WJPMR, 2026, 12(2), 469-472

Case Study

Impact Factor: 7.555

WORLD JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.wjpmr.com

ISSN (O): 2455-3301

ISSN (P): 3051-2557

Coden USA: WJPMBB

ACUTE GLOMERULONEPHRITIS COMPLICATING PULMONARY TUBERCULOSIS

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Department of Pulmonology, 20 August 1953 Hospital, Ibn Rochd University Hospital Center, Casablanca, Morocco. **DOI:** https://doi.org/10.5281/zenodo.18480696

How to cite this Article: Bougteb N., Belhaj C.*, Bamha H., Sajid I., Msika S., Arfaoui H., Jabri H., El khattabi W., Afif Mly H. (2026). Acute Glomerulonephritis Complicating Pulmonary Tuberculosis. World Journal of Pharmaceutical and Medical Research, 12(2), 469–472.

Article Received on 05/01/2026 **Article Revised on 25/01/2026** **Article Published on 04/02/2026**

ABSTRACT

Acute glomerulonephritis associated with tuberculosis is a rare but serious complication, most often resulting from immunological mechanisms triggered by Mycobacterium tuberculosis infection. Clinical presentations are heterogeneous, ranging from isolated proteinuria to rapidly progressive acute renal failure, with various histological patterns reported. We report the case of acute glomerulonephritis occurring in the aftermath of pulmonary tuberculosis in a 47-year-old patient, a chronic active smoker, revealed by the sudden onset of acute nephrotic syndrome associated with deterioration of general condition. Biological investigations showed a rapid decline in glomerular filtration rate, and the diagnosis was confirmed by renal biopsy.

KEYWORDS: Acute glomerulonephritis; Nephrotic syndrome; Pulmonary tuberculosis.

INTRODUCTION

Post-infectious acute glomerulonephritis is common in children in developing countries and less frequent in adults, in whom it is often associated with a more severe clinical presentation. Owing to the widespread use of antibiotic therapy, its incidence has been gradually declining in developed countries. Acute glomerulonephritis complicating tuberculous infection has rarely been reported in the literature.

CASE REPORT

The patient was a 47-year-old man, a chronic smoker with a 21 pack-year history, not weaned, an occasional alcohol consumer who had stopped drinking, with a history of chronic waterpipe and snuff tobacco use, both discontinued. He had been followed for bacteriologically confirmed pulmonary tuberculosis for one week and was receiving anti-tuberculous therapy, with no prior history of flu-like syndrome or pharyngitis.

The history of the present illness began four days prior to hospitalization with the onset of swelling associated with a pruritic skin eruption involving both feet and legs, along with inflammatory-type arthralgia causing functional impairment of the lower limbs. These symptoms evolved in a context of subjective fever and mild deterioration of general condition.

On admission, general examination revealed a performance status of 1, normal body mass index, afebrile status, normotension (130/70 mmHg), tachycardia at 117 beats per minute, adequate oxygen saturation on room air (97%), pitting edema of the lower limbs, and mild subicterus.

Pleuropulmonary examination showed a cavitary syndrome characterized by decreased vesicular breath sounds over both lung fields and a blowing sound over the upper third of the left lung field.

Cutaneous and mucosal examination revealed dependent petechial purpura involving the legs and feet, without pustules, vesicles, or signs of inflammation.

Urinalysis by dipstick showed proteinuria.

Chest X-ray showed diffuse bilateral nodular and micronodular infiltrates with areas of cavitation related to pulmonary tuberculosis (Figure 1).

Laboratory investigations revealed hypochromic microcytic anemia with a hemoglobin level of 10 g/dL and leukocytosis of 14,250/mm³ with neutrophil predominance. The erythrocyte sedimentation rate was elevated, and C-reactive protein was markedly increased

at 187 mg/L. Twenty-four-hour urinary protein excretion was elevated at 2 g.

Liver function tests were within normal limits. Renal investigations showed a serum creatinine level of 9.6 mg/L and a blood urea level of 0.35 g/L, with a normal estimated glomerular filtration rate (eGFR) of 98.11 mL/min/1.73 m². Serum albumin was decreased to 20 g/L.

Given the suspicion of deep vein thrombosis of the lower limbs, Doppler ultrasound of the lower extremities was performed and did not reveal any venous thrombosis.

A post-infectious or drug-induced vasculitis was considered. Antineutrophil cytoplasmic antibodies (ANCA) and anti–cyclic citrullinated peptide (anti-CCP) antibodies were negative.

