Section: General Medicine

Original Research Article

IMPACT OF TYPE 2 DIABETES MELLITUS ON TREATMENT OUTCOMES IN PULMONARY TUBERCULOSIS: CLINICAL PROFILE AND GLYCEMIC STATUS AMONG NEWLY DIAGNOSED PULMONARY TUBERCULOSIS PATIENTS

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ABSTRACT

Background: The co-epidemic of Type 2 Diabetes Mellitus (T2DM) and pulmonary tuberculosis (PTB) constitutes a major public health challenge, particularly in low- and middle-income countries. Diabetes is recognised as a significant modifiable risk factor that adversely alters the clinical presentation, immune response, and treatment outcomes in PTB patients. The objective is to characterise the clinical and radiological profile of newly diagnosed PTB patients with T2DM, assess their glycaemic status at enrolment using fasting plasma glucose (FPG) and glycated haemoglobin (HbA1c), and determine the impact of glycaemic control on treatment outcomes.

Materials and Methods: A prospective, observational, hospital-based study conducted over 18 months at a tertiary care centre. Consecutive newly diagnosed PTB patients were enrolled and stratified into TB-DM (n=95) and TB-only (n=95) groups. Glycaemic status was assessed at baseline and at 2, 5, and 6 months. Primary outcomes included sputum conversion rates at 2 months, treatment success, failure, relapse, and mortality.

Results: TB-DM patients exhibited higher symptom burden, lower BMI, and atypical radiological features including lower lobe involvement (58.9% vs 21.1%, $p<0.001$) and bilateral cavitary disease (44.2% vs 24.2%, $p=0.006$). Mean HbA1c at enrolment was 9.8 ± 2.1%. Sputum culture conversion at 2 months was significantly delayed in TB-DM (54.7% vs 78.9%, $p<0.001$). Poor glycaemic control (HbA1c ≥8%) was independently associated with treatment failure (OR 3.12, 95% CI 1.67–5.83), relapse (OR 2.94, 95% CI 1.41–6.13), and death (OR 2.17, 95% CI 1.03–4.57).

Conclusion: T2DM significantly modifies the clinical and radiological phenotype of PTB and independently worsens treatment outcomes. Routine screening for diabetes in all newly diagnosed TB patients and integrated glycaemic management are essential for improving treatment success.

Keywords: Pulmonary tuberculosis, Type 2 diabetes mellitus, HbA1c, glycaemic control, treatment outcomes, TB-DM comorbidity, sputum conversion.

INTRODUCTION

Tuberculosis (TB) and Type 2 Diabetes Mellitus (T2DM) are two of the most formidable non-communicable and communicable disease burdens confronting global health systems in the twenty-first century. Their convergence has created a syndemic of particular concern for low- and middle-income countries (LMICs), where both conditions are highly prevalent and healthcare resources remain constrained.

By 2023, TB had reclaimed its position as the leading infectious cause of death globally, affecting an estimated 10.8 million people, with Mycobacterium tuberculosis (Mtb) typically infecting the lungs.[1] Simultaneously, the International Diabetes Federation Diabetes Atlas 2023 reports 537 million adults worldwide with diabetes, with projections rising to 643 million by 2030.2 The burden of both diseases is concentrated in the same geographies: South-East Asia and sub-Saharan Africa are disproportionately affected, with an expected increase in diabetes prevalence of approximately 99% in high-TB burden countries.[2]

The biological relationship between T2DM and TB is bidirectional and well established. Diabetic individuals face a threefold increased risk of developing TB disease, associated with higher bacterial loads, delayed sputum culture conversion, increased mortality, higher relapse rates, and elevated drug resistance — even during first TB episodes.[1] Conversely, active TB infection may worsen glycemic control through systemic inflammation, cortisol dysregulation, and direct pancreatic beta-cell dysfunction.

