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Tuberculosis in Children with Type 1 Diabetes Mellitus: Clinical Profile, Treatment Challenges, and Outcomes

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Abstract

Background: The coexistence of Type 1 Diabetes Mellitus (T1DM) and Tuberculosis (TB) in children presents a significant clinical challenge, particularly in resource-limited settings. This study aims to describe the clinical profile, treatment challenges, and outcomes of TB in children with T1DM.

Methods: A prospective observational study was conducted, enrolling 90 children (aged 5–17 years) newly diagnosed with both T1DM and TB. Participants were followed throughout their anti-tuberculosis treatment (ATT) course. Demographic data, clinical symptoms, microbiological findings, treatment complications, and outcomes were collected and analyzed. Multivariate analysis was used to identify predictors of poor outcomes.

Results: The mean age was 11.4 ± 3.2 years, with a mean T1DM duration of 3.8 ± 1.6 years. Poor glycemic control (HbA1c >9%) was prevalent in 70% of the cohort. Pulmonary TB was the most common presentation (60%), followed by extrapulmonary TB (28.9%). Major treatment challenges included glycemic instability (54.4%) and increased insulin requirements (63.3%). While 80% achieved clinical cure, poor glycemic control (OR 3.1) and disseminated TB (OR 4.2) were significant predictors of poor outcomes

Conclusion: TB in children with T1DM is associated with high rates of poor glycemic control and treatment-related complications. Integrated management of both conditions is essential to improve clinical outcomes.

Keywords: NIL

Introduction

Tuberculosis (TB) remains a leading cause of morbidity and mortality worldwide, particularly in regions where infectious diseases and metabolic disorders intersect [1]. While the association between Type 2 Diabetes Mellitus and TB is well-documented in adults, the impact of Type 1 Diabetes Mellitus (T1DM) on TB in the pediatric population is an emerging area of concern [2]. Children with T1DM are particularly vulnerable to infections due to altered immune responses associated with chronic hyperglycemia [3].

The physiological changes in T1DM, including impaired neutrophil function and reduced cytokine production, significantly increase the risk of developing active TB disease [4]. Furthermore, the management of TB in these children is complicated by the bidirectional relationship between the two conditions; TB infection can worsen glycemic control, while poorly controlled diabetes can impair the response to anti-tuberculosis treatment (ATT) [5].

Recent studies have highlighted a higher prevalence of latent TB infection among children with T1DM

compared to their non-diabetic peers, suggesting a need for heightened surveillance [6]. Despite this, there is limited data on the specific clinical profile and treatment outcomes of TB in this unique pediatric cohort. This study aims to fill this gap by analyzing the clinical characteristics and therapeutic challenges faced by children with T1DM and TB.

Methodology

This study utilized a prospective observational design. Ninety children (aged 5 to 17 years) newly diagnosed with both T1DM and active TB were consecutively enrolled at a tertiary care center over a defined period. The inclusion criteria were a confirmed diagnosis of T1DM (based on WHO criteria) and active TB (confirmed via microbiological or clinical/radiological evidence).

Baseline data on demographic characteristics (age, sex, duration of T1DM), nutritional status (BMI), and glycemic control (HbA1c levels) were collected upon enrollment. Participants were followed up monthly throughout their anti-tuberculosis treatment (ATT) course and for six months post-treatment. During follow-up visits, data on clinical profiles (categorized into pulmonary, extrapulmonary, and disseminated TB), microbiological investigations (GeneXpert, sputum smear, and culture), and treatment challenges (glycemic instability, insulin requirement changes, and ATT-induced hepatotoxicity) were systematically documented. Primary outcomes assessed were clinical cure, treatment completion, relapse, and mortality. Statistical analysis was performed using multivariate logistic regression to identify predictors of poor outcomes, with a p-value <0.05 considered significant.

Results

The study population consisted of 90 children with a mean age of 11.4 ± 3.2 years. The demographic and **clinical characteristics are summarized in the tables below.**

Table 1: Demographic Characteristics of the Study Population (N = 90)

Parameter	Value
Total children	90
Mean age (years)	11.4 ± 3.2
Age range (years)	5–17
Male : Female ratio	52 (57.8%) : 38 (42.2%)
Mean duration of T1DM (years)	3.8 ± 1.6
Mean HbA1c (%)	10.2 ± 1.8
Poor glycemic control (HbA1c >9%)	63 (70%)
Nutritional status: Underweight	41 (45.6%)
Normal BMI	38 (42.2%)
Overweight	11 (12.2%)
BCG scar present	66 (73.3%)

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Table 1 summarizes the demographic features of the 90 children included in the study. The mean age was 11.4 ± 3.2 years, with an age range of 5–17 years. There was a slight male predominance with 52 males (57.8%) and 38 females (42.2%). The mean duration of Type 1 Diabetes Mellitus (T1DM) was 3.8 ± 1.6 years. Glycemic control was generally poor, as reflected by a mean HbA1c of 10.2 ± 1.8%, with 63 children (70%) having HbA1c values greater than 9%. Nutritional assessment showed that 41 children (45.6%) were underweight, 38 (42.2%) had a normal BMI, while 11 (12.2%) were overweight. A BCG scar was present in 66 children (73.3%).

