Central Nervous System Mycobacterium Infection
Tuberculosis and Beyond

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KEYWORDS

• Tuberculosis • Central nervous system • Mycobacterium • MR imaging • Computed tomography • Infection

KEY POINTS

- Tuberculosis is the most common mycobacterial infection of central nervous system (CNS) and a global concern in endemic areas, especially among patients living with HIV.
- Leptomeningitis is the most common tuberculosis, with predominant basal meningeal enhancement, hydrocephalus, and cerebral infarction.
- Tuberculomas have four stages, namely non-caseating granuloma, caseating granuloma, caseating granuloma with central liquefaction, and calcified granuloma, each having typical image features.
- Nontuberculous mycobacteria involvement of the CNS, which occurs in disseminated disease, usually presenting as meningitis or meningoencephalitis, is rare but should not be overlooked because of high mortality rates.
- Leprosy is a chronic granulomatous disease with extremely rare CNS involvement.

INTRODUCTION TO MYCOBACTERIUM

There are more than 100 species of mycobacteria, which are small, rod-shaped, acid-fast bacilli. Three groups are important for central nervous system (CNS) infection: (1) *Mycobacterium tuberculosis*, (2) *Mycobacterium leprae*, and (3) a distinct group called nontuberculous mycobacteria (NTM).[1] Among them, *M tuberculosis* is responsible for most mycobacterial infections of the CNS, whereas NTM rarely infects the CNS. Leprosy usually involves the peripheral nervous system, with CNS involvement being extremely rare (**Fig. 1**).

TUBERCULOSIS

Tuberculosis (TB) is a contagious infectious disease caused by *M tuberculosis* and is the leading cause of death from a single infectious agent, estimated to infect a quarter of the world's population.[2] According to the World Health Organization, eight endemic countries account for two-thirds of the global TB infections: India, Indonesia, China, the Philippines, Pakistan, Nigeria, Bangladesh, and South Africa.[2] The risk factors for CNS TB infection include human immunodeficiency virus (HIV) infection, malnutrition, concomitant malignancy, and the use of

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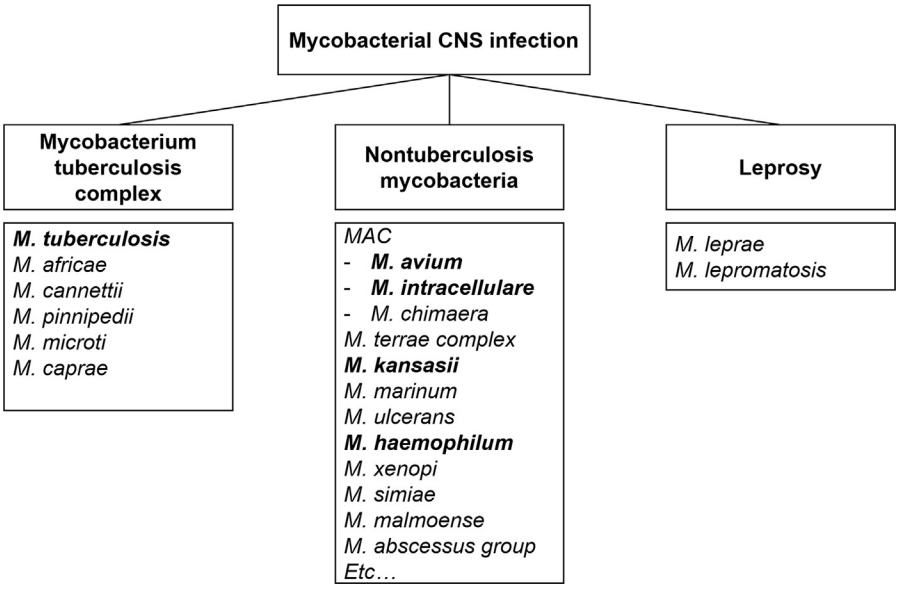

Fig. 1. Mycobacterial species associated with CNS infection.

immunosuppressive drugs; there has been a TB resurgence in nonendemic countries in recent years.[3–6] Moreover, several studies in developed countries have reported that patients born outside of developed countries are overrepresented among CNS TB patients.[7] Therefore, radiologists should be aware of the high incidence of TB and its diagnosis should be suspected with an understanding of the geographic information of pandemic areas and the patient's immune status.

Primarily affecting the respiratory system, TB spreads through the air via droplets containing tubercle bacilli that are released through coughing.[8] Although TB typically manifests as pulmonary infection, it can also affect other body parts, including the CNS. The route of disease spread to the CNS is hematogenous, usually from pulmonary TB. Classically, the rupture of a subpial or subependymal tubercle (known as a rich focus) from hematogenous spread, results in the bacilli entering the subarachnoid space.[9] Because of the lack of defensive white blood cells in the cerebrospinal fluid (CSF), TB can spread rapidly, especially in the basal cisterns.[10]

It may be hard to distinguish acute TB from other infections, and mycobacterium takes a long time to grow in laboratory culture. However, CSF findings can help rule out other pathogens. Typical laboratory results that suggest TB are lymphocytic pleocytosis, low glucose level, high protein level, and high adenosine deaminase level (**Table 1**).[11] Identification of bacilli often requires examination of large volumes of fluid from repeated lumbar punctures. Commercial kits for detecting *M tuberculosis* using genetic amplification technology can also be used: although CSF culture is often negative, polymerase chain reaction assay is positive in 60% to 90% of samples.[12,13] CNS involvement by TB can present either as a diffuse form (leptomeningitis) or as a localized form (tuberculoma, abscess, or cerebritis) (**Table 2**).

CRANIAL TUBERCULOSIS

Tuberculous Meningitis

TB leptomeningitis is the most common type of TB affecting the CNS. An inflammatory reaction with a

Table 1
Typical abnormal laboratory results of CNS TB

CSF analysis	Lymphocytic pleocytosis (50–500 cell/mm$^{3)}$ High protein level (0.5–3.0 g/L) Low glucose level (≤45 mg/dL or ≤40%–50% of serum glucose) High lactate level (5.0–10.0 mmol/L) High adenosine deaminase level (≥8 u/L)
CSF culture	Identification of AFB on stain and culture
CSF PCR	TB PCR positive (amplification of the MPB64 gene or IS6110)

Abbreviation: PCR, polymerase chain reaction.

Table 2
Types of CNS TB involvement

Location	Common	Less Common	Complications	Others
Intracranial	• Leptomeningitis • Vasculitis • Tuberculoma • Abscess	• Cerebritis • Miliary TB • Pachymeningitis • Ventriculitis	• Hydrocephalus • Acute infarction • Cranial nerve palsy	• Tuberculous encephalopathy • Paradoxic response
Intraspinal	• Spondylitis • Myelitis	• Meningitis • Intramedullary tuberculoma		• Longitudinally extensive transverse myelitis

variable admixture of exudative, proliferative, and necrotizing components in the subarachnoid cisterns is typical.[10] High viscosity and high-protein exudates are commonly found in the basal cisterns in TB leptomeningitis, and this may cause communicating hydrocephalus by obstructing CSF circulation.[10] The clinical symptoms include prodromal headache, fever, vomiting, and neck stiffness, followed by vomiting, confusion, and focal neurologic signs.[14] Evidence of concomitant extrameningeal TB is present in about three-fourths of cases.[15] In many cases, however, there is no clinical or historical clues to suggest TB. Radiologically, meningeal involvement is most pronounced at the basal cistern with involvement of the suprasellar/chiasmatic region, ambient cistern, and interpeduncular fossa.

Neuroimaging shows a thick basal meningeal enhancement, hydrocephalus, and small parenchymal infarctions, which are highly specific for TB meningitis (**Table 3**).[10,16] Computed tomography (CT) often presents with nonspecific hydrocephalus and isodense to mildly hyperdense basal and sulcal cisterns may be seen. On MR imaging, abnormal basal CSF exudates show T1 isointense signal intensity to the brain parenchyma. Fluid-attenuated inversion recovery (FLAIR) images show increased signal intensity in the sulci and cisterns when there is high CSF protein concentration.[17,18] After contrast injection, characteristic thick leptomeningeal enhancement is seen, predominantly in the basal cisterns; this can be the only imaging finding of TB CNS infection (**Fig. 2**).[19] Basal enhancement reflects microabscess formation and intense inflammation in the basal meninges and may indicate poor outcome.[20] In the evaluation of leptomeningitis, contrast-enhanced FLAIR sequence may show high sensitivity for detecting meningeal enhancement (**Fig. 3**).[21,22]

Basal enhancement in TB leptomeningitis may present differently in patients with and without HIV, with positive meningeal enhancement or enhanced meningeal nodules more frequently observed on contrast-enhanced CT scan in patients with HIV than in patients without HIV.[23,24] However, some studies have shown little or no meningeal enhancement or basal exudates on CT or MR imaging in patients with HIV, with the suggested mechanism including an impaired immunologic response.[17] However, the diagnostic sensitivity and specificity of various imaging features for TB leptomeningitis are undefined. Furthermore, approximately 30% of individuals at an early stage in TB leptomeningitis may have normal brain CT findings, and around 15% have normal brain MR imaging findings.[14]

Hydrocephalus is the most common complication of TB leptomeningitis, and may indicate poor prognosis with high morbidity and mortality.[25] In

Table 3
Summary of TB leptomeningitis

	Clinical Symptoms	Imaging Finding	Recommended Protocols
Leptomeningitis	Prodromal headache, fever, vomiting and neck stiffness, vomiting, confusion	• Thick basal meningeal enhancement • Hydrocephalus • Small parenchymal infarctions • Cranial nerve enhancement/thickening	Precontrast/ postcontrast-enhanced T1 and FLAIR images

Abbreviation: FLAIR, fluid-attenuated inversion recovery.

