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Original Research Article

ASSOCIATION BETWEEN ESTIMATED GLOMERULAR FILTRATION RATE AND CD4+ T-LYMPHOCYTE COUNT AMONG PEOPLE LIVING WITH HIV: A CROSS-SECTIONAL OBSERVATIONAL STUDY FROM A TERTIARY CARE CENTRE IN CENTRAL INDIA

Tanvi Kathed[1], Milind Chavan[2], Mansee Bhagat[3], Pranayak Panicker[4]

[1]Resident, Department of General Medicine, Government Medical College, Nagpur, India.
[2]Associate Professor, Department of General Medicine, Government Medical College, Nagpur, India.
[3]Assistant Professor, Department of General Medicine, Government Medical College, Nagpur, India.
[4]Resident, Department of General Medicine, Government Medical College, Nagpur, India.

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Corresponding Author:
Dr. Tanvi Kathed,
Resident , Department of General Medicine, Government Medical College , Nagpur, India.
Email: kathed.tanvi@gmail.com

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ABSTRACT

Background: Renal dysfunction is an increasingly recognized complication among people living with HIV (PLHIV), contributing substantially to morbidity and mortality despite widespread access to antiretroviral therapy (ART). Chronic immune activation, direct viral injury, nephrotoxic effects of ART, and traditional cardiovascular risk factors collectively influence renal function in this population. The CD4+ T-lymphocyte count remains the principal marker of immunological status in HIV infection, while estimated glomerular filtration rate (eGFR) is a reliable indicator of kidney function. However, data evaluating the association between eGFR and CD4+ T-lymphocyte count from Central India remain limited. The present study was undertaken to evaluate the association between eGFR and CD4+ T-lymphocyte count among PLHIV attending a tertiary care centre in Central India.

Materials and Methods: A hospital-based cross-sectional observational study was conducted among adult PLHIV attending the Antiretroviral Therapy (ART) Centre and Department of General Medicine at a tertiary care hospital in Central India. Demographic, clinical, treatment-related, and laboratory data were collected using a predesigned case record form. Serum creatinine was measured using standard laboratory methods, and eGFR was calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation. CD4+ T-lymphocyte count was determined by flow cytometry and categorized into predefined clinical groups. Renal function was classified according to Kidney Disease: Improving Global Outcomes (KDIGO) guidelines. Descriptive statistics, correlation analysis, group comparisons, and regression analyses were performed using IBM SPSS Statistics, with a p value <0.05 considered statistically significant.

Results: The majority of participants demonstrated preserved renal function, although 12% had reduced renal function (eGFR <60 mL/min/1.73 m²). A significant positive correlation was observed between CD4+ T-lymphocyte count and eGFR, while serum creatinine and blood urea levels showed significant inverse correlations with CD4 count. Participants with CD4 counts <200 cells/mm³ had significantly lower eGFR and higher serum creatinine compared with those having CD4 counts ≥500 cells/mm³ (p<0.001). Reduced renal function was significantly associated with older age, longer duration of HIV infection and ART exposure, advanced WHO clinical stage, opportunistic infections, and lower CD4+ T-lymphocyte counts.

Conclusion: Lower CD4+ T-lymphocyte count was significantly associated with impaired renal function among people living with HIV. Routine assessment of eGFR together with immunological monitoring may facilitate early detection of renal impairment, optimize clinical management, and reduce the burden of kidney disease in PLHIV. Larger prospective multicentre studies are warranted to further validate these findings.
Keywords: Human immunodeficiency virus; Estimated glomerular filtration rate; CD4+ T-lymphocyte count; Renal dysfunction; Antiretroviral therapy.

INTRODUCTION

Human immunodeficiency virus (HIV) infection remains a major global public health challenge despite remarkable advances in antiretroviral therapy (ART). With increasing life expectancy among people living with HIV (PLHIV), chronic non-communicable diseases have emerged as important determinants of morbidity and mortality, among which kidney disease occupies a central role. Renal involvement in HIV is multifactorial and encompasses HIV-associated nephropathy (HIVAN), HIV-associated immune complex kidney disease (HIVICK), ART-induced nephrotoxicity, opportunistic infections, and conventional risk factors such as diabetes mellitus and hypertension. These mechanisms ultimately contribute to glomerular and tubular injury, progressive nephron loss, and decline in glomerular filtration rate (GFR). Estimated glomerular filtration rate (eGFR), calculated using the CKD Epidemiology Collaboration (CKD-EPI) equation, is a validated, non-invasive marker of renal function and is recommended for routine assessment in PLHIV because early renal impairment is frequently asymptomatic and potentially reversible with timely intervention.[1-5]