From an epidemiological perspective, the prevalence of DM among TB patients ranges from 1.9% to 45%, with an overall median global prevalence of 16% (IQR 9.0%–25.3%).[3] The mechanistic basis for poor outcomes in TB-DM comorbidity is multifactorial: chronic hyperglycemia impairs macrophage phagocytosis, reduces natural killer cell activity, suppresses Th1 cytokine responses, and promotes a pro-inflammatory milieu that facilitates mycobacterial survival and dissemination.[4] Additionally, rifampicin-mediated induction of cytochrome P450 enzymes accelerates the metabolism of commonly used oral hypoglycemic agents, further destabilizing glycemic control during anti-TB therapy.

Despite the growing recognition of this comorbidity, several gaps persist in the evidence base: data on the specific clinical and radiological phenotype of TB-DM patients in tertiary hospital settings remain heterogeneous; glycemic status at the time of TB diagnosis is infrequently assessed; and the quantitative relationship between HbA1c levels and specific adverse treatment outcomes requires further characterization, particularly in South Asian populations.

The present study was therefore designed to: (1) document the clinical, biochemical, and radiological profile of newly diagnosed PTB patients stratified by diabetic status; (2) characterize the glycemic status using FPG and HbA1c at enrolment and its evolution over the treatment period; and (3) determine the impact of glycemic control on treatment outcomes at end of therapy.

MATERIALS AND METHODS

This was a prospective, observational, comparative, hospital-based study conducted at the Department of Pulmonary Medicine and the Department of General Medicine in a Tertiary Care Hospital over an 18-month period.

Adults aged ≥18 years; newly diagnosed, bacteriologically confirmed pulmonary TB (sputum smear-positive and/or CBNAAT/GeneXpert-positive); drug-sensitive TB (rifampicin-susceptible on GeneXpert); were included in the study. Previously treated TB (Category II); HIV co-infection; extrapulmonary TB without pulmonary involvement; Type 1 diabetes, gestational diabetes, or steroid-induced hyperglycaemia; active malignancy; chronic liver disease (Child–Pugh B/C); end-stage renal disease; pregnancy or lactation; immunosuppressive therapy was excluded from study.

TB-DM Group: Newly diagnosed PTB with co-existing T2DM (FPG ≥126 mg/dL on two occasions, or HbA1c ≥6.5%, or prior documented diagnosis on anti-diabetic therapy), n=95.

TB-Only Group: Newly diagnosed PTB without diabetes (FPG <100 mg/dL and HbA1c <5.7%), n=95.

A structured pro forma documented: sociodemographic characteristics (age, sex, BMI, socioeconomic status, smoking, alcohol use, occupational history); clinical symptoms (cough, haemoptysis, fever, night sweats, weight loss, dyspnoea); duration of symptoms before diagnosis; family history of TB and diabetes; and comorbidities.

Microbiological: Sputum AFB smear microscopy (Ziehl–Neelsen stain, WHO grading), GeneXpert MTB/RIF assay, and mycobacterial culture on Lowenstein–Jensen (LJ) medium at enrolment, month 2, and month 5. Radiological: Digital chest X-ray (PA view) at enrolment, month 2, and end of treatment; CT chest in cases with atypical findings. Biochemical/Glycaemic: FPG, 2-hour PPPG, HbA1c (NGSP-certified HPLC method), CBC, ESR, LFT, RFT, serum albumin, and lipid profile at baseline and specified intervals.

All patients received Category I DOTS per NTEP guidelines: Intensive Phase — 2 months of HRZE; Continuation Phase — 4 months of HR. TB-DM patients received concurrent diabetes management with insulin or metformin-based regimens and

rifampicin-drug interaction counselling. HbA1c targets aimed for ≤7% during therapy.

Treatment outcomes were defined per WHO and NTEP 2022 criteria: Treatment Success (Cured or Completed); Treatment Failure (smear/culture positive at month 5 or later); Death (any-cause mortality during treatment); Lost to Follow-up (interrupted treatment for ≥2 consecutive months); Relapse (bacteriologically confirmed TB within 12 months of treatment success).

Optimal Glycaemic Control (OGC): HbA1c <7%. Suboptimal: HbA1c 7%–7.9%. Poor Glycaemic Control (PGC): HbA1c ≥8%.

