Pulmonary tuberculosis a new look at an old disease: atypical manifestations and uncommon complications.

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Learning objectives

- To show the radiological findings (chest plain film and CT) of atypical and uncommon complications of pulmonary tuberculosis.
- To illustrate the most useful features to reach the correct diagnosis.
- To offer solutions to the common clinical dilemmas in everyday practice.

Background

Pulmonary tuberculosis (TB) is a common infection worldwide and a medical and social problem causing high mortality and morbidity, especially in developing countries. Although there is an effective treatment, TB is still prevalent and far from being eradicated. The prevalence is highest in Southeast Asia and Africa and much lower in developed countries. In Spain, 6046 new cases of TB were reported in 2012 (incidence: 13.10/100,000); 70% were located in the chest.

Some atypical manifestations can make the correct diagnosis difficult. Failure to recognize uncommon complications can lead to confusion. Sequelae and complications of tuberculosis are now rare if treatment is instituted early. Here we illustrate the atypical radiologic findings and complications, considering the following locations in the chest:

**Lung parenchyma:** tuberculoma, aspergilloma, cystic / bullous disease, ARDS, silicotuberculosis.

**Bronchial tree:** tracheobronchial stenosis, broncholithiasis, Lady Windermere syndrome.

**Mediastinum:** bronchoesophageal fistula, pericardial tuberculosis, fibrosing mediastinitis.

**Vascular:** pulmonary artery pseudoaneurysms (Rasmussen aneurysm).

**Pleural and chest wall:** concentric pleural thickening, *empyema necessitates*, bronchopleural fistula.
Findings and procedure details

PULMONARY LESIONS:

1. TUBERCULOMA. Fig. 1 on page 14

Nodular granulomatous lesions that manifest in 5% of patients in either primary or postprimary infection.

Radiological findings:

- Tuberculomas may be solitary or multiple (0.5 cm-4 cm or larger).
- Generally, they are nodular lesions with sharp borders.
- 80% have satellite lesions.
- Nodular or diffuse calcification is present in 20%-30% .
- Serial images can determine whether they are stable.

Diagnosis:

- Radiological findings must be interpreted together with clinical history and evolution.
- Fine-needle aspiration cytology can usually diagnose TB, but sometimes surgery is necessary to differentiate TB from tumors.

Treatment:

- Most tuberculomas become smaller after TB treatment.

2. ASPERGILLOMA. Fig. 2 on page 15, Fig. 3 on page 16, Fig. 4 on page 17, Fig. 5 on page 18, Fig. 6 on page 19.

It consists of a conglomerate of Aspergillus hyphae mixed with cellular debris and mucus that results from colonization of a pre-existing cavity (usually caused by TB); aspergillomas do not invade surrounding tissue.

Clinical manifestations:
• Sometimes asymptomatic, the most common clinical manifestation is hemoptysis (50%-90%).

**Radiological findings:**

**Chest X-ray:**

• **Air crescent sign:** a crescent-shaped air space of variable size separating a nodule/mass inside a cavity from its wall. Fig. 2 on page 15
• The thickening of the walls of a tuberculous cavity and/or adjacent pleura is an early sign that suggests the formation of an aspergilloma. Fig. 4 on page 17, Fig. 6 on page 19.

**CT:**

• Typically a mobile nodular lesion, generally inside a cavity (mature form) with the air crescent sign Fig. 2 on page 15, Fig. 3 on page 16 Sometimes calcified. Fig. 2 on page 15
• It may have different developmental stages. Fig. 5 on page 18.
• The immature forms present as fungal tracts attached to the cavity wall, intertwined with each other to form a network. These spongiform shapes can coalesce and lead to a mature fungus ball or remain unchanged. Fig. 2 on page 15, Fig. 3 on page 16, Fig. 5 on page 18, Fig. 6 on page 19.

**Diagnosis:**

• Clinical-radiological.

