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## Leprosy-Associated Renal Involvement: Clinical Manifestations and Histopathological Insights

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### ABSTRACT

Leprosy, a chronic infectious disease caused by *Mycobacterium leprae*, continues to pose a substantial public health challenge globally. Despite significant progress, leprosy remains prevalent in certain regions, with India accounting for a significant portion of the burden. This study delves into the less-explored realm of renal involvement in leprosy an enduring global health challenge caused by *Mycobacterium leprae*. Despite being primarily recognized for skin and nerve effects, leprosy can also impact the kidneys, leading to diverse clinical indicators like proteinuria, hematuria, altered blood parameters and reduced glomerular filtration rate. Histopathological analysis reveals various renal lesions such as glomerulonephritis, nephrosclerosis and tubulointerstitial nephritis often observed during reactive phases and within the lepromatous spectrum of the disease. While the direct invasion of renal tissue is not proven, these findings emphasize the importance of holistic patient care, early diagnosis and tailored treatment. Recognizing the interplay between leprosy and renal function contributes to enhanced patient management and informs comprehensive public health strategies for tackling leprosy's global impact.

## INTRODUCTION

Leprosy, caused by *Mycobacterium leprae*, remains a significant global public health concern, with approximately 2.19 lakh new cases reported in 2011, as documented by the WHO weekly epidemiological record<sup>[1]</sup>. Despite the world health organization's (WHO) efforts to achieve elimination targets, India bore a substantial burden, contributing 58.1% of the world's leprosy cases in the same year. Leprosy is characterized by its slow-growing nature and its capacity to infiltrate various tissues, including the skin, peripheral nerves, mucosa and eyes<sup>[2]</sup>. The incubation period between infection and clinical manifestation typically ranges from 2-20 years, affecting individuals of all ages and genders. Timely diagnosis and treatment with multidrug therapy (MDT) play a pivotal role in disease elimination as untreated leprosy can lead to irreversible damage to the skin, nerves, limbs and ocular structures. Leprosy can also entail multisystem involvement<sup>[3]</sup>.

While *Mycobacterium leprae* is not conventionally associated with direct invasion of the renal parenchyma, emerging evidence suggests significant renal functional and structural impairment in leprosy patients. Pioneering autopsy studies by Mitsuda and Ogawa in 1937 marked the first documentation of renal involvement in leprosy. They reported diverse nephritis patterns in leprosy patients and underscored renal failure as a prevalent cause of mortality<sup>[4]</sup>. Renal aberrations in leprosy patients manifest in various forms, including proteinuria, hematuria, urinary casts, elevated blood urea and serum creatinine levels and reduced glomerular filtration rates (GFR). Histopathological examinations reveal a spectrum of lesions, encompassing glomerulonephritis (GN), nephrosclerosis, tubulointerstitial nephritis, granulomas and amyloidosis. Renal lesions are more frequently reported during the reactive phase, particularly in erythema nodosum leprosum (ENL) and within the lepromatous spectrum of the disease<sup>[5]</sup>.

The present study aims to investigate the extent of renal involvement in patients diagnosed with leprosy, focusing on two primary parameters, urinary abnormalities and estimated glomerular filtration rate (eGFR). Leprosy is a chronic infectious disease caused by *mycobacterium leprae*, recognized for its dermatological and neurological manifestations<sup>[6]</sup>. However, emerging evidence suggests potential renal implications that have not been comprehensively explored. By assessing urinary abnormalities and eGFR, we seek to elucidate the scope and severity of renal dysfunction in leprosy patients, shedding light on this underrecognized aspect of the disease<sup>[7]</sup>.

Furthermore, this study aims to characterize the spectrum of renal histopathological lesions present in leprosy patients who exhibit renal involvement. Through meticulous histopathological analysis, we

intend to identify and categorize various renal lesions, including glomerulonephritis, nephrosclerosis and tubulointerstitial nephritis, among others. The insights gleaned from this study could facilitate targeted interventions and management strategies aimed at mitigating renal complications in leprosy patients. In the pursuit of leprosy elimination as a global health goal, comprehensive investigations into renal manifestations in leprosy offer a unique perspective that could shape the course of disease control policies and improve patient outcomes.

