Imaging Spectrum in Pulmonary Tuberculosis

Harsimran Bhatia[1] Ravinder Kaur[2] Irwinjit Kaur[2] Karan Garg[2]

[1] Department of Radiodiagnosis and Imaging, Post Graduate Institute of Medical Education and Research, Chandigarh, India
[2] Department of Radiodiagnosis and Imaging, Government Medical College and Hospital, Chandigarh, India

Address for correspondence Ravinder Kaur, MD, Department of Radiodiagnosis and Imaging, Government Medical College and Hospital, Sector 32, Chandigarh 160031, India (e-mail: drravinder19@gmail.com).

Indographics 2024;3:33–44.

Abstract

Pulmonary tuberculosis is one of the most common pulmonary infections prevalent in our country. Although definitive diagnosis is microbiological, characteristic imaging features help in establishing a certain diagnosis in most of the cases. While chest radiography remains the primary screening tool, computed tomography helps in clinching the diagnosis with a spectrum of findings described on cross-sectional imaging. The radiologist must be familiar with the features denoting potential complications and differential diagnosis to aid better treatment planning and case prognostication. This article describes in detail the imaging features of pulmonary tuberculosis, active disease, disease sequelae, and complications.

Keywords
- tuberculosis
- imaging
- pulmonary tuberculosis
- pulmonary TB
- CT

Introduction

Tuberculosis (TB) is a commonly encountered airborne disease caused by acid-fast bacillus, *Mycobacterium tuberculosis*, and is major cause of mortality and morbidity, particularly in developing countries of Southeast Asia.[1–3] The disease is included in the top 10 causes of death worldwide, as it is present in all countries and across all age groups.[4] Early diagnosis and treatment are thus essential to eliminate this treatable disease. Substantial efforts have been made as part of the World Health Organization End TB strategy resulting in 9% decrease in global incidence of TB between 2015 and 2019.[4]

Indian Scenario

According to the reported figures, our country has the highest estimated burden of TB infection across the globe (35–40 crores), of which nearly 18 to 36 lakh people develop TB disease every year. About 5 to 10% of the infected patients develop TB over the course of their lives, usually within the first 2 years after initial infection.[5] The risk for TB disease after infection depends on several factors, of which immunological status is the most important. This risk is increased >25 times among contacts of bacteriologically confirmed TB patients compared to general populations, 16 to 21 times in case of human immunodeficiency virus (HIV) coinfection, and 3 to 4 times in other immune-compromised status like diabetes, etc.[5]

Our policies thus not only focus on early diagnosis and treatment of active TB, instead focuses on prevention of TB as an equally important task to eliminate this disease, particularly by active case finding among high-risk groups.[6] Prevention of TB disease by treatment of TB infection is a critical component of the National Strategic Plan 2017-25 for Ending TB (NSP) in India by 2025. Radiography plays a miniscule but significant role in this program at the diagnostic level which is the most important step in the cascade of events ensuring timely and proper disease treatment.

Diagnosis

Diagnosis of TB is predominantly microbiological, with primary diagnostic tools being sputum smear microscopy and culture. While sputum smear examination using Ziehl-Neelsen stain-based and light-emitting diode-based fluorescent

DOI https://doi.org/10.1055/s-0044-1788611.
ISSN 2583-8229.

Thieme Medical and Scientific Publishers Pvt. Ltd., A-12, 2nd Floor, Sector 2, Noida-201301 UP, India

microscopy are routinely employed under Revised National Tuberculosis Control Programme (RNTCP) guidelines, culture is not routinely recommended. The latter is only to be used for follow-up of patients with drug-resistant TB. Newer methods include rapid molecular diagnostic testing that includes line probe assay and nucleic acid amplification techniques using the GeneXpert system.[5]

The Indian guidelines recommend the use of chest radiography (CXR) as a screening tool to increase the diagnostic sensitivity as well as in people with HIV before starting preventive TB treatment.[5] Cases with positive findings on the chest X-ray must undergo further microbiological confirmation.

Terms and Definitions

While the disease has been classically divided into primary and postprimary types, there are few other terms that need elaboration at this point. Location-wise, chest TB encompasses the entire spectrum of pulmonary TB (PTB) and other sites like lymph nodes (LNs) and pleura. The latter two although accompany the pulmonary form invariably, these are, however, included under the umbrella term of extrapulmonary TB. PTB can further be described as parenchymal or airway TB, depending upon the predominant site involved.[7]

Clinically, the terms primary and postprimary have been coined to differentiate the mechanism of acquisition, spread, and histological patterns that vary. Primary TB refers to a primary infection with tubercle bacilli that occurs with inhalation of bacilli on first exposure. The disease involves subpleural inflammation and cellular infiltration resulting in a localized patch of pneumonia called as Ghon's focus. Lymphatic spread to mediastinal LNs constitutes the Ghon's complex.[7,8] Although the disease is known to classically affect children with a high prevalence of patients less than 5 years of age in endemic areas, the prevalence in adults is increasing, accounting for up to 34% of the primary TB cases, with most of these occurring in adults with immunodeficiency, in particular, HIV-infected individuals.[8–10] Parenchymal lesions in these cases heal and calcify to form residual tuberculomas and fibrous scars in most of the cases.[11]

In a small percentage of cases of primary TB (5–10%), the disease may progress to involve confluent extensive multilobar disease.[12,13] This pattern is most often seen in infants or patients with immunodeficiencies, where the immune system fails to contain the primary focus of infection the first time.[11]

The third most common and the most typical form encountered is the postprimary TB (PPT) which is characterized by reactivation of a latent primary focus, leading to chronic granulomatous infection. Infection is most often seen in patients during malnutrition and systemic illnesses that compromise the immune system.[14]

Miliary TB is another less frequently encountered form of TB that occurs due to hematogenous dissemination seen in both primary and postprimary forms.[7] The radiological patterns of each of these will be discussed in detail further in the article.

Role of Imaging

Traditionally, imaging plays an indispensable role in patients suspected of disease for diagnosis or patients on treatment to look for complications.

Chest Radiograph

CXR remains the initial and primary investigation of choice in patients suspected to have PTB. Although it has lower sensitivity, according to the recent RNTCP guidelines, it is recommended in patients who are clinically symptomatic despite sputum smear negativity.[15,16] CXR is also carried out in patients who have radiographically occult disease or have clinical disease progression despite treatment to look for lung parenchymal involvement. Radiograph well demonstrates cavitation, bronchiectasis, that is parenchymal, and lung involvement with particular importance to LNs (►**Fig. 1**).

Computed Tomography

Computed tomography (CT) with its three-dimensional reconstruction and multiple planar reformatting techniques has excellent spatial resolution and submillimetric reconstruction. Contrast-enhanced CT (CECT) with intravenous contrast helps in denoting the LNs and shows central necrosis well. High-resolution lung algorithms denote the parenchyma and airways well and denote nodules, cavitation, and bronchial wall thickening.

Sonography

Role of sonography (USG) cannot be undermined as it is bedside in sick population, easily available, and radiation-free modality. It has an important role in pleural disease to look for empyema and pleural effusion or collections. USG shows pleural thickening with fluid and septations in cases of empyema. Resolution of pleural effusion and/or collection in response to treatment can be documented.[17]

Magnetic Resonance Imaging

Magnetic resonance imaging is an evolving modality that well demonstrates parenchyma and pleura. Being a radiation-free modality, it can be put to use in pediatric population.[7,18]

Classically, four entities of TB disease patterns are described. These include tubercular pleuritis, tracheobronchial TB, miliary pattern of disease, and gangliopulmonary form. While the latter is exclusive to primary TB, others are seen in postprimary disease as well.[8]

Summary of findings on various imaging modalities is mentioned in ►**Table 1**.

Imaging Features

The imaging findings follow a spectrum as described below.