CD4+ T-lymphocyte count is the principal immunological marker used to assess the severity of HIV infection, monitor disease progression, and evaluate response to ART. Progressive depletion of CD4+ T cells results in immune dysregulation, chronic inflammation, and persistent immune activation, which contribute to endothelial dysfunction, renal microvascular injury, and direct viral effects on podocytes and tubular epithelial cells. Although the incidence of classical HIVAN has declined in the ART era, kidney dysfunction remains common because of aging, prolonged ART exposure, metabolic comorbidities, and chronic immune activation. Several studies have demonstrated that lower CD4+ cell counts are associated with an increased risk of renal impairment and reduced eGFR, suggesting that immunological status may influence kidney function in PLHIV. Consequently, simultaneous assessment of eGFR and CD4+ count may facilitate early identification of individuals at risk for renal dysfunction and improve long-term clinical outcomes.[2,4-7]

India has one of the largest populations of PLHIV globally, yet data evaluating the relationship between immune status and renal function remain limited, particularly from Central India where demographic characteristics, ART practices, and comorbidity profiles may differ from those reported elsewhere. Most available Indian studies have focused on the prevalence of chronic kidney disease or ART-associated nephrotoxicity rather than exploring the association between eGFR and CD4+ T-lymphocyte count. Furthermore, regional evidence using standardized eGFR estimation methods such as the CKD-EPI equation is scarce. Therefore, the present cross-sectional observational study was undertaken to evaluate the association between estimated glomerular filtration rate and CD4+ T-lymphocyte count among people living with HIV attending a tertiary care centre in Central India. The findings may contribute to the early identification of renal dysfunction, guide risk stratification, and support routine renal monitoring as an integral component of comprehensive HIV care.[1-6]

MATERIALS AND METHODS

This hospital-based cross-sectional observational study was conducted in the Department of General Medicine in collaboration with the Antiretroviral Therapy (ART) Centre, Central India, over a period of 18 Month, after obtaining approval from the Institutional Ethics Committee. Adult people living with HIV (PLHIV) aged ≥18 years attending the ART Centre or admitted to the Department of Medicine during the study period were enrolled using consecutive sampling after obtaining written informed consent. Patients with known chronic kidney disease unrelated to HIV, acute kidney injury, pregnancy, malignancy, or any acute illness likely to independently alter renal function were excluded from the study. Demographic characteristics including age and sex, clinical variables such as duration of HIV infection, antiretroviral therapy (ART) regimen and duration, history of hypertension and diabetes mellitus, and relevant laboratory parameters were recorded using a predesigned and standardized case record form.

Venous blood samples were collected under aseptic precautions as part of routine clinical evaluation. Serum creatinine was estimated using the [enzymatic/Jaffe] method in the institutional biochemistry laboratory, and the estimated glomerular filtration rate (eGFR) was calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation and expressed as mL/min/1.73 m². Renal function was categorized

according to the Kidney Disease: Improving Global Outcomes (KDIGO) classification into G1 (≥90), G2 (60–89), G3a (45–59), G3b (30–44), G4 (15–29), and G5 (<15 mL/min/1.73 m²). CD4+ T-lymphocyte count was determined by flow cytometry and categorized as <200, 200–499, and ≥500 cells/mm³ according to standard clinical thresholds. Reduced renal function was defined as an eGFR <60 mL/min/1.73 m².

Data were entered into Microsoft Excel and analyzed using IBM SPSS Statistics version 25 (IBM Corp., Armonk, NY, USA). Continuous variables were tested for normality using the Shapiro–Wilk test and expressed as mean ± standard deviation (SD) or median with interquartile range (IQR), as appropriate, while categorical variables were presented as frequencies and percentages. Comparisons of renal function parameters across CD4+ T-lymphocyte count categories were performed using one-way analysis of variance (ANOVA) or the Kruskal–Wallis test for continuous variables and the chi-square or Fisher's exact test for categorical variables, as appropriate. The association between eGFR and CD4+ T-lymphocyte count was assessed using Pearson's or Spearman's correlation coefficient depending on data distribution. Univariable and multivariable linear regression analyses were performed to identify factors independently associated with eGFR after adjusting for clinically relevant covariates, including age, sex, duration of HIV infection, ART duration, hypertension, and diabetes mellitus. All statistical tests were two-tailed, and a p value <0.05 was considered statistically significant.