Data were analysed using SPSS version 26.0 (IBM Corp., Armonk, NY). Continuous variables were expressed as mean ± SD or median with IQR. Categorical variables were expressed as frequencies and percentages. Inter-group comparisons used Student's independent t-test or Mann–Whitney U test for continuous variables, and chi-square or Fisher's exact test for categorical variables. Logistic regression identified independent predictors of adverse outcomes, reported as OR with 95% CI. Kaplan–Meier curves with log-rank test compared time to sputum culture conversion. P<0.05 was considered statistically significant.

The study was approved by the Institutional Ethics Committee. All participants provided written informed consent. Patient anonymity and data confidentiality were maintained throughout.

RESULTS

Baseline Sociodemographic and Clinical Characteristics

A total of 210 patients were screened; 20 were excluded (8 HIV-positive, 5 drug-resistant TB, 4 Type 1 DM, 3 Category II), leaving 190 patients — 95 in each group — for final analysis.

Table 1: Baseline Sociodemographic Characteristics

Variable	TB-DM (n=95)	TB-Only (n=95)	p-value
Mean age (years ± SD)	52.4 ± 11.3	38.6 ± 14.7	< 0.001
Male sex (%)	68 (71.6%)	61 (64.2%)	0.274
BMI (kg/m² ± SD)	18.9 ± 3.1	21.4 ± 3.8	< 0.001
Current/ex-smoker (%)	49 (51.6%)	38 (40.0%)	0.115
Alcohol use (%)	31 (32.6%)	27 (28.4%)	0.546
Socioeconomic class III–V (%)	72 (75.8%)	68 (71.6%)	0.531
Median symptom duration (weeks)	10 (IQR 6–16)	6 (IQR 4–10)	0.003

[Table 1] Baseline sociodemographic characteristics of newly diagnosed pulmonary tuberculosis patients stratified by diabetic status. TB-DM patients were significantly older, more nutritionally depleted, and presented with a substantially longer symptom duration prior to diagnosis. BMI = body mass index; IQR = interquartile range; SD = standard deviation.

Clinical Symptom Profile.

Table 2: Presenting Symptoms

Symptom	TB-DM (n=95)	TB-Only (n=95)	p-value
Productive cough	91 (95.8%)	88 (92.6%)	0.377
Haemoptysis	38 (40.0%)	29 (30.5%)	0.171
Fever (≥2 weeks)	79 (83.2%)	71 (74.7%)	0.159
Night sweats	72 (75.8%)	63 (66.3%)	0.162
Significant weight loss (≥5 kg)	67 (70.5%)	52 (54.7%)	0.028
Breathlessness (MRC grade ≥2)	51 (53.7%)	31 (32.6%)	0.004
Weakness/fatigue	83 (87.4%)	68 (71.6%)	0.008

[Table 2] Presenting symptoms in TB-DM versus TB-only groups. Significant weight loss, breathlessness, and generalised weakness/fatigue were significantly more prevalent in the TB-DM group at presentation. MRC = Medical Research Council dyspnoea scale.

Microbiological Profile

Sputum grading at enrolment demonstrated a higher smear positivity burden in TB-DM patients. High-grade positivity (3+ AFB) was observed in 38.9% of TB-DM patients versus 22.1% of TB-only patients (p=0.016). GeneXpert positivity was 100% in both groups by inclusion design. All isolates were rifampicin-susceptible.

Radiological Profile

Table 3: Radiological Features on Chest X-ray at Enrolment

Feature	TB-DM (n=95)	TB-Only (n=95)	p-value
Predominantly upper lobe disease	38 (40.0%)	69 (72.6%)	< 0.001
Lower lobe involvement	56 (58.9%)	20 (21.1%)	< 0.001
Bilateral disease	52 (54.7%)	33 (34.7%)	0.006
Cavitary lesions (any)	61 (64.2%)	40 (42.1%)	0.003
Multiple/bilateral cavities	42 (44.2%)	23 (24.2%)	0.005
Far-advanced disease (ATS)	29 (30.5%)	14 (14.7%)	0.010
Pleural effusion	18 (18.9%)	11 (11.6%)	0.161

[Table 3] Radiological features on chest X-ray at enrolment. TB-DM patients demonstrated significantly higher rates of lower lobe involvement, bilateral disease, cavitary lesions, and far-advanced disease compared to TB-only patients. ATS = American Thoracic Society disease extent classification.