Consolidation

Consolidation is usually unilateral and sublobar or subpleural in location.[11] Parenchymal disease in primary TB has a predilection for middle and lower lobes due to higher ventilation.[7]

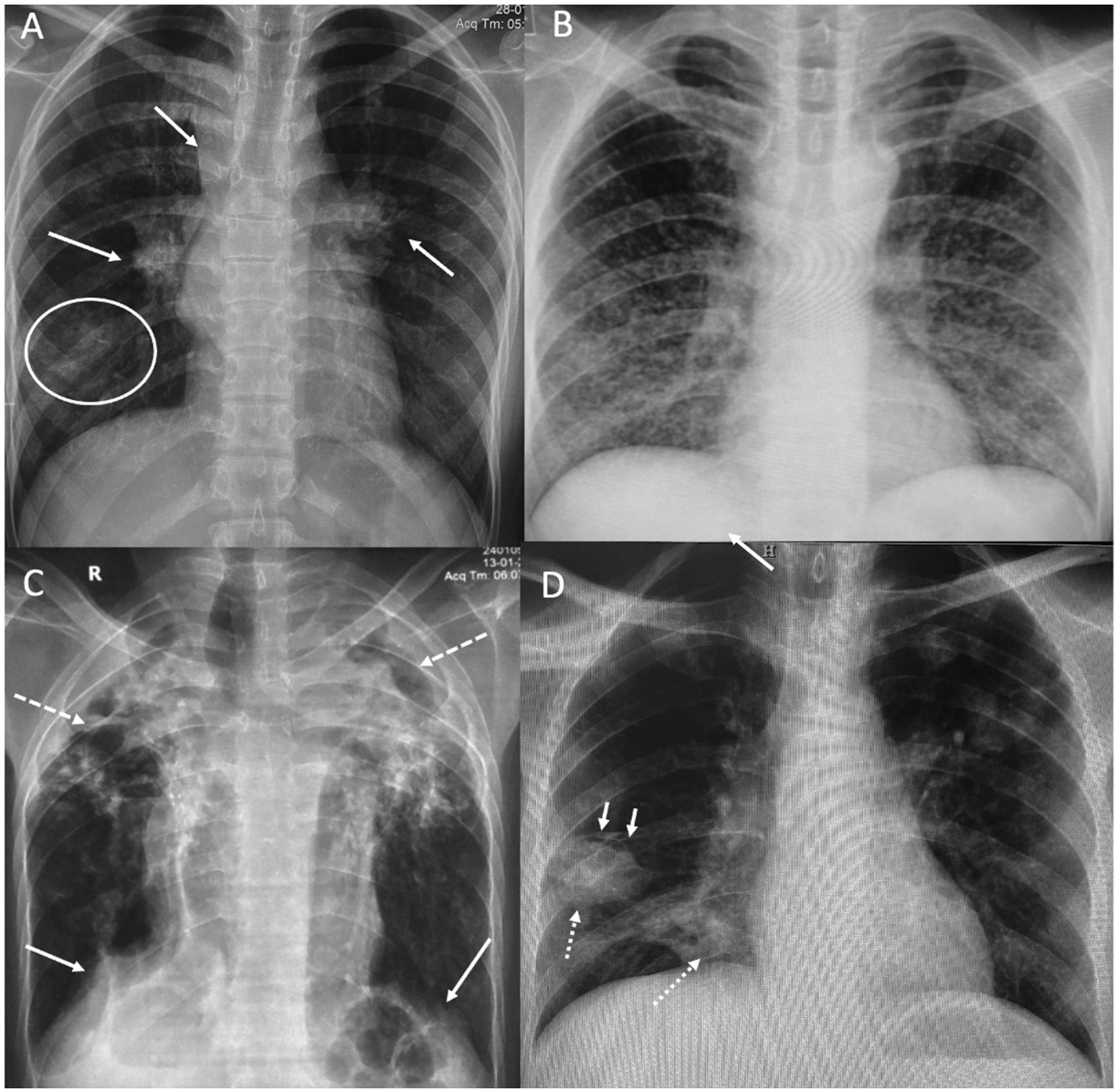

Fig. 1 Chest radiographs. (**A**) Primary tuberculosis (TB)—Enlarged right paratracheal and hilar lymph nodes (white arrows) with homogenous radio-opacity in right lower lung zone. (**B**) Miliary TB: Uniformly distributed miliary shadows in both lungs. (**C**) Post-TB lung disease: Volume loss of both upper lobes with heterogeneous opacities and cavitary changes (dashed arrows) with hyperinflated lower lung lobes and tenting of hemidiaphragms bilaterally (arrows). (**D**) Aspergillomas: Cavities in right lower lung zone (dashed arrows) with aspergilloma and air-crescent sign (white arrows).

Table 1 Summary of findings on various imaging modalities

1.	Radiograph	• Primary investigation for diagnosis • Prognosis during course of disease to look for cavities, fibrosis, lymph nodes, pleural effusion • Complications: aspergillomas
2.	Ultrasound	• Easily available • Bedside modality in acute setting • Particular use in pleural pathologies and guided interventions
3.	CT	• High sensitivity and specificity • Nodules, consolidation, lymph nodes, cavities, pleural thickening • Complications: bronchopleural fistula, CPA, Rasmussen's aneurysms
4.	MRI	• Radiation-free modality • Disease prognosis and response to treatment • Utility in pediatric population

Abbreviations: CPA, chronic pulmonary aspergillosis; CT, computed tomography; MRI, magnetic resonance imaging.

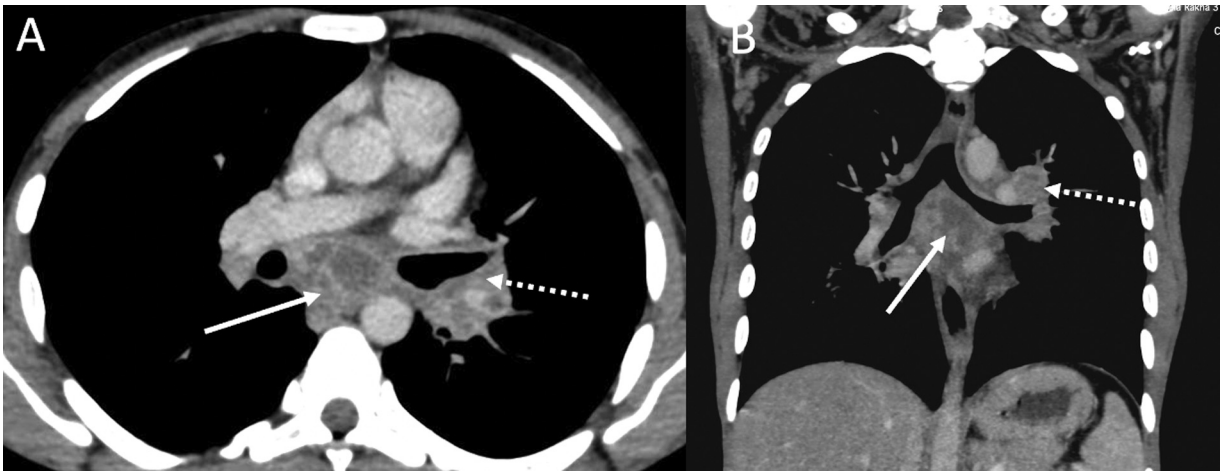

Fig. 2 Axial (**A**) and coronal (**B**) sections of a contrast-enhanced computed tomography (CECT) chest in an 18-year-old male show enlarged necrotic mediastinal (white arrows) and left hilar lymph nodes (dashed white arrows).

Dense homogenous consolidation is usually seen.[19] This primary parenchymal focus as mentioned previously constitutes the Ghon's focus.[20] These lesions may contain dormant bacilli, which can develop into PPT. The enlarged LNs are usually on the same side as that of consolidation; however, any group of LNs can be involved. About 31% cases have bilateral lymphadenopathy[19] (►**Fig. 1A**). LN enlargement is seen in up to 96% children and 43% adults with primary TB.[19,21,22]

On CECT scan, the enlarged LNs may show rim sign, heterogeneous or homogenous enhancement. The "rim sign" consisting of a low-density necrotic center and peripheral rim enhancement although characteristic, is not pathognomonic of disease[23,24] (►**Fig. 2**). This rim sign may also be seen with a number of other conditions, including atypical mycobacteria, histoplasmosis, metastases (from head/neck/testicular malignancy), and lymphoma.[8]

Since PPT results either from reinfection or from reactivation of dormant bacilli in primary infection (90% of cases), the pattern of consolidation differs from primary disease. PPT usually begins with necrotizing consolidation followed by transbronchial spread. It usually affects the apicoposterior segments of the upper lobes and superior segments of the lower lobes.[8] This typical distribution is attributed to the high concentration of oxygen and to less effective lymphatic drainage of these segments as compared to other areas of the lung.[13] Consolidation may heal with or without calcification or may cavitate.