## MATERIALS AND METHODS

The study was conducted within the Department of Medicine, Dermatology (Leprosy Clinic) and Pathology at the University College of Medical Sciences and Guru Teg Bahadur Hospital in Delhi. All study participants, aged 18 years and above were selected from the Medicine Outpatient Department (OPD), Leprosy Clinic and patients admitted to the Medicine and Dermatology wards. Informed written consent was obtained from each of the study participant and they were also provided with comprehensive patient information sheets. Ethical clearance for human research was secured from the Institutional Ethical Committee.

The study period began from October 2012 to April 2014 and it followed a cross-sectional design. A total of 100 consecutive subjects meeting the defined criteria were enrolled for the study.

The case definition of leprosy encompassed patients with active disease condition who exhibited two or more of the following cardinal signs, hypopigmented or erythematous skin lesions accompanied by definite sensory loss or impairment, involvement of peripheral nerves characterized by distinct thickening and/or tenderness with sensory impairment and demonstration of acid-fast bacilli (AFB) from lesions through slit skin smear or skin biopsy. Leprosy cases were classified according to both the NLEP and Ridley Jopling systems<sup>[8]</sup>.

A detailed medical and treatment history was taken. General physical, dermatological and systemic examination was done. Various factors which may affect the development of renal function abnormalities like duration of the disease, presence of reactions and drugs were taken into consideration. Routine biochemical tests were conducted at the Hospital Laboratory Services.

The estimated glomerular filtration rate (eGFR) was calculated using the Modification of Diet in Renal Disease (MDRD) study equation and the CKD-EPI equation.

**Renal biopsy:** Renal biopsy, a crucial diagnostic tool, holds specific indications and contraindications that guide its judicious application. Indications for renal

biopsy are abnormal renal function parameters, evident when the estimated glomerular filtration rate (eGFR) falls below  $60 \text{ mL min}^{-1} 1.73 \text{ m}^{-2}$ <sup>[9]</sup>. However, certain conditions preclude the utilization of renal biopsy. These contraindications involve an inability to procure informed consent, a predisposition to bleeding diathesis, ultrasonographically confirmed contracted kidneys (size <8 cm), pregnancy, possession of a solitary kidney and the presence of hemodynamic instability.

The renal biopsy procedure itself entails the extraction of two biopsy cores subsequent to obtaining comprehensive written consent. These extracted cores are then submitted to the department of pathology for meticulous evaluation. One of the biopsy cores is meticulously placed in 10% buffered formalin to facilitate histological examination under light microscopy. Staining techniques, including H and E, PAS, Silver Methenamine and Trichrome are meticulously employed to discern critical structural and compositional details. Meanwhile, the second biopsy core is preserved in Michel's media, a specialized medium for direct immunofluorescence study. This facet of the procedure aims to detect pertinent immunological markers such as IgG, IgA, IgM, C3 and Fibrinogen. This comprehensive approach to renal biopsy ensures a thorough assessment of both structural and immunological aspects, thus contributing to a more nuanced understanding of renal health and potential pathologies.

The statistical analysis involved calculating the frequency and percentage of renal lesions using SPSS version 20.0.

## RESULTS

**Demographic characteristics:** The study included a total of 100 patients with an age range of 18-75 years and a mean age of  $35.60 \pm 13.04$  years (Table 1). The patients were divided into two groups based on NLEP classification, 65 patients in the multibacillary (MB) group and 35 patients in the paucibacillary (PB) group. The mean ages of the MB and PB groups were

comparable ( $36.09 \pm 13.15$  and  $34.69 \pm 12.9$  years, respectively). Similarly, when classified according to the Ridley Jopling classification, the mean ages of LL, BL, BB and BT groups were  $38.41 \pm 12.48$ ,  $35.62 \pm 13.13$ ,  $41.67 \pm 17.92$  and  $33.94 \pm 12.6$  years, respectively. (Table 1 and 2).

The gender distribution showed that 61% of the patients were males and 39% were females (Fig. 1). Body mass index (BMI) ranged from  $16.4$ - $33.7 \text{ kg m}^{-2}$ , with a mean BMI of  $21.96 \pm 3.01 \text{ kg m}^{-2}$  for the entire study group. Similar to age, BMI was compared among the groups based on NLEP and Ridley Jopling classifications and was found to be comparable (Table 1 and 2).

The duration of the disease varied from 1-180 months, with a median duration of 18 months. MB and PB groups had median disease durations of 12 and 18 months, respectively. The distribution of disease duration among the Ridley Jopling groups was also found to be comparable (Table 3 and 4).

