

Research Article

Active Tuberculosis in Chronic Kidney Disease: Frequency, Clinical Spectrum, and Stage-Wise Pattern in an Observational Study

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ABSTRACT

Background: Tuberculosis Remains Clinically Important In Patients With Chronic Kidney Disease Because Uremia, Malnutrition, Diabetes, And Dialysis-Related Exposure May Weaken Host Defence And Delay Recognition Of Disease.

Objective: To Determine The Frequency, Phenotype, And Stage-Wise Pattern Of Active TB Among Patients With CKD And To Explore Selected Clinical Correlates.

Materials and Methods: This Observational Cross-Sectional Analysis Included 60 CKD Patients Enrolled Between March 2025 And February 2026. Variables Included CKD Stage, Egfr, Diabetes, Hypertension, TB Status, TB Type, Smoking, BMI, Serum Albumin, And Haemoglobin. Exact Tests And Mann-Whitney U Testing Were Used Because Active TB Events Were Few.

Results: Active TB Was Documented In 6/60 Patients (10.0%; 95% CI: 3.8-20.5). Pulmonary, Extra pulmonary, And Combined TB Were Equally Represented (2 Cases Each). Stage 5D Showed the Highest Numerical Frequency (2/5, 40.0%), Compared With 4/55 (7.3%) In Non-Stage 5D Patients (P=0.074). Active TB Patients Had Lower Mean Egfr, Serum Albumin, And Haemoglobin Than Those Without TB, Although None Reached Statistical Significance.

Conclusion: Active TB Was Not Rare In This CKD Cohort And Appeared Numerically Concentrated In Dialysis-Dependent Disease. The Findings Support Stage-Sensitive TB Vigilance In CKD Care, Particularly When Advanced CKD, Hypoalbuminaemia, Anemia, Or Dialysis Transition Is Present.

Keywords: Active Tuberculosis, Chronic Kidney Disease, Dialysis, Extra Pulmonary Tuberculosis, Serum Albumin, India.

INTRODUCTION

Tuberculosis (TB) remains a stubborn public health problem, even in health systems where diagnostic coverage and treatment notification have improved. The latest global assessment from the World Health Organization continues to describe TB as a major cause of preventable infectious morbidity, with progress still shaped by delayed diagnosis, undernutrition, diabetes, poverty, and other vulnerabilities that cluster in the same communities [1]. India occupies a particularly important position in this landscape. The National Tuberculosis Elimination Programme has expanded notification, molecular diagnosis, nutritional support, and active case-finding, yet tertiary centres continue to receive patients whose risk is amplified by chronic non-communicable diseases [2].

Chronic kidney disease (CKD) is one such condition. KDIGO defines CKD through persistent abnormal kidney structure or function and classifies severity through cause, GFR category, and albuminuria category, with GFR stages ranging from G1 to G5 [3]. The global burden of CKD has increased steadily, and Indian studies have shown that CKD is not merely a nephrology problem but a broader public health issue linked with diabetes, hypertension, ageing, and limited access to early screening [4,5]. In routine practice, the advanced CKD patient is often moving through repeated hospital visits, laboratory encounters, dialysis areas, and specialist consultations. That repeated contact with healthcare systems sits beside a biological susceptibility that is easy to underestimate.

The CKD-TB relationship has therefore been described as a syndemic rather than a simple

comorbidity. Romanowski and colleagues emphasized that TB risk in CKD is shaped by impaired cellular immunity, malnutrition, socioeconomic exposure, immunosuppression, and the dialysis environment [6]. A more recent systematic review showed that even CKD without kidney failure carries excess TB risk, and that the gradient becomes steeper in stages 4 and 5 [7]. Another meta-analysis found a high pooled TB incidence among CKD patients, with dialysis populations contributing a particularly heavy share; notably, extrapulmonary disease was not rare in these groups [8].