**Treatment:**

• Approximately 10% resolve spontaneously.
• Surgical treatment is rarely considered necessary.
• Percutaneous intracavitary instillation of amphotericin B or other antifungal drugs under CT guidance.
• Embolization of the systemic arteries in the palliative treatment of hemoptysis.

3. CYSTIC/BULLOUS LUNG DISEASE. Fig. 7 on page 20, Fig. 8 on page 21

This is one of the rarest complications of tuberculosis.

**Radiological findings:**
• Cystic lesions that grow and tend to coalesce; they can be complicated by pneumothorax and bronchopleural fistulas. Fig. 8 on page 21

**Pathogenesis:**

• Cysts occur in areas with active tuberculosis and consolidations, probably secondary to bronchiolar obstruction.

**Evolution:**

• Sometimes they are reversible, but sometimes they remain unchanged after treatment.

4. MILIARY TUBERCULOSIS AND ACUTE RESPIRATORY DISTRESS SYNDROME. Fig. 9 on page 22

Miliary tuberculosis is a life-threatening disease resulting from the hematogenous spread of *Mycobacterium tuberculosis*. It is also an unusual cause of acute respiratory distress syndrome (ARDS).

**Clinical features:**

• It is usually seen in the elderly, infants, and immunocompromised persons, manifesting within 6 months of initial exposure.

**Radiological findings:**

**Chest X-ray**

• Usually normal at the onset of symptoms; hyperinflation may be the earliest feature.
• Diffuse 2-to-3-mm nodules, with a slight lower lobe predominance.
• Diffuse or localized ground glass.

**High-resolution CT**

• Classical characteristics of miliary TB: innumerable, 1-to 3-mm nodules randomly distributed throughout both lungs, thickening of interlobular septa, and fine intralobular networks.
• Classical features of ARDS: diffuse or localized ground glass.

**Evolution:**
• High mortality (60%) for ARDS caused by miliary TB, higher than ARDS from other diseases.

**Treatment:**

• No specific treatment: corticoids have been used without good results. Thus, early suspicion and prompt diagnostic work-up are essential.

5. **SILICOTUBERCULOSIS.** Fig. 10 on page 23

Silicosis, the most prevalent of the pneumoconioses, is a diffuse interstitial lung disease caused by inhalation of crystalline silica; it is the most common occupational disease involving the lungs.

Patients with silicosis have 2.8 to 39 times greater risk of developing pulmonary TB than healthy individuals.

**Clinical features:**

• Silicosis can present in three different forms: acute, accelerated, or chronic. Hemoptysis is common in acute and chronic cavitory pulmonary TB.

**Chest X-ray:**

• Multiple, diffusely distributed nodules < 10 mm in diameter, predominantly in the superior and posterior regions of the lungs. The nodules can coalesce and form opacities > 10 mm in diameter, which is indicative of progressive massive fibrosis. Hilar and mediastinal lymphadenopathies are common; these often calcify and sometimes show an "eggshell" pattern, highly suggestive of the diagnosis. These alterations are present in the accelerated and chronic forms.

• Silicotuberculosis: thick-walled cavities and consolidations. The presence of cavitation in a conglomerate mass is a possible indication of associated TB.

**CT**

• Active TB superimposed on silicosis manifests as thick-walled cavities, consolidations, tree-in-bud pattern, nodular image asymmetry, and rapid disease progression.

**Diagnosis:**
The diagnosis of silicosis is based on a history of exposure to silica accompanied by clinical and radiological findings. Routine chest X-ray is usually sufficient.

**Evolution/treatment:**

- It is extremely important to exclude the coexistence of active TB. However, the diagnosis of active TB superimposed on silicosis can be very difficult, particularly in initial profiles, when the clinical manifestations can be benign and the radiological alterations can be indistinguishable from those resulting from the preexisting silicosis.

**BRONCHIAL LESIONS**

1. **TRACHEOBRONCHIAL STENOSIS.** Fig. 11 on page 24

Infrequent (2%-4%). Secondary to granulomatous changes in the tracheobronchial wall or extrinsic involvement. Usually affects the left main bronchus.