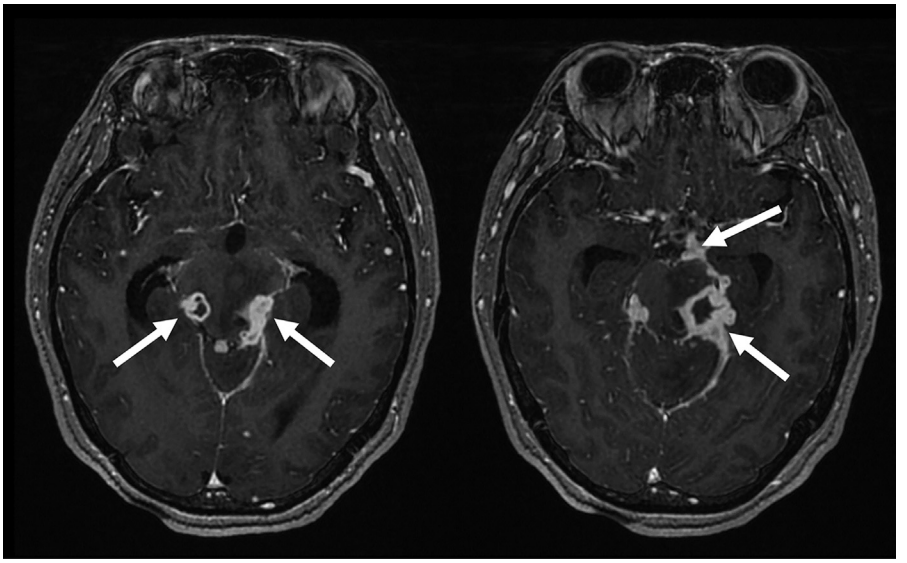

Fig. 2. Tuberculous meningitis. Axial T1-weighted postcontrast MR imaging shows thick nodular meningeal enhancement at the basal cisterns with abscess formation (*arrows*).

addition to the dilatation of lateral ventricles, periventricular T2 hyperintensity may be seen on MR imaging as a sign of transependymal edema (**Fig. 4**).[10] Although hydrocephalus may also occur transiently in bacterial meningitis, radiologists should consider the possibility of a TB cause whenever hydrocephalus is prominent.[26]

Cranial nerve involvement is reported in 17% to 40% of TB meningitis, especially cranial nerve (CN) II, III, IV, VI, and VII, considering their proximal location in the basal cistern. It occurs because of ischemic changes in the nerve or entrapment of the nerve in the basal exudates.[27] Clinically, multiple cranial nerve palsies may occur; MR imaging shows diffuse thickening and enhancement of the involved cranial nerve, usually in their cisternal segments (**Fig. 5**).[28]

Cerebral Infarction and Vasculitis

Cerebral infarction is reported in 28% to 45% of patients with TB leptomeningitis.[29–31] The inflammatory exudate in the basal cistern can involve the perivascular space and adventitia of the perforator vessels, progressing to panarteritis of the vessel wall with secondary occlusion.[32,33]

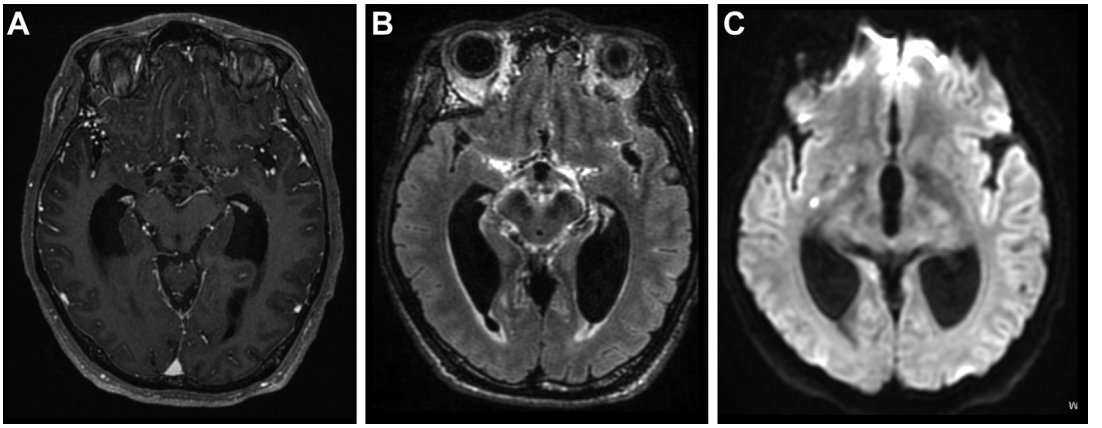

Fig. 3. Tuberculous meningitis. Axial T1-weighted postcontrast MR imaging shows subtle faint meningeal enhancement at the basal cisterns with hydrocephalus (*A*). Corresponding axial postcontrast FLAIR image shows much more prominent basal cisternal enhancement (*B*). A tiny acute infarction at the right lateral basal ganglia is also noted on diffusion-weighted imaging (DWI) (*C*).

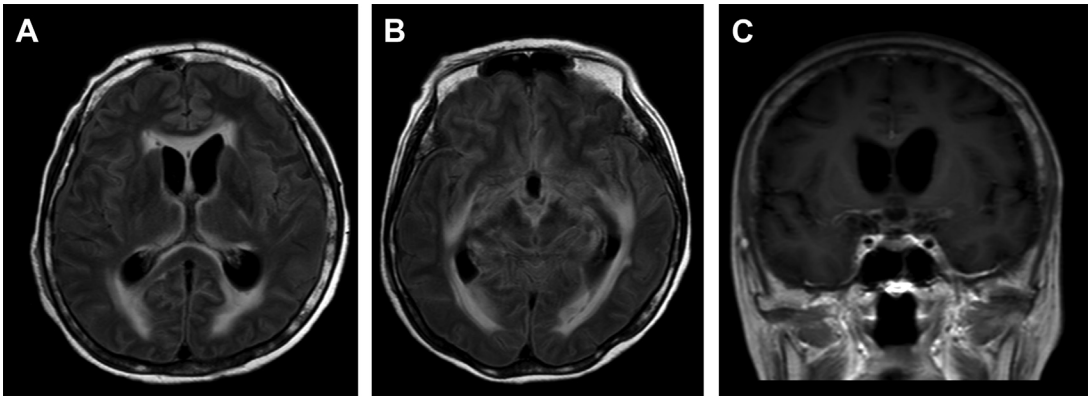

Fig. 4. Tuberculous meningitis with secondary hydrocephalus. An axial FLAIR image shows the diffuse dilatation of the bilateral lateral ventricle with periventricular hyperintensity from transependymal edema (*A, B*). Axial T1-weighted postcontrast MR imaging shows basal cisternal enhancement (*C*).

Vascular involvement of TB leptomeningitis may cause secondary vasculitis.[27,29,34] TB vasculitis may cause secondary cerebral infarction and most of the infarcts are found as small, multiple, and bilateral lesions in the basal ganglia, thalamus, and internal capsule, where the vascular territories of the basal perforators originate from the M1 segment of middle cerebral artery and from the basilar artery.[31] MR imaging patterns of acute infarction in TB typically exhibit multiple, bilateral diffusion restrictive, FLAIR high signal intense lesions, located often in the basal ganglia and the anterior thalamus (**Fig. 6**).[27,35,36]

On angiography, irregular beaded narrowing of the supraclinoid internal carotid artery and pericallosal artery, and a delayed circulation in the middle cerebral vein with a reduced collateral circulation are reported (**Table 4**).[37] Magnetic resonance angiography (MRA) may also demonstrate abnormalities, commonly in the middle cerebral artery and the posterior cerebral artery.[38,39] Patients with baseline MRA abnormalities may have a greater chance of developing infarctions than those with normal MRA results. The recently introduced high-resolution vessel wall imaging has demonstrated contrast enhancement in the involved arterial wall with predominant nodular and smooth enhancement (**Fig. 7**).[40]

Tuberculoma

Tuberculomas occur because of conglomeration and coalescence of microgranulomas, and are the second most common CNS manifestations of