The biological explanation is plausible. Uraemia alters innate and adaptive immune function through retained solutes, chronic inflammation, lymphocyte dysfunction, and impaired antigen-specific responses [9,10]. Diabetes adds further risk, and its overlap with CKD is common in India [11]. Nutritional depletion, reflected in low body mass or hypoalbuminaemia, may further weaken host defence against *Mycobacterium tuberculosis* [12]. Yet many hospital records still treat TB status as an incidental label rather than a structured risk marker in CKD clinics.

The present study was undertaken to describe the frequency, clinical spectrum, and stage-wise pattern of active TB among patients with CKD. A secondary objective was to examine whether active TB clustered with advanced CKD, dialysis dependence, diabetes, smoking, low serum albumin, and anaemia in this cohort.

MATERIALS AND METHODS

Study Design and Period: This observational cross-sectional analytical study included patients with CKD enrolled between 5 March 2025 and 10 February 2026. The manuscript should be updated with the final institution name and ethics approval details before journal submission.

Study Population: Sixty patients with recorded CKD stage, estimated glomerular filtration rate (eGFR), active TB status, and core clinical variables were included. The cohort comprised patients across Stage 1,

Stage 2, Stage 3a, Stage 3b, Stage 4, Stage 5 not on dialysis (Stage 5-ND), and dialysis-dependent Stage 5 disease (Stage 5D).

Variables Assessed: The variables analysed were age, sex, CKD stage, eGFR, diabetes mellitus, hypertension, active TB status, TB type, TB diagnosis date, enrolment date, prior TB, immunosuppressive therapy, smoking status, body mass index (BMI), serum albumin, and haemoglobin. Active TB was classified as pulmonary, extrapulmonary, both pulmonary and extrapulmonary, or absent.

Operational Definitions: Advanced CKD was defined as Stage 4, Stage 5-ND, or Stage 5D. Dialysis-dependent CKD was represented by Stage 5D. Ever smoking included former and current smoking. Low serum albumin was defined as <3.5 g/dL, and anaemia severity was explored using haemoglobin <10 g/dL as a clinically interpretable threshold.

Statistical Analysis: Continuous variables were summarized as mean $\pm$ standard deviation. Categorical variables were summarized as frequency and percentage. The exact binomial method was used to estimate the 95% confidence interval for active TB frequency. Because several contingency cells were sparse, Fisher exact testing was used for two-group comparisons and the Fisher-Freeman-Halton exact test was used for CKD stage-wise distribution. Continuous variables between active TB and non-TB groups were compared using the Mann-Whitney U test. Statistical significance was interpreted at $p < 0.05$; given the low number of TB events, multivariable modelling was not performed.

RESULTS

A total of 60 CKD patients were analysed. The mean age was 63.38 ± 8.72 years, and 33 patients (55.0%) were male. Hypertension was present in 57 patients (95.0%), while diabetes mellitus was documented in 25 patients (41.7%). The mean eGFR was 41.38 ± 27.95 mL/min/1.73 m², mean serum albumin was 3.48 ± 0.39 g/dL, and mean hemoglobin was 10.95 ± 1.65 g/dL (Table 1).

Table 1. Baseline Clinical Profile According To Active Tuberculosis Status (N=60).

Variable	Total (N=60)	Active TB (N=6)	No Active TB (N=54)	P-Value
Age (Years)	63.38 $\pm$ 8.72	64.83 $\pm$ 8.75	63.22 $\pm$ 8.79	0.622
Egfr (mL/Min/1.73 M ²)	41.38 $\pm$ 27.95	30.67 $\pm$ 24.98	42.57 $\pm$ 28.22	0.324
BMI (Kg/M ²)	25.68 $\pm$ 2.32	25.17 $\pm$ 0.75	25.74 $\pm$ 2.43	0.691

