Imaging features in pulmonary and extra-pulmonary tuberculosis

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Learning objectives

To illustrate the CT and MRI imaging findings in pulmonary and extra-pulmonary tuberculosis, with emphasis to nodal, genitourinary, adrenal, skeletal and nervous localizations.

Background

INTRODUCTION

Tuberculosis (TBC) is an airborne communicable disease caused by *Mycobacterium tuberculosis*, transmitted by patients with pulmonary TBC.

Infection occurs more commonly in adults (25-39 and 60-75 years old patients).

Population groups with increased risk: immunocompromised individuals (AIDS, lymphoma, leukemia), diabetics patients, children and the aged, alcoholics, the poor, immigrants from third-world countries, prisoners, nursing home residents, health care workers and the homeless.

Up until the mid 1980s, there was a steady decline in the prevalence of tuberculosis. Since that time, however, **there has been a resurgence of tuberculosis.** His increase has been seen not only in Africa and Asia, but also in Europe and so TBC remains an important cause of morbidity and mortality worldwide.

This resurgence is due to the acquired immunodeficiency syndrome (AIDS) epidemic and the increasing number of drug-resistant strains of *Mycobacterium tuberculosis* (**Multidrug-resistant Tuberculosis or MDR**).

- **MDR**

MDR tuberculosis is no more infective than normal tuberculosis. However, it is a more serious infection, **requiring prolonged administration of more toxic second-line drugs**, associated with higher morbidity and mortality rates. Patients also remain infectious for a longer period once treatment has been started.

- **IMMUNOCOMPROMISED PATIENTS**
They have a significantly higher prevalence of tuberculosis than does the general population and are also more likely to be infected with MDR tuberculosis. The pattern of disease is different in immunocompromised patients, who have a higher prevalence of extra-pulmonary involvement.

**SUBTYPE OF TUBERCULOSIS**

Tuberculosis is usually confined clinically to the respiratory system (pulmonary TBC, 80% of cases). However, it can affect any organ system (extra-pulmonary TBC), particularly in immunocompromised individuals.

*Pulmonary* tuberculosis is classically divided into primary and postprimary (reactivation) tuberculosis.

In *extra-pulmonary* TBC the more common sites involved are: bones, genitourinary system and central nervous system. Other sites involved are, in order of frequency: abdomen, heart (cardiac TBC), eye (ocular TBC)

**RADIOLOGIST’S ROLE**

Tuberculosis demonstrates a variety of clinical and radiologic features depending on the organ site involved and has a known propensity for dissemination from its primary site. Thus, tuberculosis can mimic a number of other disease entities, and it is important to be familiar with the various radiologic features of tuberculosis to ensure early, accurate diagnosis.

Furthermore, there is a considerable overlap in the radiologic manifestations of both primary and postprimary pulmonary TBC. However, confirming the diagnosis is more important than identifying the subtype, because this allows initiation of a correct clinical management.

**Imaging findings OR Procedure details**

**PART I - PULMONARY TBC**

**PRIMARY PULMONARY TBC**
It's seen in patients not previously exposed to *M. tuberculosis* and it's most common in infants and children. It has the highest prevalence in children under 5 years of age.

Although primary tuberculosis is the most common form of pulmonary TBC in infants and children, it has been increasingly encountered in adult patients and now accounts for 23%-34% of all adult cases of tuberculosis.

**Chest radiography** (FIG. 1) remains the mainstay of diagnosis; however, normal radiographic findings may be seen in up to 15% of patients with proved tuberculosis. **High-resolution CT** (FIG. 2) is more sensitive.

Primary tuberculosis typically manifests radiologically with these main entities:

- parenchymal disease
- lymphadenopathy
- pleural effusion (one-fourth of patients, usually unilateral; possible calcification), PNX (FIG. 1)
- miliary disease (1-7%, overall in elderly, infants and immunocompromised)
- atelectasis (either lobar or segmental, frequently seen in children under 2 years of age)

Typical presentation # "primary complex":

a) **parenchymal disease** (FIG. 1 and 2): dense, homogeneous parenchymal consolidation that affects the areas of greatest ventilation # the most common sites are the middle lobe, the lower lobes, and the anterior segment of the upper lobes; its appearance is often indistinguishable from that of bacterial pneumonia.

b) **lymphadenopathy** (up to 96% of children and 43% of adults): typically unilateral and right sided, involving the hilum and right paratracheal region, although it is bilateral in about one-third of cases.

