

Unmasking Pseudomonas-Induced Infection-Related Glomerulonephritis Presenting with Nephrotic-Range Proteinuria in an Adult Female: A Rare Case Report

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ABSTRACT

Background:

Infection-related glomerulonephritis (IRGN) is an immune-complex-mediated glomerular disease traditionally associated with streptococcal infections. In recent decades, a shift in epidemiology has been observed, with increasing recognition of non-streptococcal pathogens, particularly in adults. *Pseudomonas aeruginosa* is an uncommon cause of IRGN, and nephrotic-range proteinuria as the predominant clinical manifestation is rare.

Case

A 57-year-old female presented with progressively increasing generalized edema involving both lower limbs, abdomen, and face, accompanied by productive cough, fever, and dyspnea of twenty days' duration. Physical examination revealed anasarca and bilateral basal crepitations. Urinalysis demonstrated heavy proteinuria and microscopic hematuria. Sputum culture yielded *Pseudomonas aeruginosa*, whereas urine culture was sterile. Renal biopsy revealed diffuse endocapillary proliferative glomerulonephritis with prominent neutrophilic infiltration without crescent formation, consistent with infection-related glomerulonephritis. Empirical corticosteroid therapy initiated before biopsy confirmation was discontinued subsequently. The patient was managed with anti-pseudomonal therapy, renin-angiotensin-aldosterone system blockade, and supportive measures, resulting in significant clinical improvement.

Presentation:

Conclusion:

This case highlights the importance of recognizing pulmonary *Pseudomonas aeruginosa* infection as a rare trigger of adult IRGN. The predominance of nephrotic-range proteinuria may mimic primary nephrotic syndromes and emphasizes the indispensable role of renal biopsy in establishing an accurate diagnosis and avoiding inappropriate immunosuppression.

Keywords: Infection-related glomerulonephritis; *Pseudomonas aeruginosa*; Nephrotic-range proteinuria; Renal biopsy; Adult glomerulonephritis; Pulmonary infection.

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Introduction

Infection-related glomerulonephritis (IRGN) is an immune-complex-mediated glomerular disease characterized by glomerular inflammation occurring in association with an active or recent infection. Historically, post-streptococcal glomerulonephritis represented the classical prototype, commonly affecting children following pharyngitis or skin infections. However, the epidemiological pattern of IRGN has evolved considerably over recent decades, particularly among adults, with increasing involvement of non-streptococcal pathogens and deep-seated visceral infections.¹⁻³

Adult IRGN is frequently associated with *Staphylococcus aureus*, infective endocarditis, osteomyelitis, and visceral abscesses. Gram-negative organisms have also been implicated, although their occurrence remains relatively uncommon. *Pseudomonas aeruginosa*, an opportunistic Gram-negative bacillus commonly associated with nosocomial pulmonary infections, is rarely reported as an etiological agent for IRGN.³

The classical presentation of IRGN includes features of acute nephritic syndrome such as hematuria, hypertension, edema, and varying degrees of renal dysfunction with mild-to-moderate proteinuria. Nevertheless, atypical manifestations have increasingly been recognized. In particular, nephrotic-range proteinuria may occasionally predominate and mimic primary glomerular diseases including minimal change disease, membranous nephropathy, and focal segmental glomerulosclerosis.^{4,5} Such presentations pose significant diagnostic challenges and may result in unnecessary exposure to prolonged immunosuppressive therapy.

We report a rare case of pulmonary *Pseudomonas aeruginosa*-associated IRGN in a 57-year-old female who presented predominantly with nephrotic-range proteinuria. The case underscores the importance of maintaining a high index of suspicion for infection-related etiologies and highlights the pivotal role of renal biopsy in guiding management.

Case Presentation

A 57-year-old female presented to our tertiary care center with complaints of progressive swelling involving both lower limbs for one month. Initially confined to the feet, the edema gradually ascended to involve the abdomen and face, resulting in generalized body swelling. The patient also reported productive cough with expectoration, intermittent low-grade fever, and progressive shortness of breath for approximately twenty days.

There was no previous history of diabetes mellitus, hypertension, chronic kidney disease, connective tissue disorders, or chronic respiratory illness. She denied any history suggestive of preceding skin infections or pharyngitis.

On examination, the patient was febrile with a temperature of 38.2°C and mildly tachypneic. Blood pressure was stable. Marked generalized edema with bilateral pitting pedal edema and facial puffiness was evident. Respiratory system examination revealed bilateral basal crepitations. Cardiovascular and neurological examinations were otherwise unremarkable.

Laboratory evaluation demonstrated active urinary sediment with heavy proteinuria (++++), and microscopic hematuria (16–18 red blood cells/high-power field). Pus cells were 6–8/high-power field, and epithelial cells were 30–32/high-power field. No casts were identified. Twenty-four-hour urinary protein estimation revealed significant protein

excretion compatible with nephrotic-range proteinuria. Urine culture showed no bacterial growth.

Sputum culture grew *Pseudomonas aeruginosa*, identifying the respiratory tract as the source of infection. Renal biopsy revealed diffuse endocapillary proliferative glomerulonephritis characterized by prominent neutrophilic infiltration without crescent formation, findings consistent with infection-related glomerulonephritis.

Before biopsy confirmation, empirical corticosteroid therapy was initiated owing to the possibility of a primary glomerular disease. Following histopathological confirmation of IRGN, corticosteroids were discontinued. The patient was subsequently managed with anti-pseudomonal agents, renin-angiotensin-aldosterone system blockade, and supportive measures. Progressive resolution of fever, cough, and generalized edema was observed, and the patient was discharged in a clinically improved state.

