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Imaging in Neurotuberculosis

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Abstract

Central nervous system tuberculosis can occur in various forms and carries high morbidity despite anti-tubercular treatment. Accurate and early diagnosis based on imaging can help improve prognosis and prevent complications. Confirmation of tubercular bacteria in CSF culture methods, and molecular analysis poses challenges due to invasive and time-consuming procedures. Advanced neuroimaging techniques are greatly helpful in making presumptive diagnoses. Here we described imaging features of multiple forms of CNS tuberculosis with advanced neuroimaging. This paper will help neuroradiologists to identify CNS TB and related imaging features.

Keywords
- brain
- granuloma
- neurotuberculosis
- meningitis

Introduction

Neurotuberculosis (nTB) is a type of systemic tuberculosis that manifests in the spinal cord, meninges, brain tissue, and spine; it can be localized or diffuse.[1,2] An estimated 8 to 10 million people are diagnosed with tuberculosis (TB) annually around the globe. Among those with TB, 2 to 5% experience involvement of the nTB, with up to 15% of cases associated with TB related to acquired immunodeficiency syndrome.[3] Seventy to 80% of these manifest as tuberculous meningitis (TBM).[4,5] nTB generally develops after an initial pulmonary infection caused by the secondary hematogenous dissemination of *Mycobacterium tuberculosis*, leading to the invasion of the meninges. Occasionally, an infection may arise when a tubercle ruptures in the subarachnoid space.

Computed tomography (CT) and magnetic resonance imaging (MRI) are two cross-sectional modalities that have greatly improved the identification and assessment of nTB. CT is invaluable for detecting and screening intracranial TB and its potential complications. Additionally, it is critical for determining the severity of spinal TB. MRI is the most recommended imaging modality for evaluating nTB due to its ability to produce images in multiple planes, superior spatial and contrast resolution, and advanced sequences such as spectroscopy, magnetic resonance angiography, and perfusion imaging. This technique effectively identifies various cerebral manifestations of TB, evaluates associated complications, and quantifies neurological symptoms. Additionally, MRI can differentiate between TB and other infections affecting the central nervous system (CNS), as well as distinguish between CNS TB and neoplastic and inflammatory brain disorders.

The diagnosis of nTB is made by the presenting symptoms, neuroimaging results, laboratory and microbiological results,

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and response to antituberculous treatment (ATT). This review aims to present a thorough and current synopsis of the fundamental and advanced imaging characteristics of nTB.

Spectrum of Lesions in Neurotuberculosis

1. Meningeal TB
   a. Tuberculous leptomeningitis (TBM)
   b. Chronic tuberculous pachymeningitis
2. Parenchymal TB
   a. Tuberculoma
   b. Miliary TB
   c. Cerebritis/Tuberculous abscess
   d. Tubercular encephalopathy
3. Spinal TB
   a. Spinal intramedullary tuberculoma
   b. Tubercular myelitis
   c. Tuberculous arachnoiditis
   d. Tuberculous spondylitis (Pott's spine)
4. Miscellaneous forms of CNS TB
   a. Tuberculous hypophysitis
   b. Orbital TB
   c. Tubercular otitis media
   d. TB of skull and base of the skull
   e. TB-related immune reconstitution inflammatory syndrome (TB-IRIS)

Imaging Protocol

For patients suspected of having nTB, specifically TBM, it is recommended to undergo a comprehensive MRI protocol. This should include precontrast T1 (T1W)- and T2-weighted (T2W) sequences, fluid attenuation inversion recovery (FLAIR), diffusion-weighted images (DWIs), and susceptibility-weighted imaging (SWI).

Gadolinium-enhanced T1-turbo spin-echo (TSE) and FLAIR imaging help detect subtle leptomeningeal enhancement and enhance exudates and tuberculomas. Spin-echo three-dimensional (3D) T1 postcontrast sequence with fat suppression is particularly helpful in detecting miliary tuberculomas, cranial nerve (CN) enhancement, optic chiasmatic involvement, optic nerve sheath, and ventriculitis and these isotropic images can be rebuilt in various planes. The contrast-enhanced (CE) 3D TSE black-blood sequence improves the contrast-to-noise ratio and reduces interference from vessel signals. In cases where tuberculoma presents atypically, MRI spectroscopy (MRS) may prove beneficial, while MRI venography and angiography can assist in identifying potential vascular complications of TBM. Postcontrast vessel wall imaging (VWI) may also detect vasculitis changes before infarction occurs. In emergencies, CT may also be helpful, particularly at the beginning of treatment, to detect hydrocephalus before lumbar puncture for cerebrospinal fluid (CSF) analysis. Thin-sliced noncontrast CT (NCCT) scans with a thickness of less than 3 mm can help identify hypodense parenchymal lesions and obliteration of the basal cisterns, while contrast CT is crucial in detecting enhancing exudates and tuberculomas.

Imaging Findings in CNS Tuberculosis

Meningeal TB

Tuberculous Meningitis

TBM accounts for around 1% of all TB cases and 5% of those cases that occur outside of the lungs.[6–9] TBM is the most prevalent form of nTB, affecting infants and adolescents primarily. It is characterized by the spread of *M. tuberculosis* through the leptomeninges, often due to the rupture of a microscopic subpial or subependymal tuberculoma (known as the "Rich focus") into the subarachnoid space. Alternatively, meningeal involvement may occur due to various factors such as intravascular rupture of a tuberculoma in a subarachnoid, the rupture of one of the miliary tuberculomas in brain miliary TB, or, rarely, contiguous spread from tuberculous bone infection such as mastoiditis.[6]

TBM commonly manifests clinically as fever, headache, reduced consciousness, and meningeal symptoms as photophobia, stiff neck, and vomiting. Additionally, there may be concurrent symptoms related to CN dysfunction.

The pathological hallmark of TBM is the presence of viscous and gelatinous inflammatory exudates in the meninges. Within a loose fibrin network, neutrophils, mononuclear cells, red blood cells, and varying amounts of bacilli are present in this exudate. Lymphocytes and monocytes aggregate to form many tiny tubercles.[10,11] The exudate quickly extends along the meninges, largely through the basal cisterns, which include the perimesencephalic, interpeduncular, prepontine, and suprasellar cisterns. Consequently, the basal cisterns become obstructed, impeding CSF absorption, and potentially leading to periventricular ooze and hydrocephalus. The exudates around the vessels of the circle of Willis can cause vasculitis, potentially resulting in cerebral infarction.

Imaging

The most frequent observation on NCCT is the presence of isodense or slightly hyperdense exudates in the basal cisterns obscuring them (►**Fig. 1**). These exudates enhance significantly in CECT (►**Fig. 2**). For diagnosing TBM, the combination of basal enhancement, hydrocephalus, and infarct has a sensitivity of 34% and a specificity of 100% (►**Fig. 2**).[12,13]

MRI is more sensitive than CT in identifying these abnormalities. MR findings may include basal exudates that appear isointense on T1-WIs and bright sulci and cisterns on FLAIR images (►**Fig. 3**). A "dirty CSF" appearance may be observed when exudates fill the sulci and cisterns. Exudates can spread along the perivascular spaces, leading to meningoencephalitis, and "flow voids" of major arteries may appear irregular or reduced due to vascular complications (►**Fig. 4**). Sometimes, rather than basal cisterns, convexity sulci show enhancement secondary to leptomeningitis (►**Fig. 5**).

Post-gadolinium T1-WI may show meningeal enhancement, which is better seen in fat-saturated images (►**Fig. 3**). Additionally, inflamed meninges in the basal cisterns may

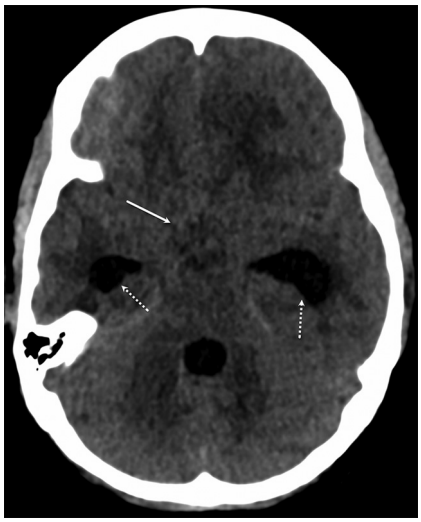

Fig. 1 Tubercular meningitis (noncontrast computed tomography [NCCT]). Noncontrast CT of the head demonstrates effacement of the suprasellar (arrow) and other basal cisterns due to isodense exudates, accompanied by hydrocephalus (dotted arrow).

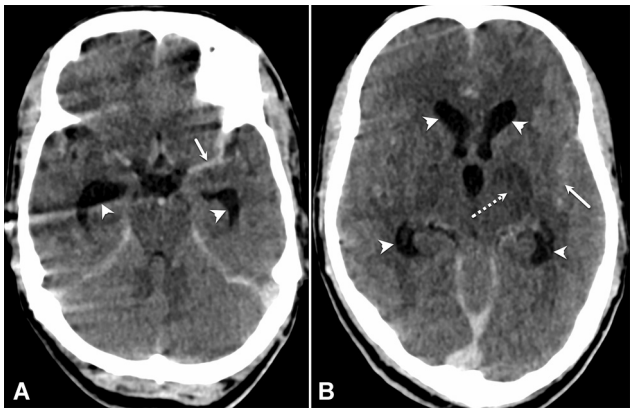

Fig. 2 Tubercular meningitis with hydrocephalus and infarct. Contrast-enhanced computed tomography (CT) of the head shows enhanced exudates in the left Sylvian cistern (arrow in **A**, **B**), accompanied by hydrocephalus (arrowheads in **A** and **B**). Additionally, there is a well-defined hypodensity in the left basal ganglia (marked by a dotted arrow in image **B**) due to an acute infarct.

display marked enhancement, resulting in a distinctive spider leg pattern on MRI (►**Fig. 3**). FLAIR-enhanced sequences are often more sensitive to detecting leptomeningeal enhancement than CE T1-WI (►**Fig. 6**).[14] In patients with acquired immunodeficiency syndrome, the meningeal enhancement may be minimal or completely absent.

