

Radiological manifestations of central nervous system tuberculosis

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Abstract

Central nervous system (CNS) tuberculosis is a potentially life-threatening condition which is curable if the correct diagnosis is made in the early stages. Its clinical and radiologic manifestations may mimic other infectious and non infectious neurological conditions. Hence, familiarity with the imaging presentations of various forms of CNS tuberculosis is essential in timely diagnosis, and thereby reducing the morbidity and mortality of this disease. Early diagnosis of CNS TB is necessary for appropriate treatment and subsequently decreases the morbidity and mortality associated with the disease. Routine diagnostic techniques involve culture and immunological tests of the tissue and biofluids, which are time-consuming and may delay definitive management. Non invasive imaging modalities such as computed tomography (CT) scan and magnetic resonance imaging (MRI) are routinely used in the diagnosis of neurotuberculosis, with MRI offering greater inherent sensitivity and specificity than CT scan. In this review, we describe the imaging characteristics of the different forms of CNS tuberculosis, including meningitis, tuberculoma, miliary tuberculosis, abscess, cerebritis, and encephalopathy

Key Word: CNS tuberculosis, radiological manifestations, non- invasive imaging, early diagnosis.

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INTRODUCTION

Mycobacteria are small rod-shaped acid-fast bacilli with more than 125 species. Most TB is caused by mycobacterium tuberculosis. It causes more than 98% of the CNS tuberculosis. Less common species that are also considered part of the M. tuberculosis complex include M. africanum, M. microti, M. canetti and M. bovis. Tuberculosis has resurged and remained a major worldwide health problem. Although Mycobacterium tuberculosis can involve any organ, most commonly the lung, central nervous system (CNS) tuberculosis is the

most devastating form of the disease. Approximately 5–10% of all patients with tuberculosis and up to 20% of patients with AIDS-related tuberculosis have CNS involvement^{1,3}. CNS tuberculosis usually results from hematogenous spread, while direct spread from intra- or extracranial focus is rare.⁴ The clinical and radiologic manifestations of CNS tuberculosis may mimic other infectious and non-infectious neurological conditions. Therefore, familiarity of infectious diseases specialists with the imaging presentations of CNS tuberculosis is essential for prompt and accurate diagnosis of this entity. Herein, we describe the different forms of CNS tuberculosis including meningitis, cerebritis, cerebral abscesses, tuberculomas, miliary tuberculosis, and spinal or calvarial involvement.^{5,7}

METHODS

The present study was to describe and get familiarized with the imaging characteristics of the different forms of CNS tuberculosis in 50 indoor patients of tuberculosis diagnosed by techniques involving culture and immunological testing of the tissue and biofluids and referred to the department of radio-diagnosis for imaging.

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Patients with spinal tuberculosis were not included in our study. Period of study: march 2017 to march 2018. Sample size–50.

OBSERVATION AND RESULTS

Males and females contributed 46% and 54% of the study population respectively. The age range in years was 10 to 76 years. The mean age of the patients was 28 years with 60% of the patients between 20-40 years. Out of the 50 patients, the commonest presentation was meningitis (82%) especially seen in the younger population – children and adolescents.

DISCUSSION

Tuberculous Meningitis

Tuberculous meningitis (TBM) is the most common cause of chronic meningitis, especially in developing countries. Meningitis is the most common manifestation of CNS tuberculosis which is most frequently seen in the children and adolescents^{8,9}. Tuberculous meningitis is mostly due to the hematogenous spread of *Mycobacterium tuberculosis*¹⁰. Tuberculous meningitis often has an insidious course with a nonspecific clinical presentation in early stages, especially in children. Therefore, the imaging plays a key role in the timely diagnosis and decreasing the morbidity and mortality. TB meningitis has a striking predilection for the basal cisterns although exudates in the superficial convexity sulci do occur. Enhancing exudate in the basal cisterns is the most common and also a relatively specific manifestation of leptomenigeal tuberculosis on computed tomography (CT) and magnetic resonance (MR) images¹¹. Meningeal enhancement has been found in up to 90% of cases and is considered to be the most sensitive feature of tubercular meningitis^{6,11,12}. The subpial exudate is primarily located in the 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 ventricle¹³. Extension to suprasellar, interpeduncular, and pontomesencephalic cisterns may also occur from these primary sites¹⁰. In most cases, some degree of meningeal involvement is seen within the sulci over the cerebral convexities, the sylvian fissures, and also the ependymal surfaces of the ventricles; the latter usually occurs in the later stages of the disease^{1,14,15}. CT findings; CT can be normal in early stages. On CT images, the obliteration of the basal cisterns by isodense or mildly hyperdense exudates is the most common finding in tuberculous meningitis.^{1,6,13,16} “blurred” ventricular margins may be seen.

