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MRI imaging findings of CNS Tuberculosis

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Abstract

Background

Tuberculosis is a common disease in developing countries, whose prevalence has increased due to population growth, overcrowding and rising cases of acquired immunodeficiency disease. It involves all the major organs of human body; however neurological involvement is the most devastating. CNS TB is a potentially life-threatening condition (comprising 10% of all tuberculosis cases) 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.[1]

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.[2]Routine diagnostic techniques involve culture and immunological tests of the tissue and biofluids, which are time-consuming and may delay definitive management.[3]Noninvasive imaging modalities such as computed Tomo graphy (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.[4]In this review, we describe the MRI imaging characteristics of the different forms of CNS tuberculosis, including meningitis, tuberculoma, abscess and spinal tuberculosis.[5]

Aims and objectives

1. To describe the various manifestations and their imaging characteristics of CNS TB
2. To find out different complications of CNS TB

Methods

22patients who presented to the hospital with symptoms like headache, altered sensorium, fever, projectile vomiting, seizures, focal neurologic deficits,

weakness(paraparesis/quadriparesis) and subsequently tested positive for tuberculosis on lab studies were included in the study. Imaging of these patients was done using MRI andvarious manifestations of tuberculosis was described.

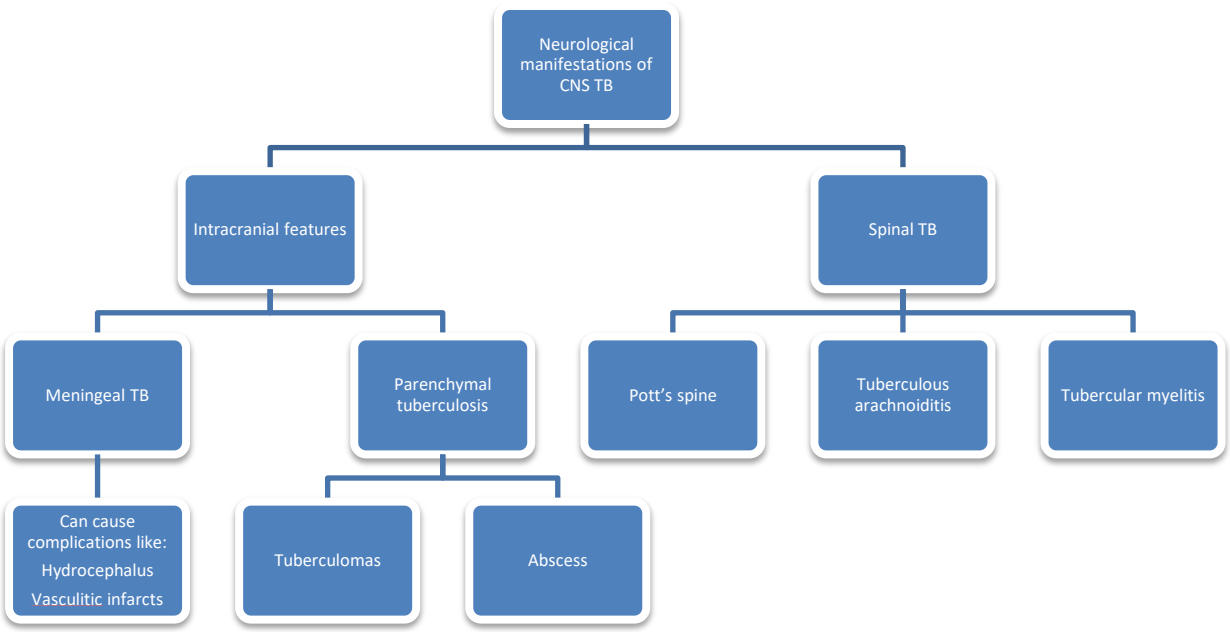
Multiplanar MR imaging was performed on 1.5 T GE MEDICAL SYSTEMS Signa HDxt. T1 weighted(T1W), T2 weighted(T2W), fluid-attenuated inversion recovery (FLAIR), pre, post-contrast fat saturated T1 weighted, diffusion weighted images (DWI) were obtained.

Study design

Observational study.

