



OPTIMIZING EARLY DIAGNOSIS OF CHRONIC GLOMERULONEPHRITIS THROUGH GENETIC SCREENING

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Abstract

Chronic glomerulonephritis (CGN) remains one of the major causes of chronic kidney disease (CKD), which occurs in nearly 80–90% of CGN patients. Despite modern treatment advances, mortality and morbidity in these patients remain high. Early detection of kidney damage and timely initiation of treatment significantly reduce the risk of irreversible inflammation and help preserve renal function. In this study, nephrotic and mixed clinical forms of CGN were compared based on laboratory and instrumental findings. The results show that early detection of renal injury using a combination of genetic screening and clinical markers plays an important role in improving quality of life and life expectancy in CGN patients.

Objective

The primary objective of this study is to optimize early diagnostic methods for chronic glomerulonephritis by conducting a comprehensive analysis of the associations between polymorphic variants of candidate genes and clinical features of the disease.

Materials and Methods

A total of 40 patients diagnosed with CGN and 30 age- and sex-matched healthy controls were enrolled in the study. All participants underwent detailed laboratory examinations including urine analysis, biochemical assays (serum creatinine, urea, lipid profile), and assessment of microalbuminuria. Functional diagnostics included electrocardiography (ECG), echocardiography (EchoCG), renal ultrasound, and Dopplerography of the renal vessels to evaluate renal hemodynamics. In addition, genetic testing was performed to analyze polymorphisms of candidate genes: IL4 C-589T, TNF G308A, and ACE insertion/deletion (allele D). Statistical analyses were performed using SPSS 18.0 software, with $p < 0.05$ considered statistically significant.

Results

The study revealed that CGN patients had significantly elevated levels of microalbuminuria, serum urea, and creatinine compared to the control group ($p < 0.05$). A decline in the glomerular filtration rate (GFR) was consistently observed among patients. Dopplerographic findings indicated increased vascular resistance and reduced renal blood flow, which correlated with



decreased GFR, highlighting the role of hemodynamic disturbances in disease progression. Genetic analysis demonstrated that the frequency of IL4 (C-589T), TNF (G308A), and ACE (allele D) polymorphisms was higher in CGN patients compared to healthy individuals. These findings suggest that genetic variants contribute to disease susceptibility and accelerate progression toward CKD.

Discussion

The findings of this study emphasize the importance of combining clinical and genetic diagnostic approaches for early detection of CGN. Conventional markers such as serum creatinine and GFR are useful but often identify kidney damage at later stages. The addition of microalbuminuria testing and renal Dopplerography enhances sensitivity for early detection. Furthermore, the genetic predisposition conferred by IL4, TNF, and ACE polymorphisms highlights the role of personalized medicine in nephrology. Patients carrying these genetic variants may benefit from closer monitoring and earlier initiation of nephroprotective therapy.

Conclusion

In conclusion, the integration of genetic screening with clinical and instrumental methods provides a more effective strategy for the early diagnosis of CGN. The identification of IL4, TNF, and ACE polymorphisms as predictive markers allows clinicians to identify high-risk patients and tailor treatment accordingly. Early recognition of kidney dysfunction and timely intervention can slow the progression of CKD, improve patient outcomes, and reduce the overall healthcare burden. Future large-scale studies are recommended to validate these findings and expand the scope of genetic testing in routine nephrological practice.

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