MR findings

The findings are better appreciated on MR imaging than on CT, especially on postcontrast MR images which show the enhancing cisternal exudates and leptomenigeal

enhancement⁵. Basilar exudates are isointense with brain on T1WI giving the appearance of “dirty” CSF. FLAIR scans show increased signal intensity in the sulci and cisterns. Marked linear or nodular meningeal enhancement is seen on T1 C + FS sequences. Focal or diffuse dura- arachnoid enhancement (pachymeningeal) with or without involvement of the underlying subarachnoid spaces may occur but is uncommon. Parmar *et al.* demonstrated that postcontrast fluid attenuation inversion recovery (FLAIR) images may have a higher specificity compared to contrast-enhanced T1-weighted images in detection of leptomenigeal enhancement¹⁷. Magnetization transfer spin echo imaging following contrast injection is superior to the conventional postcontrast imaging in demonstrating meningeal inflammation⁷. In later stages, there may be widening of subarachnoid spaces. A similar pattern of meningeal enhancement may be seen in other infective meningitis, inflammatory diseases such as rheumatoid arthritis, sarcoidosis, or carcinomatous meningitis. Carcinomatous meningitis is usually seen in older patients with a known systemic or primary CNS neoplasm. In neurosarcoidosis, infiltration of the pituitary gland, infundibulum and hypothalamus is common. Other radiologic manifestations of tuberculous meningitis may be related to its possible complications, including progressive hydrocephalus, vasculitis, infarction, and cranial neuropathies

Hydrocephalus

Hydrocephalus is the most common complication encountered in TBM and can be broadly divided into two types:¹ communicating type, which is common, secondary to an obstruction of the basal cisterns by inflammatory exudates and² obstructive type, which is less common and either secondary to a focal parenchymal lesion (tuberculoma or a brain abscess) causing mass effect or due to the entrapment of a part of the ventricle by granulomatous ependymitis.

Vasculitis

It is a complication that is commonly seen at autopsy in cranial TBM.¹³ The adventitial layer of small and medium-sized vessels develops changes similar to those of the adjacent tuberculous exudates. The intima of the vessels may eventually be affected or eroded by fibrinoid–hyaline degeneration. In later stages, the lumen of the vessel may get completely occluded by reactive subendothelial cellular proliferation.¹⁵ Ischemic cerebral infarction resulting from the vascular occlusion is a common sequelae of tuberculous arteritis. Ischemic infarct is also a common complication, being detected in 20–41% of patients on CT, mostly within the basal ganglia or internal capsule regions and resulting from vascular compression and occlusion of small perforating

vessels (necrotizing arteritis)^{14,18,19}, particularly the lenticulostriate and thalamoperforating arteries, vessels which perfuse the so-called medial tuberculosis zone¹⁶. Tuberculous meningitis may also cause dural venous sinus thrombosis with resultant hemorrhagic infarct. Rarely, tuberculosis may present as isolated dural venous sinus thrombosis without any evidence of meningitis or its complications. Cranial nerve involvement is associated with high morbidity and disability in meningitis patients. Cranial nerve involvement occurs due to vascular compromise, ischemia, or nerve entrapment in the basal exudates in 17–40% of cases, most commonly affecting the second, third, fourth, and seventh cranial nerves^{13,15}. The affected cranial nerves are best evaluated by MRI, where they may appear thickened, especially in their proximal segments, with high signal intensity on T2-weighted images and marked enhancement on postcontrast images.

