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Glycaemic control and its relationship with diabetic complications in patients with pulmonary tuberculosis: a cross-sectional study from Western India

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Abstract

Background

Type 2 diabetes mellitus and tuberculosis frequently coexist in India. It is unclear whether glycated haemoglobin (HbA1c) measured at the time of tuberculosis diagnosis reflects the burden of diabetic complications and the severity of lung disease, or whether the acute infection distorts what a single HbA1c value means. We described the clinical, radiological and complication profile of patients with pulmonary tuberculosis and type 2 diabetes mellitus and compared these features by glycaemic control.

Methods

This cross-sectional study was conducted over 18 months in the Department of General Medicine of a tertiary teaching hospital in Western India. Forty-five adults with pulmonary tuberculosis and type 2 diabetes mellitus were enrolled consecutively and assessed clinically, biochemically and radiologically. Patients were stratified by HbA1c (< 9% or ≥ 9%) and compared using the independent-samples t-test and the chi-square or Fisher's exact test; a p-value < 0.05 was considered significant.

Results

Mean age was 56.5 ± 12.5 years and 62.2% were male. Glycaemic control was poor overall (mean HbA1c 9.52 ± 1.40%). Diabetic retinopathy was present in 57.8%, albuminuria in 44.4% and peripheral neuropathy in 26.7% of patients, and 55.6% needed insulin intensification during tuberculosis treatment. Comparing HbA1c < 9% (n = 20) with ≥ 9% (n = 25), retinopathy (p = 0.55), neuropathy (p = 1.00) and radiological cavitation (p = 0.77) did not differ significantly. Insulin intensification was significantly more frequent with poorer control (84.0% vs 20.0%, p < 0.001). Unexpectedly, albuminuria was more common in the better-controlled group (70.0% vs 24.0%, p = 0.003), a group with significantly longer diabetes duration (9.2 ± 4.5 vs 6.2 ± 3.9 years, p = 0.019).

Conclusions

A single HbA1c at tuberculosis diagnosis tracked with insulin requirement but not with most microvascular complications or radiological severity, and was inversely related to albuminuria. This may reflect cumulative, duration-dependent renal injury and anaemia-related underestimation of glycaemic burden. In patients with both conditions, complication screening may need to be guided by diabetes duration rather than a single HbA1c value, and insulin-based glycaemic management should be anticipated during the acute infective illness.

Trial registration:

Not applicable.

Background

India is home to one of the largest populations of people living with type 2 diabetes mellitus (T2DM) anywhere in the world, and clinicians managing these patients routinely encounter acute intercurrent illness as a complicating factor in glycaemic assessment. Tuberculosis (TB) is among the most consequential of these illnesses in the Indian context: India also carries close to a quarter of the world's TB burden, so the two conditions converge often in everyday practice [1]. This raises a question that is easy to state but harder to answer at the bedside: when a patient with longstanding, or newly recognised, T2DM presents with active pulmonary TB, does the glycated haemoglobin (HbA1c) drawn at that visit reliably describe the patient's glycaemic risk and complication burden, or is it being distorted, in either direction, by the infection itself?

The relationship between the two diseases is bidirectional, which is precisely what makes this question difficult. Diabetes increases the risk of developing active tuberculosis by roughly two- to three-fold, an effect attributed to impaired macrophage activation, defective neutrophil chemotaxis and phagocytosis, and blunted T-cell-mediated immunity in the setting of chronic hyperglycaemia [2, 3]. Conversely, active tuberculosis itself induces a catabolic, pro-inflammatory state that worsens insulin resistance, so that patients often present with, or are unmasked to have, markedly deranged glycaemic parameters at the time of TB diagnosis [4]. Diabetes also adversely affects tuberculosis treatment outcomes, with higher risks of treatment failure, death and relapse [5]. For the treating physician, this means a single HbA1c value obtained during active infection may not carry its usual interpretive weight, yet decisions about complication screening and treatment intensification are routinely made on exactly this value.

Poor glycaemic control is the principal driver of chronic diabetic microvascular complications (retinopathy, nephropathy and peripheral neuropathy), through mechanisms well characterised by the UK Prospective Diabetes Study and subsequent literature [6]. Whether active tuberculosis, superimposed on longstanding diabetes, further compounds this burden, or whether the acute metabolic derangement of TB confounds attempts to use a single HbA1c value to predict complication risk, is clinically relevant but understudied. A related question concerns disease severity itself: diabetic patients with tuberculosis have been reported, most influentially in cohorts from Indonesia and Taiwan, to have more extensive pulmonary involvement and a higher bacillary burden than their non-diabetic counterparts, and a large Taiwanese cohort found that the risk of cavitary disease on chest radiography rose steadily as HbA1c increased above 9% [7, 8]. Whether glycaemic control at presentation similarly predicts radiological severity within an already-diabetic Indian population, where both diseases are common, is unclear, and data specifically from Western India are limited.

