Pulmonary tuberculosis: Something old, something new, something borrowed, something blue

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Authors: M. T. A. Buzan\textsuperscript{1}, M. M. Coman\textsuperscript{2}, M. M. Duma\textsuperscript{3}, A. M. Calin\textsuperscript{3}, S. A. Sfrangeu\textsuperscript{3}, C. M. Pop\textsuperscript{3}; \textsuperscript{1}Blaj/RO, \textsuperscript{2}Campia Turzii/RO, \textsuperscript{3}Cluj-Napoca/RO
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Learning objectives

To illustrate the old, classical signs and the new, atypical appearances in pulmonary tuberculosis (PTB).

To describe unusual signs for PTB, borrowed from other lung disorders, in which they were initially described.

To discuss PTB imaging features that are associated with positive or negative Acid-Fast Bacilli (AFB) smears.
Background

By 2035, the aim of WHO's Global tuberculosis (TB) Programme is to make sure TB is no longer a threat to people worldwide. Increased effort is made both in diagnosis and treatment strategies to complete TB eradication.[1] Therefore, radiological assessment plays an important part in the success of the programme. In order to interpret the radiology associated with tuberculous infection one must be aware of the wide spectrum of radiological presentations of PTB.

Classically, PTB is classified in primary and postprimary.[2] Primary TB is seen in patients not previously exposed to \textit{M tuberculosis}, most common in infants and children, with the highest prevalence under 5 years of age. Postprimary TB occurs in patients previously sensitized to \textit{M tuberculosis} and remains a disease of adolescence and adulthood. The term "postprimary TB" refers to both reinfection with \textit{M tuberculosis} and reactivation of the disease. [3] More than 50% of recurrent disease occurring in endemic areas results from reinfection, reactivation remaining an important entity among older or immunocompromised individuals, but representing a small fraction of the global TB burden.[4] Approximately 1/10 people with primary PTB present clinically and, of untreated cases, approximately 1/10 reactivate mainly at a time of relative immunodeficiency.[5]

Primary infection is respiratory of origin: after inhalation, the 2-10 μm droplets laden with bacilli (transmitted from an infected host) reach the terminal bronchioles carried by cilia. Inoculation takes place in the best ventilated areas of the lungs, most frequently in the anterior segments of the upper lobes, middle lobe and lingula, and the basal segments of the lower lobes. When the mycobacteria reach the pulmonary alveoli, they invade and replicate within alveolar macrophages; after a few weeks, this results in granulomas, which can develop into larger tuberculosis. Delayed hypersensitivity becomes manifest at 4-10 weeks after initial infection and the tuberculin reaction turns positive. The macroscopic hallmark of hypersensitivity is the development of caseous necrosis in the pulmonary focus and/or in the involved lymph nodes. The primary focus is known as the Ghon focus. The Ghon focus together with the enlarged draining lymph nodes form the primary complex, called the Ranke or Ghon complex. The Gohn focus usually undergoes healing or enlarges as disease progresses. Healing may result in a visible scar that may be dense and contain foci of calcification. [2, 6] The extent of the primary infection is dependent on different factors: number and virulence of the agent, natural and acquired resistance of the host, and hypersensitivity. [7]

Chest radiography remains the first choice of initial evaluation of patients with PTB; however, normal radiographic findings may be seen in up to 15% of patients with proved disease.[1] CT scan provides more accurate information on the extent and distribution of...
the disease, the presence of cavities and satellite lesions that cannot be visualized on chest-x ray.[8] HRCT can visualize cavities in hidden areas such as paramediastinal and retrocardiac areas, centrilobular nodules and lymphadenopathies.[9]

Primary PTB typically presents with consolidation and regional lymphadenopathy, whereas postprimary PTB typically displays inflammation and caseating necrosis, with more pronounced nodular appearances, conglomerate masses, destruction and cavitation of the parenchyma.[5]
Findings and procedure details

Something old - classical patterns

**Primary TB** manifests as four main entities: parenchymal disease, lymphadenopathy, miliary disease, and pleural effusion. Typically, *parenchymal disease* manifests as dense, homogeneous parenchymal consolidation in any lobe; predominance in the lower and middle lobes is suggestive of the disease. In children under 2 years of age, lobar or segmental atelectasis is frequently seen, most often involving the anterior segment of an upper lobe or the medial segment of the middle lobe.[3]

