JHWCR JOURNAL OF HEALTH, WELLNESS AND COMMUNITY RESEARCH 3007-0570

Volume 4, Issue 17, 2026
Open Access | CC BY 4.0
Article ID: 1788
DOI: 10.61919/jme6af80

Original Article

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

Atiqa Aziz[1], Hafiza Maria Fawad[2], Tayyaba Iqbal[3], Hira Nadeem[1], Syeda Eman Shah[1], Musfirah Khalid[1], M. Noor ul Hassan Butt[1], Mubashir Farhan[1]

[1] Al Razi Institute, Lahore, Pakistan
[2] Head of Radiography & Imaging Technology, Al Razi Institute, Lahore, Pakistan
[3] Lecturer of Radiography & Imaging Technology, Al Razi Institute, Lahore, Pakistan

Corresponding Author: Hafiza Maria Fawad
Correspondence: mariafawad20@gmail.com

Received: 08 June 2026 | Revised: 26 August 2026 | Accepted: 05 September 2026 | Published: 11 September 2026

ARTICLE INFORMATION

Article Type: Original Article
How to Cite: Aziz A, Fawad HM, Iqbal T, Nadeem H, Shah SE, Khalid M, et al. Evaluation of pulmonary and pleural changes among tuberculosis patients using high-resolution computed tomography in association with TB stage. Journal of Health, Wellness and Community Research. 2026;4(17). DOI: https://doi.org/10.61919/jme6af80.
Article ID: 1788
DOI: 10.61919/jme6af80
Article Link: https://jhwcr.com/index.php/jhwcr/article/view/1788
Declarations: See end of article.

OPEN ACCESS
This is an open access article distributed under the terms of the Creative Commons Attribution 4.0 International License (CC BY 4.0).

ABSTRACT

Background: High-resolution computed tomography (HRCT) provides detailed characterization of pulmonary, airway, nodal, and pleural abnormalities in tuberculosis, although individual imaging findings may overlap between active disease and chronic post-tuberculosis structural change. **Objective:** To evaluate pulmonary and pleural HRCT findings among adults with pulmonary tuberculosis and determine their association with active versus inactive tuberculosis stage. **Methods:** This analytical cross-sectional study included 94 adults assessed at Lahore General Hospital and Services Hospital, Lahore. Nine predefined HRCT findings were recorded as present or absent. Associations with documented active or inactive tuberculosis stage were assessed using Pearson's chi-square test, and unadjusted odds ratios with 95% confidence intervals were derived from the supplied contingency data. **Results:** Sixty participants (63.8%) were classified as having active tuberculosis and 34 (36.2%) as inactive. Tree-in-bud pattern was substantially more frequent in active tuberculosis (61.7% vs 8.8%; OR 16.62, 95% CI 4.56–60.65; p<0.001). Consolidation, pleural effusion, and lymphadenopathy were also more frequent in active disease. Fibrosis (73.5% vs 23.3%; p<0.001), calcified nodules (73.5% vs 25.0%; p<0.001), and pleural thickening were more frequent in inactive tuberculosis. Cavitary lesions showed a borderline association with active disease (p=0.055), while bronchiectasis showed no stage-related association (p=0.847). **Conclusion:** HRCT abnormalities showed distinct associations with tuberculosis stage. HRCT may support anatomical characterization of active inflammatory patterns and chronic structural sequelae when interpreted alongside clinical and microbiological assessment. **Keywords:** Pulmonary Tuberculosis; High-Resolution Computed Tomography; Tree-in-Bud Pattern; Pulmonary Fibrosis; Pleural Thickening; Post-Tuberculosis Lung Disease; Cross-Sectional Studies

INTRODUCTION

Tuberculosis (TB) remains a major global infectious disease and an important cause of preventable morbidity and mortality. The World Health Organization estimated that 10.7 million people developed TB worldwide in 2024, with approximately 1.23 million deaths, and Pakistan remains among the countries contributing substantially to the global TB burden (1). Pulmonary tuberculosis represents the predominant clinically important form of disease and may produce a broad spectrum of parenchymal, airway, nodal, and pleural abnormalities whose appearance varies according to the extent of infection, host immune response, previous treatment, and residual structural damage.