Evolution of primary complex:

- in approximately two-thirds of cases, the parenchymal focus resolves without sequelae (it can take up to 2 years);
- radiologic scar that can calcify in up to 15% of cases, entity that is known as a Ghon focus;
- persistent masslike opacities called tuberculomas, that can cavitate (approximately 9% of cases) (FIG. 2)

**LYMPHADENOPATHY (FIG. 3)**
Although lymphadenopathy is usually associated with other manifestations of tuberculosis, it can be the sole radiographic feature, a finding that is more common in infants and decreases in frequency with age.

Nodes involved often calcify (6 month/>). The combination of calcified hilar nodes and a Ghon focus is called a Ranke complex and is suggestive of previous tuberculosis, although it can also result from histoplasmosis.

CT is more sensitive than chest radiography for assessing lymphadenopathy.

Any nodes greater than 2 cm in diameter generally have a low-attenuation center secondary to necrosis at CT (FIG. 3). This finding is highly suggestive of active disease, in the appropriate clinical setting.

The radiologic differential diagnosis for tuberculous lymphadenopathy includes metastases (FIG. 4) and histoplasmosis in endemic areas.

**POST-PRIMARY TBC**

The term post-primary tuberculosis is generally used to refer to both re-infection with (30-60%) and reactivation of tuberculosis (40-70%) in patients with immunodepression.

It remains primarily a disease of adolescence and adulthood (40-60 years old) and it occurs in patients previously sensitized to M. tuberculosis.

Primary tuberculosis is usually self-limiting, whereas post-primary tuberculosis is progressive, with cavitation as its hallmark, resulting in hematogenous dissemination of the disease as well as disease spread throughout the lungs.

The features of primary and post-primary tuberculosis may overlap. However, the distinguishing features of post-primary tuberculosis include:

- predilection for the upper lobes
- absence of lymphadenopathy
- presence of cavitation

**CT FINDINGS**
**Parenchymal disease** (the earliest finding, FIG. 7, 8 and 9): patchy, poorly defined consolidation, particularly in the apical and posterior segments of the upper lobes, bilateral in one-third of cases.

**Cavitation** (FIG. 9 and 10): the hallmark of post-primary tuberculosis, that affects about 50% of patients and indicates a high likelihood of activity. 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.

**Centrilobular nodules and branching centrilobular areas of increased opacity** (FIG 5, 6 and 7): if there is airway disease and, in particular, *endobronchial spread of infection* (most common complication of TBC cavitation), "tree-in-bud" opacities may develop; this is characteristic but non pathognomonic for active TBC (can also be seen with viral, fungal, and parasitic infections and also in allergic bronchopulmonary aspergillosis).

**Airway involvement**: characterized by bronchial stenosis, leading to lobar collapse and by traction bronchiectasis.

**Pleural effusion** (18%, usually with parenchymal disease; possible calcification).

**Miliary Disease**

Miliary tuberculosis occurs in 2% to 6% of primary TBC and also occur in post-primary TBC, when the host's defence mechanisms are overwhelmed.

**High-resolution CT** (FIG. 11) is more sensitive than conventional radiography and show in 85% of cases a mixture of both sharply and poorly defined nodules of 1 to 2 mm, widely disseminated throughout the lungs (*haematogenous spread of infection*), with a slight lower lobe predominance.

The nodules usually resolve within 2-6 months with treatment, without scarring or calcification.

**PART II - EXTRA-PULMONARY TBC**

**PART IIA - MUSCULOSKELETAL TUBERCULOSIS**
Musculoskeletal system is involved in only 1%-3% of cases of TBC; however, the resultant bone and joint destruction is the cause of severe morbidity and of neurologic sequelae in spinal involvement. It affects patients of all ages and most frequently the spinal column, pelvis, hip, and knee.

Diagnosis is often difficult (there are no pathognomonic radiologic features), with an average delay of 16-19 months between the onset of symptoms and reported diagnosis. Only histologic analysis and tissue culture can help confirm the diagnosis.

MR imaging is more sensitive than conventional radiography and CT in assessing the extent of bone and joint involvement and for the diagnosis and assessment of tuberculous spondylitis (high sensitivity to soft-tissue abnormalities).

A) **TUBERCULOUS SPONDYLITIS** (FIG. 12, 13 and 14)

- 50% of skeletal tuberculosis involves the spine (probably from hematogenous spread via the venous plexus of Batson), above all the lower thoracic and upper lumbar levels.

