Primary and reactivation pulmonary tuberculosis: Up-to date imaging an management

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Learning objectives

• To review findings of pulmonary TB in the chest Xray and CT, in immunocompetent and immunocompromised patients.

• To assess the role of chest Xray, CT and PET/CT in the diagnosis of pulmonary TB.

• To describe the radiological characteristics of the most relevant complications and sequelae, including multiresistant TB and immune reconstitution inflammatory syndrome.
Background

Nowadays the tuberculosis (TB) continuous being an important problem of global health. Despite the slight decrease in incidence (2), the high prevalence of TB in immunocompetent and immunocompromised individuals makes tuberculosis a issue of concern worldwide.

In 2014, 9.6 million people fell ill with TB and 1.5 million died from the disease, and in 2015, 1 in 3 HIV deaths was due to TB. The largest number of new TB cases occurred in South-East Asia and Western Pacific Regions. However, Africa continues to have the largest number of patients with the disease, with 281 cases per 100000 population in 2014 (2).

About one-third of the world's population has latent TB, which means people have been infected by TB bacteria but are not yet ill and cannot transmit the disease (2). A person with active but untreated TB infects approximately 10-15 other people per year (3). Without proper treatment, 45% of HIV-negative people with TB on average and nearly all HIV-positive people with TB will die (2). It is for this reason that the diagnosis and early management are crucial for decreasing mortality and morbidity.

The clinical presentation of TB is insidious and can be difficult to diagnose. The symptoms of active TB disease are cough, fever, night sweats, weight loss etc., but they can be mild for many months.

Mycobacterium tuberculosis is a gram-negative non-spore forming strictly aerobic bacillus with a high lipid content to its cell wall. As a result, the growth rate of TB bacillus is dependent on oxygen availability and the cell wall renders the bacillus resistant to traditional antibiotics; the cell wall is also resistant to acids and alkali.(4)

TB is acquired by the inhalation of airborne droplet organisms transmitted from an infected host to a susceptible individual. The clinical and radiological manifestations of the disease depend upon the patients' immune response (3) and whether it is their first exposure to the tubercle bacillus.(4)

Pulmonary tuberculosis was classically divided into primary and postprimary tuberculosis. Primary pulmonary tuberculosis typically manifests radiologically as parenchymal disease, lymphadenopathy, pleural effusion, miliary disease, and atelectasis. In postprimary the finding is the development of patchy of ill-defined consolidation and cavities.
Thoracic tuberculosis has a series of patterns of presentation and healing, depending on the immunologic status. Understanding of the radiologic manifestations acute, reactivation, sequelae and complications of tuberculosis are important to facilitate a correct diagnosis and management.

Several complications are associated, such as hematogenous dissemination or extension to the pleura.

The interpretation of the radiologic manifestations of TB is difficult and sometimes it's possible that the disease expresses characteristics of other infections or mimics cancer (4).

Traditionally chest Xray has been the most frequently imaging tool used for the diagnosis and monitoring of TB; however, currently the CT has a more important role because it is much more sensitive than chest radiography, especially in the detection of TB in patients in whom the chest radiograph is normal or inconclusive in the determination of disease activity, complications, and in the management of TB by providing a roadmap for surgical treatment planning.

The role of PET-CT in the case of this disease is limited, although it has been postulated as a useful tool for the differential diagnosis of malignant causes tuberculosis infection.

Pathogeny:

TB is spread from person to person through the air mainly through coughing. The likelihood of transmission of infection depends on the number of bacilli expelled by coughing, the duration of exposure, and the virulence of M. tuberculosis.

The bacterium enters the airway host, it reaches the alveoli where it is phagocytosed by alveolar macrophages, then they interact with lymphocytes T and they are differentiate into epithelioid histiocytes, and together are grouped in small groups forming what is the granuloma, whose most common initial deployment is found in the middle and lower lung zones (4).

The aim of host immunity is the elimination of the bacteria in the body, but usually this is only inactivated, resulting in a latent infection.

In approximately 5% of the infected population, reactivation of latent infection many years after initial contact is developed. It usually presents with involvement in the apical and posterior segments of the upper lobes and the superiors of the lower lobes. This is
probably due to the higher oxygen tension and degree of lymphatic drainage alteration in these areas.
Findings and procedure details

According to the classic teaching, upper lobe cavitary disease was believed to be the hallmark of reactivation of latent infection, whereas lower lung zone disease, adenopathy, and effusions were believed to indicate recent infection (5).