Chest radiography remains an important first-line imaging examination because of its accessibility, relatively low cost, and widespread availability. However, radiographic appearances can be nonspecific, and subtle centrilobular disease, small nodules, early endobronchial spread, bronchiectatic change, limited cavitation, fibrosis, calcification, and pleural abnormalities may be incompletely characterized on conventional radiographs. High-resolution computed tomography (HRCT) provides substantially greater anatomical detail and can demonstrate pulmonary and thoracic manifestations including centrilobular nodules, tree-in-bud opacities, consolidation, cavitation, lymphadenopathy, bronchiectasis, fibrosis, calcified nodules, pleural effusion, and pleural thickening (2). HRCT should nevertheless be regarded as an adjunct to, rather than a replacement for, clinical and microbiological assessment because morphological appearances alone do not provide definitive evidence of current mycobacterial activity.

The clinical relevance of pulmonary TB extends beyond the period of active infection. Following treatment, some patients develop persistent anatomical and functional abnormalities collectively described within the spectrum of post-tuberculosis lung disease. Residual injury may involve the pulmonary parenchyma, airways, pleura, mediastinum, or pulmonary vasculature and may manifest as fibrosis, architectural distortion, bronchiectasis, residual cavities, calcified lesions, or pleural scarring (3–5). Such structural abnormalities can contribute to chronic cough, dyspnea, recurrent respiratory infections, impaired pulmonary function, reduced exercise tolerance, and diminished quality of life even after completion of anti-tuberculosis therapy (3,4). Recognition of these residual patterns is therefore important because chronic post-tuberculosis abnormalities may overlap radiologically with findings encountered during active disease.

Certain HRCT patterns occur more frequently in active pulmonary tuberculosis, although none should be interpreted independently of the overall clinical context. Centrilobular nodules and tree-in-bud opacities commonly reflect endobronchial dissemination through small airways, whereas consolidation, cavitation, lymphadenopathy, and pleural effusion may accompany ongoing inflammatory or infectious involvement (2,6). Conversely, fibrosis, volume loss, architectural distortion, calcification, traction bronchiectasis, and persistent pleural thickening are frequently encountered as chronic or post-treatment sequelae (3–5). Cavities and bronchiectasis are particularly important examples of overlap because both can be present during active disease yet may persist after treatment. Consequently, HRCT findings are more appropriately interpreted as **patterns associated with disease activity or chronic structural sequelae** rather than as stand-alone diagnostic markers of active or inactive tuberculosis.

Evidence from Pakistan has already demonstrated the clinical utility and spectrum of HRCT abnormalities in pulmonary tuberculosis. Rasheed et al. evaluated HRCT against mycobacterial culture in patients investigated for pulmonary tuberculosis and reported useful

diagnostic performance in both sputum-smear-positive and sputum-smear-negative disease (7). Ahmad et al. similarly described tree-in-bud opacities, fibrosis, consolidation, cavitation, and bronchiectatic abnormalities among patients undergoing HRCT evaluation for suspected pulmonary tuberculosis (8). More recently, Anwar et al. described a broad spectrum of HRCT patterns among patients with pulmonary tuberculosis at a tertiary-care hospital in Lahore (9). These findings indicate that local evidence concerning the radiological manifestations and diagnostic contribution of HRCT is available; therefore, the principal evidence gap is not a general absence of Pakistani HRCT research.

A more specific unresolved issue concerns the distribution of pulmonary and pleural HRCT findings according to clinically classified **active versus inactive tuberculosis** within local clinical populations. Previous work has suggested that combinations of HRCT features can provide information regarding disease activity (6), while Pakistani studies have largely concentrated on diagnostic performance, overall radiological patterns, or post-tuberculosis sequelae rather than systematically comparing a defined set of pulmonary and pleural abnormalities between active and inactive groups (5,7–9). Characterizing such associations may improve understanding of how inflammatory and chronic structural patterns are distributed across disease stages, while avoiding the assumption that individual CT findings independently establish microbiological activity.

The present study therefore aimed to evaluate pulmonary and pleural abnormalities detected on HRCT among adults with pulmonary tuberculosis and to determine their association with documented active or inactive tuberculosis stage. The evaluated findings included tree-in-bud pattern, cavitary lesions, fibrosis, consolidation, pleural effusion, lymphadenopathy, calcified nodules, bronchiectasis, and pleural thickening. It was hypothesized that findings associated with inflammatory and endobronchial involvement would occur more frequently in patients classified as having active tuberculosis, whereas fibrotic, calcified, and chronic pleural abnormalities would occur more frequently among patients classified as having inactive disease.

