



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

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ARTICLE INFO

Received: 8th May 2026

Accepted: 9th May 2026

Online: 14th May 2026

KEYWORDS

ABSTRACT

Chronic glomerulonephritis (CGN) remains one of the leading causes of chronic kidney disease (CKD) worldwide and continues to be highly prevalent in Central Asian countries, including Uzbekistan. Various clinical and morphological forms — nephrotic, hypertensive, and mixed — differ in pathogenesis and progression rate. Persistent glomerular inflammation, vascular remodeling, and nephron loss contribute to gradual deterioration of renal function. Early identification of prognostic markers for CKD progression in patients with CGN is essential for timely therapeutic intervention.

Introduction. Chronic glomerulonephritis (CGN) remains one of the leading causes of chronic kidney disease (CKD) worldwide and continues to be highly prevalent in Central Asian countries, including Uzbekistan. Various clinical and morphological forms — nephrotic, hypertensive, and mixed — differ in pathogenesis and progression rate. Persistent glomerular inflammation, vascular remodeling, and nephron loss contribute to gradual deterioration of renal function. Early identification of prognostic markers for CKD progression in patients with CGN is essential for timely therapeutic intervention.

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 aged 18–60 years with established chronic glomerulonephritis were included in the study: nephrotic form (n=20), hypertensive form (n=20), and mixed form (n=20). Renal function was assessed using serum creatinine, cystatin C, and estimated glomerular filtration rate (eGFR, CKD-EPI). Proteinuria, albumin-to-creatinine ratio (ACR), Renal Doppler ultrasonography was performed to assess the renal resistive index (RRI). Ambulatory blood pressure monitoring was used to evaluate hypertension patterns.

Results. Signs of CKD progression were identified in 27 out of 60 patients (45%). Proteinuria >1 g/day predominated in nephrotic (85%) and mixed (70%) forms. Reduced eGFR <90 mL/min/1.73 m² was observed in patients with hypertensive (40%) and mixed (50%) forms. Elevated cystatin C levels were detected in 30% of all patients, especially in the mixed form. Increased RRI (>0.70) was found in 36% of patients and was most pronounced in the hypertensive form. Persistent hypertension or nocturnal non-dipping blood pressure patterns were identified in 47% of patients and strongly correlated with proteinuria and decline in eGFR.

Discussion. Clinical progression of CKD in CGN varies depending on the disease form. The hypertensive form of CGN is characterized by significant vascular damage reflected by elevated RRI, whereas nephrotic and mixed forms are associated with marked proteinuria and tubular injury. Cystatin C and Doppler ultrasonographic indices are sensitive early markers of renal dysfunction. Persistent hypertension and inflammation accelerate renal function decline.

Conclusion. Patients with different forms of chronic glomerulonephritis demonstrate distinct clinical characteristics associated with CKD progression. Comprehensive follow-up, including assessment of cystatin C and ambulatory blood pressure monitoring, is essential for early detection and prevention of renal deterioration

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