The cerebral tissue adjacent to the tuberculous exudate demonstrates varying degrees of swelling, infiltration around blood vessels, and activation of microglial cells, referred to as "border zone encephalitis." Distinguishing border zone encephalitis can be challenging since the hyperintense signals on T2-WIs/FLAIR images in these areas merge with the hyperintense signal of the leptomeningeal exudate.[15]

Complications

The potential complications of meningitis are hydrocephalus, infarcts, and cranial neuropathies.

Hydrocephalus

TBM often results in hydrocephalus caused by disruptions in CSF circulation. This can manifest as communicating hydrocephalus, typically caused by blockages in the basal cisterns, or as noncommunicating hydrocephalus, which is less common and may be due to obstructions or pressure from tuberculomas or, rarely, tuberculous abscesses (►**Figs. 3** and **4**). If left untreated, long-term hydrocephalus can lead to cerebral atrophy.

Infarcts

The incidence of cerebral infarct in patients with TBM ranges from 15 to 57%.[16,17] Basal meningitis, which

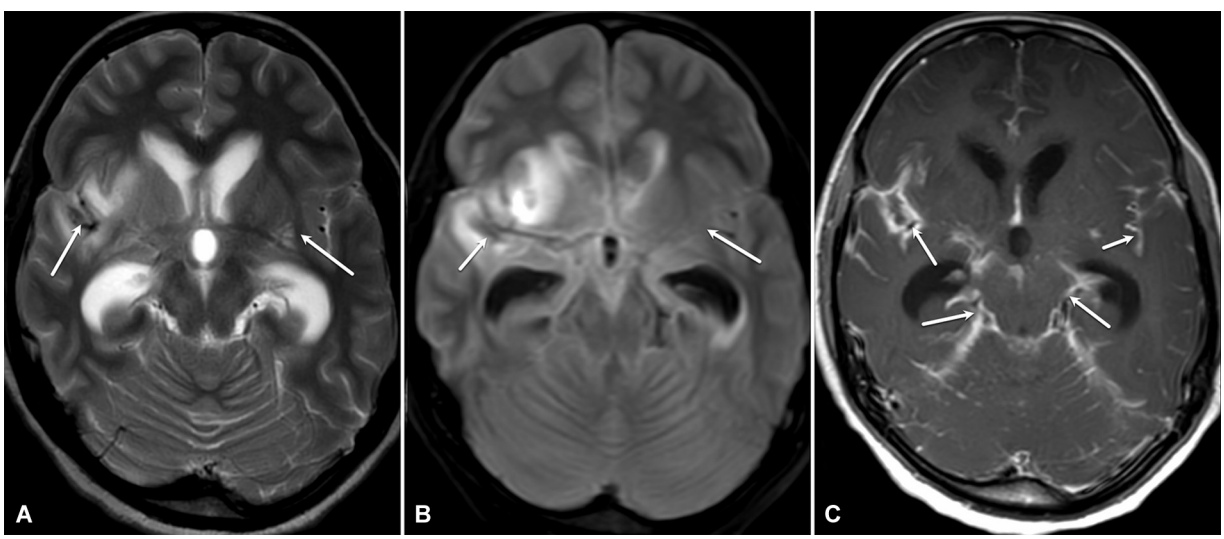

Fig. 3 Tubercular meningitis (magnetic resonance imaging [MRI]). Axial T2-weighted image (WI) (**A**) and fluid attenuation inversion recovery (FLAIR) image (**B**) demonstrate exudates occupying the basal cisterns accompanied by hydrocephalus. Post-gadolinium T1-WI (**C**) reveals widespread leptomeningeal enhancement (arrows).

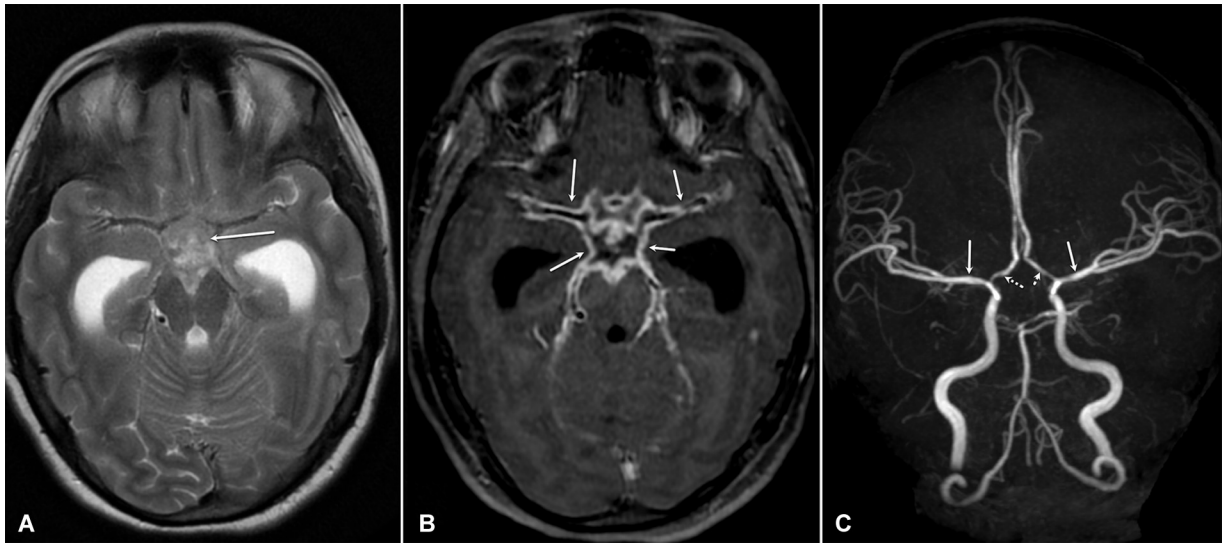

Fig. 4 Tubercular meningitis (magnetic resonance imaging [MRI]) with magnetic resonance angiography (MRA). Axial T2-weighted image (WI) (**A**) reveals iso- to hyperintense exudates within the suprasellar cistern. Post-gadolinium T1-WI (**B**) demonstrates dense enhancement of thickened meninges, giving a characteristic spider leg appearance (arrows). Time-of-flight magnetic resonance angiography of the circle of Willis (**C**) indicates attenuated bilateral distal internal carotid arteries, A1 segments of the anterior cerebral arteries (dotted arrows), and M1 segments of middle cerebral arteries (arrows).

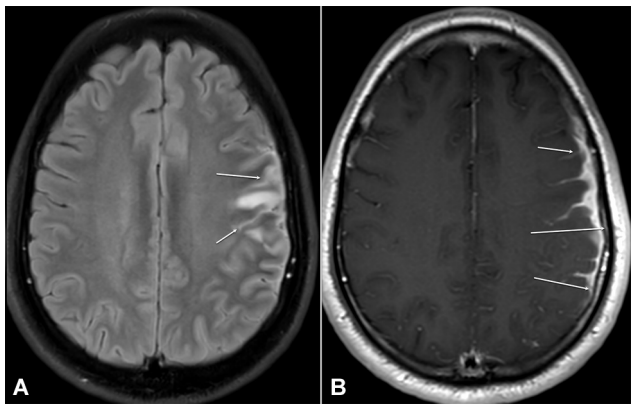

Fig. 5 Convexity meningitis. Axial fluid attenuation inversion recovery (FLAIR) image (**A**) reveals sulcal hyperintensity and adjacent parenchymal hyperintensity (arrows) in the left frontoparietal convexity. Post-gadolinium T1-weighted image (WI) (**B**) demonstrates pronounced sulcal enhancement (arrows), indicative of meningitis.

surrounds and impacts the central area of the circle of Willis, leads to inflammatory changes in the entire vessel wall, resulting in pan-arteritis and subsequent thrombosis and blockage of small perforating vessels. The lenticulostriate and thalamoperforating arteries are particularly affected. TMB often leads to multiple and bilateral infarctions, particularly in the thalami, basal ganglia, and internal capsule. In cases of acute infarcts, DWI is the preferred imaging technique, while T2W or FLAIR images are more useful in identifying subacute or chronic infarctions (►**Fig. 7**).

Cranial Neuropathy

Forty percent of nTB cases have CN involvement.[18] The nerves most commonly affected are CN II, III, IV, VI, and XII, often together due to factors such as vascular compromise, ischemia, or entrapment in basal exudates or tuberculoma mass-effect.[19] MRI can reveal thickening, T2 hyperintensity, and enhancement of the affected CNs, especially along their cisternal course in the internal auditory canal or basal cisterns (►**Fig. 8**). Fat-saturated T1-WI and CE FLAIR imaging can help detect these lesions.