MATERIALS AND METHODS

Study Design and Setting

This analytical cross-sectional observational study evaluated pulmonary and pleural abnormalities detected on high-resolution computed tomography (HRCT) and their association with active versus inactive pulmonary tuberculosis. The study was conducted at Lahore General Hospital and Services Hospital, Lahore, Pakistan, over a 90-day period following approval of the study synopsis. A cross-sectional design was used because HRCT findings and tuberculosis stage were evaluated at a single assessment point rather than longitudinally for disease progression or treatment-related change.

Study Population and Eligibility

The study population comprised adults aged 18 years or older with pulmonary tuberculosis diagnosed on the basis of available clinical and radiological assessment. Patients attending follow-up after tuberculosis treatment were also eligible when their clinical records supported classification as pulmonary tuberculosis with active or inactive disease status. Participants were required to be willing and able to provide informed consent.

Pregnant women, patients who had undergone chest computed tomography within the preceding six months, patients unable to cooperate with the imaging procedure, and patients with known active lung malignancy undergoing treatment were excluded. These exclusions were applied to reduce avoidable repeat radiation exposure, facilitate reliable image acquisition, and minimize potential diagnostic overlap between tuberculosis-related abnormalities and active pulmonary malignancy.

Potentially eligible patients presenting to the participating hospitals during the study period were assessed against the eligibility criteria. After informed consent, demographic and clinical information was recorded, including age, smoking history, previous history of tuberculosis, family history of tuberculosis, and documented tuberculosis stage.

Classification of 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 accompanied by residual structural abnormalities. The stage classification constituted the principal grouping variable for comparison of HRCT findings.

HRCT Assessment and Imaging Variables

All included participants underwent HRCT examination of the chest for evaluation of pulmonary and pleural abnormalities. Nine predefined HRCT findings were recorded as binary variables according to their presence or absence: tree-in-bud pattern, cavitary lesions, fibrosis, consolidation, pleural effusion, lymphadenopathy, calcified nodules, bronchiectasis, and pleural thickening.

These findings were selected to represent airway, parenchymal, nodal, and pleural manifestations relevant to pulmonary tuberculosis. For interpretation of the stage-related analysis, tree-in-bud pattern, consolidation, pleural effusion, and lymphadenopathy represented predominantly inflammatory or activity-associated radiological patterns, whereas fibrosis, calcified nodules, bronchiectasis, and pleural thickening represented predominantly chronic structural or post-tuberculosis abnormalities. Cavitary lesions were evaluated separately because cavitation may occur during active pulmonary tuberculosis but may also persist as a residual structural abnormality after treatment.

Sample Size

Sample size was calculated using the single-population-proportion formula:

$$n = Z^2 p(1 - p)/d^2$$

A 95% confidence level was used with a corresponding Z value of 1.96, an expected proportion of 42.4%, and a margin of error of 10%. These parameters yielded a required sample size of approximately 94 participants. All 94 included participants were retained in the final analysis.

Statistical Analysis

Data were entered and analyzed using IBM SPSS Statistics version 27.0. Age was summarized using the mean, standard deviation, minimum, and maximum. Categorical demographic, clinical, tuberculosis-stage, and HRCT variables were summarized using frequencies and percentages.

Associations between tuberculosis stage and individual HRCT findings were evaluated using Pearson's chi-square test. Each HRCT feature was analyzed as a dichotomous variable—present or absent—against the two tuberculosis-stage categories of active and inactive disease. Statistical significance was defined as a two-sided p-value <0.05.

Because several HRCT abnormalities were examined individually, statistical findings were interpreted in conjunction with the magnitude and direction of stage-specific differences rather than on statistical significance alone.

Bias Control and Data Quality

Eligibility criteria were applied uniformly to participants before inclusion. HRCT findings were recorded using predefined binary categories to standardize data entry, and patients with active lung malignancy under treatment were excluded to reduce potential diagnostic overlap in interpretation of pulmonary imaging abnormalities. Data were checked before analysis for completeness and consistency across demographic, clinical, tuberculosis-stage, and HRCT variables.

Ethical Considerations

Ethical approval for the study was obtained from Al Razi Institute, Lahore, Pakistan. Written informed consent was obtained from all participants before inclusion. Participant information was handled confidentially throughout data collection, processing, and analysis.

RESULTS

Participant Characteristics

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

Table 1. Age Characteristics of Study Participants (N = 94)

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

Of the 94 participants, 38 (40.4%) had a history of smoking and 56 (59.6%) did not. Previous tuberculosis was reported by 51 participants (54.3%), whereas 43 (45.7%) had no previous tuberculosis history. A family history of tuberculosis was reported by 24 participants (25.5%). Sixty participants (63.8%) were classified as having active tuberculosis and 34 (36.2%) as having inactive tuberculosis.