The World Health Organization and the International Union Against Tuberculosis and Lung Disease have, for over a decade, recommended bidirectional screening for TB in diabetic patients and for diabetes in TB patients, and more recent guidance has pushed for integrated management of the two conditions

rather than parallel, siloed care [9, 10]. Translating this into everyday practice requires knowing which parameters, measured when, actually predict complications and severity in this comorbid population, and that knowledge remains limited.

Against this background, we undertook a cross-sectional study of patients with concomitant pulmonary tuberculosis and T2DM attending a tertiary care teaching hospital in Western India. Our aims were twofold: first, to describe the clinical, biochemical, radiological and treatment profile of this population, which remains sparsely documented from Western India; and second, to determine whether the degree of glycaemic control at presentation, using a pragmatic HbA1c cut-off of 9%, was associated with the burden of diabetic microvascular complications, radiological severity, or the need for insulin intensification during anti-tubercular therapy. Beyond describing a regional cohort, the study addresses a question of wider relevance: how far a single HbA1c value taken during active infection, in a population in which anaemia is very common, can be relied upon to guide complication screening and treatment decisions.

Methods

Study design and setting

This hospital-based, observational, cross-sectional study was conducted in the Department of General Medicine at a tertiary care teaching hospital in Western India (D. Y. Patil Medical College, Hospital and Research Centre, Navi Mumbai) over a period of 18 months. The study is reported in line with the STROBE statement for cross-sectional studies.

Participants

Patients attending the outpatient department or admitted under the Department of General Medicine during the study period were screened consecutively for eligibility. Patients aged 18 years or older with microbiologically confirmed pulmonary tuberculosis (sputum acid-fast bacilli positivity and/or a positive cartridge-based nucleic acid amplification test) or with radiologically compatible pulmonary tuberculosis in an appropriate clinical setting, and with either previously diagnosed or newly detected T2DM, were included. Patients with type 1 diabetes mellitus, pregnant women, patients with active malignancy, and those unwilling to provide consent were excluded. Forty-five patients meeting these criteria were enrolled.

Data collection

Demographic details, presenting symptoms, duration of symptoms, personal history (smoking, alcohol use), duration of diabetes, and comorbid illnesses were recorded on a structured proforma. All patients underwent a general physical and systemic examination, fundus examination, and a clinical assessment

for peripheral neuropathy. Laboratory investigations included a complete blood count, liver and renal function tests, a fasting lipid profile, fasting and post-prandial blood glucose, HbA1c, and a urine routine examination for albuminuria. Chest radiographs were obtained in all patients and reviewed for the presence, distribution and pattern (cavitary versus non-cavitary consolidation) of pulmonary lesions. Sputum was tested for acid-fast bacilli and by GeneXpert MTB/RIF wherever feasible.

Definitions

T2DM was diagnosed using American Diabetes Association criteria or a prior documented diagnosis on treatment [11]. Diabetic retinopathy was defined on dilated fundus examination, nephropathy by the presence of albuminuria on urine routine examination and/or an elevated serum creatinine, and peripheral neuropathy by clinical examination. For the purposes of comparative analysis, glycaemic control was dichotomised using an HbA1c threshold of 9%, a cut-off used in comparable published cohorts and one that separates patients with markedly uncontrolled diabetes from those with moderate-to-poor but less severe hyperglycaemia [8].

Statistical analysis

Data were entered in Microsoft Excel and analysed using SPSS version 27.0. Continuous variables are expressed as mean ± standard deviation (SD) and were compared between the HbA1c < 9% and HbA1c ≥ 9% groups using the independent-samples t-test. Categorical variables are expressed as frequencies and percentages and were compared using the chi-square test, or Fisher's exact test where an expected cell frequency was less than five. A two-tailed p-value < 0.05 was considered statistically significant. No formal sample-size calculation was performed; the sample comprised all eligible consecutive patients during the study period.

