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Case Report

Crescentic Glomerulonephritis in a 65-year-old Male Patient with Lepromatous Leprosy: Case Report

P Bhutani[1], A Kumar[2], M Dhar[3], M Pathania[4]

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Leprosy, a chronic infectious disease caused by *Mycobacterium leprae*, can involve multiple organ systems; however, renal manifestations are uncommon and often under-recognized, especially in the geriatric population. We report a rare case of a 65-year-old male with untreated multibacillary leprosy who presented with acute kidney injury and respiratory distress. Further evaluation uncovered an uncommon glomerular pathology (necrotising crescentic glomerulonephritis) necessitating both immunosuppressive therapy and multi-drug treatment for leprosy. This case highlights the diagnostic and therapeutic challenges in the geriatric population, where age-related changes, comorbidities, and atypical presentations can delay diagnosis. It underscores the importance of maintaining a high index of suspicion for leprosy and its systemic complications—including glomerulonephritis—especially in older patients, where it is often overlooked. Early diagnosis through renal biopsy facilitated appropriate management in this patient. The case emphasizes the importance of integrated, multidisciplinary, and accessible healthcare approaches for older adults with leprosy, particularly those presenting with multi-organ involvement.

Key words: Multibacillary Leprosy, Older Adults, Necrotising Crescentic Glomerulonephritis, Anti-GBM Disease, Leprosy Related Nephropathy, Multi-drug Therapy

Introduction

Leprosy, also called Hansen's disease, is a chronic infectious disease caused by *Mycobacterium leprae (M. leprae)* which is an obligate intracellular bacillus and acid fast in nature. *M. leprae* thrives at temperatures between 27°C and 30°C, which is why it primarily affects surface areas of the body, such as the skin, peripheral nerves, testicles, and upper airways, while internal organs are less commonly involved (Loscalzo et al 2022). Clinical manifestations of leprosy can vary significantly, depending on the individual's immune response to pathogen, resulting in a spectrum of disease that ranges from tuberculoid to lepromatous leprosy (Gelber & Andres 2024, Silva Junior et al 2015).

Renal complications in adults with leprosy have been reported which include both acute and chronic glomerulonephritis, interstitial nephritis, secondary amyloidosis, and pyelonephritis. However, the exact mechanisms causing renal impairment in leprosy remain uncertain (Silva Junior et al 2015).

[1] Dr P Bhutani, MBBS, Senior Resident
[2] Dr A Kumar, MBBS, Junior Resident
[3] Dr M Dhar, MBBS, MD, Professor and Head
[4] Dr M Pathania, MBBS, MD, Additional Professor
Department of Geriatric Medicine, All India Institute of Medical Sciences Rishikesh, Uttarakhand, 249201, India
Corresponding Author: Dr Parul Bhutani, **Email**: parulbhutani2510@gmail.com

In this report, we present a unique case of lepromatous leprosy presenting as renal failure in an older adult.

Case Report

A 65-year-old male, a chronic reformed smoker, presented with acute onset breathlessness mMRC (modified medical research council) grade II to IV with pink frothy sputum for the last 3 days. It was associated with orthopnea and reduced urine output. There was no history of fever, chest pain, haemoptysis, diaphoresis, lower limb swelling, history of immobilisation, facial puffiness, frothy urine, haematuria, or burning micturition.

On general examination, the patient was thin-built, conscious, and oriented. Pulse rate-64/minute; Blood pressure in supine position-100/66mmHg; O2 Saturation on room air-70 %; Respiratory rate-30/minute; Glasgow Coma Scale-E4V5M6.

During general physical examination, the patient exhibited complete loss of eyebrows and eyelashes, a saddle nose deformity, moderate to severe pallor with grade 2 clubbing present in fingers of both hands. There were multiple ill-defined purpuric lesions of varying shapes and sizes on both legs, localized to the dorsum of the feet and shin areas. He also had few well-defined ulcers ranging from 3x3 cm to 4x5 cm with sloping edges over the malleolus of both feet. There were no other skin lesions present. Peripheral nerve thickening was noted, involving the bilateral greater auricular, ulnar, radial, and peroneal nerves which were non- tender and rope-like in consistency (grade 2 thickening) with loss of pain, touch and temperature in distal parts of upper and lower limbs. All toenails showed yellow to brown discoloration, thickened nail plates, subungual hyperkeratosis, longitudinal and transverse ridging, and a few Beau's lines. Systemic examination was unremarkable.

