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Incidence and Pattern of Obstructive Airway Disease in Treated Patients of Pulmonary Tuberculosis: A Retrospective Study

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ABSTRACT

Introduction: Post-tuberculosis lung disease (PTLD) is increasingly recognized as a significant cause of chronic respiratory morbidity following successful treatment of pulmonary tuberculosis (PTB). Obstructive airway disease (OAD) is an important manifestation of PTLD, resulting from persistent airway inflammation, fibrosis, bronchiectasis, and structural lung damage. This study aimed to determine the incidence and pattern of OAD among patients who had completed anti-tubercular therapy (ATT).

Materials and Methods: This retrospective observational study included 50 adults with microbiologically cured PTB who presented with persistent respiratory symptoms after completion of ATT at a tertiary care teaching hospital. Demographic, clinical, radiological, and spirometric data were retrieved from medical records. Spirometry findings were analysed from case records. Chest radiographs were reviewed for presence of post-tuberculous sequelae. Statistical analysis was performed by using SPSS version 23.0 version. For statistical purposes p-value less than 0.05 was considered significant.

Results: Amongst the studied cases there were 34 (68%) males and 16 (32%) females. The mean age of studied cases was found to be 45.2±13.4 years. Most common presenting symptom was dyspnea (44%) followed by cough (34%) and wheeze (22%). Spirometry demonstrated OAD in 11 patients (22%). Radiological abnormalities included extensive fibrosis (8%), bronchiectasis (8%) and cavitary fibrosis (6%). 39 (78%) patients showed no significant residual sequelae on chest radiography. OAD was more frequent among males (26.5% vs. 12.5%) and tended to increase with longer duration after ATT completion, reaching 60% in patients evaluated more than five years after treatment, although these associations were not statistically significant.

Conclusion: Obstructive airway disease is a common long-term sequela of treated pulmonary tuberculosis, even in non-smokers without pre-existing respiratory disease. Persistent respiratory symptoms after ATT should prompt spirometric evaluation to facilitate early diagnosis and appropriate management. Routine post-treatment respiratory follow-up may improve quality of life and reduce long-term respiratory morbidity among tuberculosis survivors.

Keywords: *Airway Obstruction, Post-Tuberculosis Lung Disease, Pulmonary Function Tests, Pulmonary Tuberculosis, Spirometry.*

INTRODUCTION

Pulmonary tuberculosis (PTB) remains one of the major public health concerns particularly in developing countries. According to the World Health Organization (WHO), tuberculosis continues to rank among the leading infectious causes of morbidity and mortality worldwide particularly in low- and middle-income countries.[1] While microbiological cure is the primary objective of anti-tubercular therapy (ATT), there is increasing evidence that suggests many patients continue to experience persistent respiratory symptoms even after successful completion of treatment. These post treatment sequelae

include chronic cough, dyspnea and reduced exercise tolerance. Post-tuberculosis lung disease (PTLD) has emerged as a distinct clinical entity characterized by chronic respiratory impairment following microbiological cure of tuberculosis. Among the various manifestations of PTLD, obstructive airway disease (OAD) has gained increasing recognition because of its substantial contribution to chronic respiratory disability and healthcare utilization. The burden of post-tuberculosis obstructive airway disease is expected to rise with improving tuberculosis treatment success rates, emphasising the need for better understanding of its epidemiology and clinical characteristics. [2]

The pathophysiological basis of obstructive airway disease following pulmonary tuberculosis is multifactorial. It may involve factors such as extensive airway inflammation, fibrosis, bronchiectasis and destruction of lung parenchyma. Persistent inflammatory responses initiated during active Mycobacterium tuberculosis infection may continue even after microbiological cure thereby leading to progressive remodeling of both large and small airways. Unlike classical smoking-related COPD, post-tuberculosis obstructive airway disease often develops in younger individuals and may present with heterogeneous radiological and spirometric abnormalities. Pulmonary function testing commonly demonstrates irreversible airflow limitation with reduced forced expiratory volume in one second (FEV_1), decreased FEV_1/FVC ratio, and varying degrees of mixed restrictive and obstructive ventilatory defects depending upon the extent of parenchymal involvement. Recognition of these mechanisms has shifted attention toward long-term respiratory monitoring of patients successfully treated for pulmonary tuberculosis.[3]

