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Original Article

Evaluation of Pulmonary and Pleural Changes Among Tuberculosis Patients Using High-Resolution Computed Tomography in Association with TB Stage

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ABSTRACT

Background: Tuberculosis remains a major cause of respiratory morbidity, and conventional chest radiography may underestimate subtle pulmonary and pleural structural abnormalities. High-resolution computed tomography provides detailed assessment of parenchymal, airway, nodal, and pleural changes and may help characterize disease activity and chronic post-tuberculosis sequelae. Objective: To evaluate pulmonary and pleural HRCT findings among tuberculosis patients and determine their association with active and inactive tuberculosis stage. Methods: This cross-sectional observational study included 94 adult pulmonary tuberculosis patients assessed at Lahore General Hospital and Services Hospital, Lahore. HRCT findings were recorded as present or absent for tree-in-bud pattern, cavitary lesions, fibrosis, consolidation, pleural effusion, lymphadenopathy, calcified nodules, bronchiectasis, and pleural thickening. Data were analyzed using SPSS version 27.0, and associations between HRCT findings and tuberculosis stage were assessed using chi-square analysis with $p < 0.05$ considered statistically significant. Results: The mean age was 50.36 ± 19.83 years. Active tuberculosis was present in 60 patients (63.8%), while 34 patients (36.2%) had inactive disease. Consolidation and pleural thickening were the most frequent findings, each observed in 44 patients (46.8%). Tree-in-bud pattern, consolidation, pleural effusion, and lymphadenopathy were significantly more frequent in active tuberculosis, whereas fibrosis, calcified nodules, and pleural thickening were significantly more frequent in inactive tuberculosis. Conclusion: HRCT demonstrated distinct pulmonary and pleural patterns associated with tuberculosis stage and may support differentiation between active inflammatory disease and chronic post-tuberculosis structural sequelae. Keywords: High-Resolution Computed Tomography; Pulmonary Tuberculosis; Tree-in-Bud Pattern; Pleural Thickening; Fibrosis; Diagnostic Imaging

INTRODUCTION

Tuberculosis remains a major infectious cause of respiratory morbidity and mortality, particularly in high-burden countries where delayed diagnosis and incomplete assessment of residual lung damage contribute to preventable clinical deterioration. Pulmonary tuberculosis may present with a wide spectrum of parenchymal, airway, nodal, and pleural abnormalities, and its radiological appearance varies according to disease activity, host immune response, prior treatment exposure, and chronic sequelae. Conventional chest radiography is often used as the first imaging investigation because it is accessible and inexpensive; however, it has limited sensitivity for subtle airway-centered disease, early parenchymal involvement, small nodules, bronchiectatic change, fibrosis, calcification, and pleural

abnormalities. High-resolution computed tomography provides a more detailed evaluation of lung architecture and can identify characteristic findings such as centrilobular nodules, tree-in-bud pattern, consolidation, cavitary lesions, lymphadenopathy, bronchiectasis, fibrosis, calcified nodules, pleural effusion, and pleural thickening, which may help clinicians characterize the extent and pattern of pulmonary involvement in tuberculosis (1).

The global burden of tuberculosis continues to create substantial pressure on diagnostic and follow-up services. Recent international estimates indicate that millions of people develop tuberculosis annually, with major setbacks in case detection and treatment continuity following disruptions in health services during the coronavirus pandemic period. These delays are clinically important because untreated or inadequately monitored pulmonary tuberculosis may lead to progressive parenchymal destruction, persistent airway disease, pleural complications, transmission within the community, and long-term respiratory impairment. In high-burden settings, imaging strategies that can detect structural abnormalities more accurately than plain radiography are therefore relevant not only for initial assessment but also for identifying patients who may require closer follow-up after treatment completion (2).

