Imaging Spectrum of Extrathoracic Tuberculosis

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KEYWORDS

• Musculoskeletal TB • CNS TB • TB lymphadenitis • Abdominal TB • Genitourinary TB • Peritoneal TB

KEY POINTS

- The incidence of extrathoracic tuberculosis (ETB) continues to increase slowly, especially in immunocompromised and multidrug-resistant tuberculosis (TB) patients.
- ETB has nonspecific clinical presentations, and being less frequent, is less familiar to most physicians.
- The most common sites of ETB include lymphadenopathy, peritoneum, ileocecal region, hepatosplenic, genitourinary, central nervous system (CNS), and musculoskeletal (MSK) regions; multisystem involvement is common.
- For early and correct diagnosis of ETB, imaging plays a vital role.
- Imaging modalities of choice are computed tomography (lymphadenopathy and abdominal TB) and MR imaging (CNS and MSK TB).

INTRODUCTION

Even today, tuberculosis (TB) remains a global health problem despite availability of effective antituberculous treatment. This global resurgence of TB is mainly owing to the AIDS epidemic, increasing migration from endemic areas to the developed world where TB is uncommon, and the increasing number of drug-resistant strains of *Mycobacterium tuberculosis*. Although pulmonary involvement is the most common form of TB, almost any organ can be involved by TB, particularly in immunocompromised patients.[1] The incidence of extrathoracic TB (ETB) continues to increase slowly, especially in immunocompromised and multidrug-resistant TB patients.[2,3] The most common sites of ETB include lymph nodes, ileocecal (IC) region, peritoneum, liver, spleen, genitourinary system, central nervous system (CNS), and musculoskeletal system.

The diagnosis of ETB may be missed or delayed owing to nonspecific patient symptoms as well as a lack of familiarity of protean manifestations to most physicians. ETB involves relatively inaccessible sites; thus, fewer bacilli can cause much greater damage. In addition, multisystem involvement is common with ETB. For early and correct diagnosis of ETB, imaging plays a vital role. In this article, we discuss the imaging features of ETB.

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CENTRAL NERVOUS SYSTEM TUBERCULOSIS

Tuberculous involvement of the CNS is a serious form of extrapulmonary disease accounting for 1% of all TB and 10% to 15% of extrapulmonary TB. CNS TB is a leading cause of morbidity and mortality in endemic regions, particularly in children.[4,5] Depending on the site of involvement and pathologic manifestations, varied forms of CNS TB have been described such as tuberculous meningitis (TBM) and its complications, focal cerebritis, tuberculoma, and tuberculous abscess (**Table 1**). Spinal infection is less common and causes either arachnoiditis and/or intramedullary tuberculomas. Imaging plays a vital role in diagnosis of CNS TB, in the detection of early complications and also in follow-up. Computed tomography (CT) and MR imaging are used in the diagnosis of CNS TB; however, MR has greater sensitivity and specificity than CT in detection of CNS TB.

Pathophysiology

TB bacilli reach the CNS by an hematogeneous route from distant active TB sites elsewhere in the body. During bacteremia, small Rich's focus develops in the meninges, in the subpial or subependymal surface of the brain or the spinal cord. It may remain dormant for a long period. Rupture of Rich's focus produces various types of CNS TB.[6] Infrequently, CNS TB can occur by direct spread from adjacent infected paranasal sinuses or mastoid air cells. Rupture of TB granuloma into the subarachnoid space and cerebrospinal fluid (CSF) results in leptomeningitis. Leptomeningitis can lead to obstructive hydrocephalus or vasculitis. Depending on the virulence of the organisms and host immunity, parenchymal cerebral tuberculous foci may develop into tuberculoma or tuberculous brain abscess.[7]

Table 1
Neurologic TB spectrum

Intracranial TB	Intraspinal TB
Meningeal TB	Intraspinal TBM
TBM	Tuberculous myelitis
Pachymeningitis	Intramedullary tuberculoma
Parenchymal TB	—
Tuberculoma	
Tuberculous abscess	
Tuberculous cerebritis	
Tuberculous encephalopathy	
Complications of TBM: vasculitis, hydrocephalus, cranial neuropathy	

Abbreviations: TB, tuberculosis; TBM, tuberculous meningitis.

TUBERCULOUS MENINGITIS

In developing countries, TBM is the most common cause of chronic meningitis. The diagnosis of TBM is challenging and is based on a constellation of clinical features, imaging findings, and CSF abnormalities, which include detection of acid-fast bacilli by direct staining of CSF or positive CSF culture for bacilli and response (both clinical and CSF) to antituberculous medications.[8] However, the clinical features are often nonspecific and microbiological detection of the organisms is difficult owing to paucibacillary CSF. Imaging plays a crucial role in the diagnosis of TBM and early detection of its complications. Focal or diffuse cerebral atrophy, areas of gliosis secondary to infarcts, hydrocephalus, meningeal, or ependymal calcifications, and occasionally syringomyelia are the sequelae of TBM.

Imaging characteristics suggestive of TBM are basal meningeal enhancement, hydrocephalus, tuberculomas, and infarcts on CT and MR imaging (**Figs. 1** and **2**).[9,10] Basal exudates in TBM appear mildly hyperdense on plain scans and reveal intense homogenous postcontrast enhancement.[11] Basal exudates in TBM appear hyperintense on fluid-attenuated inversion recovery MR imaging and show intense enhancement on postcontrast T1-weighted images. Linear enhancement along the ventricular margins confirms ependymitis. MR imaging is superior to CT in diagnosing suspected meningitis and associated complications.[12] The magnetization transfer (MT) technique is reported to be superior in differentiating TBM from other nontuberculous causes of meningitis. Meninges appear hyperintense on precontrast T1-weighted MT images and enhance further on postcontrast T1-weighted MT images. MT ratio in TBM is significantly higher than in viral meningitis, and fungal and pyogenic meningitis show higher MT ratio compared with TBM.[13] Patients with acquired AIDS may show minimal or absent meningeal enhancement, likely owing to lack of immunologic response.[14]

TUBERCULOUS PACHYMENINGITIS

Chronic TBM can infrequently lead to localized or diffuse dura matter involvement and may present as tuberculous pachymeningitis. There may be pial or parenchymal extension. The cavernous sinuses, floor of middle cranial fossa, and tentorium are frequently involved. Tuberculous pachymeningitis