Traditionally, it was believed that the clinical, pathologic, and radiologic manifestations of reactivation TB were quite distinct from those of primary TB. This concept has been challenged on the basis of DNA fingerprinting and the analysis about restriction fragment length polymorphism (RFLP) of M. tuberculosis. Isolates from patients infected with epidemiologically unrelated strains of TB have different RFLP patterns, whereas those from patients with epidemiologically linked strains generally have identical RFLP patterns (3).

The time from acquisition of infection to the development of clinical disease does not reliably predict the radiographic appearance of TB. The only independent predictor of radiographic appearance may be integrity of the host immune response; namely, severely immunocompromised patients show a tendency to have the primary form of TB, whereas immunocompetent patients tend to have the reactivation form (3).

Primary TB:

This type of radiological pattern is more common in children and immunocompromised patients, mainly watching lymphadenopathy, which are usually unilateral, hilar or mediastinal and generally they are ipsilateral to parenchymal abnormalities (4), and more commonly involves the right side. In the active stage, the nodes have central low attenuation and peripheral rim enhancement at CT, which correspond to caseation or liquefaction necrosis and and granulation tissue with inflammatory hypervascularity (1). This pattern of lymphadenopathy is very characteristic of TB in the appropriate clinical setting, but we must remember that there are some other inflammatory entities so as neoformativas, such as lymphoma, metastasis and other infections (6), which may have the same appearance (Fig.1). Obstructive atelectasis due to bronchial compression by lymphadenopathy is seen mainly in children and it can also occur in adults due to endoluminal bronchial occupation, being able to simulate a neoplastic disease (2).

Usually, the parenchymal involvement shown as a consolidation area, usually unilateral and homogeneous. You can locate in any area of the lung being more frequent in the right hemithorax.
To a lesser extent we found pleural effusion, which occurs unilaterally and ipsilateral to the primary or sole focus involvement. TB should always be considered in the presence of a unilateral effusion and TB pleural disease may cause pleural thickening extensive enough to mimic mesothelioma (4) (Fig.2).

**Postprimary TB:**

This radiological pattern occurs mainly as patchy, poorly defined segmental consolidation. These pulmonary abnormalities are mainly located in the apical and posterior segments of the upper lobes, and less frequently in the superior segment of the lower lobe (6) and in most cases with involvement of multiple segments simultaneously.

Cavities may be seen, they can be single or multiple, thick or thin walled, and up to 25% contain an air-fluid level. When possible the comparison with previous imaging, may reveal thickening of the wall of a pre-existing cavity or cavitation developing within consolidation or a nodule, these are findings suggestive of active disease (Fig.3). It is usually seen in an infected immunocompetent host.

The presence of small nodules in the region of the cavity suggests endobronchial spread, and it is best appreciated on thin section CT presenting as a tree in bud pattern (Fig.4). All these findings indicate active disease.

Thickening in the wall of a pre-existing cavity could also indicate colonisation such as a developing aspergilloma (4).

In the TC we can find three types of small nodules associated with TB infection, tuberculomas, tree in bud pattern and miliary TB.

*Tuberculoma* may be a manifestation of either primary or postprimary tuberculosis. It is a round or oval granuloma caused by acid-fast bacilli with a wall lined by granulomatous inflammatory tissue or encapsulated by connective tissue (2) Tuberculomas can be solitary or multiple and range in diameter from 0.5 to 4.0 cm or greater. They are smooth or sharply defined and calcification is found in 20%-30% of tuberculomas and is usually nodular and diffuse (1) (Fig.5).

Because of its high metabolism can give false positives in the 18F-FDG PET and can be confused with a malignant lesion. The 11C-choline PET can help differentiate a malignant lesion.
'Tree in bud' describes the CT scan appearance of plugging of the distal airways and adjacent acini; the result is a branching pattern terminated by small nodules (4).

*Miliary TB*, due to the haematogenous spread of TB in the lungs, results in the widespread random distribution of active TB granulomata throughout the lung and it can occur in both primary and postprimary TB and is commonly seen in the elderly, in under 2-year-old children and in immunocompromised patients (4) (Fig.6).