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

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

Distribution of HRCT Findings

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

Table 3. Frequency of Pulmonary and Pleural HRCT Findings (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)

Association Between HRCT Findings and Tuberculosis Stage

Seven of the nine evaluated HRCT findings showed statistically significant associations with tuberculosis stage.

Tree-in-bud pattern demonstrated the largest stage-related difference. It was present in 37 of 60 participants with active tuberculosis (61.7%) compared with 3 of 34 participants with inactive tuberculosis (8.8%; $\chi^2 = 24.790$, $p < 0.001$). Fibrosis showed the opposite distribution, occurring in 25 of 34 participants with inactive tuberculosis (73.5%) compared with 14 of 60 participants with active tuberculosis (23.3%; $\chi^2 = 22.525$, $p < 0.001$). Calcified nodules were similarly more frequent in inactive tuberculosis, occurring in 25 of 34 participants (73.5%) compared with 15 of 60 participants with active disease (25.0%; $\chi^2 = 20.908$, $p < 0.001$). Consolidation was present in 33 of 60 participants with active tuberculosis (55.0%) and 11 of 34 with inactive tuberculosis (32.4%; $\chi^2 = 4.471$, $p = 0.034$). Pleural effusion occurred in 21 active cases (35.0%) compared with 5 inactive cases (14.7%; $\chi^2 = 4.467$, $p = 0.035$), while lymphadenopathy occurred in 33 active cases (55.0%) and 10 inactive cases (29.4%; $\chi^2 = 5.725$, $p = 0.017$). Pleural thickening was more frequent among participants with inactive tuberculosis, occurring in 21 of 34 cases (61.8%) compared with 23 of 60 active cases (38.3%; $\chi^2 = 4.786$, $p = 0.029$). Cavitary lesions were identified in 26 of 60 participants with active tuberculosis (43.3%) and 8 of 34 with inactive tuberculosis (23.5%). This difference did not reach the prespecified threshold for statistical significance ($\chi^2 = 3.687$, $p = 0.055$). Bronchiectasis showed little difference between stages, occurring in 27 of 60 active cases (45.0%) and 16 of 34 inactive cases (47.1%; $\chi^2 = 0.037$, $p = 0.847$).

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: Pearson chi-square statistic; TB: tuberculosis. Percentages are calculated within tuberculosis-stage groups.

Effect Estimates for Active Versus Inactive Tuberculosis

Unadjusted odds ratios were calculated from the supplied 2×2 contingency data, using inactive tuberculosis as the comparison group. Tree-in-bud pattern showed the largest positive association with active tuberculosis (OR 16.62, 95% CI 4.56–60.65). Active tuberculosis was also associated with higher odds of consolidation (OR 2.56, 95% CI 1.06–6.16), pleural effusion (OR 3.12, 95% CI 1.05–9.26), and lymphadenopathy (OR 2.93, 95% CI 1.20–7.19). Fibrosis (OR 0.11, 95% CI 0.04–0.29), calcified nodules (OR 0.12, 95% CI 0.05–0.31), and pleural thickening (OR 0.38, 95% CI 0.16–0.91) showed lower odds in the active-tuberculosis group and correspondingly greater occurrence in the inactive group. Cavitary lesions showed higher estimated odds in active tuberculosis, but the confidence interval included the null value (OR 2.49, 95% CI 0.97–6.38). Bronchiectasis showed no material stage-related association (OR 0.92, 95% CI 0.40–2.14).

Table 5. Unadjusted Association of HRCT Findings With 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

Odds ratios >1 indicate higher odds of the HRCT finding among participants classified as having active tuberculosis; values <1 indicate lower odds in the active group. CI: confidence interval.

Integrated Stage-Related HRCT Pattern

The largest absolute stage difference was observed for tree-in-bud pattern, which increased from 8.8% in inactive tuberculosis to 61.7% in active tuberculosis, corresponding to a 52.9-percentage-point difference. Fibrosis decreased from 73.5% in inactive tuberculosis to 23.3% in active tuberculosis, a 50.2-percentage-point difference, while calcified nodules decreased from 73.5% to 25.0%, a 48.5-percentage-point difference. More modest active-minus-inactive differences were observed for lymphadenopathy (+25.6 percentage points), pleural thickening (−23.5 percentage points), consolidation (+22.6 percentage points), and pleural effusion (+20.3 percentage points). Cavitary lesions differed by +19.8 percentage points but did not reach statistical significance, whereas bronchiectasis differed by only −2.1 percentage points.

