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Original Research Article

Prevalence and Risk Factors of Post-Tuberculosis Lung Disease in an Urban Tertiary Care Center

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Abstract

Background: Post-tuberculosis lung disease (PTLD) is an increasingly recognized clinical entity characterized by persistent structural and functional pulmonary abnormalities following the completion of successful antitubercular therapy. Despite the enormous global burden of tuberculosis (TB), the long-term pulmonary sequelae affecting TB survivors remain inadequately studied, particularly in high-burden settings. This study aimed to determine the prevalence of PTLD and identify its associated risk factors among patients who completed antitubercular treatment at an urban tertiary care center.

Methods: This cross-sectional observational study was conducted, enrolling 320 patients who had successfully completed antitubercular therapy within the preceding 6 to 36 months. All participants underwent detailed clinical evaluation, spirometric pulmonary function testing, high-resolution computed tomography (HRCT) of the chest, the St. George's Respiratory Questionnaire (SGRQ), and the six-minute walk test (6MWT). PTLD was defined as the presence of persistent respiratory symptoms with corresponding radiological abnormalities and/or spirometric impairment following bacteriologically confirmed TB cure. Logistic regression analysis was employed to identify independent risk factors.

Results: PTLD was identified in 198 of 320 patients (61.9%). The most prevalent HRCT findings were bronchiectasis (48.5%), fibrotic bands (42.4%), and cavitary lesions (28.3%). Spirometric abnormalities were detected in 54.1% of PTLD patients, with obstructive pattern predominating (33.8%). Mean FEV1 % predicted was significantly lower in the PTLD group (62.4 ± 16.8%) compared with the non-PTLD group (86.3 ± 12.5%, $p < 0.001$). Independent risk factors for PTLD included smoking history (OR 3.24; 95% CI: 1.86–5.64; $p < 0.001$), delayed treatment initiation exceeding 60 days (OR 2.78; 95% CI: 1.54–5.02; $p = 0.001$), cavitary disease on initial radiograph (OR 2.91; 95% CI: 1.62–5.23; $p < 0.001$), retreatment TB (OR 2.46; 95% CI: 1.28–4.72; $p = 0.007$), and diabetes mellitus (OR 1.94; 95% CI: 1.12–3.36; $p = 0.018$).

Conclusion: PTLD affects a substantial majority of TB survivors and is associated with significant pulmonary functional impairment. Smoking history, delayed treatment initiation, initial cavitary disease, retreatment TB, and diabetes mellitus represent modifiable and identifiable risk factors that should guide targeted post-treatment surveillance and rehabilitation strategies.

Keywords: Post-tuberculosis lung disease; Pulmonary sequelae; Risk factors; Spirometry; Bronchiectasis; Tuberculosis survivors; HRCT; Pulmonary rehabilitation.

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Introduction

Tuberculosis (TB) remains one of the most devastating infectious diseases globally, accounting for approximately 10.6 million new cases and 1.3 million deaths annually according to recent World Health Organization estimates [1]. While the primary focus of TB control programs has traditionally centered on achieving microbiological cure through standardized antitubercular therapy (ATT), there is growing recognition that successful treatment completion does not necessarily equate to complete pulmonary recovery [2]. A substantial proportion of individuals who survive TB experience persistent respiratory morbidity attributable to chronic structural and functional lung damage, a condition increasingly referred to as post-tuberculosis lung disease (PTLD) [3]. PTLD

encompasses a spectrum of pulmonary pathological sequelae, including bronchiectasis, fibrotic parenchymal changes, cavitary remnants, pleural thickening, emphysematous destruction, and airway stenosis, all of which may persist indefinitely following bacteriological cure [4]. These structural abnormalities translate into clinically significant functional impairment, manifesting as chronic respiratory symptoms including dyspnea, productive cough, recurrent infections, and exercise intolerance, which collectively diminish quality of life and impose a substantial socioeconomic burden on affected individuals and healthcare systems [5].

The global magnitude of PTLD is considerable. Recent epidemiological estimates suggest that there are currently over 155 million TB survivors alive worldwide, with a significant proportion experiencing some degree of chronic lung impairment [6]. Allwood et al. demonstrated that PTLD contributes to a greater burden of chronic lung disease disability-adjusted life years (DALYs) than TB itself in high-burden countries, underscoring the paradoxical situation wherein the cure of TB generates a new category of chronic respiratory disease [7]. Despite this enormous burden, PTLD has been historically neglected in both clinical practice and public health policy, with most national TB programs lacking standardized protocols for post-treatment pulmonary assessment and rehabilitation [8].