Period of study- May 2021 to March 2022

Figure 1: Imaging- Neurological manifestations of CNS TB



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. Tuberculous meningitis is mostly due to the Hematogenous spread of Mycobacterium tuberculosis. The organism seeds into a subpial (rarely subependymal) focus of infection called the 'Rich focus', which subsequently ruptures into the subarachnoid (or ventricular) space and triggers an inflammatory response. Meningitis usually presents with features of meningeal irritation such as neck stiffness and photophobia. 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. The Leptomeningeal enhancement shows a distinctive predilection to involve the basal cisterns in TB, in particular the peri mesencephalic, interpeduncular, prepontine, and suprasellar cisterns. It frequently extends along the inferomedial surface of the frontal lobes, the anteromedial surface of the temporal lobes, the floor of the third ventricle, the superior aspect of the tentorial surface, and along the sylvian fissures. Meningeal enhancement has been found in up to 90% of cases and

is considered to be the most sensitive feature of tubercular meningitis.

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 LeptoMeningeal enhancement. 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.

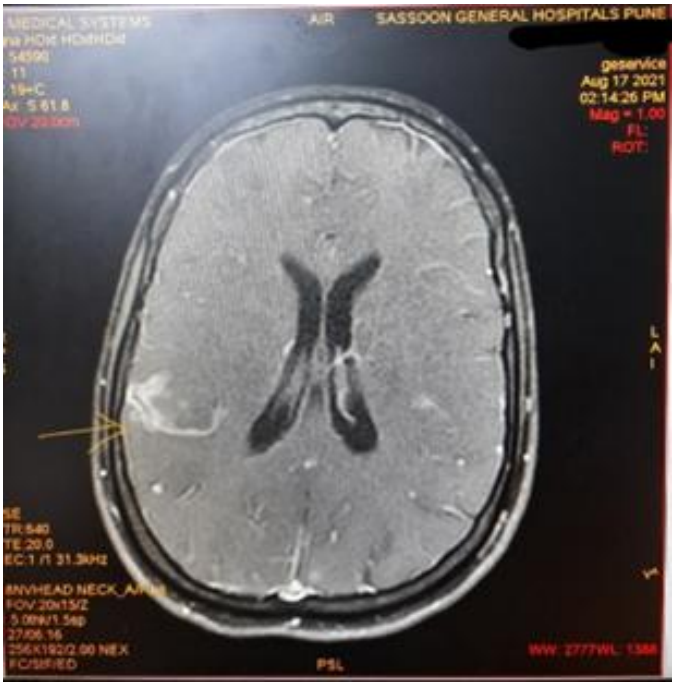


Figure 2: Axial T1W +C showing Leptomeningeal enhancement in right parietal region

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 and 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.

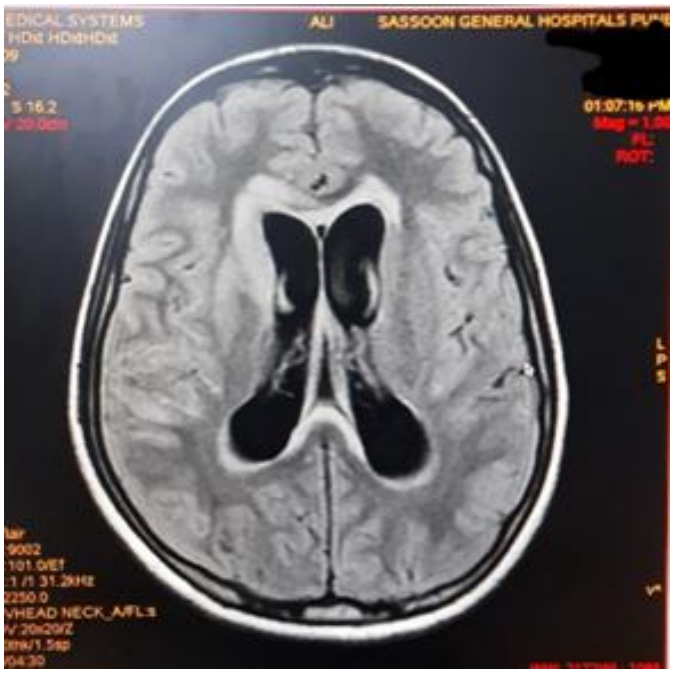


Figure 3: T2W FLAIR axial image showing hydrocephalus with periventricular ooze

Vasculitis

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.