Cavities

Liquefaction of caseous necrosis results in low attenuation areas within the consolidation. The liquefied central necrosis on communication with the tracheobronchial tree forms air cavities in half of the cases.[25,26] The walls of the cavities are usually thick and irregular. While active infection may present with thick-walled cavities and pericavitary consolidation, thin-walled cavities on the other hand may represent healed infection and are difficult to distinguish from other air-containing mimics like pneumatoceles, cysts, or bullae[27] (►**Figs. 3** and **4**). Intracavitary air-fluid levels are seen secondary to superimposed bacterial or fungal infection

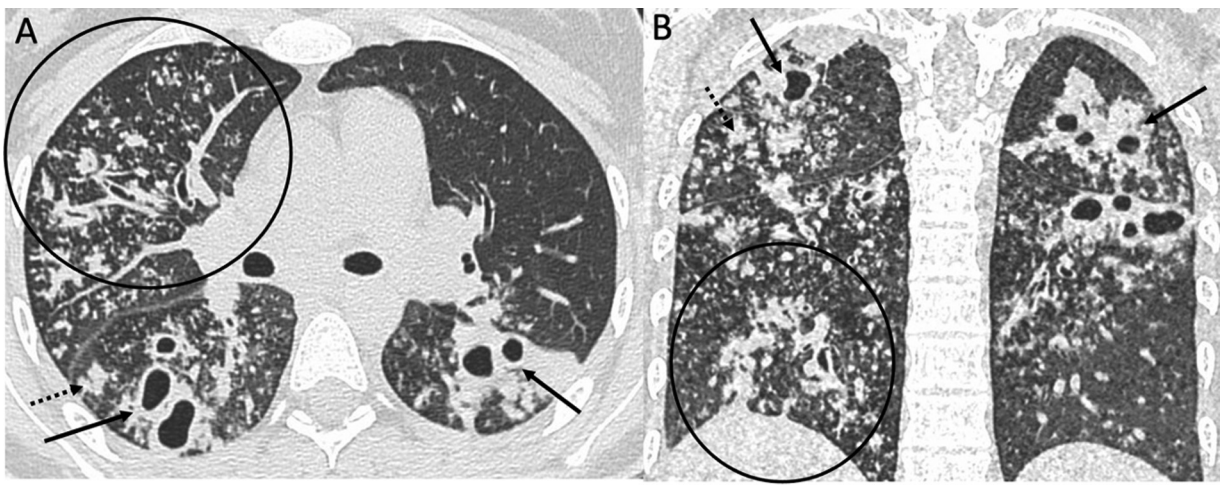

Fig. 3 Active tuberculosis (TB): Axial (**A**) and coronal (**B**) sections of high-resolution computed tomography (HRCT) chest in a sputum positive TB case show multiple centrilobular nodules with tree-in-bud appearance (encircled) in both lungs, with tiny patches of consolidation (dashed black arrows). Black arrows denote cavitation in both lungs.

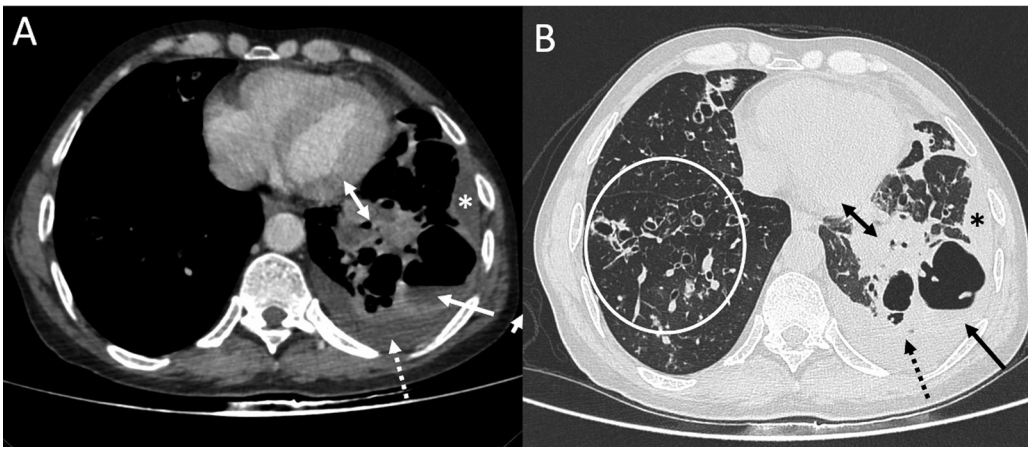

Fig. 4 Axial mediastinal (**A**) and lung window (**B**) sections denote cavities with air-fluid levels in left lower lobe (white arrows in **A** and black arrows in **B**); consolidation (double-headed arrow); associated pleural effusion (*). Bronchiectatic changes are encircled in right lower lobe.

in about 10% of the cases.[21,28] On follow-up, these cavities may heal with fibrosis leading to architectural distortion and/or cicatricial bronchiectasis, characteristic of PPT.[29]

Nodules

All types of nodules can occur in TB, ranging from centrilobular (CLN), random, and perilymphatic to miliary nodules. Size may vary from micronodules to large nodules.

(1) *CLNs*: These nodules occur due to the bronchogenic spread of infection to other segments of the lungs which can occur due to coughing and result in inflammatory exudates filling the terminal bronchioles, respiratory bronchioles, and the alveolar ducts. Although seen anywhere in the affected lung, these nodules are often seen as ill-defined micronodules in a segmental or lobar distribution, often involving the lower lobes and away from the cavity.[30] These are often considered a reliable marker of the disease activity. These are evident radiographically in 20% of cases on X-ray.[28] High-resolution CT (HRCT) is the imaging modality of choice to detect early bronchogenic spread. These can give "tree-in-bud" appearance which typically consists of 2- to 4-mm sharply marginated linear branching CLNs around terminal and respiratory bronchioles[31] (►**Figs. 5** and **6**). Although CLN and tree-in-bud opacities can be seen in other infections (bacterial, viral, fungal, and parasitic infections), chronic bronchiolitis, aspiration, and inhalation of foreign substances, these, however when associated with cavitation or nodular opacities in the appropriate clinical setting point to a specific diagnosis of PTB. Healing of cavities occur by fibrosis or nodules.[14]

(2) *Perilymphatic nodules*: Perilymphatic nodules are seen around the bronchovascular bundle or in subpleural location. Perilymphatic nodules and the "galaxy sign" though typically seen in sarcoidosis have been recently reported to occur in TB as well.[32] These may thus suggest TB as a diagnosis in an appropriate clinical setting.[29]

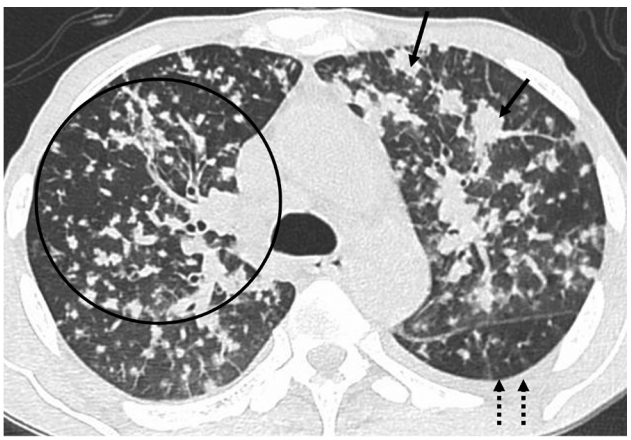

Fig. 5 Axial lung window in a 34-year-old male patient with multidrug-resistant (MDR) tuberculosis (TB) shows multiple centrilobular nodules scattered in both lungs (encircled) with tiny patches of consolidation in left upper lobe (black arrows) and minimal left pleural effusion (dashed black arrows).

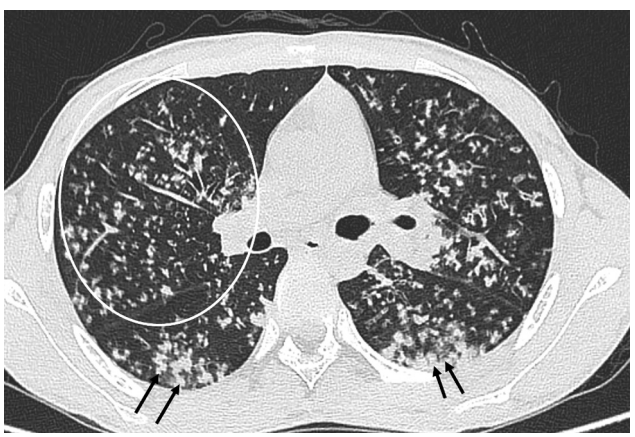

Fig. 6 Axial lung window in an active case of tuberculosis (TB) shows multiple centrilobular nodules giving tree-in-bud appearance (encircled) with clustering and surrounding ground-glass opacities (GGOs) in superior segments of both lower lobes (black arrows).

