Central Nervous System Tuberculosis

JLS Khoo, KY Lau, CM Cheung, TH Tsoi

1Department of Radiology, and 2Department of Medicine, Pamela Youde Nethersole Eastern Hospital, Hong Kong

ABSTRACT

The imaging findings of 9 patients with central nervous system tuberculosis are presented to highlight the typical findings and some complications. Lesions may involve the meninges, brain, and spinal cord. Contrast-enhanced magnetic resonance imaging is the best imaging modality for detection and follow-up of lesions and assessment of complications.

Key Words: Central nervous system, Computed tomography, Magnetic resonance imaging, Meningitis, Tuberculosis

INTRODUCTION

Tuberculosis (TB) is an infectious disease that continues to be a significant public health concern in Hong Kong. According to the statistics published by the Department of Health, there were 7262 notified cases in 2001, which accounted for 28% of the total notifiable infectious diseases, and the number of deaths from TB was reported to be 311. The cumulative number of notifications was 6665 in the year 2002. According to the 2001 Annual Report of the Tuberculosis and Chest Service of the Department of Health, 25 cases of “tuberculosis of the meninges” were reported.

Central nervous system (CNS) TB infection causes a granulomatous inflammatory reaction that involves the CNS meninges and parenchyma. This paper presents the computed tomography (CT) and magnetic resonance imaging (MRI) features of 9 patients with CNS TB managed at the Pamela Youde Nethersole Eastern Hospital during the past 7 years.

PATHOLOGY

In most patients, TB involving the leptomeninges is thought to spread by haematogenous dissemination from a primary source outside the CNS such as the lung or gastrointestinal tract. The meningitic process may affect the cranial cerebrospinal fluid (CSF) pathway, the spinal subarachnoid pathway, or both. CNS involvement by the tuberculous bacilli can also manifest as hard or soft granulomas, tuberculous abscess, tuberculous cerebritis, pachymeningitis, spinal arachnoiditis, and intraspinal tuberculoma.

Hydrocephalus may result from blockage of the basal subarachnoid cisterns by the dense basal exudate, or narrowing of the aqueduct and third ventricle by tuberculomas. Extension of the inflammatory exudate along proliferating blood vessels into the brain substance causes ischaemic brain damage due to vasculitis.

Parenchymal disease usually presents as either solitary or multiple tuberculoma, and can occur with or without meningitis. Tuberculomas are histologically round or oval masses, or they may assume a more lobular configuration with the fusion of several smaller nodules.

A tuberculous granuloma is typically small, measuring approximately 0.5 to 2.0 mm in size. The granuloma consists of epithelioid cells harbouring tuberculous bacilli, with a peripheral collar of fibroblasts and mononuclear inflammatory cells. Langhans’ giant cells, which contain multiple nuclei, is a characteristic feature. A hard tubercle does not show central necrosis, but with cell-mediated delayed hypersensitivity reaction, the core undergoes caseous necrosis, which is a coagulative and liquefactive necrosis resembling cheesy material. This forms the soft tubercle of tuberculosis.
Spinal tuberculous meningitis (STBM) may originate in 3 ways:
- Haematogenous spread from a source outside the CNS
- Caudal extension of intracranial tuberculous basal meningitis
- Intraspinal extension from osseous or discal TB.

**IMAGING FEATURES**

**Cranial Tuberculous Meningitis**

On CT scan, the most common finding in cranial tuberculous meningitis (CTBM) is obliteration of the basal cisterns by isodense or mildly hyperdense exudate. After the administration of contrast medium, there is dense homogeneous enhancement of the basal meninges (Figure 1). Extension of the meningeal enhancement over the surface of the cerebral and cerebellar hemispheres may also be observed. Extension into the ventricular system with ependymitis may be seen as a linear enhancement along the ventricular margin.

Communicating hydrocephalus, the most frequent complication of CTBM, usually caused by obstruction of CSF flow by the meningeal exudate in the basal cisterns, may also be seen at the time of diagnosis. In non-contrast enhanced CT and MRI, hydrocephalus may be the only clue for diagnosis at the initial presentation (Figure 2).

At MRI, although the meningeal inflammation or basal exudate may not be readily apparent on the pre-contrast MR images, post-contrast T1-weighted MR images show diffuse meningeal enhancement, mainly at the basal cisterns (Figure 3).

With extension into the ventricular system, abnormal enhancement of the ependymal lining of the ventricles and the choroid plexi may be seen (Figure 4).
Parenchymal involvement is another complication of CTBM, manifesting as TB cerebritis and granuloma formation (Figure 5). This will be further discussed later in this article, together with parenchymal involvement of the spinal cord.