Serum Albumin (G/Dl)	3.48 ± 0.39	3.25 ± 0.41	3.51 ± 0.39	0.145
Hemoglobin (G/Dl)	10.95 ± 1.65	10.18 ± 1.80	11.04 ± 1.62	0.257
Male Sex	33 (55.0)	3 (50.0)	30 (55.6)	1.000
Diabetes Mellitus	25 (41.7)	2 (33.3)	23 (42.6)	1.000
Hypertension	57 (95.0)	6 (100.0)	51 (94.4)	1.000
Ever Smoker	24 (40.0)	2 (33.3)	22 (40.7)	1.000
Prior TB	2 (3.3)	0 (0.0)	2 (3.7)	1.000
Immunosuppressive Therapy	3 (5.0)	0 (0.0)	3 (5.6)	1.000

Values are mean ± SD or n (%). Mann-Whitney U test was used for continuous variables; Fisher exact test was used for categorical variables.

Active TB was recorded in 6/60 patients, giving a frequency of 10.0% (95% CI: 3.8-20.5%). The remaining 54 patients (90.0%) had no active TB at evaluation. This distribution is shown in Figure 1.

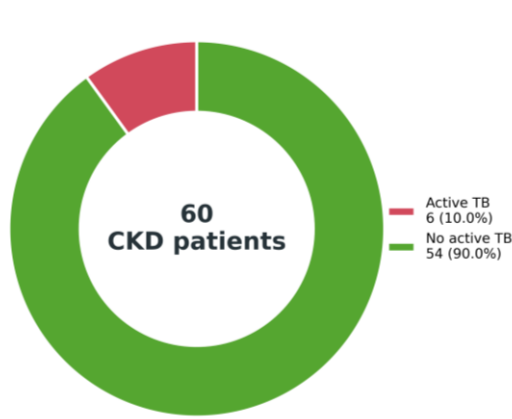


Figure 1. Frequency of Active Tuberculosis among Patients with CKD.

*Donut chart shows the proportion of active TB and non-active TB cases; values are displayed as n (%). *CKD stage distribution was skewed toward moderate-to-advanced disease: Stage 4 accounted for 15 patients (25.0%), Stage 3b for 12 (20.0%), and Stage 3a for 10 (16.7%). Active TB occurred in one patient each in Stage 2, Stage 3a, Stage 3b,

and Stage 4, and in two patients in Stage 5D; no active TB was recorded in Stage 1 or Stage 5-ND. The highest stage-specific proportion was seen in Stage 5D (2/5, 40.0%), although the overall stage-wise association did not reach statistical significance (Fisher-Freeman-Halton exact test, $p=0.512$) (Table 2, Figure 2).

Table 2. CKD Stage-Wise Clinical Profile and Active TB Distribution.

CKD Stage	N	Egfr Mean ± SD	Albumin Mean ± SD	Hb Mean ± SD	Active TB N (%)	TB Phenotype
Stage 1	5	99.4 ± 7.0	4.12 ± 0.19	13.68 ± 0.51	0 (0.0)	None
Stage 2	8	76.8 ± 8.5	3.93 ± 0.17	12.91 ± 0.56	1 (12.5)	Pulmonary: 1
Stage 3a	10	51.4 ± 4.6	3.67 ± 0.18	11.83 ± 0.53	1 (10.0)	Extra pulmonary: 1
Stage 3b	12	37.0 ± 4.7	3.53 ± 0.15	11.12 ± 0.45	1 (8.3)	Both: 1
Stage 4	15	22.0 ± 4.5	3.22 ± 0.15	9.80 ± 0.46	1 (6.7)	Extra pulmonary: 1
Stage 5-ND	5	10.6 ± 2.4	2.92 ± 0.08	8.60 ± 0.27	0 (0.0)	None
Stage 5D	5	6.2 ± 1.9	3.00 ± 0.16	8.74 ± 0.40	2 (40.0)	Pulmonary: 1; Both: 1

Fisher-Freeman-Halton exact test for CKD stage-wise active TB distribution: $p=0.515$. ND: not on dialysis; Hb: hemoglobin.