**Radiological findings:**

**CT:**

- Concentric thickening with irregular narrowing of the bronchial lumen and wall enhancement of a long bronchial segment.
- Persistent collapse of a segment or lobe, which can cause distal bronchiecstasis.
- Secondary pulmonary hyperinflation.
- Mediastinal lymphadenopathy.
- It must be differentiated from bronchogenic carcinoma. Involvement of long segments and of several bronchi, circumferential narrowing, and absence of endobronchial mass favor the diagnosis of inflammatory stenosis.

**Treatment:**

- Early diagnosis and treatment prevent bronchostenosis.
2. BRONCHOLITHIASIS. Fig. 12 on page 25, Fig. 13 on page 26

It is defined as calcified material within the bronchial lumen. Calcium and lymph node inflammation or a granuloma erodes the bronchial wall and can fall inside. It is more frequent in the middle lobe and anterior segments of the upper lobes.

Clinical manifestations:

- Nonspecific. Chronic cough, hemoptysis, recurrent pneumonia.

Radiological findings:

Chest X-Ray:

- A change in the position of a calcium focus on serial radiographs.
- Lobar or segmental atelectasis, mucus impaction or air trapping.

CT:

- Endobronchial or peribronchial calcification associated with distal bronchial obstruction (atelectasis, mucous impaction, obstructive pneumonitis, or bronchiectasis).

Treatment:

- Observation or removal by bronchoscopy or surgery if symptoms persist.

3. LADY WINDERMERE SYNDROME. Fig. 14 on page 27

Infection with nontuberculous bacteria (Mycobacterium avium complex) in immunocompetent patients, usually women over 50 years old without preexisting lung disease. Diagnosis is complicated because the microorganism is difficult to isolate and culture; radiologists should alert clinicians in the appropriate context.

Clinical manifestations:

- Nonspecific symptoms: cough, fatigue, fever, weight loss, hemoptysis, dyspnea.

Radiological findings:
Chest X-Ray:

- Opacities in the middle lobe and/or lingua.

CT:

- Centrilobular nodules, usually accompanied by tree-in-bud opacities and bronchiectasis affecting the middle lobe and/or lingua.

Treatment:

- At least one triple treatment maintained at least 12 months after sputum cultures become negative.

MEDIASTINAL LESIONS

1. ESOPHAGOMEDIASTINAL FISTULA.

Esophagomediastinal fistulas are rare, even in tuberculous esophagitis. The most frequent cause is erosion of the esophageal wall through contiguous extension of a caseous mediastinal adenopathy.

Clinical manifestations:

- Symptoms may include fever, cough, weight loss, dysphagia, chest pain.

Radiological findings:

CT:

- Mediastinal collection/esophageal thickening.
- Pneumomediastinum. Lymphadenopathy.
- Most often subcarinal.

Treatment:

- TB treatment is usually sufficient.

2. PERICARDIAL TUBERCULOSIS. Fig. 15 on page 28, Fig. 16 on page 29
It is caused by retrograde lymphatic dissemination from mediastinal lymph nodes. Constrictive pericarditis occurs in about 10% of patients with tuberculous pericarditis. It is rare in our environment, but is the most common cause of pericarditis in Africa.

**Clinical manifestations:**
- Constrictive pericarditis: Signs of diastolic heart failure.

**Radiological findings:**

**CT:**

**Main finding:**
- Pericardial thickening with or without effusion, often associated with mediastinal lymphadenopathy.
- Pericardial calcification (Does not in itself indicate constriction).

**Findings secondary to cardiac constriction:**
- Dilation of the vena cava (superior and inferior). Contrast reflux to the inferior vena cava and to the azygos and hepatic veins.
- Deformity and compression of heart chambers.
- Leftward displacement of the interventricular septum.
- Signs of heart failure: interstitial edema (septal thickening), pleural effusion, liver findings secondary to stasis.

**Treatment:**
- Tuberculostatics. Pericardiocentesis if tamponade. Pericardiectomy in cases of constrictive pericarditis or recurrent cardiac tamponade.