Several epidemiological studies have demonstrated a significant association between previous pulmonary tuberculosis and subsequent development of chronic airflow obstruction independent of smoking status. Population-based surveys and longitudinal cohort studies have consistently shown that individuals with a history of pulmonary tuberculosis have a two- to four-fold higher risk of developing chronic obstructive airway disease compared with those without prior tuberculosis. The prevalence of airflow obstruction among treated tuberculosis patients has been reported to range from 18% to over 60%, depending upon study population, disease severity, duration of follow-up, and spirometric criteria employed. Factors such as delayed diagnosis, recurrent tuberculosis, multidrug-resistant disease, extensive radiographic involvement, cavitary lesions, smoking, biomass fuel exposure, diabetes mellitus, malnutrition, and advancing age further increase the likelihood of chronic airway dysfunction. In countries with high tuberculosis prevalence, post-tuberculosis lung disease is increasingly recognized as an important contributor to the global burden of chronic respiratory diseases. However, despite growing awareness, many patients remain undiagnosed because respiratory symptoms after ATT completion are frequently attributed to residual scarring rather than objective pulmonary function impairment. Early identification through spirometry and radiological assessment may facilitate timely institution of bronchodilator therapy, pulmonary rehabilitation, smoking cessation counseling, vaccination, and long-term follow-up strategies aimed at reducing disease progression and improving functional outcomes.[4]

The pattern of obstructive airway disease in treated pulmonary tuberculosis patients is heterogeneous. Some patients may exhibit isolated small airway dysfunction, whereas others develop moderate-to-severe fixed airflow obstruction resembling COPD. The severity of spirometric abnormalities in these cases may correlate with extent of parenchymal destruction caused by the disease.[5] Retrospective hospital-based studies provide valuable insight into the incidence, spectrum, and predictors of post-tuberculosis obstructive airway disease by utilizing routinely available clinical records, radiological findings, and pulmonary function test data. Such evidence is particularly relevant in resource-limited settings where tuberculosis remains endemic and systematic post-treatment respiratory surveillance is not routinely practiced. Therefore, the present retrospective study aims to determine the incidence and characterize the pattern of obstructive airway disease among patients who have completed treatment for pulmonary tuberculosis, thereby contributing to the existing body of knowledge and helping bridge the current evidence gap regarding post-tuberculosis chronic airway dysfunction.

MATERIALS AND METHODS

This was a retrospective observational study conducted in the Department of Medicine at a tertiary-care teaching hospital. The study included 50 patients with a documented history of successfully treated pulmonary tuberculosis (PTB) who subsequently attended the Medicine outpatient department with persistent respiratory symptoms. These symptoms included dyspnea, wheezing or chronic cough. Only those patients who had completed a full course of anti-tubercular therapy (ATT) and had been declared microbiologically cured. Patient confidentiality was maintained throughout the study by anonymizing all personal and identifiable information during data collection and analysis.

Medical records were reviewed using a predetermined structured data collection proforma. Demographic characteristics such as age and gender were recorded along with clinical details such as presenting complaints, duration since completion of ATT and previous tuberculosis treatment history. A detailed clinical examination performed during the outpatient visit was documented from the case records. Particular attention was given to the presence and duration of respiratory complaints which developed after completion of tuberculosis treatment.

Radiological evaluation was performed using plain posteroanterior chest radiographs available in the hospital records. Chest radiographs were reviewed for evidence of post-tuberculous structural sequelae including extensive fibrosis, cavitary

lesions with fibrosis, bronchiectasis, and the absence of significant residual abnormalities. Radiological findings were categorized according to the predominant abnormality observed and were correlated with pulmonary function findings.

Pulmonary function assessment was performed in all patients using spirometry according to the American Thoracic Society/European Respiratory Society (ATS/ERS) recommendations. Only spirometry recordings fulfilling standard acceptability and reproducibility criteria were included in the final analysis. Forced vital capacity (FVC), forced expiratory volume in one second (FEV_1), and the FEV_1/FVC ratio were recorded. Obstructive airway disease was diagnosed on the basis of persistent respiratory symptoms together with spirometric evidence of airflow obstruction and compatible radiological findings attributable to previous pulmonary tuberculosis. Patients without evidence of airflow obstruction on spirometry were categorized as having normal lung function.