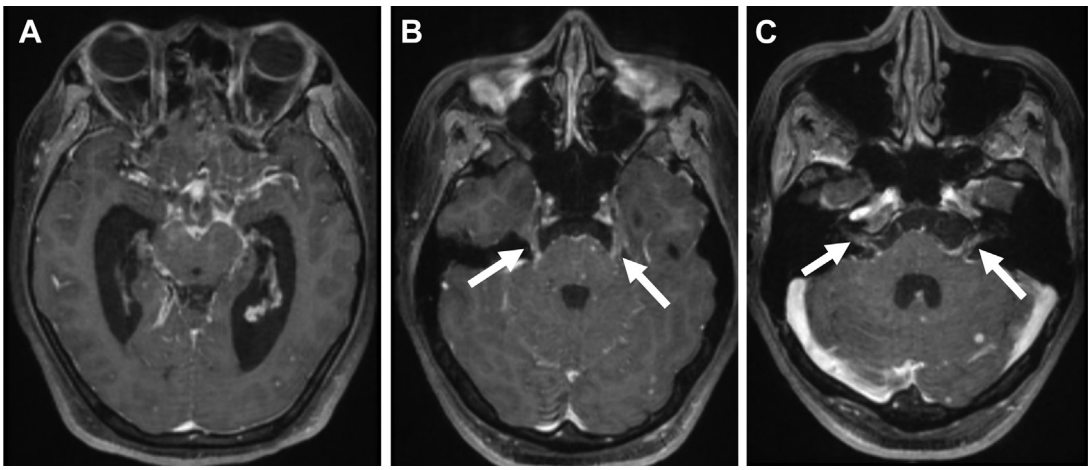

Fig. 5. Tuberculous meningitis with cranial nerve involvement. Axial T1-weighted postcontrast MR images show thick basal cisternal enhancement with hydrocephalus (*A*), diffuse thick enhancement along the cranial nerve V (*B*), and VII/VIII complexes (*arrows*) (*C*).

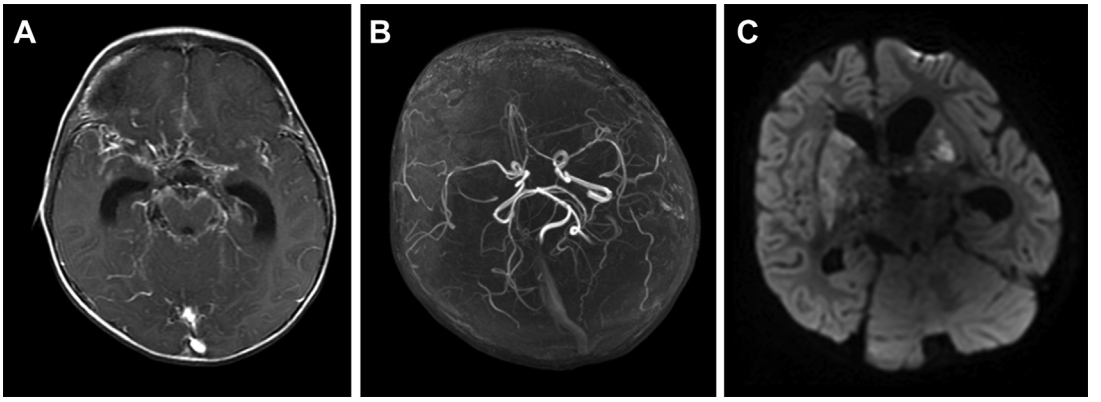

Fig. 6. Tuberculous meningitis with secondary acute infarction. Axial T1-weighted postcontrast MR imaging shows extensive enhancement in the basal cisterns (*A*). On time-of-flight MR angiography, there is luminal irregularity in the bilateral middle cerebral artery (MCA) (*B*). Axial DWI shows multifocal acute infarctions in the bilateral basal ganglia and thalamus (*C*).

TB after leptomenigitis.[10,35] They are mostly found at the gray matter and white matter junction as the hematogenous spreading microbes are captured in the distal arteries (Fig. 8).[10] A small number of tuberculomas can also gain entry into the brain parenchyma via the perivascular space around small penetrating arteries, from direct extension of leptomeningitis.[10,32] Tuberculomas are more common in children and are predominantly located in the infratentorial region, whereas in adults they tend to be predominantly supratentorial.[41] Tuberculomas can vary in size, but most are small, and the miliary nodules are often a few millimeters in diameter (discussed later). Tuberculomas typically pass through four different stages: (1) noncaseating granuloma, (2) solid caseating granuloma, (3) caseating granuloma with central liquefaction, and (4) calcified granuloma. Pathologically, tuberculoma consist of a typical granuloma with epithelioid cells, Langerhans giant cells, a peripheral rim of lymphocytes surrounding central caseous necrosis, followed by liquefaction.[42] Tuberculomas may resolve completely, or form calcified granulomas.

Imaging findings of tuberculomas depend on their stage (Table 5). Noncaseating tuberculomas typically present as homogeneously enhancing nodular lesions with low signal on T1-weighted images and high signal intensity on T2-weighted images, usually surrounded by vasogenic edema (Fig. 9).[10,35] Solid caseating tuberculoma showed ring enhancement with central heterogeneous enhancement with hypointensity/isointensity on T1-weighted images (Fig. 10).[10,35] Sold caseating granulomas may show slightly hypointense T2 signal intensity because of the paramagnetic free radicals released from the inflammatory cells, with a combination of fibrosis and gliosis.[43,44] When caseating granuloma show liquefaction, the central enhancement disappears, leaving irregular, thick ring enhancement with central hyperintensity on T2-weighted images,[35] and

Table 4
Summary of TB vasculitis

	Clinical Symptoms	Imaging Finding	Recommended Protocols
Vasculitis	Focal neurologic signs	• Small, multiple acute infarction in the basal ganglia, thalamus, and internal capsule • Irregular beaded narrowing of distal ICA, proximal MCA, and ACA • Nodular and smooth vessel wall enhancement	DWIs, intracranial TOF MRA or CTA, high-resolution vessel wall MR imaging

Abbreviations: ACA, anterior cerebral artery; CTA, CT angiography; DWI, diffusion-weighted imaging; ICA, internal carotid artery; MCA, middle cerebral artery; MRA, magnetic resonance angiography; TOF, time of flight.

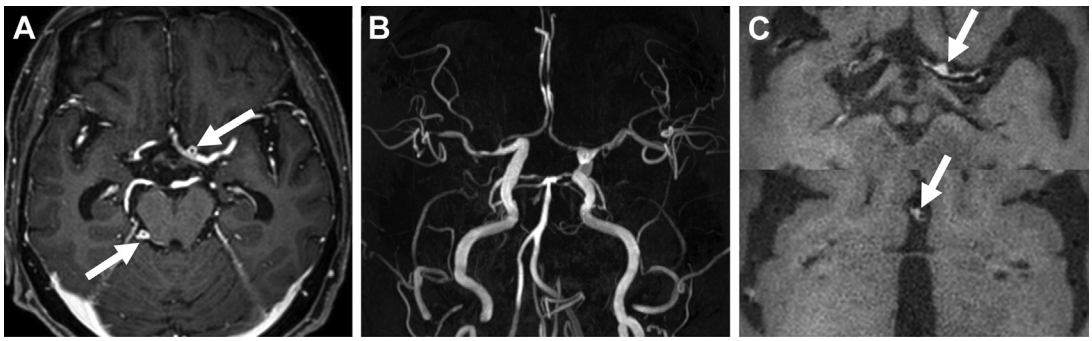

Fig. 7. Tuberculous meningitis with secondary vasculitis. Axial T1-weighted postcontrast MR imaging shows a few rim-enhancing nodular lesions in the basal cisterns (*arrows*) (*A*). Irregular narrowing of the bilateral anterior cerebral artery (ACA), middle cerebral artery (MCA), and posterior cerebral artery (PCA) is seen on MRA (*B*). High-resolution vessel wall imaging (*C*) reveals nodular thick enhancement along the left MCA and bilateral ACA (*arrows*).