Glycaemic Status at Enrolment

Table 4: Baseline Glycaemic Parameters in the TB-DM Group

Parameter	Value
Fasting Plasma Glucose (mg/dL)	214.6 ± 68.4
2-hr Post-Prandial Glucose (mg/dL)	286.3 ± 74.2
HbA1c (%) mean ± SD	9.8 ± 2.1
Newly detected T2DM at TB diagnosis	38 (40.0%)
Known T2DM (pre-existing)	57 (60.0%)
HbA1c <7% (Optimal Glycaemic Control)	11 (11.6%)
HbA1c 7%–7.9% (Suboptimal Control)	23 (24.2%)
HbA1c ≥8% (Poor Glycaemic Control)	61 (64.2%)

[Table 4] Baseline glycaemic parameters in the TB-DM group (n=95). Notably, 40.0% of TB-DM patients were newly detected at the time of TB diagnosis, underscoring the importance of universal glycaemic screening. The majority (64.2%) had poor glycaemic control (HbA1c ≥8%). HbA1c = glycated haemoglobin; SD = standard deviation; T2DM = type 2 diabetes mellitus.

Biochemical Parameters

TB-DM patients showed significantly lower serum albumin (3.1±0.6 vs 3.6±0.5 g/dL, p<0.001), higher ESR (98.4±32.1 vs 76.3±28.7 mm/hr, p<0.001), higher CRP (64.2±28.4 vs 41.7±22.6 mg/L, p<0.001), and lower total lymphocyte count (1240±410 vs 1680±520 cells/µL, p<0.001), consistent with a more pronounced systemic inflammatory and immunosuppressed state.

Sputum Conversion at 2 Months

Table 5: Sputum Smear/Culture Conversion at Month 2

	TB-DM (n=95)	TB-Only (n=95)	p-value
Smear conversion (%)	58 (61.1%)	78 (82.1%)	0.001
Culture conversion (%)	52 (54.7%)	75 (78.9%)	< 0.001

[Table 5] Sputum smear and culture conversion at month 2. Conversion rates were significantly lower in the TB-DM group at both time points. Among TB-DM patients, those with optimal glycaemic control had higher 2-month culture conversion (81.8%) compared to suboptimal (65.2%) and poor control (40.9%) (p<0.001).

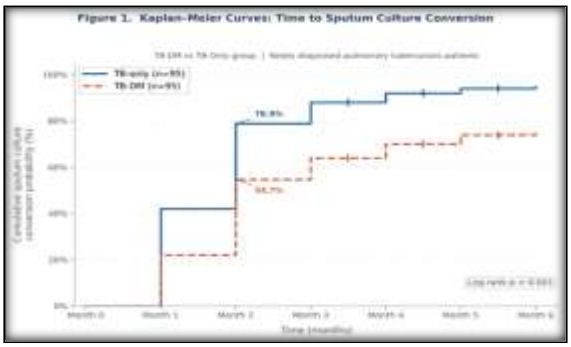

Figure 1: Kaplan–Meier curves showing the probability of sputum culture conversion

[Figure 1] Kaplan–Meier curves showing the probability of sputum culture conversion over the course of anti-TB therapy (months 0–6) in newly diagnosed pulmonary tuberculosis patients with T2DM (TB-DM group, dashed coral line, n = 95) versus without diabetes (TB-only group, solid blue line, n = 95). Conversion probability was significantly lower at all-time points in the TB-DM group. Tick marks (|) indicate censored observations. Log-rank test p < 0.001. TB = tuberculosis; DM = diabetes mellitus; T2DM = type 2 diabetes mellitus.

End-of-Treatment Outcomes

[Table 6] End-of-treatment outcomes stratified by diabetic status. Treatment success was significantly lower in the TB-DM group (67.4% vs 88.4%), falling well below the WHO 90% target for drug-susceptible TB. See Figure 2 for visual comparison.