**Lymphadenopathy:** Is typically unilateral, involving the right hilum and paratracheal region, or can be bilateral in about one-third of cases. It can either be associated with other manifestations of TB, or may represent the sole radiographic feature. Nodes greater than 2 cm in diameter may present with a low-attenuation center, secondary to caseation necrosis, and peripheral rim enhancement at CT and are highly suggestive of active disease.[3] Associated parenchymal infiltrates are encountered on the same side as nodal enlargement, especially in the subpleural areas, in approximately two-thirds of pediatric cases of primary PTB. Their small volume renders them difficult to see on conventional radiography; therefore, CT is often necessary to detect these subtle peripheral lung infiltrates.[2] Gangliopulmonary TB may be complicated by perforation of an adenopathy into a bronchus, retroobstructive pneumonia, and/or atelectasis (epituberculosis;). On HRCT scans, acute tracheobronchial TB manifests as irregular or smooth circumferential bronchial narrowing associated with mural thickening. A retroobstructive infiltrate in primary TB most commonly appears as an area of homogenous consolidation. Obstructive atelectasis or overinflation results from the compression of a bronchus by an adjacent enlarged node, with a typical right sided distribution, at the level of the right lobar bronchus or bronchus intermedius [2,10].
Fig. 1: Something old - Right paratracheal and mediastinal adenopathy associated with right middle lobe consolidation in a 6 year old patient

References: Department of Pediatric Pulmonology, "Leon Daniello" Pulmonology and Tuberculosis Care Clinics, Cluj-Napoca, Ro

Postprimary TB: Classical HRCT findings in postprimary PTB include centrilobular or airspace nodules, branching linear and nodular opacities ("tree-in-bud" sign), areas of consolidation, ground-glass opacities, cavitations, bronchial wall thickening, miliary nodules, tuberculomas, calcifications, parenchymal bands, interlobular septal thickening, pericicatricial emphysema, and fibrotic changes.[11]

The earliest findings are patchy heterogeneous, poorly defined areas of consolidation, mainly in the apical and posterior segments of the upper lobes and less frequent in the apical segments of the lower lobes. Usually, more than one pulmonary segment is involved, with bilateral disease seen in 1/3 to 2/3 of cases.[3] HRCT-pathologic correlations revealed that air space consolidations consist of centrally located granulomas containing caseation necrosis and marginal nonspecific inflammation, seen as ground-glass opacities. In their evolution, these regions liquefy and form cavities by draining through the tracheobronchial tree.[8,12]
Bronchogenic spread is radiographically identified in 1/5 cases of postprimary TB. It manifests as multiple, ill-defined nodules, with tendency to coalesce and a segmental or lobar distribution, typically involving the lower lung zones and the peripheral areas of consolidations or cavities. While on CT scan, it is identified in 95% of cases, HRCT is the imaging technique of choice to reveal early bronchogenic spread.[2] Micronodules, most often seen in the acute early stages of TB, are not visible on standard chest radiography.[13] Typical findings are 2-4 mm **centrilobular nodules** and sharply marginated linear branching opacities, "**tree-in-bud**".[2] Centrilobular nodules reflecting granulomatous lesions may show coalescence and enlargement of multiple foci to make an airspace nodule.[14] The term "tree-in-bud" was first used by Im et al. [12] to describe the appearance of the endobronchial spread of TB. The terminal tufts might represent caseation necrosis within the bronchioles and alveolar ducts, while the stalk might represent a lesion that affected the last order bronchus within the secondary lobule. The tree-in-bud appearance is characteristic, but not pathognomonic for active TB.[13,15]

**Airway involvement** is characterized by bronchial stenosis, leading to lobar collapse or hyperinflation, obstructive pneumonia, and mucoid impaction. Bronchial stenosis is seen in 10%-40% of patients with active TB and is best demonstrated with CT, which usually shows long segment of circumferential narrowing with irregular wall thickening, luminal obstruction, and extrinsic compression. It also results in tree-in-bud opacities and traction bronchiectasis, particularly of the upper lobes.[3] In active disease, the airways are irregularly narrowed and have thick walls, whereas in fibrotic disease, the airways are smoothly narrowed and have thin walls. The left main bronchus is involved more frequently in fibrotic disease, whereas both main bronchi are equally involved in active disease.[6]

**Cavitation**, the hallmark of postprimary TB, affects about 50% of patients. The cavities typically have thick, irregular walls, which become smooth and thin with successful treatment. Cavities are usually multiple and occur within areas of consolidation. Cavities may demonstrate air-fluid levels; this can also indicate superinfection.[3]
**Fig. 2:** Something old: Classical CT pattern showing a large thick-wall cavity, tree-in-bud and bronchial wall thickening.