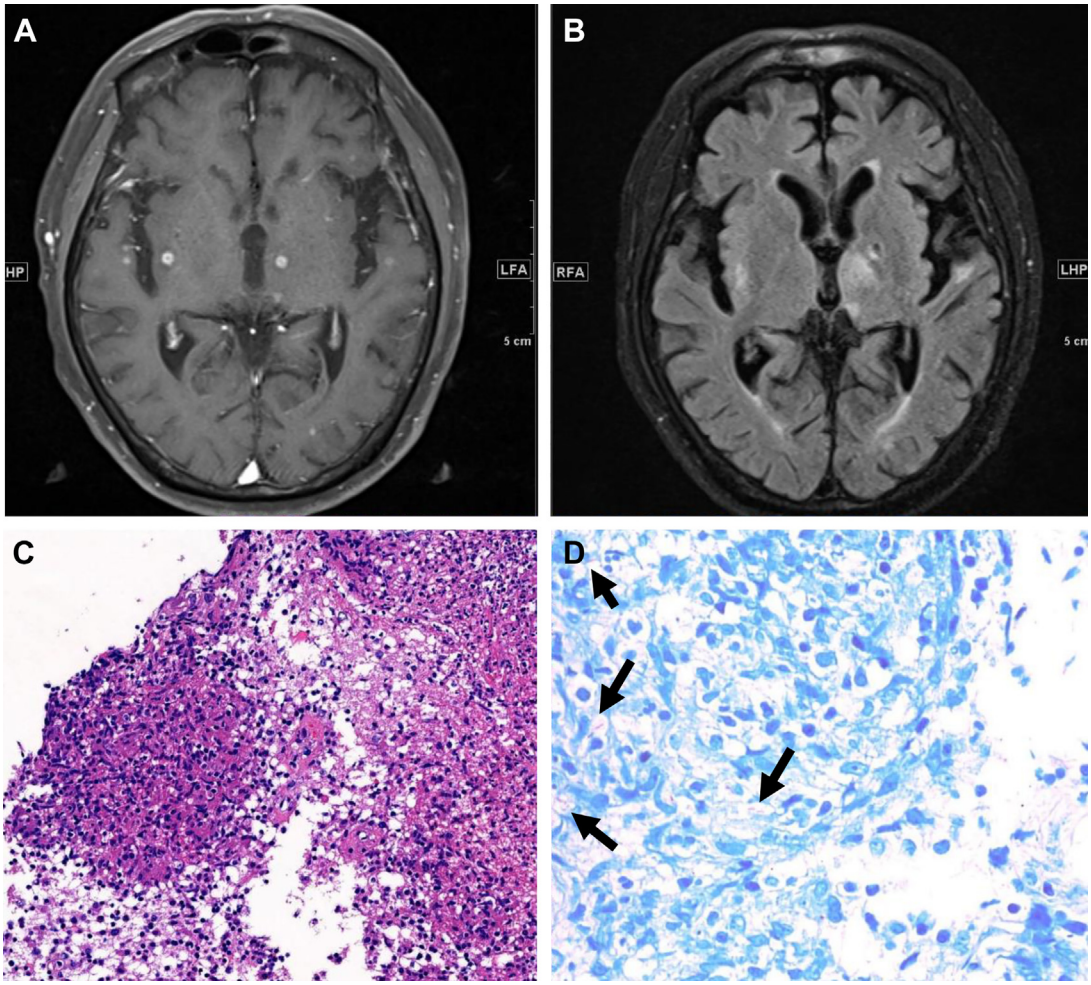

Fig. 8. Noncaseating tuberculoma with histopathologic findings. Axial T1-weighted postcontrast MR imaging and unenhanced FLAIR MR imaging show several rim-enhancing lesions in the right basal ganglia, left thalamus, and bilateral temporo-occipital lobe (*A*, *B*). The lesions were surgically resected. (*C*) Granulomatous inflammation with necrosis and suppurative inflammation (hematoxylin-eosin, original magnification ×200). (*D*) Ziehl-Neelsen staining reveals a few acid-fast bacilli under higher magnification (*arrows*) (original magnification ×600).

Table 5
Stage of tuberculomas with histopathologic/MR imaging appearance

Stage	T1-Weighted/ T2-Weighted Images	Contrast-Enhancement	DWI
Noncaseating granuloma	T1: hypointense T2: hyperintense	Homogeneous enhancement	No diffusion restriction
Caseating granuloma	T1: isointense/ hypointense T2: hypointensity	Ring enhancement with central heterogeneous enhancement	No diffusion restriction
Caseating granuloma with central liquefaction	T1: isointense to hypointense T2: central hyperintensity	Ring enhancement	Diffusion restriction (+/–)
Calcified granuloma	T2: dark signal intensity Blooming artifact (+)	No enhancement	No diffusion restriction

restricted diffusion on diffusion-weighted imaging because of high viscosity; unlike pyogenic abscess, there is less edema (Fig. 11).[35] Magnetization transfer (MT) imaging has demonstrated higher MT ratio in the nonenhancing cores of TB than in abscesses, likely related to their higher cellularity.[45]

Tubercular Abscess

CNS involvement of TB may rarely present as a true pyogenic abscess; they can occur in less than 10% of patients with CNS TB.[46] TB abscess is characterized by cavity formation with central necrosis, containing more bacilli, macrophages, liquefied necrotic debris, and pus than caseating granuloma.[47] Imaging finding of TB abscess includes central T1 hypointensity and T2 hyperintensity with central diffusion restriction and rim enhancement, which is nonspecific, making it difficult to differentiate it from pyogenic abscesses. Compared with TB granulomas, TB abscesses are larger in size, and show a multiloculated thin-walled lesion (Fig. 12, see also Table 5),[27,48] with a lower MT ratio than tuberculoma. MR spectroscopy may help in differential diagnosis, with a high lipid peak in TB abscess.[48]

Miliary Tuberculosis

Miliary TB are mostly seen in immunocompromised patients. They are usually accompanied by extracranial and meningeal involvement of TB.[49] Miliary TB show innumerable scattered foci in the brain parenchyma, predominantly located at the gray matter and white matter junction because of hematogenous spread, similar to tuberculomas. On MR imaging, numerous, small noncaseating granulomas are scattered at the gray-white matter junction with T2 hypointensity and homogeneous enhancement (Fig. 13).[50]

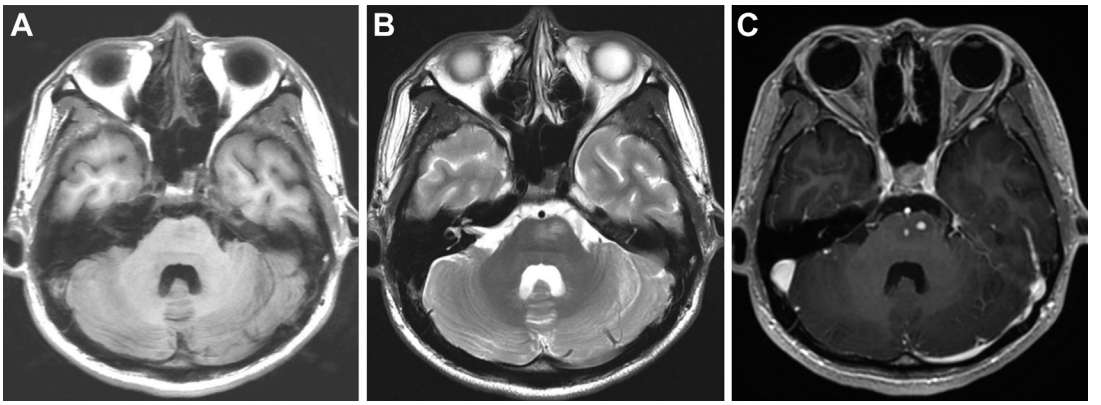

Fig. 9. Noncaseating tuberculoma. Axial T1-weighted MR imaging (*A*) shows a hypointense lesion in the ventral pons. Corresponding axial T2-weighted MR imaging (*B*) shows infiltrative high signal intensity, with homogeneous contrast enhancement on axial T1-weighted postcontrast MR imaging (*C*).

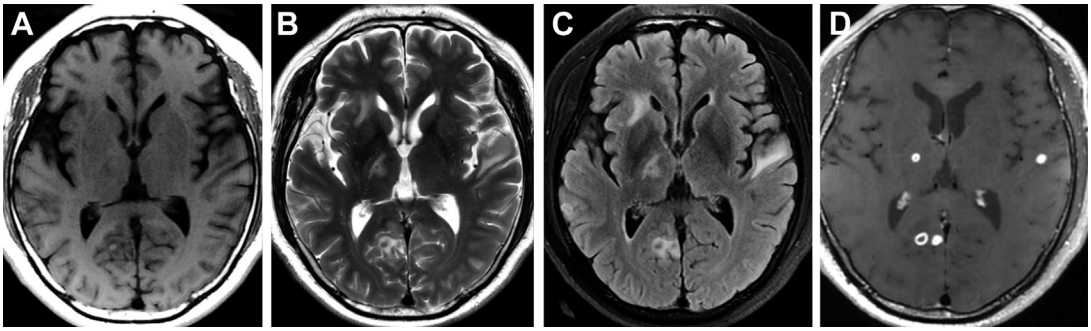

Fig. 10. Solid caseating tuberculoma. Multiple small nodular lesions are scattered in the right thalamus, right occipital lobe, and left frontal operculum. Axial T1-weighted MR imaging (*A*) shows hyposignal or isosignal intensity, whereas axial T2-weighted (*B*) and FLAIR (*C*) images shows central hyposignal intensity surrounded by high signal. There is solid or rim enhancement on axial T1-weighted postcontrast MR imaging (*D*).

Tuberculosis Cerebritis

TB cerebritis is a rare presentation involving focal brain parenchyma reported in patients without HIV infection, with or without concurrent leptomeningitis.[27] On MR imaging, parenchymal swelling with T1 hypointensities and T2 hyperintensities is noted with patchy contrast enhancement.[28] Histopathologically, it is composed of tubercular microgranulomata with scarce tubercular bacilli and without the associated caseous necrosis.

Tuberculosis Encephalopathy

TB encephalopathy is typically found in children with clinical symptoms of seizures and altered mental status.[42] It may be associated with type IV hypersensitivity, in which patients are reactive to a tuberculin protein.[37] Histopathologic examination reveals extensive white matter injury and perivascular demyelination, but without invasion by tuberculous bacilli. On imaging, TB encephalopathy presents severe unilateral or bilateral cerebral edema with T2 hyperintensity in the white matter without meningeal involvement (**Fig. 14**).[51] Diffuse contrast enhancement of the involved white matter may be observed.[52,53] Acute disseminated encephalomyelitis is an important differential diagnosis of TB encephalopathy.