The nodules are uniform in size, measuring from 1-4 mm, and are usually discernable on chest X-ray around 4 weeks following the onset of symptoms (4). When they are cured there is no residual scarring or calcification (6).

We can also find plural tuberculous effusion, which is typically loculated and size can be stable for years.

Pneumothorax secondary to tuberculous infection is seen in approximately 5% of patients with post-primary TB and occurs following liquefaction of caseous pleural disease, resulting in pleural necrosis and rupture (4).

**Role of different imaging tests in TB:**

Chest radiographs play a major role in the screening, diagnosis, and response to treatment of patients with TB. However, the radiographs may be normal or show only mild or nonspecific findings in patients with active disease (3).

With CT, the diagnosis of pulmonary TB is correct in 91% of patients and TB is correctly excluded in 76% of patients. CT is particularly helpful in the detection of small foci of cavitation in areas of confluent pneumonia and in areas of dense nodularity and scarring.

CT plays an important role in the detection of TB in patients in whom the chest radiograph is normal or inconclusive, in the determination of disease activity, in the detection of complication, and in the management of TB by providing a roadmap for surgical treatment planning (3).

PET scans using 18F-FDG or 11C-choline can sometimes help differentiate tuberculous granuloma from lung malignancy (3) (Fig 7).

The integration of CT and PET hybrid system in PET / CT can acquire in one session of anatomical and metabolic images, combining the benefits of both while
minimizing the limitations of each modality. It has demonstrated the contribution of 18F-FDG-PET-CT in the diagnosis of tuberculosis. Because leukocytes and activated macrophages metabolize many glucose for energy chemotaxis and phagocytosis, and the use fibroblasts for proliferation (15). These cells therefore also show 18FDG avidity, resulting in increased uptake at sites of active infection or inflammation.

The 18-FDG PET / CT may be useful in differentiating the aftermath of the latent form of tuberculosis or infection to active particularly in patients with isolated radiological findings (1).

The SUV max alone cannot predict with certainty the etiology, as there may be overlap in uptake between benign and malignant lesions.

The 11C-choline PET scans can help differentiate between lung cancer and tuberculoma. Because the uptake value of tuberculoma is low in 11C-choline PET scans (3).

Described as the decrease in 18F-FDG uptake is correlated with treatment response, 18 FDG-PET can help guide the monitoring and the duration of antimicrobial therapy, particularly in patients with multidrug-resistant TB (7).

The regression of imaging abnormalities following adequate treatment is often a slow process, and it is not unusual to witness an initial deterioration before improvement. The localised areas of consolidation resolve within a month; the TB cavities respond to treatment with enlargement but with thinning of the wall such that there is an overall reduction in the amount of abnormal soft tissue. On completion of treatment the cavity may persist or become contracted by fibrosis (4) (Fig.8).

The volume of lymphadenopathy is a poor indicator of response to treatment as nodes often enlarge initially and may take a long time to show signs of resolution. The resolution of a tuberculous pleural effusion is readily identified on imaging and to be expected following effective therapy; persistence or increase in size of an effusion suggests either resistance to therapy or that there is an alternative cause for the effusion.

Complete resolution is rare, and residual areas of scarring, focal nodularity, and volume loss are the more commonly seen end points of treatment(4)

**TB in immunosuppressed patients and HIV:**

The deterioration of host immunity has been considered as a predisposing factor for tuberculosis. Some of the risk factors for the development of active tuberculosis
are defects in cell-mediated immunity, such as HIV infection, malnutrition, drugs and alcohol, end-stage renal disease, diabetes mellitus, and intake of corticosteroids or other immunosuppressive therapy. People who are infected with HIV are 20 to 30 times more likely to develop active TB (2). Immunocompromised patients are clearly at increased risk of TB and tend to display radiological features more typically associated with infection in the TB naïve host. (4)

It has been observed that the viral load during the course of tuberculosis decreases with proper treatment. In addition, HIV patients with TB have a shorter survival and faster than those without tuberculosis develop AIDS (8).

Unusual or atypical manifestations of pulmonary tuberculosis are common in patients with impaired immunity. The radiographic manifestations of pulmonary TB associated with HIV are dependent on the level of immunosuppression. A miliary pattern and pleural effusions is also more commonly seen (4) (Fig.9 and 10).