- Infection usually begins in the anterior part of the vertebral body adjacent to the end plate # demineralization and scalloping of the end plate # spread of infection to adjacent intervertebral disk # potential spread to additional spinal segments and subsequent spread into the paraspinal tissues, with formation of a paravertebral abscess (**Pott's abscess**, FIG. 12 and 13) +/- with calcification;

- if there is anterior subligamentous involvement of the spine, infection can extend both superiorly and inferiorly to more segments, with **sparing or delayed involvement of intervertebral disks**;

- Characteristically associated with little or no reactive sclerosis/local periosteal reaction, in contrast to pyogenic infections;

- Rarely affects the posterior vertebral elements (including the pedicles), in contrast to metastasis

- In the early stage, imaging appearances are entirely non specific; in differentiating TBC from pyogenic infection, the clinical picture is important: insidious onset of symptoms, a normal erythrocyte sedimentation rate, relevant respiratory symptoms, and slow disease progression (favoring the diagnosis of tuberculosis)
B) **TUBERCULOUS OSTEMYELITIS (EXTRASPINAL TBC OSTEOMYELITIS)** (FIG. 15)

- tuberculous osteomyelitis is **usually hematogenous** in origin and the isolated form in the absence of associated tuberculous arthritis is relatively rare

- is most commonly seen in **bones of the extremities** (femur, tibia), including the small bones of the hands and feet

- in long, tubular bones, often involves the epiphyses; in children metaphyseal foci can involve the growth plate (this feature differentiates tuberculosis from pyogenic infection)

- initial radiologic appearance is similar to that of other types of osteomyelitis and includes **foci of osteolysis with varying degrees of eburnation and periostitis.**

- the diagnosis is usually made after considerable delay, and radiographic changes are seen at clinical presentation; in contrast, in pyogenic infection, radiographic changes occur 2-3 weeks after presentation.

C) **TUBERCULOUS ARTHRITIS** (FIG. 16)

- may be secondary to **direct invasion** from an adjacent focus of tuberculous osteomyelitis or may result from **hematogenous dissemination**

- is typically **monoarticular** and primarily involves **large weight-bearing joints** (hip and knee)

- the imaging findings are similar to those of other infectious and inflammatory arthritides and are, therefore, non specific; these findings include **osteopenia, synovitis and other soft-tissue swellings, marginal erosions, and varying degrees of cartilage destruction**

- the end result is usually fibrous ankylosis of the joint

- the differential diagnosis includes pyogenic and fungal infections factors # favoring a diagnosis of TBC include insidious onset, minimal sclerosis, the relative absence of periosteal reaction and bone proliferation
PART IIB - **TUBERCULOSIS INVOLVING THE CNS**

Most tuberculous infections of the central nervous system (neurotuberculosis, 5% of patients) are usually a result of **hematogenous spread**. It results in two related pathologic processes: tuberculous meningitis (the most common manifestation, across all age groups) and intracranial tuberculomas and abscesses. Its prevalence is greater in immunocompromised patients.

A) **MENINGEAL INVOLVEMENT (TUBERCULOUS MENINGITIS)**

- typical radiographic finding, better seen at gadolinium-enhanced MR imaging than at CT # **anormal meningeal enhancement** (FIG. 16)

- appearance **non specific and with wide differential diagnosis**: meningitis from other infective agents, inflammatory diseases (rheumatoid arthritis and sarcoidosis) and neoplastic causes (both primary and secondary)

- most common complication # **communicating hydrocephalus**

B) **PARENCHYMAL INVOLVEMENT** (can exist in conjunction with tuberculous meningitis)

- most common parenchymal lesion # **tuberculoma (non-caseating granuloma)**, solitary, multiple, or miliary, anywhere within the brain parenchyma (above all within frontal and parietal lobes)

- it demonstrate **homogeneous enhancement**, with irregular wall of varying thickness and in one-third with central calcification

- **caseating tuberculoma with necrotic center** demonstrate a **ring enhancement** (FIG. 17) and usually have a variable amount of surrounding edema; abscesses can be similar in appearance

PART IIC - **GENITOURINARY TUBERCULOSIS**

TBC may involve the genitourinary tract as a secondary site following **hematogenous dissemination** from the lung. Genitourinary TBC is the **most common clinical manifestation of extrapulmonary tuberculosis**.