Tuberculosis Pachymeningitis

Pachymeningitis in CNS TB is not commonly observed, and it usually occurs secondary to acute or chronic leptomeningitis. TB pachymeningitis is localized or in a diffuse form. On MR imaging, it is characterized by focal or diffuse dural thickening with prominent contrast enhancement and a FLAIR sequence may be helpful for detecting dural thickening.[54] Isolated TB pachymeningitis is difficult to differentiate from other types of pachymeningitis, such as

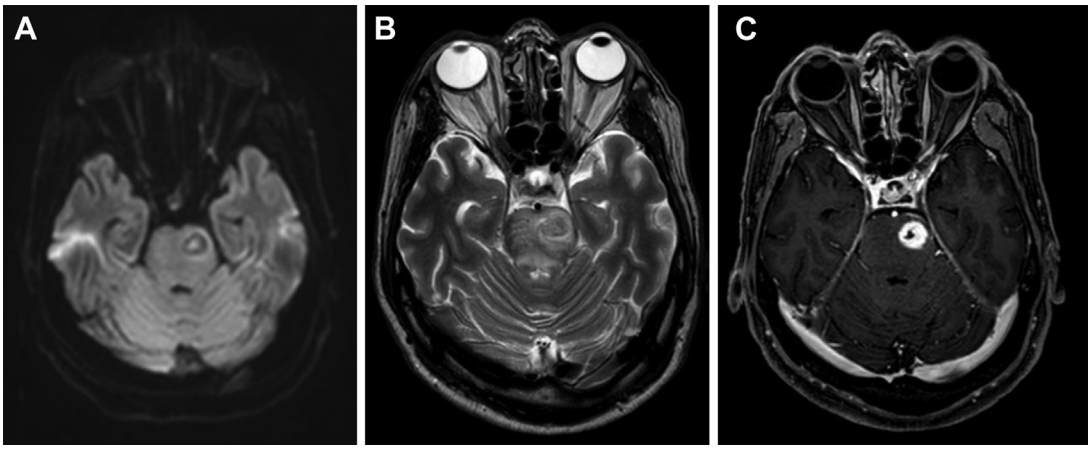

Fig. 11. Caseating granuloma with central necrosis. Axial DWI (*A*) shows a centrally diffusion-restrictive lesion in the left side pons with mixed T2 high signal intensity (*B*) and irregular thick rim enhancement (*C*).

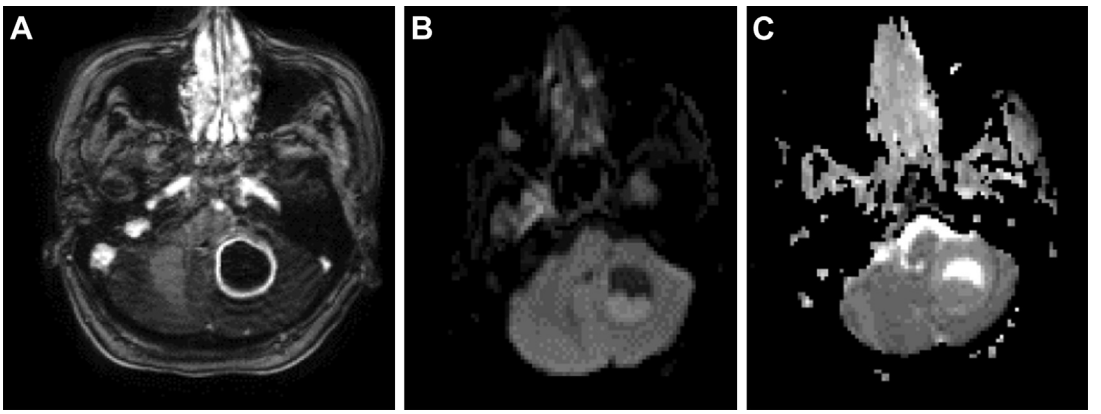

Fig. 12. Tuberculous abscess. A large, solitary abscess shows thin rim enhancement on axial T1-weighted postcontrast MR imaging (*A*) with central diffusion restriction on DWI (*B*, *C*).

neurosarcoid, neurosyphilis, and idiopathic hypertrophied pachymeningitis.[42]

Tuberculosis Ventriculitis

Subependymal TB focus may rupture into the ventricles or late-stage TB leptomeningitis can spread retrogradely to the ventricles. On MR imaging, CSF in the ventricles shows heterogeneous T1 signal intensity with diffuse thickening and enhancement along the ependymal lining (**Fig. 15**).[55] It is usually associated with hydrocephalus.

SPINAL TUBERCULOSIS

Tuberculosis Spondylitis

TB spondylitis is the most common presentation of skeletal infection of TB. TB spondylitis results from hematogenous spread of disease to the vertebral body through paravertebral venous plexus. TB spondylitis usually presents as multiple vertebral body involvement with intervertebral disk sparing in the early stage because mycobacteria are unable to degrade the collagenous annulus of the disk because of lack of proteolytic enzymes (**Table 6**).[56] TB spondylitis commonly shows paraspinal soft tissue extension and paravertebral abscess formation is also often detected.[28] There is a predilection of TB for the anterior vertebral body.[57] Severe complications of vertebral collapse resulting in kyphosis and cord compression is observed.[56]

Imaging findings of TB spondylitis show bone edema with low signal intensity on T1-weighted and hyperintense on T2-weighted/STIR images with contrast enhancement in its early stage (**Fig. 16**).[27] Later, extensive bony destruction of vertebral body becomes evident typically involving three or more vertebral levels of subligamental and paravertebral soft tissue enhancement or abscess formation (**Fig. 17**).[56,58] A well-defined paraspinal abnormal signal intensity and thin, smooth,

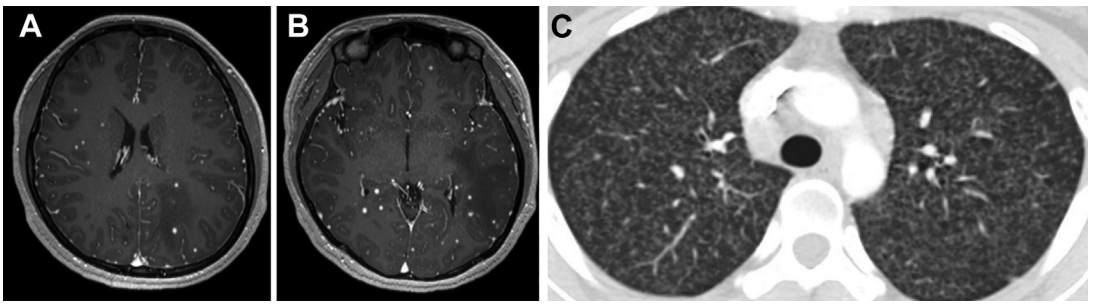

Fig. 13. Miliary TB. Axial T1-weighted postcontrast MR imaging show innumerable tiny enhancing foci scattered in the gray and white matter junction of the brain (*A*, *B*). Chest CT also shows diffuse miliary micronodules distributed throughout both lungs (*C*).

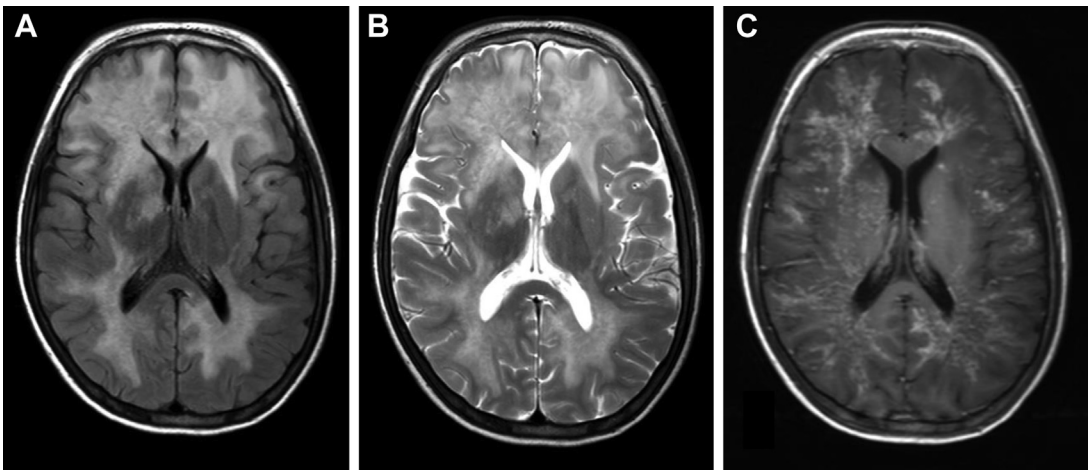

Fig. 14. Tuberculous encephalopathy. Axial FLAIR (*A*) and T2-weighted images (*B*) show severe symmetric diffuse white matter edema. Axial T1-weighted postcontrast MR imaging shows diffuse white matter contrast enhancement (*C*).

enhancing paraspinal and intraosseous abscess are also well-known imaging findings of TB spondylitis (**Fig. 18**).[58,59]