**Laboratory parameters:** Blood urea levels were examined and the mean blood urea was  $25.84 \pm 8.97 \text{ mg dL}^{-1}$  for the entire study group. When categorized based on NLEP classification, mean blood urea levels were comparable between the MB and PB groups. Similar results were observed when classified based on Ridley Jopling's classification (Fig. 2 and 3).

Serum creatinine levels were also analyzed and the mean serum creatinine was found to be  $0.91 \pm 0.20 \text{ mg dL}^{-1}$ . Just like blood urea, serum creatinine levels were comparable between the different groups, regardless of the classification used (Fig. 4 and 5).

**eGFR:** eGFR was calculated using both MDRD and CKD EPI equations. Using MDRD equation, mean eGFR of study group was found to be  $94.99 \pm 23.14 \text{ mL min}^{-1} 1.73 \text{ m}^{-2}$ . When classified according to NLEP classification, it was found that mean eGFR (MDRD) of MB group and PB group in the study was  $97.7 \pm 26.7$  and  $92.28 \pm 22.5 \text{ mL min}^{-1} 1.73 \text{ m}^{-2}$ , respectively. The difference in mean eGFR of the two groups was statistically not significant (Table 5 and Fig. 6). When

Table 1: Age and BMI distribution among the study subjects according to NLEP classification

Distribution	MB (n = 65)	PB (n = 35)	Total (n = 100)	p-value
<b>Age (years)</b>				
Range	18-75	18-60	18-75	-
Mean $\pm$ SD	$36.09 \pm 13.15$	$34.69 \pm 12.9$	$35.6 \pm 13$	0.61
<b>BMI (<math>\text{kg m}^{-2}</math>)</b>				
Range	16.8-33.7	16.4-30.6	16.4-33.7	-
Mean $\pm$ SD	$21.86 \pm 2.96$	$22.14 \pm 3.16$	$21.99 \pm 3.02$	0.66

Table 2: Age and BMI distribution among the study subjects according to Ridley Jopling's classification

Distribution	LL (n = 17)	BL (n = 26)	BB (n = 6)	BT (n = 51)	Total	p-value
<b>Age (years)</b>						
Range	18-60	18-65	27-75	18-60	18-75	-
Mean $\pm$ SD	$38.41 \pm 12.48$	$35.62 \pm 13.13$	$41.67 \pm 17.92$	$33.94 \pm 12.6$	$35.6 \pm 13.04$	0.410
<b>BMI (<math>\text{kg m}^{-2}</math>)</b>						
Range	17.5-33.7	16.8-27.5	19.8-30.6	16.4-28.4	16.4-33.7	-
Mean $\pm$ SD	$22.62 \pm 3.85$	$21.66 \pm 2.93$	$23.43 \pm 3.80$	$21.72 \pm 2.65$	$21.96 \pm 3.01$	0.428

