CLINICAL FEATURES OF CHRONIC KIDNEY DISEASE PROGRESSION IN PATIENTS WITH DIFFERENT FORMS OF CHRONIC GLOMERULONEPHRITIS

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Clinical progression of CKD in CGN varies by form. Hypertensive CGN demonstrates significant vascular damage reflected by RRI elevation, while the nephrotic and mixed forms show marked proteinuria and tubular injury

ABSTRACT

Chronic glomerulonephritis (CGN) remains one of the major causes of chronic kidney disease (CKD) worldwide and continues to be highly prevalent in Central Asian countries, including Uzbekistan. Various clinical-morphological forms-nephrotic, hypertensive, and mixed-differ in pathogenesis and progression rate. Persistent glomerular inflammation, vascular remodeling, and nephron loss contribute to gradual renal dysfunction. Early identification of prognostic markers for CKD progression in CGN patients is essential for timely therapeutic interventions. Aim. To evaluate the clinical features and early predictors of chronic kidney disease progression in patients with different clinical forms of chronic glomerulonephritis

Introduction. Chronic glomerulonephritis (CGN) remains one of the major causes of chronic kidney disease (CKD) worldwide and continues to be highly prevalent in Central Asian countries, including Uzbekistan. Various clinical-morphological forms-nephrotic, hypertensive, and mixed-differ in pathogenesis and progression rate. Persistent glomerular inflammation, vascular remodeling, and nephron loss contribute to gradual renal dysfunction. Early identification of prognostic markers for CKD progression in CGN patients is essential for timely therapeutic interventions. Aim. To evaluate the clinical features and early predictors of chronic kidney disease progression in patients with different clinical forms of chronic glomerulonephritis.

Materials and Methods. A total of 60 patients (18-60 years) with established chronic glomerulonephritis were included: nephrotic form (n=20), hypertensive form (n=20), mixed form (n=20). Renal function was assessed by creatinine, cystatin C, and eGFR (CKD-EPI). Proteinuria, ACR, NGAL, KIM-1, inflammatory markers (CRP, IL-6), and endothelial markers (ADMA, sICAM-1) were studied. Renal Doppler ultrasonography assessed renal resistive index (RRI). Ambulatory blood pressure monitoring was used to evaluate hypertension patterns. Results. Progressive CKD indicators were detected in 27/60 patients (45%). Proteinuria >1 g/day predominated in nephrotic (85%) and mixed (70%) forms. Reduced eGFR <90 mL/min/1.73 m^2: hypertensive (40%), mixed (50%). Elevated cystatin C: 30% of all patients, especially in mixed form. Increased RRI (>0.70): 36%, most pronounced in hypertensive form. High NGAL and KIM-1 ($p<0.05$) were more common in nephrotic and mixed forms. Persistent

hypertension or nocturnal non-dipping was found in 47% and strongly correlated with proteinuria and eGFR decline.

Discussion. Clinical progression of CKD in CGN varies by form. Hypertensive CGN demonstrates significant vascular damage reflected by RRI elevation, while the nephrotic and mixed forms show marked proteinuria and tubular injury. Cystatin C, tubular biomarkers, and Doppler indices are sensitive early markers. Persistent hypertension and inflammation accelerate renal decline.

Conclusion. Patients with various CGN forms show distinct clinical patterns associated with CKD progression. Comprehensive follow-up including cystatin C, tubular markers, RRI, and blood pressure monitoring-is essential for early detection and prevention of renal deterioration.

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