Tuberculosis Spinal Meningitis

Infrequently, TB causes spinal meningitis from cranial extension or from extension of adjacent TB spondylitis as a gelatinous granulomatous exudate that fills the subarachnoid space, causing focal spinal cord and nerve root inflammation.[10,35] In chronic cases, the exudate produces adhesions and fibrosis. Similar to cranial leptomeningitis, spinal meningitis can also cause local vasculitis and focal segmental infarction.[10,60] Imaging findings of spinal meningitis include spinal subarachnoid space loculations/obliteration, nerve root thickening or clumping, combined with irregular meningeal or nerve root enhancement (**Fig. 19**).[61] In the late stage of the disease, syringomyelia can occur

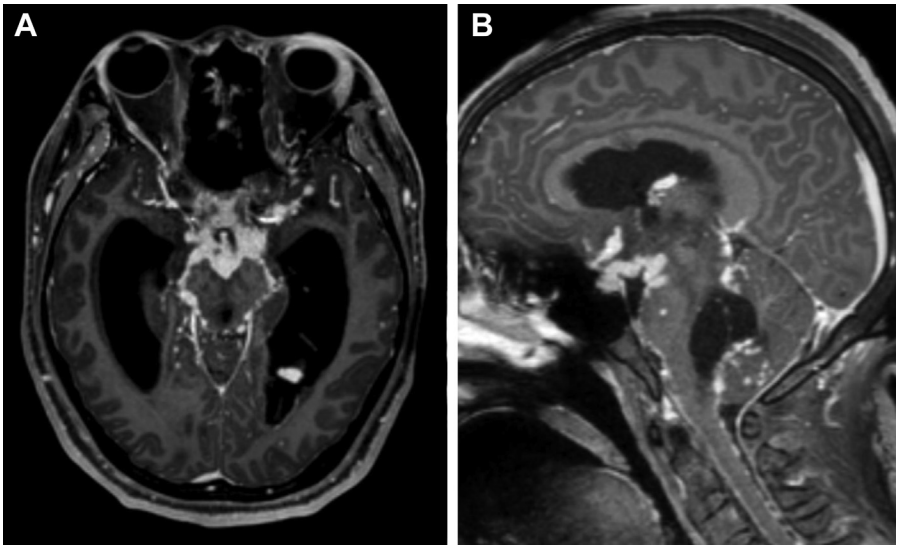

Fig. 15. Tuberculous ventriculitis and meningitis. Axial T1-weighted postcontrast MR imaging shows diffuse extensive thick enhancement of the basal cisterns that extends to the occipital horn of the left lateral ventricle and secondary hydrocephalus (*A*). Sagittal T1-weighted postcontrast MR imaging shows involvement of the suprasellar cistern, inferior ependymal lining of the fourth ventricle, and dorsal surface of the upper cervical spinal cord (*B*).

Table 6
Summary of TB spondylitis

	Clinical Symptoms	Imaging Finding	Recommended Protocols
Spondylitis	Back pain, stiffness, lower limb weakness, paraplegia	• 3 or more vertebral bodies involvement sparing intervertebral disks • Bone marrow edema with contrast enhancement (early phase) • Paraspinal and paravertebral soft tissue/abscess formation • Vertebral body collapse	Precontrast/postcontrast-enhanced T1 T2-weighted and fat suppressed T2-weighted

secondary to chronic arachnoiditis and appears as cystic central canal dilatation on MR imaging (**Fig. 20**).[10]

Tuberculosis Myelitis

TB myelitis is the second most common involvement of the spine, after spondylitis. In most cases, it appears in individuals younger than the age of 30, and involves intracranial TB. Clinical symptoms may vary from radicular pain to weakness. The thoracic spinal cord is most commonly affected, followed by lumbar and cervical cord. An affected spinal cord appears hyperintense on T2-weighted images and hypointense or isointense on T1-weighted images, with or without cord expansion.[61] Obliteration of spinal subarachnoid space, clumping nerve roots, and nodular intradural enhancement may be seen when meningitis is also present (**Fig. 21**).[62] Rarely, longitudinally extensive transverse myelitis as contiguous immune-mediated inflammatory lesion has been reported without pathologic evidence of active TB invasion of the spinal cord.[63,64]

Tuberculosis Intramedullary Tuberculoma

TB intramedullary spinal tuberculoma is a rare neurologic manifestation of TB and usually arises from hematogenous dissemination.[10] The most

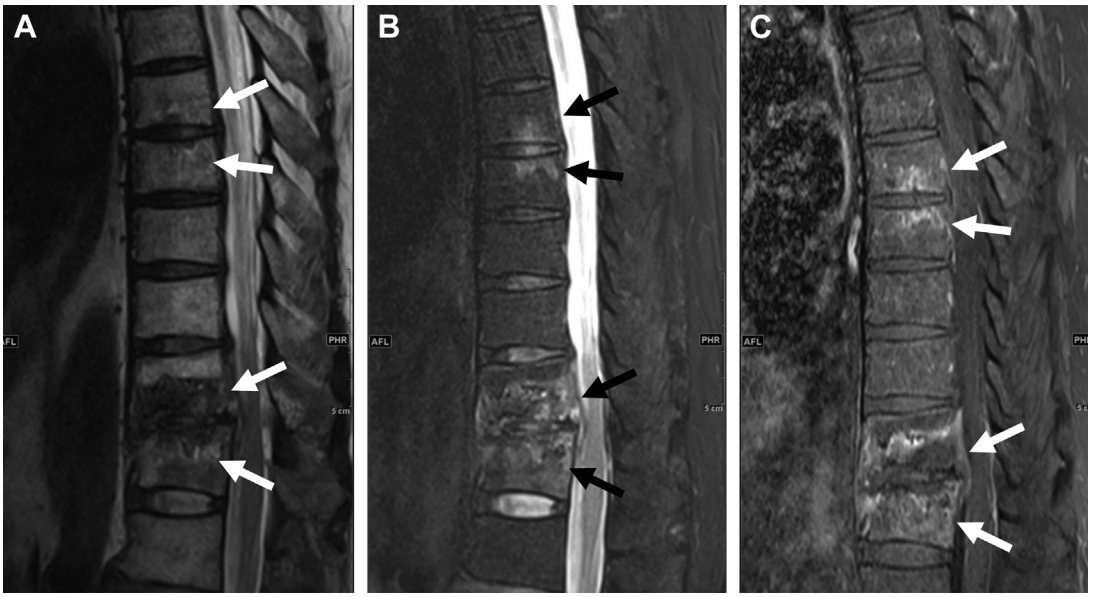

Fig. 16. Early stage of tuberculous spondylitis. Sagittal T2-weighted (*A*) and fat-suppressed T2-weighted MR imaging (*B*) show high signal bone marrow edema in the T7, T8, T11, and T12 vertebral bodies (*arrows*), skipping the intervertebral disks and intervening normal segments. (*C*) The lesions show enhancement on sagittal T1-weighted postcontrast MR imaging.

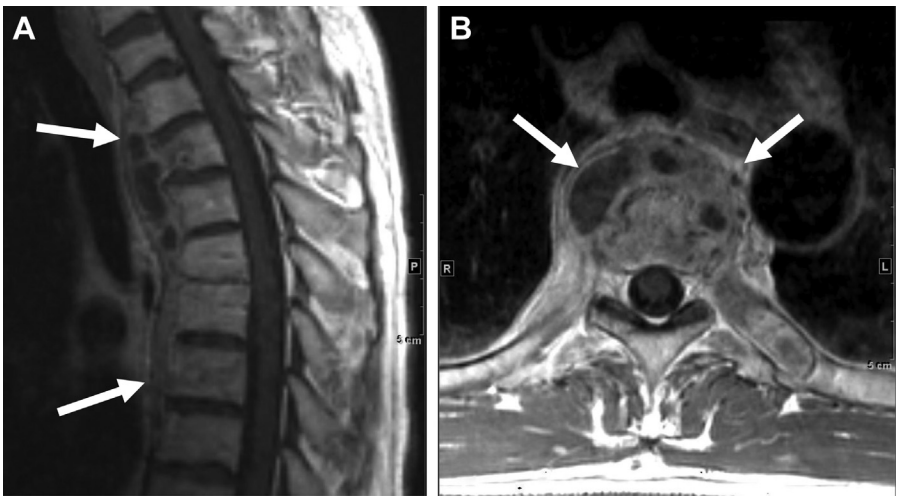

Fig. 17. Tuberculous spondylitis. Sagittal (*A*) and axial T1-weighted postcontrast MR imaging (*B*) show subligamentous extension of the enhancing prevertebral abscess (*arrows*) along the anterior longitudinal ligament at multiple vertebral levels.

common symptom is motor and sensory deficit, depending on the location of the lesion.[37,65,66] The MR imaging findings are similar to the characteristic appearance of intracranial tuberculomas, usually showing focal rim enhancement on postcontrast study.[67]

Paradoxic Reaction

Paradoxic reaction is defined as paradoxic clinical and/or radiologic deterioration after effective TB treatment initiation, and occurs because of excessive inflammatory response against mycobacterial antigens.[68,69] It is common in HIV-infected and HIV-uninfected patients with CNS TB and approximately one-third of patients with TB leptomeningitis undergo paradoxic reaction.[69] Mycobacterial cell wall antigens are present in the affected brain tissues and trigger an exaggerated inflammatory reaction following treatment.[69] Patients exhibit aggravated clinical symptoms and imaging findings, such as basal cisternal enhancement, increased tuberculoma, development of infarctions, and worsening ventriculomegaly (**Fig. 22**).[69]

