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Original Research Article

Metabolic risk in drug-resistant tuberculosis: prevalence of type 2 diabetes and its association with disease severity

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ABSTRACT

Background: Tuberculosis (TB), caused by *Mycobacterium tuberculosis*, remains a global health challenge, further complicated by the co-prevalence of Type 2 Diabetes Mellitus (T2DM), particularly in drug-resistant TB (DR-TB) cases. This dual burden worsens clinical severity, leads to poor treatment outcomes, and prolongs infectivity. This study assessed the prevalence of T2DM among DR-TB patients and evaluated clinical-radiological correlations, alongside the relationship between HbA1c and 7-point glucose profiles.
Methods: A cross-sectional study of 104 microbiologically confirmed DR-TB patients. Diabetes was diagnosed using ADA criteria. Demographics, clinical features, radiology, HbA1c, and 7-point glucose profiles were analyzed.
Results: T2DM prevalence was 41.3% in DR-TB patients, with significant associations found between T2DM, older age, and higher BMI. Radiological abnormalities included infiltrates (32.7% upper, 32.7% middle zones), consolidation (30.8%), and cavities (20.2%). No significant association was found between diabetes and radiographic extent ($p> 0.05$). HbA1c demonstrated a strong positive correlation with mean 7-point glucose values ($r=0.579$; $p=0.03$).
Conclusions: T2DM is highly prevalent among DR-TB patients. Routine screening and integrated TB–diabetes care are essential. HbA1c remains reliable for diagnosis and monitoring even during active TB disease.

Keywords: Drug-resistant tuberculosis, Type 2 diabetes mellitus, Clinical profile, Radiological findings

INTRODUCTION

Tuberculosis (TB) continues to pose an immense global health challenge despite decades of progress in diagnostics, therapeutics, and public health interventions. The World Health Organization (WHO) estimates that TB remains one of the top infectious killers worldwide, with millions affected annually despite the ambitious End TB Strategy aimed at global elimination.[1,2] This challenges compounded by the growing burden of drug-resistant tuberculosis (DR-TB), which represents a particularly formidable barrier to TB control due to its prolonged treatment duration, increased toxicity of second-line regimens, higher case fatality, and greater potential for community transmission.[3,4] Parallel to this, type 2 diabetes mellitus (T2DM) has emerged as one of the most pervasive non-communicable diseases globally, with prevalence nearly quadrupling in the last four decades and disproportionately rising in low- and middle-income nations that also bear the highest TB burden.[5-7] Compelling epidemiological evidence demonstrates a strong and multifaceted interaction between TB and diabetes, wherein

T2DM increases the risk of developing active TB by nearly three-fold, exacerbates disease progression, and adversely influences treatment outcomes.[8-11] The pathophysiological mechanisms responsible for this enhanced susceptibility include impaired cellular immunity, altered cytokine signaling, dysfunctional macrophage activation, and compromised phagocytic capability, which collectively diminish host defenses against Mycobacterium tuberculosis.[12-15]

The TB–diabetes intersection is now recognized as a "syndemic," wherein two epidemics interact synergistically within vulnerable populations, thereby amplifying morbidity, delaying diagnosis, and undermining treatment efficacy. Notably, India—harboring the world's largest number of TB and diabetes patients—represents the epicenter of this syndemic, making integrated management an urgent public health priority.[16-18] Studies from various Indian cohorts have consistently reported a rising prevalence of diabetes among TB patients, ranging between 25% and 40%, with even higher rates observed in drug-resistant TB populations due to prolonged disease course, recurrent infections, and corticosteroid exposure.[19-22] Evidence also suggests that coexistent diabetes may modify the clinical and radiological phenotype of pulmonary TB, contributing to more extensive parenchymal involvement, higher bacillary loads, cavitary lesions, and delayed sputum culture conversion.[23-25]

However, the available data on the clinical-radiological spectrum of DR-TB in diabetic patients remain limited, heterogeneous, and regionally variable. Furthermore, the diagnostic reliability of glycemic markers—particularly HbA1c—in the inflammatory milieu of active TB is an area of increasing clinical interest, given potential interactions between chronic infection, acute stress hyperglycemia, and erythrocyte turnover. In this context, the present study was undertaken to comprehensively evaluate the prevalence of T2DM among DR-TB patients, delineate their clinical and radiological characteristics, and examine the correlation between HbA1c and detailed glucose profiling. Understanding these interrelationships is essential to inform integrated TB–diabetes management strategies, guide risk stratification, and improve clinical outcomes in high-burden settings.