Pulmonary damage associated with tuberculosis is not limited to the active infectious phase. A previous history of pulmonary tuberculosis is increasingly recognized as a risk factor for chronic respiratory symptoms, impaired lung function, reduced exercise capacity, and post-tuberculosis lung disease. The pathophysiology of post-tuberculosis lung damage is multifactorial and may involve persistent inflammation, host immune-mediated tissue injury, airway remodeling, parenchymal scarring, bronchiectasis, and pleural fibrosis. These abnormalities may remain clinically silent in some patients but can contribute to chronic cough, dyspnea, recurrent healthcare use, and reduced quality of life. Because chest radiography and spirometry may not fully capture the anatomical extent of post-tuberculosis structural damage, computed tomography-based evaluation has become important for characterizing residual disease patterns and differentiating potentially active inflammatory changes from chronic sequelae (3,4).

High-resolution computed tomography is particularly useful because different imaging patterns may suggest different stages or biological behavior of tuberculosis. Findings such as tree-in-bud appearance, consolidation, pleural effusion, and lymphadenopathy are commonly associated with active endobronchial spread, inflammatory parenchymal involvement, or ongoing immune response, whereas fibrosis, calcified nodules, architectural distortion, bronchiectasis, and pleural thickening are more frequently interpreted as sequelae of healed or inactive disease. Although these findings should always be interpreted in combination with clinical assessment and microbiological evidence where available, their distribution and frequency can provide clinically meaningful information for staging, follow-up planning, and identification of patients at risk of persistent respiratory morbidity (5,6).

Despite the recognized value of computed tomography in pulmonary tuberculosis, local evidence from Pakistan describing the frequency of HRCT pulmonary and pleural findings and their association with active versus inactive tuberculosis remains limited. This gap is important because disease burden, access to diagnostic facilities, treatment delays, and follow-up practices may differ across settings, and local data can support more contextually appropriate imaging recommendations. A structured evaluation of HRCT findings in adult pulmonary tuberculosis patients can therefore help clarify which abnormalities are most frequently observed and which radiological patterns are associated with active or inactive disease in routine clinical practice.

The present study was conducted to evaluate pulmonary and pleural changes detected on high-resolution computed tomography among adult patients with pulmonary tuberculosis and to determine their association with tuberculosis stage. The study specifically examined whether HRCT findings including tree-in-bud pattern, cavitary lesions, fibrosis, consolidation, pleural effusion, lymphadenopathy, calcified nodules, bronchiectasis, and pleural thickening differed between active and

inactive tuberculosis. It was hypothesized that active tuberculosis would show higher frequencies of inflammatory and endobronchial spread-related findings, whereas inactive tuberculosis would show higher frequencies of fibrotic, calcified, and chronic pleural changes.

MATERIAL AND METHODS

This cross-sectional observational study was conducted to evaluate pulmonary and pleural abnormalities detected on high-resolution computed tomography among adult patients with pulmonary tuberculosis and to assess the association of these findings with active and inactive tuberculosis stage. The study was carried out at Lahore General Hospital and Services Hospital, Lahore, over a period of 90 days after approval of the study synopsis. The cross-sectional design was selected because the objective was to describe the frequency of HRCT findings and examine their association with tuberculosis stage at a single assessment point rather than to determine temporal progression or treatment-related change.

The study population consisted of patients aged 18 years or above with pulmonary tuberculosis diagnosed on the basis of clinical evaluation and radiological findings. Patients presenting for follow-up assessment after tuberculosis treatment were also eligible when their clinical record supported classification as pulmonary tuberculosis with active or inactive disease status. Participants were included if they were willing and able to provide informed consent. Pregnant women, patients with a prior chest computed tomography scan within the preceding six months, uncooperative patients, and patients with known active lung malignancy under treatment were excluded to reduce unnecessary radiation exposure, avoid duplication of imaging, improve image acquisition reliability, and minimize diagnostic overlap with malignant pulmonary disease.

Eligible participants were selected using the sampling method specified in the study protocol from patients presenting to the participating hospitals during the study period. After eligibility assessment and informed consent, demographic and clinical information was recorded, including age, smoking history, previous tuberculosis history, family history of tuberculosis, and tuberculosis stage. Tuberculosis stage was categorized as active or inactive according to the documented clinical and radiological assessment available at the time of evaluation. Active tuberculosis represented patients with current clinical-radiological evidence of ongoing pulmonary tuberculosis, whereas inactive tuberculosis represented patients with post-treatment or healed disease status with residual structural findings. HRCT findings were then assessed as binary variables according to their presence or absence on imaging.