Blood investigations revealed severe anemia with thrombocytopenia, azotemia, and hyperphosphatemia (Table 1). Urine routine microscopy revealed 2+proteinuria, 8-10 white blood cells/high power field and 20-25 red blood cells/high power field. 24-hour urine protein revealed few dysmorphic red blood cells and granular cast. Chest X-ray was suggestive of pulmonary edema. Patient underwent a session

Table 1: Result of blood investigations of case reported in this study

TABLE 1: BLOOD INVESTIGATION	
Haemoglobin (g/dl)	8.7
Total leucocyte counts	9.8k
Platelet count	25k
Blood Urea (mg/dl) / Creatinine (mg/dl)	220/6.83
Sodium/Potassium (meq/L)	141/4
Calcium/Phosphate (mg/dl)	7.7/6.9
Uric Acid (mg/dl)	9.4
Total / Direct Bilirubin (mg/dl)	0.81/0.17
SGPT/SGOT (U/L)	44/18
ALP/GGT (IU/L)	141/73
Total protein / Serum albumin (g/dl)	5.2/2.6

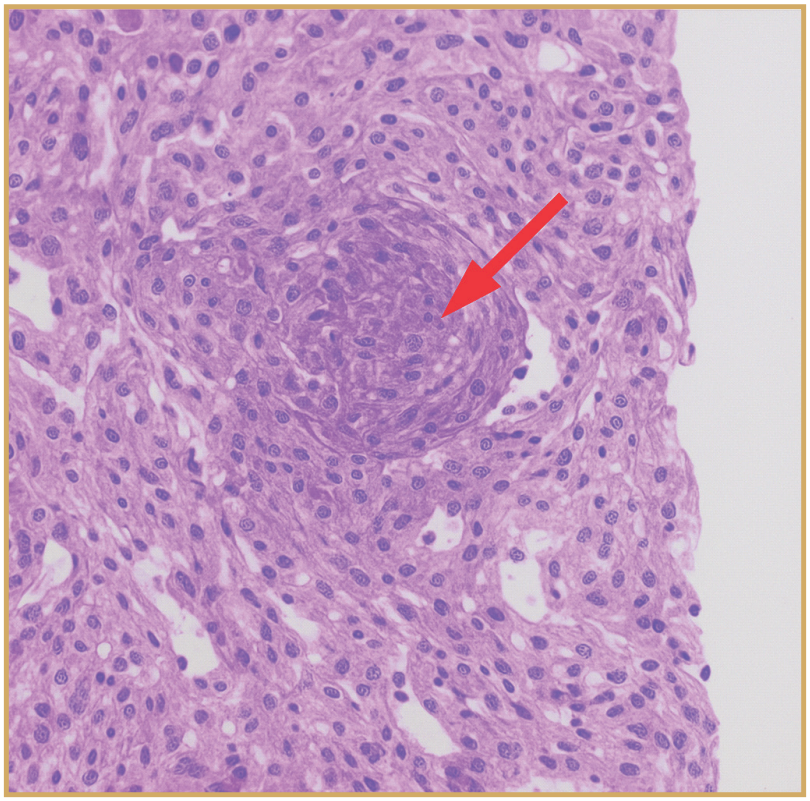

Fig. 1: Renal biopsy showing Necrotising Crescentic Glomerulonephritis with crescents over all sampled glomeruli.

of hemodialysis in view of anuria and non-cardiogenic pulmonary edema. Patient was put on alternate day hemodialysis for persistent anuria. He was investigated for suspected glomerulonephritis with pulmonary involvement and chronic skin lesion. His vasculitis workup in the form of anti-nuclear antibodies, anti-neutrophil cytoplasmic antibodies, cryoglobulins and anti-glomerular basement membrane (anti-GBM) antibodies were negative with reduced complement component 3 (C3) levels and normal complement component 4 (C4) levels. Schistocytes were not found on peripheral blood smear and microangiopathy hemolytic anemia was ruled out. A skin slit smear was done which showed dense dermal infiltrate suggestive of multibacillary lepromatous leprosy. High-resolution computed tomography thorax was suggestive of non-cardiogenic pulmonary edema. Ultrasound whole abdomen revealed bilateral renal parenchymal disease with ascites with bilateral mild pleural effusion. Diagnostic thoracocentesis revealed transudative pleural effusion. He underwent renal biopsy which showed (Fig. 1) features of necrotising crescentic glomerulonephritis with crescents over all sampled glomeruli with acute tubular injury and multifocal chronic tubulo-interstitial inflammation. Direct immunofluorescence (DIF) studies revealed linear staining for IgG along glomerular capillaries, suggestive of anti-GBM disease.

The patient was diagnosed as lepromatous leprosy with nephropathy in the form of crescentic glomerulonephritis with anti GBM disease. He was started on multi-drug therapy (MDT) and planned for immunosuppressive therapy for anti-GBM disease. Patient was planned for bronchoscopy to rule out diffuse alveolar haemorrhage which couldn't be done in view of severe thrombocytopenia. So patient was discharged on MHD and MDT only. Patient was non- compliant to dialysis and finally succumbed to death.