DISCUSSION

This analytical cross-sectional study demonstrated substantial differences in the distribution of HRCT abnormalities between participants classified as having active and inactive pulmonary tuberculosis. Seven of the nine evaluated findings were significantly associated 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 disease. Cavitary lesions showed a higher frequency and greater estimated odds in active tuberculosis but did not reach the prespecified threshold for statistical significance, while bronchiectasis showed almost no stage-related difference. Collectively, these findings indicate that combinations of HRCT abnormalities

may provide useful structural information regarding the pattern of pulmonary involvement, although individual imaging findings should not be interpreted as independent evidence of microbiological disease activity.

Tree-in-bud pattern showed the largest separation between the two tuberculosis-stage groups, occurring in 61.7% of participants with active tuberculosis compared with only 8.8% of those with inactive disease. The corresponding unadjusted odds ratio of 16.62 further demonstrated the magnitude of this association. Tree-in-bud opacity represents bronchiolar impaction and branching centrilobular abnormalities and is well recognized in pulmonary tuberculosis as an imaging manifestation associated with bronchogenic or endobronchial dissemination (2). Previous HRCT studies have similarly reported tree-in-bud and centrilobular nodular patterns among the characteristic abnormalities encountered in active pulmonary tuberculosis (6,11,12). More recent imaging reviews continue to identify tree-in-bud nodularity as an important manifestation of active bronchogenic dissemination and emphasize that CT patterns should be interpreted together with microbiological and clinical information rather than as isolated diagnostic signs (14,15). The present finding therefore supports the established association between airway-centered nodular disease and active pulmonary involvement while acknowledging that tree-in-bud morphology is not specific to tuberculosis.

Consolidation was also more frequent in active disease, occurring in 55.0% of active cases compared with 32.4% of inactive cases. Consolidative abnormalities are commonly described in active pulmonary tuberculosis and may accompany bronchiolar dissemination, cavitation, and other manifestations of ongoing parenchymal inflammation (2,11,12,14,15). Ahmad et al. also identified consolidation among the common HRCT abnormalities in Pakistani patients investigated for pulmonary tuberculosis (8), while other regional HRCT studies have reported combinations of consolidation, nodules, and tree-in-bud abnormalities in active or microbiologically positive disease (7). Contemporary imaging literature similarly emphasizes the heterogeneity of active pulmonary tuberculosis, in which consolidation frequently coexists with small-airway nodularity, cavitation, and multifocal parenchymal disease rather than functioning as an isolated radiological marker (14,15). The present data extend these observations by demonstrating a significant stage-related difference within the study population rather than simply describing the overall frequency of consolidation.

Lymphadenopathy and pleural effusion were likewise significantly more frequent in active tuberculosis. Lymphadenopathy was present in 55.0% of active cases compared with 29.4% of inactive cases, while pleural effusion occurred in 35.0% and 14.7%, respectively. Both findings are recognized components of the radiological spectrum of pulmonary and thoracic tuberculosis, although their frequency varies according to disease phenotype, immune status, age, and clinical setting (2,15). Recent evidence from patients with tuberculous pleuritis has also demonstrated frequent pleural thickening or nodularity on CT together with imaging features of active pulmonary disease, reinforcing the importance of integrating pleural and parenchymal abnormalities during assessment (16). Their association with the active group in the present cohort is therefore compatible with greater contemporaneous nodal and pleural involvement. Nevertheless, neither lymphadenopathy nor pleural effusion should be regarded as stage-defining in isolation because both occur in multiple infectious, inflammatory, and neoplastic conditions.

In contrast, fibrosis was strongly associated with inactive tuberculosis, occurring in 73.5% of inactive cases compared with 23.3% of active cases. Calcified nodules showed a similarly marked distribution, being present in 73.5% of inactive cases compared with 25.0% of active cases. These findings are compatible with the established spectrum of post-tuberculosis structural lung disease, in which residual parenchymal fibrosis, volume loss, architectural distortion, calcified granulomatous lesions, cavities, and airway abnormalities may remain after completion of treatment (3–5,10). More recent work has reinforced the concept that post-tuberculosis lung disease represents a heterogeneous structural and functional syndrome rather than a single radiological entity. Persistent abnormalities may involve the airways, parenchyma, pleura, and pulmonary vasculature and can remain clinically relevant after microbiological cure (17–19). The marked predominance of fibrosis and calcified nodules in the inactive group therefore accords with contemporary understanding of residual post-tuberculosis structural disease.