Antiretroviral therapy is successful in suppressing HIV replication and cellular immunity improvement, these immunological changes correlate with the reduction in the frequency of opportunistic infections and prolongs survival (9).

However, a subgroup of patients experience clinical deterioration due to the rapid restoration of the specific immune response during treatment, the resulting condition is called immune reconstitution inflammatory syndrome (IRIS) (8). The etiology is unknown, it is thought to be related to the reconstitution of immunity, leading to abnormal to any specific infectious or non-infectious antigens immune response with symptoms and clinical signs consistent with an inflammatory process.

IRIS-TB frequently occurs within the first two months. The patient presents worsening or onset of new clinical or radiological symptoms. The most common manifestations include return of symptoms such as fever, enlarged lymph nodes, appearance of infiltrates or pleural effusion (8). Most patients with IRIS have a course of self-limiting disease. The mortality associated with this is relatively uncommon; however, high morbidity is considerable.

**Multidrug-resistant TB (MDR-TB):**

TB is a treatable and curable disease. Active, drug-susceptible TB disease is treated with a standard 6 month course of 4 antimicrobial. (2) But it occasionally develops resistance to the commonly given treatment. It is a form of TB caused by bacteria that does not
respond to isoniazid and rifampicin, the first-line anti-TB drugs. The causes of MDR-TB are the inappropriate treatment or use of poor quality medicines (2).

MDR-TB is treatable and curable by using second-line drugs. However second-line treatment options are limited and recommended medicines may not be always available. The chemotherapy requires up to 2 years of treatment. This is more costly and can produce severe adverse drug reactions in patients.

In some cases, more severe drug resistance can develop. Extensively drug-resistant TB, XDR-TB, is a form of multi-drug resistant tuberculosis which is resistant to the two above mentioned medicines, the fluoroquinolone and at least one of three injectable second-line drugs (capreomycin, kanamycin and amikacin).

Imaging findings of MDR TB can have multiple cavities and findings of chronicity, such as bronchiectasis and calcified granulomas (3). In adults, failure of improvement in the extent of lung parenchymal involvement following 3 months of therapy may suggest drug resistance or a co-existent pathology (3).

Complications and sequelae:

Fibrocavitary disease and bronchiectasis:

The most significant pulmonary parenchymal sequelae, both render the lung susceptible to further infection.(4) The fibrotic response, which manifests as atelectasis of the upper lobe, retraction of the hilum, compensatory lower lobe hyperinflation, and mediastinal shift toward the fibrotic lung (Fig.11). A nonspecific radiologic pattern of fibrosis consisting of parenchymal bands, fibrotic nodules and cavities, or traction bronchiectasis is occasionally encountered (1).

Bronchiectasis may develop as a result of tuberculous involvement of the bronchial wall and subsequent fibrosis. Bronchiectasis is seen in 71%-86% of patients with inactive disease. Bronchiectasis in postprimary TB can be a result of cicatricial bronchostenosis after local infection. It more commonly occurs by destruction and fibrosis of the lung parenchyma with secondary bronchial dilatation (traction bronchiectasis) (1) (Fig 12).

Aspergilloma:

Chronic cavities in the pulmonary parenchyma can be colonized by Aspergillus. It is usually located within a cavity or ectatic bronchus and consists of masses of fungal hyphae with mucus and cellular debris. This mass is surrounded by a crescentic air
shadow is noted inside a lung cavity (air-crescent sign). CT demonstrates a mobile fungus ball (1). These may exist for years without symptoms. Hemoptysis is the most common clinical complication, with a prevalence of 50%-90% (Fig 13).

**Pneumothorax:**

It may occur in 5% of patients with post primary TB, which usually have severe cavitary disease. It can also be seen as the first manifestation of the disease. The pathogenesis involves pleural caseous infiltrates that undergo liquefaction, resulting in pleural necrosis and rupture (1).

**Bronchogenic carcinoma:**

TB can favor the development of cancer and it can also make its injuries are misinterpreted as disease progression (1) (Fig. 14). The histopathologic diagnoses in reported cases have been malignant lymphoma, squamous cell carcinoma, mesothelioma, malignant fibrous histiocytoma, liposarcoma, rhabdomyosarcoma, angiosarcoma, and hemangioendothelioma, in order of frequency. The pathogenesis of malignancy developing a long-standing severe inflammatory process or chronic stimulation of mesothelial cells (1) (Fig. 15 and 16).