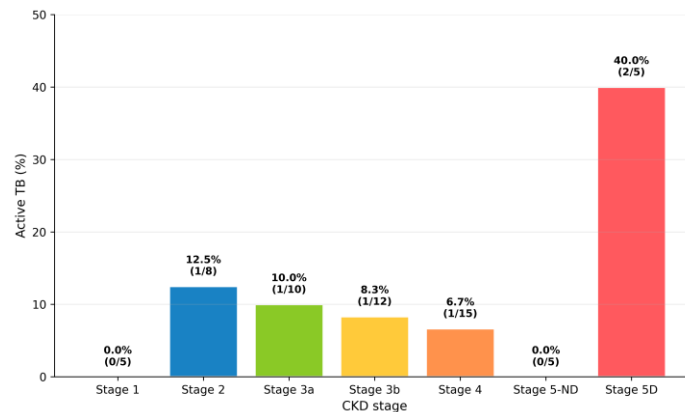


Figure 2. Stage-Wise Frequency of Active Tuberculosis among Ckd Patients.

Bars show stage-specific active TB percentages; bold labels indicate percentage and active TB count/total for each stage. TB phenotype was evenly distributed. Pulmonary TB, extra pulmonary TB, and combined pulmonary plus extra pulmonary TB

were each present in two patients (33.3% each). The stage-wise distribution of TB phenotype is illustrated in Figure 3, and the clinical characteristics of all six active TB cases are summarized in Table 3.

Table 3. Clinical Profile of Patients with Active Tuberculosis.

Patient ID	Age	Sex	CKD Stage	EGFR	TB Type	Diabetes	Smoking	Albumin	Hb
P008	49	Female	Stage 2	68	Pulmonary	No	Never	3.8	12.5
P019	63	Female	Stage 3a	50	Extrapulmonary	No	Never	3.6	11.8
P027	70	Male	Stage 3b	33	Both	No	Former	3.4	10.9
P042	74	Female	Stage 4	22	Extrapulmonary	No	Never	3.0	9.2

Patient ID	Age	Sex	CKD Stage	EGFR	TB Type	Diabetes	Smoking	Albumin	Hb
P057	64	Male	Stage 5D	7	Pulmonary	Yes	Never	2.9	8.5
P059	69	Male	Stage 5D	4	Both	Yes	Former	2.8	8.2

EGFR in mL/min/1.73 m²; albumin in g/dL; Hb in g/dL.

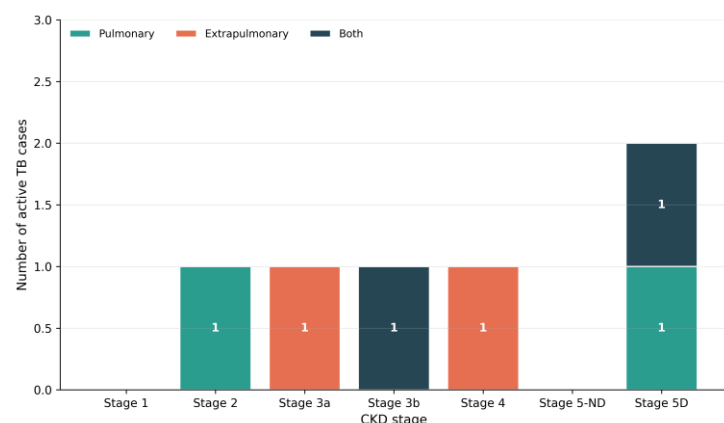


Figure 3. Tuberculosis Phenotype across CKD Stages.