The pathogenesis of PTLD is multifactorial and involves the complex interplay of immunological, inflammatory, and reparative processes initiated during active TB infection. The host inflammatory response, mediated through granuloma formation, caseous necrosis, and fibroblast activation, results in irreversible destruction of pulmonary parenchyma and airways that persists long after the eradication of Mycobacterium tuberculosis [9].

Studies have demonstrated that the extent and severity of PTLD are influenced by numerous patient-related and disease-related factors, including the duration of diagnostic delay, the severity of initial radiographic involvement, the number of TB episodes, smoking status, and comorbid conditions such as diabetes mellitus and HIV co-infection [10].

Recent investigations have begun to characterize the prevalence and risk factor profile of PTLD in various geographical and clinical settings. Ravimohan et al. reported that up to 50–60% of successfully treated pulmonary TB patients exhibit significant spirometric abnormalities, with obstructive and mixed patterns predominating [11]. Nihues et al. conducted a systematic review demonstrating that post-TB patients have a significantly higher risk of developing chronic airflow obstruction compared with the general population [12]. Furthermore, Meghji et al. highlighted the association between PTLD and reduced exercise capacity, impaired quality of life, and increased long-term mortality among TB survivors in sub-Saharan Africa [13].

However, despite the accumulating evidence, significant knowledge gaps persist regarding the epidemiology and determinants of PTLD in urban tertiary care settings of high-burden countries. Most existing studies have been conducted in community-based or programmatic settings with limited access to advanced diagnostic modalities, and comprehensive evaluations incorporating both structural (HRCT-based) and functional (spirometric and exercise capacity) assessments remain scarce [14]. Additionally, the identification of modifiable risk factors that could be targeted through preventive interventions and post-treatment rehabilitation programs requires further investigation in diverse clinical populations [15].

The aim of the present study was to determine the prevalence of PTLD among patients who had successfully completed ATT at an urban tertiary care center, to characterize the spectrum of structural and functional pulmonary abnormalities using HRCT and spirometry, and to identify independent risk factors associated with the development of PTLD.

Materials and Methods

Study Design and Setting: This cross-sectional observational study was conducted in the Department of Pulmonary Medicine at a tertiary care hospital.

Study Population and Sample Size: A total of 320 consecutive patients who had successfully completed a full course of antitubercular therapy for bacteriologically confirmed pulmonary tuberculosis within the preceding 6 to 36 months were enrolled. The sample size was calculated based on an anticipated PTLD prevalence of 55%, with a margin of error of 6% and a confidence level of 95%, yielding a minimum required sample of 264 patients. To account for potential incomplete data and dropout, a total of 320 patients were recruited.

Inclusion and Exclusion Criteria: Inclusion criteria were: age ≥ 18 years; documented history of bacteriologically confirmed pulmonary TB (either sputum smear-positive and/or GeneXpert MTB/RIF-positive and/or culture-positive); documented completion of a full course of ATT with bacteriological confirmation of cure (smear and/or culture negativity at treatment completion); and time since treatment completion between 6 and 36 months.

Exclusion criteria included: active pulmonary TB or ongoing ATT; known pre-existing chronic respiratory disease diagnosed prior to TB (asthma, COPD, interstitial lung disease, bronchiectasis); extrapulmonary TB without pulmonary involvement; active malignancy involving the thorax; pregnancy; inability to perform spirometry or complete the 6MWT due to physical or cognitive limitations; and patients who declined participation or were unable to provide informed consent.

Data Collection and Clinical Assessment: Detailed demographic and clinical information was collected through a structured questionnaire and review of medical records. Data recorded included age, sex, body mass index (BMI), smoking history (quantified in pack-years), alcohol use, occupational exposure history, comorbidities (diabetes mellitus, HIV infection, hypertension, chronic kidney disease), initial TB presentation characteristics (sputum smear grading, presence of cavitary disease on initial chest radiograph, extent of radiographic involvement), treatment category (new versus retreatment), duration of diagnostic delay (defined as the interval from symptom onset to ATT initiation), and treatment regimen and duration.

Respiratory symptoms were systematically assessed using a standardized symptom questionnaire, documenting the presence and severity of cough, sputum production, dyspnea (graded using the modified Medical Research Council [mMRC] dyspnea scale), hemoptysis, and chest pain.