METHODS

This observational cross-sectional study was conducted at Himalayan Hospital, Dehradun, over a period of 12 months i.e. from 1st April 2022 to 31st May 2023. A total of 104 adults with microbiologically confirmed DR-TB (including at least rifampicin resistance) were enrolled. Exclusion criteria included pregnancy, age under 18 years, and inability to classify diabetic status. Data collected included demographic profile, symptomatology, past TB history, prior ATT exposure, fasting plasma glucose (FPG), HbA1c, and chest X-ray findings. Diabetes was defined using ADA criteria: FPG≥126 mg/dL or HbA1c ≥6.5%. A 7-point glucose profile was recorded for newly diagnosed diabetics before and after ATT initiation.

Statistical analysis was performed using SPSS 22. Qualitative data were expressed in frequencies and percentages, and quantitative data in descriptive terms. The One-sample Kolmogorov-Smirnov Test assessed normality, and the Chi- square test determined associations between clinical and radiological features in diabetic and non-diabetic patients, with $p<0.05$ considered significant. Correlation between HbA1c and mean glucose values were evaluated using Spearman's coefficient.

RESULTS

This study analyzed 104 patients diagnosed with DR-TB, with a focus on the prevalence of DM and the associated clinical and radiological features.

Prevalence of diabetes mellitus

The prevalence of DM among DR-TB patients was found to be 41.3%, indicating a significant comorbidity within this population (Table 1). The remaining 58.7% of patients were non-diabetic.

Table 1: Prevalence of diabetes mellitus among DR TB patients.

Diabetes mellitus	Frequency (N)	Percentage (%)
Present	43	41.3
Not present	61	58.7
Total	104	100

Table 2: Distribution of DR-TB patients by age, past history, ATT intake, and symptoms.

Category	Frequency (N)	Percentage (%)
Age group (years)		
<20	5	4.81
21-40	47	45.19
41-60	37	35.58
61-80	15	14.2
Past TB history		
Present	75	72.12
Not present	29	27.88
ATT intake		
HRZE	52	69.33
MDR – longer course	5	6.67
MDR - short course	18	24.00
Symptoms		
Cough	98	93.00
Fever	68	44.00
Shortness of breath	37	10.00
Hemoptysis	20	17.60
Chest pain	3	9.00

Age distribution

A notable observation was the higher prevalence of DR-TB with DM in younger adults, particularly those aged 21-40 years, who comprised 45.19% of the total sample (Table 2). Patients aged 41-60 years followed closely at 35.58%, while patients under 20 years and over 60 years made up smaller proportions at 4.81% and 14.42%, respectively.

Clinical symptoms

The clinical presentation of DR-TB patients commonly included symptoms such as cough (93.00%), fever (44.00%), and shortness of breath (10.00%) (Table 2). Other symptoms such as hemoptysis (17.60%) and chest pain (9.00%) were less frequently observed.

Past Tuberculosis History and Anti tuberculosis Therapy (ATT) Intake A significant number of DR-TB patients (72.12%) had a past history of tuberculosis (Table 2). Regarding ATT intake, 69.33% had received the HRZE regimen, while the remaining had undergone either the longer course (6.67%) or the shorter course (24.00%) of multidrug-resistant TB treatment.

Laboratory findings

Laboratory investigations revealed that 32.7% of the patients had fasting plasma glucose (FPG) levels ≥126 mg/dL, with the mean FPG being 136.12±77.90 mg/dL. The FPG range varied from 67 to 520 mg/dL. The mean HbA1c among patients was 8.45±2.41%, with 37.5% having HbA1c ≥ 6.5% (Table 3). However, HbA1c values were missing for 50% of the patients (non-diabetics).

Table 3: Laboratory and radiological findings in DR-TB patients.

