# Imaging Findings of Tuberculosis of the Brain and Its Coverings

# 13

Mohammad Ali Karimi, Morteza Sanei Taheri, and Ahmet T. Turgut

## Contents



## Abbreviations

| | |
|---|---|
| AIDS | Acquired immunodeficiency syndrome |
| CNS | Central nervous system |
| CSF | Cerebrospinal fluid |
| CT | Computed tomography |
| FLAIR | Fluid attenuation inversion recovery |
| MRA | Magnetic resonance angiography |
| MRI | Magnetic resonance imaging |
| MT | Magnetization transfer |
| TB | Tuberculosis |
| TBA | Tuberculous brain abscess |
| TBM | Tuberculous meningitis |

## 13.1 Introduction

Tuberculosis (TB) is a historical disease which has reemerged and considered as a major worldwide health problem due to increasing frequency of immunocompromising conditions such as diabetes, alcoholism, cancer, and acquired immunodeficiency syndrome (AIDS) in recent decades. *Mycobacterium tuberculosis* may involve any organ, and the lungs are the most common location; however, central nervous system (CNS) TB is the most tremendous form of this disease. CNS TB consists about 5–10% of all patients with TB and up to 20% of patients with AIDS-related TB [1–3].

CNS TB usually has a distant origin, e.g., the lung, and the organism reaches to the brain or meninges via hematogenous spread. Sometimes, it

M.A. Karimi, MD (✉) • M.S. Taheri, MD
Department of Radiology, Shohada-e-Tajrish Hospital, Shahid Beheshti University of Medical Sciences, Tehran, Iran
e-mail: mkarimidr@yahoo.com; saneim@gmail.com

A.T. Turgut, MD
Department of Radiology, Hacettepe University School of Medicine, Ankara, Turkey
e-mail: ahmettuncayturgut@yahoo.com


M. Turgut et al. (eds.), *Tuberculosis of the Central Nervous System*,
DOI 10.1007/978-3-319-50712-5_13

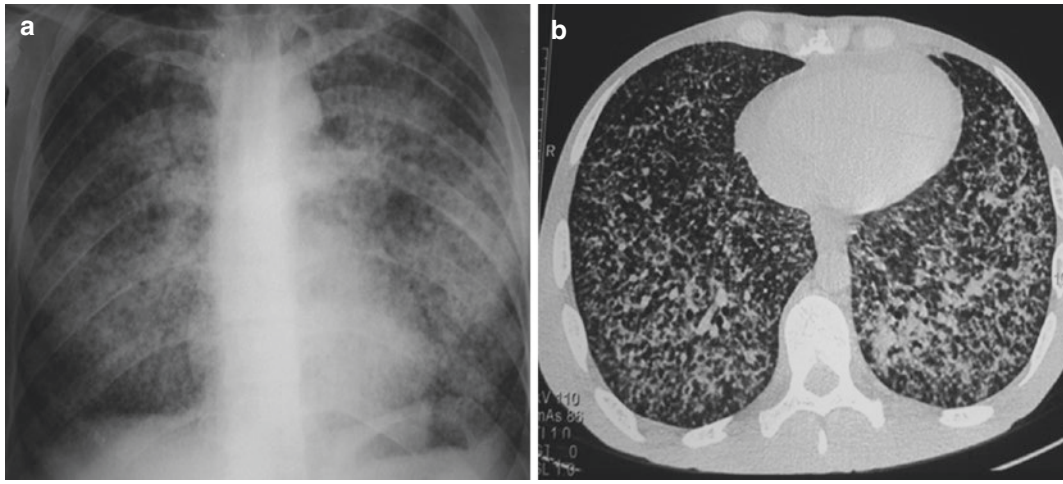


**Fig. 13.1** Miliary tuberculosis (TB). Frontal chest radiograph (**a**) shows bilateral diffuse fine tiny nodules. Axial high-resolution chest CT scan (**b**) demonstrates innumerable randomly distributed fine discrete nodules bilaterally.

may result from direct spread from intra- or extracranial foci [4]. Clinical and imaging manifestations of CNS TB may simulate other neurological diseases such as tumors and other infectious and noninfectious conditions [5]. Since CNS TB has no unique characteristics on neuroimaging and clinical manifestation, diagnosis of this disease remains a challenging issue. Therefore, familiarity with the radiological manifestations of CNS TB has a key role in timely and precise diagnosis of this disease. CNS TB may present as tuberculous meningitis (TBM), cerebritis, ventriculitis, cerebral abscesses, tuberculomas, miliary TB, and spinal and calvarial involvement [6–8]. In this chapter, we describe imaging findings of TB of the brain and its coverings.

## 13.2 Pulmonary Tuberculosis as a Diagnostic Clue

Most of patients with CNS TB have a diagnostic clue in their lung imaging, and sometimes a plain radiograph can help us to make a correct diagnosis. Therefore, we first briefly describe the imaging manifestations of pulmonary TB. Pulmonary TB may be primary or postprimary (reactivation). Primary form is common in children, and postprimary infection usually presents in adults.

Primary TB characteristically presents as hilar (and/or mediastinal) lymphadenopathy which is typically unilateral but may be asymmetrically bilateral. Other findings are pleural effusion (typically unilateral), miliary nodules, and consolidation (Fig. 13.1). In primary infection, the parenchymal disease and adenopathy may completely resolve, or there may be a residual focus of scarring or calcification [9, 10].

Postprimary TB typically involves apical and posterior segments of upper lobes and to a lesser degree superior segment of lower lobes. The main manifestations include cavitations, nodular infiltrations, typically as tree-in-bud appearance, multifocal patchy opacities, lymphadenopathies, and pleural effusion. Cavitations usually indicate active and transmissible disease (Fig. 13.2) [9, 10].

When we encounter patients with CNS TB, lung radiograph or computed tomography (CT) may show the sequels of old pulmonary TB, including parenchymal fibrosis, architectural distortion, bronchiectasis, and volume loss in the upper lobes.

## 13.3 Tuberculous Meningitis

Tuberculous Meningitis (TBM) is the most common type of CNS TB. It is most frequent in children or immunocompromised individuals [11, 12].

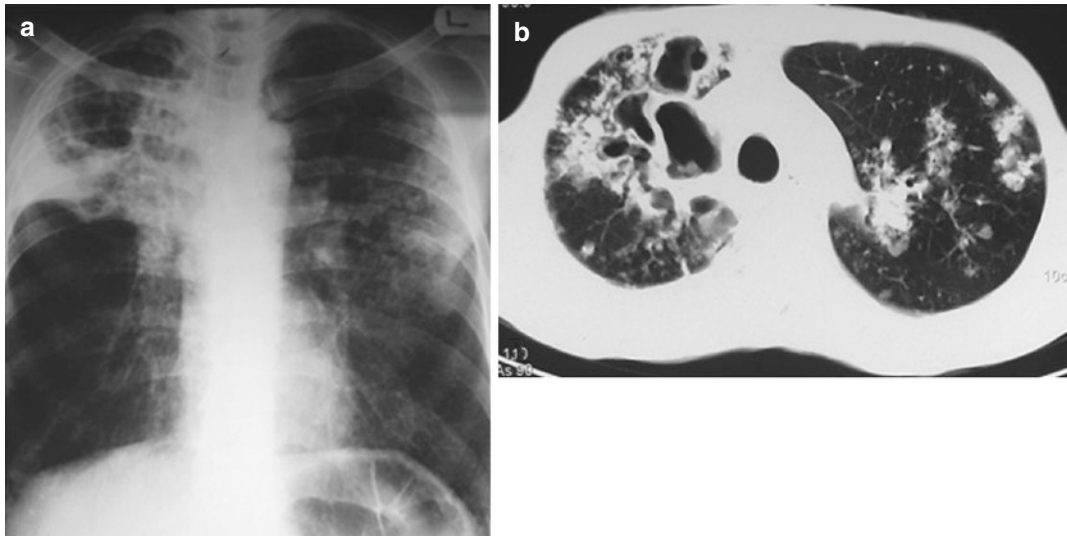


**Fig. 13.2** Reactivated pulmonary TB. (**a**) Frontal chest radiograph shows typical location of TB involvement in *right* upper lobe with cavitary consolidation and volume loss. There are also some nodular opacities in *left* upper lobe. (**b**) Axial chest CT scan of another HIV-positive patient demonstrates multiple cavities in *right* upper lobe associated with clumped nodular and some linear opacities in both upper lobes

TBM results from hematogenous spread of *M. tuberculosis*; however, it may also result from spread of adjacent focus (Rich focus) in cortical subpial or subependymal into the subarachnoid spaces or into the ventricular system [13]. Insidious course of TBM along with its nonspecific manifestations may result from misdiagnosis or delayed diagnosis of TBM. Hence, high index of suspicion and early imaging play important role in the timely detection of TBM and thereby reducing its morbidity and mortality rate.