Parenchymal Tuberculosis:

Parenchymal disease may be isolated or associated with tuberculous meningitis. Parenchymal involvement usually presents as tuberculoma. It can also manifest as cerebritis, cerebral abscess, miliary tuberculosis, or tuberculosis encephalopathy.^{20,21,22} a. Cerebritis and Cerebral Abscess: Parenchymal tuberculosis may occur with or without accompanying meningitis. Tuberculosis cerebritis or abscess may have an appearance similar to that of pyogenic bacterial infection on neuroimaging studies. Focal tuberculous cerebritis is very rare⁵, cerebritis is a term that represents inflammation of the brain in the setting of infection before the development of abscess. CT: Hypoattenuation due to edema is seen. Small areas of hemorrhage can sometimes be seen. Faint heterogeneous contrast enhancement can be seen. MRI: T1 iso to hypointensity, T2 hyperintensity, and minimal post contrast enhancement on T1 C + images is seen. Patchy restricted diffusion is seen. Tuberculous pseudo-abscess is rare and is characterized by a central area of liquefaction with pus. It may be solitary or multiple and is frequently multiloculated¹⁵. Tuberculous abscess is different from tuberculomas which contain central caseation and liquefaction mimicking pus. On CT: Tb pseudoabscesses are hypodense on NECT with significant mass effect and surrounding edema. Ring enhancement is seen on CECT. On MRI: Unlike tuberculomas, Tb pseudoabscesses are usually hyperintense to brain on T2/FLAIR images and restrict on DWI. A ring enhancing multi-loculated lesion or multiple separate lesions is the typical finding on T1 C + images. MRS shows lipid and lactate peaks without evidence of cytosolic amino-acids. TB pseudoabscesses appear identical to pyogenic abscesses on standard imaging studies. Both show restricted diffusion. On MR spectroscopy, the peak of

amino acids, which is the spectral hallmark in pyogenic abscess, is not usually seen in tuberculous pseudoabscess^{22,27}. Magnetization transfer (MT) images improve the conspicuity of all CNS tuberculosis lesions^{22,27}. b. Tuberculoma: Tuberculoma is the most common parenchymal lesion in CNS tuberculosis which could be found in any portion of the intracranial space. The lesion may be solitary or multiple and may be seen with or without meningitis. Histologically, the mature tuberculoma is composed of a necrotic caseous center surrounded by a capsule that contains fibroblasts, epithelioid cells, Langhans giant cells, and lymphocytes²⁸. On CT: NECT scans show one or more iso to slightly hyperdense round, lobulated or crenated masses with variable perilesional edema. Calcification can be seen in healed granulomas. CECT scans demonstrate punctate, solid or ring like enhancement. MRI: Most TB granulomas are solid caseating lesions that appear hypo or isointense with brain on T1WI and hypointense on T2WI. Liquefied areas may be T2 hyperintense with a hypointense rim. Enhancement is variable ranging from small punctate foci to multiple rim enhancing lesions. Mild to moderate round or lobulated ring like enhancement around a non enhancing center is the most typical pattern. pMR shows elevated rCBV in the cellular, hypervascular enhancing rim. Solid caseating tuberculomas do not restrict on DWI although liquefied foci may restrict. MRS is helpful in characterizing tuberculomas and distinguishing them from neoplasm or pyogenic abscess. A prominent decrease in the NAA: Cr with a modest decrease in the NAA: Cho is typical. A large lipid peak with absence of other metabolites such as amino acids and succinate is seen in 85-90 % of the cases. The major differential for multiple parenchymal tuberculomas is seen is neurocysticercosis (NCC). NCC usually shows multiple lesions in different stages of evolution. Tuberculomas can also resemble pyogenic abscesses or neoplasms. Abscesses restrict on DWI. Tuberculomas have a large lipid peak on MRS and lack the elevated Cho typical of neoplasm.²⁸ Follow-up CT or MR studies are useful in monitoring the response to medical treatment. Paradoxical enlargement of a preexisting tuberculoma or evolution of a new intracranial and spinal tuberculoma in patients receiving adequate treatment may be occasionally seen. However, with continuation of antituberculous therapy, eventual resolution of the tuberculoma usually occurs^{29,30}. Sometimes, healed tuberculomas appear as calcified foci on nonenhanced CT. Similarly, calcification in the basal cisterns has been demonstrated a few years after meningeal tuberculosis³¹. c. Miliary Tuberculosis: Miliary tuberculosis is seen mostly in severely immunocompromised patients and is usually associated