Pleural thickening also occurred more frequently in inactive tuberculosis, affecting 61.8% of inactive cases compared with 38.3% of active cases. Persistent pleural thickening and pleural calcific or fibrotic abnormalities are recognized sequelae of previous tuberculous pleural involvement (5,13). Contemporary thoracic imaging literature similarly recognizes pleural fibrosis and thickening within the spectrum of chronic tuberculosis-related structural abnormalities, while emphasizing that pleural changes may coexist with active parenchymal or pleural infection (15,16). The present stage distribution is therefore compatible with residual pleural remodeling among patients classified as having inactive disease. Nevertheless, pleural thickening alone cannot establish inactivity, and interpretation should consider previous pleural disease, treatment history, clinical status, microbiological findings, and temporal imaging information where available.

The cavitation finding illustrates the overlap between active disease and post-treatment structural change. Cavitary lesions occurred in 43.3% of active tuberculosis compared with 23.5% of inactive tuberculosis, corresponding to an unadjusted odds ratio of 2.49; however, the 95% confidence interval included the null and the p-value was 0.055. Cavitation is a recognized manifestation of active pulmonary tuberculosis and has been associated with active or microbiologically positive disease in previous imaging studies (2,7,11,12,14). At the same time, residual cavities may persist after treatment, and recent reviews of post-tuberculosis lung disease continue to identify cavitation among clinically relevant chronic structural abnormalities (17–19). This dual biological and radiological context may partly explain why cavitation showed a substantial numerical difference between groups without achieving conventional statistical significance in the present sample.

Bronchiectasis showed a different pattern. It was present in 45.0% of active cases and 47.1% of inactive cases, with an odds ratio close to unity and no statistically significant association with tuberculosis stage. Bronchiectasis is a well-established component of post-tuberculosis lung disease and may arise from irreversible airway destruction, traction related to fibrosis, or chronic airway remodeling after pulmonary infection (4,5,10,13). Recent PTLD literature continues to identify bronchiectasis as one of the most important structural phenotypes after pulmonary tuberculosis, particularly among patients with persistent airflow obstruction and recurrent respiratory morbidity (17–19). However, bronchiectatic changes can also coexist with active tuberculosis. The nearly identical frequencies between groups in the present study therefore suggest that bronchiectasis represented accumulated structural airway damage rather than a reliable stage-discriminating feature within this cohort.

The overall pattern is clinically relevant because it emphasizes interpretation of HRCT as a constellation of findings rather than reliance on a single radiological sign. In this cohort, tree-in-bud pattern showed the clearest association with active tuberculosis, while fibrosis and calcified nodules showed the strongest inverse associations with active status. Consolidation, lymphadenopathy, and pleural effusion provided additional stage-related separation, whereas cavitation and bronchiectasis demonstrated substantial potential for overlap between active and chronic disease. Contemporary thoracic imaging reviews similarly emphasize that pulmonary tuberculosis demonstrates heterogeneous combinations of airway, parenchymal, nodal, and pleural abnormalities and that definitive assessment of

disease activity requires integration with microbiological and clinical information (14,15). HRCT may consequently be most useful as an adjunct for anatomical characterization, assessment of disease extent, recognition of complications, and evaluation of residual structural disease rather than as a replacement for microbiological confirmation.

Several methodological considerations affect interpretation of these results. First, the cross-sectional design establishes associations between HRCT findings and contemporaneously documented tuberculosis stage but cannot determine temporal evolution of the abnormalities or demonstrate that tuberculosis stage caused the observed imaging patterns. Longitudinal imaging studies following patients from active disease through treatment completion would be better suited to characterizing the resolution, persistence, or progression of individual HRCT abnormalities. This approach is increasingly relevant because contemporary PTLD research recognizes persistent structural and functional abnormalities after microbiological cure as an important source of long-term respiratory morbidity (17–19).

Second, the operational distinction between active and inactive tuberculosis is particularly important. Stage classification in this study was based on documented clinical and radiological assessment available at the time of evaluation. Because several of the same radiological abnormalities were subsequently tested for association with tuberculosis stage, incorporation or classification bias cannot be excluded if HRCT information contributed materially to the original stage assignment. Current imaging literature continues to emphasize that radiological findings support but do not replace microbiological confirmation of tuberculosis activity (14,15). Consequently, the magnitude of the observed associations, particularly for highly stage-separated findings such as tree-in-bud pattern, should be interpreted as association estimates within the study's classification framework rather than measures of independent diagnostic accuracy.