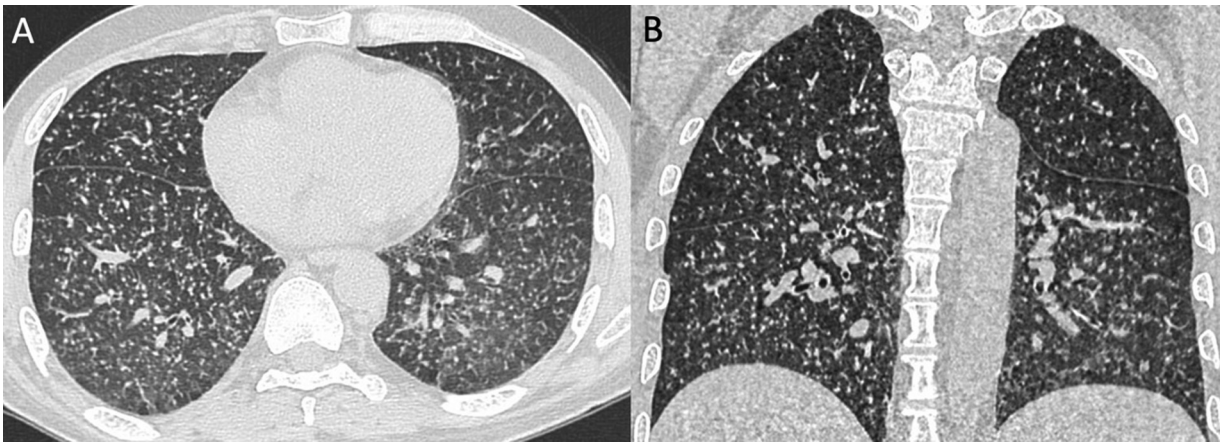

Fig. 7 Miliary tuberculosis (TB): Axial (**A**) and coronal (**B**) sections of high-resolution computed tomography (HRCT) chest in a 16-year-old male patient with sputum smear positive TB show miliary nodules scattered in both lungs.

(3) *Miliary nodules*: These result from hematogenous dissemination of tubercle bacilli and are seen as 1 to 3 mm sharply defined nodules in random distribution representing innumerable small granulomas in the lungs (►**Fig. 7**). These may be associated with interstitial septal thickening.[33] Though classically described in children, there is an increase in the percentage of adults presenting with this radiological disease form.[8,34] CXR is often normal in the initial stage of disease. In such cases, HRCT is the investigation of choice that clearly shows miliary nodules in the lungs.[34] These nodules increase gradually from a size of about 1 mm to 3 to 5 mm, when they become confluent and mimic the "snow-storm" appearance of sarcoid nodules.[7] This miliary pattern needs to be clearly differentiated from the tree-in-bud nodules. While the former are well defined and uniform in distribution and represent hematogenous dissemination, the latter are poorly defined in comparison, have a patchy distribution, and suggest air-borne spread.[14] Miliary TB often resolves with proper antitubercular treatment leaving no residual scars or calcifications.[14]

(4) *Larger nodules*: Larger nodules of 1 to 4 cm in size can also be seen due to coalescence of smaller nodules. Peribronchial distribution of these nodules indicates active disease. They can have irregular margins and may have satellite nodules. Potential differential diagnosis includes metastasis, lymphoma, etc.

Tracheobronchial TB

It can occur as a complication of primary TB as well as PPT due to perforation of an involved LN into a bronchus or by ascending endobronchial spread.[8] The incidence of tracheobronchial TB is about 2 to 4% in PTB cases.[35]

(1) *Larger airways*: In acute tracheobronchitis, CT may show smooth or irregular enhancing wall thickening leading to symmetric circumferential reduction of the airway diameter.[36,37] Adjacent lymphadenopathy may be seen. Rarely, peribronchial soft tissue or ulcerated polypoidal mass may be seen.[37] During the course of the disease, airway involvement often heals as severe bronchial stenosis due to fibrosis and/or associated bronchiectasis. Bronchus intermedius is the most frequently involved followed by left main bronchus.[29,35] Collapse of the distal lung segments can occur due to bronchial stenosis. Rarely, a calcified LN can erode into an adjacent bronchus and produce broncholiths with resultant collapse of respective segment/lobe.[38]

(2) *Smaller airways*: Involvement of smaller airways often results in bronchiolitis which may present as CLNs with tree-in-bud appearance.

Imaging Features of Disease/Response Assessment

A radiologist is frequently required to answer the important question pertaining to activity of the infection on imaging.

Active Disease

The following imaging findings suggest that the disease is active but definitive diagnosis is always made by isolation of *M. tuberculosis* from the sputum.

(1) *Consolidation*: While these can be detected on radiographs, CT is definitely more sensitive and picks up smaller and subtle patches of consolidation well. Consolidation located in apicoposterior segments of bilateral upper lobes and/or superior segments of lower lobe with associated ipsilateral hilar/paratracheal lymphadenopathy are more likely in favor of TB.[39] Lower lobe (basal segment) affection though rare, may be seen in elderly patients.[40]

(2) CLN or clustered nodules in the above described location

(3) Miliary nodules in bilateral lungs

(4) Cavities (with thick walls) and/or surrounding consolidation suggest active infection (►**Fig. 8**)

(5) Air-fluid level in a cavity may be seen in superimposed bacterial infection (►**Fig. 9**)

Findings denoting active disease are summarized in ►**Table 2**.

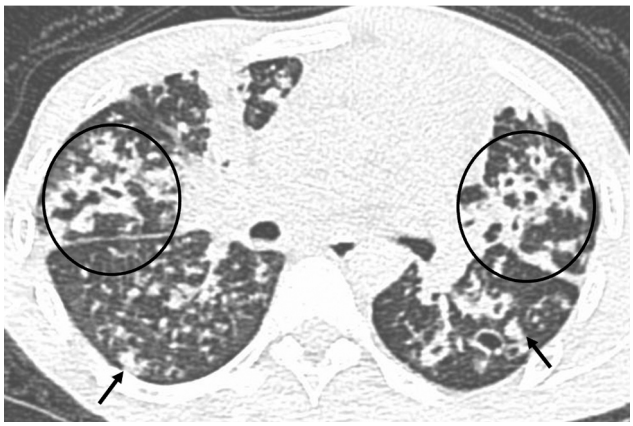

Fig. 8 Axial lung window in a 26-year-old male with sputum positive pulmonary tuberculosis (PTB) shows multiple centrilobular nodules in both lungs with clustering at places (black arrows) with cavitary breakdown at places (encircled).

Table 2 Summary of findings denoting active disease

1.	Patches of consolidation
2.	Centrilobular nodules with tree in bud appearance[a]
3.	Miliary nodules
4.	Cavities with surrounding consolidation
5.	Necrotic mediastinal lymphadenopathy with lung changes
6.	Fibrocavitary changes in upper lobes with consolidation and/or centrilobular nodules[a,b]

[a]In an appropriate clinical setting favoring tubercular etiology.
[b]Suggest reactivation of disease in a postprimary tuberculosis (TB) case.

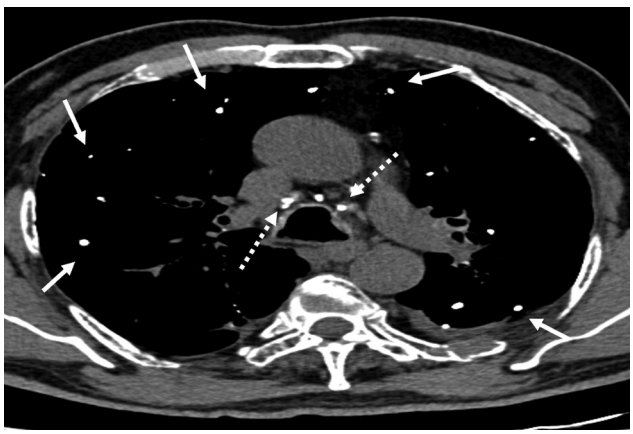

Fig. 10 Calcified nodules: Axial mediastinal window of noncontrast computed tomography (CT) in a 36-year-old female with healed tuberculosis (TB) shows multiple calcified nodules (white arrows). Dashed white arrows denote calcified mediastinal lymph nodes.