All participants underwent HRCT evaluation of the chest for assessment of pulmonary and pleural abnormalities. The imaging variables recorded for analysis were tree-in-bud pattern, cavitary lesions, fibrosis, consolidation, pleural effusion, lymphadenopathy, calcified nodules, bronchiectasis, and pleural thickening. These variables were selected because they represent common parenchymal, airway, nodal, and pleural manifestations of pulmonary tuberculosis and may differ according to disease activity. Tree-in-bud pattern, consolidation, pleural effusion, and lymphadenopathy were considered inflammatory or activity-related radiological features for analytical interpretation, whereas fibrosis, calcified nodules, bronchiectasis, and pleural thickening were considered chronic structural or post-tuberculosis sequelae when interpreted in relation to inactive disease. Cavitary lesions were assessed separately because they may occur in active disease but can also persist as residual structural change.

The sample size was calculated using the single population proportion formula, $n = Z^2p(1 - p)/d^2$, with a 95% confidence level, Z value of 1.96, expected prevalence of 42.4%, and margin of error of 10%. The calculated sample size was 94 participants, and all 94 patients were included in the final analysis. Data were entered and analyzed using SPSS version 27.0. Continuous variables were summarized using mean and standard deviation, while categorical variables were summarized using frequencies and percentages. The association between tuberculosis stage and each HRCT pulmonary or pleural finding was assessed using the chi-square test. A p-value of less than 0.05 was considered statistically significant. Where expected cell counts were small, Fisher's exact test would be more appropriate for confirmatory analysis.

Because multiple HRCT features were assessed, the findings were interpreted with emphasis on clinical pattern consistency rather than isolated p-values alone.

To reduce measurement and classification bias, all variables were defined before analysis and HRCT findings were recorded in a structured binary format. Eligibility criteria were applied uniformly to all participants, and patients with conditions likely to confound pulmonary imaging interpretation, such as active lung malignancy under treatment, were excluded. Data checking was performed before analysis to ensure completeness of demographic, clinical, staging, and HRCT variables. Confidentiality of participant information was maintained throughout data handling and analysis, and informed consent was obtained before inclusion in the study.

RESULTS

A total of 94 patients with pulmonary tuberculosis were included in the final analysis. The age of participants ranged from 18 to 96 years, with a mean age of 50.36 ± 19.83 years.

Table 1. Descriptive Statistics of Age Among Study Participants (N = 94)

Variable	N	Minimum	Maximum	Mean ± SD
Age, years	94	18	96	50.36 ± 19.83

The study population represented a broad adult age range, indicating that pulmonary tuberculosis and its HRCT-detectable pulmonary and pleural changes were evaluated across both younger and older adult patients. The wide standard deviation reflects substantial age variability within the included sample.

Table 2. Clinical History and Tuberculosis Stage Among Study Participants (N = 94)

Variable	Category	n (%)
Smoking history	No	56 (59.6)
Smoking history	Yes	38 (40.4)
Previous tuberculosis history	No	43 (45.7)
Previous tuberculosis history	Yes	51 (54.3)
Family history of tuberculosis	No	70 (74.5)
Family history of tuberculosis	Yes	24 (25.5)
Tuberculosis stage	Inactive	34 (36.2)
Tuberculosis stage	Active	60 (63.8)

More than half of the participants had a previous history of tuberculosis, reported in 51 patients (54.3%), while 38 patients (40.4%) had a history of smoking. Family history of tuberculosis was reported by 24 patients (25.5%). Active tuberculosis was more frequent than inactive tuberculosis in the study sample, accounting for 60 patients (63.8%) compared with 34 patients (36.2%) classified as inactive.