**Bronchial Stenosis:**

The most common cause of inflammatory stricture of the bronchus is TB. It is seen as a uniform bronchial wall thickening with luminal narrowing. In active disease, the airways are irregularly narrowed in their lumina 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 (3) (Fig. 17).

**Broncholithiasis:**

It is an uncommon complication of pulmonary TB. Calcified and necrotic material found in lymph nodes, affected by TB, can erode the bronchial wall, pass airway and it can produce obstruction and atelectasis (1) (Fig.18).

**Fibrosing mediastinitis:**

It is uncommon and it is seen as a fibrotic reaction extensive that can compress mediastinal mediastinal structures occurs. It can be seen as a widened mediastinum or as a localized mass with calcifications. The symptoms that occur are due to compression of
mediastinal structures such as the superior vena cava, esophagus, and tracheobronchial tree (1) (Fig. 19).

**Empyema necessitatis:**

Pleural infection is usually caused by rupture of a subpleural caseous focus into the pleural space; less commonly, it is caused by hematogenous dissemination and contamination by adjacent infected lymph nodes (1).

It is a rare complication and is seen as an inflammatory mass that communicates with the pleural cavity, soft tissue and sometimes skin. When spontaneous discharge of empyema through the parietal pleura into the chest wall forms a subcutaneous abscess, it is termed empyema necessitatis (1) (Fig.20).

**Aneurysm Rasmussen:**

It is caused by weakening of the wall of the pulmonary artery due to contact with an adjacent tuberculous cavitation (6).

It is also possible to find that pulmonary arteries and veins in an area of active tuberculous infection may demonstrate vasculitis and thrombosis (1) (Fig.21).
Fig. 1: a) Chest X-ray shows increased density right paratracheal (arrow) b) CT shows right paratracheal and left supraclavicular lymphadenopathy with peripheral contrast enhancement and hypodense center (arrow).

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Fig. 2: Male 36 years with a fever and without known medical history. a) Chest Xray shows right pleural effusion. b) CT shows right pleural thickening with extensive pleural effusion (arrow). The patient is placed a chest tube which presents positive for TB culture. Correct outcome after medical and surgical treatment.

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**Fig. 3:** a) - b) On chest Xray and CT are identified multiple pulmonary cavitations of different sizes in right chest, predominantly they are in upper segments and some them have air-fluid levels.

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Fig. 4: Male 47 years with weakness, pain chest and coughing up of 18 months duration. Dyspnoea in recent weeks. a) Chest Xray shows caverns in both vertices and diffuse nodular lung involvement. b) TC shows cavitations in both upper lobes and centrilobular bilateral involvement compatible with bronchogenic dissemination. Positive sputum for TB.

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**Fig. 5:** Female 34 years with cough and hemoptysis. a) CT shows a pulmonary mass in the right upper lobe with centrilobular affectation adjacent to the lesion (arrow). b) PET-CT performed within two weeks, shows enlargement and cavitation of the lesion, identifying hypermetabolism on the walls and metabolic center, this translates necrosis. Good clinical progress with TB treatment.

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**Fig. 6:** a) - b) CT shows nodules of up to 3 mm in diameter, they are distributed randomly in both lungs. c) Disappearance of parenchymal involvement 6 months after starting TB treatment.

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Fig. 7: Female 21 years old with chronic cough without other symptoms. a) Chest Xray evidences right mediastinal mass. b) PET-CT showed hypermetabolic mediastinal mass. The case is oriented as possible lymphoma. Mediastinoscopy with biopsy is performed, which was positive for TB. c) Chest Xray after TB treatment shows lesion resolution.

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**Fig. 8:** Male 28 years with hemoptysis. a) Chest X Ray shows pulmonary cavity in the right upper lobe and lingula condensation b) CT also shows centrilobular affectation pericaverna and other condensation in the right upper lobe with adjacent areas of ground glass that could translate in relation to residues hematological (arrow). c) TC post treatment. Good performance, leaving only residual scar lesions and some calcifications in the right apex.

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**Fig. 9:** Male 48 year old, diagnosed of HIV in 2000 and without medical check ups for 2 years. Presents with fever, cough, weight loss of 8 kg from 1 month ago and profuse night sweats. Sputum sample shows abundant acid-fast bacilli. a) CT with MIP reconstruction shows multiple millimetric nodules randomly distributed by the lung, they are suggestive of miliary TB. b) CT also evidence mediastinal lymph nodes with central necrosis (arrow).