Radiologically, the most common finding is enhancing exudate in the basal cisterns. This is a relatively specific neuroimaging finding of leptomeningeal TB on CT and magnetic resonance images (MRIs) [14]; however, sometimes this feature may also be seen in meningitis due to other infectious including fungal ones and granulomatous (sarcoidosis) and neoplastic (lymphoma; carcinomatosis) diseases [1]. The tuberculous exudate is composed of bacilli and the host immune cells.

The most sensitive imaging finding of tubercular meningitis is meningeal enhancement that has been reported in up to 90% of patients [7, 14, 15]. The subpial exudate is commonly located in the lateral cerebral fossa and the sylvian fissure, inferomedial surface of the frontal lobes, the anteromedial surface of the temporal lobes, the superior aspect of the cerebellum, and the floor of the third ventricle [16]. Sometimes, extension to adjacent areas such as suprasellar, pontomesencephalic, or interpeduncular cisterns may also occur [17]. Meningeal enhancement over the cerebral convexities and the sylvian fissures is another common finding. Involvement of ependymal surfaces of the ventricles usually occurs in the later stages of the TBM [1, 17, 18]. In later stages, also there may be widening of subarachnoid spaces.

CT images of TBM usually demonstrate obliteration of basal cisterns by exudates of iso- to mild hyperattenuation [1, 6, 16, 19]. MRI is more sensitive than CT for detection of the findings, especially post-contrast MRIs which will demonstrate the leptomeningeal enhancement and enhancing cisternal exudates (Figs. 13.3, 13.4, and 13.5) [6].

Some studies reported that in comparison to contrast-enhanced T1-weighted images, post-contrast fluid attenuation inversion recovery (FLAIR) images show a higher specificity for discovery of leptomeningeal enhancement [20]. Also, contrast-enhanced magnetization transfer

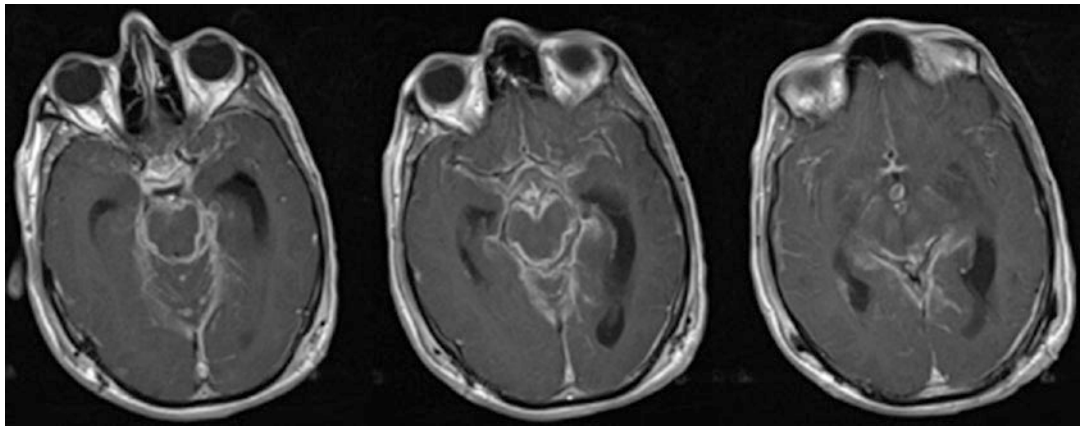

**Fig. 13.3** Meningeal TB. Axial post-contrast T1-weighted MRIs demonstrate enhancing basilar exudates and leptomeningeal enhancement

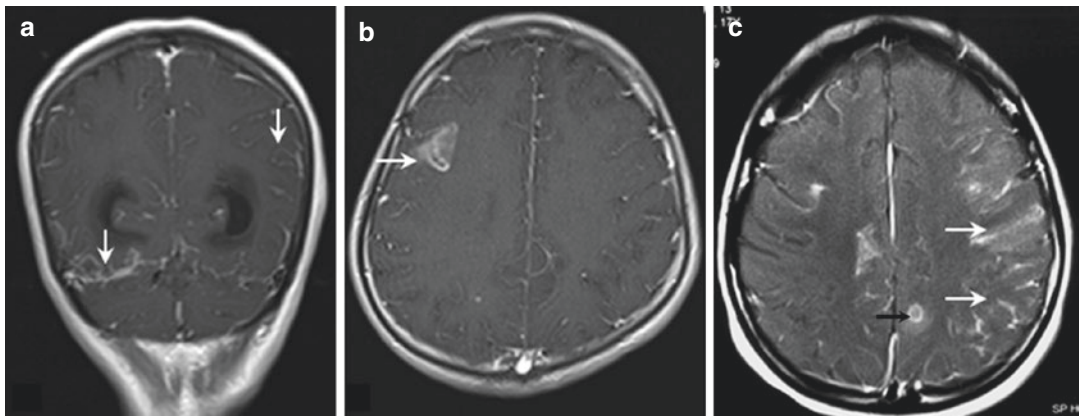


**Fig. 13.4** Meningeal TB. Coronal (**a**) and axial (**b**, **c**) post-contrast T1-weighted MRIs show leptomeningeal enhancement (*white arrows*). A small tuberculoma in *left* parasagittal parietal region (*black arrow* in **c**) is also evident

(MT) imaging is superior to the conventional post-contrast imaging in detecting meningeal involvement [11]. As mentioned above, TBM imaging findings may overlap with other conditions; this form of meningeal enhancement may be seen in carcinomatous meningitis, other infective meningitis, and inflammatory diseases such as rheumatoid arthritis or sarcoidosis [11].

Progressive hydrocephalus, cranial neuropathies, infarction, and vasculitis with their own imaging features are the complications of TBM which may alter the face of the disease [8, 12]. Obstruction of cerebrospinal fluid (CSF) flow in the basal cisterns results to the most common complication of TBM, communicating hydrocephalus (Fig. 13.6) [1, 4, 8, 11]. Noncommunicating hydrocephalus may be seen in some cases which is due to obstruction by tuberculoma or TB abscess.

Ischemic infarct (Fig. 13.6) secondary to vascular compression and obstruction of perforating vessels (necrotizing arteritis) [17, 21, 22], especially the lenticulostriate and thalamoperforating arteries, is also a common complication with detection rate of 20–40% of patients. This event is seen mostly in the basal ganglia or internal capsule which is enriched by vessels that perfuse the so-called medial TB zone [19]. Dural venous sinus thrombosis and secondary hemorrhagic infarct may be seen in patients with TBM. We encountered a patient with CNS TB in our institute in which the only finding was dural venous sinus thrombosis (Fig. 13.7).