Stacked bars display counts of pulmonary, extra pulmonary, and combined TB among active TB cases; segment labels show counts. When selected risk strata were compared, dialysis-dependent CKD showed the clearest numerical separation: active TB was present in 2/5 Stage 5D patients (40.0%) compared with 4/55 non-Stage 5D patients (7.3%), with a borderline exact test result ($p=0.074$). Active

TB was also more frequent among patients with albumin <3.5 g/dL (4/29, 13.8%) than among those with albumin ≥ 3.5 g/dL (2/31, 6.5%), and among patients with hemoglobin <10 g/dL (3/19, 15.8%) compared with hemoglobin ≥ 10 g/dL (3/41, 7.3%); neither contrast was statistically significant. These crude comparisons are presented in Table 4 and Figure 4.

Table 4. Crude Comparison of Selected Clinical Strata with Active Tuberculosis.

Clinical Stratum	Active TB In Exposed N/N (%)	Active TB In Unexposed N/N (%)	Exact P-Value
Advanced CKD (Stage 4/5-ND/5D)	3 (12.0)	3 (8.6)	0.686
Dialysis-Dependent CKD (Stage 5D)	2 (40.0)	4 (7.3)	0.074
Diabetes Mellitus	2 (8.0)	4 (11.4)	1.000
Ever Smoker	2 (8.3)	4 (11.1)	1.000
Albumin <3.5 G/Dl	4 (13.8)	2 (6.5)	0.417
Hemoglobin <10 G/Dl	3 (15.8)	3 (7.3)	0.370

Exact p-values are from Fisher exact tests. These are unadjusted comparisons;

multivariable modelling was avoided because only six active TB events were present.

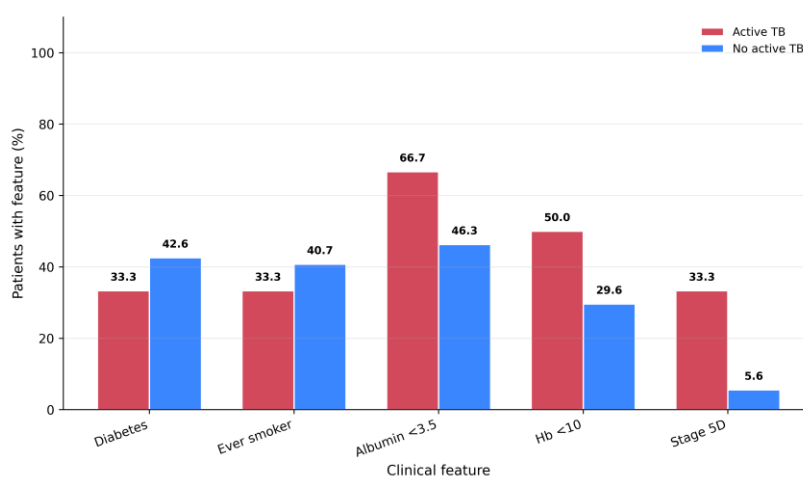


Figure 4. Selected Clinical Features According To Active Tuberculosis Status

Grouped bars show the percentage of patients with each feature in the active TB and non-active TB groups; values above bars represent percentages.

Continuous clinical variables showed directionally poorer renal and nutritional-haematological profiles among patients with active TB. Mean eGFR was lower in the active TB group (30.67 ± 24.98 vs 42.57 ± 28.22 mL/min/ 1.73 m ^{2}), serum albumin was lower (3.25 ± 0.41 vs 3.51 ± 0.39 g/dL), and haemoglobin was lower (10.18 ± 1.80 vs 11.04 ± 1.62 g/dL), but the differences were not statistically significant (all $p > 0.05$).

DISCUSSION

The study shows that one in ten CKD patients in this cohort had active TB, with all three TB phenotypes represented: pulmonary, extrapulmonary, and combined disease. The finding is clinically meaningful, although it must be read with caution because the cohort is small and the number of TB events is limited. The signal was most visible in dialysis-dependent CKD. Stage 5D patients formed only 8.3% of the total cohort, yet they accounted for one-third of active TB cases. The exact p-value was borderline rather than conventionally significant, but the magnitude

is difficult to ignore from a clinical screening perspective.

