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FACTORS ASSOCIATED WITH ACUTE AND LONG-TERM RENAL DYSFUNCTION IN PEDIATRIC POSTINFECTIOUS GLOMERULONEPHRITIS

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Abstract

Postinfectious glomerulonephritis (PIGN) remains one of the most common causes of acute glomerulonephritis in children worldwide. Although the disease generally has a favorable prognosis, some pediatric patients may develop acute kidney injury (AKI) and long-term renal impairment. Early identification of risk factors associated with kidney damage is important for improving clinical management and preventing chronic kidney disease.

These findings highlight the importance of early identification of clinical and laboratory predictors of kidney damage in children with PIGN. Recognizing high-risk patients may improve clinical outcomes and guide long-term monitoring strategies.

Keywords: Postinfectious glomerulonephritis, children, acute kidney injury, complement system, C3, proteinuria, hypertension.

Introduction

Postinfectious glomerulonephritis (PIGN) is an immune-mediated kidney disease that develops after bacterial or viral infections. It is characterized by inflammatory damage to the renal glomeruli caused by immune complex

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deposition and complement activation. Despite improvements in healthcare systems and infection control measures, PIGN remains one of the leading causes of acute glomerulonephritis in pediatric populations, particularly in developing countries.

The most common etiological factor associated with PIGN is infection caused by group A beta-hemolytic streptococci. However, other pathogens, including staphylococci, viruses, and parasites, have also been implicated in the development of this condition. The clinical presentation of PIGN may include hematuria, proteinuria, edema, and arterial hypertension. In severe cases, patients may develop acute kidney injury, requiring hospitalization and intensive monitoring.

Although most children with PIGN recover completely within several weeks or months, a subset of patients may develop persistent renal abnormalities. These complications may include chronic proteinuria, hypertension, or decreased glomerular filtration rate, which can ultimately lead to chronic kidney disease. Identifying factors associated with adverse outcomes is therefore crucial for optimizing patient management.

Several studies have suggested that activation of the complement system plays an important role in the pathogenesis of postinfectious glomerulonephritis. Reduced levels of complement component C3 are frequently observed during the acute phase of the disease and may reflect immune complex-mediated activation of the alternative complement pathway. Persistent complement abnormalities may also indicate underlying complement regulatory disorders.

Understanding the clinical and laboratory predictors of disease severity is essential for identifying high-risk patients who require closer monitoring and long-term follow-up. Therefore, the aim of this study was to evaluate the risk factors associated with short- and long-term kidney damage in children diagnosed with postinfectious glomerulonephritis.

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Materials and Methods

This study was conducted as a retrospective cohort analysis of pediatric patients diagnosed with postinfectious glomerulonephritis. Medical records were reviewed to obtain demographic, clinical, and laboratory data.

Children diagnosed with postinfectious glomerulonephritis and referred to a specialized nephrology center were included in the study. The diagnosis of PIGN was established based on clinical presentation, laboratory findings, and evidence of preceding infection.

Inclusion criteria were:

- age under 18 years;
- clinical diagnosis of postinfectious glomerulonephritis;
- availability of laboratory data at the time of diagnosis and during follow-up.

Patients with pre-existing chronic kidney disease or other primary glomerular diseases were excluded.

Data collection

Clinical data collected from patient records included age, sex, blood pressure, hospitalization status, and clinical symptoms at presentation. Laboratory parameters included serum creatinine levels, complement component C3 concentration, urinalysis results, and proteinuria levels.

Estimated glomerular filtration rate (eGFR) was calculated using the Schwartz formula.

The primary outcome of the study was acute kidney injury (AKI) at the time of initial presentation.

The secondary outcome was composite kidney damage at the final follow-up visit, defined as the presence of at least one of the following conditions:

- decreased estimated glomerular filtration rate (eGFR);
- persistent proteinuria;
- arterial hypertension.

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Statistical analysis

Statistical analysis was performed using logistic regression models. Independent risk factors associated with the development of acute kidney injury and long-term kidney damage were identified. Odds ratios (OR) and 95% confidence intervals (CI) were calculated.

Results

A total of 125 pediatric patients diagnosed with postinfectious glomerulonephritis were included in the study. The mean age at presentation was 8.3 ± 3.5 years. The average duration of follow-up was approximately 252 days.

Among the patients included in the study, acute kidney injury was observed in 66% of cases. Additionally, 57% of patients required hospitalization due to disease severity.

Multivariate logistic regression analysis identified several significant predictors associated with the development of acute kidney injury.

The following factors were independently associated with AKI:

- decreased complement C3 levels;
- nephrotic-range proteinuria;
- the need for antihypertensive therapy;
- severe clinical presentation at diagnosis.

These findings suggest that complement activation and significant glomerular damage play a key role in the development of acute kidney injury in children with PIGN.

At the final follow-up visit, approximately 35% of patients demonstrated persistent renal abnormalities, including reduced eGFR, proteinuria, or arterial hypertension.Older age at presentation and significantly decreased complement C3 levels were associated with long-term renal impairment.

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Discussion

The results of this study confirm that postinfectious glomerulonephritis remains an important cause of acute kidney injury in children. Although the disease is generally considered self-limiting, a considerable proportion of patients may develop significant renal complications.

The complement system appears to play a critical role in the pathogenesis of this condition. Reduced complement C3 levels observed in many patients reflect activation of the alternative complement pathway, which contributes to inflammatory injury of the glomeruli.

Proteinuria is another important marker of glomerular damage. Nephrotic-range proteinuria indicates severe disruption of the glomerular filtration barrier and is associated with a higher risk of kidney dysfunction.

Hypertension is frequently observed in children with PIGN and may reflect both acute and chronic kidney injury. Persistent hypertension may further contribute to progressive renal damage.

The identification of clinical and laboratory predictors of adverse outcomes is essential for improving patient management. Early recognition of high-risk patients may allow clinicians to implement more intensive monitoring and timely therapeutic interventions.

Conclusion

Postinfectious glomerulonephritis remains a significant cause of acute kidney injury in children. The severity of the initial disease presentation, decreased complement C3 levels, and nephrotic-range proteinuria are important predictors of kidney damage.

Early identification of these risk factors may help clinicians determine which patients require closer monitoring and prolonged follow-up. Improved understanding of disease predictors may contribute to better prevention of long-term renal complications in pediatric patients with PIGN.

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