RESULTS

Characteristics of the cohort

Three hundred and sixteen adults with prediabetes were studied. Age ranged from 23 to 66 years with a mean of 47.5 years; the largest single band was 41–50 years (98 participants, 31.0%), and only 13 participants (4.1%) were aged 30 years or younger. There were 168 men (53.2%) and 148 women (46.8%). Mean HbA1c was 5.9 ± 0.19%, all values lying within the prediabetic range. Overweight or obesity was present in 168 participants (53.2%), hypertension in 173 (54.7%) and raised total cholesterol in 164 (51.9%). Cohort characteristics are shown in Table 1.

Table 1: Baseline demographic, clinical, HIV-related, treatment, and laboratory characteristics of the study population

Characteristic	Summary value
Demographic and clinical characteristics	
Age, years, median (IQR)	40 (33–49)
Male sex, n (%)	62 (62.0)
Female sex, n (%)	38 (38.0)
Body mass index, kg/m², median (IQR)	22.9 (20.8–25.6)
Hypertension, n (%)	12 (12.0)
Diabetes mellitus, n (%)	8 (8.0)
Current smoking, n (%)	17 (17.0)
Alcohol consumption, n (%)	22 (22.0)
HIV-related characteristics	
Duration of HIV infection, years, median (IQR)	4.0 (2.0–7.0)
WHO clinical stage III–IV, n (%)	35 (35.0)
History of opportunistic infection, n (%)	27 (27.0)
CD4+ T-lymphocyte count, cells/mm³, mean ± SD	438.7 ± 221.4
CD4+ T-lymphocyte count, cells/mm³, median (IQR)	412 (268–598)
Antiretroviral therapy characteristics	
Receiving ART, n (%)	100 (100.0)
Duration of ART, years, median (IQR)	4.0 (2.0–6.5)
Tenofovir-based regimen, n (%)	82 (82.0)
Kidney function parameters	
Blood urea, mg/dL, median (IQR)	28 (22–36)
Serum creatinine, mg/dL, median (IQR)	0.91 (0.78–1.08)
Serum sodium, mEq/L, median (IQR)	138 (136–141)
Serum potassium, mEq/L, median (IQR)	4.2 (3.9–4.5)
eGFR, mL/min/1.73 m², mean ± SD	94.8 ± 21.6
eGFR, mL/min/1.73 m², median (IQR)	96.5 (81–108)
eGFR range, mL/min/1.73 m²	38–142

A total of 100 people living with HIV (PLHIV) were included in the study. The median age of the participants was 40 (IQR: 33–49) years, with 62% being males. The median body mass index was 22.9 (IQR: 20.8–25.6) kg/m². The median duration of HIV infection and antiretroviral therapy (ART) was 4 (IQR: 2–7) years and 4 (IQR: 2–6.5) years, respectively. Hypertension and diabetes mellitus were present in 12% and 8% of the participants, while 27% had a history of opportunistic infections. The mean CD4+ T-lymphocyte count was 438.7 ± 221.4 cells/mm³, and the mean eGFR was 94.8 ± 21.6 mL/min/1.73 m². The median serum creatinine and blood urea levels were 0.91 mg/dL and 28 mg/dL, respectively. Overall, 12% of the study participants had reduced renal function (eGFR <60 mL/min/1.73 m²). [Table 1]

Table 2: Distribution of CD4+ T-lymphocyte count and estimated glomerular filtration rate categories

Classification	Category	n (%)	95% CI, %
CD4+ T-lymphocyte count	<200 cells/mm³	14 (14.0)	8.5–22.2
	200–349 cells/mm³	24 (24.0)	16.7–33.2
	350–499 cells/mm³	28 (28.0)	20.0–38.0
	≥500 cells/mm³	34 (34.0)	25.4–43.8
KDIGO eGFR category	G1: ≥90 mL/min/1.73 m²	67 (67.0)	—
	G2: 60–89 mL/min/1.73 m²	21 (21.0)	—
	G3a: 45–59 mL/min/1.73 m²	8 (8.0)	—
	G3b: 30–44 mL/min/1.73 m²	4 (4.0)	—
	G4: 15–29 mL/min/1.73 m²	0 (0.0)	—
	G5: <15 mL/min/1.73 m²	0 (0.0)	—
Overall kidney function status	eGFR ≥60 mL/min/1.73 m²	88 (88.0)	80.0–93.7
	eGFR <60 mL/min/1.73 m²	12 (12.0)	6.3–20.0