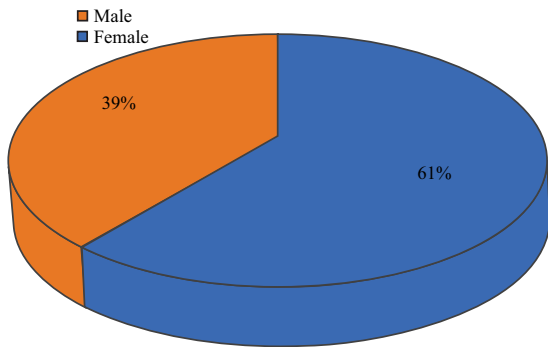


Fig. 1: Sex distribution of the study subjects

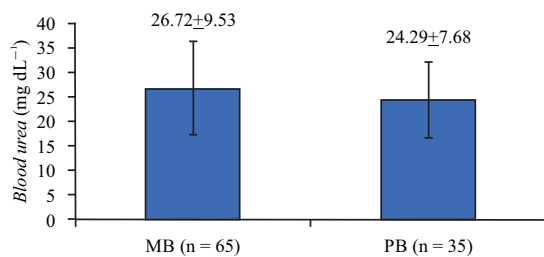


Fig. 2: Distribution of blood urea according to NLEP classification

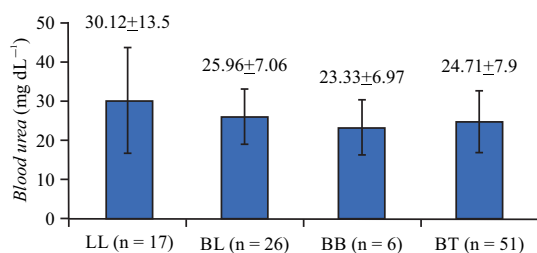


Fig. 3: Distribution of blood urea according to Ridley Jopling classification

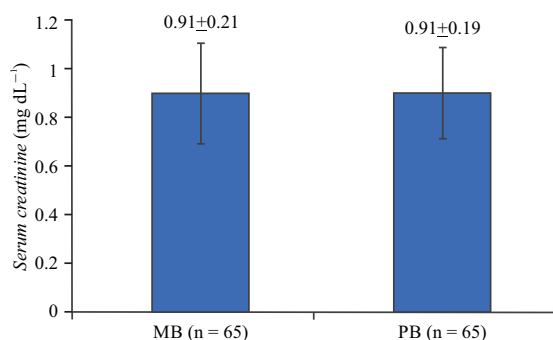


Fig. 4: Distribution of serum creatinine according to NLEP classification

classified according to Ridley Jopling classification, it was found that the mean eGFR of LL, BL, BB and BT groups was  $100.62 \pm 31.47$ ,  $97.14 \pm 21.12$ ,  $90.11 \pm 26.46$  and  $92.60 \pm 20.69$  mL min<sup>-1</sup> 1.73 m<sup>-2</sup>, respectively. The difference in mean eGFR of the groups was statistically not significant (Table 5 and Fig. 6).

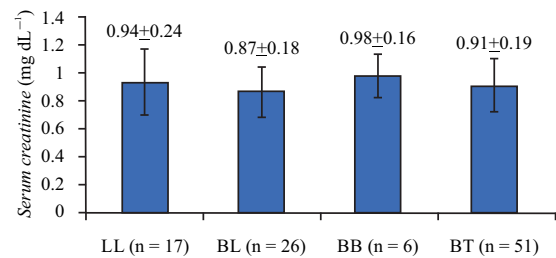


Fig. 5: Distribution of serum creatinine according to Ridley Jopling classification

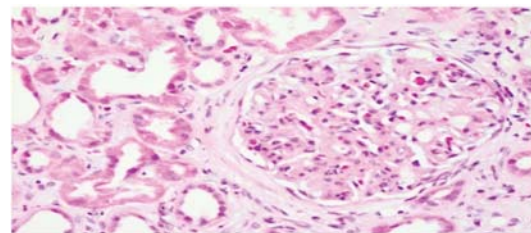


Fig. 6: H and E stained section of kidney biopsy showing mild to moderate increase in mesangial matrix and increase in cellularity with neutrophilic infiltration suggestive of mesangioproliferative glomerulonephritis

Table 3: Duration of disease among the study subjects according to NLEP classification

Duration	MB	PB	Total
Range (months)	1-156	3-180	1-180
median	12	18	18

Using the CKD EPI equation, the mean eGFR of the study group was found to be  $97.89 \pm 20.10$  mL min<sup>-1</sup> 1.73 m<sup>-2</sup>. When classified according to NLEP classification, it was found that the mean eGFR of the MB group was  $99.03 \pm 20.76$  mL min<sup>-1</sup> 1.73 m<sup>-2</sup>, while for the PB group, it was  $96.42 \pm 20.42$  mL min<sup>-1</sup> 1.73 m<sup>-2</sup>. The difference in mean eGFR of the two groups was statistically not significant. When classified according to Ridley Jopling classification, it was found that the mean eGFR of LL, BL, BB and BT groups was  $98.83 \pm 24.50$ ,  $100.17 \pm 18.25$ ,  $91.05 \pm 23.33$  and  $97.21 \pm 19.44$  mL min<sup>-1</sup> 1.73 m<sup>-2</sup>, respectively. However, the difference in mean eGFR of the groups was statistically not significant (Table 6).

Using the MDRD equation, it was observed that out of 100 leprosy patients, 2 patients had eGFR <60, both of these patients were in the multibacillary group using NLEP classification and one was in the LL group and one in the BL group as per Ridley Jopling classification. About 47 patients had eGFR in the range 60-89 while 51 patients had eGFR >90 mL min<sup>-1</sup> 1.73 m<sup>-2</sup>. In accordance with the defined criteria, 2% of the patients were found to have renal involvement in the form of decreased eGFR

Table 4: Duration of disease among the study subjects according to Ridley Jopling classification

Duration	LL	BL	BB	BT	Total
Range (months)	2-108	1-156	6-180	2-96	1-180
Median	24	15	18	18	