Ischemic cerebral infarction resulting from the vascular occlusion is a common sequela 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) particularly the lenticulostriate and Thalamo perforating arteries, vessels which perfuse the so-called medial tuberculosis zone

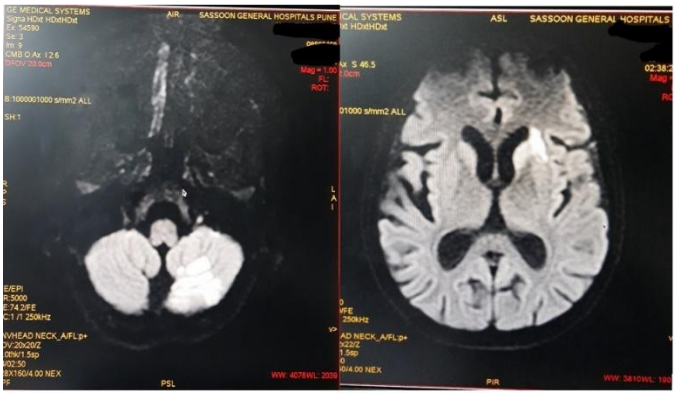


Figure 4**:** DWI images showing diffusion restriction in left cerebellar hemisphere and left basal ganglia s/o vasculitic infarcts.

Parenchymal tuberculosis

Tuberculoma

They occur due to conglomeration and coalescence of tubercular microgranulomas, which tend to occur at the grey-white matter junction due to arrest of the Hematogenous spread of microbes caused by a reduction in calibre of vessels in that region. Occasionally, lesions can develop in the brain parenchyma secondary to spread of CSF infection through the perivascular (Virchow Robin) spaces. They can, however, occur in almost any possible location in the brain, including the sulcal spaces, brainstem, cerebellar hemispheres, basal cisterns, and the ventricular system. They also show a tendency to conglomerate and occur in clusters or coalesce into larger tuberculomas. Tuberculomas are more common in children and are predominantly inFra tentorial. In adults they tend to be predominantly supratentorial. Pathologically tuberculoma consists of a typical granuloma with central caseous necrosis. Clinically they present with headache, seizures, raised intracranial tension (ICT), focal neurological deficits, and fever.

Stages of tuberculoma

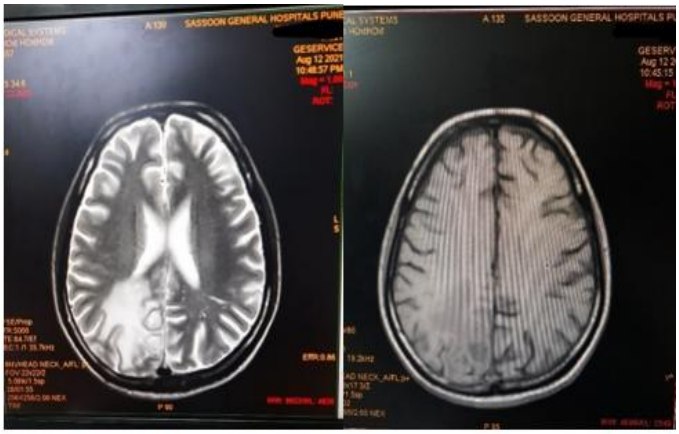
Tuberculomas occur in four stages

- non caseating granuloma
- caseating granuloma
- caseating granuloma with central liquefaction
- calcified granuloma

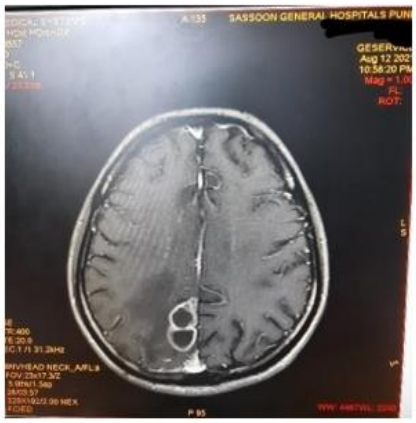
Tuberculoma consist of a typical granuloma with epithelioid cells, Langhans giant cells, and a peripheral rim of lymphocytes. Central caseous necrosis followed by liquefaction develops in subsequent stages. Post treatment a paradoxical increase in size of tuberculoma can occur. They may resolve completely, but in most cases they resolve with the formation of calcified granulomas. Their MRI appearance, although somewhat variable, in the majority of cases is described below.

Table 1: Imaging appearances of different stages of tuberculoma

Lesion	T1W	T2W	FLAIR	DWI	T1WCE
Non caseating granuloma	Iso- to hypointense	Hyperintense	No suppression	No restriction	Homogeneous enhancement
Caseating granuloma	Iso- to hypointense with hyperintense rim	Hypointense	No suppression	No restriction	Homogeneous or ring enhancement
Caseating granuloma with central liquefaction	Isointense to hypointense with hyperintense rim	Hypointense rim with central hyperintensity	Partial suppression	May or may not show restriction	Ring enhancement
Calcified granuloma	Iso- to hypointense	Hypointense	No suppression	No restriction	No enhancement



(a)T2W (b)T1W



(c) T1W + C

Figure 5: a,b and c- showing caseating granuloma with liquefied Centre in right parietal lobe

Abscess

Tuberculous abscesses are more common in elderly and immunocompromised patients. On conventional imaging,

TB abscess may be indistinguishable from pyogenic abscess, both appear large (>5 cm), multiloculated, show thick rind enhancement and perilesional oedema with mass effect.