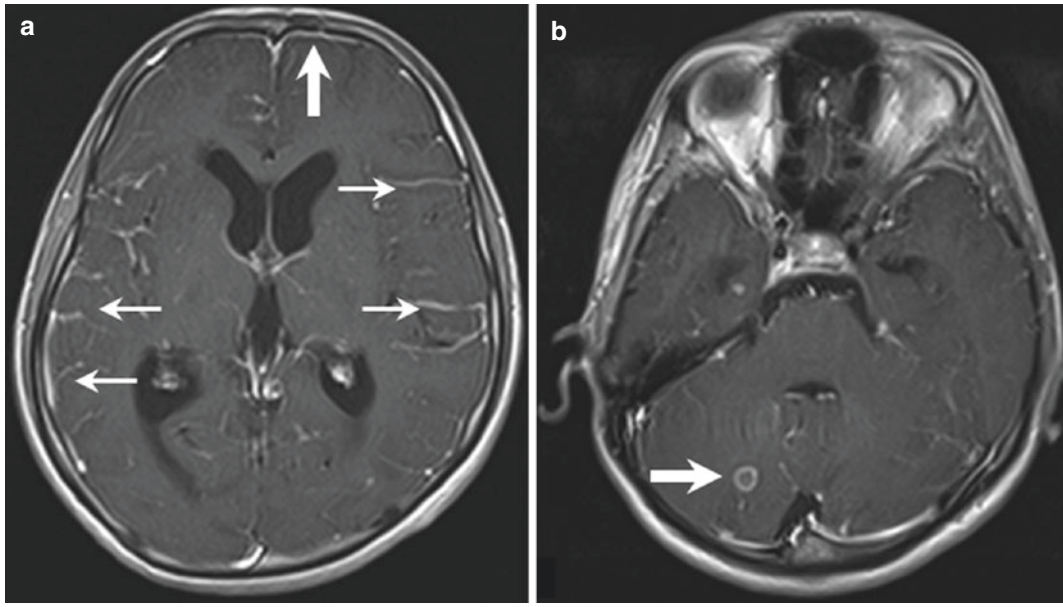


**Fig. 13.5** Meningeal TB. (**a**) Leptomeningeal (*small arrows*) and dural pachymeningeal (*large arrow*) enhancements are seen in this axial post-contrast T1-weighted MRIs. (**b**) A small tuberculoma is seen in *right* cerebellar hemisphere (*arrow*)

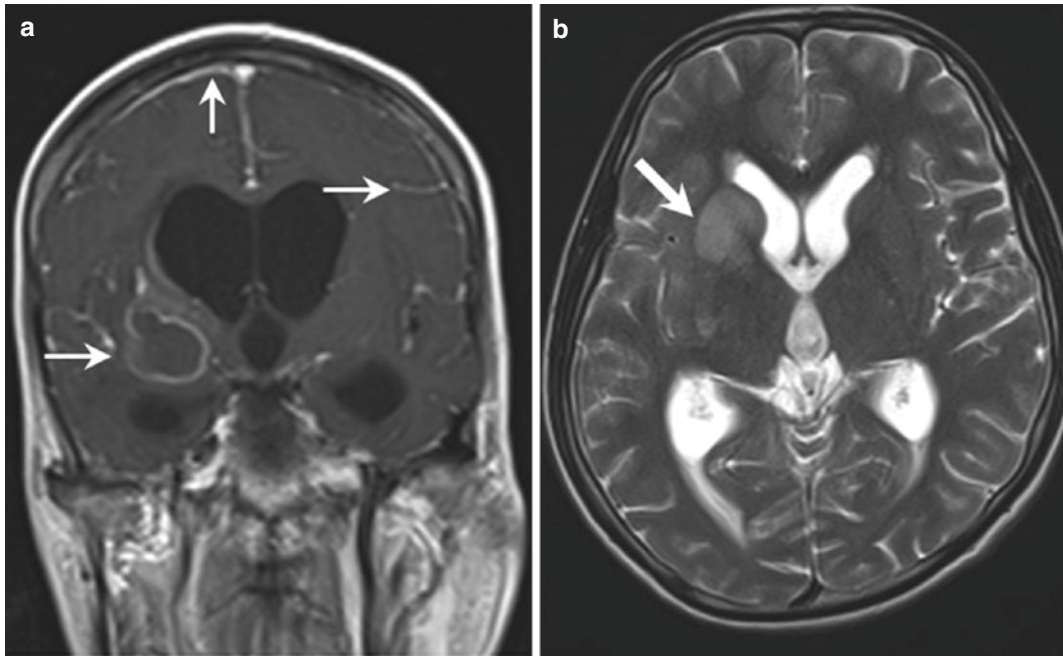


**Fig. 13.6** Complications of meningeal TB. (**a**) Coronal post-contrast T1-weighted MRI demonstrates leptomeningeal and pachymeningeal enhancement (*arrows*). Hydrocephalus is also seen which is secondary to the obstruction of cerebrospinal fluid (CSF) flow in the basal cisterns. (**b**) Axial T2-weighted MRI of another patient with TBM shows a lesion in *right* basal ganglia due to ischemic infarct

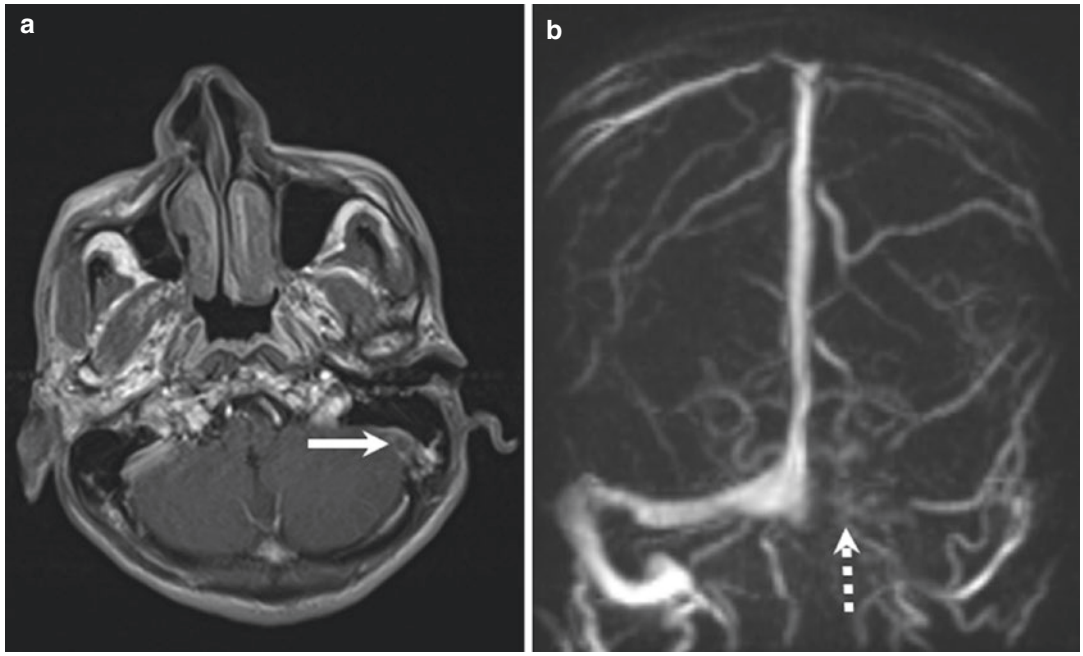


**Fig. 13.7** Dural venous sinus thrombosis. (**a**) Axial post-contrast T1-weighted MRI demonstrates a filling defect within *left* sigmoid sinus (*white arrow*). (**b**) MRA reveals nonvisualization of transverse and sigmoid sinuses in *left* side (*white arrow*). This was the only imaging evidence of TBM in a man presented with headache. Because the patient was an immigrant from an endemic area for TB, we assessed CSF PCR for *M. tuberculosis* which revealed a positive result

TBM may also complicate by cranial nerve involvement which occurs in 17–40% of cases. It occurs secondary to ischemia, vascular compromise, or nerve entrapment within basal exudates. Second, third, fourth, and seventh cranial nerves are the most commonly involved nerves [16, 18]. MRI is the preferred imaging for evaluation of the affected cranial nerves; they usually appear as thickened enhanced nerves, particularly in proximal segments. These thickened nerves have high signal intensity on T2-weighted images.

