



Renal Lesions in Leprosy

Dear Editor,

Leprosy is caused by *Mycobacterium leprae*, an intracellular organism that affects the skin and peripheral nerves. It causes chronic debilitating sequelae and systemic complications.¹ There are limited data on renal manifestations of *M. leprae* infection. Here, we describe the pathological findings in leprosy patients with renal involvement.

Sixteen renal biopsies of patients diagnosed with leprosy were retrieved from the biopsy database at Renopath, Center for Renal and Urological Pathology (2013-2025). Clinical and laboratory data at the time of biopsy was collected. For routine diagnostic evaluation, paraffin-embedded sections were cut and stained with hematoxylin and eosin, periodic acid-Schiff, Jones methenamine silver, and Masson's trichrome. All cases were stained with Congo red and modified Ziehl-Neelsen. Biopsies underwent routine immunofluorescence staining for IgG, IgM, IgA, C3, C1q, and κ and λ light chains. Two pathologists (J.P.K.S. and A.A.K) independently reviewed the histopathologic findings.

The cohort of patients with *M. leprae* infection who underwent kidney biopsy included 11 males and five females. The median age at the time of renal biopsy was 48.5 years (range: 33,63). Subnephrotic and nephrotic-range proteinuria were observed in six and seven patients, respectively. Nine patients presented with AKI, and two had hematuria. The mean serum creatinine at presentation was 3.15 ± 2.65 mg/dL. One patient required hemodialysis. Four patients had hypertension, and one had a history of diabetes mellitus.

The pathological diagnoses of the 16 cases have been enumerated in Table 1. Most patients had glomerular disease. Diagnoses included infection-associated glomerulonephritis (IRGN) (n=6), membranous nephropathy (MN) (n=3), C3 GN (n=1), AA amyloidosis (n=1), focal segmental glomerulosclerosis (FSGS) with concurrent acute tubular injury (ATI) (n=1), minimal change disease with concurrent acute interstitial nephritis (n=1), acute pyelonephritis (n=1), intravascular hemolysis-associated ATI (n=1), and necrotizing vasculitis (due to Lucio phenomenon, n=1). In patients with IRGN, immunofluorescence revealed C3 and IgG deposits in glomerular capillary loops and/or mesangium. Four biopsies revealed endocapillary proliferative GN [Figure 1a, 1b]. One case of resolving IRGN with mesangial hypercellularity was noted. IgA-dominant IRGN with C3 and IgA deposits was found in another. None of the biopsies exhibited crescents or necrotizing glomerular lesions. No biopsy showed granuloma formation. Interstitial fibrosis and tubular atrophy were absent to mild in most cases and of moderate degree in two biopsies. Necrotizing

Table 1: Histopathological diagnosis in renal biopsies from patients with leprosy

Etiology	Pathologic diagnosis	
Infection	Infection-related glomerulonephritis/IRGN	6 (37.5)
	IRGN (4)	
	IgA dominant IRGN (1)	
	Resolving IRGN (1)	
Podocytopathy + Tubulointerstitial involvement	Acute pyelonephritis	1 (6.2)
	Focal segmental glomerulosclerosis/Acute tubular injury	1 (6.2)
	Minimal change disease/Acute interstitial nephritis	1 (6.2)
Immune complex Complement	Membranous nephropathy	3 (18.7)
	C3 glomerulonephritis	1 (6.2)
Deposits	AA Amyloidosis	1 (6.2)
	Drug (Rifampicin)	1 (6.2)
Drug (Rifampicin)	Intravascular hemolysis associated acute tubular injury	1 (6.2)
	Lucio phenomenon	1 (6.2)
Lucio phenomenon	Necrotizing vasculitis	1 (6.2)
Total		16

AA: Amyloid A, IRGN: Infection-related glomerulonephritis.

vasculitis and arteriosclerosis were present in one and four cases, respectively. In patients with membranous nephropathy, one was PLA2R positive [Figure 1c] and two were negative for PLA2R and NELL1.

The patient with hemolysis-associated ATI had a history of rifampicin use, which was discontinued. Two patients were on multidrug therapy for multibacillary leprosy, and one for the paucibacillary form at the time of biopsy. Two patients were on steroids for erythema nodosum leprosum (ENL). One patient showed Lucio phenomenon with systemic vasculitis, mixed connective tissue disorder and severe pulmonary hypertension.