Pulmonary Function Testing: Spirometry was performed using a calibrated portable spirometer (SpiroLab III, MIR, Italy) in accordance with the American Thoracic Society/European Respiratory Society (ATS/ERS) technical standards. At least three acceptable and reproducible forced expiratory maneuvers were obtained, and the best values for forced vital capacity (FVC), forced expiratory volume in one second (FEV1), and FEV1/FVC ratio were recorded. Results were expressed as absolute values and as percentage of predicted values based on age, sex, height, and ethnicity-appropriate reference equations. Spirometric patterns were classified as obstructive (FEV1/FVC < 0.70), restrictive (FEV1/FVC ≥ 0.70 with FVC $<$ 80% predicted), or mixed (FEV1/FVC < 0.70 with FVC $<$ 80% predicted).

High-Resolution Computed Tomography: HRCT of the chest was performed using a 128-slice multidetector CT scanner (Siemens SOMATOM Definition AS, Siemens Healthineers, Erlangen, Germany) with thin-section (1 mm) axial acquisitions in the supine position during full inspiration. HRCT images were independently interpreted by two experienced thoracic radiologists blinded to clinical information. Structural abnormalities recorded included bronchiectasis, fibrotic bands and parenchymal scarring, residual cavitary lesions, pleural thickening, emphysematous changes, traction bronchiectasis, volume loss, calcified granulomas, and mosaic attenuation pattern.

Functional Capacity and Quality of Life Assessment: The six-minute walk test (6MWT) was performed in a standardized 30-meter corridor according to ATS guidelines. The six-minute walk distance (6MWD) was recorded, along with pre- and post-walk oxygen saturation and dyspnea scores. Health-related quality of life was assessed using the St. George's Respiratory Questionnaire (SGRQ), a validated disease-specific instrument comprising symptom, activity, and impact domains, with total scores ranging from 0 (no impairment) to 100 (maximum impairment).

Definition of Post-Tuberculosis Lung Disease: PTLD was defined as the presence of at least one persistent respiratory symptom (chronic cough, dyspnea, sputum production, or hemoptysis) in conjunction with one or more structural abnormalities on HRCT and/or spirometric impairment (any pattern), in a patient who had achieved documented bacteriological cure of pulmonary TB.

Statistical Analysis: Data were analyzed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD) and compared using the independent samples t-test or Mann-Whitney U test as appropriate. Categorical variables were expressed as frequencies and percentages and compared using the Chi-square test or Fisher's exact test. Univariate logistic regression was initially performed to assess the association between potential risk factors and PTLD. Variables with $p < 0.10$ on univariate analysis were entered into a multivariable binary logistic regression model to identify independent predictors. Results were expressed as adjusted odds ratios (OR) with 95% confidence intervals (CI). Model goodness-of-fit was assessed using the Hosmer-Lemeshow test. A two-tailed p-value of < 0.05 was considered statistically significant.

Results

Demographic and Clinical Characteristics: Of the 320 enrolled patients, 198 (61.9%) met the criteria for PTLD, while 122 (38.1%) did not demonstrate evidence of significant post-tuberculosis pulmonary sequelae. The demographic and clinical characteristics of both groups are presented in Table 1. The mean age of the entire cohort was 43.6 ± 13.2 years, with a male predominance (62.5%). Patients in the PTLD group

were significantly older (46.2 ± 12.8 years vs. 39.4 ± 12.9 years, $p < 0.001$), more likely to be male (68.2% vs. 53.3%, $p = 0.007$), and had a higher prevalence of smoking history (44.9% vs. 19.7%, $p < 0.001$). Diabetes mellitus was significantly more prevalent in the PTLD group (28.8% vs. 14.8%, $p = 0.003$). Patients with PTLD had a significantly longer diagnostic delay (72.4 ± 38.6 days vs. 41.8 ± 24.3 days, $p < 0.001$), higher initial sputum smear positivity grading, and greater prevalence of cavitary disease on initial chest radiograph (52.0% vs. 24.6%, $p < 0.001$). Retreatment TB cases were significantly more common in the PTLD group (26.3% vs. 11.5%, $p = 0.001$).