Based on immunological status, 34% of participants had a CD4+ T-lymphocyte count of ≥500 cells/mm³, followed by 28% with 350–499 cells/mm³, 24% with 200–349 cells/mm³, and 14% with <200 cells/mm³. According to the KDIGO classification, 67% of participants were categorized as G1 (eGFR ≥90 mL/min/1.73 m²), 21% as G2 (60–89 mL/min/1.73 m²), 8% as G3a (45–59 mL/min/1.73 m²), and 4% as G3b (30–44 mL/min/1.73 m²). None of the participants belonged to G4 or G5 categories. Overall, 88% of participants had preserved renal function, whereas 12% had reduced renal function (eGFR <60 mL/min/1.73 m²). [Table 2]

Table 3: Comparison of kidney function parameters across CD4+ T-lymphocyte count categories

Parameter	CD4 <200 (n=14)	CD4 200–349 (n=24)	CD4 350–499 (n=28)	CD4 ≥500 (n=34)	*p*-value
eGFR, mL/min/1.73 m²	68 (54–81)	84 (72–96)	99 (88–110)	112 (101–121)	<0.0001
Blood urea, mg/dL	39 (32–48)	31 (25–38)	27 (22–32)	23 (19–28)	<0.0001
Serum creatinine, mg/dL	1.18 (0.98–1.42)	1.01 (0.88–1.16)	0.88 (0.77–0.98)	0.79 (0.70–0.91)	<0.0001
Serum sodium, mEq/L	137 (135–139)	138 (136–140)	138 (136–141)	139 (137–141)	0.2784
Serum potassium, mEq/L	4.4 (4.0–4.8)	4.3 (4.0–4.6)	4.2 (3.9–4.5)	4.1 (3.8–4.4)	0.1842
Reduced kidney function, n (%)	6 (42.9)	4 (16.7)	2 (7.1)	0 (0.0)	0.0002†

A significant difference in renal function parameters was observed across the CD4+ T-lymphocyte count categories. Participants with CD4 counts <200 cells/mm³ had the lowest median eGFR (68 mL/min/1.73 m²) and the highest median serum creatinine (1.18 mg/dL) and blood urea (39 mg/dL) levels. In contrast, participants with CD4 counts ≥500 cells/mm³ demonstrated the highest median eGFR (112 mL/min/1.73 m²) and the lowest serum creatinine (0.79 mg/dL) and blood urea (23 mg/dL) levels. Significant differences were observed in eGFR, serum creatinine, and blood urea across the CD4 categories (p<0.001 for all). However, serum sodium and potassium levels did not differ significantly between the groups (p=0.278 and p=0.184, respectively). The proportion of participants with reduced renal function progressively decreased with increasing CD4+ T-lymphocyte count, ranging from 42.9% in those with CD4 counts <200 cells/mm³ to 0% among participants with CD4 counts ≥500 cells/mm³. [Table 3]

Table 4: Correlation of CD4+ T-lymphocyte count with kidney function parameters

Kidney function parameter	Spearman's ρ	95% CI	*p*-value
eGFR, mL/min/1.73 m²	0.524	0.361 to 0.651	<0.0001
Blood urea, mg/dL	−0.381	−0.534 to −0.193	0.0001
Serum creatinine, mg/dL	−0.468	−0.607 to −0.290	<0.0001
Serum sodium, mEq/L	0.094	−0.104 to 0.285	0.3521
Serum potassium, mEq/L	−0.118	−0.306 to 0.080	0.2416

Correlation analysis revealed a statistically significant moderate positive correlation between CD4+ T-lymphocyte count and eGFR (ρ = 0.524, p<0.001), indicating that higher CD4 counts were associated with better renal function. Conversely, CD4 count demonstrated significant negative correlations with serum creatinine (ρ = –0.468, p<0.001) and blood urea (ρ = –0.381, p<0.001). No significant correlation was observed between CD4+ T-lymphocyte count and serum sodium (ρ = 0.094, p=0.352) or serum potassium (ρ = –0.118, p=0.242). [Table 4]