## 13.4 Brain Parenchymal Tuberculosis

The most common form of brain parenchymal TB disease is tuberculoma. Other forms include cerebritis, cerebral abscess, miliary TB, or TB encephalopathy. Parenchymal disease may occur with or without TBM.

### 13.4.1 Cerebritis and Cerebral Abscess

Some parenchymal TB is associated with concomitant TBM, and sometimes it occurs without accompanying meningitis. Cerebritis refers to pyogenic inflammation of the brain parenchyma and may lead to abscess formation in the setting of inadequate or incorrect treatment. TB cerebritis or abscess may mimic pyogenic bacterial infection on CT or MRI.

TB cerebritis is infrequent and usually appears as a single or multiple focal ill-defined hypoattenuated lesion(s) on CT images. These lesions appear hyposignal on T1-weighted and hypersignal on T2-weighted images. On post-contrast MRI, areas of patchy enhancement may be seen (Fig. 13.8) [6, 23].

Tuberculous brain abscess (TBA) (Fig. 13.9) is also infrequent and consists of a central zone of pus and liquefied material. The abscess may be single or multiple and often has multilocu-

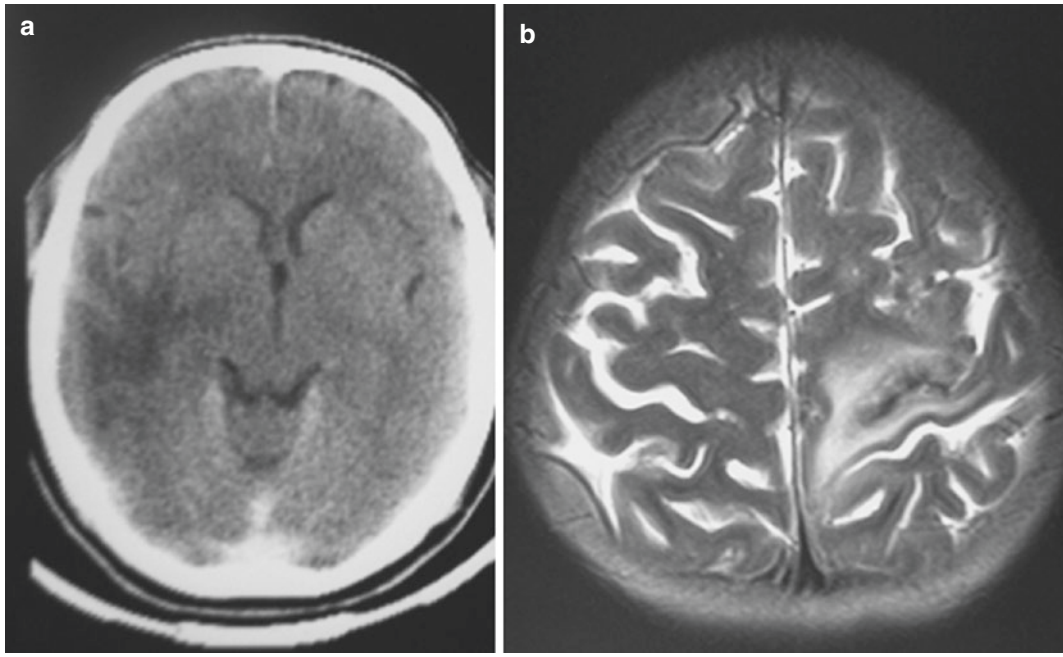


**Fig. 13.8** TB cerebritis. (**a**) Axial post-contrast CT image demonstrates an irregular hypodensity in *right* temporal region with faint peripheral enhancement in anterior part. (**b**) Axial T2-weighted MRI in another patient shows nonspecific hypersignality in *left* frontoparietal region

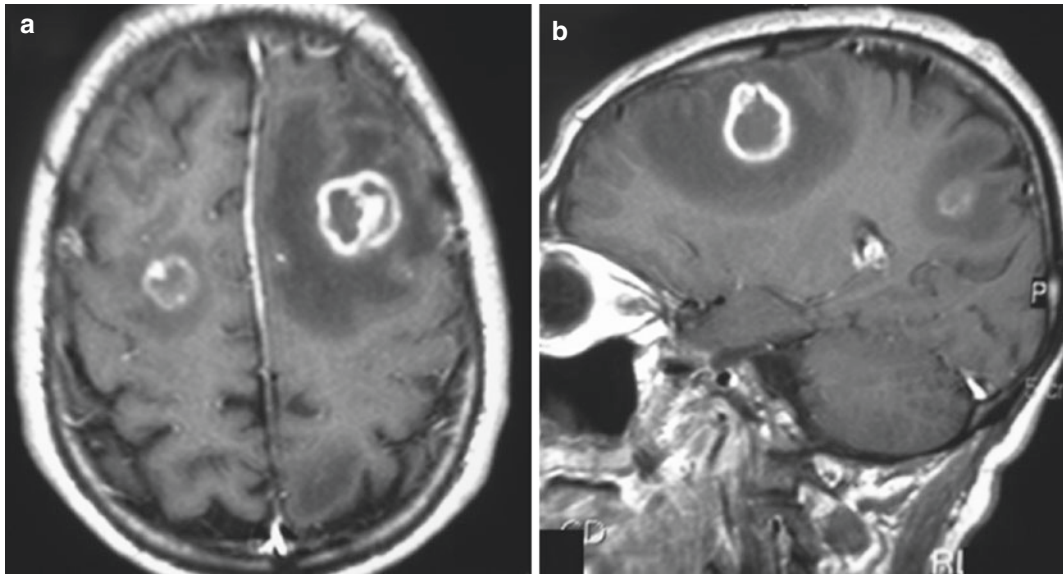


**Fig. 13.9** Tuberculous brain abscess (TBA). Axial (**a**) and sagittal (**b**) post-contrast T1-weighted MRIs show multiple TBAs as irregular thick ringlike enhancing lesions in both frontal lobes and *left* parietal region with peripheral edema

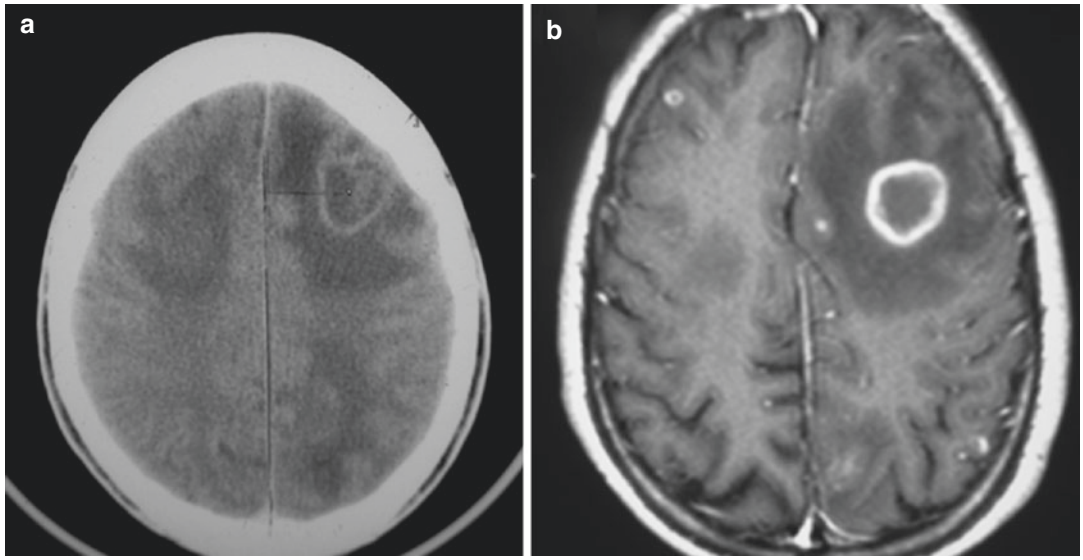


**Fig. 13.10** TBA. (**a**) Axial post-contrast CT image shows ring-enhancing lesions in *left* frontal lobe with marked peripheral edema. (**b**) Axial post-contrast T1-weighted MRI in another patient also reveals a lesion in *left* frontal lobe with thick ring enhancement and marked peripheral edema and mass effect leading to midline shift

lar appearance [15]. On CT images the TBA presents as hypodense round or multiloculated lesion(s) accompanied with peripheral edema. It may show some mass effect. Since the abscess contains a central necrotic and liquefied zone, it shows an increased signal intensity on T2-weighted images. Ring enhancement is a dominant feature in post-contrast CT or MRIs (Fig. 13.10). This enhancement is often thin and uniform. However, it may be irregular and thick, particularly in immunodeficient patients [1, 3, 7, 8, 24, 25].