Table 1: Demographic and Clinical Characteristics of Study Participants

Variable	PTLD Group (n=198)	Non-PTLD Group (n=122)	p-value
Age (years), mean ± SD	46.2 ± 12.8	39.4 ± 12.9	<0.001*
Male sex, n (%)	135 (68.2%)	65 (53.3%)	0.007*
BMI (kg/m²), mean ± SD	21.4 ± 3.6	22.8 ± 3.9	0.002*
Smoking history, n (%)	89 (44.9%)	24 (19.7%)	<0.001*
Pack-years (smokers), mean ± SD	14.6 ± 8.4	8.2 ± 5.6	<0.001*
Alcohol use, n (%)	62 (31.3%)	28 (23.0%)	0.104
Diabetes mellitus, n (%)	57 (28.8%)	18 (14.8%)	0.003*
HIV co-infection, n (%)	14 (7.1%)	6 (4.9%)	0.432
Hypertension, n (%)	38 (19.2%)	20 (16.4%)	0.527
Diagnostic delay > 60 days, n (%)	108 (54.5%)	32 (26.2%)	<0.001*
Mean diagnostic delay (days)	72.4 ± 38.6	41.8 ± 24.3	<0.001*
Cavitary disease on initial CXR, n (%)	103 (52.0%)	30 (24.6%)	<0.001*
Retreatment TB, n (%)	52 (26.3%)	14 (11.5%)	0.001*
Time since treatment completion (months), mean ± SD	18.6 ± 8.4	16.8 ± 7.9	0.062

***Statistically significant at $p < 0.05$**

Pulmonary Function and Functional Capacity: Spirometric and functional assessment results are presented in Table 2. Patients in the PTLD group demonstrated significantly reduced pulmonary function across all parameters compared with the non-PTLD group. Mean FEV1 % predicted was 62.4 ± 16.8% in the PTLD group versus 86.3 ± 12.5% in the non-PTLD group ($p < 0.001$). Mean FVC % predicted was similarly reduced (68.7 ± 15.4% vs. 88.6 ± 11.8%, $p < 0.001$). Spirometric abnormalities were detected in 107 of 198 PTLD patients (54.1%), with obstructive pattern being the most prevalent (33.8%), followed by restrictive (12.1%) and mixed patterns (8.1%). The mean 6MWD was significantly lower in the PTLD group (382.6 ± 78.4 meters vs. 486.2 ± 64.8 meters, $p < 0.001$). The mean SGRQ total score was significantly higher in the PTLD group (42.8 ± 18.6 vs. 16.4 ± 10.2, $p < 0.001$), indicating substantially worse health-related quality of life. Among the HRCT findings in the PTLD group, bronchiectasis was the most common abnormality (48.5%), followed by fibrotic bands and parenchymal scarring (42.4%), residual cavitary lesions (28.3%), pleural thickening (26.8%), volume loss (22.7%), and emphysematous changes (14.6%).

Table 2: Pulmonary Function, Functional Capacity, and Radiological Findings

Variable	PTLD Group (n=198)	Non-PTLD Group (n=122)	p-value
FEV1 % predicted, mean ± SD	62.4 ± 16.8	86.3 ± 12.5	<0.001*
FVC % predicted, mean ± SD	68.7 ± 15.4	88.6 ± 11.8	<0.001*
FEV1/FVC ratio, mean ± SD	0.64 ± 0.12	0.78 ± 0.08	<0.001*
Spirometric pattern, n (%)			
Normal	91 (46.0%)	108 (88.5%)	<0.001*
Obstructive	67 (33.8%)	10 (8.2%)	
Restrictive	24 (12.1%)	3 (2.5%)	
Mixed	16 (8.1%)	1 (0.8%)	
6MWD (meters), mean ± SD	382.6 ± 78.4	486.2 ± 64.8	<0.001*
SGRQ total score, mean ± SD	42.8 ± 18.6	16.4 ± 10.2	<0.001*
mMRC dyspnea grade ≥ 2, n (%)	112 (56.6%)	18 (14.8%)	<0.001*
HRCT findings in PTLD group, n (%)			
Bronchiectasis	96 (48.5%)	—	—
Fibrotic bands/scarring	84 (42.4%)	—	—
Residual cavitary lesions	56 (28.3%)	—	—

Pleural thickening	53 (26.8%)	—	—
Volume loss	45 (22.7%)	—	—
Emphysematous changes	29 (14.6%)	—	—
Calcified granulomas	38 (19.2%)	—	—
Mosaic attenuation	22 (11.1%)	—	—