with meningeal involvement or extracranial primary sites³². Since the dissemination is hematogenous, the lesions are usually located at the corticomedullary junctions. The lesions are tiny (2-3 mm in diameter) scattered lesions that may be invisible on noncontrast MR sequences. In visible lesions, MRI shows small lesions that are hypointense on T2-weighted sequences. These lesions occasionally can be seen as small hypodensities on CT scan¹³. Postcontrast T1-weighted MR images show numerous, round, small, homogeneous, enhancing (usually ring enhancement) lesions²². Invisible lesions that may or may not enhance after intravenous injection of gadolinium can be clearly visible on magnetization transfer spin echo T1-weighted imaging with or without contrast²³. d. Tuberculous Encephalopathy: Tuberculous encephalopathy typically occurs in young children who may present with convulsion, stupor, and coma with no signs of meningeal irritation or focal neurological deficit. Neuroimaging studies show severe cerebral edema, which may be unilateral or bilateral. Myelin loss in the white matter may result in hypodensity on CT images and hyperintensity on T2-weighted MR images^{7,10,33}.

Miscellaneous Forms of CNS Tuberculosis:

Osseous and nonosseous spinal/spinal cord tuberculosis, subdural/epidural abscess, and calvarial tuberculosis are other forms of tuberculosis that may involve CNS with

direct or indirect pathways. Tuberculous spinal meningitis presents on MR imaging as a CSF loculation and obliteration of the spinal subarachnoid space, with loss of the outline of the spinal cord in the cervicothoracic spine and matting of the nerve roots in the lumbar region. Contrast-enhanced imaging reveals nodular, thick, linear intradural enhancement, which may completely fill the subarachnoid spaces⁵. Longstanding arachnoiditis may result in the development of syringomyelia (spinal cord cavitation) that typically demonstrates CSF signal intensity on all MR sequences^{5,29}. Tuberculous spondylitis results from hematogenous spread of infection to the vertebral body via paravertebral venous plexus of Batson. Typical presentation is involvement of multiple vertebral bodies with sparing of intervertebral disc in early stage and disc involvement in later stages. Paraspinal extension and resultant paravertebral abscess (Pott's abscess) as well as subdural/epidural abscess formation with associated spinal cord compression are other common findings. Intracranial subdural or epidural abscess formation may or may not be associated with a primary CNS tuberculous focus and have imaging findings identical to that of other pyogenic abscesses, that is, iso- to hypointensity on T1-weighted images, hyper- or mixed signal intensity on T2-weighted images, and rim enhancement on postcontrast images²⁹.

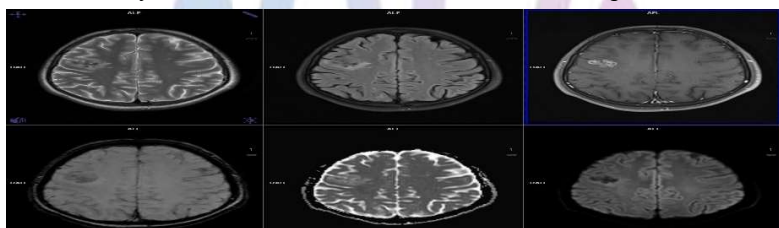


Figure 1: T2W axial, T2W FLAIR axial, T1W C+, SWI, ADC and DWI images showing tuberculomas in the right frontal lobe.

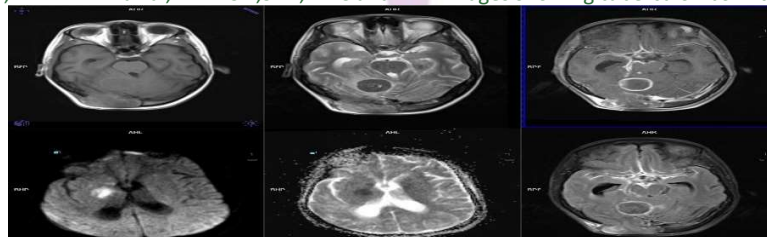


Figure 2: T1W axial, T2W axial, T1W C+, DWI, ADC and T2 C+ FLAIR images showing right cerebellar tuberculoma. Vasculitic infarct in right thalamo-capsular region. Extensive basilar meningitis. Post operative defect with meningo-encephalocele is also noted involving the right occipital lobe and right cerebellar hemisphere at the post operative site.