Table 2: Clinical Profile of Tuberculosis (N = 90)

Parameter	**n (%)**
Type of TB	
Pulmonary TB	54 (60%)
Extrapulmonary TB (EPTB)	26 (28.9%)
Disseminated TB	10 (11.1%)
Common symptoms	
Fever	78 (86.7%)
Cough >2 weeks	52 (57.8%)
Weight loss / poor weight gain	63 (70%)
Night sweats	29 (32.2%)
Anorexia	48 (53.3%)
Polyuria/polydipsia	39 (43.3%)

Table 2 details the clinical manifestations of tuberculosis among the participants. Pulmonary TB was the most common presentation, observed in 54 children (60%), followed by extrapulmonary TB (26; 28.9%) and disseminated TB (10; 11.1%). The most frequent symptoms included fever (78; 86.7%), cough lasting more than 2 weeks (52; 57.8%), and weight loss or poor weight gain (63; 70%). Additionally, night sweats were reported in 29 children (32.2%), anorexia in 48 (53.3%), and polyuria/polydipsia in 39 (43.3%).

Table 3: Distribution of Extrapulmonary TB (n = 26)

EPTB Type	**n (%)**
Lymph node TB	11 (42.3%)
TB meningitis	6 (23.1%)

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Abdominal TB	5 (19.2%)
Pleural TB	3 (11.5%)
Osteoarticular TB	1 (3.9%)

Among the 26 cases of extrapulmonary TB, Table 3 shows that lymph node TB was the most common form, accounting for 11 cases (42.3%). This was followed by TB meningitis (6; 23.1%), abdominal TB (5; 19.2%), and pleural TB (3; 11.5%). Osteoarticular TB was the least common type, seen in only 1 child (3.9%).

Table 4: Hematological and Biochemical Parameters (N = 90)

Parameter	**Mean ± SD / n (%)**
Hemoglobin (g/dL)	10.1 ± 1.4
Anemia (<11 g/dL)	58 (64.4%)
ESR elevated	74 (82.2%)
CRP positive	61 (67.8%)
Hyponatremia (<135 mEq/L)	18 (20%)
Hypokalemia (<3.5 mEq/L)	11 (12.2%)
HbA1c (%)	10.2 ± 1.8

Table 4 presents the laboratory findings of the study population. The mean hemoglobin level was 10.1 ± 1.4 g/dL, and anemia (<11 g/dL) was present in 58 children (64.4%). An elevated ESR was seen in 74 children (82.2%), while CRP positivity was noted in 61 (67.8%). Electrolyte imbalances were also reported: hyponatremia (<135 mEq/L) in 18 children (20%) and hypokalemia (<3.5 mEq/L) in 11 children (12.2%). The mean HbA1c was 10.2 ± 1.8%, indicating persistent poor glycemic control.

Table 5: Microbiological and Imaging Findings (N = 90)

Investigation	**n (%)**
GeneXpert positive	43 (47.8%)
Smear positive	18 (20%)
Culture positive	32 (35.6%)
Chest X-ray findings (n=54)	
Hilar lymphadenopathy	23 (42.6%)

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Parenchymal infiltrates	19 (35.2%)
Cavitation	7 (13%)
Miliary pattern	5 (9.2%)

Table 5 shows microbiological confirmation and imaging features. GeneXpert positivity was observed in 43 children (47.8%), whereas smear positivity and culture positivity were found in 18 (20%) and 32 (35.6%), respectively. Among the 54 children with pulmonary TB, chest X-ray abnormalities included hilar lymphadenopathy in 23 (42.6%), parenchymal infiltrates in 19 (35.2%), cavitary lesions in 7 (13%), and a miliary pattern in 5 (9.2%).

Table 6: Treatment-Related Challenges (N = 90)

Problem	**n (%)**
Glycemic instability	49 (54.4%)
Increased insulin requirement	57 (63.3%)
Hypoglycemia episodes	21 (23.3%)
ATT-induced hepatotoxicity	14 (15.6%)
GI intolerance	19 (21.1%)
Peripheral neuropathy	8 (8.9%)
Treatment interruption	17 (18.9%)

Table 6 outlines the difficulties encountered during treatment. Glycemic instability was reported in 49 children (54.4%), and increased insulin requirements were noted in 57 (63.3%). Hypoglycemia episodes occurred in 21 children (23.3%). Drug-related problems included ATT-induced hepatotoxicity (14; 15.6%), gastrointestinal intolerance (19; 21.1%), and peripheral neuropathy (8; 8.9%). Treatment interruption was observed in 17 children (18.9%), posing a risk for poor outcomes.