The primary outcome measure was the incidence of obstructive airway disease among previously treated pulmonary tuberculosis patients. Secondary outcome measures included the pattern of radiological sequelae associated with airflow obstruction and the relationship between the interval since completion of ATT and the occurrence of obstructive airway disease.

All collected data was entered into a structured database and SPSS version 23.0 was used for statistical analysis. Continuous variables were expressed as mean ± standard deviation (SD). Categorical variables were documented as frequencies and percentages. Associations between categorical variables were analysed using the Chi-square test. A p-value less than 0.05 was considered statistically significant.

Inclusion criteria

- Patients aged ≥18 years.
- Documented history of successfully treated pulmonary tuberculosis with completion of anti-tubercular therapy.
- Patients presenting with respiratory symptoms such as dyspnea, wheezing, or chronic cough after completion of ATT.
- Availability of complete clinical records, chest radiograph, and acceptable spirometry.

Exclusion criteria

- Current or former smokers.
- History of bronchial asthma, chronic obstructive pulmonary disease, or any obstructive airway disease prior to pulmonary tuberculosis.
- Active pulmonary tuberculosis at the time of evaluation.
- Incomplete clinical, radiological, or spirometry records.
- Inability to perform acceptable and reproducible spirometry according to ATS/ERS criteria.
- Presence of other significant chronic respiratory diseases unrelated to tuberculosis.

RESULTS

A total of 50 patients previously treated for pulmonary tuberculosis were studied. There were 34 (68%) males and 16 (32%) females. There was a male predominance in studied cases with a M:F ratio of 1:0.36 (Figure 1).

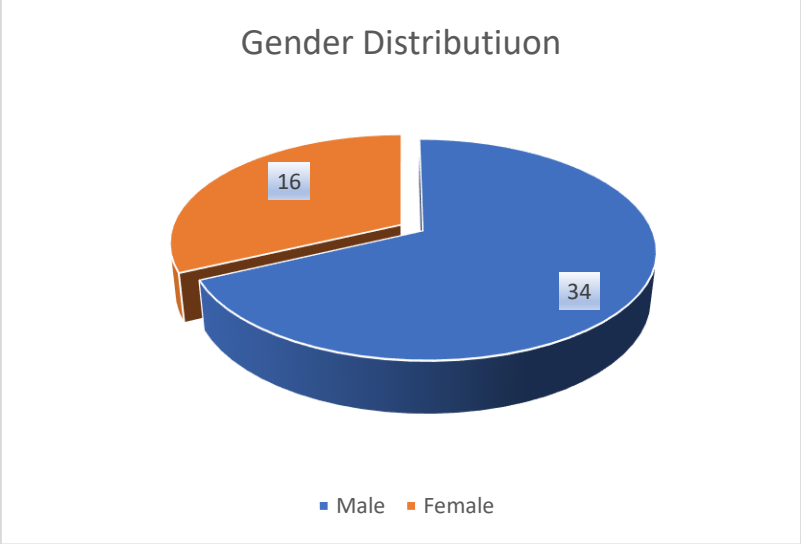

Figure 1: Gender Distribution of the studied cases.

The analysis of the age distribution of the studied cases showed that the largest proportion of patients belonged to the 31–45 years age group, accounting for 19 cases (38.0%), followed by the 46–60 years age group with 15 cases (30.0%). Patients aged ≤30 years and those aged >60 years each comprised 8 cases (16.0%). The mean age of the study population was 45.2 ± 13.4 years, indicating that the majority of treated pulmonary tuberculosis patients were middle-aged (Table 1).

Table 1. Demographic characteristics of the study population (N = 50).

Age group (years)	≤30	8 (16.0)
	31–45	19 (38.0)
	46–60	15 (30.0)
	>60	8 (16.0)
Mean age ± SD	45.2 ± 13.4	

The analysis of the predominant symptoms and interval since ATT completion of the studied cases showed that dyspnea was the most common symptom, observed in 22 cases (44.0%), followed by cough in 17 cases (34.0%) and wheeze in 11 cases (22.0%). The most common interval since completion of ATT was 1–3 years, seen in 23 cases (46.0%), followed by <1 year and 3–5 years with 11 cases (22.0%) each, while >5 years was reported in 5 cases (10.0%) (Table 2).