A) **KIDNEYS** (75% unilateral) (FIG. 18)

- the most common CT finding is renal **calcification** (50%)
- renal parenchymal **cavitation** may be detected as irregular pools of contrast material
- "moth-eaten" abnormality of calix at urography, due to erosions

B) **BLADDER** (tuberculous cystitis)

It manifests as a shrunken bladder with wall thickening and reduced bladder capacity.

C) **GENITALIA**

- **female**: obstruction of the fallopian tubes (**salpingitis**, 94% cases) with multiple areas of constriction, and calcified lymph nodes in the adnexal region, **endometrial adhesions** with deformity and obliteration of endometrial cavity

- **male**: involvement of the prostate gland or seminal vesicles (FIG. 19) may lead to **necrosis**, **calcification**, **caseation** (hypoattenuating lesions at CT), and cavitation

PART IIIC - **ABDOMINAL TUBERCULOSIS**

The abdomen is the most common focus of extrapulmonary tuberculosis, with the solid viscera being affected more often than the gastrointestinal tract. Although abdominal tuberculosis is usually secondary to pulmonary tuberculosis, radiologic evaluation often shows no evidence of lung disease. **CT is the mainstay** for investigating possible abdominal tuberculosis.

A) **LYMPHADENOPATHY**

The most common manifestation (FIG. 20) of abdominal TBC (55-60%), with **enlarged nodes with hypoattenuating centers and hyperattenuating enhancing rims** at CT (40-60% of cases), findings that are characteristic of, but not pathognomonic for, caseous necrosis.

The mesenteric, omental, and peripancreatic lymph nodes are most commonly involved.

B) **GASTROINTESTINAL TBC** (rare)

It can occur elsewhere in the small bowel, but almost always involves ileocecal region (90% of cases), usually both the terminal ileum and the cecum. Rare localizations are esophagus, stomach and proximal small bowel. The most common CT finding (FIG. 21) is **concentric mural thickening, with associated luminal narrowing with or without proximal dilatation**.
C) **HEPATOSPLENIC TBC**

- common in patients with **disseminated disease**, either miliary or macronodular

- *micronodular/miliary involvement*: seen in patients with miliary pulmonary TBC, characterized by innumerable 0.5-2.0-mm **hypoattenuating** nodules. The liver appears hyperechoic at US.

- *macronodular involvement*: uncommon, with liver or splenic enlargement and **hypoattenuating** lesions at CT, with irregular ill-defined margins and minimal but definite **peripheral contrast enhancement** (imaging appearances non specific and similar to those of multiple metastases and abscesses). At MR imaging, these lesions are hypointense with T1-weighted sequences and hyperintense with T2-weighted sequences.

- hepatic tuberculomas eventually tend to **calcify**; the presence of calcified granulomas at CT in patients with known risk factors and in the absence of a known primary tumor should raise suspicion for TBC

D) **ADRENAL TBC**

- it's seen in up to 6% of patients with active tuberculosis.

- bilateral adrenal involvement and an Addisonian type clinical picture

- CT signs: **enlarged glands associated with large, hypoattenuating necrotic areas**, with or without dotlike calcification

**Images for this section:**
Frontal chest radiograph demonstrates dense and homogeneous tuberculous consolidation in the right upper lobe, indistinguishable from a bacterial pneumonia. From this side is also evident pneumothorax. Other subpleural, poorly defined consolidations are present in the middle and upper left lung.

**Fig. 1:** Primary pulmonary TBC, chest X-Ray
Axial CT scan show two dense and homogeneous parenchymal consolidation in the right and left upper lobes, with little cavitations.

**Fig. 2:** Primary pulmonary TBC at CT
Fig. 3: Lymphadenopathy in TBC
Fig. 4: Lymphadenopathy in TBC, differential diagnosis (lung metastases)
Axial CT scan show multiple nodules (size from mm to 1 cm), in the right lung, particulary in the upper lobe, consistent with active TBC with endobronchial spread.

**Fig. 5:** Post-primary pulmonary TBC, endobronchial spread of infection with centrilobular nodules
**Fig. 6:** Post-primary pulmonary TBC, endobronchial spread of infection with centrilobular nodules (same patient of FIG. 5)
Fig. 7: Post-primary pulmonary TBC, parenchymal disease
Axial CT scan demonstrate “tree-in-bud” opacities (multiple, centrilobular nodules) red arrow) in the anterior segment of the right upper lobe and more homogeneous parenchymal consolidation (yellow arrow) in the apical segment of right lower lobe, with an eccentric cavitation.