Tubercular Ventriculitis

Tuberculous ventriculitis is a rare and devastating complication that arises when a tuberculoma grows, extends, and bursts in the ventricular system. It is accompanied by severe deterioration in clinical conditions. Symptoms can be seizure, papilledema, meningismus, and worsening of the underlying disease. Imaging can reveal hydrocephalus, ventricular sludge/debris, choroid plexitis, and enhancement of the ependymal and meningeal tissues. In some cases, restricted diffusion may be observed in the dependent portion of the sludge or the occipital horn.[6,13]

Differential Diagnosis

Other infectious meningitis and inflammatory conditions, such as rheumatoid arthritis, sarcoidosis, or carcinomatous meningitis, may exhibit similar patterns of meningeal enhancement. Focal or diffuse dural thickening can also indicate neurosarcoidosis, meningioma, or lymphoma and should be considered important differential diagnoses.

The radiological findings of enhancement of basal meninges, hydrocephalus, and parenchymal infarction strongly suggest TBM.[12,13]

Pachymeningeal Tuberculosis

Pachymeningeal TB, a chronic infection of the dura caused by TB, is typically a result of tuberculous leptomeningitis. Although rare, there have been documented cases of isolated pachymeningitis caused by direct invasion of TB bacilli through hematogenous spread. This condition is defined by

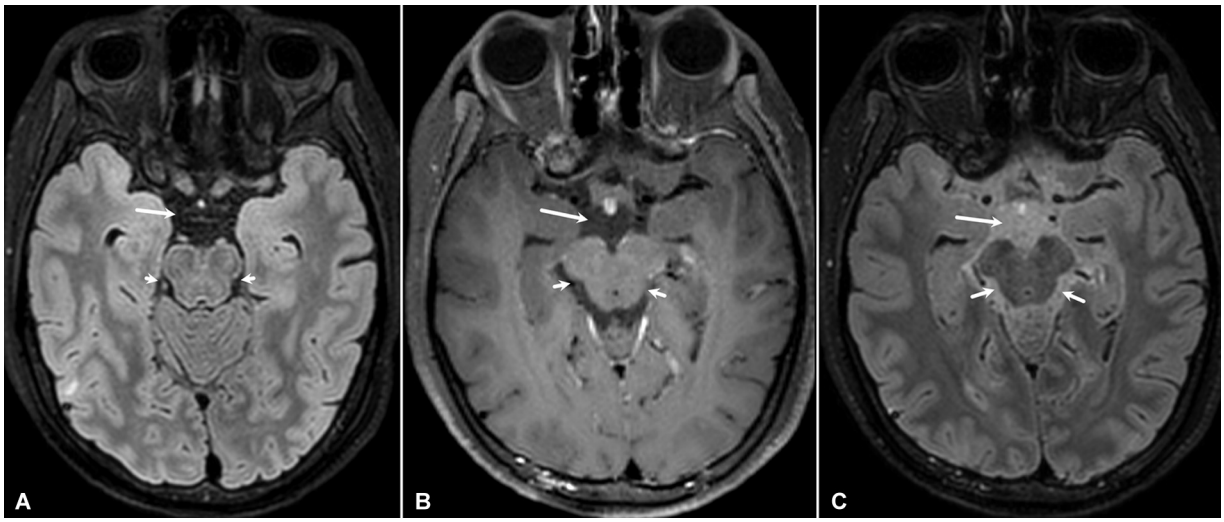

Fig. 6 Post-gadolinium fluid attenuation inversion recovery (FLAIR). Axial FLAIR (**A**) and post-gadolinium T1-weighted images (WIs) (**B**) reveal no clear abnormalities in the suprasellar (arrow) and perimesencephalic cisterns (short arrows). However, post-gadolinium FLAIR (**C**) imaging shows enhancement in the suprasellar and perimesencephalic cisterns, indicating meningitis. It is noted that postcontrast FLAIR imaging is more sensitive in detecting meningitis compared to post-gadolinium T1-weighted images.

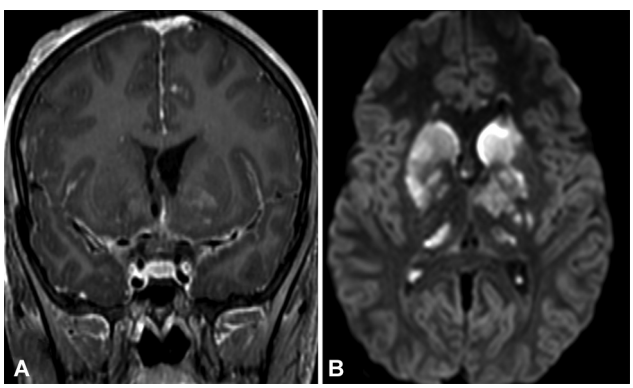

Fig. 7 Tuberculous meningitis (TBM) with infarct. Coronal postcontrast T1-weighted image (WI) (**A**) demonstrates enhancement around both middle cerebral arteries within the Sylvian fissures. Axial diffusion-weighted imaging (**B**) reveals diffusion restriction in both basal ganglia, indicative of acute infarcts (**B**).

focal or diffuse dural thickening, with or without inflammation. On CT scans, the focal lesion, known as "en plaque tuberculoma," appears hyperdense and shows homogenous or peripheral enhancement on CE imaging, while on MRI, it appears isointense to hypointense on T1- and T2-WIs (►**Figs. 9** and **10**).[20] Meningeal and pachymeningeal calcifications have been observed in adult patients. Commonly affected locations include the cavernous sinus, middle cranial fossa floor, tentorium, interhemispheric fissure, and cerebral convexity.

The presence of TB pachymeningitis on imaging is a nonspecific finding. When there is no proof of TB elsewhere in the body, it is important to consider other potential diagnoses, such as neurosarcoidosis, meningioma, and lymphoma. Meningioma may exhibit hyperostosis of nearby calvarial or skull base bone and increased perfusion on arterial spin labeling (ASL) maps, while neurosarcoidosis or lymphoma may require a comprehensive systemic evaluation, which may include

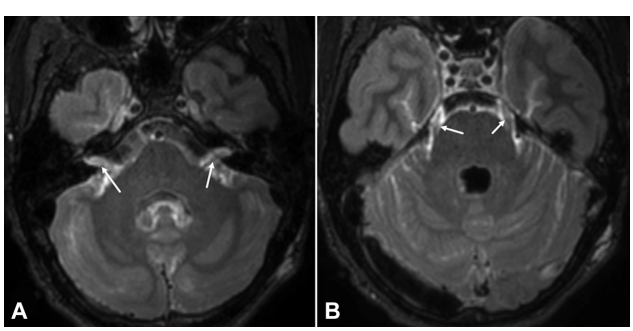

Fig. 8 Cranial neuritis. Axial postcontrast fluid attenuation inversion recovery (FLAIR) imaging reveals significant enhancement of the bilateral 7th–8th nerve complexes (arrows in **A**), the right 6th, and bilateral 5th nerves (arrows in **B**), indicative of neuritis. Additionally, enhancement is observed in both cerebellopontine angle cisterns (**A**), the fourth ventricle, and the cerebellar folia.

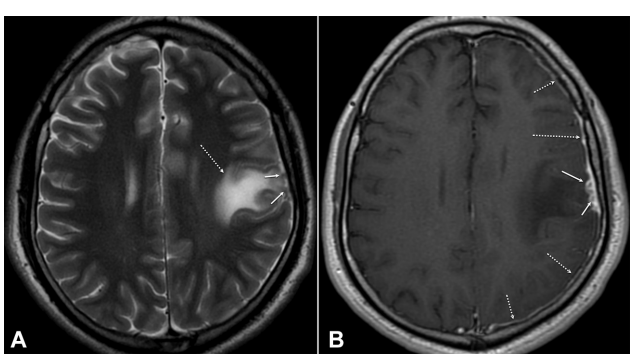

Fig. 9 Pachymeningitis. Axial T2-weighted image (WI) (**A**) reveals nodular thickening with hypointensity (arrows) along the left cerebral convexity and underlying edema (dotted arrow). Postcontrast T1-WI (**B**) demonstrates nodular (arrows) in conjunction with diffuse pachymeningeal (dotted arrows) enhancement.

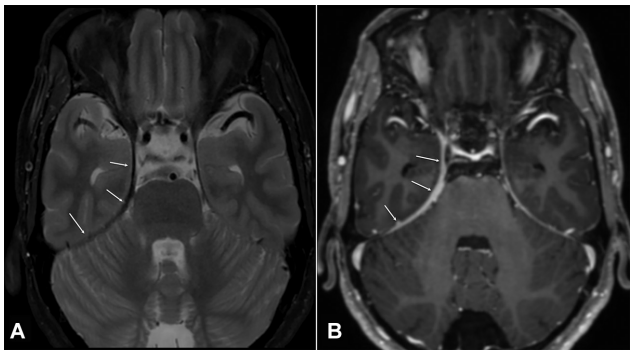

Fig. 10 Basal pachymeningitis. Axial T2 (**A**) and post-gadolinium T1-weighted image (WI) (**B**) demonstrate T2-hypointense pachymeningeal thickening with enhancement along the left tentorial and cavernous sinus.

taking samples of tissue. If the lesion shows a favorable response to anti-TB treatment, the most likely diagnosis is TB pachymeningitis.[21]

Parenchymal Tuberculosis

Parenchymal TB may manifest alone or together with TBM. It may present as tuberculoma, cerebritis, abscess, military TB, or tuberculous encephalopathy.