Table 2. Presenting symptoms and interval since completion of anti-tuberculosis treatment (ATT).

Variable	**Category**	**n (%)**
Predominant symptom	Dyspnea	22 (44.0)
	Cough	17 (34.0)
	Wheeze	11 (22.0)
Interval since ATT completion	<1 year	11 (22.0)
	1–3 years	23 (46.0)
	3–5 years	11 (22.0)
	>5 years	5 (10.0)

The analysis of the functional spirometry outcome of the studied cases showed that normal lung function was observed in 39 cases (78.0%), while obstructive airway disease was present in 11 cases (22.0%) (Table 3).

Table 3. Functional outcome on spirometry among treated pulmonary tuberculosis patients (N = 50).

Functional (spirometry) outcome	**n**	**%**
Obstructive airway disease present	11	22.0
Normal lung function	39	78.0
Total	**50**	**100.0**

The analysis of chest radiography findings of the studied cases showed that no significant sequela was observed in 39 cases (78.0%), followed by extensive fibrosis and bronchiectasis in 4 cases (8.0%) each, while cavitation with fibrosis was seen in 3 cases (6.0%) (Table 4).

Table 4. Distribution of radiological sequelae on chest radiography among treated pulmonary tuberculosis patients.

Radiological finding	**n**	**%**
Extensive fibrosis	4	8.0
Cavitation with fibrosis	3	6.0
Bronchiectasis	4	8.0
No significant sequela	39	78.0
Total	**50**	**100.0**

The distribution of obstructive airway disease by gender is shown in Table 5. Obstruction was numerically more common among men than women, but the difference did not reach statistical significance (Table 5).

Table 5. Distribution of obstructive airway disease (OAD) by Gender (Fisher's exact test).

Sex	**Total, n**	**With OAD, n (%)**	**Without OAD, n (%)**	**p-value**
Male	34	9 (26.5)	25 (73.5)	0.47
Female	16	2 (12.5)	14 (87.5)	
Total	50	11 (22.0)	39 (78.0)	

The proportion of patients with obstructive airway disease increased with a longer interval since completion of anti-tuberculosis treatment, from about one-tenth of those presenting within a year to over half of those presenting beyond five years; in this small sample the trend did not reach statistical significance (Table 7).

Table 7. Relationship between interval since anti-tuberculosis treatment (ATT) completion and obstructive airway disease (OAD) (chi-square test).

Interval since ATT	Total, n	With OAD, n (%)	p-value
<1 year	11	1 (9.1)	0.13
1–3 years	23	4 (17.4)	
3–5 years	11	3 (27.3)	
>5 years	5	3 (60.0)	
Total	50	11 (22.0)	

DISCUSSION

Pulmonary tuberculosis is increasingly recognized as an important precursor of chronic respiratory impairment even after successful microbiological cure. The persistence of airflow limitation despite completion of anti-tubercular therapy reflects the cumulative effects of chronic inflammation, airway remodeling, parenchymal destruction, and abnormal tissue repair rather than persistent infection alone. In the present study, obstructive airway disease (OAD) was detected in 22% of previously treated pulmonary tuberculosis patients, supporting the concept that tuberculosis itself is an independent risk factor for chronic airflow obstruction. Similar observations have been reported by Allwood et al[6] that previous pulmonary tuberculosis significantly increases the risk of chronic airflow obstruction independent of cigarette smoking. Likewise, Amaral et al[7], analysing data from the multinational Burden of Obstructive Lung Disease (BOLD) study, demonstrated that individuals with a history of pulmonary tuberculosis had significantly lower lung function and a higher prevalence of airflow obstruction compared with subjects without previous tuberculosis, even after adjustment for smoking and other confounding variables. More recently, Gai X et al [8] further emphasized that post-tuberculosis lung disease (PTLD) represents an important contributor to the global burden of COPD, particularly in low- and middle-income countries where tuberculosis remains highly prevalent. The incidence observed in the present study therefore falls within the range reported in published literature and further highlights that successful treatment of tuberculosis should not be considered synonymous with complete respiratory recovery.