Some studies have shown that application of MT spine echo images make the CNS TB lesions more obvious [26–31]. By increasing the detectability of the lesions, MT images improve the assessment of extent of CNS TB. MT ratio measurements on MRI help to distinguish CNS TB from other infectious brain lesions; MT ratios in TB are lower than those in pyogenic infections and higher than those in viral infections, with the difference related to variations in protein content [5]. Furthermore, on MR spectroscopy, unlike the pyogenic abscess, the peak of amino acids is not a usual finding in TBA [26, 31].

### 13.4.2 Tuberculoma

Tuberculoma is the most common form of parenchymal involvement in CNS TB. The lesion may be single or multiple and may be seen in any part of the intracranial space. Tuberculoma and TBM may occur concomitantly (Fig. 13.11).

Unlike the TBA which contains central area of pus, tuberculoma has a necrotic caseous center. Its peripheral capsule contains Langerhans cells, epithelioid cells, fibroblasts, and lymphocytes [32]. Tuberculoma in nonenhanced CT images may be isodense, hyperdense, or of mixed density. On contrast-enhanced CT, tuberculoma shows a ringlike enhancement. It may also exhibit irregular or nodular nonhomogeneous enhancement. Some studies have shown that presence of a central calcification within a ringlike enhancement (target sign) is suggestive of tuberculoma [22]. T2-weighted or FLAIR images show a mixed intensity lesion (mainly low signal) which contains a central zone of high signal intensity [33]. The central caseating necrosis makes high signal intensity zone, and peripheral collagenous capsule presents as low signal intensity rim. The capsule is a layer with low water content and high protein content [33].

Surrounding edema may be seen as high signal intensity edema. Post-contrast MRIs often show a ring enhancement (Figs. 13.12 and 13.13).

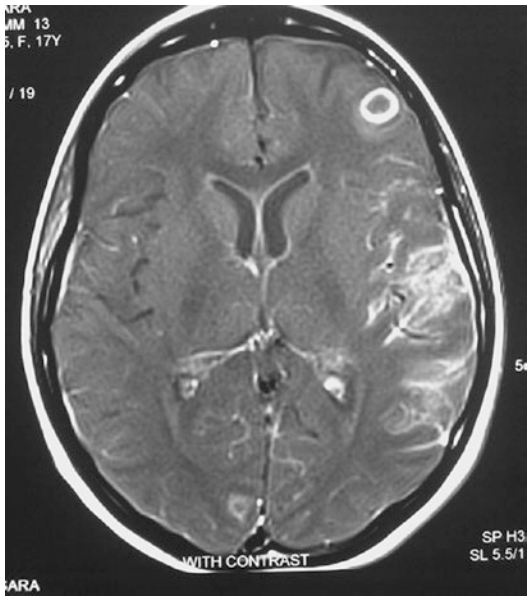


**Fig. 13.11** Tuberculoma with TBM. Axial post-contrast T1-weighted image through the lateral ventricles shows a tuberculoma as a cortical ring-enhancing lesion in *left* frontal lobe. There is also another small tuberculoma in *right* occipital lobe. Dural and leptomeningeal enhancements in *left* frontoparietal region indicate associated TBM

Caseating solid granulomas are hypointense on T1-weighted images and strikingly hypointense on T2-weighted images. This hypointensity is due to the granulation tissue and a compact cellularity which is higher than that of brain parenchyma. These typical imaging findings are not seen in noncaseating granulomas; they usually are hypointense to isointense on T1-weighted and hyperintense on T2-weighted images. On post-contrast images homogeneous enhancement is typical [8].

Evaluation of response to medical treatment can be performed by follow-up CT or MRI studies. Regression of the lesions is not a role, and some patients who receive suitable treatment may show paradoxical enlargement of a tuberculoma. Sometimes, a new intracranial and spinal tuberculoma may be seen. Nevertheless, with continuation of anti-TB treatment, the lesions often eventually resolve [33, 34].

Occasionally, healed tuberculomas appear as calcified points on noncontrast CT (Fig. 13.14). Also, several years after healed TBM, calcifications within basal cisterns or brain sulci may be seen [35]. On MR spectroscopy tuberculomas demonstrate lipid level peaks at 0.9 ppm, 1.3 ppm, 2.0 ppm, and 2.8 ppm; these peaks are due to presence of the high lipid content of the mycolic

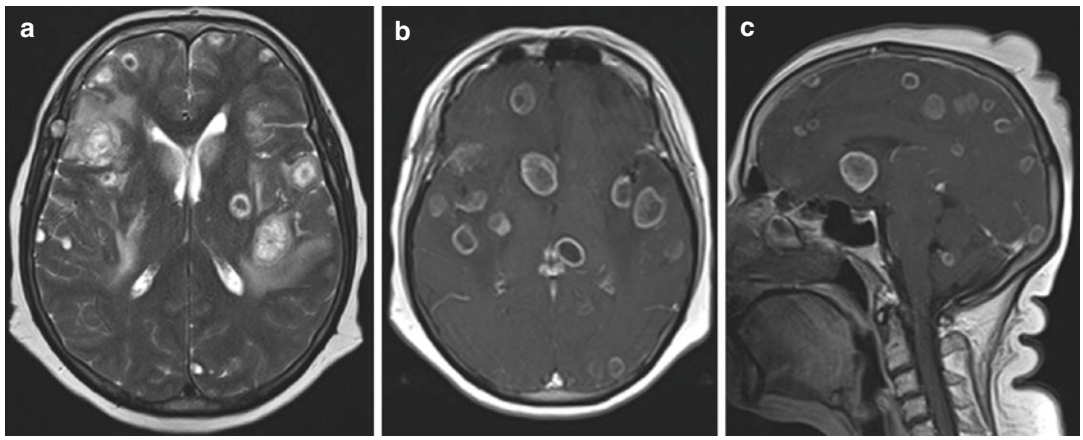


**Fig. 13.12** Multiple tuberculomas. (**a**) T2-weighted image through the lateral ventricles shows lesions with hypointense core and peripheral hyperintensity in bilateral frontotemporal regions. Axial (**b**) and sagittal (**c**) post-contrast T1-weighted images show scattered bilateral cerebral tuberculomas as ring-enhancing lesions without peripheral edema. A cerebellar tuberculoma is also seen (**c**)