**References:** "Leon Daniello" Pulmonology and Tuberculosis Care Clinics, Cluj-Napoca, Ro

**Miliary** TB refers to haematogenous dissemination, resulting in widespread random distribution of active TB granulomas throughout the lung. The nodules are uniform in size, measuring from 1-4 mm, with a slight lower lobe predominance, often associated with intra- and interlobular septal thickening [16,17]. They may coalesce to form focal or diffuse consolidation.[3]. Chest radiography is usually normal at the onset of symptoms, and hyperinflation may be the earliest feature.[3,16] The nodules are usually discernable on CXR around 4 weeks following the onset of symptoms,[5] CT can demonstrate miliary disease before it becomes radiographically apparent.[2,16] It occurs in 2-6% of primary TB and also occurs somewhat more frequently in reactivation TB. In the latter situation, miliary TB may be seen in association with typical parenchymal changes or may be the only pulmonary abnormality.[6] Miliary disease has been reported to be associated with severe immunosuppression [2,6], elderly and infants, within 6 months of initial exposure [3]. The more widespread location of these micronodules, including subpleural location, excludes the diagnosis of lymphangitis carcinomatosa and bronchiolitis.[2] This pattern
can be differentiated from tree-in-bud due to its uniform distribution, whereas tree-in-bud nodules have a patchy distribution.[18]

Fig. 3: Something old - Miliary PTB

References: Radiology Department, Guy's and St Thomas' Hospital, London, UK
**Fig. 4: Something old - Miliary PTB with associated apical consolidation in the right upper lobe**

References: "Leon Daniello" Pulmonology and Tuberculosis Care Clinics, Cluj-Napoca, Ro

**Something new - atypical patterns**

There is a changing radiological pattern of PTB, with fading of the strict classical distinction between primary and postprimary disease.[2] Adolescents and young adults are at risk of developing cavities in the lung apices within 6 months to 2 years of documented primary infection.[19] Cavitary TB may be a manifestation of
recent primary infection or, more commonly in endemic settings, of reinfection.[4] In industrialized countries, however, there seems to be a shift of primary TB towards adults. Primary infection in adults most frequently results in parenchymal consolidation without adenopathy.[2]

Known risk factors for development of more atypical patterns of PTB include conditions that are associated with defects in cell-mediated immunity: HIV infection, malnutrition, drug and alcohol abuse, malignancy, end-stage renal disease, transplant recipients, diabetes mellitus and corticosteroid or other immunosuppressive therapy.[6]

Fig. 5: Something new - Primary focus in the right upper lobe and associated right paratracheal adenopathy with rim-enhancement in a kidney transplant patient

References: Radiology Department, Guy's and St Thomas' Hospital, London, UK

Immunodeficient and elderly patients can present with the childhood type (hilar/mediastinal adenopathies and parenchymal abnormalities), frequently combined with formation of cavities (mixed type).
Fig. 6: Something new - two tuberculomas in the right lower lobe and associated mediastinal adenopathy with rim-enhancement in an elderly patient

References: Radiology Department, Guy’s and St Thomas’ Hospital, London, UK

Atypical presentations such as gangliopulmonary forms, ventrobasal infiltrates, hilar-mediastinal adenopathy, exudative pleuritis and extra-thoracic manifestations are frequent in the elderly and HIV-infected adults. This results in late diagnosis and delayed therapy.[2,6] Bacilli in dormant position, at low metabolic rate, maintain the hypersensitivity to tuberculous antigen. In situations of immunodepression, these bacilli can reactivate. In immunocompromised patients, the widespread lymphogenic and hematogenous dissemination, results in adenopathy and peripheral involvement. If immunity is inadequate, clinically active disease can develop within 5 years after infection, called progressive primary TB.[2]
Fig. 7: Something new - Right basal infiltrates and cavities on a conventional tomography in a patient suffering from diabetes mellitus

References: "Leon Daniello" Pulmonology and Tuberculosis Care Clinics, Cluj-Napoca, Ro

