Extrapulmonary Tuberculosis: a 10-year review in our hospital.

Poster No.: C-2396
Congress: ECR 2013
Type: Educational Exhibit
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Keywords: Infection, Abscess, Diagnostic procedure, Ultrasound, MR, CT, Lymph nodes, Head and neck, CNS
DOI: 10.1594/ecr2013/C-2396

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Learning objectives

To discuss and illustrate the radiological manifestations of extrapulmonary tuberculosis (EPTB) in a series of 20 patients.

Background

Tuberculosis (TB), discovered in 1882 by Robert Koch, is considered by some as the most common infectious-contagious disease in the world.

Although its incidence has been on the decline since 2002, among the 8.8 million cases of tuberculosis reported worldwide in 2010, an estimated 418 000 individuals were located in the European Region.

Undoubtedly the disease still remains a major threat despite control efforts and its resurgence in the mid 90’s has been mainly attributed to different causes, such as increased global migrations, Human Immunodeficiency Virus and the appearance of multidrug-resistant forms.

TB can involve any organ system in the whole body and it can be life-threatening. While pulmonary TB is the most common presentation, extrapulmonary tuberculosis (EPTB) is also an important clinical problem (it accounts for 10-20% of all cases of tuberculosis in immunocompetent patients, and up to 60% in AIDS patients).

The term EPTB has been used to describe isolated occurrence of TB at body sites other than the lung. However, practically in all the cases of EPTB a primary pulmonary focus exists, which can be visible or not in chest radiographs. EPTB is the result of dissemination of tubercle bacilli from this initial focus in the lungs soon after primary infection. The dissemination is mainly by contiguity, or by way of the lymphatic or hematogenous route.

Excepting the pleural involvement, the nodal, urogenital and osteoarticular are the most frequent locations of EPTB. Other localizations are highly infrequent.

Early detection is crucial to successful management and requires a high-clinical suspicion. Imaging plays a key role in its diagnosis, however the course of TB and its resultant radiological pattern fall out of the interaction between the organism (Mycobacterium tuberculosis) and the patient response.
Imaging findings OR Procedure details

We revised the patients with proven EPTB in a retrospective 10-year (2002-2012) study performed in our hospital.

We retrieved 20 patients with microbiologically confirmed EPTB and we performed a posterior revision of their imaging data and clinical histories.

We present a pictorial review of different locations of EPTB, according to our experience: 9 tuberculous lymphadenitis, 2 central nervous system (CNS) TB, 3 urinary TB, 1 tuberculous pericarditis, 3 musculoskeletal TB (2 spondylitis and 1 arthritis), 1 splenic TB, and 1 gastrointestinal TB.

**Tuberculous Lymphadenitis**

Lymphadenitis is the most commonly occurring form of EPTB.

Cervical nodes are most frequently affected, accounting for approximately 63% of cases, followed by mediastinal (27%) [Figure 1] and axillary ones (8%) [Figure 2 and Figure 3], but submandibular, intraabdominal, inguinal and intramammary locations were also described.

TB cervical lymphadenitis (also known as Scrofula or King’s evil) is the most common manifestation of EPTB and is a very frequent cause of peripheral lymphadenitis. Within the neck, the nodes in the posterior triangle (51%) [Figure 4 and Figure 5] and deep upper cervical (48%) are more frequently involved than others. In the majority of cases lymphadenitis is unilateral.

Ultrasound (US) is an excellent first line investigation, able to assess cervical lymphadenopathies and to enable guided fine needle aspiration cytology if necessary.

On gray-scale US, TB nodes tend to be hypoechoic, round, without echogenic hilum and tend to show intranodal cystic necrosis, nodal matting, and adjacent soft-tissue edema. On color Doppler, power Doppler, and 3D sonography, the vascular distribution of TB nodes is varied. However, displacement of hilar vascularity is common and is due to the high incidence of intranodal cystic necrosis, which displaces the vessels (Figure 6).
CT appearance of TB lymphadenitis is variable, depending on the degree of caseation present in the node. Nodes may initially appear merely enlarged (Figure 7), often with attenuation similar to muscle. Eventually central caseation develops and the nodes become centrally low density and eventually frankly cystic, with peripheral rim enhancement (Figure 8). They are usually matted together with only minor surrounding inflammatory changes.

On MRI the involved nodes are initially homogeneously enlarged but later undergo central necrosis, manifesting with central hypointensity and hyperintensity on T1- and T2-weighted MR images, respectively. Finally, cystic transformation with peripheral enhancement is seen.