Figure 2: Treatment Outcomes at end of therapy

[Figure 2] Grouped bar chart comparing end-of-treatment outcomes (%) between the TB-only group (blue, n = 95) and the TB-DM group (coral, n = 95). Treatment success was significantly lower in the TB-DM group (67.4% vs 88.4%, p < 0.001). Treatment failure (12.6% vs 3.2%, p = 0.015) and 12-month relapse (14.1% vs 3.6%, p = 0.018) were markedly higher in the TB-DM group. Asterisks denote level of statistical significance (*p < 0.05;

**p < 0.001). TB = tuberculosis; DM = diabetes mellitus.

Relapse Rates at 12 Months Post-Treatment

Among patients who achieved treatment success, relapse within 12 months was observed in 14.1% (9/64) of the TB-DM group versus 3.6% (3/84) in the TB-only group (p=0.018). All relapsing TB-DM patients had HbA1c ≥8% at enrolment.

Predictors of Adverse Treatment Outcomes

Table 6: Treatment Outcomes at End of Therapy (Month 6)

Outcome	TB-DM (n=95)	TB-Only (n=95)	p-value
Treatment success (cured + completed)	64 (67.4%)	84 (88.4%)	< 0.001
Treatment failure	12 (12.6%)	3 (3.2%)	0.015
Death (all-cause)	9 (9.5%)	3 (3.2%)	0.075
Lost to follow-up	6 (6.3%)	5 (5.3%)	0.764
Not evaluated	4 (4.2%)	0 (0%)	0.043

Table 7: Multivariate Logistic Regression — Predictors of Treatment Failure

Variable	Adjusted OR	95% CI	p-value
T2DM (vs no DM)	2.74	1.31–5.73	0.007
Poor glycaemic control (HbA1c ≥8%)	3.12	1.67–5.83	< 0.001
Cavitary disease (bilateral)	2.68	1.34–5.37	0.005
Far-advanced disease (ATS)	2.41	1.18–4.91	0.016
Low serum albumin (<3.0 g/dL)	2.03	1.02–4.05	0.044
High AFB smear grade (3+)	1.87	0.96–3.63	0.066
Age >50 years	1.62	0.83–3.16	0.158

[Table 7] Multivariate logistic regression identifying independent predictors of treatment failure. Poor glycaemic control (HbA1c ≥8%) was the strongest independent predictor (OR 3.12), followed by T2DM per se, bilateral cavitary disease, and hypoalbuminaemia.

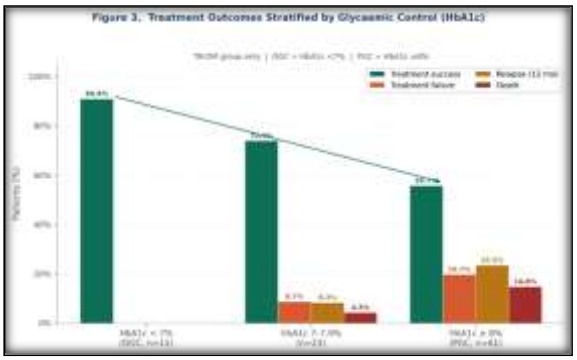

Figure 3: Treatment Outcomes Stratified by Glycaemic Control (Hba1c)

[Figure 4] Grouped bar chart showing treatment outcomes stratified by baseline HbA1c categories in the TB-DM group (n = 95): optimal glycaemic control (OGC, HbA1c < 7%, n = 11), suboptimal control (HbA1c 7%–7.9%, n = 23), and poor glycaemic control (PGC, HbA1c ≥ 8%, n = 61). A clear dose–response relationship is observed: treatment success declines (90.9% → 73.9% → 55.7%) and adverse outcomes — treatment failure, relapse at 12 months, and all-cause mortality — increase progressively with worsening glycaemic control.