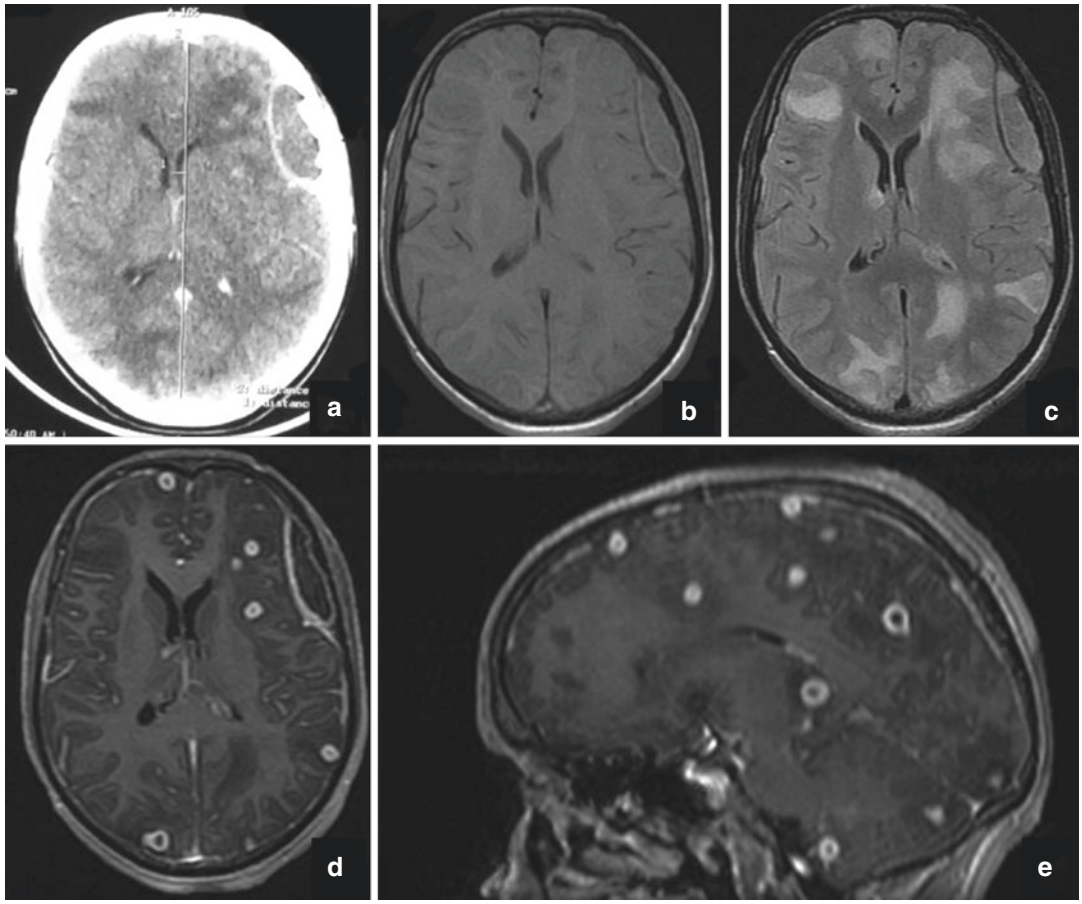


**Fig. 13.13** Contrast-enhanced CT of the head showing multiple enhancing lesions with perilesional edema and an extra-axial collection in *left* frontal region, with suggestion of midline shift to the *right* (**a**). The extra-axial collection is iso- to hypointense on T1 W (**b**) and T2-FLAIR (**c**). SPGR-contrast images (axial, **d**; sagittal, **e**) show multiple ring lesions (tuberculomas) with an extra-axial enhancing collection (en plaque tuberculoma) (Courtesy of R. K. Garg, M.D.)

acid in the mycobacterial cell wall. In contrast to pyogenic abscesses which show amino acid resonances at 0.9 ppm at MR spectroscopy, this feature is not seen in TBAs [5].

### 13.4.3 Miliary Tuberculosis

Brain miliary TB is a very rare form of TB which is seen mainly in severely immunodeficient patients. It is often accompanied with meningitis or an extracranial primary TB infection [36]. The dissemination is always hematogenous, and therefore the military lesions often lodge at the corticomedullary junctions.

Miliary tuberculomas are usually tiny (2–3 mm) dispersed lesions. The lesions usually are not seen on CT images and may be also invisible on noncontrast MRIs. In visible lesions, MRI shows small lesions that are hypointense on T2-weighted sequences. These lesions occasionally can be hardly seen as small hypodense foci on CT images [16]. Post-contrast MRIs usually demonstrate innumerable homogeneously enhancing small round lesions (usually ring enhancing) (Fig. 13.15) [26]. MT spin echo

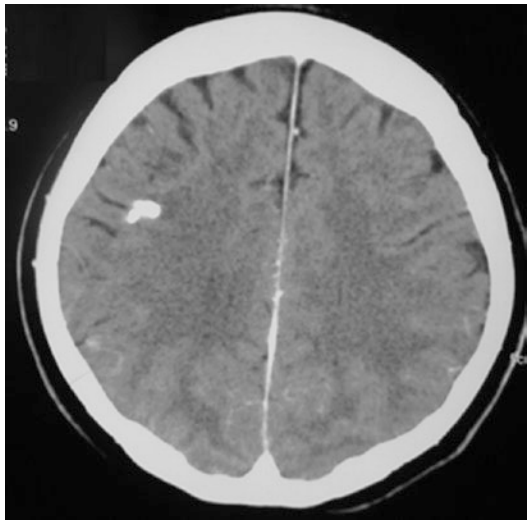

**Fig. 13.14** Treated tuberculoma. Axial CT image in a patient with history of treated TB shows a calcified lesion in *right* frontal lobe without edema or mass effect

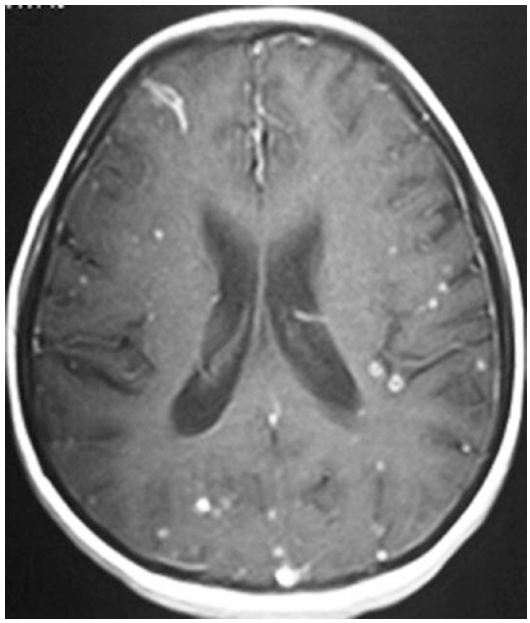

**Fig. 13.15** Miliary brain TB. Axial post-contrast T1-weighted image shows numerous bilateral tiny enhancing nodules. This young female patient presented with chronic cough, headache, dizziness, nausea, and vomiting

T1-weighted images (with or without contrast) can detect invisible lesions on routine MRIs even those without enhancement [27].

### 13.4.4 Tuberculous Encephalopathy

This type of CNS TB characteristically involves the young children. These patients may present with neurologic signs such as convulsion, stupor, and coma. There may be no signs of focal neurological deficit or meningeal irritation. Severe cerebral edema, which may be unilateral or bilateral, is the main neuroimaging finding. Sometimes, hypoattenuating areas on CT images and hyperintensity areas on T2-weighted MRIs may be seen due to myelin loss in the white matter [8, 13, 37].

## 13.5 Tuberculous Ventriculitis

In addition to the subarachnoid space and brain parenchyma, *M. tuberculosis* can infect the ventricles called "ventriculitis." Despite high incidence of TBM in endemic areas, there are a few reports of TB ventriculitis [38, 39], and it seems to be underestimated [38]. Main CT and MRI findings include intraventricular debris (mostly in occipital horns), ventricular dilatation, periventricular edema, subependymal (or choroidal) enhancement, and restricted diffusion (Fig. 13.16). Singh et al. [38] reported five patients of TB ventriculitis. Enhancement of ependymal wall of lateral ventricle or fourth ventricle was seen on MRIs of these patients along with restricted diffusion and hydrocephalus. Intraventricular septations, sequestered ventricles, and ventricular sludge were also seen in some patients. They concluded that sequestered ventricles (intraventricular septations) and enhanced or hyperintense ependymal wall on MT images are suggestive for TB ventriculitis.