Central Nervous System (CNS) Tuberculosis

CNS tuberculosis (TB) is uncommon; involvement of the CNS is seen in approximately 5% of patients with TB, with a greater prevalence in immunocompromised ones (up to 15% of cases of AIDS-related TB).

CNS TB usually results from hematogenous spread, or from direct rupture or extension of a subependymal or subpial focus (Rich focus) and may be located in the meninges, brain, or spinal cord. CNS TB can manifest in a variety of forms, including tuberculous meningitis, tuberculomas, tuberculous abscesses, tuberculous cerebritis, spinal tuberculous arachnoiditis and miliary tuberculosis.

The most common parenchymal lesion of CNS TB is tuberculoma (tuberculous granuloma). This lesion may be solitary, multiple, or miliary and may be seen anywhere within the brain parenchyma, although it most commonly occurs within the frontal and parietal lobes.

At CT, tuberculomas appear as round or lobulated masses with low or high attenuation. They demonstrate homogeneous or ring enhancement and have irregular walls of varying thickness. One-third of patients demonstrate the "target sign" (central calcification or punctate enhancement with surrounding hypoattenuation and ring enhancement). This finding is highly suggestive of, but not pathognomonic for, tuberculosis.

The MRI findings depend on whether the tuberculoma is caseating. It is thought that there is a progression from a noncaseating (solid) to a caseating (liquid) center.
A noncaseating tuberculoma is hypointense relative to gray matter on T1W and hyperintense on T2W images, with homogeneous gadolinium enhancement.

Caseating tuberculomas with a solid center are isointense to hypointense on both T1- and T2W images. They usually have a variable amount of surrounding edema (hyperintense on T2W images) (Figure 9).

Caseating tuberculomas with a liquid center are hypointense on T1-W images and centrally hyperintense on T2-W images, with a peripheral capsule (seen as a hypointense rim on T2-W images). Rim enhancement is usually seen at gadolinium-enhanced MRI (Figure 10).

After treatment, tuberculomas can completely resolve; however, calcification is seen in up to one-fourth of cases and is identified most clearly at CT.

**Urinary Tuberculosis**

Urinary TB is a common clinical manifestation of EPTB, more frequently seen in young males. The kidney, the epididymis and the collecting system are usually first infected; the other genitourinary organs may be affected by ascendant or descendant dissemination.

Approximately 75% of renal TB involvement is unilateral, the most common CT finding being renal calcification (50% of cases) (Figure 11). At intravenous urography, the earliest abnormality is a "moth-eaten" calix due to erosions, which progresses to papillary necrosis. Hydronephrosis tends to have irregular margins and filling defects owing to caseous debris. Renal parenchymal cavitation may be detected as irregular pools of contrast material. Dilated calices (hydrocalicosis) with related infundibular stricture at one or more sites within the collecting system may be seen. Characteristic calcifications in a lobar distribution are often seen in end-stage tuberculosis (tuberculous autonephrectomy).

Sometimes an iliopsoas abscess may be originated from kidney involving (Figure 12).

**Musculoskeletal Tuberculosis**

The musculoskeletal TB accounts for up to 35% of all forms of EPTB, and up to 2% of all cases of tuberculosis.

**Tuberculous Spondylitis**


Approximately 50% of skeletal tuberculosis involves the spine (mostly the lower thoracic and upper lumbar levels), with TB spondylitis.

Infection usually begins in the anterior part of the vertebral body adjacent to the end plate, with subsequent demineralization, seen on conventional radiographs as loss of definition of its dense margins. The classic pattern is the involvement of more than one vertebral body together with the intervening disks. Tuberculosis rarely affects the posterior vertebral elements (including the pedicles) and is characteristically associated with little or no reactive sclerosis or local periosteal reaction, a feature that helps distinguish it from pyogenic infections of the spine.

TB spondylitis also allows spread into the paraspinal tissues, resulting in the formation of a paravertebral abscess known as a Pott abscess. Calcification within the abscess is virtually diagnostic for tuberculosis. If left untreated, the infection eventually results in vertebral collapse and anterior wedging, leading to kyphosis and gibbus formation. With healing, ankylosis of the vertebral bodies occurs, with obliteration of the intervening disk space.

CT is especially useful for guided fine needle aspiration cytology or biopsy.