NONTUBERCULOUS MYCOBACTERIAL INFECTION

NTM are ubiquitous microorganisms that are widely distributed in water and soil.[70] CNS

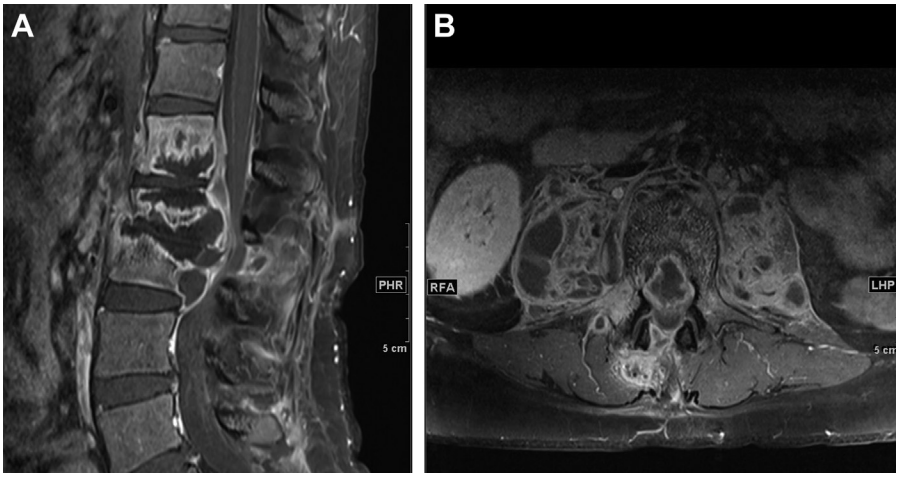

Fig. 18. Tuberculous spondylitis with abscess formation. Sagittal (*A*) and axial T1-weighted postcontrast MR imaging (*B*) show well-defined thin, rim-enhancing paraspinal, intraosseous, and epidural abscess with nonenhancing central cavity. There is also bilateral psoas abscess formation.

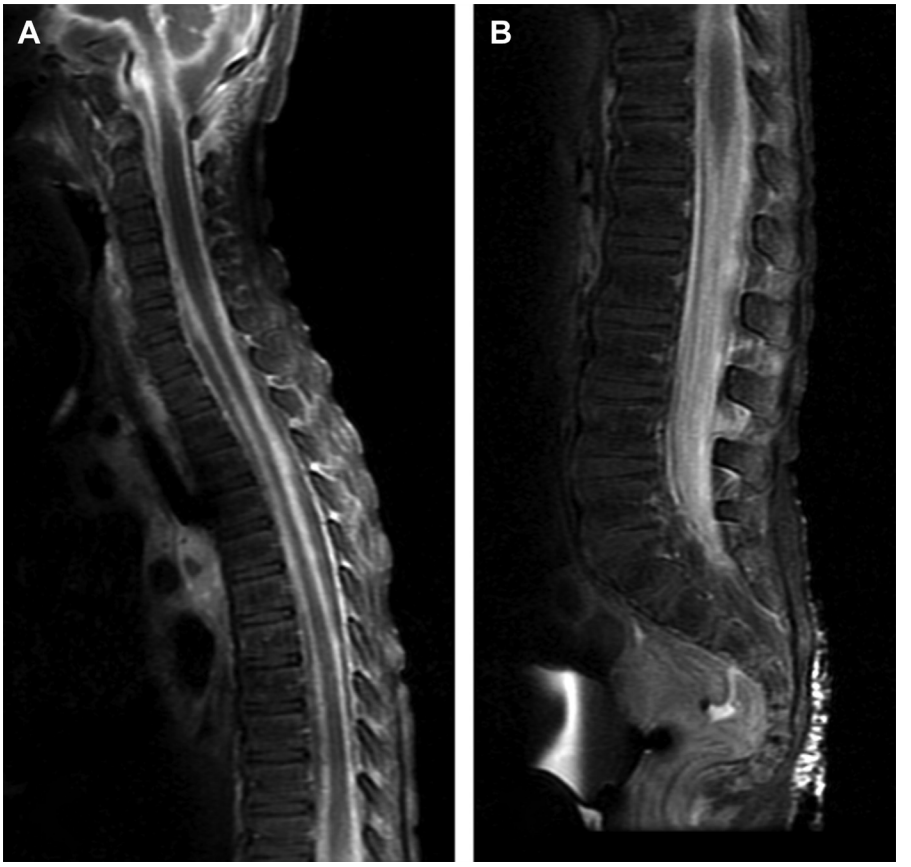

Fig. 19. Tuberculous spinal meningitis. Sagittal T1-weighted postcontrast (*A,B*) imaging shows extensive diffuse, thick meningeal and cauda equina enhancement.

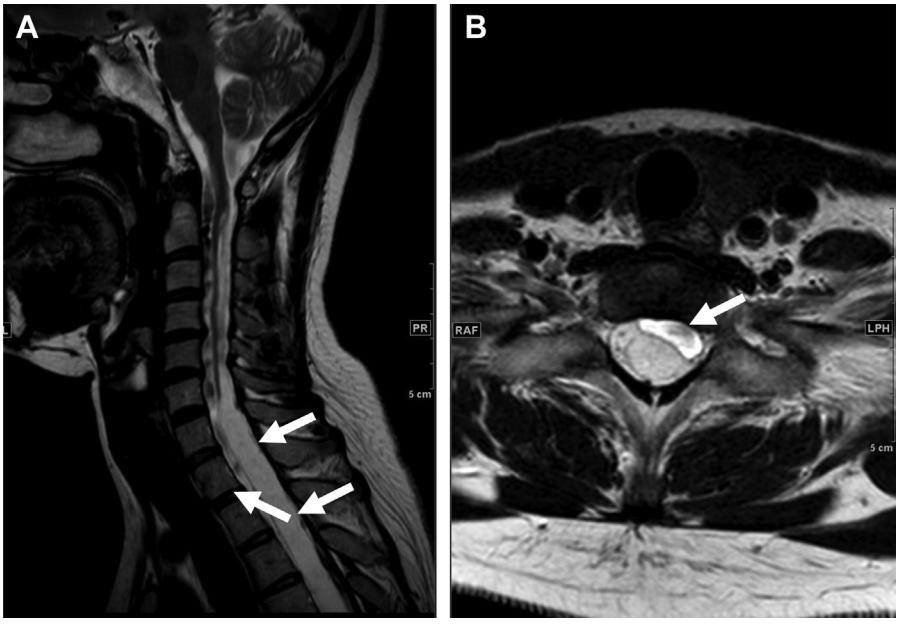

Fig. 20. Chronic arachnoiditis after remote TB spinal meningitis. Sagittal T2-weighted MR imaging (*A*) and axial T2-weighted MR imaging (*B*) show a syrinx formation in the cervicothoracic spinal cord with arachnoid cysts (*arrows*).

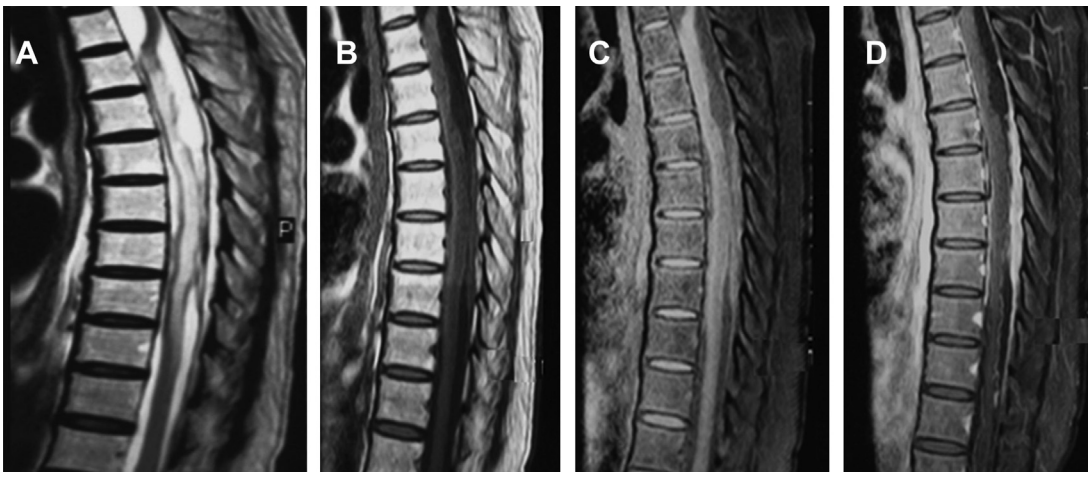

Fig. 21. Tuberculous myelitis. Sagittal T2-weighted MR imaging (*A*) and sagittal T1-weighted MR imaging (*B*) show diffuse cord swelling with T2 hyperintensity and T1 isointensity and ventral subarachnoid space obliteration. There is diffuse dural enhancement on sagittal T1-weighted postcontrast MR imaging (*C*, *D*).

infections caused by NTM are extremely rare, but there are increasing reports; high mortality is often associated with CNS involvement.[71] NTM usually infect immunosuppressed individuals, primarily those with HIV infection or transplant-associated immunosuppression, or those with hematologic malignancies.[70,72] CNS infection of NTM occurs in the settings of disseminated disease.[73] It usually presents as meningitis or meningoencephalitis.[74,75] The suggested mechanism of NTM-CNS infection is either through the hematogenous route because of high-grade bacteremia associated with disseminated NTM infections, or through direct inoculation, such as trauma, neurosurgery, and otolaryngologic infection.[1,76] *Mycobacterium avium* is the most common species of NTM-CNS infection, followed by *Mycobacterium fortuitum*, *Mycobacterium kansasii*, and *Mycobacterium abscessus*. The clinical presentations of NTM-CNS infection vary from headache, fever, and vomiting to hemiplegia, seizures, and altered mental state.[76–78] Diagnosis of NTM-CNS infection may be challenging because of the nonspecific clinical features and rarity of the disease.