Discussion

Leprosy is an age-old communicable disease from ancient times, known for causing significant morbidity and disability. Over the years, it has been found not only as a curable disease but also as a preventable one. India officially achieved leprosy elimination in 2005, as per the WHO definition (a prevalence of less than 1 case per 10,000 population). However, a substantial number of new cases continue to be reported annually, particularly in developing countries like India.

Leprosy remains one of the Neglected Tropical Diseases (NTDs) listed by the World Health Organization due to its disproportionate impact on impoverished and marginalized communities, who often face social stigma and discrimination which often leads to patient isolation, reluctance to seek timely treatment, and social exclusion, further perpetuating the neglect of this disease.

The renal complications of leprosy were first recognized in the early 20th century through autopsy studies that uncovered glomerulonephritis and tubulointerstitial lesions. Over the period of time, a variety of renal pathologies have been found to be associated with leprosy in adult patients. These include acute and chronic glomerulonephritis, interstitial nephritis, secondary amyloidosis, and pyelonephritis (Silva Junior et al 2015). But acute kidney injury is a rare type of renal impairment in patients of leprosy. When it does occur, it usually results from acute tubular necrosis, drug-induced interstitial nephritis, or, less commonly, crescentic glomerulonephritis (Nigam et al 1986). The precise mechanism by which leprosy leads to kidney impairment is still not fully understood. However, our review of the literature points towards three possible mechanisms, which are linked to reactional states, multibacillary forms of leprosy, and advanced age. Erythema nodosum leprosum (ENL) is one such reactional state in leprosy, characterized by the formation of circulating immune complexes that deposit in vessels and tissues. These immune complexes sometimes result from Hansen's bacilli antigens, which are released into circulation after the initiation of antibiotic therapy. Host antibodies recognize these antigens, forming immune complexes that can either deposit in the glomerulus or develop in situ. However, not all cases of glomerulonephritis in leprosy are linked to ENL, suggesting that leprosy nephropathy may have a multifactorial origin (Silva Junior et al 2015). Other mechanisms which can lead to nephropathy in leprosy includes, direct renal injury by the bacillus with granuloma formation and causing tubulointerstitial lesions that are found in multibacillary leprosy, use of nephrotoxic drugs, renal amyloidosis due to leprosy, skin infections leading to circulatory immune complex causing glomerulonephritis and vasculitis associated with leprosy (Silva Junior et al 2015). Advanced age is also linked to nephropathy in leprosy as one of the mechanisms, but clinically these conditions are under-recognised in older adult population. In this population, leprosy-related kidney disease can substantially increase morbidity

and mortality due to existing health conditions, weakened immunity due to immunosenescence, and age-related kidney decline.

Although several forms of glomerulonephritis have been reported in adults with leprosy, crescentic glomerulonephritis is extremely rare. Only one instance of crescentic glomerulonephritis has been documented in an adult with erythema nodosum leprosum (Nigam et al 1986), but it has not been observed in older adults. Therefore, whenever a patient with renal dysfunction came with underlying history of long-standing skin lesion and nerve involvement, we should suspect leprosy as the cause of renal dysfunction. Suspecting leprosy and doing skin smears is important and needs to be undertaken to clinch the diagnosis of leprosy. In such patient with leprosy with acute kidney injury, it is essential to consider a prompt renal biopsy. This not only enables accurate diagnosis but also guides appropriate management for such acute cases, which may require immunosuppressive treatment, especially in older adults.

In this report, we present a case of leprosy in an older patient who developed acute kidney injury, ultimately diagnosed as crescentic glomerulonephritis caused by anti-GBM disease. Sharma et al (2010) also reported progression from diffuse proliferative to crescentic glomerulonephritis in a 25-year-old young male patient with leprosy. To our knowledge, this is the first reported case of leprosy with anti-GBM disease as systemic manifestation in an older adult, although idiopathic anti-GBM disease has been documented in elderly populations (Silva Junior et al 2015). A Chinese study noted that anti-GBM disease is less frequent in older adults and often presents with severely impaired kidney function. Many elderly patients with this condition are found to have advanced chronic kidney disease (CKD) or require dialysis. Thus, older adults with anti-GBM disease tend to have a poorer renal outlook, and pulmonary involvement is less common compared to younger patients with the same disease (Cui et al 2011).

The most important take home message is that all patients who are suspected cases of leprosy should be examined thoroughly, and tests should be conducted even on slight suspicion of leprosy. If the tests came positive and patients diagnosed as leprosy, MDT should be initiated promptly and completed as required. Additionally, close contacts should be screened for signs and symptoms, with treatment provided for any detected cases. These measures are crucial and help to curtail the transmission of leprosy.

Conclusion

We have presented a case of an older adult with anti-GBM disease with multibacillary leprosy. This case report highlights the importance of considering glomerular involvement when managing acute kidney injury in leprosy patients. Older adults with anti-GBM disease tend to have a poorer renal prognosis. Timely diagnosis might help in planning the management of such patients to preserve their renal function.

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