MRI is the preferred imaging modality in the diagnosis of TB spondylitis, in order to assess the soft-tissue abnormalities and the possible medullary compression. In a Pott abscess the disc and the adjacent vertebral plates are hyper-intense on STIR and T2W MRI, and hypointense on T1W MRI, with intense Gadolinium-enhancement (Figure 13 and Figure 14).

**Tuberculous Arthritis**

Tuberculous arthritis is characteristically a monoarthritis affecting large weight-bearing joints like hip and knee.

There are no pathognomonic radiological findings for TB arthritis. Nonspecific changes may be seen, such as juxtaarticular osteopenia, synovitis and other soft-tissue swellings, marginal erosions, and varying degrees of cartilage destruction. Joint space narrowing is usually delayed. The end result is usually fibrous ankylosis of the joint. Factors favoring a diagnosis of tuberculosis include insidious onset, minimal sclerosis, the relative absence of periosteal reaction and bone proliferation, and relative preservation of joint space in the early stages (Figure 15).
Tuberculous Pericarditis

Tuberculosis involving the heart is rare, accounting for only 0.5% of cases of EPTB.

The main presenting finding is pericardial involvement, particularly in immunocompromised patients. The primary sign of TB pericarditis is pericardial thickening of more than 3 mm in adults, seen in the majority of cases. CT demonstrates a thickened, irregular pericardium, frequently with associated mediastinal lymphadenopathy.

Most patients have distention of the inferior vena cava to a diameter exceeding 3 cm; pleural effusions, typically bilateral; and deformities of the intraventricular septum. Less than 20% of patients have pericardial effusions (Figure 16) or develop localized pericardial calcification.

Splenic Tuberculosis

Tuberculous splenic involvement is very rare and it is most likely secondary to hematogenous dissemination of the primary form of the disease.

Miliary splenic TB manifests as multiple tiny (0.5-2.0-mm), low-attenuation foci on CT (Figure 17) and multiple hypoechoic nodules on US.

The macronodular form is rare and manifests as diffuse splenic enlargement with multiple low-attenuation lesions or a single tumorlike mass. On contrast-enhanced CT, early-stage lesions may demonstrate central enhancement whereas more advanced lesions may demonstrate calcification.

Intraabdominal lymphadenopathies are usually seen.

Gastrointestinal Tuberculosis

Gastrointestinal tuberculosis is rare; when present, however, it almost always involves the ileocecal region (90% of cases), usually both the terminal ileum and the cecum, with or without proximal dilatation.
The most common CT finding is mural thickening, which is typically concentric but if eccentric tends to involve the medial cecal wall.

Localized lymphadenopathy is usually seen (with hypodense center that represents the caseum). (Figure 18)

Images for this section:

**Fig. 1:** Multiple mediastinal tuberculous lymphadenopathies (right paratracheal, subcarinal and pre-vascular spaces) (arrows). Axial contrast-enhanced thoracic CT.
**Fig. 2:** Right axillary tuberculous lymphadenopathies on Ultrasound. Round, hypoechoic nodes, without echogenic hilum and with prominent vascularity.

**Fig. 3:** Same patient as in Figure 2 on axial plain (A) and contrast-enhanced (B) thoracic CT. Right axillary tuberculous lymphadenopathies (arrows) with adjacent collection (asterisk) that extends to the anterior thoracic wall, affecting the pectoralis minor muscle.
The collection presents a well-defined, contrast-enhancing wall, internal septa and central hypodensity (related to central necrosis).

**Fig. 4:** Cervical tuberculous abscess on ultrasound. Large cystic lesion with thick septa and internal echogenic material.
Fig. 5: Same patient as in Figure 4, on axial contrast-enhanced cervical CT with soft-tissue (A, B) and bone window settings (C, D). Large heterogeneous (mainly hypodense, due to central necrosis) collection located in the left paravertebral cervical space (asterisk). The collection has a thick contrast-enhancing wall, and produces C5-C6 vertebral destruction, with enlarged left foramen (arrow), but without medullary canal invasion.
Fig. 6: Multiple submandibular lymphadenopathies on Ultrasound. Round, hypoechoic and mildly heterogeneous nodes.
**Fig. 7:** Tuberculous cervical lymphadenitis on axial contrast-enhanced cervical CT. Intense contrast enhancement of right posterior triangle (A) and submandibular (B) lymphadenopathies (arrows).