Renal involvement in patients with leprosy has been described from around the world.¹⁻³ *Lepra* bacilli are infrequently demonstrated in renal biopsies.⁴ Factors responsible for renal injury in leprosy include circulating immune complexes, infections, drug toxicity, disease duration, and *lepra* reactions.⁵

IRGN was the most common cause of renal failure in our cohort (n=6). Three of these patients had non-healing trophic ulcers of the feet. Infections and neoplastic conditions have been reported in association with GN in leprosy.⁴ However, many studies have observed GN more commonly in lepromatous leprosy and ENL. ENL, a type II *lepra* reaction, has an immune-mediated inflammatory pathogenesis.⁶ We found acute pyelonephritis [Figure 1d] and IgA-dominant IRGN in two patients with ENL.

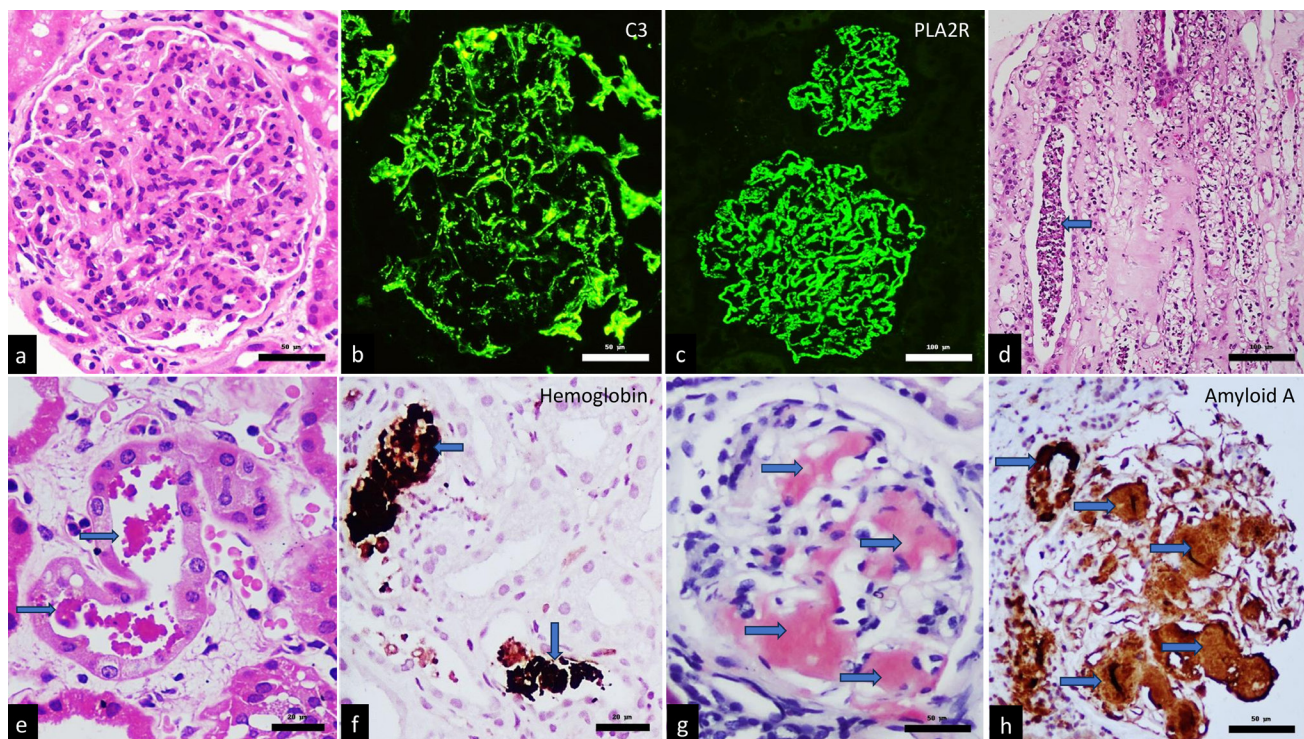


Figure 1: (a) Endocapillary hypercellularity and neutrophilic infiltration in a patient with IRGN (40X, hematoxylin and eosin stain), (b) C3 immunofluorescence stain shows intense granular staining along the capillary loops and mesangium in a case of IRGN (40X, C3 immunofluorescence), (c) PLA2R immunostain is positive on the glomerular capillary loops in a granular pattern in a patient with membranous nephropathy (20X, PLA2R immunostain), (d) acute pyelonephritis with pus cell cast (blue arrow) in medullary tubules (10X, hematoxylin and eosin stain), (e) densely eosinophilic granular to globular tubular pigment cast (blue arrows) in a patient with rifampicin associated intravascular hemolysis (40X, hematoxylin and eosin stain), (f) immunohistochemical stain for hemoglobin is strongly positive on the pigment casts (blue arrows), confirming the diagnosis of intravascular hemolysis associated tubular injury (40X, hemoglobin immunohistochemical stain), (g) amyloid deposits (blue arrows) showing Congo red positivity (40X, Congo red stain) and (h) amyloid A immunohistochemical stain is positive (blue arrows) in the amyloid deposits (40X, amyloid A immunohistochemical stain).