Table 5: Distribution of eGFR using NLEP classification

Variables	Mean±SD			p-value
	Total	MB (n = 65)	PB (n = 35)	
eGFR MDRD ( $\text{mL min}^{-1} 1.73 \text{ m}^{-2}$ )	94.9±25.3	97.7±26.7	92.28±22.5	0.37
eGFR CKD-EPI ( $\text{mL min}^{-1} 1.73 \text{ m}^{-2}$ )	97.1±20.6	99.03±20.76	96.42±20.42	0.60

Table 6: Distribution of eGFR using Ridley Jopling classification

Variables	Mean±SD				p-value
	LL (n = 17)	BL (n = 26)	BB (n = 6)	BT (n = 51)	
eGFR MDRD ( $\text{mL min}^{-1} 1.73 \text{ m}^{-2}$ )	100.62±31.47	97.14±21.12	90.11±26.46	92.60±20.69	0.570
eGFR CKD-EPI ( $\text{mL min}^{-1} 1.73 \text{ m}^{-2}$ )	98.83±24.50	100.17±18.25	91.05±23.33	97.21±19.44	0.777

Table 7: Staging of kidney disease in patients with renal involvement according to MDRD and CKD EPI equation

Stage of kidney disease	eGFR ( $\text{mL min}^{-1} 1.73 \text{ m}^{-2}$ )	MDRD		CKD EPI	
		No.	Percentage	No.	Percentage
Stage 1	>90	51	51	64	64
Stage 2	60-89	47	47	35	35
Stage 3	30-59	2	2	1	1
Stage 4	15-29	0	0	0	0
Stage 5	<15	0	0	0	0

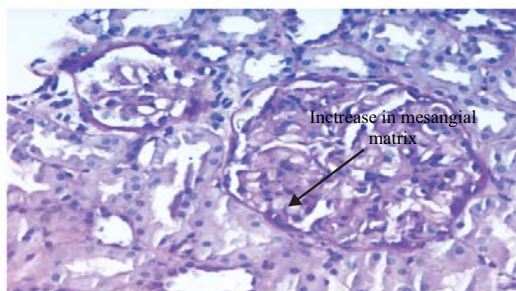


Fig. 7: PAS stain of kidney biopsy showing an increase in mesangial matrix

(Table 7). Using the CKD EPI equation, it was observed that out of 100 leprosy patients, 1 patient had eGFR  $<60 \text{ mL min}^{-1} 1.73 \text{ m}^{-2}$ , this patient was in the MB group as per NLEP classification and LL group as per Ridley Jopling Classification. About 35 patients had eGFR in the range 60-89 while 64 patients had eGFR  $>90 \text{ mL min}^{-1} 1.73 \text{ m}^{-2}$ . In accordance with the defined criteria, 1% of the patients were found to have renal involvement in the form of decreased eGFR (Table 7).

**Histopathological analysis:** It was conducted on a subset of patients meeting the criteria for kidney biopsy, with one patient ultimately undergoing the procedure due to consent-related issues. The established criteria encompassed estimated glomerular filtration rate (eGFR) below  $60 \text{ mL min}^{-1} 1.73 \text{ m}^{-2}$ . The kidney biopsy findings of the analyzed patient revealed various notable aspects. Microscopic examination unveiled the presence of 10 glomeruli,

appearing normal-sized and featuring an unremarkable glomerular basement membrane. However, a mild to moderate elevation in the mesangial matrix was observed across nearly all glomeruli, accompanied by increased cellularity characterized by neutrophilic infiltration. Tubular observations demonstrated an unremarkable status with occasional mild inflammation manifesting as scattered lymphocytes and neutrophils. Arteries were found to be unremarkable, while arterioles exhibited minimal thickening. Notably, staining for acid-fast bacilli (AFB) lepra exhibited a 1+ result.

## DISCUSSIONS

Leprosy remains an important public health problem in India, which bears the maximum burden of disease in the world<sup>[10]</sup>. Visceral involvement in leprosy was reported as early as in 1895 by Hansen and Looft<sup>[11]</sup>. *Mycobacterium leprae* ordinarily does not invade renal parenchyma but considerable functional and structural impairment of kidneys is well known in leprosy patients<sup>[12]</sup>.