Category	Frequency (N)	Percentage (%)	Category	Frequency (N)	Percentage (%)
Laboratory findings			**Radiological findings**		
Fasting plasma glucose < 126 mg/ dl	70	67.3	Normal chest x-ray	6	5.8
Fasting plasma glucose ≥126 mg/ dl	34	32.7	Consolidation	32	30.8
Mean baseline FPG (mg/dl)	136.12±77.90	-	Cavity	21	20.2
FPG range (mg/dl)	67-520	-	Upper lung zone infiltrates	34	32.7
HbA1c < 6.5%	13	12.5	Middle lung zone infiltrates	34	32.7
HbA1c ≥ 6.5%	39	37.5	Lower lung zone infiltrates	12	11.5
HbA1c missing values	52	50	Fibrotic lesion	10	9.6
Mean HbA1c (%)	8.45±2.41	-	Pleural effusion	1	0.9
HbA1c range (%)	5.2-15	-			

Table 4: Correlation between quadrant involved, chest X ray finding, T2DM.

Correlation matrix				
		Number of quadrant(s) involved	Diabetes mellitus status	Chest x-ray finding
Number of quadrant(s) involved	Spearman's RHO	--		
	DF	--		
	P-value	--		
Diabetes mellitus status	Spearman's RHO	0.173	--	
	DF	102	--	
	P-value	0.079	--	
Chest X-ray finding	Spearman's RHO	-0.011	0.09	--
	DF	102	102	--
	P value	0.911	0.362	--

Radiological Findings

Radiological assessment done using chest X ray, primarily showed lung infiltrates, with 32.7% of patients exhibiting upper lung zone involvement and another 32.7% showing middle lung zone infiltrates (Table 3). Consolidation was present in 30.8% of cases, while cavitary lesions were observed in 20.2%. Other radiological findings included fibrotic lesions (9.6%) and pleural effusion (0.9%). Despite these findings, there was no statistically significant correlation between diabetes status, lung involvement, and X- ray results (p value>0.05) (Table 4).

Table 5: Association of quadrant involvement with diabetes mellitus in patients with DR TB.

Number of quadrant(s) involved	Total	Diabetes mellitus present N (%)	Diabetes mellitus not present N (%)	P value
1	9	3(33.33)	6 (66.67)	0.073
2	48	11 (22.92)	37 (77.08)	
3	31	13 (41.94)	18 (58.06)	
4	16	9 (56.25)	7 (43.75)	

Table 6: 7-point glucose profile (newly diagnosed diabetics) - for 48 hours.

7-point glucose profile (mg/dl)	Day 1		Day 2	
	Mean	SD	Mean	SD
Before breakfast	131.36	35.65	122.50	30.14
After breakfast	198.57	64.69	195.43	63.89
Before lunch	141.86	32.92	139.79	27.74
After lunch	230.86	67.64	219.36	54.19
Before dinner	137.79	34.03	132.07	36.71
After dinner	248.86	57.78	241.93	55.50
Before sleeping	182.79	43.29	173.07	36.83
Mean glucose	175.14 +/- 40.95			

Table 7: Correlation of HbA1c with fasting blood glucose profile in drug resistant pulmonary tuberculosis patients with newly diagnosed diabetes mellitus.

HbA1c	Mean 7- point blood glucose (mg/dl)	Pearson's correlation coefficient (R)	P value
8.87±2.09	175.14±40.95	0.579	0.03

Correlation analysis

The correlation analysis between the number of lung quadrants involved, diabetes mellitus status, and chest X-ray findings revealed weak and non-significant correlations (Table 4). For instance, the correlation between the number of quadrants involved and DM status yielded a Spearman's RHO of 0.173 (p=0.079), indicating a weak relationship. Similarly, the correlation between diabetes status and chest X-ray findings had a Spearman's RHO of 0.09 (p=0.362), also showing a non-significant association.

These results suggest that while DM is a common comorbidity in DR-TB patients, its impact on the radiological presentation and the extent of lung involvement may not be strongly correlated. TB patients with involvement of all 4 quadrants had highest prevalence of diabetes mellitus (56.25%). There was increase in prevalence of diabetes in TB patients with involvement of more quadrants. However, there was no significant association between quadrant involvement and diabetes in TB patients (p>0.05). 7 Point Glucose Profile was done for DR-TB patients who were newly diagnosed with diabetes mellitus for 48 hours, with day 1 being before initiation of anti-tubercular drugs and day 2 being after initiation of anti-tubercular drugs, as shown in Table 6. The total mean glucose for 48 hours was found to be 175.14±40.95 mg/dL.