## 13.6 Miscellaneous Forms of CNS Tuberculosis

Beside the abovementioned presentations, CNS TB may present as other forms such as subdural or epidural abscess, spinal or spinal cord

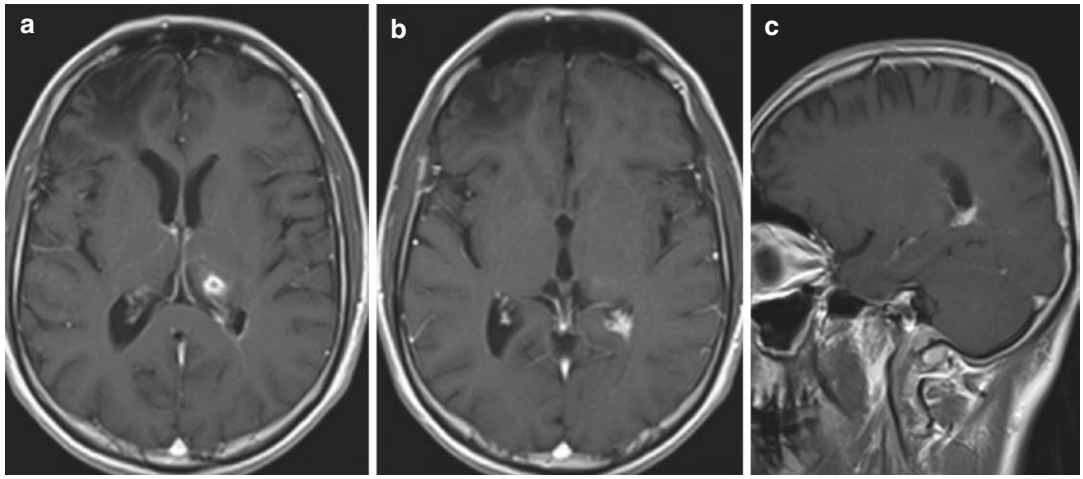


**Fig. 13.16** Axial (**a**, **b**) and sagittal (**c**) post-contrast T1-weighted MRIs demonstrate marked enhancement in posterior horn of *left* lateral ventricle and adjacent subtle ventricular wall enhancement. There is also a ring-enhancing tuberculoma in *left* thalamus

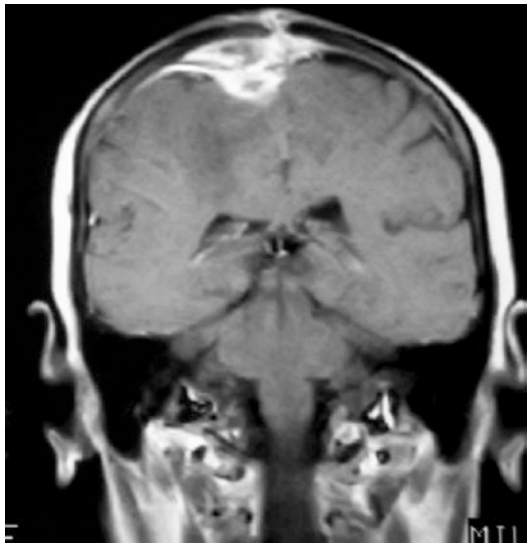

**Fig. 13.17** TB epidural and subdural empyema. Coronal post-contrast T1-weighted MRI demonstrates dural enhancement and epidural and subdural collections with thick enhancing walls in *right* frontal region. The epidural component extended to the *left* side. Involvement of diploe indicates calvarial involvement

TB, and calvarial TB. Intracranial subdural or epidural abscess may be seen with or without a primary CNS TB. Imaging features of the abscess are similar to that of other pyogenic abscesses, i.e., isosignal to hyposignal intensity on T1-weighted images and hypersignal or mixed signal intensity on T2-weighted images. These lesions typically show rim enhancement on post-contrast MRI or CT images (Fig. 13.17) [33]. Atypical imaging presentation of CNS TB is not infrequent, and brain TB sometimes mimics a mass lesion leading to unnecessary surgery (Fig. 13.18).

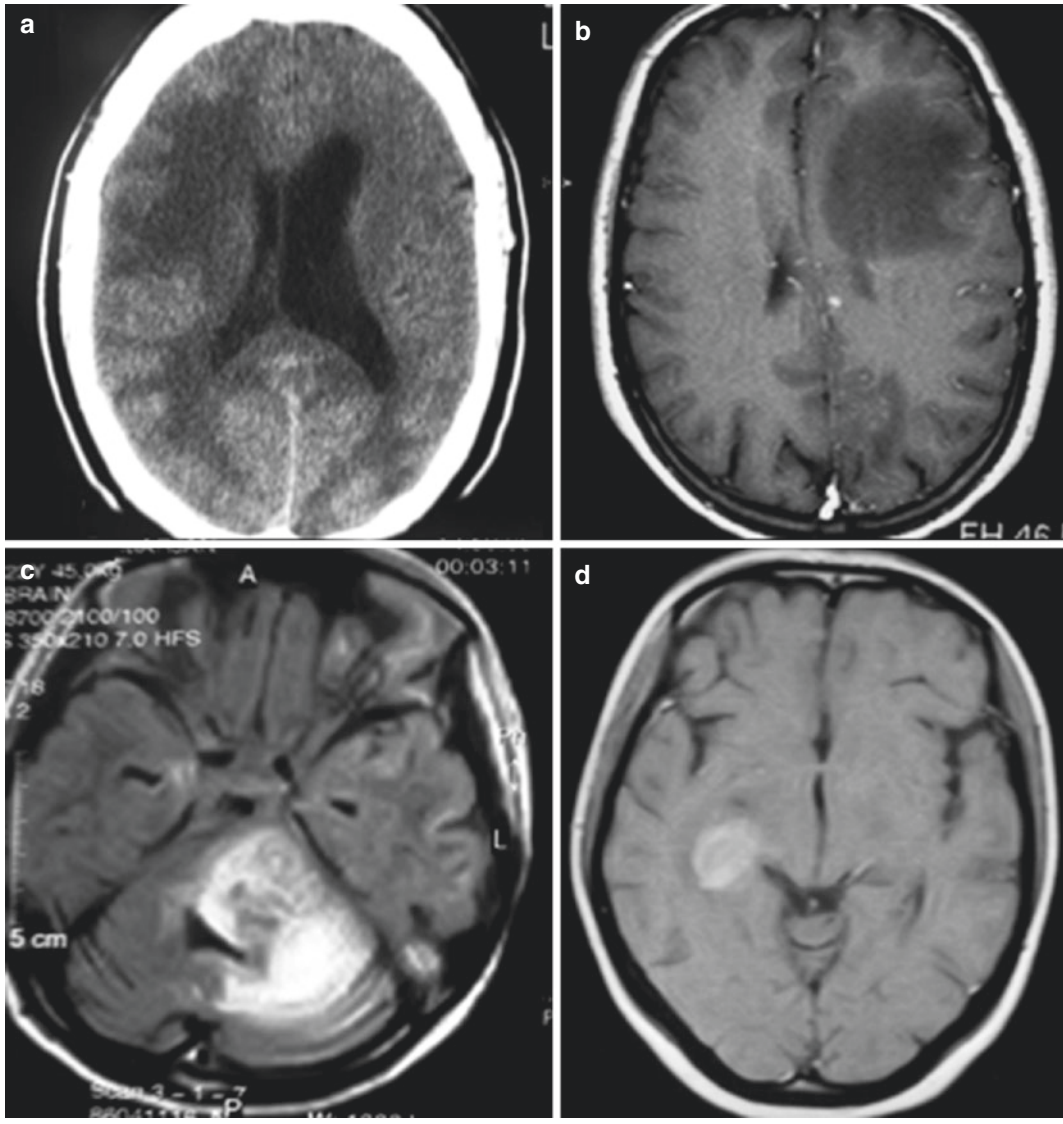


**Fig. 13.18** Atypical presentations of CNS TB. (**a**–**d**) These are four different patients with CNS TB mimicking cerebral or cerebellar mass lesions. In case of (**c**) the diagnosis of TB was made after a complicated surgery

## Conclusion

TBM is the most common form of CNS TB, followed by tuberculoma. Enhancing exudate in the basal cisterns is the hallmark of TBM. Tuberculoma often presents as single or multiple ring-enhancing lesion(s) with or without evidence of TBM. Diagnosis of CNS TB is a challenging issue because of its insidious course and nonspecific clinical presentations and also sometimes atypical imaging presentations such as a mass-like lesion. Therefore, the neuroimaging plays a key role in early diagnosis of this curable disease and reducing its morbidity and mortality rates.