Table 3. Frequency of HRCT Pulmonary and Pleural Findings Among Study Participants (N = 94)

HRCT finding	Absent, n (%)	Present, n (%)
Tree-in-bud pattern	54 (57.4)	40 (42.6)
Cavitary lesions	60 (63.8)	34 (36.2)
Fibrosis	55 (58.5)	39 (41.5)
Consolidation	50 (53.2)	44 (46.8)
Pleural effusion	68 (72.3)	26 (27.7)
Lymphadenopathy	51 (54.3)	43 (45.7)
Calcified nodules	54 (57.4)	40 (42.6)
Bronchiectasis	51 (54.3)	43 (45.7)
Pleural thickening	50 (53.2)	44 (46.8)

Consolidation and pleural thickening were the most frequently observed HRCT findings, each present in 44 patients (46.8%). Lymphadenopathy and bronchiectasis were each identified in 43 patients (45.7%), while tree-in-bud pattern and calcified nodules were each observed in 40 patients (42.6%). Fibrosis was present in 39 patients (41.5%), cavitary lesions in 34 patients (36.2%), and pleural effusion in 26 patients (27.7%), making pleural effusion the least frequent HRCT abnormality in the overall sample.

Seven of the nine HRCT findings showed statistically significant associations with tuberculosis stage. Tree-in-bud pattern showed the strongest association with active tuberculosis, being present in 37 of 60 active cases (61.7%) compared with 3 of 34 inactive cases (8.8%). Consolidation, pleural effusion, and lymphadenopathy were also more frequent among active tuberculosis patients, affecting 33 (55.0%), 21 (35.0%), and 33 (55.0%) active cases, respectively. In contrast, fibrosis, calcified nodules, and pleural thickening were more frequent in inactive tuberculosis, with fibrosis and calcified nodules each present in 25 of 34 inactive cases (73.5%) and pleural thickening present in 21 inactive cases (61.8%). Cavitary lesions were more common in active tuberculosis than inactive tuberculosis, but the association did not reach statistical significance. Bronchiectasis showed nearly similar frequencies between inactive and active tuberculosis groups.

Table 4. Association Between HRCT Findings and Tuberculosis Stage

HRCT finding	Inactive TB, n/N (%)	Active TB, n/N (%)	χ^2	p-value
Tree-in-bud pattern	3/34 (8.8)	37/60 (61.7)	24.790	<0.001
Cavitary lesions	8/34 (23.5)	26/60 (43.3)	3.687	0.055
Fibrosis	25/34 (73.5)	14/60 (23.3)	22.525	<0.001
Consolidation	11/34 (32.4)	33/60 (55.0)	4.471	0.034
Pleural effusion	5/34 (14.7)	21/60 (35.0)	4.467	0.035
Lymphadenopathy	10/34 (29.4)	33/60 (55.0)	5.725	0.017
Calcified nodules	25/34 (73.5)	15/60 (25.0)	20.908	<0.001
Bronchiectasis	16/34 (47.1)	27/60 (45.0)	0.037	0.847
Pleural thickening	21/34 (61.8)	23/60 (38.3)	4.786	0.029

χ^2: chi-square statistic; TB: tuberculosis. Percentages are calculated within tuberculosis-stage groups.

Table 5. Derived Effect Estimates for Association Between HRCT Findings and Active Tuberculosis

HRCT finding	Odds Ratio	95% CI	p-value
Tree-in-bud pattern	16.62	4.56–60.65	<0.001
Cavitary lesions	2.49	0.97–6.38	0.055
Fibrosis	0.11	0.04–0.29	<0.001
Consolidation	2.56	1.06–6.16	0.034
Pleural effusion	3.12	1.05–9.26	0.035
Lymphadenopathy	2.93	1.20–7.19	0.017
Calcified nodules	0.12	0.05–0.31	<0.001
Bronchiectasis	0.92	0.40–2.14	0.847
Pleural thickening	0.38	0.16–0.91	0.029

Tree-in-bud pattern demonstrated the largest association with active tuberculosis, with active cases showing 16.62 times higher odds of this finding compared with inactive cases. Consolidation, pleural effusion, and lymphadenopathy were also associated with higher odds of active tuberculosis, with odds ratios of 2.56, 3.12, and 2.93, respectively. Conversely, fibrosis and calcified nodules showed markedly lower odds among active tuberculosis patients, supporting their stronger association with inactive or post-tuberculosis structural change. Pleural thickening also showed lower odds among active tuberculosis patients, consistent with its predominance in inactive disease. Cavitary lesions showed increased odds in active tuberculosis, but the confidence interval crossed 1.00, while bronchiectasis showed no meaningful stage-related difference.