Imaging findings of NTM-CNS infection are not established because there are only a few reports of the disease. The common imaging manifestations include meningeal enhancement and/or cerebral involvement (**Fig. 23**).[79–81] However, radiologic features may not distinguish NTM

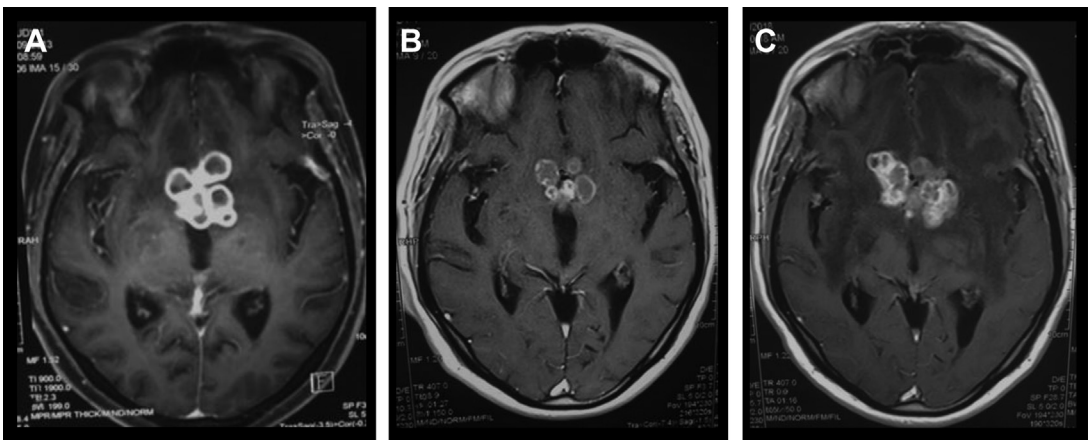

Fig. 22. Paradoxic reaction. Axial T1-weighted postcontrast MR imaging at symptom presentation (*A*) shows tuberculoma formation at the basal cistern. After treatment with antitubercular therapy, for 8 months the lesion decreased in size (*B*), but on follow-up after a year, repeat MR imaging shows paradoxic increase in size of the lesion (*C*).

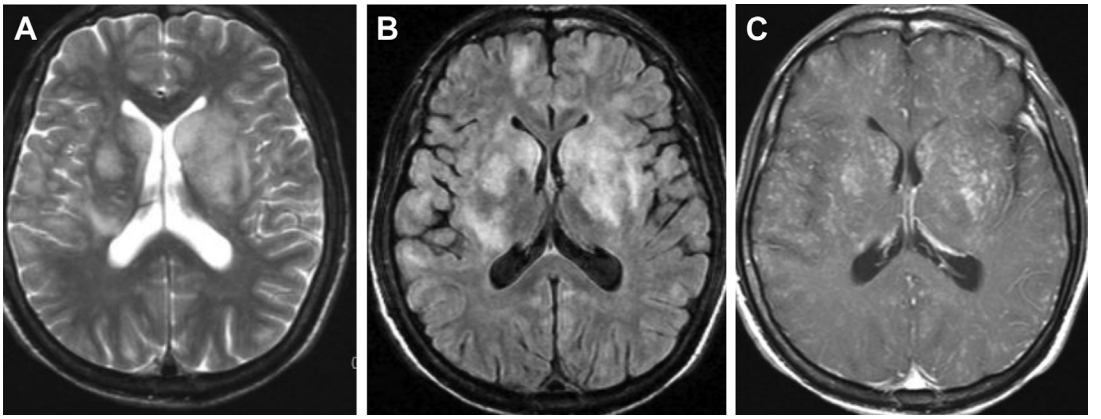

Fig. 23. NTM infection. Axial T2-weighted MR imaging (*A*) and FLAIR MR imaging (*B*) show multifocal patchy signal abnormality in the bilateral subcortical white matter and deep gray matter. Axial T1-weighted postcontrast MR imaging (*C*) shows widespread infiltrative contrast enhancement.

meningitis from other forms of meningitis, including tuberculous meningitis, because NTM infection may also exhibit basal nodular enhancement.[77,80] NTM infection of the CNS has rarely been reported as rhombencephalitis or an abscess that is indistinguishable from other causes of abscess.[78,82] An NTM infection may also manifest as hydrocephalus or brain atrophy, with chronic meningitis.[74,83] Therefore, if an immunocompromised patient has a sign of CNS infection and the pathogen for meningitis is difficult to detect, the clinician should suspect the NTM infection to avoid devastating complications.

LEPROSY

Leprosy is a chronic granulomatous disease caused by *M leprae*. It primarily affects the skin and peripheral nerves (rather than the CNS) and may cause significant functional impairments and deformities.[84,85] Although the incidence of leprosy has decreased in the past decades, it still remains a global public health issue.[85] A few case reports of CNS leprosy present as leprous ganglionitis and myelitis, mostly at the cervical spine.[86–88] The pathogenesis of spinal cord or brain involvement of leprosy is still unknown and hematogenous spread with direct infection or inflammatory immunologic reactions are suggested.[86]

On imaging, ganglionitis presents as the dorsal root ganglia swelling and contrast enhancement.[87,88] Combined brachial plexus thickening and enhancement may be found.[86] In some cases, there were asymmetrical T2 hyperintense cord signal changes with contrast enhancement at the adjacent cord, suggesting combined myelitis. On brain MR imaging, bilateral symmetric T2/FLAIR hyperintensity is observed in the dorsal brainstem with focal enhancement, where the cranial nucleus is located (**Fig. 24**).[86]

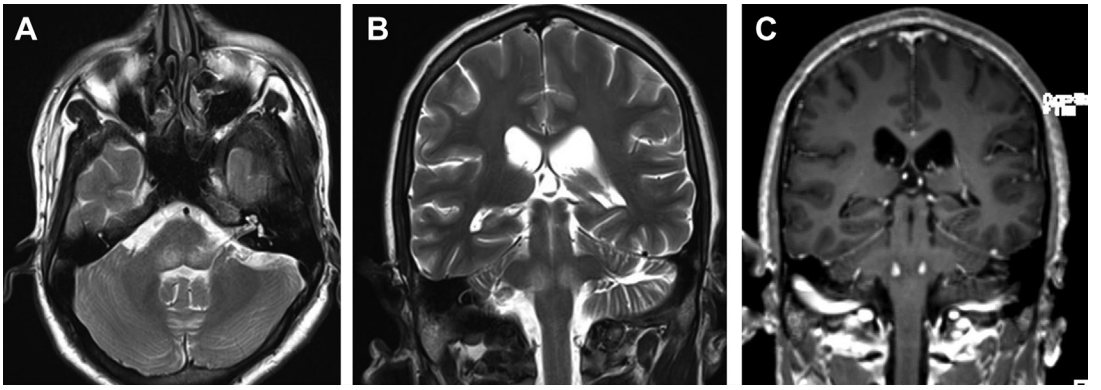

Fig. 24. CNS leprosy. Axial T2-weighted MR imaging (*A*) and coronal T2-weighted MR imaging (*B*) show symmetric T2 hyperintensities in the dorsal pons with corresponding contrast enhancement (*C*).

SUMMARY

TB is a common infectious disease of the CNS in developing countries and developed countries, in association with HIV infection. Cranial and spinal imaging are important in CNS tuberculous infection because understanding typical and atypical imaging findings may help in early diagnosis and disease monitoring in clinical practice. NTM infection is extremely rare in the CNS, and there are no consistent imaging findings. However, a high index of suspicion for CNS-NTM infection in indicative clinical conditions may be helpful.

CLINICS CARE POINTS

- Typical neuroimaging pattern of predominantly basal meningeal enhancement with hydrocephalus, with complications of cerebral infarction, should raise suspicion of tuberculous leptomeningitis, especially in immunocompromised patients and patients living in endemic areas.
- Tuberculomas have different imaging findings according to their clinical staging and radiologists should be aware of their stage-related characteristic imaging findings.
- Destructive, noncontiguous involvement of multiple vertebral bodies sparing intervertebral disks, especially in thoracic spine, is characteristic of tuberculous spondylitis.

DISCLOSURE

The authors have nothing to disclose.

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