The radiographic appearance of HIV-associated PTB is dependent on the level of immunosuppression (table 1).[2,5,6,20] Most of the TB infections in HIV patients are postprimary TB.[2] The AIDS patients can have massive hematogenous dissemination following primary infection, and thus have high risk of more fulminant course and development of progressive primary TB during the first year after infection. Because of deficient cellular immunity, they are also prone to reactivation TB.[2]
<table>
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<th>Group:</th>
<th>Appearances:</th>
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| AIDS patients | During the first year after infection=> risk of developing progressive primary TB
| severe level of immunosuppression | =>10-20% of patients have normal chest films [2]
| | =>findings usually associated with primary disease, regardless of prior TB exposure status [2].
| | =>a miliary pattern of disease [2]
| | =>mediastinal and hilar adenopathy occur in 75% of cases [2]
| | =>extrapulmonary localizations (brain, pericardium, intestine, peritoneum, testes) [2]
| CD4 lymphocyte < 200 cells/mm3 | primary TB pattern (adenopathy, pleural effusion, areas of non-cavitary consolidation, more frequent in the middle-lower lobes, but often randomly distributed in the lung).[21]
| CD4 lymphocyte < 50 cell/mm3 | miliary, atypical patterns, or normal chest radiographs[21]
| Elderly patients | malnutrition (hypoalbuminemia) - deficient cellular immunity=> promotes reactivation TB[2,22]
| | an exogenic reinfection or "second" primary infection: elderly patients may have outlived their initial infecting organisms, as manifested by the negative tuberculin skin test, and become vulnerable to a "second primary infection" [2].
| | exogenic reinfection or infected for the first time in their life=> presentation similar to that found in children.[2]
The incidence of TB in patients with **idiopathic pulmonary fibrosis** (IPF) is >4x higher than that of the general population. Atypical manifestations (subpleural nodules or a lobar or segmental airspace consolidation) are common in patients with IPF and may mimic lung cancer or bacterial pneumonia.[6]

PTB in patients with **systemic lupus erythematosus** (SLE) has a higher incidence and prevalence due to abnormal function of alveolar macrophages and exposure to corticosteroid and cytotoxic drugs. TB in patients with SLE may show radiologic findings of miliary dissemination, diffuse consolidation, or primary TB.[6]

Most patients with **primary drug resistance** showed a primary pattern such as noncavitary consolidation, pleural effusion, and lymphadenopathy, whereas cavitary disease was common in patients who acquired multidrug-resistant TB secondary to noncompliance with therapy.[23] In one review article [6], the authors note from their experience, that the extensively-drug-resistant PTB manifests as an advanced pattern of primary TB (extensive consolidation with or without lymphadenopathy) in AIDS patients. [6]

**Something borrowed - unusual patterns**

The radiological features of TB may mimic those of other diseases, leading to a series of "borrowed" signs, initially described as specific for other pathological entities.

The Fleischner Society defines the **"reversed-halo sign"** (RHS) as "a focal, rounded area of ground-glass opacity surrounded by a more or less complete ring of consolidation" seen on CT images.[24] In 1999, a similar CT finding was described in a cryptogenic organizing pneumonia (COP) case. Zompatori et al. used the term "atoll sign". They noted that this CT appearance resembled the photographic negative of the "halo sign" and suggested that it should be regarded as a highly specific sign for COP.[25] Kim et al. named this finding the RHS and considered it to be relatively specific for COP.[26] Recently, the presence of RHS lesions in patients with PTB was described [27,28]. Patients with TB present the RHS with nodular walls and small nodules inside the ground-glass component of the RHS. Histopathologic analysis has revealed the presence of granulomas within the ring portion of the RHS and/or inside the RHS.[29,30] Organizing pneumonia is the most frequent cause of the RHS, but it was later observed in several other infectious and noninfectious diseases. A wide spectrum of diseases can manifest with RHS, including infectious conditions: paracoccidioidomycosis, TB, zygomycosis, and aspergillosis, and non-infectious conditions: Wegener's granulomatosis, lymphomatoid granulomatosis or bronchioloalveolar carcinoma.[31] The morphological aspect of the ring and ground-glass component, particularly by the presence of small nodules, usually indicates active TB or sarcoidosis, rather than organizing pneumonia.[29] The association
with parenchymal abnormalities such as consolidation, multifocal ground-glass opacities, or linear opacities would favor a TB diagnosis. [30]

**Fig. 8:** Something borrowed - Reversed-halo sign subpleural in the left upper lobe

**References:** Radiology Department, Guy's and St Thomas' Hospital, London, UK

The "**sarcoid galaxy sign**" (SGS) is a large parenchymal nodule arising from coalescent small nodules, and surrounded by many tiny satellite nodules. The "**sarcoid cluster sign**" (SCS) is also characterized by clusters of multiple small nodules in the pulmonary parenchyma but, in contrast to the SGS, the nodules do not tend to coalesce. These two signs were initially described in sarcoidosis, but were subsequently also identified in TB.[31]
SGS as a tomographic aspect in sarcoidosis corresponding to large parenchymal nodules arising from a coalescence of small nodules. At the periphery of the large nodules, each constituent small nodule had a relatively distinct margin. Pathologically, the nodules represent numerous coalescent granulomas, which were more concentrated toward the center of the sarcoid galaxy, than at the periphery. Heo et al. described a similar aspect in active TB, and suggested the term "clusters of small nodules" instead of SGS as a better description of the pathological morphology.[31,32] Radiologically, tuberculous clusters of small nodules seem to be postprimary due to their location mainly in the upper lobe and the superior segment of the lower lobe and lack of associated lymph node enlargement (table 2).[32]