Tuberculoma

Tuberculoma is the most common type of parenchymal lesion found in nTB. These lesions are initially formed by merging granulomas caused by *M. tuberculosis*. They are characterized pathologically by the presence of epithelial and multinucleated cells inside a granuloma, encircled by a layer of lymphocytes, and have a central region of caseous necrosis. Throughout the brain, they can occur as single or multiple lesions, and their distribution correlates with the hematogenous spread. They are more common in the corticomedullary junction and in the periventricular region.

On NCCT scans, a tuberculoma might present as an iso- to hypodense mass (►**Fig. 11A**), while CECT scans could reveal solid or ring-enhancing lesions, indicative of the tuberculoma or abscess. A notable diagnostic feature, the target sign, characterized by a central calcified nidus with a peripheral rim of enhancement, further suggests the presence of a tuberculoma. MRI characteristics of tuberculomas may vary according to their stage of maturation. Noncaseating tuberculomas, solid- caseating tuberculomas, tuberculomas with central liquefaction, and calcified tuberculomas are the four subtypes.[12]

A noncaseating tuberculoma is a solid lesion without necrosis. It typically presents as iso-hypointense on T1-WI, hyperintense on T2-WI, and uniform nodular or homogeneous enhancement. On the other hand, a solid caseating tuberculoma has a core of caseating necrosis surrounded by a collagenous tissue capsule made up of epithelioid cells, multinucleated giant cells, and mononuclear inflammatory cells.[22] It often appears as iso-hypointense on T1-WI, mixed but predominantly hypointensity on T2-WI/ FLAIR images, and has a ring-enhancing capsule on postcontrast T1WI (►**Fig. 11**).[12] The next stage is central liquefaction of caseous material in tuberculoma. This lesion is similar to other infectious abscesses and typically has a low bacilli concentration in the caseous material. At CECT, it manifests as a ring-enhancing lesion with thick irregular wall and hypodense core. In MRI, the core is hypointense on T1-WI and hyperintense on T2-WI, and the peripheral capsule appears T2-hypointense with a peripheral, irregular, thick enhancement (►**Fig. 12**). Varying levels of vasogenic edema may be observed in all presentations. Healed tuberculomas may be identified as calcified foci on nonenhanced CT scans. In some cases, calcification in the basal cisterns may be observed years after an episode of meningeal TB.

Perfusion MRI frequently demonstrates increased relative cerebral blood volume, whereas MRS shows a decreased N-acetyl aspartate (NAA) to creatine ratio together with a

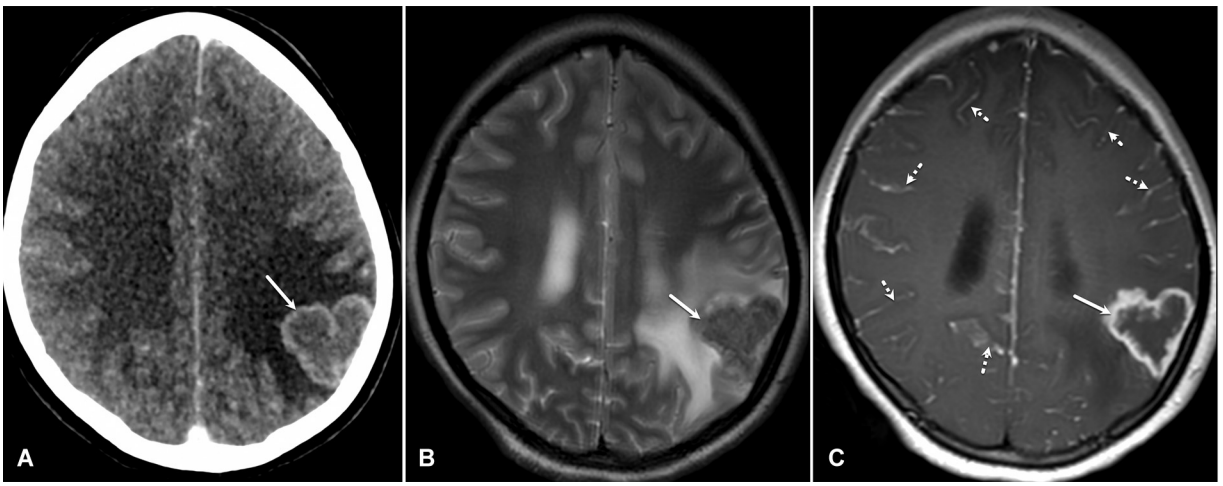

Fig. 11 Caseating granuloma with a solid center. A caseous solid granuloma, indicated by arrows, is present in the left parietal lobe, displaying iso-to-hyperdensity on noncontrast computed tomography (NCCT) (**A**) and pronounced hypointensity on T2-weighted image (WI) (**B**), with rim enhancement observed on postcontrast T1-WI (**C**) and associated mass effect. In addition, diffuse leptomeningeal enhancement is seen (dotted arrows in **C**).

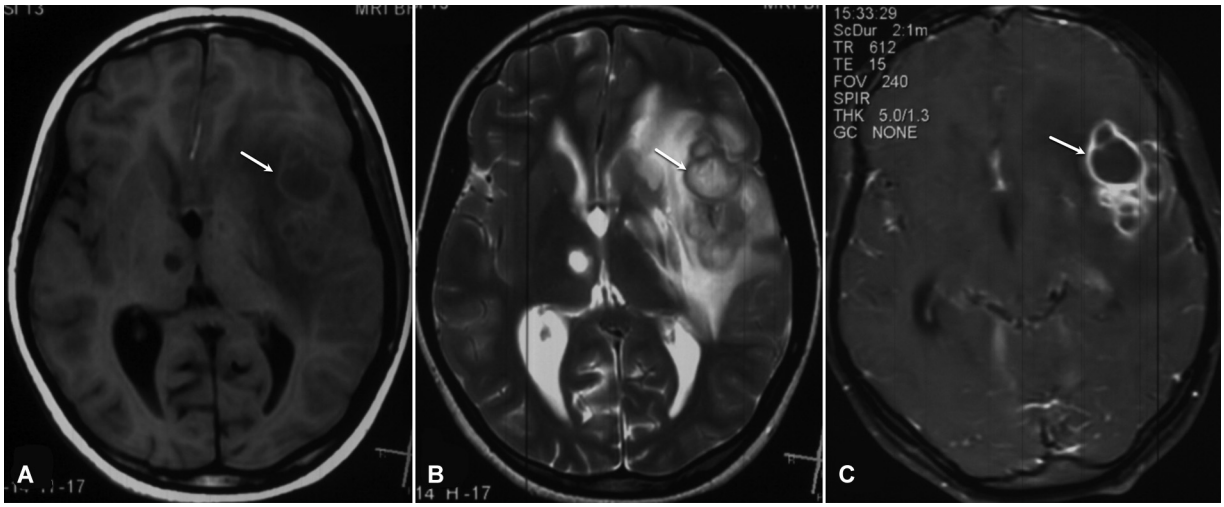

Fig. 12 Caseating granuloma with a liquid center. Caseating granulomas with central liquefaction (arrow) are present in the left frontal lobe; hypointense on T1-weighted image (WI) (**A**) and hyperintense on T2-WI (**B**), surrounded by hypointense peripheral rim indicative of a collagenous capsule and perilesional edema. The lesions show rim enhancement after intravenous administration of contrast.

prominent lipid peak, which is distinct from the profiles seen in typical pyogenic abscesses.

Differential Diagnosis

The differentiation of tuberculomas from other intracranial pathologies such as neurocysticercosis, fungal granulomas, pyogenic brain abscesses, primary brain tumors (including glioma and lymphoma), and metastatic brain tumors is challenging due to overlapping MRI features. Advanced imaging techniques are crucial for accurately diagnosing tuberculomas among these possibilities.

DWI is particularly useful; for example, T2-hypointense caseating granulomas with a solid center, which do not exhibit diffusion restriction, can be distinguished from T2-hypointense lymphomas. In contrast, T2-hyperintense caseating granulomas that have central liquefaction may show restricted diffusion, making them difficult to differentiate from pyogenic abscesses and high-grade gliomas.

MRS offers additional differentiation capabilities, identifying tuberculomas through specific spectral signatures. T2-hypointense tuberculomas reveal a unique lipid peak at 0.9 and 1.3 ppm, a highly specific indicator of tuberculoma. Conversely, T2-hyperintense tuberculomas exhibit both a choline peak at 3.22 ppm and a prominent lipid peak. Pyogenic abscesses are characterized by an amino acid peak (e.g., valine, leucine, isoleucine) at 0.9 ppm and a lipid-lactate peak at 1.3 ppm, while tumors show a lipid peak alongside elevated choline and decreased NAA and creatine peaks.

Characteristics Imaging Appearance of Tuberculomas

Lesion	T1-WI	T2-WI	FLAIR	DWI	T1-WI + C
Noncaseating tuberculoma	Iso- to hypointense	Hyperintense	No suppression	No restriction	Homogeneous enhancement
Solid caseating tuberculoma (►**Fig. 11**)	Iso- to hypointense along with hyperintense rim	Hypointense	No suppression	No restriction	Homogeneous or ring-like enhancement
Caseating tuberculoma with central liquefaction (►**Fig. 12**)	Isointense to hypointense along with hyperintense rim	Hypointense rim with central hyperintensity	Partial suppression	May or may not show a restriction	Ring-like enhancement
Calcified tuberculoma (►**Fig. 13**)	Iso- to hypointense	Hypointense	No suppression	No restriction	No enhancement

Cerebritis and Abscesses

Tuberculous cerebritis is a rare, particularly focal form, characterized by the presence of micronodular granulomas with or without caseous components in a specific area, without tuberculoma formation. This condition may or may not be accompanied by meningitis and can be diagnosed by imaging. MRI can show swelling, alternations in the signal intensity (high T2 signal and low T1 signal), and irregular contrast enhancement in the affected gyrus. However, the specificity of these findings is limited.