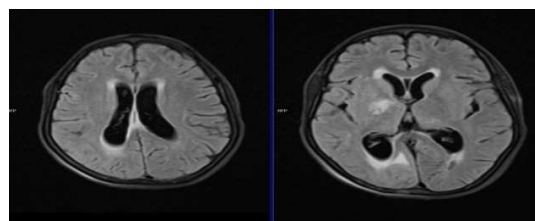


Figure 3: T2W FLAIR axial images in the same patient showing hydrocephalus.

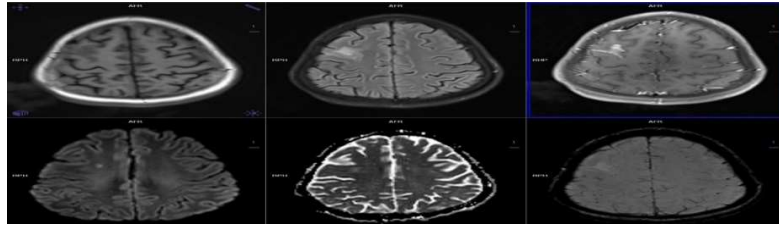


Figure 4: T1W axial, T2W FLAIR axial, T1W C +, DWI, ADC and SWI images showing T2W FLAIR hyperintense enhancing exudates in the right superior frontal sulcus with thickening of the adjacent cortical gyri suggestive of meningo-encephalitis. Small vasculitic infarct in the right frontal white matter involving the centrum semiovale

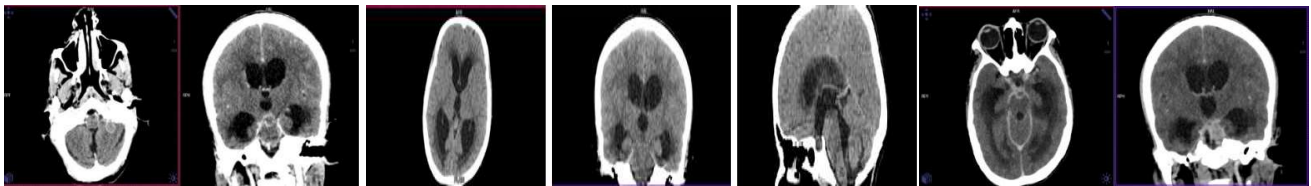


Figure 5: CT brain axial, sag and coronal plain and delayed post contrast images showing tuberculomas in the left cerebellar hemisphere and in the midbrain with enhancing basilar exudates and communicating hydrocephalus.

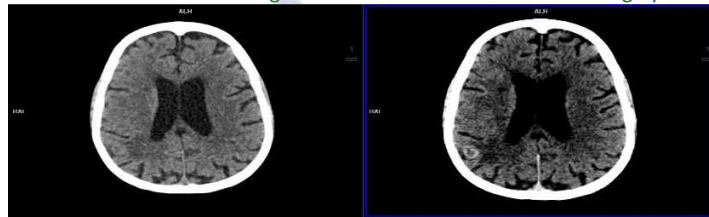


Figure 6: CT brain plain and delayed post contrast axial images showing a small sub centimeter sized ring enhancing lesion in the right parietal sub-cortical white matter with mild adjacent edema in this patient with past history of sputum positive pulmonary tuberculosis. The patient presented with convulsions.

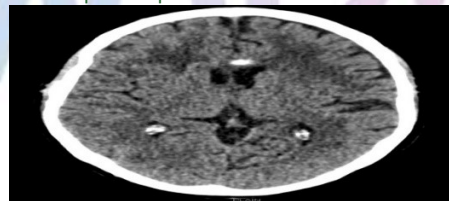


Figure 7: CT brain plain axial image showing a calcified granuloma in the genu of corpus callosum in a case of RVD patient with PMLE and TB.

CONCLUSION

CNS tuberculosis has various imaging appearances, including meningitis, tuberculoma, miliary tuberculosis, abscess, cerebritis, and encephalopathy. In addition, the radiologic manifestations of this disease are not always typical and sometimes may be mistaken with other lesions. Familiarity with the various imaging presentations of CNS tuberculosis is of key importance for the radiologists and infectious diseases specialists in timely diagnosis, thereby reducing the morbidity and mortality of this potentially life-threatening disease.

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