**Fig. 8:** Post-primary pulmonary TBC, parenchymal disease and endobronchial spread of infection with centrilobular nodules
Axial CT scans of the same patient show multiple and bilateral nodules (green arrow) and parenchymal consolidation, particularly in both upper lobes. Some of these consolidations demonstrate cavitation (red arrow).

**Fig. 9:** Post-primary pulmonary TBC, parenchymal disease and cavitation
Fig. 10: Post-primary pulmonary TBC, cavitation
High-resolution CT showing the presence of widely diffuse small nodules, in patient with miliary disease

Fig. 11: Miliary tuberculosis, HRCT
Axial and coronal CT scan demonstrate a lytic destruction of a dorsal vertebral body (red arrow), with an adjoining soft-tissue abscess in the right anterior paravertebral space.

**Fig. 12:** Tuberculous spondylitis with Pott's abscess
Axial, coronal and sagittal CT scan of the same patient show lytic destruction of another vertebral body (L3, red arrow). Multiloculated bilateral iliopsoas abscesses (particularly of the right one, green arrow) and in the adjoining soft-tissue of vertebral body (yellow arrow) are present.

**Fig. 13:** Tuberculous spondylitis with Pott's abscess
Sagittal T2-weighted, T1-weighted and contrast-enhanced T1-weighted MR image show the presence of spondylitis of L5-S1, without a certain involvement of the adjacent intervertebral disk. An abscessual cavity (red arrow) is evident in the presacral space.

CT scans demonstrate better than MRI the presence of cortical erosion of both end plate and the narrowing of adjacent intervertebral space.

**Fig. 14:** Tuberculous spondylitis, MRI
Axial, T1-weighted (A), fat-sat T2-weighted (STIR) (B) and axial and sagittal contrast-enhanced T1-weighted (C, B) MR image show the presence of intramedullary focus of osteolysis (T1-hypointense, STIR-hyperintense and with peripheral enhancement) of TBC, involving the distal lateral femur; the focus is associated with irregular cortical interruptions.

**Fig. 15:** Tuberculous osteomyelitis
A) Axial and coronal gadolinium-enhanced T1-weighted MR image demonstrates peripheral enhancement around and along the left sacroiliac joint. In front of the joint, between iliac vessels and iliac muscle, is present an abscess (red arrow), with thick, regular and peripheral rim enhancement and central hypointense area.

B) Axial and coronal contrast-enhanced CT scans demonstrate better than MRI marginal erosion of the left sacroiliac joint. It also recognizable the abscess (red arrow) in front of the joint.

Fig. 16: CT and MRI features of tuberculous arthritis of left sacroiliac joint with abscess
Axial contrast-enhanced T1-weighted MR image shows multiple ring-enhancing lesions within both cerebral hemispheres, with variable amount of surrounding edema. It is also present a leptomeningeal enhancement along the interhemispheric fissure.

**Fig. 17:** MRI features of tuberculous infection of the central nervous system: tuberculous meningitis and multiple absceses
Unenhanced (A) and contrast-enhanced (B, C) CT scan show the presence, in right kidney, of parenchymal calcifications (red arrow) and cavitation (green arrow), seen as hypoattenuating lesion within the parenchyma. Regular enhancement of left kidney.

**Fig. 18:** Genitourinary TBC: kidney’s involvement
Fig. 19: Male genitourinary TBC, involving prostate gland and seminal vesicles
Axial and coronal contrast-enhanced CT scan that demonstrate enlarged nodes (celiac and omental), with hypoattenuating central areas and peripheral rim enhancement (red arrows). These findings represented the only abnormality that was seen in this patient.

**Fig. 20:** Abdominal TBC with lymphadenopathy
Fig. 21: Gastrointestinal TBC

Axial contrast-enhanced CT scan that demonstrate a diffuse concentric wall thickening (11 mm) of the small bowel, with an homogeneous enhancement.
Conclusion

The infection of *Mycobacterium tuberculosis* had in the last years an evident recrudescence, due to HIV epidemic, to constant migrant population and to the spread of drug resistant strains (MDR).

The two main pathologic form include pulmonary and extra-pulmonary infection. Both have a variety of clinical and radiologic features depending on the organ site involved, also in the patients with decreased immunity.

The radiologist has an important role in the evaluation of the several manifestations and sites of infection, keeping in mind that tuberculosis can mimic a number of other disease entities.

Personal Information

References