Tuberculous abscesses are a rare complication that affects less than 10% of patients diagnosed with nTB. The elderly and debilitated patients are more susceptible to developing this complication, which presents acutely as headache, fever, and focal neurological deficits. Tuberculous abscesses progress more rapidly than tuberculomas. These abscesses form due

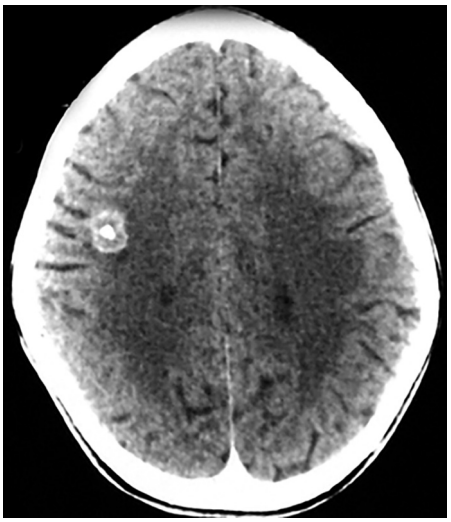

Fig. 13 Calcified tuberculoma of the brain. The nonenhanced axial computed tomography (CT) reveals a hyperdense lesion featuring a hyperdense periphery and an inner calcification zone located at the gray-white matter junction of the right frontal lobe. There is no notable surrounding edema.

to the liquefaction of tuberculous granulomas in the brain tissue or the extension of tuberculous lesions from the meninges. Pathologically, they are confined collection of pus that contains bacteria, both dead and alive, as well as cellular debris, and caseous material. Unlike tuberculous granulomas, they do not have the characteristic giant cell epithelioid granulomatous reaction in the wall.[19]

On imaging studies, tuberculous abscesses present as well-defined, multiloculated lesions larger than 3 cm, with surrounding edema. CT scans reveal hypodense masses with peripheral edema. The core of the abscess is generally hypointense on T1-WIs and hyperintense or heterogeneous on T2-WIs, with varying degrees of suppression on FLAIR sequences. DWI typically shows restricted diffusion indicated by a low apparent diffusion coefficient value (►**Fig. 14**). CECT and MRI detect enhancement in the outer wall of the abscess, which is typically thin and uniform in appearance but may become irregular and thick in individuals with compromised immune systems.

Distinguishing tuberculous abscesses from caseating tuberculomas can be challenging, but features such as size larger

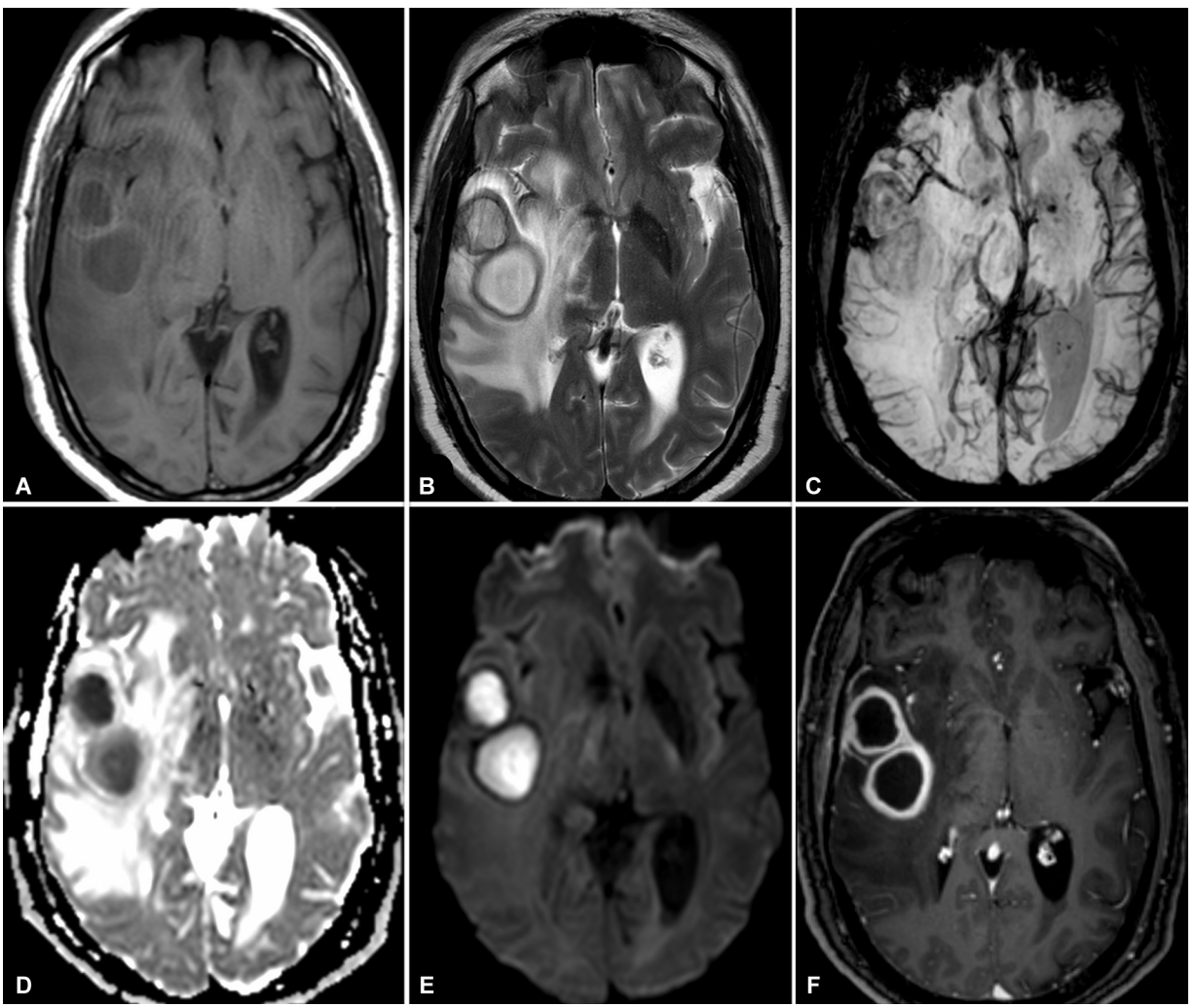

Fig. 14 Abscess. Two abscesses are located in the right temporal lobe, accompanied by edema. The core of these abscesses appears hypointense on T1-weighted image (WI) (**A**) and hyperintense on T2-WI (**B**), with a hypointense collagenous rim. No hemorrhage is observed on susceptibility-weighted imaging (**C**). The core exhibits high signal intensity on diffusion-weighted imaging (**E**) and low signal intensity on the apparent diffusion coefficient map (**D**), indicating restricted diffusion. Additionally, the abscess wall demonstrates smooth enhancement (**F**).

than 3 cm, diffusion restriction, a thin and smooth wall, and multiloculation generally suggest an abscess diagnosis. Magnetization transfer (MT) imaging and MRS can be utilized to distinguish tuberculous abscesses from pyogenic abscesses by detecting an amino acid peak at 0.9 ppm that is specific to pyogenic abscesses and absent in tuberculous abscesses. In addition, the MT ratio (MTR) value of tuberculous abscesses is considerably lower compared to pyogenic abscesses.

Miliary Tuberculosis

Miliary TB primarily affects immunocompromised patients and can occur alongside TBM or a primary TB infection in another part of the body. Despite its significant pathology, clinical symptoms indicating brain involvement are often absent, making it difficult to diagnose without imaging. Miliary TB is characterized by numerous lesions, typically between 2 and 3 mm in diameter, dispersed throughout the brain. These lesions are commonly found at the corticomedullary junction and along the pathways of perforating blood vessels, indicating a hematogenous spread. These lesions may manifest as high or low signal intensities on T2-WI; however, noncontrast sequences might fail to detect most granulomas. Several scattered foci of punctate enhancement, some with a ring-like enhancement and others without, can be seen in post-gadolinium T1-WI (►**Fig. 15**).

Tuberculous Encephalopathy

Tuberculous encephalopathy is a condition that primarily affects infants and children and is caused by a cell-mediated immune response to tuberculin protein. This results in a delayed type IV hypersensitivity reaction and can lead to symptoms such as progressive cognitive decline, convulsions, stupor, and coma. Unlike other forms of meningitis, this condition does not typically present with symptoms of meningeal irritation or focal neurological deficits.

Neuroimaging is crucial in diagnosing this condition, as it can reveal diffuse unilateral or bilateral white matter hyperintensity in T2W/FLAIR images.[23] Additionally, the affected white matter may exhibit diffuse postcontrast enhancement. MT imaging can show a significantly reduced MTR in the affected white matter, providing further insight into the extent of demyelination and edema associated with this condition.