Disease Sequelae

Post-tubercular lung disease (PTLD) refers to chronic respiratory abnormalities affecting the airways, parenchyma, pleura, and vasculature in patients with previous TB. Tubercular lesions may heal with the following imaging features[41]:

(1) *Tuberculomas* are persistent nodules or mass-like lesions seen in primary TB as well as PPT. These may be solitary or multiple in the affected lungs with size ranging from millimetric lesions to large-sized masses (about > 5 cm).[7] These often have a smooth wall with well-defined margins and remain stable in size over the years. These may undergo calcification (20–30% cases) and cavitation (10–50% cases) over the years that follow (►**Fig. 10**). A large number of these lesions have satellite lesions that consist of small round opacities located in the immediate vicinity of the main lesion.[8]

(2) *Fibro-parenchymal lesions* occur secondary to healing of active disease. These may consist of architectural distortion, bronchiectasis, and atelectasis[8] (►**Fig. 11**). Cases with end-stage disease may present as complete lung or multilobar destruction which often occurs due to a combination of severe involvement of the airway as well as parenchyma. Although this destructive form of parenchymal involvement is expected in PPT, recent literature reports children and adolescents to be equally prone to undergo this variety of lung destruction.[33] Thin-walled

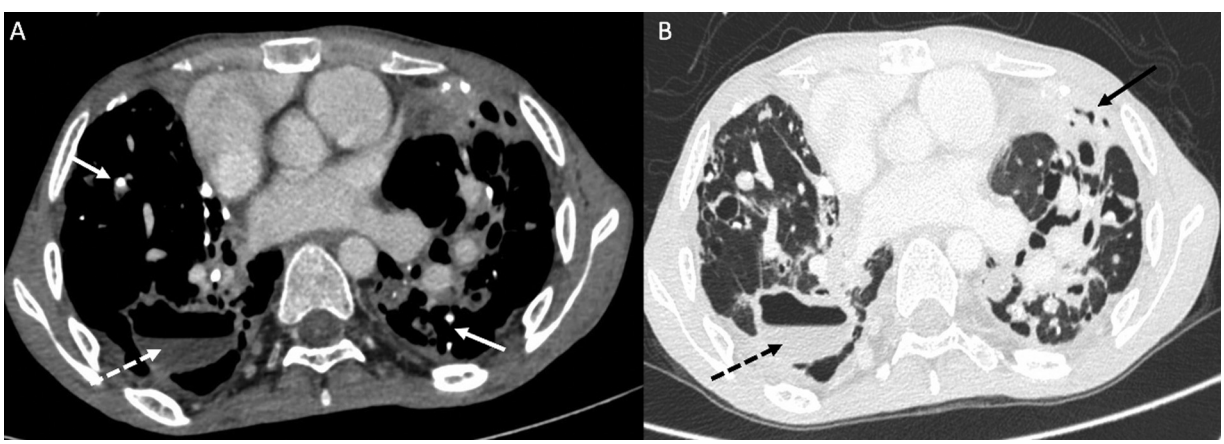

Fig. 9 Axial contrast-enhanced computed tomography (CT) (mediastinal window: **A**; lung window: **B**) in a case with partially treated tuberculosis (TB) shows multiple calcified nodules in both lungs (white arrows in **A**). Dashed white and black arrows in **A** and **B** denote thick-walled cavity with air-fluid level suggestive of secondary infection. Fibro-bronchiectatic changes in the lingula (black arrow in **B**).

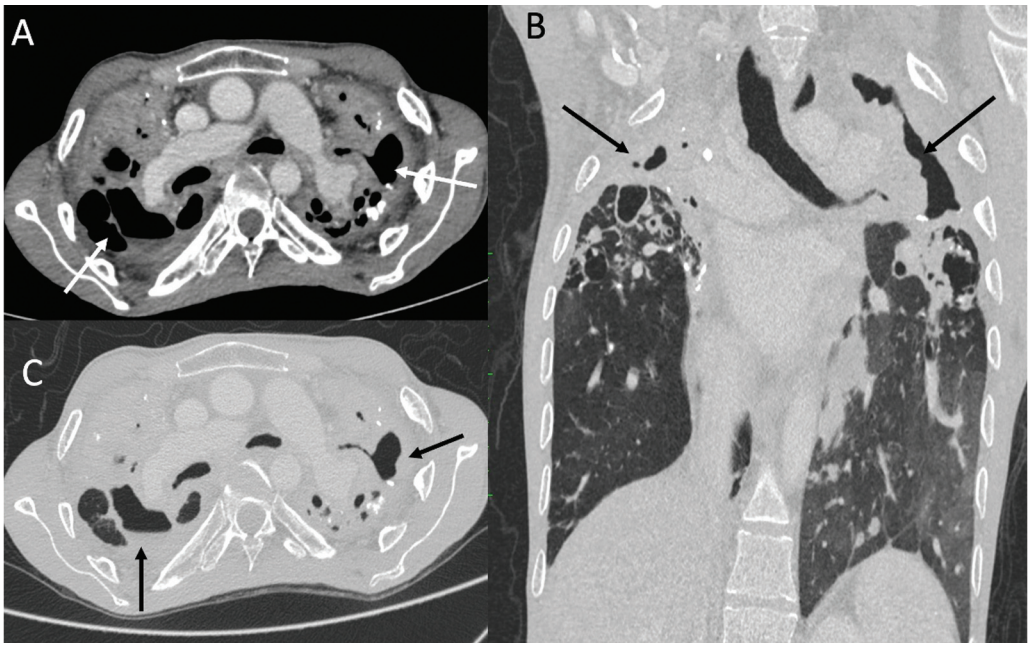

Fig. 11 Post-tubercular lung disease. Axial mediastinal (**A**), coronal (**B**), and axial (**C**) lung window sections in a case of untreated tuberculosis (TB) show volume loss of both upper lobes with extensive fibrocavitary changes (white arrows in **A** and black arrows in **B**, **C**). Multiple calcific foci are also seen bilaterally.

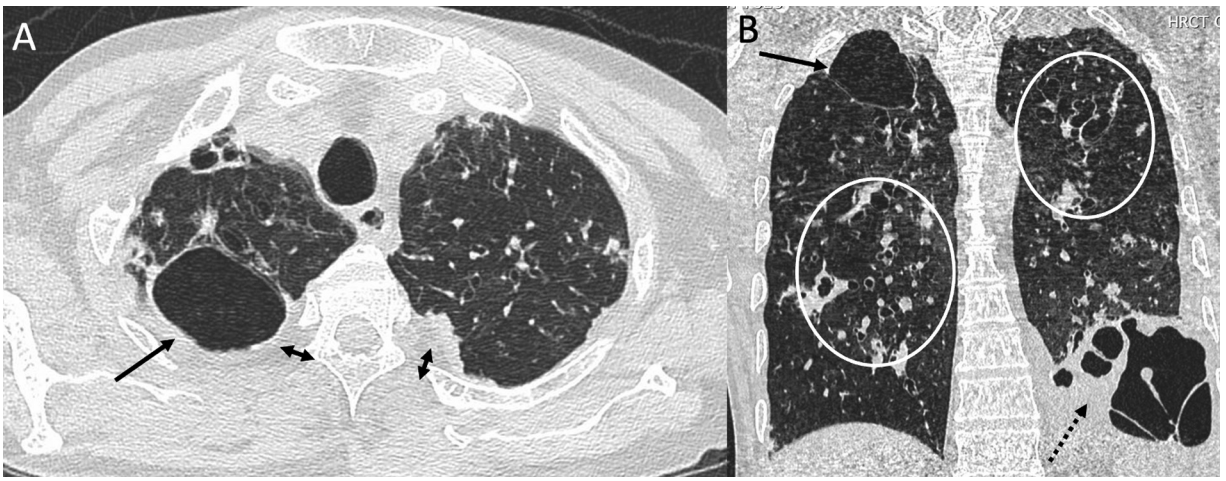

Fig. 12 Post-tubercular lung disease. Axial and coronal lung window of high-resolution computed tomography (HRCT) chest in a treated case of tuberculosis (TB) shows a pneumatocele (black arrows in **A**) in right upper lobe with associated pleural thickening bilaterally (double-headed arrows in **A**); thick-walled cavity with intracavitary strands in left lower lobe (dashed black arrow in **B**) and bronchiectatic changes (encircled in **B**).

cavities may persist which may result in colonization of saprophytic fungi like aspergillus.[29]

(3) *Well-defined nodules* with or without calcification may persist in healed TB[29] (►**Fig. 10**).

Complications

Acute Respiratory Distress Syndrome

Findings of miliary or endobronchial dissemination of TB may be superimposed on substantial areas of bilateral ground-glass opacity or consolidation as a consequence of acute respiratory distress syndrome (ARDS) owing to TB. Patients recuperating from ARDS or those experiencing significant consolidation as a result of TB may develop many cystic lesions.[42] The cystic lesions, which may dissolve over many months or remain persistent, may resemble bullae or pneumatoceles[42] (►**Fig. 12**).