Dyspnea was the predominant presenting symptom, followed by chronic cough and wheeze, indicating that respiratory morbidity frequently persists after microbiological cure. These symptoms probably reflect residual airway narrowing, impaired elastic recoil, ventilation-perfusion mismatch, respiratory muscle dysfunction, and loss of functional lung parenchyma resulting from healed tuberculosis. Byrne et al[9] found that tuberculosis survivors consistently experience chronic respiratory symptoms and reduced health-related quality of life long after completion of therapy. These findings emphasize the importance of assessing presence of respiratory disability in tuberculosis patients who successfully completed ATT.[10] Recent evidence summarized by Meghji et al[11] further indicates that almost half of pulmonary tuberculosis survivors continue to have residual respiratory impairment requiring long-term clinical follow-up. The predominance of dyspnea observed in the present series is therefore biologically plausible and supports current recommendations advocating routine post-treatment respiratory assessment rather than limiting follow-up to confirmation of microbiological cure alone. Incorporation of symptom assessment together with spirometry may facilitate early diagnosis of airflow limitation and timely institution of bronchodilator therapy, pulmonary rehabilitation, vaccination, and other supportive interventions aimed at improving functional outcomes.

Imaging studies in these cases demonstrated fibrosis, bronchiectasis and cavitary fibrosis as the dominant abnormalities associated with airflow obstruction. These findings are consistent with pathological mechanisms underlying PTLD. Persistent inflammation promotes destruction of alveolar architecture, bronchial distortion and permanent narrowing of both large and small airways. Khan R et al12 reviewed imaging findings following pulmonary tuberculosis and identified fibrosis, bronchiectasis, cavitation and parenchymal destruction as the predominant imaging abnormalities in these cases . Similarly, van Kampen SC et al[13] in their systematic review of post-tuberculosis chronic lung disorders, emphasized that structural abnormalities detected on imaging correlate closely with long-term functional impairment and should prompt objective pulmonary function assessment.

An increasing proportion of airflow obstruction was observed with longer duration after completion of anti-tubercular therapy, although statistical significance was not achieved. This trend is consistent with the evolving concept that PTLD represents a dynamic rather than static disease process. Ongoing airway remodeling, chronic immune activation, repeated respiratory infections, and progressive fibrotic changes may continue to impair pulmonary function for several years after microbiological cure. Earlier evidence from Hnizdo et al[14] demonstrated that pulmonary tuberculosis is associated with chronic impairment of lung function independent of smoking and that recurrent tuberculosis episodes further accelerate functional decline. The authors further emphasized that tuberculosis survivors remain at increased risk of developing chronic airflow obstruction long after treatment completion.

Several strengths and limitations should be considered while interpreting these findings. Exclusion of smokers and patients with pre-existing obstructive airway disease minimized important confounding factors and strengthened the likelihood that airflow obstruction was attributable primarily to previous tuberculosis. Furthermore, diagnosis was based on standardized spirometry together with compatible clinical and radiological findings, thereby improving diagnostic accuracy. Nevertheless, the retrospective design, relatively small sample size, single-centre setting, and absence of baseline pulmonary function before tuberculosis limit the ability to establish the magnitude of lung function decline attributable to the disease. High-resolution computed tomography and diffusion capacity measurements were also unavailable and may have detected additional abnormalities not evident on conventional chest radiography. Despite these limitations, the findings add to accumulating evidence that PTLD constitutes a significant but frequently neglected cause of chronic respiratory disability. Kirakosyan O et al[15] recently highlighted that structured interventions directed toward prevention, early diagnosis, pulmonary rehabilitation, and long-term respiratory care are likely to reduce the burden of PTLD and improve quality of life among tuberculosis survivors. Routine spirometry for symptomatic patients after completion of anti-tubercular therapy therefore appears justified, particularly in high tuberculosis burden countries where early identification and management of post-tuberculosis airflow obstruction may substantially reduce long-term morbidity.

CONCLUSION

Obstructive airway disease is an important sequela of treated pulmonary tuberculosis cases. COAD occurs even in non-smokers and without presence of pre-existing respiratory disease. Persistent respiratory symptoms, particularly dyspnea, should prompt spirometric evaluation. This will facilitate early diagnosis as well as appropriate management. Routine post-ATT respiratory follow-up may help identify patients with post-tuberculosis lung disease. Early diagnosis of post-tuberculosis lung disease improve quality of life and reduce long-term respiratory morbidity through timely therapeutic interventions and pulmonary rehabilitation.

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