Spinal Tuberculosis

Spinal Tuberculomas

Nonosseous spine involvement can be categorized as intramedullary, intradural extramedullary, or extradural. The incidence of these tuberculomas is influenced by geographical location, with studies reporting extradural granulomas in 64% of patients and arachnoid lesions without dural involvement in 20% of patients.[10] Additionally, 8% of patients had intramedullary lesions and 8% had subdural extramedullary lesions.[10] Intramedullary tuberculomas are usually detected on CE MRI as either nodular or ring-enhancing lesions, often accompanied by cord edema, syrinx formation, or arachnoiditis (►**Fig. 16**). On the other hand, intradural extramedullary tuberculomas might manifest as a solitary lesion with ring-shaped enhancement on the dura, a long segment of enhancing soft tissue mass, or might even resemble an en-plaque meningioma. The spinal cord and nerve roots are frequently compressed by these lesions, which may or may not be accompanied by arachnoiditis.

Tuberculous Spondylitis

Tubercular spondylitis is the most common form of bone TB infection and a significant cause of paraplegia, affecting individuals of all ages. Tuberculous spondylitis occurs due to the spread of the disease to the vertebral body via the paravertebral venous plexus. The most commonly affected vertebrae are the lower dorsal and lumbar, followed by the cervical. Involvement of the atlantoaxial region is rare. It shows a predilection for the anterior aspect of the vertebral bodies. This condition commonly affects several adjacent vertebrae, sparing the intervertebral discs at first, and frequently bypasses several levels before advancing beneath the

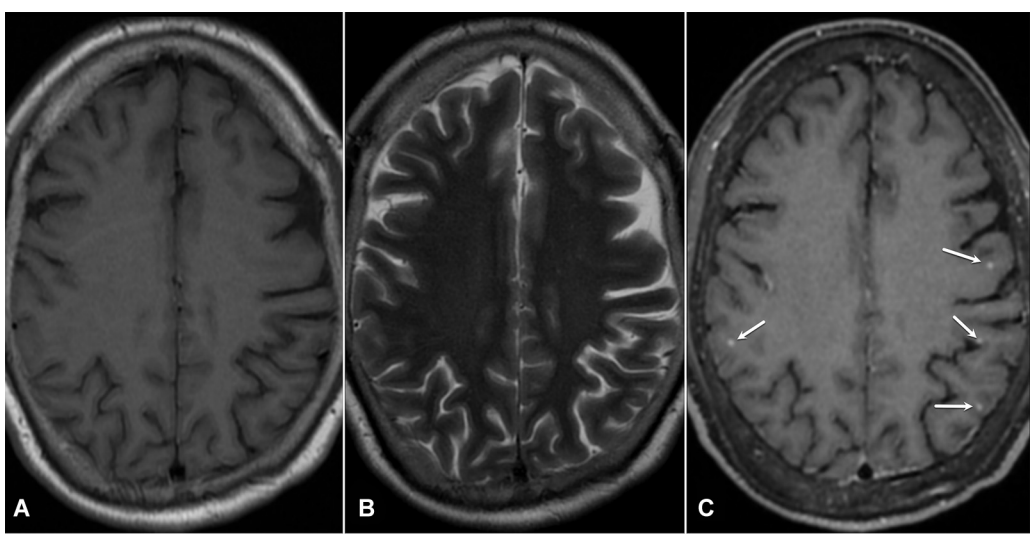

Fig. 15 Miliary tuberculomas. Following gadolinium administration, T1-weighted image (WI) (**C**) reveals numerous punctate-enhancing lesions across both cerebral hemispheres, indicated by arrows. These lesions are not visible in the axial T1 (**A**) and T2-WIs (**B**).

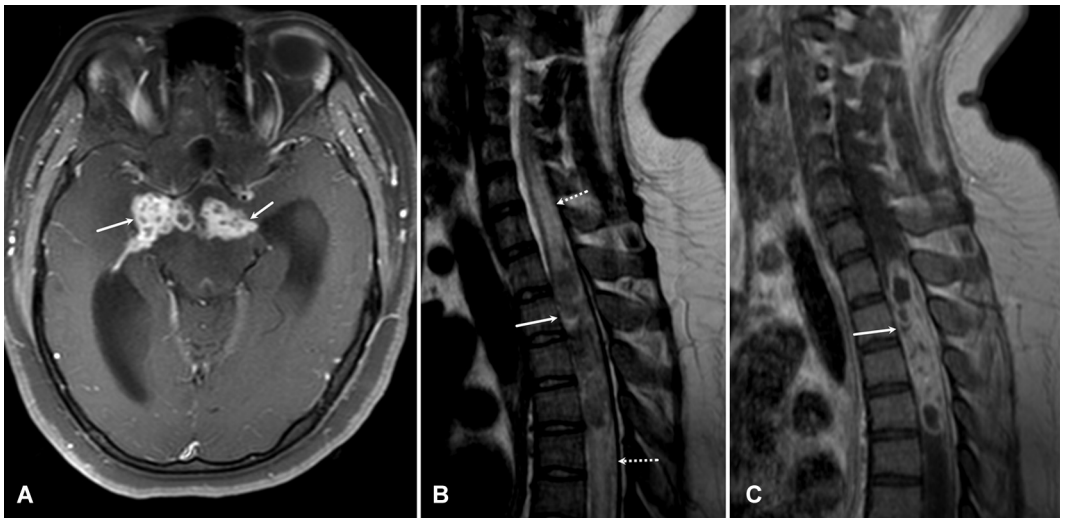

Fig. 16 Brain and spinal intramedullary tuberculoma. The axial postcontrast T1-weighted image (WI) (**A**) of the brain reveals several clustered ring-enhancing lesions within both temporal lobes, accompanied by hydrocephalus. Additionally, the sagittal T2-WI (**B**) of the dorsal spine displays a distinctly outlined ellipsoid hypointense lesion (arrow) situated in the intramedullary space opposite the dorsal vertebra, with nearby cord edema evident (dotted arrow). Furthermore, the sagittal postcontrast T1-WI (**C**) illustrates predominantly heterogeneous enhancement, featuring areas of central nonenhancement.

anterior longitudinal ligament (and occasionally the posterior longitudinal ligament). The paraspinal extension is a frequently observed complication that may result in abscess formation in the paravertebral, subdural, or epidural regions, ultimately compressing the spinal cord. In more advanced stages, this condition can progress to kyphosis and cord compression due to vertebral body collapse.

Imaging findings in cases of TB spondylitis reveal bone edema characterized by low signal intensity on T1-WIs, high signal intensity on T2-W/STIR images, and contrast enhancement in the initial phase (►**Fig. 16**). As the condition progresses, there is notable extensive destruction of the vertebral body, usually affecting three or more vertebral levels, with accompanying subligamental and paravertebral soft tissue or the formation of abscesses (►**Fig. 17**). Additionally, distinct paraspinal abnormal signal intensity and the presence of a thin, smooth, enhancing paraspinal and intraosseous abscess are recognized imaging features of TB spondylitis (►**Fig. 18**). CT scans are effective in determining the extent of bone involvement. The presence of bone fragments within or close to the lesion is a distinguishing feature of CNS TB, as *M. tuberculosis* lacks the lytic enzymes necessary for bone degradation.[24] CT is the preferred method for visualizing these lesions, although T2 sequences may reveal magnetic susceptibility artifacts without abscesses.[25]

Pyogenic spondylodiscitis is the primary differential diagnosis to consider for tuberculous spondylitis. This condition

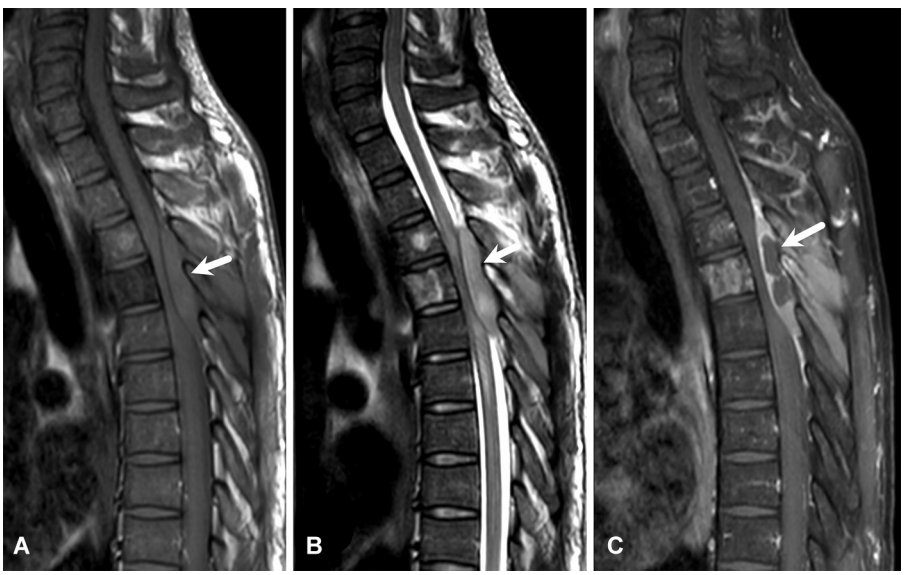

Fig. 17 Tubercular spondylitis with dorsal epidural abscess. Sagittal magnetic resonance (MR) images of the spine reveal changes in signal intensity affecting the upper dorsal vertebrae, characterized by T1 hypointensity (**A**) and T2 hyperintensity (**B**). Additionally, there is a peripherally enhancing dorsal epidural collection (indicated by arrows) accompanied by meningeal thickening in postcontrast T1-weighted images (**C**).