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Fig. 10: a) - b) Patient HIV with positive sputum sample for TB, who debuts with condensation LII (arrow) and hydropneumothorax.

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**Fig. 11:** a)- b) Chest Xray and CT show pulmonary destructive pattern in the left lung with ipsilateral volume loss and calcified pleural thickening.

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**Fig. 12**: Male 57 years with syndrome constitutional of 4 months evolution. a) Chest Xray shows right hydropneumothorax. b) - c) CT shows communication between bronchus and pleural cavity. The findings correspond to a bronchopleural fistula.

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**Fig. 13:** Female 36 years with cough and hemoptysis. a) - b) Chest Xray and CT show a cavitary lesion in the right lower lobe with dense content on your inner suggestive of fungal colonization of preexisting tuberculous cavity (aspergilloma).

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**Fig. 14:** Male 71 years old with a history of TB in youth and ex-smoker since 2 years, who presents progressive chest pain of 4 months of evolution. a) Chest Xray shows right apical opacity (arrow) b) CT shows poorly defined increased density in the right upper lobe with destruction of adjacent ribs (arrow). The result of the biopsy reported adenocarcinoma.

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**Fig. 15:** Female 32 years constitutional syndrome. a) Chest Xray shows right paramediastinal increased density (arrow). b) - c) PET-CT shows hypermetabolic mass paramediastinal right, together with pulmonary lesions, some of them cavitated (arrow). Taking into account the findings, we can do a differential diagnosis between TB and lymphoma. Mediastinoscopy with biopsy confirms the diagnosis of Hodgkin lymphoma, which presents good clinical evolution to chemotherapy. This case was initially a false positive of TB.

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**Fig. 16:** Male 75 year old with history of therapeutic pneumothorax in youth by TB and who presents apparition of mass in the right chest wall of 3 months evolution. a) Chest Xray shows increased density in the right base with involvement of the rib (arrow) b) - c) CT and MRI show right pleural collection with a soft tissue mass and destruction of adjacent rib.(arrow) . The result of the lesion biopsy was lymphoma.

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**Fig. 17:** Female 40 years old and HIV-positive with dyspnea and progressive stridor and positive sputum sample for TB a) CT shows thickening of the walls of the trachea which conditions stenosis.

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Fig. 18: a) CT shows endobronchial right lithiastic (arrow) corresponding to broncholithiasis. b) - c) Correlation with bronchoscopy.

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Fig. 19: Female 73 years old with history of TB in youth, who presents symptoms consistent with superior vena cava syndrome. a) CT shows multiple calcified lymph nodes in the mediastinum (arrow) and b) venous collaterals (arrow).

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Fig. 20: Male 81 years old with history of therapeutic pneumothorax because TB in youth, who presents purulent wound in chest wall of 6 weeks of evolution. Hard and painful tumor consistency feels right chest wall. a) Chest Xray shows right pleural effusion with increased adjacent soft tissue. b) CT shows right calcified pleural thickening and pleural collection that extends into the adjacent soft tissue.

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**Fig. 21:** Male 51 years old with diagnosis and treatment of tuberculosis in 2004. Patient presents multiple episodes of hemoptysis. a) Chest X-ray shows cavitation and scarring lesions in the left upper lobe b) Angiography shows an aneurysm of a branch of left superior pulmonary artery, which is embolized. Findings regarding Rasmussen aneurysm.

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Conclusion

- Thoracic tuberculosis has a series of patterns of presentation and resolution depending on the immune status of the host. Understanding the radiological manifestations of primary and postprimary tuberculosis, reactivation, and complications is important to help diagnose and correct management.
- In people with normal immune response, radiologic findings are of post-primary TB and in immunosuppressed patients are of primary TB.
- Unusual or atypical manifestations of pulmonary tuberculosis are common in patients with impaired immunity.
- CT has an important role in the detection and characterization of radiological signs of TB because it is much more sensitive than chest radiography, as well as being important as a roadmap for managing complicated TB and MDR TB.
- The role of PET-CT is postulated as a useful tool for the differential diagnosis of malignant causes tuberculosis infection.
References