Vasculitis may be caused by direct invasion of the vessel by mycobacteria, or from extension of adjacent arachnoiditis. These may lead to spasm or thrombosis of the vessels, with resulting infarction, most frequently seen at the basal ganglia and internal capsule. On CT scan, infarctions are demonstrated as areas of
hypodensities, while on MR images, areas of high signal intensity on T2-weighted images are observed. MRI is more sensitive than CT for the demonstration of areas of infarction.\textsuperscript{4,12}

Granulomatous basal meningitis is another complication of intracranial tuberculosis. This is characterised by diffuse or circumscribed granulomatous involvement of the meninges at the skull base.\textsuperscript{4,13} On CT scan, an irregular lumpy enhancing mass can be seen superimposed on the dense basal enhancement. On MR imaging, T1-weighted images reveal a mass isointense to brain, and post-gadolinium scans show intense heterogeneous lumpy enhancement of the basal cisterns (Figure 6).\textsuperscript{3}

**Figure 5.** Tuberculosis causes midbrain signal change in a 25-year-old woman. (a) Axial T1-weighted magnetic resonance imaging after intravenous gadolinium injection reveals multiple tuberculosis granulomas in the right temporal lobe, and meningeal enhancement in the right ambient cistern. (b) Axial T2-weighted magnetic resonance imaging shows hyperintense changes in the right midbrain, which may represent cerebritis or infarction. (c) Follow-up scan performed 19 months later. Axial T2-weighted magnetic resonance imaging shows resolution of the hyperintense signals in the midbrain, suggesting that the signal change was due to cerebritis rather than infarction.

**Figure 6.** Granulomatous tuberculous meningitis, ventriculitis, and spinal arachnoiditis in a 34-year-old man. Sagittal paramedian T1-weighted magnetic resonance imaging after intravenous gadolinium injection reveals irregular lumpy enhancement of the tentorium cerebelli suggestive of granulomatous meningitis. Enhancement of the walls of the lateral ventricle and spinal canal are consistent with ventriculitis and spinal arachnoiditis.
Caseation of the granuloma will produce rim-enhancing lesions. Contrast-enhanced MRI is superb for lesion demonstration and offers the advantage of multiplanar capability (Figure 7). In the chronic phase of CTBM, calcification of the granulomas will be demonstrated as hyperdense foci on non-contrast CT scan, while on MRI, lesions are hypointense on T1-weighted and T2-weighted images due to the calcium deposition (Figure 8).

**Spinal Tuberculous Meningitis**

The unenhanced MR images of spinal tuberculous meningitis (STBM) may appear unremarkable, or may show CSF loculation and obliteration of the spinal subarachnoid space with loss of outline of the spinal cord in the cervico-thoracic spine, and thickening and clumping of the nerve roots in the lumbar spine.³,¹⁴,¹⁵

Gadolinium-enhanced MR images show linear enhancement of the surface of the spinal cord and nerve roots, or plaque-like enhancement of the dura-arachnoid mater complex which obliterates the subarachnoid space (Figures 9 and 10).³,¹⁴,¹⁵

**Parenchymal Tuberculosis in the Brain and Spinal Cord**

Parenchymal TB granulomas or abscesses may be seen in the brain and spinal cord, and may be single or multiple. The changes may be seen in association with leptomeningitis.³,⁴,⁶ Intracranially, parenchymal TB may be in the form of cerebritis and tuberculomas (Figure 11). Tuberculomas may appear in various sizes and locations. They may appear as non-enhancing

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Reference:

or enhancing lesions, as solid enhancing or ring-enhancing lesions, and as disseminated tuberculoma.\textsuperscript{3,4}

A variable degree of vasogenic oedema surrounds the lesions, which is better appreciated on T2-weighted MR images.\textsuperscript{4} Enhancement of the granulomas on the post-gadolinium scans improves their conspicuity and enables their differentiation from the adjacent vasogenic oedema (Figures 12 and 13). Parenchymal tuberculomas can also obstruct the CSF pathways and cause hydrocephalus (Figure 14).\textsuperscript{5}

Tuberculous abscess is an uncommon complication of brain tuberculosis. There is usually irregular perilesional oedema adjacent to the lesion. Dense peripheral ring enhancement of the wall is demonstrated on post-contrast studies (Figure 15).\textsuperscript{4} It may be difficult to differentiate tuberculous abscesses from caseating tuberculomas and pyogenic abscesses, since rim enhancement may be seen in all 3 conditions.\textsuperscript{3,4,6}

The spinal cord may be involved with parenchymal TB myelitis and tuberculoma formation, or as complications of arachnoiditis (e.g. infarction or syrinxomyelia) [Figures 16 and 17].\textsuperscript{3,14}

Contrast-enhanced MRI is superior to CT scanning or unenhanced MRI for the demonstration of CNS TB.\textsuperscript{4}
Figure 10. Spinal tuberculosis meningitis affecting the lumbar spine in a 34-year-old man. (a) Sagittal T1-weighted magnetic resonance imaging of the lower thoracic and lumbar spine shows no abnormality. (b) Sagittal T2-weighted magnetic resonance imaging is also unremarkable. (c) Sagittal and (d) axial enhanced T1-weighted magnetic resonance imaging with fat suppression show intense enhancement of the subarachnoid space indicating arachnoiditis. Loculation of cerebrospinal fluid is demonstrated posteriorly in the lower thoracic spinal canal (arrows).
Figure 11. Caseating tuberculosis granuloma involving the left temporal lobe in a 48-year-old woman. Contrast-enhanced computed tomography scan shows a rim-enhancing lesion in the left temporal lobe consistent with a caseating tuberculosis granuloma.