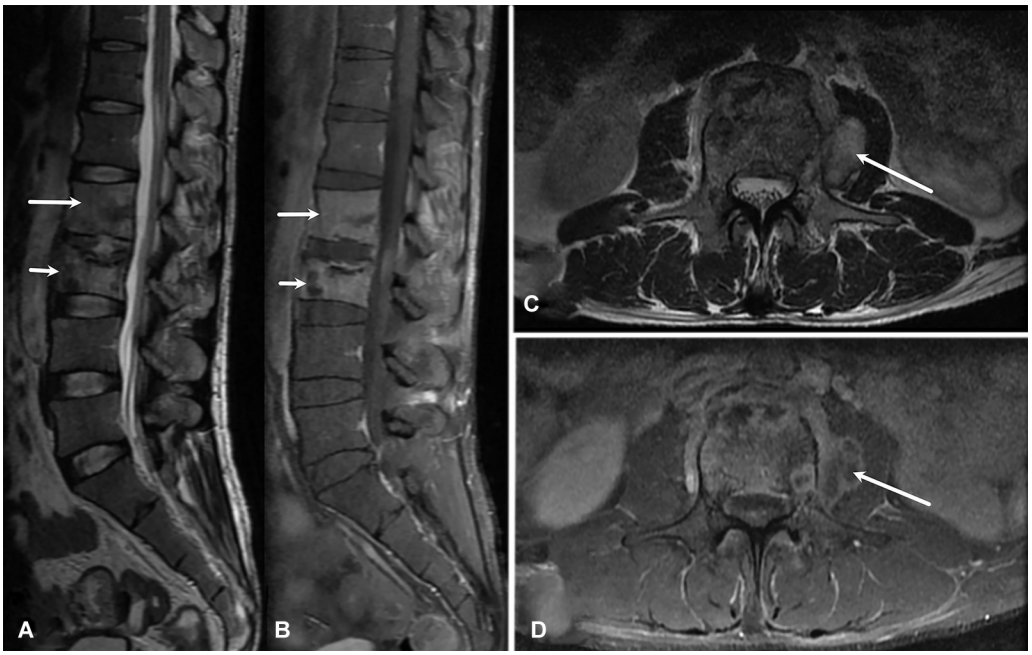

Fig. 18 Tubercular spondylitis with paravertebral abscess. Sagittal (**A**) and axial (**C**) T2-weighted images reveal widespread signal abnormalities characterized by T2 hyperintense signals affecting the L2 and L3 vertebrae (arrows in **A**), accompanied by a T2 hyperintense collection (arrow in **C**) adjacent to the left paravertebral area (**C**). Postcontrast imaging demonstrates abnormal enhancement of the vertebrae (arrows in **B**) and peripheral enhancement of the collection, including intraosseous extension (arrows in **D**).

predominantly affects the lumbar spine, which tends to affect the intervertebral disc first and vertebral body endplates. It typically involves up to two vertebral bodies. Tuberculous spondylitis typically initiates from the anterior cancellous bone of the vertebral body, destroying the vertebral body and the formation of an abscess near it, which extends beneath the anterior longitudinal ligament. Distinguishing features that indicate TB spondylitis over pyogenic spondylitis include the presence of para-/intraspinal spinal abscesses, thin and smooth walls of abscess, the well-defined margin of the wall, abscess within the vertebra, sparing of the disc, involvement of more than two vertebrae, and greater bone destruction. Conversely, these features favor pyogenic spondylitis.

Tuberculous Spinal Meningitis

Tubercular spinal meningitis is usually an extension of cerebral TBM or a contiguous spread of tuberculous spondylitis or presents as a primary lesion through hematogenous spread. It results in the accumulation of a gelatinous exudate within the spinal subarachnoid space, leading to localized inflammation of the cord and nerve roots.[26] In chronic cases, this exudate may lead to the development of adhesions and fibrosis. Spinal meningitis, akin to cranial leptomeningitis, can potentially trigger local vasculitis and focal segmental infarction.

Imaging may reveal inflammatory exudates that obliterate the spinal subarachnoid space, obscure the spinal cord's outline, and thicken or clump nerve roots. CE MRI may show nodular or linear enhancement along the surface of the spinal cord or nerve roots, thickening of dural resembling plaques, and irregular enhancement of the meninges or nerve roots. Arachnoiditis, when it reaches an advanced stage, can lead to the formation of CSF loculations, cord infarction, and syringomyelia (►**Fig. 19**).

Tuberculous Myelitis

Tubercular myelitis primarily affects the thoracic spinal cord and, less commonly, the lumbar and cervical cords. Paraparesis is the most common clinical presentation. With or without cord expansion, the affected spinal cord shows hyperintensity on T2-WI and hypointensity or isointensity on T1-WI.[27] Diffuse or patchy postcontrast enhancement is seen that can persist for several months. The associated meningitis may manifest as obliteration of the spinal subarachnoid space, clumping of nerve roots, and nodular intradural enhancement. Occasionally, there have been reports of longitudinally extensive transverse myelitis as a contiguous immune-mediated inflammatory lesion without any histopathologic evidence of active TB invading the spinal cord.[28] Associated conditions such as intramedullary abscess, cavitation, cord atrophy, and syringomyelia are associated with unfavorable prognoses.

Miscellaneous Forms of nTB

Miscellaneous forms of nTB encompass a range of rare conditions that can mimic other diseases, making accurate diagnosis challenging but critical.

1. *Tuberculous hypophysitis is* a condition that bears resemblance to a pituitary adenoma. On MRI, it manifests as a diffusely enlarged gland with a thickened stalk. The key differentiating factors from a pituitary adenoma are the thickened enhancing stalk and the surrounding dura.

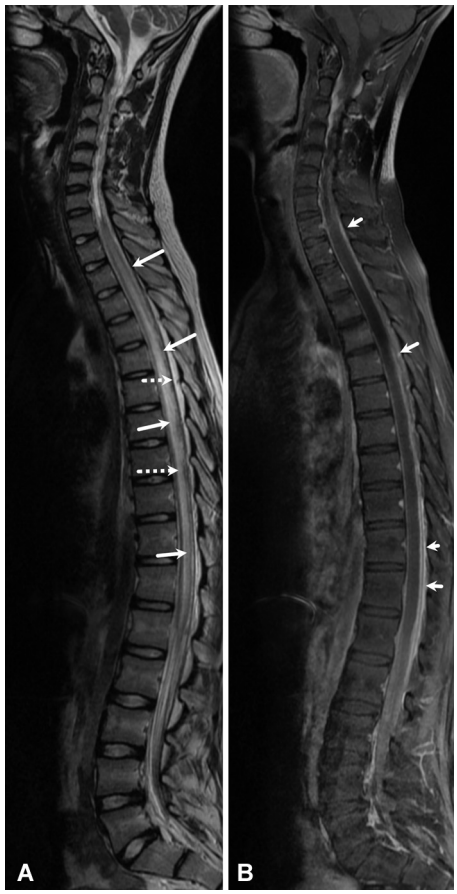

Fig. 19 Tubercular spinal meningitis with myelitis. Sagittal T2-weighted (**A**) and T1 postcontrast (**B**) magnetic resonance (MR) images of the spine reveal meningeal enhancement and thickening along the dorsal aspect of the dorsolumbar vertebral levels (indicated by arrows), accompanied by extensive, long-segment T2 hyperintense swelling of the spinal cord (indicated by dotted arrows).

Other differential diagnoses include sarcoidosis, syphilis, germinoma, and eosinophilic granuloma.

2. *Tuberculous otitis media* is a rare condition that primarily affects infants and children. It is characterized by painless otorrhea and can be identified through CT or MRI of the temporal bone. These imaging techniques usually show an enhancing soft tissue mass in the middle ear and external auditory canal. In some cases, there may be signs of bone erosion or sclerosis, while the scutum remains intact. If left untreated, this condition can progress to mastoiditis and the formation of sinus tracts. It is crucial to differentiate tuberculous otitis media from other conditions, such as bacterial infections, fungal granulomas, cholesteatomas, Wegener's granulomatosis, and Langerhans cell histiocytosis.
3. *Orbital TB* is a rare condition that usually affects only one eye and is more frequently seen in children. MRI findings may show a mass-like extraconal inflammatory deposit or abscess, preseptal part thickening, inflammation in lacrimal gland, and bony orbit damage or sclerosis.
4. *Calvarial TB* is a rare manifestation primarily affecting the frontal and parietal bones.[29] It usually appears as a subgaleal swelling, referred to as Pott's puffy tumor, associated with a discharging sinus. Distinct borders and a central sequestrum characterize the osteolytic lesion, which can affect both the outer and inner tables of the skull. Complications may include extensive bone destruction and occasional sclerosis and often cooccur with subdural empyema, meningitis, and parenchymal granuloma.