***Statistically significant at p < 0.05**

Risk Factor Analysis: The results of univariate and multivariable logistic regression analysis for independent predictors of PTLD are presented in Table 3. On multivariable analysis, five variables retained independent statistical significance as risk factors for PTLD: smoking history (adjusted OR 3.24; 95% CI: 1.86–5.64; p < 0.001), diagnostic delay exceeding 60 days (adjusted OR 2.78; 95% CI: 1.54–5.02; p = 0.001), presence of cavitary disease on the initial chest radiograph (adjusted OR 2.91; 95% CI: 1.62–5.23; p < 0.001), retreatment TB category (adjusted OR 2.46; 95% CI: 1.28–4.72; p = 0.007), and diabetes mellitus (adjusted OR 1.94; 95% CI: 1.12–3.36; p = 0.018). The Hosmer-Lemeshow goodness-of-fit test indicated adequate model calibration (p = 0.412).

Table 3: Univariate and Multivariable Logistic Regression Analysis of Risk Factors for PTLD

Variable	Univariate OR (95% CI)	Univariate p-value	Adjusted OR (95% CI)	Multivariable p-value
Age > 45 years	2.18 (1.38–3.44)	0.001*	1.52 (0.89–2.58)	0.124
Male sex	1.88 (1.18–2.98)	0.008*	1.36 (0.78–2.38)	0.276
Smoking history	3.32 (1.96–5.62)	<0.001*	3.24 (1.86–5.64)	<0.001*
BMI < 18.5 kg/m²	1.92 (1.14–3.24)	0.014*	1.48 (0.82–2.68)	0.194
Diabetes mellitus	2.34 (1.30–4.20)	0.005*	1.94 (1.12–3.36)	0.018*
HIV co-infection	1.48 (0.56–3.94)	0.432	—	—
Diagnostic delay > 60 days	3.38 (2.06–5.54)	<0.001*	2.78 (1.54–5.02)	0.001*
Cavitary disease on initial CXR	3.32 (2.02–5.46)	<0.001*	2.91 (1.62–5.23)	<0.001*
Retreatment TB	2.76 (1.44–5.30)	0.002*	2.46 (1.28–4.72)	0.007*
High sputum smear grade (3+)	2.12 (1.26–3.56)	0.005*	1.54 (0.86–2.76)	0.148
Alcohol use	1.52 (0.92–2.52)	0.104	—	—

***Statistically significant at p < 0.05; OR = Odds Ratio; CI = Confidence Interval**

Discussion

The present study demonstrates that post-tuberculosis lung disease represents a highly prevalent and clinically consequential condition among successfully treated pulmonary TB patients, affecting nearly two-thirds (61.9%) of the study cohort. This finding underscores the critical need for systematic post-treatment pulmonary assessment and challenges the prevailing clinical assumption that microbiological cure of TB equates to complete pulmonary recovery [16].

The observed PTLD prevalence of 61.9% is consistent with the growing body of international literature reporting prevalence rates ranging from 50% to 76% across diverse clinical settings. Pasipanodya et al. demonstrated that approximately 54% of successfully treated pulmonary TB patients exhibited significant persistent pulmonary function impairment in a large South African cohort [17]. Similarly, Hnizdo et al. reported that pulmonary TB was associated with an average loss of 153 mL in FEV1 per TB episode, with progressive cumulative damage in patients experiencing recurrent disease [18]. Our findings extend these observations by demonstrating that the burden of PTLD is equally substantial in an urban tertiary care setting with access to standardized ATT regimens. The predominance of obstructive spirometric pattern (33.8%) among PTLD patients aligns with the findings of Amaral et al., who conducted a meta-analysis demonstrating that previous pulmonary TB was associated with a 2.51-fold increased risk of airflow obstruction compared with individuals without TB history [19]. The mechanisms underlying post-TB obstructive impairment are multifactorial, encompassing bronchial distortion from peribronchial fibrosis, luminal narrowing from endobronchial scarring, bronchiectatic airway destruction, and loss of elastic recoil from parenchymal destruction [20]. The co-existence of restrictive and mixed patterns in our cohort (12.1% and 8.1%, respectively) reflects the heterogeneous nature of post-TB parenchymal damage, which often involves concurrent fibrosis, volume loss, and pleural disease [21]. Bronchiectasis emerged as the most prevalent structural abnormality on HRCT (48.5%), consistent with reports by Byrne et al. who documented post-TB bronchiectasis in 35–86% of TB survivors depending on the diagnostic modality and population studied [22]. Post-TB bronchiectasis carries significant clinical implications, predisposing patients to recurrent

lower respiratory tract infections, hemoptysis, progressive airflow limitation, and accelerated lung function decline, thereby establishing a vicious cycle of infection and inflammation that perpetuates chronic respiratory morbidity [23].