Table 2. Differential diagnosis between PTB and sarcoidosis in the presence of SGS

<table>
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<tr>
<th>PTB</th>
<th>Sarcoidosis</th>
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<td>single cluster of small nodules</td>
<td>multiple clusters of small nodules</td>
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upper lobes and the superior segment of the lower lobes; usually in the upper and middle lobes; rare in the lower lobes
associated with tree-in-bud associated with lymphadenopathy

Ortega et al. described the SCS as a new HRCT sign of sarcoidosis.[33] It represents rounded/long clusters of multiple small nodules in the pulmonary parenchyma that are close to each other but not coalescent. Pathologically, these tiny nodules correspond to noncaseating granulomas. Marchiori et al. described a similar aspect in a patient with proven PTB. Unlike the case by Ortega et al., this patient did not show perilymphatic nodules or lymph node enlargement.[34]

Something blue - patterns associated with positivity of AFB smears

Determination of diagnosis and activity of PTB is usually based on the detection of acid-fast bacilli (AFB) in sputum smears or culture.[35] The concentration of infectious organisms is an important factor in the transmission of PTB, and the frequency of transmission from patients with presence of AFB on Ziehl-Neelsen stain is 22% higher than that from patients with negative smear.[36] The bacterial count in the stained sputum might indicate the degree of infectiousness.[13]

There is a significant correlation between radiologic extent of disease and the degree of smear positivity. Different HRCT findings such as GGO, consolidation, cavitation, nodules and bronchial lesions are significantly associated with smear-positive PTB.[8]

Three independent factors are predictive of smear-positive sputum results: cavitation, nodule clusters without relation to the upper or lower lung fields and consolidation over the upper lung fields. Furthermore, the clusters of nodules present the highest odds ratio for smear-positive after multivariate analysis.[36] The more the size of a nodule, the more related it is to smear positivity.[8] The clusters of nodules representing peribronchovascular nodules are significant findings in patients with positive AFB sputum smear.[37] One study found that as the number of AFB in sputum smears increased, the number of lobes involving micronodules and nodules also increased.[38]
Fig. 10: Something blue - Tree-in-bud pattern in the right lung (A) associated with positive sputum smear (B)

References: "Leon Daniello" Pulmonology and Tuberculosis Care Clinics, Cluj-Napoca, Ro

Consolidations involving multiple segments/lobes are likely to have positive AFB-smear results.[36] Ors et al. found a significant difference between smear-positive and smear-negative patients for consolidation score. The same authors also found a significant correlation between GGO score and the degree of smear positivity.[8]

Another study showed a statistically significant relationship between mycobacterial load and cavitary volume. Larger cavities, determined by maximum lumen diameter, were associated with a more positive smear.[39] Also, patients with 4-6 cavities had a higher mycobacterial load than those with fewer overall cavities. Other significant correlations were found between the degree of smear positivity and the thickness of cavity wall and between the degree of smear positivity and distance of cavity from nearest airway.[8] The latter is an expected finding as the opening and discharging of central cavities into airways is easier than that of peripheral cavities. This aspect may explain smear negativity in some patients with peripheral cavities on HRCT.[8] Cavitation is also associated with a prolonged time to smear conversion from positive to negative after 2 months of treatment.[39]

The frequency of centrilobular opacity is not associated with positive sputum smear results.[36] This may be explained either by the smaller total affected area compared to, for example, air space consolidation, leading to a lower amount of AFB-rich exudation and necrotic material, and by the longer distance from the affected area to the central airway in centrilobular nodules than in air space consolidation and cavitation.[40]

Matsuoka et al. also found consolidation in less than two lobes, and cavitation only in a single pulmonary lobe, if at all, in smear-negative patients.[38]
Images for this section:

![Image](image_url)

**Fig. 1:** Something old - Right paratracheal and mediastinal adenopathy associated with right middle lobe consolidation in a 6 year old patient

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Conclusion

The extremely heterogeneous spectrum of radiologic manifestations of PTB can pose a variety of radiological presentations and diagnostic challenges.
References