Overall, the results indicate that HRCT findings differed substantially according to tuberculosis stage. Active tuberculosis was characterized mainly by tree-in-bud pattern, consolidation, pleural effusion, and lymphadenopathy, suggesting ongoing inflammatory, endobronchial, parenchymal, or nodal involvement. Inactive tuberculosis was characterized mainly by fibrosis, calcified nodules, and pleural thickening, reflecting chronic structural sequelae. Cavitary lesions showed a clinically suggestive but statistically borderline association with active disease, whereas bronchiectasis appeared to represent a persistent airway abnormality that was not specific to either active or inactive tuberculosis in this sample.

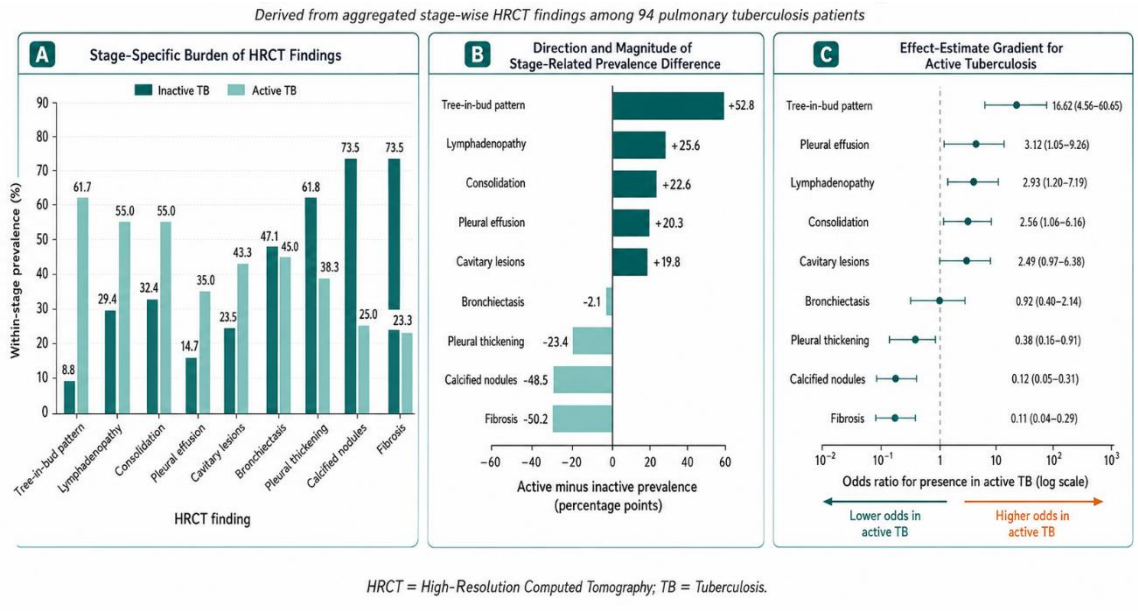

Figure 1 Integrated HRCT Pattern Profile by Tuberculosis Stage

The panelled figure demonstrates a distinct stage-related HRCT pattern among 94 pulmonary tuberculosis patients. Tree-in-bud pattern showed the largest active-stage gradient, increasing from 8.8% in inactive tuberculosis to 61.7% in active tuberculosis, corresponding to a +52.8 percentage-point difference and an odds ratio of 16.62. Consolidation, lymphadenopathy, and pleural effusion also showed higher active-stage prevalence, with differences of +22.6, +25.6, and +20.3 percentage points, respectively. In contrast, fibrosis and calcified nodules were strongly concentrated in inactive tuberculosis, each present in 73.5% of inactive cases compared with 23.3% and 25.0% of active cases, respectively. Pleural thickening also favored inactive disease, with a −23.4 percentage-point active-minus-inactive difference. Cavitary lesions showed a clinically suggestive active-stage increase but with borderline statistical support, while bronchiectasis showed minimal stage separation, indicating that it may represent persistent airway damage rather than a stage-specific HRCT marker.