The mean blood glucose before breakfast was 131.36±35.65 mg/dL, after breakfast 198.57 ±64.69 mg/dL, before lunch 141.86±32.92 mg/dL, after lunch 230.86±67.64 mg/dL, before dinner 137.79±34.03 mg/dL, after dinner 248.86±57.78mg/dL and before sleeping 182.79±43.29 mg/dL. The highest mean blood glucose was after dinner while the lowest mean blood glucose was before breakfast. The mean 7-point blood glucose for 48 hours in DR TB patients with newly diagnosed T2DM was found to be 175.14± 40.95 mg/dL and HbA1c was found to be 8.87±2.09%. On studying the correlation between HbA1c and blood glucose profile, a positive correlation was found between them (r=0.579) with statistical significance (p=0.03).

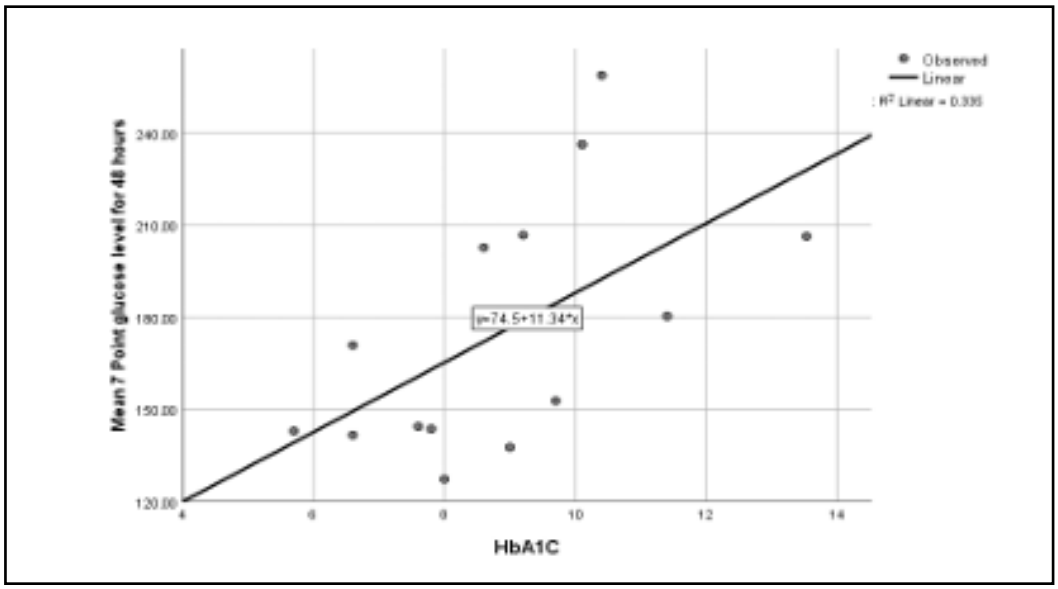

Figure 1: Scatter plot showing relation between mean 7-point glucose level for 48 hours and HbA1c.