## References


1. Bernaerts A, Vanhoenacker FM, Parizel PM, Van Goethem JW, Van Altena R, Laridon A, De Roeck J, Coeman V, De Schepper AM (2003) Tuberculosis of the central nervous system: overview of neuroradiological findings. Eur Radiol 13:1876–1890
2. Berenguer J, Moreno S, Laguna F, Vicente T, Adrados M, Ortega A, González-LaHoz J, Bouza E (1992) Tuberculous meningitis in patients infected with the human immunodeficiency virus. N Engl J Med 326:668–672
3. Vidal JE, Penalva de Oliveira AC, Bonasser Filho F, Schiavon Nogueira R, Dauar RF, Leite AG, Lins DL, Coelho JF (2005) Tuberculous brain abscess in AIDS patients: report of three cases and literature review. Int J Infect Dis 9:201–207
4. Rock RB, Olin M, Baker CA, Molitor TW, Peterson PK (2008) Central nervous system tuberculosis: pathogenesis and clinical aspects. Clin Microbiol Rev 21:243–261
5. Shih RY, Koeller KK (2015) Bacterial, fungal, and parasitic infections of the central nervous system: radiologic-pathologic correlation and historical perspectives. Radiographics 35:0000–0000
6. Burrill J, Williams CJ, Bain G, Conder G, Hine AL, Misra RR (2007) Tuberculosis: a radiologic review. Radiographics 27:1255–1273
7. Bathla G, Khandelwal G, Maller VG, Gupta A (2001) Manifestations of cerebral tuberculosis. Singapore Med J 52:124–130
8. Ahluwalia VV, Sagar GD, Singh TP, Arora N, Narayan S, Singh M (2013) MRI spectrum of CNS tuberculosis. JIACM 14:83–90
9. Harisinghani MG, McLoud TC, Shepard JA, Ko JP, Shroff MM, Mueller PR (2000) Tuberculosis from head to toe. Radiographics 20:449–470
10. Bhalla AS, Goyal A, Guleria R, Gupta AK (2015) Chest tuberculosis: Radiological review and imaging recommendations. Indian J Radiol Imaging 25:213–225
11. Raviglioni MC, Snider DE, Kochi A (1995) Global epidemiology of tuberculosis: morbidity and mortality of a worldwide epidemic. JAMA 273:220–226
12. Andronikou S, Wilmshurst J, Hatherill M, VanToorn R (2006) Distribution of brain infarction in children with tuberculous meningitis and correlation with outcome score at 6 months. Pediatr Radiol 36:1289–1294
13. Dastur D, Manghani D, Udani PM (1995) Pathology and pathogenetic mechanisms in neurotuberculosis. Radiol Clin North Am 33:733–750
14. Andronikou S, Smith B, Hatherhill M, Douis H, Wilmshurst J (2004) Definitive neuroradiological diagnostic features of tuberculous meningitis in children. Pediatr Radiol 34:876–885
15. Uysal G, Köse G, Güven A, Diren B (2001) Magnetic resonance imaging in diagnosis of childhood central nervous system tuberculosis. Infection 29:148–153
16. Arbeláez A, Medina E, Restrepo F, Castillo M (2004) Cerebral tuberculosis. Semin Roentgenol 39:474–481
17. Jinkins JR, Gupta R, Chang KH, Rodriguez-Carbajal J (1995) MR imaging of central nervous system tuberculosis. Radiol Clin North Am 33:771–786
18. Morgado C, Ruivo N (2005) Imaging meningoencephalic tuberculosis. Eur J Radiol 55:188–192
19. Shah GV (2000) Central nervous system tuberculosis: imaging manifestations. Neuroimaging Clin N Am 10:355–374
20. Parmar H, Sitoh YY, Anand P, Chua V, Hui F (2006) Contrast-enhanced FLAIR imaging in the evaluation of infectious leptomeningeal diseases. Eur J Radiol 58:89–95
21. Dastur DK, Lalitha VS, Udani PM, Parekh U (1970) The brain and meninges in tuberculous meningitis - gross pathology in 100 cases and pathogenesis. Neurology 18:86–100
22. Whiteman ML (1997) Neuroimaging of central nervous system tuberculosis in HIV-infected patients. Neuroimaging Clin N Am 7:199–214
23. Rath TJ, Hughes M, Arabi M, Shah GV (2012) Imaging of cerebritis, encephalitis, and brain abscess. Neuroimaging Clin N Am 22:585–607
24. Sharma P, Garg RK, Verma R, Singh MK, Shukla R (2011) Incidence, predictors and prognostic value of cranial nerve involvement in patients with tuberculous meningitis: a retrospective evaluation. Eur J Intern Med 22:289–295
25. Garg RK (1999) Tuberculosis of central nervous system. Postgrad Med J 75:133–140
26. Trivedi R, Saksena S, Gupta RK (2009) Magnetic resonance imaging in central nervous system tuberculosis. Indian J Radiol Imaging 19:256–265
27. Gupta RK, Husain N, Kathuria MK, Datta S, Rathore RK, Husain M (2001) Magnetization transfer MR imaging correlation with histopathology in intracranial tuberculomas. Clin Radiol 56:656–663
28. Saxena S, Prakash M, Kumar S, Gupta RK (2005) Comparative evaluation of magnetization transfer contrast and fluid attenuated inversion recovery sequences in brain tuberculoma. Clin Radiol 60:787–793
29. Gupta RK, Kathuria MK, Pradhan S (1999) Magnetization transfer MR imaging in CNS tuberculosis. AJNR Am J Neuroradiol 20:867–875
30. Gupta R (2002) Magnetization transfer MR imaging in central nervous system infections. Indian J Radiol Imaging 12:51–58
31. Gupta RK, Vatsal DK, Husain N, Chawla S, Prasad KN, Roy R, Kumar R, Jha D, Husain M (2001) Differentiation of tuberculous from pyogenic brain abscesses with in vivo proton MR spectroscopy and magnetization transfer MR imaging. AJNR Am J Neuroradiol 22:1503–1509
32. Kim TK, Chang KH, Kim CJ, Goo JM, Kook MC, Han MH (1995) Intracranial tuberculoma: comparison of MR with pathologic findings. AJNR Am J Neuroradiol 16:1903–1908
33. Ku BD, Yoo SD (2009) Extensive meningeal and prenchymal calcified tuberculoma as long-term residual sequelae of tuberculous meningitis. Neurol India 57:521–522

34. Afghani B, Lieberman JM (1994) Paradoxical enlargement or development of intracranial tuberculomas during therapy: case report and review. Clin Infect Dis 19:1092–1099
35. Neumann R, Pohanka P, Prunyi E (1963) Intracranial calcification following tuberculous meningitis in children. Acta Radiol 18:487–492
36. Krishnan N, Robertson BD, Thwaites G (2010) The mechanisms and consequences of the extrapulmonary dissemination of *Mycobacterium tuberculosis*. Tuberculosis 90:361–366
37. Udani PM, Dastur DK (1970) Tuberculous encephalopathy with and without meningitis clinical features and pathological correlations. J Neurol Sci 10:541–561
38. Singh P, Paliwal VK, Neyaz Z, Srivastava AK, Verma R, Mohan S (2014) Clinical and magnetic resonance imaging characteristics of tubercular ventriculitis: an under-recognized complication of tubercular meningitis. J Neurol Sci 342:137–140
39. Kumar S, Kumar R, Radotra BD, Singh M (2014) Tubercular ventriculitis: an uncommon entity. Indian J Pediatr 81:608–610