Figure 12. Tuberculosis granulomas and cerebritis in a 21-year-old woman. (a) Axial T2-weighted magnetic resonance imaging demonstrates a mixed hypo- and hyperintense mass in the left posterior parasagittal region, associated with hyperintense oedema. Other small hyperintense foci are noted in the left periventricular region. (b) Axial T1-weighted magnetic resonance imaging after intravenous gadolinium injection reveals irregular rim enhancement of the caseating tuberculosis granuloma. Other periventricular foci of T2 signal changes shown in Figure 12a, which did not show enhancement, are presumably foci of tuberculous cerebritis.

Figure 13. Miliary cerebral and cerebellar tuberculosis in a 21-year-old woman. Coronal enhanced T1-weighted magnetic resonance imaging reveals intense nodular enhancement in the cerebrum and cerebellum.

Figure 14. Caseating tuberculosis granuloma involving the left thalamus and causing obstructive hydrocephalus in a 21-year-old woman. Coronal T1-weighted magnetic resonance imaging after intravenous gadolinium injection reveals thick irregular rim enhancement of the caseating tuberculosis granuloma compressing onto the third ventricle and causing obstructive hydrocephalus.
Figure 15. Tuberculosis abscess and granulomas in a 21-year-old woman. (a) Axial T2-weighted magnetic resonance imaging reveals a large hypointense left cerebellar lesion with associated oedema. Another small low-signal lesion containing a central dot-like high signal is noted in the right cerebellar hemisphere (arrow), also with surrounding hyperintense oedema. (b) Axial T1-weighted magnetic resonance imaging after intravenous gadolinium injection reveals a uniformly thin smooth wall of enhancement surrounding the large left cerebellar lesion consistent with a tuberculosis abscess, and solid nodular enhancement of several contiguous tuberculosis granulomas. The tiny right cerebellar lesion shows rim enhancement and is consistent with a caseating soft tuberculous granuloma.

Figure 16. Non-caseating tuberculous granulomas involving the cervical spinal cord, with associated oedema, in a 63-year-old man. (a) Sagittal T2-weighted magnetic resonance imaging shows hyperintensity in the cervical spinal cord extending from C2 to C7 levels consistent with oedema. A hypointense nodule representing the granuloma is noted at the C4 level. Underlying cervical spondylotic changes are present. (b) Sagittal T1-weighted magnetic resonance imaging and (c) axial T1-weighted magnetic resonance imaging with fat suppression after intravenous gadolinium injection reveal an area of solid nodular enhancement representing non-caseating tuberculosis granuloma of the spinal cord. A smaller enhancing granuloma is also noted at the C2 level on the sagittal image.
and is currently the best imaging modality for the demonstration of the meningeal disease, parenchymal abnormalities, complications, and for the follow-up of lesions. The multiplanar capability of MRI offers additional advantage for the localisation of lesions (Figure 18).

Other conditions which may give rise to MRI findings similar to cranial and spinal TB include other infectious and non-infectious inflammatory diseases such as fungal infection and sarcoidosis, metastatic tumours (arising within or outside the CNS), and multicentric primary neoplastic disease (such as gliomata).\(^3\)

CONCLUSION

Imaging features of CNS TB have been outlined. MRI with contrast administration is more sensitive than CT scanning or unenhanced MRI for the detection of lesions, and has the advantage of multiplanar capability. Dense basal meningeal enhancement is a typical finding, and complications include hydrocephalus, abscess formation, and vasculitis leading to infarction. TB meningitis and parenchymal tuberculosis are important differential diagnoses when hydrocephalus and multiple enhancing lesions are noted in the CNS. Radiologists and clinicians should be familiar with the imaging features of this disease entity in order to achieve early diagnosis and treatment. Radiological imaging plays an important role in the diagnosis and monitoring of the disease, and for assessment of complications.

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REFERENCES


Figure 17. Spinal tuberculosis meningitis in a 64-year-old woman. (a) Sagittal T1-weighted magnetic resonance imaging after intravenous gadolinium injection shows intense enhancement of the subarachnoid space indicating arachnoiditis. (b) Sagittal T2-weighted magnetic resonance imaging shows hyperintense changes in the cervical spinal cord that did not enhance on the post-contrast study, presumably representing myelitis and associated oedema.
Figure 18. Combined tuberculosis of basal meninges and cerebral parenchyma in a 25-year-old woman. (a) Axial and (b) coronal T1-weighted magnetic resonance imaging after intravenous gadolinium injection shows multiple small right temporal lobe granulomata, and intense meningeal enhancement in the right ambient cistern consistent with leptomeningeal tuberculosis. (c) Contrast-enhanced computed tomography scan and (d) axial T1-weighted magnetic resonance imaging after intravenous gadolinium injection, performed 10 months later for follow-up, show striking nodular enhancing lesions in the right temporal lobe, indicating unsatisfactory response to treatment. Mild linear enhancement in the right ambient cistern is suggestive of partial resolution of the tuberculosis meningitis.