The clinical course was marked by the development of acute renal failure, with serum creatinine rising from 9 mg/L to 18 mg/L over three days, accompanied by a decrease in estimated glomerular filtration rate (eGFR) to 46.14 mL/min/1.73 m², and further decline to 23.13 mL/min/1.73 m² by day five.

Urine cytobacteriological examination did not reveal hematuria; however, urinary lymphocytes were elevated and urine culture was sterile.

Renal and bladder ultrasound showed both kidneys of normal size, with regular contours, preserved corticomedullary differentiation, no dilatation of the collecting systems, and no identifiable obstruction. Doppler ultrasound of the renal arteries revealed no abnormalities.

An urgent renal biopsy was performed, revealing endocapillary and extracapillary glomerulonephritis with intense C3 deposition and no immunoglobulin deposits. Complement levels (C3 and C4) were within normal limits.

A diagnosis of post-infectious glomerulonephritis was established, and the patient was started on intravenous methylprednisolone boluses at 1 g/day for three days, followed by oral prednisone at 1 mg/kg/day with gradual tapering, under coverage of broad-spectrum antibiotic therapy (ceftriaxone 2 g/day) and adjustment of anti-tuberculosis treatment doses.

At one-month follow-up, the patient showed marked clinical and laboratory improvement, including resolution of lower limb edema, normalization of white blood cell count and C-reactive protein levels, and an increase in estimated glomerular filtration rate to 40.66 mL/min/1.73 m². Follow-up chest radiography at one month demonstrated partial resolution of pulmonary tuberculosis lesions (Figure 2).

DISCUSSION

Post-infectious glomerulonephritis, a nephritic syndrome, is the most common cause of acute glomerulonephritis in children aged 3 to 15 years. It is more frequent in densely populated and economically disadvantaged regions.[1]

Most cases are caused by nephritogenic beta-hemolytic group A streptococcal strains, notably type 12 (associated with pharyngitis) and type 49 (causing impetigo). A latency period of 6 to 21 days between the infection and the onset of glomerulonephritis is typical, although it can extend up to 6 weeks. This latency is longer than that observed between IgA nephropathy and prior infection. (1) Less commonly implicated pathogens include other non-streptococcal bacteria, viruses, parasites, rickettsiae, and fungi (see Table: Causes of Glomerulonephritis). In our case, glomerulonephritis was secondary to pulmonary tuberculosis.

The pathophysiological mechanism is not fully understood, but microbial antigens are thought to bind to the glomerular basement membrane and initially activate the alternative complement pathway, both directly and via interaction with circulating antibodies, leading to glomerular injury that may be focal or diffuse. Alternatively, circulating immune complexes may deposit on the glomerular basement membrane.[1]

Clinical manifestations range from asymptomatic hematuria with mild proteinuria to a full nephritic syndrome, including microscopic or macroscopic hematuria (cola-colored, brown, dark, or frankly hemorrhagic), proteinuria (sometimes nephrotic-range), oliguria, edema, hypertension, and acute renal failure. Fever is uncommon and usually suggests persistent infection. Clinical features of non-streptococcal post-infectious glomerulonephritis may mimic other disorders, such as polyarteritis nodosa, renal emboli, or drug-induced acute interstitial nephritis.[2]

In our study, the clinical presentation was characterized by lower limb edema and deterioration of general condition.

The definitive diagnosis is based on evidence of recent infection, urinalysis typically showing dysmorphic red blood cells, red blood cell casts, proteinuria, white blood cells, and tubular epithelial cells, often associated with hypocomplementemia.[3] Renal biopsy is not indicated in typical cases but is recommended in patients with massive proteinuria and nephrotic syndrome persisting beyond seven days, or in cases of acute renal failure lasting more than three days.[4] Biopsy findings reveal features of post-infectious glomerulonephritis, including enlarged glomeruli and hypercellularity, with initial neutrophilic infiltration followed later by mononuclear cell infiltration.[3]

Treatment of glomerulonephritis depends on its type and severity, with the main goal being to control inflammation and preserve renal function.[5]

Corticosteroids often remain the first-line treatment. Prednisolone, administered in tapering doses, helps reduce glomerular inflammation. However, prolonged use requires close monitoring for side effects such as weight gain, osteoporosis, and diabetes.[6]

Immunosuppressive agents frequently complement corticosteroid therapy. Cyclophosphamide, mycophenolate mofetil, or cyclosporine may be prescribed depending on the type of glomerulonephritis. These medications require regular laboratory monitoring to adjust doses and ensure tolerability.[7]