There is wide variation in the incidence and type of histologic lesions in kidneys reported from different geographical areas<sup>[13]</sup>. Renal involvement may occur in the form of glomerulonephritis, amyloidosis and tubular functional defects<sup>[14]</sup>. The exact pathogenesis of renal involvement remains uncertain. However, glomerular lesions have been attributed to immune complex formation secondary to the episodes of ENL, which is supported by visualization of immune complexes in renal biopsy specimens and decreased serum complement levels as well<sup>[15]</sup>. The development of amyloidosis is held to be secondary to chronic



granulomatous reactions caused by *Mycobacterium leprae*, which usually manifests as significant proteinuria and may progress to chronic kidney disease and may ultimately lead to fatal outcomes<sup>[16]</sup>.

Leprosy patients with renal involvement present a large spectrum of clinical manifestations which range from asymptomatic patients to classical nephrotic syndrome. Most of the patients have only asymptomatic urinary abnormalities<sup>[17]</sup>. Patients with early involvement are at high risk for progression to end-stage renal disease (ESRD), a condition requiring renal replacement therapy<sup>[18]</sup>. The enormous cost of treatment leads to a large burden on the healthcare system, particularly in developing countries. Due to the asymptomatic nature in the early course of the disease, CKD is not frequently detected until the late stages, resulting in lost opportunities for prevention. Progress to renal failure or other adverse outcomes could be prevented or delayed through early detection and treatment<sup>[19]</sup>.

There exists a lot of disparity and inadequacy in the available literature. Also, the nature of the disease is incompletely understood. Hence, to understand the nature, distribution and risk factors of renal involvement in leprosy patients, the present study was designed to estimate the prevalence of renal involvement in leprosy patients to correlate it with the type and duration of leprosy and to study the spectrum of histopathological lesions in leprosy patients with renal involvement.

**Baseline characteristics:** A total of 100 consecutive skin biopsy-proven cases of leprosy, 18 years or above attending medicine OPD, leprosy clinic and the patients admitted in medicine and dermatology wards were recruited. About 61 of our study subjects were male while 39 were female. The mean age among our study subjects was  $35.60 \pm 13.04$  years and it ranged between 18 and 75 years. Mean age of various groups was comparable when study group was classified by NLEP and Ridley Jopling classification. Most of our study subjects had normal BMI (mean BMI  $21.96 \pm 3.01 \text{ kg m}^{-2}$ ).

The duration of disease in our study population varied from 1-180 months and the median duration of disease was 18 months. Comparing to mean duration of disease of  $2.85 \pm 2.45$  years in the study conducted by Aggarwal *et al.*<sup>[20]</sup>. Our study group had relatively shorter duration of disease of the patients were receiving multi drug therapy (MDT), rest of them were newly diagnosed cases who were recruited before the initiation of MDT. Multidrug therapy, as recommended by the world health organization includes rifampicin and dapsone for paucibacillary patients and rifampicin, dapsone and clofazimine for multibacillary patients. The patients with ENL were treated with corticosteroids and/or thalidomide<sup>[21]</sup>.

**Renal involvement in leprosy patients:** In the present study, eGFR (both by MDRD and CKD EPI equations) were used to define renal involvement in leprosy patients.  $\text{eGFR} < 60 \text{ mL min}^{-1} 1.73 \text{ m}^{-2}$  were set as the criteria to define renal involvement.

Overall, renal involvement in 100 consecutive patients of leprosy in the present study was found to be 19%. Out of these 19 patients, 17 had renal involvement in the form of micro/macroalbuminuria, one had decreased eGFR both by MDRD and CKD EPI equation, while one had decreased eGFR only by MDRD equation. The number of patients with eGFR in the range 60-89 classified as stage 2 CKD was found to be 47 and 36 as calculated with MDRD and CKD EPI equation, respectively.

**Glomerular filtration rate (GFR):** In the present study, eGFR was calculated both by MDRD and CKD EPI equations,  $\text{eGFR} < 60 \text{ mL min}^{-1} 1.73 \text{ m}^{-2}$  was defined as the criteria for renal involvement. Patients having renal involvement were classified into various stages of CKD. Taking into account the MDRD equation, out of 100 study subjects, we found only 2 with renal involvement. They were classified into stage 3 CKD and both of them belonged to MB group as per NLEP classification while one was in LL and one in BL as per Ridley Jopling classification, thus strengthening the presence of renal involvement with higher bacillary load in leprosy patients. One of these two patients did not show renal involvement when CKD EPI equation was used for eGFR calculation. The other patient was in MB group and LL group as per NLEP and Ridley Jopling classification respectively. Also we observed 47 patients in stage 2 CKD ( $\text{eGFR} 60-90 \text{ mL min}^{-1} 1.73 \text{ m}^{-2}$ ) while using MDRD equation and 35 patients in same while using CKD EPI equation.