Aspergilloma

Intrapulmonary complications usually occur due to the cavitating lesions. Chronically persisting cavities are often colonized by *Aspergillus spp*. resulting in the development of a "fungal ball." Patients with aspergilloma often have persistent symptoms of productive cough.[14] Although a radiograph can well detect an aspergilloma with an "air crescent sign," CT proves to be a more sensitive investigation while adding additional information about the affected lung. The

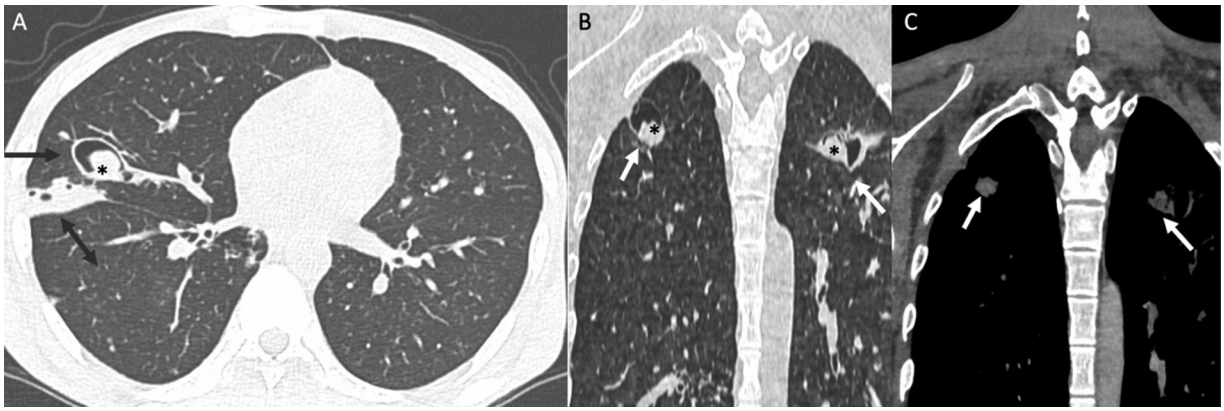

Fig. 13 Aspergilloma: Axial (**A**) and coronal high-resolution computed tomography (HRCT) lung (**B**) and coronal mediastinal window (**C**) sections in a 45-year-old male with past history of tuberculosis (TB) show a thin-walled cavity (black arrow in **A**) in right middle lobe and both upper lobes (white arrows in **B** and **C**) with intracavitary soft tissue (denoted by * in **A** and **B**) with surrounding air crescent.

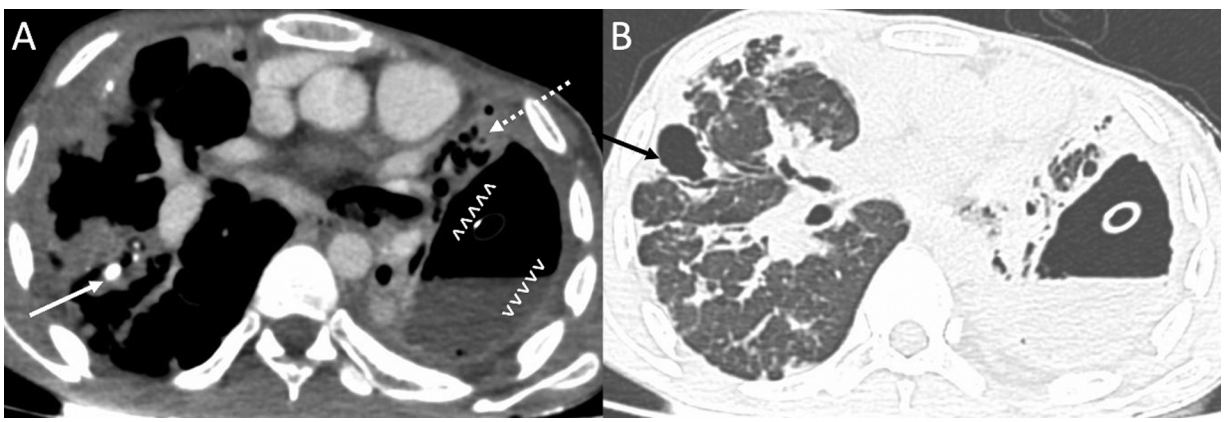

Fig. 14 Axial mediastinal and lung window sections of contrast-enhanced computed tomography (CECT) chest show volume loss of left hemithorax with bronchiectatic changes (dashed white arrow in **A**) and pleural collection with air-fluid level (arrowheads denote enhancing pleural layers) with intercostal drainage tube in situ; fibrocalcific and cavitary changes in right lung (white arrow in **A** and black arrow in **B**).

radiological appearance often varies with time period since colonization of cavity. Immature aspergilloma appears as network or mesh with multiple scattered irregular pockets of intracavitary air. This network then coalesces to form a mature fungus ball classically seen as a round mobile intracavitary soft tissue mass with a surrounding crescent of air (Monod sign)[43,44] (►**Fig. 13**).

Bronchopleural Fistula

Bronchopleural fistula may develop both in the active disease stage due to extensive lung involvement and in the healing phase after treatment. The latter occurs due to rupture of a cavity into pleura leading to empyema and subsequent bronchopleural fistula formation[14] (►**Figs. 14** and **15**).

Vascular Complications

These occur due to erosion of a pulmonary artery branch forming Rasmussen pseudoaneurysm (►**Fig. 16**). These patients present with hemoptysis which may be life-threatening in certain cases. Necrotizing granulomatous vasculitis develops in the pulmonary artery branches and veins in the vicinity of the cavity and leads to vascular thrombosis.[29]

Bronchial Stenosis

Bronchial stenosis can occur due to the involvement of the tracheobronchial tree. The reported prevalence of tuberculous bronchial stenosis is 10% in patients undergoing routine bronchofibroscopy.[14] CT often shows concentric luminal diametric reduction of the bronchi with symmetric wall thickening (►**Fig. 17**).

Chronic Pulmonary Aspergillosis

Chronic pulmonary aspergillosis (CPA) is an often overlooked complication occurring in patients with preexisting structural lung disease, most commonly TB in our setting. Imaging spectrum ranges from simple aspergilloma to chronic cavitary pulmonary aspergillosis and chronic fibrotic pulmonary aspergillosis depending upon the status of the patient's immunity. It is characterized by multiple thick-walled cavities with aspergillomas, pleural thickening, and architectural distortion.[45] Prompt diagnosis by clinical, radiological, and

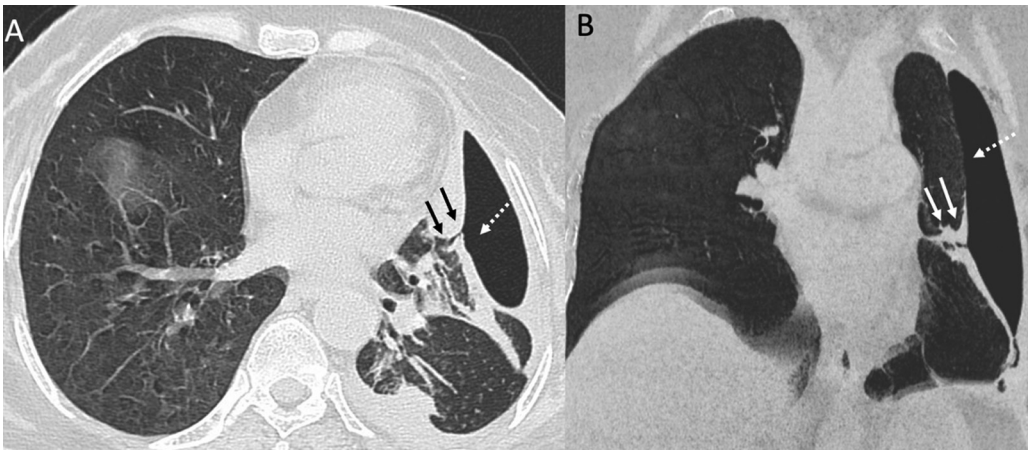

Fig. 15 Bronchopleural fistula. Axial lung (**A**) and coronal minimum intensity projection images (**B**) in a 43-year-old male with past history of tuberculosis (TB), presenting with new-onset breathlessness show volume loss of left hemithorax with ipsilateral mediastinal shift, loculated pneumothorax (dashed white arrow) communicating with the lingular segmental bronchus (black arrows in **A** and white arrows in **B**).