DISCUSSION

The present study provides compelling evidence of a substantial coexistence of type 2 T2DM among patients with drug-resistant pulmonary tuberculosis (DR-TB), demonstrating a prevalence of 41.3%—markedly higher than estimates from the several regional and global cohorts.[14-16,21] This disproportionate burden reflects the complex syndemic interaction between TB and diabetes, wherein metabolic dysfunction amplifies susceptibility to Mycobacterium tuberculosis, exacerbates disease pathophysiology, and compromises immune effector functions central to mycobacterial clearance. Extensive literature attests that chronic hyperglycemia disrupts macrophage activation, impairs phagolysosome fusion, attenuates T-cell–mediated immunity, and blunts pro-inflammatory cytokine responses including TNF-α and IL-1β.[12-15] These mechanisms collectively predispose diabetic patients not only to primary infection but also to more severe and persistent forms of TB. Our demographic findings align with previous work showing that DR-TB disproportionately affects younger adults in India. Nearly half of our cohort belonged to the 21–40-year age group, a pattern corroborated by Venkatesh et al who noted similar age trends among MDR-TB patients.[18] The predominance of disease in economically productive age groups signals substantial societal and economic consequences, particularly when compounded by concomitant diabetes, which may prolong disability and impair treatment adherence. The prevalence of hyperglycemia in our cohort (fasting plasma glucose ≥126 mg/dL in 32.7% of patients) parallels findings from Sharma et al, reinforcing mounting evidence that occult diabetes or stress-related hyperglycemia frequently accompanies active TB.[19] Experimental and the clinical research suggests that the TB-associated inflammation may itself induce transient dysglycaemia via stress pathways, while persistent hyperglycemia further impairs pathogen containment.[9-11]

These interlocked pathways underscore the bidirectional nature of the TB–diabetes relationship. Radiological manifestations in our study were dominated by upper- and middle-zone infiltrates and consolidation, a pattern consistent with earlier Indian studies.[19-22] Although prior investigations have posited that diabetic individuals tend to present with more extensive parenchymal destruction, bilateral cavitation, and higher bacillary loads, our findings did not reveal a statistically significant association between diabetes status and radiographic severity. Similar heterogeneity has been reported in multi-country analyses, suggesting that radiological patterns may vary by ethnicity, glycemic control, duration of diabetes, and strain virulence.[23-25]

The lack of significant radiological associations in our study may also reflect the overarching severity of DR-TB itself, which could overshadow metabolic-driven variations. One of the most clinically salient findings of this study is the strong positive correlation between HbA1c and the 7-point glucose profile among newly diagnosed diabetics. Despite concerns that TB-related inflammation, altered erythrocyte turnover, or nutritional deficiencies might affect HbA1c reliability, our results support its continued utility in evaluating chronic glycemic burden in DR-TB patients. Similar conclusions have been drawn by Buasroung et al and Hall et al, who observed that HbA1c remains a useful metric for risk stratification and monitoring within TB–diabetes dual pathology.[4-14] Taken together, these findings highlight the urgent need for integrated TB–diabetes screening and management strategies. Global public health frameworks, including the WHO End TB Strategy, increasingly emphasize the incorporation of non-communicable disease surveillance—particularly diabetes screening—into TB programs.[2] Given the mounting evidence linking diabetes with delayed sputum conversion, increased relapse rates, higher mortality, and greater treatment complexity, early

identification and stringent glycemic control may confer significant benefits for DR-TB outcomes.[17-20]

Limitations of study

This study was conducted at a single tertiary care center with a limited sample size, which may restrict the generalizability of the findings. The cross-sectional design limits the ability to infer a causal relationship between diabetes mellitus and drug-resistant tuberculosis. Glycemic status was assessed at a single time point, raising the possibility of misclassification due to stress-related hyperglycemia. Additionally, the influence of residual confounding from unmeasured variables cannot be entirely excluded.

CONCLUSION

This study underscores the profound interplay between DR-TB and T2DM, revealing an exceptionally high prevalence of diabetes within the DR-TB population. The confluence of these two epidemics—each independently associated with high morbidity—creates a synergistic pathological environment that exacerbates disease severity and complicates management. Although radiological patterns did not differ significantly by diabetic status in our cohort, the broader body of evidence continues to suggest that glycemic dysregulation may influence disease extent, bacillary burden, and treatment response. Furthermore, the demonstrated correlation between HbA1c and detailed glucose profiling supports the ongoing use of HbA1c as an effective tool for metabolic assessment during active TB disease.

Given the significant burden of T2DM among DR-TB patients, systematic diabetes screening, early glycemic optimization, and integrated TB–diabetes care models are indispensable to improving patient outcomes. Strengthening collaborative frameworks between TB control programs and non-communicable disease services will be essential to mitigating the compounding impact of this syndemic on vulnerable populations. Continued research should focus on mechanistic pathways, longitudinal outcomes, and targeted interventions capable of interrupting the deleterious bidirectional cycle between metabolic dysfunction and drug-resistant tuberculosis.

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