DISCUSSION

This prospective study comprehensively describes the clinical phenotype, glycaemic status, and treatment outcomes in newly diagnosed PTB patients with and without T2DM at a tertiary care centre. Our findings broadly confirm, extend, and contextualise the growing body of evidence on the TB-DM syndemic.[8]

TB-DM patients were significantly older, more nutritionally depleted (lower BMI and albumin), and had a prolonged symptom duration before diagnosis. This delay — a median of 4 additional weeks — likely reflects atypical clinical presentation, diagnostic anchoring on chronic diabetic symptoms such as fatigue and weight loss, and the altered immune response that blunts the classic febrile response.[9-11]

The radiological differences between the two groups deserve emphasis. Lower lobe predominance was observed in 58.9% of TB-DM patients. Since 1927, when Sosman and Steidl first reported that diabetic patients with TB had more lower lung involvement, clinicians have recognised this important distinction.[14] Lower lobe tuberculosis may be misdiagnosed as community-acquired pneumonia or malignancy, and a high degree of suspicion is required in diabetic patients presenting with lower lobe opacities.[14] Bilateral cavitary disease was significantly more common in the TB-DM cohort (44.2% vs 24.2%), which has direct implications for infectiousness and treatment complexity.[13-17]

A critical finding was that 40.0% of TB-DM patients were newly detected at the time of TB diagnosis. This underscores the urgent need for bidirectional screening: all TB patients should be screened for diabetes, and all diabetic patients should be evaluated for TB symptoms. Point-of-care HbA1c testing has been validated for identifying hyperglycaemia severity among individuals with dual TB and DM, and should be integrated into TB diagnostic algorithms.[18-21]

The predominance of poor glycaemic control (HbA1c ≥8%) in our cohort (64.2%) reflects the compounding effect of TB infection itself on glycaemic homeostasis. Active TB triggers a pro-

inflammatory state characterised by elevated cortisol and counter-regulatory hormones, worsening insulin resistance. Simultaneously, rifampicin — a potent inducer of CYP450 enzymes — accelerates hepatic metabolism of sulphonylureas, reducing their bioavailability and efficacy. The analysis of optimal versus poor glycaemic control consistently reveals significant improvement in treatment outcomes among patients with better glycaemic control.[5]

Our treatment success rate of 67.4% in the TB-DM group falls well below the WHO target of 90% for drug-susceptible TB. Our multivariate analysis identified HbA1c ≥8% as the most powerful modifiable predictor of treatment failure, lending strong support to the hypothesis that glycaemic optimisation is not merely adjunctive but therapeutically essential in TB-DM management.[22-26]

Studies on the effect of poor glycaemic control in newly diagnosed smear-positive PTB patients with T2DM have reported a mean HbA1c of 10±2.6% in the poorly controlled group, with significantly higher symptom scores compared to optimally controlled patients,[3] — findings mirrored in our cohort. Baker et al. demonstrated that diabetes significantly increases the combined risk of failure and death among TB patients (RR 1.89 unadjusted, rising to 4.95 after adjustment), alongside an increased risk of relapse (RR 3.89).[12]

This was a single-centre study, limiting generalisability. Drug plasma concentration monitoring was not performed, precluding pharmacokinetic analysis. HOMA-IR was not calculated for all participants. Long-term follow-up beyond 12 months' post-treatment for relapse was not included. Quality-of-life measures and mental health outcomes were not assessed.

CONCLUSION

This study establishes that T2DM significantly modifies the clinical, radiological, and microbiological phenotype of newly diagnosed pulmonary tuberculosis and independently worsens treatment outcomes. Key findings are:

1. TB-DM patients present with atypical radiological features — predominantly lower lobe involvement, bilateral cavitary disease, and far-advanced disease — complicating timely diagnosis.
2. The majority of TB-DM patients had poor glycaemic control (HbA1c ≥8%) at enrolment, with 40% being newly detected diabetics.
3. Sputum culture conversion rates at 2 months were significantly lower in TB-DM patients, particularly among those with poor glycaemic control.
4. Treatment success rates were significantly reduced (67.4% vs 88.4%), with treatment failure and relapse rates markedly elevated in the TB-DM group.
5. Poor glycaemic control (HbA1c ≥8%) is the strongest independent predictor of treatment failure.

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