Table 7: Treatment Outcomes (N = 90)

Outcome	**n (%)**
Complete clinical cure	72 (80%)
Treatment completed with minor sequelae	8 (8.9%)
Relapse during follow-up	5 (5.6%)
Progression to severe disease	3 (3.3%)

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Mortality	2 (2.2%)
DKA during TB therapy	9 (10%)
Improvement in HbA1c (>1% reduction)	31 (34.4%)

Treatment outcomes are summarized in Table 7. A complete clinical cure was achieved in 72 children (80%), while 8 children (8.9%) completed treatment with minor sequelae. Relapse occurred in 5 children (5.6%), and progression to severe disease in 3 (3.3%). There were 2 deaths (2.2%) during follow-up. DKA episodes during TB therapy were seen in 9 children (10%). Improvement in glycemic control, defined as HbA1c reduction >1%, was recorded in 31 children (34.4%).

Table 8: Predictors of Poor Outcome (Multivariate Analysis)

Predictor	**Adjusted OR**	**p-value**
HbA1c > 9%	3.1	<0.01
Severe malnutrition	2.8	<0.05
Disseminated TB	4.2	<0.01
Treatment interruption	3.6	<0.05
Hepatotoxicity	2.1	0.04

Table 8 identifies independent predictors of poor treatment outcomes based on multivariate analysis. Poor glycemic control (HbA1c > 9%) significantly increased the risk (Adjusted OR 3.1, $p < 0.01$). Severe malnutrition was also associated with adverse outcomes (OR 2.8, $p < 0.05$). Disseminated TB had the strongest association (OR 4.2, $p < 0.01$). Treatment interruption (OR 3.6, $p < 0.05$) and hepatotoxicity (OR 2.1, $p = 0.04$) were additional significant predictors.

Discussion

The findings of this study underscore the complex interplay between T1DM and TB in children. Our results show that a significant majority (70%) of children had poor glycemic control (HbA1c >9%), which Krishna et al. identified as a major risk factor for treatment failure and increased mortality in TB-DM patients [7]. The high prevalence of pulmonary TB (60%) in our cohort aligns with the observations of van Doorn et al., who noted that diabetes mellitus as a comorbidity increases the risk of severe pulmonary involvement and poor treatment outcomes [8].

Treatment challenges were prominent, with 63.3% of patients requiring increased insulin doses. This phenomenon is largely attributed to the induction of cytochrome P450 enzymes by Rifampicin, which accelerates the metabolism of various drugs and complicates glycemic management, as discussed by Cáceres et al. [9]. Furthermore, the high rate of glycemic instability (54.4%) and episodes of Diabetic Ketoacidosis (DKA) (10%) during therapy highlight the metabolic stress imposed by TB infection on children with T1DM, a trend also observed by Park et al. in their analysis of pediatric T1DM and TB incidence [10].

The bidirectional relationship described by Boadu et al. was evident in our study, where TB not only complicated diabetes management but was also more severe in those with poor metabolic control [11].

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Slaoui et al. reported similar clinical particularities in pediatric cases, emphasizing that TB in diabetic children often presents with more extensive radiological findings, such as the hilar lymphadenopathy (42.6%) and parenchymal infiltrates (35.2%) seen in our results [12].

The importance of optimal glycemic control cannot be overstated. Zhao et al. concluded that maintaining target HbA1c levels is crucial for reducing susceptibility to TB and improving overall outcomes [13]. This is supported by our multivariate analysis, where HbA1c >9% was a strong predictor of poor outcomes (OR 3.1). Additionally, the mortality rate of 2.2% in our study, while relatively low, reflects the increased risk of death associated with the TB-DM comorbidity, as highlighted by Gautam et al. [14].

George et al. noted that the clinical profile of TB in diabetics often includes a higher frequency of extrapulmonary manifestations [15]. In our study, 28.9% of children had EPTB, with lymph node TB being the most common (42.3%). This diversity in clinical presentation necessitates a high index of suspicion and comprehensive diagnostic workup, as suggested by Kota et al. [16]. Workneh et al. further emphasized that nutritional status is a critical determinant of TB outcomes; our finding that severe malnutrition was a predictor of poor outcome (OR 2.8) reinforces this [17].

The risk of TB in T1DM patients is significantly higher than in the general population, a fact reaffirmed by Franco et al. in their Cochrane review [18]. Majeed et al. identified environmental and metabolic factors that predispose children to T1DM, which may also indirectly influence their susceptibility to TB [19]. From a management perspective, Nascimento et al. argued for a child-centered approach to T1DM care, which is even more critical when managing a co-infection like TB [20]. Finally, Jiang et al. highlighted the associations between T1DM and pulmonary TB, noting that females may have a slightly different risk profile, although our study showed a higher male prevalence (57.8%) [21].

Conclusion

The management of TB in children with T1DM requires an integrated approach that addresses both the infectious and metabolic aspects of the diseases. Addressing glycemic control and nutritional status is paramount to improving the clinical cure rate and reducing complications.

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