3. FIBROSING MEDIASTINITIS (FM). Fig. 17 on page 30

Benign chronic inflammatory process characterized by the proliferation of fibrous tissue in the mediastinum. Focal FM is the most common in TB, with paratracheal, subcarinal, or hilar calcified masses. Focal FM might be due to an idiosyncratic fibroinflammatory reaction. Although TB can cause focal FM, focal FM is more commonly associated with histoplasmosis.

**Clinical manifestations:**
• Progresses insidiously with cough, fever, and symptoms due to compression of structures. It is the most common benign cause of obstruction of the superior vena cava.

Radiological findings:

CT:

• Calcified mediastinal or hilar mass obliterating planes and invading adjacent structures (vena cava, pulmonary artery, bronchi).
• Lung lesions secondary to bronchial or vascular involvement (atelectasis, infarcts...).

Diagnosis:

• Histological, but in the right clinical context, CT findings including a calcified mediastinal mass may be sufficient.

Treatment:

• Ketoconazole therapy and tuberculous drugs may stabilize the disease in cases with active granulomas. Interventional treatment of the stenotic structures (stents, prosthesis).

VASCULAR LESIONS

1. RASMUSSEN’S ANEURYSM. **Fig. 18** on page 31

This pseudoaneurysm occurs in areas of active TB when granulation tissue weakens and erodes the wall of a pulmonary artery. Often located in the walls of tuberculous cavities; found in 5% of autopsies of patients with a history of chronic cavitary TB.

Clinical manifestations:

• The main symptom is hemoptysis, which can be massive and life-threatening.

Radiological findings:

Chest X-ray:
Intracavitary protrusion, the replacement of a cavity by a nodule, or a rapidly growing mass inside a cavity can suggest pseudoaneurysm formation.

**CT angiography:**

- Rounded images that enhance with contrast material (pseudoaneurysm) in the same phase as the pulmonary arteries; generally located in the periphery of tuberculous cavities.

**Treatment:**

- Embolization.

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**PLEURAL AND CHEST WALL LESIONS**

1. **CHRONIC TUBERCULOUS EMPYEMA.** Fig. 19 on page 32

Pleural infection is usually caused by the rupture of a caseous subpleural focus and less often by hematogenous spread. If untreated, pleural involvement can encapsulate and form a chronic collection with thickening and calcification of the pleural layers; the material inside the capsulated lesion usually includes latent TB bacilli (difficult to demonstrate in cultures). Occasionally, complications such as fistulas to neighboring structures occur.

**Radiological findings:**

**Chest X-ray:**

- Calcified encapsulated pleural collection.

**CT:**

- Encapsulated pleural collection, with pleural thickening and/or calcification.

**Diagnosis:**

- Radiological.

**Treatment:**
• If there are no complications, no treatment is usually necessary. If there are complications, pleural drainage, although more aggressive treatment like pleurotomy or thoracotomy may be necessary in some cases.

2. BRONCHOLEURAL FISTULA. Fig. 19 on page 32

Usually occur in the active phase (rupture of a cavity in the pleura) or more commonly as a consequence of sequela after infection (generally secondary to fibrothorax or chronic pleural empyema).

Clinical manifestations:

• Increased sputum production, cough.

Radiological findings:

Chest X-ray:

• Air-fluid level in a pleural collection.

CT:

• Air-fluid level in a pleural collection.
• Sometimes the communication between the pleural space and the airway can be demonstrated in thin slices.

Treatment:

• Some fistulas (usually very small caliber) can cicatrize spontaneously.
• Persistent fistulas require drainage of the pleural cavity and/or surgery.

3. EMPYEMA NECESSITATIS. Fig. 20 on page 33

This is a chronic tuberculous empyema that extends beyond the pleural space to form a collection in the chest wall; it can cause bone destruction.

Clinical manifestations:

• A mass in the chest wall, with or without signs of inflammation.
Radiological findings:

CT

- Communication between the calcified chronic pleural collection and the collection in the chest wall (pathognomic).