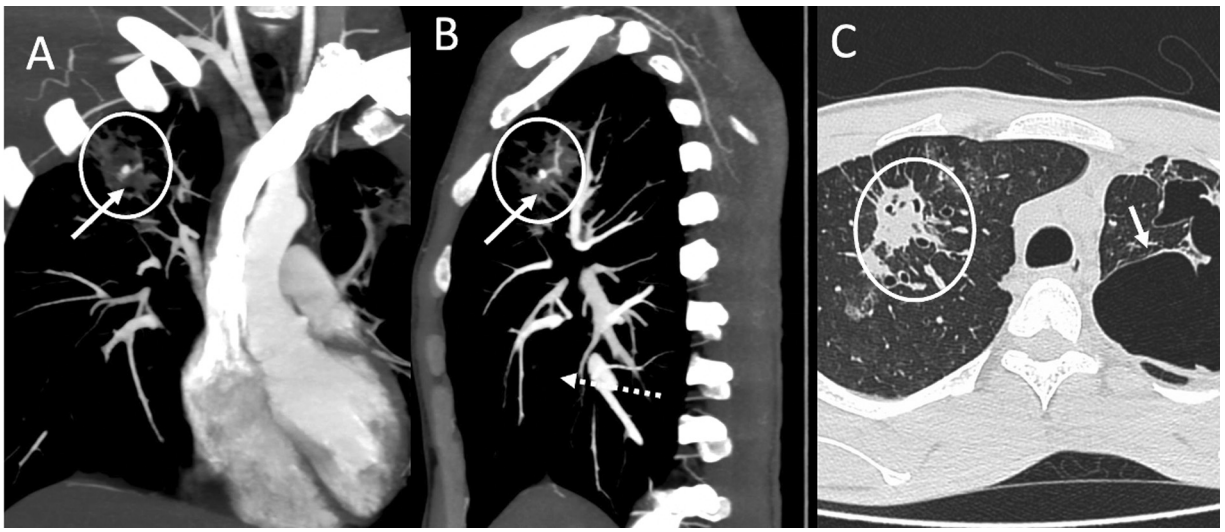

Fig. 16 Rasmussen's aneurysm: Coronal (**A**) and sagittal maximum intensity projection images (**B**). Axial lung window sections of contrast-enhanced computed tomography (CECT) chest show a fibrotic patch (encircled) in right upper lobe with a contrast-filled outpouching arising from a subsegmental branch of the upper lobe right pulmonary artery.

microbiological criteria is essential to initiate antifungal treatment and prognosticate the patient.

Scar Carcinoma

Concurrent or subsequent malignancy may develop in patients with past PTB. Scarring is associated with increased cancer risk in ipsilateral lung.[46] The most common malignancy in peripheral lung scars is adenocarcinoma. The clinician needs to be more sensitive in ruling out an underlying malignancy in patients with parenchymal scarring.

Differential Diagnosis

Although we have described in detail the typical imaging features of PTB on radiograph as well as cross-sectional imaging, there is often a considerable overlap in the findings of other infective etiologies and TB. Thus, presence of characteristic imaging features in an appropriate clinical setting (patient history, symptoms, and laboratory parameters) favors PTB over other infective organisms.

Primary and Postprimary Disease

Lymphadenopathy, per se has a number of differential diagnosis including lymphoma, fungal infections, and metastatic cancers. Characteristic parenchymal findings constituting the Ghon's complex may point toward a diagnosis of primary TB, particularly in pediatric population.[11]

Lung findings: Imaging features of patchy heterogeneous consolidation and associated pleural effusion may be seen in any other bacterial pneumonias as well and is difficult to differentiate the two. Necrotic lymphadenopathy and associated cavitation favors tubercular etiology in cases showing poor response to conventional antibiotic therapy.[9] Multilobar multisegmental cavitation with surrounding CLNs

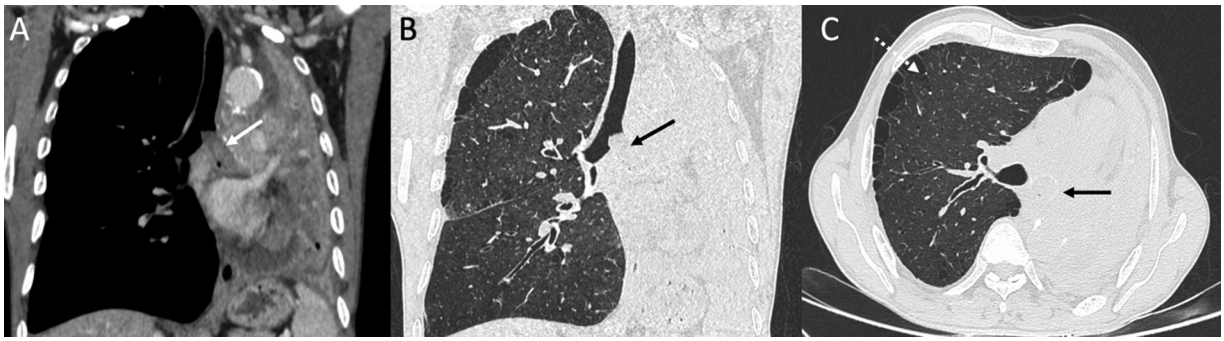

Fig. 17 Bronchial involvement: Coronal mediastinal (**A**) and lung window (**B**), axial lung window section (**C**) in a treated case of tuberculosis (TB) with sequelae show complete obliteration of left main bronchus with soft tissue (white arrow in **A** and black arrows in **B** and **C**) and collapse of left lung. Dashed white arrow in **C** denotes paraseptal emphysematous changes in the right lung.

favors TB over other cavitating pneumonias like staphylococcal and fungal infections.[4] While miliary disease may be seen in systemic fungal infections, a strong clinical suspicion and microbiological confirmation is necessary in cases where miliary nodules are the only parenchymal abnormality.[8,42]

Atypical mycobacteria: Radiographic and CT findings of TB may substantially overlap with atypical mycobacterial infections. While the classic cavitary form mimics TB in all features, characterized by upper lobe cavitation, distortion, and CLNs, the nonclassic or bronchiectatic form has distinguishing features including lower lobe predominant bronchiectasis.[47]

Perilymphatic nodules (including the galaxy sign and cluster sign) with associated septal thickening may be seen in both sarcoidosis and TB often complicating the final diagnosis.[4]

Mimics of Disease Sequelae

Features of lung architectural distortion, parenchymal destruction, and fibro-parenchymal scarring may be observed as an end result of a cavitating lung disease including TB. A strong clinical suspicion is often required to raise a red flag for CPA in patients with PTLD for better prognostication, with features like aspergillomas, pleural thickening, and thick irregular cavities favoring the latter.[45]

Table 3 Summary of imaging findings in tuberculosis on CT

1.	Location	• Upper lobes/superior segments of lower lobes • Subpleural consolidation in primary TB
2.	Nodules	• Centrilobular (tree in bud) • Miliary • Perilymphatic (cluster sign/galaxy sign)
3.	Pleural involvement	• Pleural effusion • Empyema • Bronchopleural fistula
4.	Lymph nodes	• Mediastinal and hilar • Enhancing • Central necrosis (rim sign) • Calcified

Abbreviations: CT, computed tomography; TB, tuberculosis.

Table 4 Key points of the article

1.	Optimal utilization of available imaging modalities in the right clinical context helps in establishing an accurate diagnosis
2.	CT is the investigation of choice and clearly demonstrates imaging features pertaining to specific clinical type (primary TB, postprimary TB, miliary TB)
3.	Miliary nodules can be seen in both primary and postprimary disease types and reflect hematogenous dissemination of disease
4.	Features of primary TB (consolidation and lymph nodes) are not only seen in children, instead are being increasingly diagnosed in the adult population owing to immunosuppression
5.	Signs of active disease include patches of consolidation, centrilobular, or miliary nodules, cavities with surrounding consolidation

Abbreviations: CT, computed tomography; TB, tuberculosis.

Conclusion

Due to the high burden of TB in our country, a radiologist in the present scenario must be familiar with the typical as well as atypical findings of the disease and establish a key role in patient management by looking for complications and features of active disease.

►**Table 3** summarizes the imaging findings of TB. ►**Table 4** summarizes the key points of the article.

Conflict of Interest
None declared.