Results

Forty-five patients with concomitant pulmonary tuberculosis and T2DM were enrolled over the study period. The mean age was 56.5 ± 12.5 years (range 35–73 years), and nearly half the cohort (48.9%) was between 61 and 80 years of age. Males predominated (62.2%). Using Asian-specific cut-offs, 40.0% of patients were obese and 35.6% were overweight; only 11.1% were underweight. Cough and expectoration were each reported by 80.0% of patients, followed by fever (77.8%), weight loss (73.3%) and breathlessness (68.9%); most patients (68.9%) had been symptomatic for less than four weeks before presentation. Hypertension was present in 26.7%, a previous history of tuberculosis in 17.8%, and chronic obstructive pulmonary disease in 6.7%. Smoking and alcohol use were reported by 44.4% and 40.0% of patients, respectively (Table 1).

The diabetic profile reflected a population with long-standing and generally poorly controlled disease: the mean duration of diabetes was 7.5 ± 4.4 years, with 33.3% having had diabetes for more than 10 years. Mean fasting and post-prandial blood glucose were 180.9 ± 39.3 mg/dL and 256.7 ± 61.9 mg/dL

respectively, and the mean HbA1c was 9.52 ± 1.40% (range 7.3–12.9%), confirming that glycaemic control was suboptimal across the cohort as a whole. Anaemia (haemoglobin < 13 g/dL) was present in 91.1% of patients, and transaminase elevation was common (SGOT raised in 75.6%, SGPT in 62.2%), while dyslipidaemia was also frequent (elevated LDL cholesterol in 73.3%, low HDL cholesterol in 46.7%) (Table 1).

Table 1
Baseline demographic, clinical and biochemical characteristics of study participants (n = 45)

Variable	Value (n = 45)
Demographic and clinical characteristics	
Age (years), mean ± SD	56.5 ± 12.5
Age 61–80 years, n (%)	22 (48.9)
Male sex, n (%)	28 (62.2)
Body mass index (kg/m²), mean ± SD	26.7 ± 5.4
Obese (BMI > 27.5), n (%)	18 (40.0)
Overweight (BMI 23–27.5), n (%)	16 (35.6)
Symptom duration < 4 weeks, n (%)	31 (68.9)
Previous history of tuberculosis, n (%)	8 (17.8)
Hypertension, n (%)	12 (26.7)
Chronic obstructive pulmonary disease, n (%)	3 (6.7)
Smoking history, n (%)	20 (44.4)
Alcohol use, n (%)	18 (40.0)
Presenting symptoms	
Cough, n (%)	36 (80.0)
Expectoration, n (%)	36 (80.0)
Fever, n (%)	35 (77.8)
Weight loss, n (%)	33 (73.3)
Breathlessness, n (%)	31 (68.9)
Diabetic and biochemical profile	
Duration of diabetes (years), mean ± SD	7.5 ± 4.4
Diabetes duration > 10 years, n (%)	15 (33.3)
Fasting blood glucose (mg/dL), mean ± SD	180.9 ± 39.3
Post-prandial blood glucose (mg/dL), mean ± SD	256.7 ± 61.9
HbA1c (%), mean ± SD	9.52 ± 1.40
Anaemia (haemoglobin < 13 g/dL), n (%)	41 (91.1)

Variable	Value (n = 45)
Demographic and clinical characteristics	
Elevated SGOT, n (%)	34 (75.6)
Elevated SGPT, n (%)	28 (62.2)
Elevated LDL cholesterol, n (%)	33 (73.3)
Low HDL cholesterol, n (%)	21 (46.7)

SD, standard deviation; BMI, body mass index; HbA1c, glycated haemoglobin; SGOT, serum glutamic-oxaloacetic transaminase; SGPT, serum glutamic-pyruvic transaminase; LDL, low-density lipoprotein; HDL, high-density lipoprotein.

All 45 patients were sputum smear-positive for acid-fast bacilli, and GeneXpert testing confirmed rifampicin-sensitive disease in every case tested. Chest radiography showed an upper- and middle-zone predominant pattern: cavitary lesions were seen in 19 patients (42.2%), most often in the left upper lobe (15.6%), and consolidation was seen in 26 patients (57.8%), most often in the right upper lobe (24.4%). On fundus examination, 26 patients (57.8%) had evidence of diabetic retinopathy (classical in 46.7%, mixed in 11.1%). Peripheral neuropathy was present in 26.7% and albuminuria in 44.4% of patients, and 31.1% had an elevated serum creatinine. Diastolic dysfunction was noted on echocardiography in 35.6% of patients. Cutaneous markers of chronic hyperglycaemia (diabetic dermopathy, acanthosis nigricans and skin tags) were seen in 35.6%, 33.3% and 11.1% of patients respectively (Table 2).