Third, the reported odds ratios are unadjusted. Potential confounding by characteristics such as age, smoking history, previous tuberculosis, disease duration, treatment history, or other clinical factors was not addressed through a supplied multivariable model. The observed associations therefore describe crude differences between the active and inactive groups and should not be interpreted as independent effects after adjustment for potentially influential patient characteristics.

Fourth, nine HRCT findings were tested separately using a conventional p-value threshold of 0.05 without a supplied multiplicity-adjustment strategy. This increases the possibility that one or more statistically significant findings could arise through repeated testing. The strongest associations—particularly those involving tree-in-bud pattern, fibrosis, and calcified nodules, all with $p<0.001$ and large stage differences—are less likely to be explained solely by borderline statistical fluctuation, whereas associations with p-values closer to 0.05, including consolidation, pleural effusion, and pleural thickening, warrant more cautious interpretation. This consideration does not invalidate the reported analyses but supports greater emphasis on effect magnitude, confidence intervals, biological plausibility, and consistency across findings rather than dichotomous statistical significance alone.

Fifth, detailed HRCT acquisition parameters, reader blinding, the number and experience of image interpreters, procedures for resolving disagreements, and interobserver reliability were not available in the study evidence. These factors are relevant to reproducibility because recognition of some HRCT features depends on acquisition technique and reader interpretation. Contemporary imaging guidance continues to emphasize standardized CT technique, systematic pattern recognition, and integration of radiological findings with the broader clinical context (15). The two-hospital Lahore setting provides locally relevant evidence but may also limit generalizability to populations with different tuberculosis epidemiology, referral patterns, treatment histories, disease severity, or access to cross-sectional imaging. Despite these limitations, the study contributes stage-stratified local data across a broad range of pulmonary and pleural HRCT abnormalities. A particular strength is the simultaneous assessment of nine predefined findings across active and inactive groups, allowing inflammatory-appearing and chronic structural patterns to be evaluated within the same analytical framework. The inclusion of pulmonary, airway, nodal, and pleural manifestations also provides a more comprehensive representation of thoracic tuberculosis than assessment of isolated parenchymal features alone. Future studies should use independently defined microbiological and clinical reference criteria for tuberculosis activity, standardized HRCT acquisition and interpretation protocols, blinded image assessment where feasible, and explicit assessment of interobserver agreement. Larger multicenter cohorts would permit multivariable analysis of potential confounders and more precise estimation of associations. Longitudinal follow-up from active disease through treatment completion would further clarify which abnormalities resolve, persist, or evolve into post-tuberculosis structural disease. Contemporary PTLD literature also supports integrating radiological assessment with pulmonary function, respiratory symptoms, exercise capacity, and patient-reported outcomes to characterize the clinical consequences of residual disease more comprehensively (17–19).

CONCLUSION

High-resolution computed tomography demonstrated distinct stage-associated patterns among adults with pulmonary tuberculosis. Tree-in-bud pattern, consolidation, pleural effusion, and lymphadenopathy were more frequent in participants classified as having active tuberculosis, whereas fibrosis, calcified nodules, and pleural thickening were more frequent in those with inactive disease. Cavitary lesions showed a higher frequency in active tuberculosis but did not reach statistical significance, while bronchiectasis showed no meaningful stage-related difference. These findings support the use of HRCT as an adjunctive imaging modality for characterizing pulmonary and pleural abnormalities across tuberculosis stages, while interpretation should remain integrated with clinical and microbiological assessment rather than relying on individual HRCT features alone.

DECLARATIONS

Author Contributions:	Concept: AA, HMF; Design: HMF, TI; Data Collection: HN, SES, MK, MNUHB, MF; Analysis: AA, HMF; Drafting: AA, HMF, TI.
Ethical Approval:	Ethical approval was obtained from Al Razi Institute, Lahore, Pakistan.
Informed Consent:	Written informed consent was obtained from all participants.
Conflict of Interest:	The authors declare no competing interests.
Funding:	This research received no external funding.
Data Availability:	Data supporting the findings of this study are available from the corresponding author upon reasonable request, subject to applicable ethical and confidentiality restrictions.
Acknowledgments:	Not applicable.
AI Declaration:	Generative AI tools were used solely for language and readability improvement. The authors reviewed and verified the final manuscript and take full responsibility for its content and integrity.