Decreased GFR as estimated by endogenous creatinine clearance or by calculation of eGFR has repeatedly been observed in patients of leprosy. Aggarwal *et al.*<sup>[20]</sup> reported decreased GFR (estimated by creatinine clearance) in 8 (26.66%) patients out of 30. Out of these, 4 were in multibacillary group and 4 in paucibacillary group. In a study of 59 patients by Oliveira *et al.*<sup>[22]</sup>, they reported GFR (estimated by MDRD equation)  $< 80 \text{ mL min}^{-1} 1.73 \text{ m}^{-2}$  in 30 (50%) patients. They found that MB patients had lowest GFR in comparison with the controls and PB patients. Higher prevalence of decreased GFR in these studies as compared to ours may be explained by the lower cut off for defining decreased GFR in our study. In a study, Peter *et al.*<sup>[23]</sup> 17 lepromatous and 13 non lepromatous patients of leprosy reported decreased creatinine clearance in 10 non lepromatous and all lepromatous patients. Thus, it may be concluded that renal involvement in the form is decreased eGFR is common in leprosy patients and the prevalence increases as the bacillary status of the disease increases.

## CONCLUSION

The occurrence of renal complications among leprosy patients is a noteworthy phenomenon, with our study revealing that 19% of the examined subjects exhibited renal involvement, characterized by either micro/macro albuminuria or a reduction in estimated glomerular filtration rate (eGFR)<sup>[24]</sup>. Notably, this renal involvement was more prevalent within the multibacillary group in accordance with the National Leprosy Eradication Program (NLEP) classification. Similarly, as per the Ridley Jopling classification, the LL and BL groups displayed a higher incidence of renal involvement. A particularly intriguing observation was the heightened frequency of renal complications in patients experiencing erythema nodosum leprosum (ENL)<sup>[25]</sup>.

Remarkably, a positive correlation between the duration of the disease and the likelihood of renal involvement was evident, indicating a potential progressive impact<sup>[26]</sup>. However, it is important to note that no significant association between drug regimens and renal involvement was discerned, suggesting the need for further investigation.

The underlying mechanisms driving renal involvement appear to be multifaceted. Immunological factors came to the fore, with evidence of immune complex formation demonstrated by the presence of immunoglobulins and complement components within renal biopsies<sup>[27]</sup>. This suggests an immune-mediated process contributing to renal pathology. Furthermore, the potential direct influence of lepra bacilli on renal complications cannot be dismissed, implicating the bacteria in the development of renal manifestations<sup>[28]</sup>.

Overall, our findings shed light on the intricate interplay of immunological processes and potential microbial contributions in the genesis of renal involvement among leprosy patients. Further research is warranted to unravel the precise mechanisms and interactions underlying this intriguing clinical association.