With regard to treatment, 44.4% of patients continued on oral hypoglycaemic agents alone, while 55.6% required intensification of anti-diabetic therapy with insulin during the course of illness: 22.2% were shifted to human actrapid insulin, 20.0% to human mixed insulin, and 13.3% required a combination of actrapid insulin with insulin glargine (Table 2).

Table 2
Radiological, microbiological, diabetic complication and treatment profile of study participants (n = 45)

Variable	Value (n = 45)
Microbiological and radiological findings	
Sputum acid-fast bacilli positive, n (%)	45 (100)
GeneXpert rifampicin-sensitive, n (%)	45 (100)
Cavitary lesion (any), n (%)	19 (42.2)
Left upper lobe	7 (15.6)
Right middle lobe	6 (13.3)
Right upper lobe	6 (13.3)
Consolidation (any), n (%)	26 (57.8)
Right upper lobe	11 (24.4)
Right middle lobe	9 (20.0)
Left upper lobe	6 (13.3)
Diabetic complications	
Diabetic retinopathy (any), n (%)	26 (57.8)
Classical diabetic retinopathy	21 (46.7)
Mixed retinopathy	5 (11.1)
Peripheral neuropathy, n (%)	12 (26.7)
Albuminuria, n (%)	20 (44.4)
Elevated serum creatinine, n (%)	14 (31.1)
Diastolic dysfunction on echocardiography, n (%)	16 (35.6)
Diabetic dermopathy, n (%)	16 (35.6)
Acanthosis nigricans, n (%)	15 (33.3)
Anti-diabetic treatment	
Oral hypoglycaemic agents alone, n (%)	20 (44.4)
Insulin intensification (any), n (%)	25 (55.6)
Human actrapid insulin	10 (22.2)
Human mixed insulin	9 (20.0)

Variable	Value (n = 45)
Microbiological and radiological findings	
Actrapid insulin + insulin glargine	6 (13.3)

Percentages for lobar cavitary and consolidative lesions are calculated on the total cohort (n = 45) and are not mutually exclusive, as some patients had lesions in more than one location.

Comparison by glycaemic control status

Patients were stratified into those with HbA1c < 9% (n = 20) and HbA1c ≥ 9% (n = 25). The two groups were comparable for age, sex distribution, body mass index and smoking history (all p > 0.05). Notably, the better-controlled group had a significantly longer duration of diabetes (9.2 ± 4.5 years vs 6.2 ± 3.9 years, p = 0.019) (Table 3).

Table 3
Comparison of clinical, radiological and treatment characteristics according to glycaemic control status

Variable	HbA1c < 9% (n = 20)	HbA1c ≥ 9% (n = 25)	p value
Age (years), mean ± SD	58.0 ± 10.3	55.3 ± 14.1	0.473
Male sex, n (%)	13 (65.0)	15 (60.0)	0.767
BMI (kg/m²), mean ± SD	26.8 ± 5.4	26.6 ± 5.5	0.923
Duration of diabetes (years), mean ± SD	9.2 ± 4.5	6.2 ± 3.9	0.019
Smoking history, n (%)	9 (45.0)	11 (44.0)	1.000
Cavitary lesion on chest X-ray, n (%)	9 (45.0)	10 (40.0)	0.770
Diabetic retinopathy (any), n (%)	13 (65.0)	13 (52.0)	0.545
Peripheral neuropathy, n (%)	5 (25.0)	7 (28.0)	1.000
Albuminuria, n (%)	14 (70.0)	6 (24.0)	0.003
Insulin intensification required, n (%)	4 (20.0)	21 (84.0)	< 0.001

Continuous variables compared using the independent-samples t-test; categorical variables compared using the chi-square test or Fisher's exact test, as appropriate. Values in bold indicate statistical significance (p < 0.05). HbA1c, glycated haemoglobin; SD, standard deviation; BMI, body mass index.

Radiological severity did not differ meaningfully between groups: cavitary lesions were present in 45.0% of the HbA1c < 9% group and 40.0% of the HbA1c ≥ 9% group (p = 0.77). Diabetic retinopathy (65.0% vs 52.0%, p = 0.55) and peripheral neuropathy (25.0% vs 28.0%, p = 1.00) were also similar between groups.

Insulin intensification, by contrast, was strongly and significantly associated with poorer glycaemic control, required in 84.0% of the HbA1c ≥ 9% group compared with only 20.0% of the HbA1c < 9% group ($p < 0.001$). Albuminuria showed a significant association in the opposite direction to what is conventionally expected: it was more common in the better-controlled group (70.0% vs 24.0%, $p = 0.003$) (Table 3).