   Extradural collections and punched-out lesions in the skull are common radiographic findings. There are three types of tubercular osteitis: (1) circumscribed lytic lesions—small punched-out lesions with granulation tissue that affects both the inner and outer tables of the skull, with no associated periosteal reaction, but they can spread (►**Fig. 20**); (2) diffuse TB of the cranium—lesions that cause extensive destruction of the inner table of the skull; and (3) spreading type—when the previous lesions are

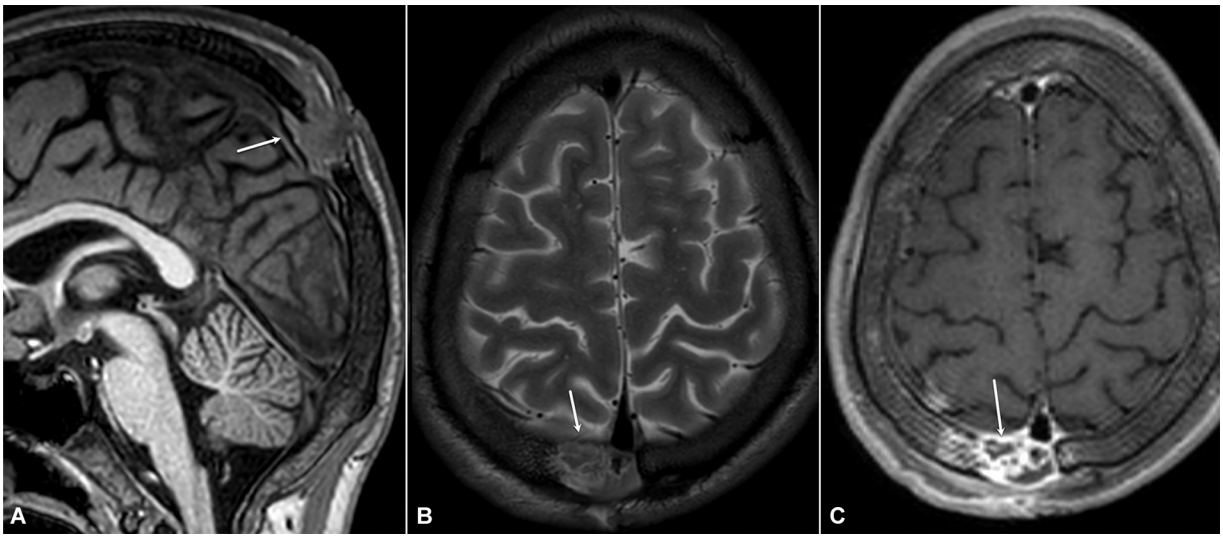

Fig. 20 Calvarial tuberculosis. On the sagittal T1-weighted image (**A**) and axial T2-weighted image (**B**), there is a focal lytic lesion located on the right side of the parietal bone. After gadolinium administration, this lesion shows enhancement (**C**).

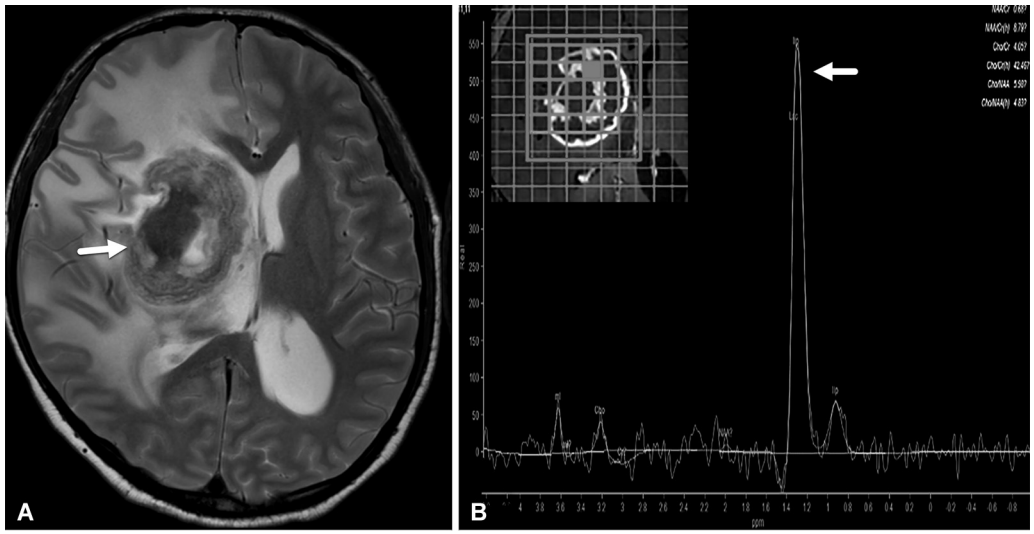

Fig. 21 Magnetic resonance spectroscopy (MRS) in tuberculoma. Axial T2-weighted image shows a large hypointense tuberculoma (bold arrow) in the right basal ganglia with significant perilesional edema. MRS shows large lipid peak (arrow) at 1.33 ppm level.

accompanied by granulation tissue outside the dura mater.

5. *TB-IRIS*
   TB-IRIS is a commonly observed complication in human immunodeficiency virus (HIV) patients who are undergoing antiretroviral therapy (ART), characterized by an exaggerated immune reaction against MTB. Treatment-related manifestations of this condition can occur either during or after treatment. Diagnosis is made by assessing specific criteria, including initial improvement of symptoms or radiographic findings related to TB with appropriate anti-TB treatment. This is followed by a paradoxical decline in clinical symptoms and radiologic findings, either at the original sites or new locations. Other potential causes of clinical deterioration, such as noncompliance, drug malabsorption, and medication side effects, must be ruled out.
   Neurological symptoms may occur in 10% of cases.[30] The prevalence of TB-IRIS ranges from 2 to 23%.[31,32] IRIS usually occurs 20 to 50 days after the start of ATT and 2 to 4 weeks after beginning ART in HIV-infected patients.[33] Common neuroimaging findings in TB-IRIS include worsening of basal meningitis, presence of mass lesions, and infarction, without formation of tuberculomas. Without a history of ART, it would be challenging to differentiate basal meningeal enhancement from primary TBM or TB-IRIS. In such situations, comparing with previous scans and reviewing the patient's medical history is crucial. Treatment involves modifying antituberculous medications and incorporating anti-inflammatory agents, such as corticosteroids, into the antiretroviral treatment plan.[30]

Role of New Imaging Technique in TBM

1. *VWI*: Advanced imaging methods, such as VWI, allow for precise assessment of the submillimeter vessel wall and its associated diseases. VWI is performed using conventional sequences, including a pre- and postcontrast black-blood T1W sequence. TBM is known to cause arterial damage through various pathological processes, resulting in constriction of arteries located at the base of the brain (terminal internal carotid artery, M1 middle cerebral artery [MCA], perforator or MCA, and posterior cerebral artery) and smaller vessels.[34] VWI reveals vessel wall enhancement.
2. *MRS*: The MRS analysis of a tuberculoma may reveal a prominent lipid peak in the core, while the wall of the tuberculoma may show increased levels of choline and lipids, as well as a decrease in NAA and creatinine. Neurocysticercosis demonstrates increased levels of lactate, alanine, succinate, and choline, and decreased levels of NAA and creatine.[35] Short TE MRS (20–35 ms) provides superior resolution due to its high signal-to-noise ratio (►**Fig. 21**).
3. *Perfusion imaging*: ASL can be incorporated into standard imaging protocols to evaluate cerebral blood flow (CBF) in the brain without the need for contrast injection. In patients with TBM and basal exudates, there is a decrease in CBF in the basal ganglia and affected vessel territories due to the presence of exudates around the basal cisterns, indicating potential early tuberculous arteritis. As symptoms may be lacking in the initial stages of arteritis in nTB, ASL can aid in the early detection of hypoperfusion and prompt adjustments to treatment to prevent irreversible vascular damage.[36]
4. *Amide proton transfer*: Using the principles of chemical exchange saturation transfer and MT, amide proton transfer imaging creates tissue contrast by identifying the mobile amide protons found in intracellular proteins and native peptides. In tuberculomas, which can sometimes mimic gliomas, a lower MTR is observed compared to gliomas and other malignant lesions, indicating a lower concentration of mobile amide protons in the surrounding microenvironment. Placing the voxel in the wall of the tuberculoma allows for the detection of elevated MTR_{asym} values in the perilesional parenchyma, which may serve as a unique indicator of the inflammatory environment.[37]

Treatment Response after ATT and Follow-Up Imaging

Radiological evaluations following the initiation of treatment involve comparing previous CT and/or MRI scans. These assessments should be conducted at 6 and 12 weeks after treatment begins. Positive indicators of a favorable response include a decrease in edema, tuberculoma size, and enhancement, as well as the appearance of calcification and disappearance of small tuberculomas. Clinical and bacteriological markers can also be utilized. Serial imaging in patients on ATT may demonstrate a decrease in the size of the lesion within a period of 3 to 4 months and complete resolution after 12 months.[38] Additionally, dynamic CE MRI has shown that alternations in k^{trans} and v_e are correlated with therapeutic response, even in the presence of a paradoxical increase in lesion volume.

Conclusion

The timely identification and effective management of nTB is crucial in preventing the high mortality and morbidity associated with the disease. A thorough understanding of the different imaging manifestations of CNS TB is crucial for prompt diagnosis. The use of conventional imaging techniques, along with advanced MRI methods, can help accurately identify tuberculous lesions, leading to a more confident diagnosis.

Ethical Approval

This work has been approved by the Institute Ethical Committee (IEC).

Authors' Contributions

S.A., S.A.S., E.S., and B.D.C. contributed to the acquisition, analysis, conception, design, and drafting of the work. S.A., S.A.S., E.S., along with A.G. contributed to the final draft, revisions, upload, and submission of the final revised work. All authors have agreed both to be personally accountable for own contributions and ensured that questions related to the accuracy or integrity of any part of the work, even ones in which one was not personally involved, are appropriately investigated, resolved, and the resolution documented in the literature.

Conflict of Interest

None declared.

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