58 in Group 2. Cystatin C-based GFR showed a similar decrease from 112 in controls to 93 in Group 1 and 52 in Group 2. The combined creatinine/cystatin C GFR also decreased, from 112 in the control group to 93 in Group 1 and 56 in Group 2.

These results indicate that renal filtration impairment becomes more pronounced with progression from uncomplicated gout to gouty nephropathy. Importantly, cystatin C and KIM-1 abnormalities were already evident in Group 1, suggesting that these markers may detect renal impairment earlier than conventional functional decline alone.

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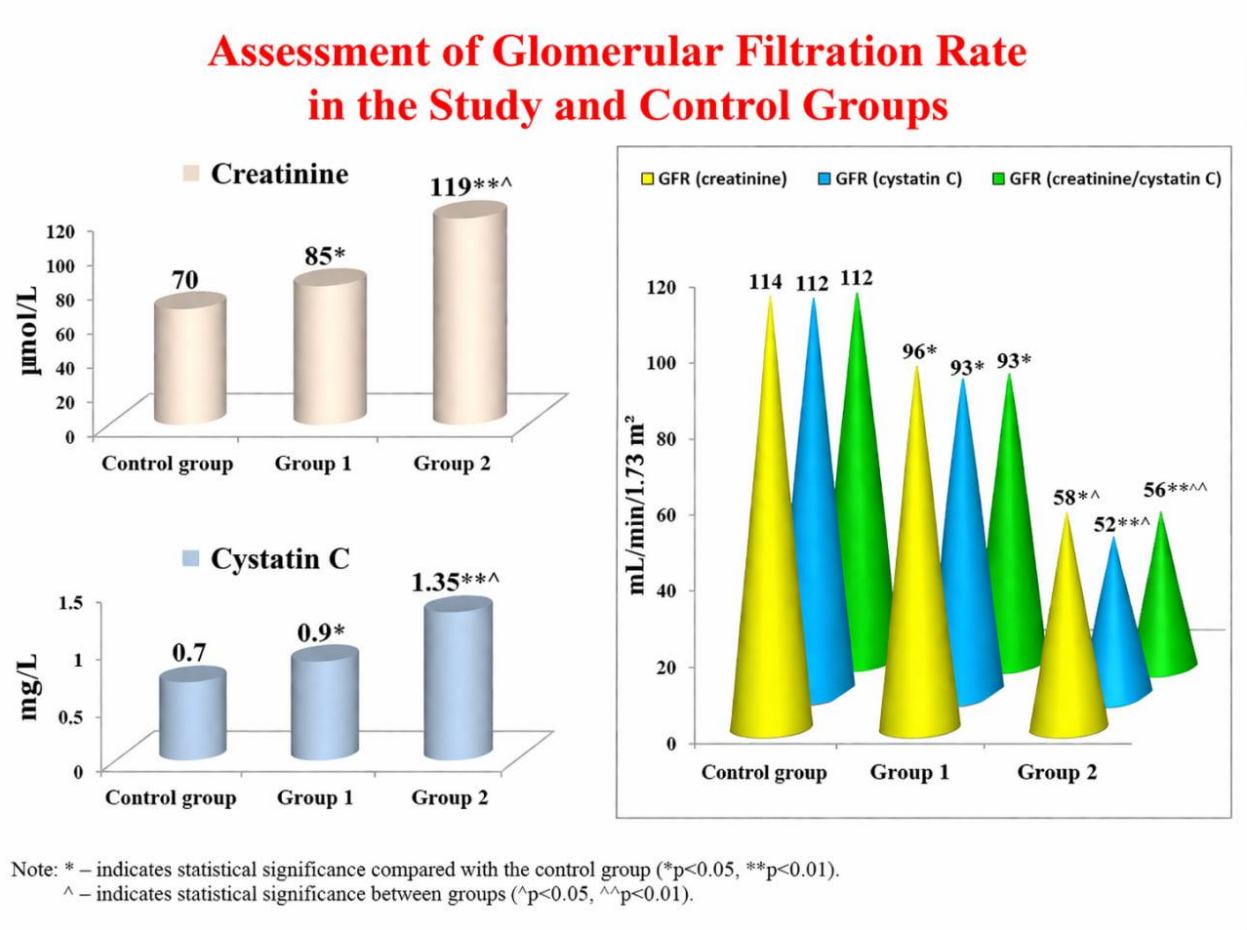

Figure 2. Serum creatinine, cystatin C, and glomerular filtration rate in the control group, patients with gout without nephropathy (Group 1), and patients with gouty nephropathy (Group 2). *Note:* * — compared with the control group; ^ — Group 2 compared with Group 1.

Discussion. The present study demonstrated that markers of early renal injury are substantially altered in patients with gout and become more pronounced in those with gouty nephropathy. The progressive increase in KIM-1, cystatin C, microalbuminuria, and serum creatinine, together with the decline in GFR, reflects the gradual worsening of renal damage from early functional disturbances to clinically significant nephropathy.

KIM-1 showed a marked elevation already in patients without clinically evident nephropathy and increased further in patients with gouty nephropathy. This supports its role as an early marker of tubular epithelial injury. Since tubular damage is one of the central mechanisms of urate-related kidney involvement, elevation of KIM-1 may indicate the onset of renal injury before overt decline in standard renal function tests becomes apparent.

Cystatin C also demonstrated a steady rise across the groups. Compared with creatinine, cystatin C is considered a more sensitive marker of changes in glomerular filtration, especially in early stages of kidney dysfunction. The increase observed even in Group 1 suggests that subtle renal impairment may already be present in gout patients without clinically established nephropathy.

Microalbuminuria showed the most substantial increase, particularly in Group 2. This finding suggests that glomerular barrier dysfunction and endothelial injury are prominent features of gouty nephropathy. MAU may therefore serve as a useful indicator of progression from early renal involvement to more advanced nephropathic changes.

The decrease in GFR estimated by creatinine, cystatin C, and combined formulas confirms progressive renal dysfunction. Notably, cystatin C-based and combined GFR estimates appeared to reflect renal impairment more sensitively than creatinine alone. This supports the view that

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integrated laboratory assessment may improve detection of early kidney injury in patients with gout.

From a clinical perspective, the combined use of KIM-1, cystatin C, microalbuminuria, and GFR estimation may provide a valuable approach for early diagnosis of gouty nephropathy. Timely identification of patients at risk may help optimize monitoring strategies and allow earlier intervention aimed at preserving renal function.

This study has several limitations. The number of patients was moderate, and the analysis was based on selected biomarkers. Additional studies with larger populations and longitudinal follow-up are needed to determine the prognostic value of these markers and to clarify their role in predicting progression of renal involvement in gout.

Conclusion. KIM-1, cystatin C, and microalbuminuria are informative early markers of renal injury in patients with gout. Their values increase progressively from healthy controls to patients with gout without nephropathy and are highest in patients with gouty nephropathy.

At the same time, serum creatinine rises and glomerular filtration rate declines, confirming progressive deterioration of renal function. The combined assessment of KIM-1, cystatin C, MAU, and GFR may improve early detection of gouty nephropathy and facilitate identification of patients at increased risk of renal complications.

These findings support the use of integrated laboratory and functional renal assessment in patients with gout for timely diagnosis and risk stratification of gouty nephropathy.

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