Table 5: Factors associated with reduced kidney function, defined as eGFR <60 mL/min/1.73 m²

Variable	Reduced kidney function (n=12)	eGFR ≥60 mL/min/1.73 m² (n=88)	Effect estimate (95% CI)	*p*-value
Age, years, median (IQR)	52 (46–59)	38 (31–47)	—	0.0004
Male sex, n (%)	8 (66.7)	54 (61.4)	—	0.7238
WHO clinical stage III–IV, n (%)	8 (66.7)	27 (30.7)	OR 4.51 (1.29–15.75)	0.0181

History of opportunistic infection, n (%)	7 (58.3)	20 (22.7)	OR 4.76 (1.39–16.24)	0.0130
Duration of HIV infection, years, median (IQR)	6.5 (4.0–9.0)	3.5 (2.0–6.0)	—	0.0217
Duration of ART, years, median (IQR)	6.0 (4.0–8.0)	3.5 (2.0–6.0)	—	0.0328
Tenofovir exposure, n (%)	11 (91.7)	71 (80.7)	—	0.6814
CD4 count, cells/mm³, median (IQR)	218 (142–328)	452 (312–618)	—	<0.0001
CD4 count <350 cells/mm³, n (%)	10 (83.3)	28 (31.8)	OR 10.68 (2.18–52.26)	0.0034
Blood urea, mg/dL, median (IQR)	46 (40–58)	26 (21–33)	—	<0.0001
Serum creatinine, mg/dL, median (IQR)	1.42 (1.26–1.78)	0.86 (0.74–0.98)	—	<0.0001

Participants with reduced renal function (eGFR <60 mL/min/1.73 m²) were significantly older than those with preserved renal function (median age 52 vs. 38 years, p=0.0004). They also had a longer duration of HIV infection (6.5 vs. 3.5 years, p=0.0217) and ART exposure (6.0 vs. 3.5 years, p=0.0328). Advanced WHO clinical stage (66.7% vs. 30.7%, p=0.018), history of opportunistic infections (58.3% vs. 22.7%, p=0.013), and CD4+ T-lymphocyte count <350 cells/mm³ (83.3% vs. 31.8%, p=0.003) were significantly more common among participants with reduced renal function. Blood urea and serum creatinine concentrations were also significantly higher in this group (p<0.001 for both). No statistically significant association was observed between reduced renal function and male sex or tenofovir-based ART regimen. [Table 5]

DISCUSSION

The present study demonstrated a significant positive association between CD4+ T-lymphocyte count and estimated glomerular filtration rate (eGFR), with participants having lower CD4 counts exhibiting significantly lower eGFR, higher serum creatinine, and a greater prevalence of reduced renal function. These findings support the concept that deterioration in immunological status is accompanied by progressive renal dysfunction in people living with HIV (PLHIV). HIV-mediated renal injury results from direct viral infection of podocytes and tubular epithelial cells, persistent immune activation, chronic inflammation, and exposure to nephrotoxic antiretroviral agents, all of which contribute to progressive nephron loss and declining kidney function.[8-11]

In the present study, the majority of participants had preserved renal function, while 12% had reduced renal function (eGFR <60 mL/min/1.73 m²). This observation is comparable to the multicentre ART cohort reported by Scherzer et al., in which the incidence of CKD (eGFR <60 mL/min/1.73 m²) was approximately 10.5 per 1000 person-years, with lower CD4 counts and higher viral loads independently predicting CKD development.[12] Similarly, Ibrahim et al. evaluated 3,714 HIV-infected patients and reported a CKD prevalence of 4.1%, with affected patients being older, having lower CD4 counts, longer HIV duration, hypertension, and more rapid eGFR decline.[8] The slightly higher frequency of reduced renal function in our cohort may reflect differences in patient characteristics, regional disease burden, referral patterns, and healthcare access.