## REFERENCES

1. WHO., 2012. Weekly epidemiological record. Relevé Épidémiologique Hebdomadaire, 87: 129-144.
2. Drutz, D.J. and R.A. Gutman, 1973. Renal manifestations of leprosy: Glomerulonephritis, a complication of erythema nodosum leprosum. Am. J. Trop. Med. Hyg., 22: 496-502.
3. Aggarwal, H.K., P. Sharma, T.S. Jaswal, V.K. Jain and N. Nand *et al.* 2004. Evaluation of renal profile in patients of leprosy. J. Indian Acad. Clin. Med., 5: 316-321.
4. Saha, K. and A.K. Chakraborty, 1977. Serum complement profile in human leprosy and its comparison with immune complex diseases. Int. J. Lepr. Other Mycobact. Dis., 45: 327-337.
5. Date, A., K.V. Johny, R. Mathai and A. Thomas, 1977. Glomerular pathology in leprosy: An electron microscopic study. Am. J. Trop. Med. Hyg., 26: 266-272.
6. Date, A. and K.V. Johny, 1975. Glomerular subepithelial deposits in lepromatous leprosy. Am. J. Trop. Med. Hyg., 24: 853-856.
7. Bajaj, A.K., S.C. Gupta, S.N. Sinha, D.C. Govil, U.C. Gaur and R. Kumar, 1981. Renal functional status in lepromatous leprosy. Int. J. Lepr. Other Mycobact. Dis., 49: 37-41.
8. Gutman, R.A., W.H. Lu and D.J. Drutz, 1973. Renal manifestations of leprosy: Impaired acidification and concentration of urine in patients with leprosy. Am. J. Trop. Med. Hyg., 22: 223-228.
9. Bullock, W.E., M.L. Callera and B.J. Panner, 1974. Immunohistologic alteration of skin and ultrastructural changes of glomerular basement membranes in leprosy. Am. J. Trop. Med. Hyg., 23: 81-86.
10. Iveson, J.M., A.C. McDougall, A.J. Leatham and H.J. Harris, 1975. Lepromatous leprosy presenting with polyarthritis, myositis, and immune-complex glomerulonephritis. Br. Med. J., 3: 619-621.
11. Hansen, A.G. and C. Looft, O. Die Lepra Vom Clinischen Und Pathologisch Anatomischen Standpunkte. N. Walker. Bristol: John Wright and Co, Pages: 45.
12. Gupta, S.C., A.K. Bajaj, D.C. Govil, S.N. Sinha and R. Kumar, 1981. A study of percutaneous renal biopsy in lepromatous leprosy. Lepr. India, 53: 179-184.
13. Hedfors, E. and R. Norberg, 1974. Evidence for circulating immune complexes in sarcoidosis. Clin. Exp. Immunol., 16: 493-496.
14. Ozturk, S., T. Ozturk and I. Can, 2017. Renal involvement in leprosy: Evaluation of patients in Turkey. Adv. Dermatol. Allergol., 3: 240-244.
15. Goldman, L., 1971. Leprosy in five young men. Arch. Pediatr. Adolesc. Med., Vol. 122, No. 2. 10.1001/archpedi.1971.02110020117027
16. Ahsan, N., D.E. Wheeler and B.F. Palmer, 1995. Leprosy-associated renal disease. J. Am. Soc. Nephrol., 5: 1546-1552.
17. McAdam, K.P.W.J., R.F. Anders, S.R. Smith, D.A. Russell and M.A. Price, 1975. Association of amyloidosis with erythema nodosum leprosum reactions and recurrent neutrophil leucocytosis in leprosy. Lancet, 306: 572-576.
18. Ng, W.L., D.M. Scollard and A. Hua, 1981. Glomerulonephritis in leprosy. Am. J. Clin. Pathol., 76: 321-329.
19. Editorial, 1975. Amyloidosis and leprosy. Lancet, 2: 589-290.
20. Aggarwal, H.K., P. Sharma, T.S. V.K. Jaswal, N. Jain, R. Nand, V. Sehgal and M.K. Bharti, 2004. Evaluation of renal profile in patients of leprosy. J. Indian Acad. Clin. Med., 5: 316-321.

21. Weiner, I.D. and A.D. Northcutt, 1989. Leprosy and glomerulonephritis: Case report and review of the literature. *Am. J. Kidney Dis.*, 13: 424-429.
22. Oliveira, R.A., G.B. Silva, C.J. Souza, E.F. Vieira and R.M.S. Mota *et al.*, 2007. Evaluation of renal function in leprosy: A study of 59 consecutive patients. *Nephrology Dialysis Transplant.*, 23: 256-262.
23. Peter, K.S., T. Vijayakumar, D.M. Vasudevan, K.R. Devi, M.T. Mathew and T. Gopinath, 1981. Renal involvement in leprosy. *Lepr. India*, 53: 163-178.
24. Dedhia, N.M., A.F. Almeida, U.B. Khanna, B.V. Mittal and V.M. Acharya, 1986. Acute renal failure: A complication of new multidrug regimen for treatment of leprosy. *Int. J. Lepr. Other Mycobact. Dis.*, 54: 380-382.
25. Kanwar, A.J., S.C. Bharija and M.S. Belhaj, 1984. Renal functional status in leprosy. *Indian J. Leprosy*, 56: 595-599.
26. Alam, F. and S.A. Emadi, 2014. Case of arthritis secondary to leprosy. *Springerplus*, Vol. 3, No. 734. 10.1186/2193-1801-3-734
27. Chugh, K.S., P.B. Damle, S. Kaur, B.K. Sharma and B. Kumar *et al.*, 1983. Renal lesions in leprosy amongst north Indian patients. *Postgraduate Med. J.*, 59: 707-711.
28. Powell, C.S. and L.L. Swan, 1955. Leprosy: Pathologic changes observed in fifty consecutive necropsies. *Am. J. Pathol.*, 31: 1131-1147.