Discussion

This study asked a practical question relevant to anyone managing diabetes in a high-TB-burden setting: at the time a patient with T2DM is diagnosed with active pulmonary tuberculosis, does the HbA1c drawn at that visit meaningfully predict their microvascular complication burden and disease severity, or should it be interpreted with caution? Three findings stand out. Glycaemic control in this cohort was uniformly poor, confirming that active TB and poor diabetes control cluster together in practice. A single HbA1c value did not distinguish radiological severity or most microvascular complications. And, unexpectedly, albuminuria was more frequent among the better-controlled rather than the poorly controlled patients, a finding with direct implications for how complication screening should be timed and interpreted in this population.

The demographic profile, with a mean age in the late fifties and male predominance, is consistent with the broader epidemiology of tuberculosis-diabetes comorbidity reported from India and elsewhere, reflecting the rising prevalence of diabetes in middle-aged and older adults and their consequent vulnerability to active TB [2, 7]. The mean HbA1c of 9.52% in our cohort was well above conventional treatment targets, and every patient had at least one elevated glycaemic parameter. This is in keeping with the recognised bidirectional relationship between the two diseases: chronic hyperglycaemia predisposes to active tuberculosis, while the systemic inflammatory and catabolic effects of active infection further worsen insulin resistance, so that many patients are effectively at their metabolic worst at the point of TB diagnosis [3, 4].

Radiologically, our cohort showed the upper-lobe, cavity-predominant pattern classically associated with post-primary tuberculosis, rather than the atypical lower-zone or diffuse patterns sometimes attributed to diabetic hosts. When we compared radiological severity across the HbA1c cut-off, however, we found no significant association: cavitation was present in a similar proportion of well- and poorly-controlled patients. This contrasts with the findings of Chiang et al., whose large Taiwanese cohort demonstrated a graded increase in cavitation risk with rising HbA1c above 9% [8]. Several factors could explain the discrepancy. Chiang and colleagues compared diabetic against non-diabetic patients across a spectrum of glycaemic control; we, by contrast, restricted our comparison to patients who already had established diabetes and already had active tuberculosis, a narrower and arguably harder comparison to power with 45 patients. It is also plausible that a chest radiograph obtained at presentation reflects cumulative structural damage accrued over the preceding weeks to months of untreated infection, while a single HbA1c value reflects only the preceding roughly three months of glycaemic exposure, so the two measures may simply be capturing different time windows of a dynamic disease process, particularly

when, as here, bacillary burden, nutritional status, smoking and duration of symptoms before diagnosis were not adjusted for.

The burden of diabetic microvascular complications in our cohort was substantial (retinopathy in 57.8%, albuminuria in 44.4% and neuropathy in 26.7% of patients), underscoring that these were not newly diagnosed, mild diabetics but a population with considerable pre-existing metabolic injury. Retinopathy and neuropathy did not differ significantly by HbA1c category, which is broadly consistent with what is already known about these complications: they develop over years of cumulative glycaemic exposure and are also shaped by disease duration, hypertension, dyslipidaemia and genetic susceptibility, so a single contemporaneous HbA1c reading, especially one drawn during the metabolic stress of active TB, is unlikely to fully capture the glycaemic history responsible for their development [6].

The albuminuria finding merits closer scrutiny because it ran counter to what we, and the existing literature, would ordinarily expect. Patients with better current glycaemic control (HbA1c < 9%) had significantly more albuminuria than those with poorer control, and this same better-controlled group also had a significantly longer duration of diabetes. Read together, these two results suggest a coherent, if counter-intuitive, explanation: albuminuria in this cohort is more likely a marker of longstanding, cumulative diabetic nephropathy acquired over years of disease than a reflection of glycaemia at the moment of TB diagnosis. Patients with more advanced nephropathy may also have been under closer medical follow-up, with treatment already intensified or adjusted in ways that lowered their HbA1c by the time of enrolment, a form of reverse causality in a cross-sectional design. A further, biologically distinct possibility is that HbA1c itself becomes a less reliable index of true glycaemic burden in patients with anaemia, which was present in 91.1% of our cohort; a shortened erythrocyte lifespan lowers measured HbA1c independent of actual glucose exposure, a limitation explicitly recognised in current diagnostic guidance [11]. Whichever mechanism (or combination) is responsible, the practical implication is the same: an isolated HbA1c value at the time of TB diagnosis should not be used to reassure a patient, or their treating physician, that renal screening can be deferred.