Our findings demonstrated that eGFR increased progressively with increasing CD4+ T-lymphocyte count, whereas serum creatinine and blood urea decreased significantly across CD4 categories. A moderate positive correlation between CD4 count and eGFR further supports the close relationship between immune recovery and preservation of renal function. These observations agree with the findings of Post et al., who reported that a baseline CD4 count ≥200 cells/mm³ was associated with significantly better renal outcomes, with 64% of patients maintaining eGFR >60 mL/min/1.73 m² compared with only 35% among those with CD4 counts <200 cells/mm³ during follow-up.[13] Likewise, Choi et al. demonstrated that CD4 counts <200 cells/mm³ increased the risk of end-stage renal disease by nearly 50% (HR 1.5, 95% CI 1.2–2.0), highlighting the importance of immune status in determining long-term renal prognosis.[10]

Reduced renal function in the present study was also associated with older age, longer duration of HIV infection, advanced WHO clinical stage, opportunistic infections, and lower CD4 counts. Similar observations have been reported in several longitudinal cohorts. Choi et al. found that hypertension (HR 1.9), diabetes mellitus (HR 1.7), cardiovascular disease (HR 2.2), CD4 count <200 cells/mm³ (HR 1.5), and high HIV viral load (HR 2.0) were independent predictors of progression to end-stage renal disease.[10] Scherzer et al. further observed that lower time-updated CD4 counts, higher viral load, and tenofovir-containing regimens significantly increased CKD risk, although effective ART slowed the rate of eGFR decline.[12] These findings emphasize that both HIV-related and traditional cardiovascular risk factors contribute to renal impairment in PLHIV.

Although tenofovir exposure was not significantly associated with reduced renal function in our study, prolonged ART exposure was more common among participants with impaired kidney function. Ryom et al., in the large D:A:D cohort of 22,603 HIV-positive individuals, demonstrated that cumulative exposure to tenofovir, ritonavir-boosted atazanavir, and

lopinavir independently increased the risk of chronic renal impairment, although the absolute incidence of confirmed CKD remained relatively low (0.6% during a median follow-up of 4.5 years).[14] In contrast, Lucas et al. reported that initiation of ART improved the annual eGFR slope from −4.7 to −1.9 mL/min/1.73 m², suggesting that viral suppression mitigates—but does not completely prevent—progressive renal dysfunction.[11] Differences in ART regimens, treatment duration, and baseline renal function may explain the variation between studies.

Overall, the present study reinforces the growing evidence that declining immune status is associated with impaired renal function in PLHIV. The observed positive relationship between CD4+ T-lymphocyte count and eGFR, together with the higher burden of renal dysfunction among individuals with advanced immunosuppression, supports routine monitoring of renal function alongside immunological assessment during HIV care. Early identification of patients with low CD4 counts and declining eGFR may facilitate timely interventions, optimize antiretroviral therapy, and potentially reduce the progression of HIV-associated kidney disease.

CONCLUSION

The present cross-sectional study demonstrated a significant positive association between CD4+ T-lymphocyte count and estimated glomerular filtration rate (eGFR) among people living with HIV. Participants with lower CD4 counts had significantly reduced eGFR, higher serum creatinine levels, and a greater prevalence of reduced renal function, indicating that worsening immunological status is associated with declining kidney function. In addition, older age, longer duration of HIV infection, advanced disease stage, and opportunistic infections were associated with impaired renal function. These findings highlight the importance of integrating routine renal function assessment with immunological monitoring to facilitate early detection and management of kidney dysfunction in PLHIV.

Recommendations: Routine estimation of eGFR should be incorporated into the standard clinical evaluation of all people living with HIV, particularly those with low CD4+ T-lymphocyte counts, prolonged HIV duration, or additional renal risk factors. Periodic monitoring may enable early identification of renal impairment, optimization of antiretroviral therapy, and timely nephrology referral when indicated. Further prospective multicentre studies with larger sample sizes, inclusion of viral load assessment and urinary biomarkers, and longitudinal follow-up are recommended to better establish the temporal relationship between immunological recovery and renal function decline.

Limitations: This study has certain limitations. The cross-sectional design precludes establishing a causal relationship between CD4+ T-lymphocyte count and renal dysfunction. Being a single-centre study, the findings may not be generalizable to all populations of people living with HIV. Renal function was assessed using a single eGFR measurement without longitudinal follow-up, and additional markers of kidney damage such as albuminuria or proteinuria were not evaluated. Furthermore, viral load and cumulative exposure to potentially nephrotoxic antiretroviral drugs were not comprehensively analyzed, which may have influenced the observed associations. Despite these limitations, the study provides important regional evidence regarding the relationship between immune status and renal function among people living with HIV in Central India.

Conflict of Interest

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