ULTRASONOGRAPHY

- Useful for assessing pleural involvement and the association with the soft-tissue abscess.

Diagnosis:

- Clinical and radiological findings. The combination of a chest wall mass and a calcified chronic pleural empyema on the chest X-ray should alert radiologists to the diagnosis.
- Large proportion of false-negatives in aspiration and even surgical specimens.

Treatment:

- Abscesses require debridement and wide resection; occasionally, more surgical treatment is required.

Images for this section:
**Fig. 1:** A 32-year-old woman studied for a right apical opacity. (a) and (b). Noncontrast chest CT: (a) (mediastinal window) and (b) (lung window). Multiple clustered nodules in the upper right lobe (blue arrows), some of which are calcified (red arrows).
**Fig. 2:** Patient with a history of tuberculosis who presented hemoptysis. (a) Chest X-ray and (b) chest CT (coronal reconstruction, lung window): Post-TB changes in both upper lobes, with marked loss of volume and bilateral lung cavities (yellow arrow). Note the oval lesion (aspergilloma) surrounded by air (air crescent sign) in the cavity in the upper left lobe (red arrows); (c) and (d). Axial CT (c) (lung window) and (d) (mediastinal window): Large cavity in upper left lobe with aspergillomas inside (red arrows). Hypertrophy of the bronchial arteries (yellow arrows).
**Fig. 3:** (e) and (f). Coronal MIP reconstructions. (e) Hypertrophy of the bronchial arteries (red arrows); note the right bronchial artery arising from the descending portion of the aortic arch and an ectopic branch arising from the right vertebral artery. (f) Hypertrophy of the right intercostal arteries.
**Fig. 4:** Chest X-ray: (a). Cavitated lesion in the right upper lobe (arrows) with loss of lobar volume. (b). Thickening of the walls of the cavitated lesion, suggesting aspergilloma formation (blue arrows).
**Fig. 5:** Formation of an aspergilloma. Patient with a tuberculous cavity in the upper left lobe. CT (lung window) (a) Initial appearance of linear opacities in the cavity, corresponding to Aspergillus hyphae. (b) Progressive growth of the fungus until it occupies nearly the entire cavity. (c) Oval soft-tissue-density mass separated from the wall of the cavity by air (air crescent sign). (d) Finally, the mass separates from the walls of the cavity and moves when the patient changes position.
**Fig. 6:** Chest X-ray. Detail of the left upper lobe showing thickening of the costal pleura and of the walls of the cavity between (a) 23-03-2009 and (b) 28-9-2009 (arrows); (c) and (d). CT (lung window) images showing progressive thickening of the pleura and of the walls of the aspergilloma cavity (*).
Fig. 7: (a) and (b). 27-year-old woman with active tuberculosis (a) PA Chest X-ray shows extensive bilateral consolidations, predominantly in the left hemithorax, with volume loss. (b) Follow-up X-ray 10 months after starting treatment shows significant improvement in the opacities, with cystic images appearing in the medial and basal fields of the left hemithorax (red arrows).
Fig. 8: (c) and (d). Coronal and axial chest CT images show bilateral cystic structures with left predominance (red arrows) and ipsilateral pneumothorax (blue arrow) that required talc pleurodesis because of persistence due to a bronchopleural fistula.
**Fig. 9:** ARDS and Milliary tuberculosis. 86 year-old patient. (a). High-resolution CT image (1.0-mm section thickness) and (b) and (c) (MIP reformations) show randomly distributed small nodules and patchy ground-glass opacities in both lungs. (d) Chest radiograph performed five days after the CT shows worsening of the bilateral opacities, the patient died 2 days later.
Fig. 10: 40-year-old man. (a) Chest X-ray: Bilateral nodular pattern, predominantly in the upper fields, right apical lung opacity (arrow). (b) and (c) Chest CT (lung window) shows multiple well-defined nodules ranging in size from 1 mm to 5 mm and diffusely distributed, although with a clear upper lobe predominance, and a cavitated lesion with thickened walls in the right upper lobe (arrows). (d) Multiple large mediastinal lymph nodes, some with high density suggesting calcification (star).