Insulin intensification, in contrast to the complication measures, tracked glycaemic control exactly as expected, required in 84.0% of the poorly-controlled group versus only 20.0% of the better-controlled group. This internal consistency is reassuring: it confirms that HbA1c in our cohort was measuring something clinically meaningful about current metabolic state, even where it failed to predict complications shaped by longer time horizons, and it reinforces that active tuberculosis frequently precipitates a step-up in diabetic therapy, presumably reflecting both the direct hyperglycaemic stress of infection and the catabolic physiology of active disease.

This finding is consistent with current Indian guidance on managing hyperglycaemia in hospitalised and acutely unwell patients. The Research Society for the Study of Diabetes in India’s clinical practice guidelines for hyperglycaemia in hospitalised patients recommend insulin as the preferred therapy in this setting, with individualised glucose targets, over continuation of oral agents alone [12]. Our data support extending this expectation specifically to patients with active pulmonary tuberculosis: more than half of

our cohort required insulin intensification regardless of their prior treatment, and among patients with HbA1c ≥ 9% this rose to the large majority. For the practising clinician, the practical message is that oral therapy alone should not be assumed adequate once active TB is diagnosed, and glucose monitoring should be intensified proactively rather than reactively during anti-tubercular treatment, particularly in the first weeks when the catabolic stress of infection is greatest.

This study has important limitations. It was conducted at a single centre with a modest sample size of 45 patients, which limits statistical power, particularly for the subgroup comparisons, and generalisability. The cross-sectional design precludes any causal inference, and HbA1c was measured only once, at a point when active infection may itself have been distorting glycaemic parameters. Radiological severity was assessed qualitatively rather than with a validated scoring system, and we did not include a non-diabetic TB comparator group, so we cannot distinguish the effect of diabetes itself from the effect of glycaemic control within diabetes. Duration of diabetes, nutritional status, bacillary load and socioeconomic factors were not adjusted for in a multivariable model. These limitations notwithstanding, the dataset was collected prospectively using a standardised proforma at a single institution, which reduces measurement variability, and it adds a contemporary, well-characterised description of this comorbidity from a part of India, Western India, where such data remain scarce.

Conclusions

Patients with concomitant pulmonary tuberculosis and type 2 diabetes mellitus in this cohort had poor glycaemic control and a substantial burden of diabetic microvascular complications. A single HbA1c value obtained at the time of TB diagnosis tracked closely with the need for insulin intensification but did not reliably predict retinopathy, neuropathy or radiological severity, and was, if anything, inversely associated with albuminuria, a pattern most plausibly explained by the cumulative, duration-dependent nature of diabetic nephropathy and by the confounding effect of anaemia on HbA1c interpretation. For the practising clinician, these findings suggest that screening for diabetic complications in patients with TB-DM comorbidity should be guided by diabetes duration and standard complication surveillance rather than by a single contemporaneous HbA1c value, that insulin-based glycaemic management should be anticipated during the acute infective illness regardless of prior oral therapy, and that bidirectional screening and integrated management of both diseases should remain the standard of care.

Abbreviations

BMI
body mass index
HbA1c
glycated haemoglobin
HDL
high-density lipoprotein
LDL

low-density lipoprotein
SD
standard deviation
SGOT
serum glutamic-oxaloacetic transaminase
SGPT
serum glutamic-pyruvic transaminase
T2DM
type 2 diabetes mellitus
TB
tuberculosis
TB-DM
tuberculosis with diabetes mellitus.

Declarations

Ethics approval and consent to participate

This study was approved by the Institutional Ethics Committee for Biomedical and Health Research, D. Y. Patil Deemed to be University School of Medicine, Navi Mumbai, India (reference number DYP/IECBH/2023/268; approved 09 December 2023). The study was conducted in accordance with the Declaration of Helsinki. Written informed consent to participate was obtained from all participants prior to enrolment.

Consent for publication

Not applicable. The manuscript contains no individual person's data, images or videos.

Competing interests

The authors declare that they have no competing interests.

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Author Contribution

A.A and S.T conceptualised the study, collected and curated the data, performed the formal analysis, designed the methodology and wrote the original draft of the manuscript. S.P conceptualised the study, supervised the work, and reviewed and edited the manuscript. All authors read and approved the final manuscript.

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Data Availability

The datasets generated and/or analysed during the current study are available from the corresponding author on reasonable request.

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