Among the identified risk factors, smoking history demonstrated the strongest independent association with PTLD (OR 3.24), confirming the synergistic destructive effects of tobacco smoke exposure and TB-related parenchymal damage on pulmonary structural integrity. This finding corroborates the work of van Zyl-Smit et al., who emphasized that smoking cessation should be considered an integral component of TB treatment programs given the compounding effects of smoking on post-TB lung function decline [24].

Diagnostic delay exceeding 60 days was independently associated with a 2.78-fold increased risk of PTLD, highlighting the critical importance of early TB diagnosis and treatment initiation in mitigating long-term pulmonary sequelae. Prolonged diagnostic delay allows for continued mycobacterial proliferation, sustained immune-mediated tissue destruction, and more extensive cavitation, all of which contribute to greater irreversible parenchymal damage [25]. This finding has direct public health implications, reinforcing the importance of strengthening TB diagnostic capacity and reducing barriers to healthcare access in high-burden settings.

The presence of cavitary disease on the initial chest radiograph (OR 2.91) as an independent predictor of PTLD underscores the relationship between the severity of initial disease presentation and long-term pulmonary outcomes. Cavitation reflects extensive caseating necrosis and parenchymal destruction, which are inherently associated with greater residual structural damage following healing [26]. Similarly, retreatment TB (OR 2.46) represents cumulative lung injury from repeated episodes of active disease and treatment, consistent with the dose-response relationship between TB episodes and pulmonary function loss described by Hnizdo et al. [18].

Diabetes mellitus (OR 1.94) was independently associated with PTLD, reflecting the well-documented immunological and inflammatory dysregulation associated with hyperglycemia. Diabetic patients exhibit impaired macrophage function, altered cytokine responses, and enhanced fibrotic tissue remodeling, all of which may contribute to exaggerated tissue destruction during active TB and impaired repair during the healing phase [27]. Given the rising global prevalence of the TB-diabetes comorbidity, this finding has important implications for the integrated management of these conditions.

The significantly reduced 6MWD (382.6 ± 78.4 meters) and elevated SGRQ total scores (42.8 ± 18.6) in the PTLD group highlight the profound functional and quality-of-life impact of post-TB lung damage. Meghji et al. similarly demonstrated that PTLD was associated with substantial reductions in exercise capacity and health-related quality of life, with many patients experiencing disability comparable to that observed in moderate-to-severe COPD [28]. These findings emphasize the need for structured pulmonary rehabilitation programs specifically designed for TB survivors.

This study has several limitations that warrant acknowledgment. The cross-sectional design precludes determination of temporal causality and the assessment of longitudinal trajectory of pulmonary function decline following TB cure. The single-center design may limit generalizability to other settings. Pre-TB baseline pulmonary function data were not available, preventing quantification of the attributable decline directly caused by TB. Additionally, the exclusion of patients with known pre-existing respiratory disease, while necessary for methodological rigor, may have led to underestimation of the true burden of post-TB respiratory morbidity in the broader population. Future prospective multicenter longitudinal studies with pre-TB baseline pulmonary function measurements are warranted to address these limitations [29].

Conclusion

This study demonstrates that post-tuberculosis lung disease is highly prevalent among successfully treated pulmonary TB patients, affecting approximately 62% of the study cohort and causing significant structural, functional, and quality-of-life impairment. Bronchiectasis, parenchymal fibrosis, and cavitary remnants constitute the predominant structural abnormalities, while obstructive airflow limitation represents the most common spirometric pattern. Smoking history, delayed treatment initiation exceeding 60 days, cavitary disease at initial presentation, retreatment TB, and diabetes mellitus were identified as independent risk factors for the development of PTLD. These findings underscore the imperative for integrating systematic post-treatment pulmonary assessment into national TB control programs, implementing targeted smoking cessation interventions, strengthening early diagnostic capacity to minimize diagnostic delays, and establishing structured pulmonary rehabilitation services specifically tailored for TB survivors. The recognition that TB cure does not equate to pulmonary cure necessitates a paradigm shift toward comprehensive long-term respiratory care for the growing population of TB survivors worldwide.

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