**Fig. 11:** 56-year-old woman with persistent cough. (a) and (b). Initial CT showing nodular thickening of the trachea (arrows) and collapse of the lingua (asterisk) (b). Tuberculosis was not suspected in the initial CT; (c), (d) and (e). Follow-up CT 3 months later showed worsening of the bilateral tree-in-bud pattern (red arrows) and persistence of the involvement of the trachea, main left bronchus, and lingua (black arrows).
**Fig. 12:** Broncholithiasis. Chest CT (a) and (b). Calculus occluding a subsegmental bronchus in the left lower lobe (red arrow); mucous impactions are seen distally (black arrow).
Fig. 13: Broncholithiasis. (a) and (b). axial and sagittal chest CT images. Calcifications in the left anterior apical bronchus (red arrow) causing distal mucous impaction.
Fig. 14: 59-year-old woman with hemoptoic sputum. Chest CT (lung window) (a) and (b) showing bronchiectasis in the middle lobe and lingua, with bronchial impactions.
Fig. 15: 30-year-old Moroccan woman with active tuberculosis involving the pericardium. (a), (b) and (c). Chest CT (mediastinal window) shows diffuse adenopathic involvement (blue arrows) and nodular thickening of the pericardium (red arrows) causing constrictive pericarditis with compression of the heart chambers, especially the left ventricle. (d). CT (lung window) shows interstitial edema with septal thickening due to the difficulty of venous drainage e. Heterogeneous appearance of the liver suggestive of liver stasis.
**Fig. 16:** Asymptomatic 74-year-old patient with a history of tuberculosis in his youth. (a) and (b). Chest X-ray showing extensive pericardial calcification. (c) and (d). CT (mediastinal window) showing the extensive calcification, mainly surrounding the right ventricle and the base. The patient had no clinical or radiological signs of constriction.
**Fig. 17:** Focal mediastinal fibrosis secondary to tuberculosis in a 62-year-old man. (a). Unenhanced CT scan at the level of the right hilum shows a highly calcified mass that encases the right pulmonary artery (curved arrow) and involves the left lower lobe vein (straight arrow) and left atrium (not shown). (b). CT scan (lung window) shows multiple linear opacities perpendicular to the pleural surface (black arrowhead), enlarged septa (white arrowhead) due to systemic supply by collateral vessels (secondary to involvement of the right pulmonary artery), and enlarged septal veins (secondary to involvement of the left lower lobe vein). Note the striking contrast in appearance between the right and left lungs.
Fig. 18: Rasmussen aneurysm in a 42-year-old man with active postprimary tuberculosis and massive hemoptysis. (a) and (b) Contrast-enhanced CT scan at the level of the upper lobes shows, in the area of cavitation (blue arrow), a small rounded bilobed enhancing lesion (red arrow) that arises from a branch of the pulmonary artery. (c). Conventional angiogram shows contrast material filling two aneurysms (yellow arrow) in a segmental branch of the right upper lobe pulmonary artery. (d) Posteroanterior chest radiograph obtained after embolization shows coils (white arrow) in the wall of the tuberculous cavity.
Fig. 19: A 84 patient with tuberculosis in youth treated with thoracoplasty (a). Chest X-ray showing thoracoplasty changes with loss of volume and deformity of the left hemithorax. Calcified pleural thickening and fluid level (arrows) (b). CT with contrast (mediastinal window): Calcified pleural collection (chronic pleural empyema) with fluid level suggestive of bronchopleural fistula.
Fig. 20: A 90-year-old man presented with persistent left chest pain due to a chronic tuberculous pleural empyema. (a) Chest X-ray shows a large chronic pleural collection with calcified walls (black arrows). CT (mediastinal window) in the coronal (b) and axial (c) planes shows the collection in the chest wall (red arrows).
Conclusion

TB has a wide spectrum of manifestations on chest imaging. Knowledge about atypical manifestations and complications can help us reach an early diagnosis to enable appropriate treatment.

Personal information

References