REFERENCES

1. World Health Organization. Global tuberculosis report 2025. Geneva: World Health Organization; 2025.
2. Nachiappan AC, Rahbar K, Shi X, Guy ES, Mortani Barbosa EJ Jr, Shroff GS, et al. Pulmonary tuberculosis: role of radiology in diagnosis and management. Radiographics. 2017;37(1):52–72.
3. Ravimohan S, Kornfeld H, Weissman D, Bisson GP. Tuberculosis and lung damage: from epidemiology to pathophysiology. Eur Respir Rev. 2018;27(147).
4. Allwood BW, Byrne A, Meghji J, Rachow A, Van Der Zalm MM, Schoch OD. Post-tuberculosis lung disease: clinical review of an under-recognised global challenge. Respiration. 2021;100(8):751–763.
5. Khan R, Malik NI, Razaque A. Imaging of pulmonary post-tuberculosis sequelae. Pak J Med Sci. 2020;36(ICON-Suppl). doi:10.12669/pjms.36.ICON-Suppl.1722.
6. Majmudar DK, Rajput DK. Role of HRCT in diagnosing disease activity of pulmonary tuberculosis. Int J Contemp Med Res. 2017;4(8):1724–1727.
7. Rasheed W, Qureshi R, Jabeen N, Shah HA, Khan RN. Diagnostic accuracy of high-resolution computed tomography of chest in diagnosing sputum smear positive and sputum smear negative pulmonary tuberculosis. Cureus. 2020;12(6):e8467. doi:10.7759/cureus.8467. PMID:32642373.
8. Ahmad MW, Rashid N, Arooj S, Shahzadi S. Diagnostic accuracy of high-resolution computed tomography in assessing activity of pulmonary tuberculosis. J Sheikh Zayed Med Coll. 2020;11(4). doi:10.47883/jszmc.v11i04.99.
9. Anwar S, Nawaz A, Moinuddin IA, Kashif M, Mumtaz A, Bhatti MI. Patterns of HRCT in patients with pulmonary tuberculosis at a tertiary care hospital. J Rawalpindi Med Coll. 2024;28(2). doi:10.37939/jrmc.v28i2.2480.
10. Meghji J, Simpson H, Squire SB, Mortimer K. A systematic review of the prevalence and pattern of imaging-defined post-TB lung disease. PLoS One. 2016;11(8):e0161176.
11. Soekardi A, Icksan AG, Ernes A. The role of chest HRCT in diagnosis active tuberculosis and lung destruction. J Prima Medika Sains. 2023;5(2):169–174.
12. Biswas SS, Borpatragohain D, Thoumoung C. Findings of HRCT thorax in patients of sputum-positive pulmonary tuberculosis in a tertiary health care centre of North East India. J Med Sci. 2022;8(1):47.
13. Yadav S, Rawal G. Understanding the spectrum and management of post-tuberculosis lung disease: a comprehensive review. Cureus. 2024;16(6).
14. Wetscherek MTA, Sadler TJ, Lee JY, Karia S, Babar JL. Active pulmonary tuberculosis: something old, something new, something borrowed, something blue. Insights Imaging. 2022;13. doi:10.1186/s13244-021-01138-8. PMID: 35001143.
15. Pattamapaspong N, Kanthawang T, Peh WCG, Hammami N, Bouaziz MC, Ladeb MF. Imaging of thoracic tuberculosis: pulmonary and extrapulmonary. BJR Open. 2024. doi:10.1093/bjro/tzae031. PMID: 39363908.
16. Young S, Chua B, Tan Q, Leong C, Wong J, Phua I, et al. Pleural and parenchymal radiological characteristics of tuberculous pleuritis and correlation with microbiological and molecular diagnostic yield. BMC Pulm Med. 2025. doi:10.1186/s12890-025-03995-1. PMID: 41239354.
17. Singh S, Allwood BW, Chiyaka T, Kleyhans L, Naidoo C, Moodley S, et al. Immunologic and imaging signatures in post tuberculosis lung disease. Tuberculosis (Edinb). 2022. doi:10.1016/j.tube.2022.102244. PMID: 36007338.
18. Sehgal IS, Dhooria S, Muthu V, Salzer HJF, Agarwal R. Burden, clinical features, and outcomes of post-tuberculosis chronic obstructive lung diseases. Curr Opin Pulm Med. 2023. doi:10.1097/MCP.0000000000001026. PMID: 37902135.
19. Fumagalli G, Mencarini J, Sini I, Allavena L, Tadolini M, Mantero M, et al. Post-tuberculosis lung disease: a guide for clinicians. Infection. 2025. doi:10.1007/s15010-025-02645-2. PMID: 40996670.