Central Nervous System Tuberculosis

Pathophysiology and Imaging Findings

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KEYWORDS

• CNS tuberculosis • Tuberculoma • Intracranial infection • Spinal tuberculosis

KEY POINTS

- Tuberculosis (TB) is a global clinical concern, particularly after the human immunodeficiency virus pandemic.
- Imaging, particularly magnetic resonance imaging, is a cornerstone in the diagnosis as well as follow-up of central nervous system (CNS) tuberculosis.
- Imaging appearance of CNS TB is becoming more and more complex and atypical with the onset of multidrug-resistant tuberculosis.
- Early, accurate diagnosis can help in preventing morbidity and mortality.
- Newer imaging techniques like magnetic resonance spectroscopy help to improve characterization and thus aid in diagnosis of atypical CNS TB.

INTRODUCTION

Tuberculosis (TB) remains a prominent global problem especially because of the increasing incidence of human immunodeficiency virus (HIV) and drug-resistant strains, although its incidence seems to have declined recently.[1] According to the World Health Organization report, 1.3 million deaths were caused by TB in 2008, which is equivalent to 20 deaths per 10,000 population.[2] Among all other forms of TB, central nervous system (CNS) TB accounts for approximately 1% and has the highest mortality.[3] Although diagnostic evaluation includes various microbiological, pathologic, molecular, and biochemical investigations,[4] imaging modalities have an important diagnostic role. Imaging helps in early diagnosis and helps in preventing morbidity and mortality. Imaging is essential in showing complications in addition to diagnosis of CNS TB.[5]

PATHOGENESIS

Mycobacterium TB is the most common organism causing tuberculous infection of CNS. Other species of mycobacteria may be involved in immunocompromised patients.[6] Based on the observations of Rich and McCordock,[6] a 2-step model has been proposed for the pathogenesis of CNS TB. During the initial pulmonary infection, tuberculous bacteria may enter the systemic circulation and subsequently reach the oxygen-rich CNS, establishing a focus called the Rich focus. This focus may be in the meninges, subpial or subependymal

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region of the brain, or the spinal cord. Later, this may rupture into the subarachnoid space or ventricular system leading to meningitis.[7] The probability of the organism reaching the brain depends on the extent of bacteremia and the immune response of the host.[8,9]

Meninges may be secondarily involved because of rupture of a tuberculoma into a vessel in the subarachnoid space, or rupture of miliary tubercles in miliary TB. There can be contiguous spread of infection from the adjacent bone. but this is uncommon. Cell-mediated immunity is responsible for the formation of dense, gelatinous, inflammatory exudate along the basal surface of the cerebrum. Severe cases may show leptomeningeal involvement over the cerebral convexities, and extension into the ventricular system can cause ependymitis and choroid plexitis. Parenchymal tuberculous focus can develop into tuberculoma or brain abscess in the absence of adequate immunity or in the presence of a sizable tuberculous focus.[10–15]

Imaging of CNS TB can be divided into types of involvement, as listed in **Box 1**.

Box 1
Types of CNS TB involvement

Intracranial TB

- Meningeal TB
  - Tuberculous leptomeningitis
  - Pachymeningeal TB
- Complications of TB meningitis (TBM)
  - Hydrocephalus
  - Tuberculous vasculitis
  - Cranial nerve involvement
- Sequel of TBM
- Parenchymal TB
  - Tuberculomas
  - Tuberculous abscess
  - Tuberculous cerebritis
  - Tuberculous encephalopathy

Intraspinal TB

- Typical spinal TB
  - Spondylodiscitis
- Atypical spinal TB
  - Intramedullary TB
  - Solitary vertebral body involvement
  - Pure posterior-element TB
  - Tubercular arachnoiditis

TUBERCULOUS MENINGITIS

Tuberculous Leptomeningitis

After reaching the subarachnoid space, tuberculous focus leads to formation of thick, gelatinous, inflammatory exudate. It affects basal cisterns, sylvian fissures, and, rarely, leptomeninges over cerebral convexities. The exudate in the basal cisterns can cause obstruction to cerebrospinal fluid (CSF) flow, causing hydrocephalus, and can compress cranial nerves. Cerebral infarction can occur because of obliterative vasculitis, the vessels at the base of the brain being severely affected. Granulomas may coalesce to form tuberculomas or, rarely, an abscess.[1,14] Thus, common imaging triad includes abnormal meningeal enhancement predominantly in the basal regions of brain and its associated complications of hydrocephalus and infarcts. This triad is specific for the diagnosis of TBM.[16]

Kumar and colleagues[16] found that the presence of basal enhancement, hydrocephalus, tuberculoma, and infarction were more common in TBM than in children with pyogenic meningitis. They reported that basal enhancement, tuberculomas, or both were 100% specific and 89% sensitive for the diagnosis of TBM.[17] Andronikou and colleagues[17] suggested 9 criteria for the diagnosis of TBM on computed tomography (CT). Przybojewski and colleagues[18] evaluated these 9 criteria and showed high specificity for all the criteria, and 100% specificity for 4 individual criteria. It has been shown that sensitivity has been improved when more than 1 criterion was present.[19] Presence of hyperdensity on precontrast scans in the basal cisterns might be the specific sign of TBM in children.[18]

Magnetic resonance (MR) imaging has been shown to be superior to CT in evaluating patients with suspected meningitis and its associated complications.[20,21] In the early stages, noncontrast MR imaging shows little or no evidence of meningitis. Mild shortening of T1 and T2 relaxation times of CSF occurs as the disease progresses. Contrast-enhanced MR imaging has the added advantage of showing meningitis and its associated complications compared with contrast-enhanced CT and noncontrast MR imaging.[21–24] Abnormal meningeal enhancement is seen in the basal cisterns, and sylvian fissures, and severe and late-stage TBM can show enhancement over the convexities (**Fig. 1**). Tentorial and cerebellar meningeal involvement is less common.[22,25]