![Fig. 7: Tuberculous cervical lymphadenitis on axial contrast-enhanced cervical CT.](image)

**Fig. 8:** Tuberculous cervical lymphadenitis at axial contrast-enhanced cervical CT. Multiple cervical nodes with peripheral enhancement and central hypoattenuation representing central necrosis. A - Left superficial laterocervical, posterior cervical triangle and carotid space (asterisks). B, C - Adenopathic conglomerate in the left vascular space (arrows), with anterior displacement of the jugular vein and common carotid artery. D - Necrotic lymphadenopathy in the anterior midline (asterisks).

![Fig. 8: Tuberculous cervical lymphadenitis at axial contrast-enhanced cervical CT.](image)
Fig. 9: CNS parenchymal tuberculosis. Two tuberculomas, seen as contrast-enhancing focal lesions on Coronal (A, B) and Sagittal (C) Gadolinium-enhanced T1W MRI, and as ill-defined hyperintense focal lesions with perilesional edema on axial FLAIR (D) and Coronal T2W (E, F) MRI.
**Fig. 10:** CNS parenchymal tuberculosis. Multiple bilateral small ring-enhancing focal lesions (tuberculomas) (arrows) in cerebral and cerebellar parenchyma on coronal Gadolinium-enhanced T1W MRI.

**Fig. 11:** Renal tuberculosis on plain axial abdominal CT. Small kidneys, both with important decrease of parenchymal thickness. Small calcification in right kidney and coarse calcification (arrow) in left kidney. Cystic cavities in right kidney (asterisk).

**Fig. 12:** Abscess of the psoas muscle likely originating from left tuberculous kidney. Left kidney with coarse parenchymal calcification (arrow), which continues with a well-defined collection (asterisk), that extends caudally along the psoas muscle, through the lumbar zone into the gluteal region. The collection presents a contrast-enhancing wall and central
low-attenuation (representing necrosis) collection. Axial contrast-enhanced abdominal CT.

**Fig. 13:** Dorsal Pott abscess in a patient with TB spondylitis on axial contrast-enhanced dorsal CT with soft-tissue (A, B) and bone (C, D) window settings. Bone destruction of the dorsal vertebral bodies with lytic irregular lesions and paravertebral abscess, seen as a soft-tissue mass (asterisk) that surround the vertebral body. The mass presents peripheral wall enhancement, central low-attenuation related to necrosis. Medullary canal invasion with epidural abscess (arrow) which produces anterior spinal cord compression.
Fig. 14: Dorsal Pott abscess in a patient with TB spondylitis (same patient as in Figure 13) on sagittal plain (A) and gadolinium-enhanced (B) T1W MRI. D8, D9 and D10 vertebral wedging. Hypo-intense discs and adjacent vertebral plates on T1W MRI, with intense heterogeneous Gadolinium-enhancement. Medullary canal invasion with epidural abscess that produces spinal cord compression (arrow).
Fig. 15: Tuberculous arthritis on axial plain CT with soft-tissue (A) and bone window (B, C) settings. Widening of the left sternoclavicular join with marginal erosions in the sternal edge and proximal clavicle osteolysis (arrow). Adjacent soft-tissue swelling.

Fig. 16: Tuberculous pericarditis on axial contrast-enhanced thoracic CT. Mild pericardial thickening with pericardial effusion. Left pleural effusion.
Fig. 17: Miliary splenic tuberculosis on axial contrast-enhanced abdominal CT. Innumerable tiny hypoattenuating foci in the splenic parenchyma. Multiple hypodense (necrotic) enlarged nodes (periportal and peripancreatic spaces).

Fig. 18: Intestinal tuberculosis on axial contrast-enhanced abdominal CT. Concentric mural thickening of the terminal ileon, with associated luminal narrowing (A, B - short arrows). Irregular thickening of the cecal wall (C - large arrow). Multiple mesenteric and retroperitoneal lymphadenopathies (mostly necrotic - hypodense).
Conclusion

Tuberculosis can affect any organ system in the body and can be devastating if left untreated.

The unusual finding of extrapulmonary tuberculosis (EPTB) can mimic numerous other disease entities. The radiological imaging plays an important role in identifying EPTB patients.

A high degree of clinical suspicion and familiarity with the various radiologic manifestations of EPTB might be helpful for the exact diagnosis.

Early diagnosis and appropriate treatment are the only effective tools in order to diminish transmission and lead towards gradual disease elimination.

References


Personal Information