DISCUSSION

The present cross-sectional observational study evaluated HRCT-detected pulmonary and pleural abnormalities among 94 adult patients with pulmonary tuberculosis and examined their association with active and inactive tuberculosis stage. The findings demonstrate a clear radiological separation between activity-related inflammatory patterns and chronic post-tuberculosis structural changes. Among the nine HRCT variables assessed, seven showed statistically significant associations with tuberculosis stage. Tree-in-bud pattern, consolidation, pleural effusion, and lymphadenopathy were more frequent in active tuberculosis, whereas fibrosis, calcified nodules, and pleural thickening were more frequent in inactive tuberculosis. Cavitary lesions showed a clinically suggestive but statistically borderline association with active disease, while bronchiectasis showed no meaningful stage-related difference. These findings support the role of HRCT as an adjunctive imaging modality for characterizing tuberculosis-related pulmonary and pleural abnormalities, particularly when conventional imaging is insufficient to define disease extent or residual structural damage.

Tree-in-bud pattern showed the strongest association with active tuberculosis in this study, occurring in 61.7% of active cases compared with 8.8% of inactive cases. The derived odds ratio indicated substantially higher odds of tree-in-bud appearance among active tuberculosis patients, supporting its interpretation as a marker of endobronchial spread and active small-airway inflammatory involvement. This finding is consistent with established radiological descriptions of pulmonary tuberculosis, where centrilobular nodules and tree-in-bud opacities are commonly linked to bronchogenic dissemination and active disease. Previous imaging literature has emphasized that HRCT is more sensitive than chest radiography

for detecting small airway-centered lesions, nodular disease, and early parenchymal abnormalities that may be overlooked on plain radiographs (1,5). Similar observations have also been reported in studies describing tree-in-bud appearance, consolidation, and cavitary change as important HRCT features of active pulmonary tuberculosis (7,8).

Consolidation was also significantly more frequent in active tuberculosis, affecting 55.0% of active cases compared with 32.4% of inactive cases. This pattern is biologically plausible because consolidation reflects active parenchymal inflammatory involvement, alveolar exudation, and disease activity when interpreted alongside clinical and microbiological context. Lymphadenopathy was similarly associated with active disease, being present in 55.0% of active cases compared with 29.4% of inactive cases. In tuberculosis, nodal enlargement may occur due to ongoing immune activation and mycobacterial involvement, particularly in active primary or post-primary disease patterns. Pleural effusion was less frequent overall than several parenchymal findings, but it was significantly more common in active tuberculosis, occurring in 35.0% of active cases compared with 14.7% of inactive cases. Together, these findings indicate that active tuberculosis in this cohort was characterized not by a single isolated HRCT marker but by a broader inflammatory profile involving airway-centered spread, parenchymal consolidation, nodal response, and pleural fluid accumulation.

In contrast, fibrosis and calcified nodules were strongly associated with inactive tuberculosis. Each was present in 73.5% of inactive cases, compared with 23.3% and 25.0% of active cases, respectively. These results are consistent with the expected radiological behavior of healed or previously treated tuberculosis, where chronic parenchymal scarring, architectural distortion, and calcified granulomatous residues may persist after microbiological cure. Post-tuberculosis lung disease has increasingly been recognized as a clinically important condition, with residual abnormalities involving lung parenchyma, airways, pleura, and pulmonary vasculature contributing to persistent respiratory symptoms and functional impairment (3,4). Imaging-based studies and clinical reviews have similarly emphasized that fibrosis, calcification, bronchiectatic distortion, and pleural thickening are common structural sequelae after pulmonary tuberculosis and may remain relevant even after treatment completion (9–11).