Symptomatic treatment is equally important. Angiotensin-converting enzyme inhibitors (ACE inhibitors) or angiotensin II receptor blockers (ARBs) protect renal function and help control blood pressure. Diuretics assist in eliminating excess fluid, and a low-salt diet is often recommended.[8]

Complete recovery is possible in 70–80% of cases, particularly in children.[9] Several factors influence prognosis: age at diagnosis (better in children), severity of initial renal involvement, response to treatment during the first few months, and the presence of hypertension. Recent therapeutic innovations continue to improve these outcomes.[10]

A scoping review including 130 patients with tuberculosis who developed glomerulonephritis and underwent renal biopsy reported a mean age of 42.3 years, with women representing a minority. Acute glomerulonephritis occurred immediately following the tuberculosis diagnosis in most cases (88.9%) and rarely after 24 weeks (5.7%). The most frequent site of tuberculosis involvement was the lung (78.9%), followed by lymph nodes (8.8%). Management was based on corticosteroid and/or immunosuppressive therapy as well as adjustments to anti-tuberculosis medications. Improvement in glomerular filtration rate was incompletely reported.[11]

The conclusions of this study are consistent with the clinical findings observed in our reported case.

Prevention of infections remains essential. Prompt treatment of streptococcal pharyngitis, particularly in children, is important, and the full course of prescribed antibiotics should be completed. Hepatitis B vaccination provides protection against certain forms of secondary glomerulonephritis.[12]

CONCLUSION

Acute glomerulonephritis caused by Mycobacterium tuberculosis remains rare in our context. Early detection and effective treatment of infections, particularly tuberculosis, could help reduce the incidence of this condition. Recognition of this association is crucial to prevent progression to chronic renal failure.

Figures

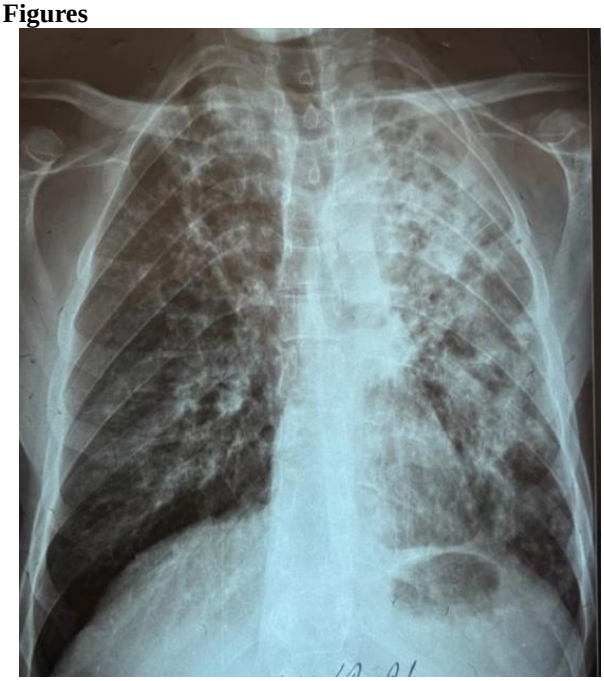

Figure 1: Chest radiograph showing diffuse bilateral nodular and micronodular infiltrates with cavitary lesions, consistent with pulmonary tuberculosis.

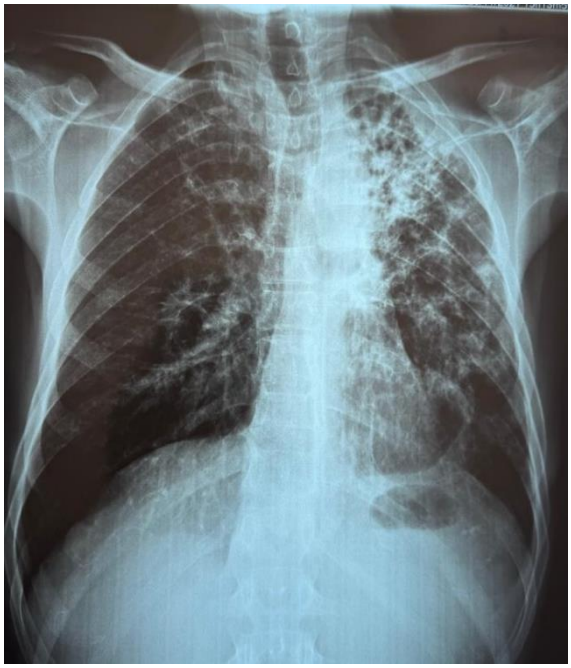

Figure 2: Follow-up chest radiograph at one month showing partial resolution of pulmonary tuberculosis lesions after treatment.

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