References

1 Cegielski JP, Chin DP, Espinal MA, et al. The global tuberculosis situation. Progress and problems in the 20th century, prospects for the 21st century. Infect Dis Clin North Am 2002;16(01):1–58
2 Corbett EL, Watt CJ, Walker N, et al. The growing burden of tuberculosis: global trends and interactions with the HIV epidemic. Arch Intern Med 2003;163(09):1009–1021
3 Tufariello JM, Chan J, Flynn JL. Latent tuberculosis: mechanisms of host and bacillus that contribute to persistent infection. Lancet Infect Dis 2003;3(09):578–590

4 Wetscherek MTA, Sadler TJ, Lee JYJ, Karia S, Babar JL. Active pulmonary tuberculosis: something old, something new, something borrowed, something blue. Insights Imaging 2022;13(01):3
5 tbcindia.gov.in. [Internet]. Guidelines for programmatic management of tuberculosis preventive treatment in India. July 2021. Accessed July 5, 2024 at: https://tbcindia.gov.in
6 Getahun H, Matteelli A, Chaisson RE, Raviglione M. Latent Mycobacterium tuberculosis infection. N Engl J Med 2015;372(22): 2127–2135
7 Bhalla AS, Goyal A, Guleria R, Gupta AK. Chest tuberculosis: radiological review and imaging recommendations. Indian J Radiol Imaging 2015;25(03):213–225
8 Van Dyck P, Vanhoenacker FM, Van den Brande P, De Schepper AM. Imaging of pulmonary tuberculosis. Eur Radiol 2003;13(08): 1771–1785
9 Restrepo CS, Katre R, Mumbower A. Imaging manifestations of thoracic tuberculosis. Radiol Clin North Am 2016;54(03): 453–473
10 Havlir DV, Barnes PF. Tuberculosis in patients with human immunodeficiency virus infection. N Engl J Med 1999;340(05):367–373
11 Beigelman C, Sellami D, Brauner M. CT of parenchymal and bronchial tuberculosis. Eur Radiol 2000;10(05):699–709
12 Rubin SA. Tuberculosis and atypical mycobacterial infections in the 1990s. Radiographics 1997;17(04):1051–1059
13 McAdams HP, Erasmus J, Winter JA. Radiologic manifestations of pulmonary tuberculosis. Radiol Clin North Am 1995;33(04): 655–678
14 Andreu J, Cáceres J, Pallisa E, Martinez-Rodriguez M. Radiological manifestations of pulmonary tuberculosis. Eur J Radiol 2004;51 (02):139–149
15 Swingler GH, du Toit G, Andronikou S, van der Merwe L, Zar HJ. Diagnostic accuracy of chest radiography in detecting mediastinal lymphadenopathy in suspected pulmonary tuberculosis. Arch Dis Child 2005;90(11):1153–1156
16 Graham SM, Ahmed T, Amanullah F, et al. Evaluation of tuberculosis diagnostics in children: 1. Proposed clinical case definitions for classification of intrathoracic tuberculosis disease. Consensus from an expert panel. J Infect Dis 2012;205 Suppl 2(Suppl 2): S199–S208
17 Akhan O, Demirkazik FB, Ozmen MN, et al. Tuberculous pleural effusions: ultrasonic diagnosis. J Clin Ultrasound 1992;20(07): 461–465
18 Naranje P, Bhalla AS, Sherwani P. Chest tuberculosis in children. Indian J Pediatr 2019;86(05):448–458
19 Leung AN. Pulmonary tuberculosis: the essentials. Radiology 1999;210(02):307–322
20 Burrill J, Williams CJ, Bain G, Conder G, Hine AL, Misra RR. Tuberculosis: a radiologic review. Radiographics 2007;27(05): 1255–1273
21 Woodring JH, Vandiviere HM, Fried AM, Dillon ML, Williams TD, Melvin IG. Update: the radiographic features of pulmonary tuberculosis. AJR Am J Roentgenol 1986;146(03):497–506
22 Choyke PL, Sostman HD, Curtis AM, et al. Adult-onset pulmonary tuberculosis. Radiology 1983;148(02):357–362
23 Mukund A, Khurana R, Bhalla AS, Gupta AK, Kabra SK. CT patterns of nodal disease in pediatric chest tuberculosis. World J Radiol 2011;3(01):17–23
24 Kim WS, Moon WK, Kim IO, et al. Pulmonary tuberculosis in children: evaluation with CT. AJR Am J Roentgenol 1997;168(04): 1005–1009
25 Lee KS, Im JG. CT in adults with tuberculosis of the chest: characteristic findings and role in management. AJR Am J Roentgenol 1995;164(06):1361–1367
26 Miller WT, Miller WT Jr. Tuberculosis in the normal host: radiological findings. Semin Roentgenol 1993;28(02):109–118
27 Raniga S, Parikh N, Arora A. Is HRCT reliable in determining disease activity in pulmonary tuberculosis. Ind J Radiol Imag 2006;16:221–228
28 Hadlock FP, Park SK, Awe RJ, Rivera M. Unusual radiographic findings in adult pulmonary tuberculosis. AJR Am J Roentgenol 1980;134(05):1015–1018
29 Bhalla AS, Jana M. Clinico-Radiologic Series: Imaging of Chest Infections. 1st ed. India: Jaypee Publishers; 2018
30 Curvo-Semedo L, Teixeira L, Caseiro-Alves F. Tuberculosis of the chest. Eur J Radiol 2005;55(02):158–172
31 Im JG, Itoh H, Shim YS, et al. Pulmonary tuberculosis: CT findings–early active disease and sequential change with antituberculous therapy. Radiology 1993;186(03):653–660
32 Oda M, Saraya T, Shirai T, Ishikawa N, Fujiwara M, Takizawa H. Multiple huge “cluster” and “galaxy” signs on chest radiography in a patient with pulmonary tuberculosis. Respirol Case Rep 2019; 7(03):e00398
33 Veedu PT, Bhalla AS, Vishnubhatla S, et al. Pediatric vs adult pulmonary tuberculosis: A retrospective computed tomography study. World J Clin Pediatr 2013;2(04):70–76
34 Kwong JS, Carignan S, Kang EY, Müller NL, FitzGerald JM. Miliary tuberculosis. Diagnostic accuracy of chest radiography. Chest 1996;110(02):339–342
35 Lee KS, Kim YH, Kim WS, Hwang SH, Kim PN, Lee BH. Endobronchial tuberculosis: CT features. J Comput Assist Tomogr 1991;15 (03):424–428
36 Moon WK, Im JG, Yeon KM, Han MC. Tuberculosis of the central airways: CT findings of active and fibrotic disease. AJR Am J Roentgenol 1997;169(03):649–653
37 Arora A, Bhalla AS, Jana M, Sharma R. Overview of airway involvement in tuberculosis. J Med Imaging Radiat Oncol 2013; 57(05):576–581
38 Conces DJ Jr, Tarver RD, Vix VA. Broncholithiasis: CT features in 15 patients. AJR Am J Roentgenol 1991;157(02):249–253
39 Yeh JJ, Yu JK, Teng WB, et al. High-resolution CT for identify patients with smear-positive, active pulmonary tuberculosis. Eur J Radiol 2012;81(01):195–201
40 Yeh JJ, Chen SC, Chen CR, et al. A high-resolution computed tomography-based scoring system to differentiate the most infectious active pulmonary tuberculosis from community-acquired pneumonia in elderly and non-elderly patients. Eur Radiol 2014;24(10):2372–2384
41 Allwood BW, Byrne A, Meghji J, Rachow A, van der Zalm MM, Schoch OD. Post-tuberculosis lung disease: clinical review of an underrecognised global challenge. Respiration 2021;100(08):751–763
42 Jeong YJ, Lee KS. Pulmonary tuberculosis: up-to-date imaging and management. AJR Am J Roentgenol 2008;191(03):834–844
43 Logan PM, Müller NL. CT manifestations of pulmonary aspergillosis. Crit Rev Diagn Imaging 1996;37(01):1–37
44 Thompson BH, Stanford W, Galvin JR, Kurihara Y. Varied radiologic appearances of pulmonary aspergillosis. Radiographics 1995;15 (06):1273–1284
45 Garg M, Bhatia H, Chandra T, et al. Imaging spectrum in chronic pulmonary aspergillosis. Am J Trop Med Hyg 2022;108(01):15–21
46 Yu YY, Pinsky PF, Caporaso NE, et al. Lung cancer risk following detection of pulmonary scarring by chest radiography in the prostate, lung, colorectal, and ovarian cancer screening trial. Arch Intern Med 2008;168(21):2326–2332, discussion 2332
47 Erasmus JJ, McAdams HP, Farrell MA, Patz EF Jr. Pulmonary nontuberculous mycobacterial infection: radiologic manifestations. Radiographics 1999;19(06):1487–1505