Pleural thickening was also significantly more frequent in inactive tuberculosis, occurring in 61.8% of inactive cases compared with 38.3% of active cases. This supports its interpretation as a chronic pleural sequela, particularly in patients with previous pleural inflammation or treated tuberculous pleuritis. Although pleural abnormalities can occur in active disease, persistent pleural thickening more often reflects fibrotic remodeling and residual pleural scarring. This distinction is clinically important because chronic pleural thickening may contribute to restrictive respiratory symptoms and may not necessarily indicate current infectious activity. Therefore, HRCT interpretation should be integrated with symptom duration, treatment history, microbiological results, inflammatory markers, and prior imaging where available.

Cavitary lesions were more frequent in active tuberculosis than inactive tuberculosis, affecting 43.3% of active cases compared with 23.5% of inactive cases; however, the association did not reach conventional statistical significance. The borderline p-value suggests that a larger sample may have clarified whether cavitation was independently associated with active disease in this population. Cavities are clinically important because they may indicate high bacillary burden and potential infectiousness in active pulmonary tuberculosis, but they can also persist as residual structural defects after treatment. This dual interpretation may explain why cavitary lesions showed a trend toward active-stage predominance without definitive statistical separation. Bronchiectasis also did not differ meaningfully between active and inactive tuberculosis, with frequencies of 45.0% and 47.1%, respectively. This finding suggests that bronchiectasis may represent persistent airway damage that remains after tuberculosis rather than a reliable marker of current activity. Similar interpretations have been described in post-tuberculosis lung disease, where bronchiectasis can persist as a long-term consequence of airway destruction and remodeling (10,11).

The findings should be interpreted in light of several methodological considerations. First, the cross-sectional design allows assessment of associations between HRCT findings and tuberculosis stage but cannot establish temporal progression or causality. Second, active and inactive tuberculosis classification should ideally be supported by a standardized combination of clinical, microbiological, treatment-history, and radiological criteria. If HRCT findings contributed to disease-stage classification, incorporation bias may have influenced the strength of observed associations. Third, the sample was drawn from two hospitals in Lahore, which provides useful local clinical data but limits generalizability to other populations and healthcare settings. Fourth, HRCT protocol details, radiologist blinding, interobserver agreement, and microbiological confirmation were not fully reported in the available manuscript, and future studies should address these elements to improve reproducibility and diagnostic validity.

Despite these limitations, the study provides clinically meaningful local evidence on the distribution of pulmonary and pleural HRCT abnormalities among tuberculosis patients. The results suggest that HRCT can help differentiate activity-related inflammatory patterns from chronic post-tuberculosis structural changes when interpreted as part of a broader clinical evaluation. In high-burden settings, this distinction may support better follow-up planning, identification of patients with residual structural lung disease, and more careful evaluation of patients whose chest radiographs do not adequately explain clinical symptoms. Larger multicenter studies with standardized staging criteria, microbiological correlation, low-dose CT protocols, pulmonary function testing, and longitudinal follow-up are needed to validate these findings and determine the prognostic significance of HRCT abnormalities in tuberculosis survivors.

CONCLUSION

High-resolution computed tomography demonstrated clinically meaningful differences in pulmonary and pleural findings between active and inactive tuberculosis in this cohort of 94 adult patients. Active tuberculosis was characterized mainly by tree-in-bud pattern, consolidation, pleural effusion, and lymphadenopathy, while inactive tuberculosis was characterized by fibrosis, calcified nodules, and pleural thickening. Cavitary lesions showed a borderline association with active disease, whereas bronchiectasis appeared to represent persistent airway damage rather than a stage-specific marker. These findings support the use of HRCT as an adjunctive imaging modality for detailed assessment of tuberculosis-related structural lung and pleural changes, particularly when conventional radiography is insufficient or when differentiation between active inflammatory disease and chronic post-tuberculosis sequelae is clinically important.

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