Lucio phenomenon is a rare vasculonecrotic reaction occurring in untreated or inadequately treated leprosy. Endothelial injury due to a heavy bacterial load has been proposed as the cause of vasculitis or thrombosis in vessels. Histologically, endothelial proliferation, thrombosis, and vasculitis have been reported in dermal biopsies.⁷ Acid-fast lepra bacilli have been demonstrated in the endothelial cells of dermal vessels.⁸ The kidney biopsy in our patient with Lucio phenomenon showed necrotizing vasculitis. No acid-fast bacilli were identified on modified Ziehl-Neelsen staining.

Drug toxicity can cause renal failure. Rifampicin is known to cause hemoglobin casts due to intravascular hemolysis.⁹ One case had granular to ropy pigment casts with associated ATI [Figure 1e]. Immunostaining for hemoglobin was positive in the tubular casts [Figure 1f].

Amyloidosis associated with leprosy has been described by Nakayama *et al.*⁴ Repeated complications related to leprosy, such as lepra reactions and trophic ulcers, are considered possible triggers for amyloid fibril production [Figure 1g, 1h].

Three MN cases were reported in our series. Our spectrum also included one case each of minimal change disease and FSGS. These presented with proteinuria and AKI due to concurrent tubulointerstitial pathology [Table 1]. Podocytopathy and C3 GN in leprosy were less evident in the literature. Given the limited number of cases in our study, it remains difficult to interpret these as primary glomerular pathologies or lesions secondary to leprosy.

To conclude, the spectrum of renal lesions in leprosy is diverse. IRGN is the most common cause of renal failure.

Conflicts of interest: There are no conflicts of interest.

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Atypical Hemolytic Uremic Syndrome in Patients with Pregnancy Related Renal Cortical Necrosis: An Observational Study From Western Part of India

Dear Editor,

Renal cortical necrosis (RCN) is the most catastrophic form of AKI, associated with increasing maternal morbidity and mortality. Prolonged low renal perfusion causes ischemic cortical injury via spasm, clotting, and capillary damage. Obstetric RCN is an important sequela of pregnancy-related AKI, particularly in developing countries. and is most linked to massive blood loss from conditions such as placenta previa, abruptio placentae, post-partum hemorrhage, amniotic fluid embolism, and septic abortion.¹

Of late, complement-mediated thrombotic microangiopathy (CM-TMA) or atypical hemolytic uremic syndrome (aHUS) are distinct causes of AKI occurring in late pregnancy or postpartum.² In this study, we describe the clinico-epidemiological profile and outcomes of pregnancy-related RCN, emphasizing the potential contribution of aHUS, diagnosed clinically or through renal histology, and its impact on renal recovery in postpartum RCN patients from the western part of India. Materials and methods of the study have been provided in the Supplementary File.

A total of 20 patients with pregnancy-related AKI and biopsy-proven RCN were followed. The mean age at presentation was 28.1 (range: 19-42) years. Preeclampsia was seen in four patients (20%). Post-partum hemorrhage occurred in six patients (30%). A majority of patients (17/20) had an institutional delivery. The time from

first medical contact to referral to our centre, as well as the delay in referral, were associated with an adverse outcome [Supplementary Table S1]. All patients presented in an anuric state after delivery and were initiated on hemodialysis. About 65% had sepsis; 17 (85%) exhibited classical microangiopathic hemolytic anemia (MAHA). Of these, 13 had histological changes of TMA on kidney biopsy. Five patients had low C3 levels, while five had normal C3 levels [Supplementary Table S2]. Nine patients were treated with plasma exchange. Patients with diffuse RCN with or without TMA on biopsy remained dialysis-dependent at 3 months. No deaths were observed during follow-up; one patient was lost to follow-up. Neonatal outcomes included intrauterine death (9), molar pregnancy (1), and live birth (10).

We assessed the relationship of various clinical and laboratory parameters with dialysis dependency at 3 months [Table 1]. In multivariate regression analysis, diffuse RCN was a statistically significant independent predictor ($p=0.006$) of dialysis dependency at 3 months. Among the factors associated with the diffuse RCN, only age > 30 years had a significant association ($p=0.01$) [Supplementary Table S3]. Both clinical HUS and histological TMA were not associated with diffuse cortical necrosis ($p=1.00$ and $p=0.16$, respectively).

The median age at presentation was 28 years, similar to the findings from the systematic review by Gupta *et al.*³