Discussion

Infection-related glomerulonephritis (IRGN) represents an immune-complex-mediated glomerular disease that develops during or following an active infection. Although historically regarded as a post-streptococcal disease affecting children, adult IRGN has emerged as a distinct clinical entity with changing epidemiological characteristics. Non-streptococcal pathogens and deep-seated visceral infections are increasingly recognized as important triggers in adults.¹⁻³

The present case is noteworthy because of two unusual features: the rarity of *Pseudomonas aeruginosa* as the underlying pathogen and the predominance of nephrotic-range proteinuria at presentation. To the best of our knowledge, pulmonary *Pseudomonas* infection precipitating biopsy-proven IRGN with nephrotic-range proteinuria has been infrequently described in the literature.

Pseudomonas as an Uncommon Etiological Agent

Most adult cases of IRGN are associated with *Staphylococcus aureus*, infective endocarditis, osteomyelitis, diabetic foot infections, and visceral abscesses.^{2,4} Gram-negative organisms are less frequently implicated. *Pseudomonas aeruginosa* is primarily recognized as an opportunistic pathogen responsible for nosocomial pneumonia and chronic pulmonary infections. Reports linking *Pseudomonas* infection with glomerulonephritis remain exceedingly rare.⁵

In the present patient, sputum culture demonstrated growth of *Pseudomonas aeruginosa*, establishing the lungs as the primary source of infection. The absence of bacterial growth in urine culture and the histopathological findings on renal biopsy strongly

supported the diagnosis of pulmonary infection-associated IRGN.

Pathophysiology

The pathogenesis of IRGN involves the formation of circulating immune complexes containing bacterial antigens. These immune complexes deposit within the glomeruli and activate complement pathways, leading to recruitment of inflammatory cells and subsequent glomerular injury.⁶

Histologically, diffuse endocapillary proliferation with prominent neutrophilic infiltration is a characteristic finding in IRGN. Crescent formation may occur in severe cases but was absent in our patient. The biopsy findings in the present case were classical for infection-related glomerulonephritis and played a decisive role in confirming the diagnosis.

Nephrotic-Range Proteinuria as the Predominant Manifestation

Classically, IRGN presents with nephritic syndrome characterized by hematuria, edema, hypertension, and mild-to-moderate proteinuria. However, increasing evidence suggests that heavy proteinuria and even nephrotic-range proteinuria may occur in a subset of adult patients.^{2,7}

The present case is particularly unusual because nephrotic-range proteinuria constituted the dominant clinical manifestation. Such a presentation may closely mimic primary nephrotic syndromes including minimal change disease, membranous nephropathy, focal segmental glomerulosclerosis, and IgA nephropathy. Consequently, diagnosis based solely on clinical features may be misleading.

Massive proteinuria in IRGN has been attributed to severe inflammatory injury to the glomerular capillary wall and podocytes, resulting in disruption of the filtration barrier.⁸ Recognition of this atypical phenotype is important because management differs substantially from that of primary nephrotic syndromes.

Importance of Renal Biopsy

Renal biopsy remains the gold standard for diagnosis in atypical presentations. In the present case, empirical corticosteroid therapy was initiated because of concern for an underlying primary glomerular disease. Histopathological confirmation of IRGN prompted withdrawal of steroids and a shift toward pathogen-directed therapy.

This distinction is clinically relevant because prolonged immunosuppression in the presence of active infection may worsen bacterial proliferation and adversely affect outcomes.⁹ Early renal biopsy prevented unnecessary continuation of corticosteroid therapy and facilitated appropriate treatment.

Therapeutic Considerations

Management of IRGN primarily focuses on eradication of the underlying infection and supportive

care. Treatment strategies include appropriate antimicrobial therapy, optimization of blood pressure, control of edema, and reduction of proteinuria through renin-angiotensin-aldosterone system blockade.¹⁰

The role of corticosteroids remains controversial and is generally limited to selected cases after careful exclusion of ongoing infection. In our patient, withdrawal of steroids following biopsy confirmation and institution of targeted therapy resulted in marked clinical improvement with resolution of respiratory symptoms and edema.

Clinical Significance

This case highlights the evolving spectrum of adult IRGN and demonstrates that nephrotic-range proteinuria does not exclude the diagnosis of infection-related glomerulonephritis. Awareness of such atypical presentations is essential to avoid misclassification as primary nephrotic syndrome and inappropriate administration of prolonged immunosuppressive therapy.

Conclusion

The present case illustrates a rare association between pulmonary *Pseudomonas aeruginosa* infection and biopsy-proven infection-related glomerulonephritis in an adult female. Unlike the classical nephritic presentation, nephrotic-range proteinuria was the predominant manifestation, creating a diagnostic dilemma and mimicking primary glomerular disease. Renal biopsy proved indispensable in establishing the diagnosis and guiding appropriate management. Early recognition of infection-related etiologies and prompt institution of pathogen-directed therapy remain crucial for favorable outcomes.

Learning Points

1. *Pseudomonas aeruginosa* is a rare but important cause of adult infection-related glomerulonephritis.
2. IRGN may present predominantly with nephrotic-range proteinuria.
3. Heavy proteinuria does not exclude infection-related glomerulonephritis.
4. Renal biopsy is essential in atypical presentations.
5. Early diagnosis prevents unnecessary prolonged immunosuppressive therapy.

Declaration of Patient Consent

Appropriate informed consent was obtained from the patient for publication of clinical details. Efforts have been made to ensure anonymity, and no identifying information has been disclosed.

Conflict of Interest

None declared.

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KIDNEY BIOPSY SLIDE :
https://digiscan.app/DSSStore/HtmlViewerMTA0MDQ%3d_c-3

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