The pattern fits the broader evidence that TB risk rises as kidney function declines. Shu et al. reported a severity relationship between impaired kidney function and incident TB, with risk increasing from CKD Stage 3 and becoming particularly prominent in more advanced disease [13]. Min et al., using a nationwide cohort, found that dialysis patients with end-stage renal disease had a markedly higher risk of active TB than matched non-dialysis controls [14]. Our study cannot estimate incidence or causal risk because follow-up person-time was not available, but the Stage 5D clustering is directionally consistent with that literature.

The equal distribution of pulmonary, extrapulmonary, and combined TB also deserves attention. CKD patients do not always present with classic pulmonary disease alone; extrapulmonary disease may be missed when screening relies heavily on respiratory symptoms. A meta-analysis of CKD populations similarly noted that extrapulmonary TB contributes substantially to the disease profile [8]. In Indian tertiary-care practice, this point is practical. Fever, weight loss, lymphadenopathy, effusion, abdominal symptoms, or unexplained inflammatory illness in CKD should not be dismissed as uraemia, volume overload, or malnutrition without considering TB, especially when dialysis or advanced CKD is present.

The biomarker profile in the active TB group was also suggestive, though statistically imprecise. Patients with active TB had lower mean albumin and haemoglobin than those without TB. This is biologically plausible because malnutrition, chronic inflammation, reduced erythropoietin activity, and anaemia of CKD often overlap. Cegielski and McMurray described the reciprocal relationship between malnutrition and TB, where impaired cell-mediated immunity increases susceptibility, and TB itself worsens nutritional depletion [12]. In CKD clinics, serum albumin and haemoglobin are already monitored for renal care; using them as soft warning markers for infection risk may therefore be feasible without adding major cost.

Diabetes was common in the cohort, but it was not enriched among active TB cases. This should not be interpreted as absence of diabetes-related TB risk. The cohort was underpowered for such comparisons, and diabetes is a well-established TB risk factor in

larger systematic evidence [11]. Similarly, smoking and prior TB did not show a clear association here, largely because event counts were too small. The study is better viewed as a descriptive clinical signal than as a risk prediction model.

A key implication is that TB assessment in CKD should be stage-sensitive. Ruzangi et al. reported an association between CKD and TB in a comparative cohort from England, reinforcing that kidney disease itself can carry risk even outside the highest-burden settings [15]. More recent reviews of latent TB screening in CKD have argued for structured screening when patients approach dialysis initiation, precisely because the transition to kidney replacement therapy concentrates both biological susceptibility and healthcare exposure [16]. In India, where TB background prevalence remains high, this argument becomes stronger. Screening cannot rely on cough alone. It should include symptom review, nutritional and inflammatory clues, chest imaging when indicated, microbiological confirmation through NTEP-linked molecular diagnostics, and a low threshold for evaluating extrapulmonary disease.

This study has limitations. The sample size was modest, and only six active TB events were recorded, preventing adjusted modelling. The design was cross-sectional, so temporal inference and incidence estimation are not possible. The sampling method, institutional setting, microbiological confirmation pathway, drug-susceptibility profile, treatment outcomes, and ethics approval details should be inserted from the study protocol before submission. Albuminuria category was not available, so KDIGO CGA risk classification could not be fully reconstructed. Despite these constraints, the study offers a clinically coherent message: active TB is not rare among CKD patients, and dialysis-dependent CKD may represent a practical screening priority.

CONCLUSION

Active TB was recorded in 10.0% of CKD patients in this observational cohort, with pulmonary, extrapulmonary, and combined disease occurring in equal proportions. The highest numerical burden was seen among dialysis-dependent Stage 5D patients, while active TB cases also showed lower mean eGFR, albumin, and haemoglobin. Although statistical certainty was limited by small event numbers, the findings support stage-sensitive

TB screening in CKD care, particularly around advanced disease and dialysis transition.

Source of Funding

Nil.

Conflict of Interest

None declared.

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