However, MR spectroscopy may help in differentiating these 2 lesions. Pyogenic abscess demonstrates amino acid peak which is absent in TB abscess.

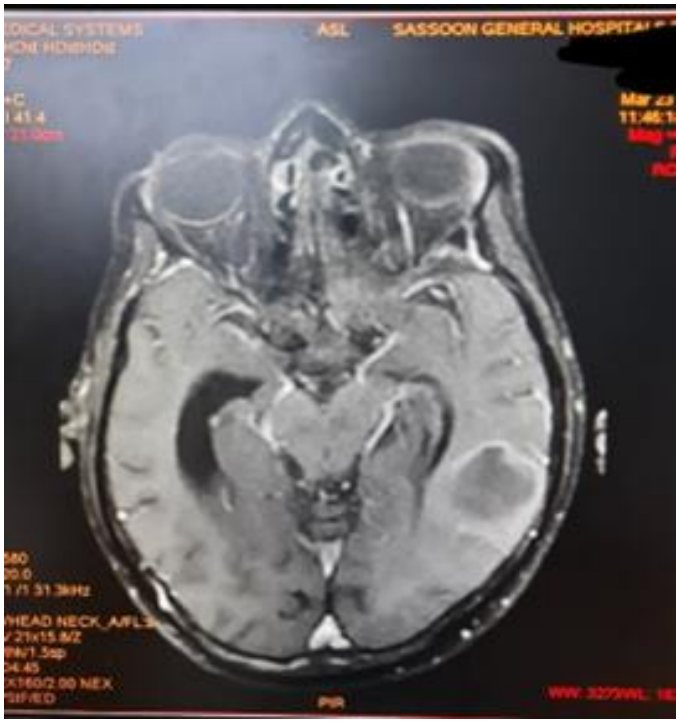


Figure 6: Thick walled peripherally enhancing abscess in left temporal lobe

Spinal TB

Osseous and non-osseous spinal/spinal cord tuberculosis, subdural/epidural abscess, and Calverial tuberculosis are other forms of tuberculosis that may involve CNS with direct or indirect pathways.

a). 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.

Figure 6

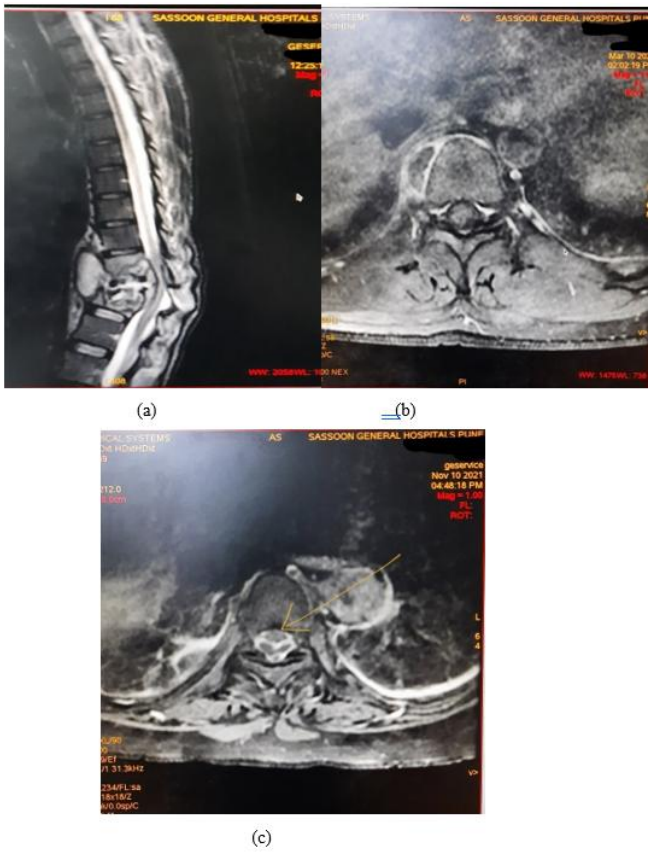


Figure 7: (a)Altered signal intensity in intervertebral disc and adjacent vertebral bodies with hyperintense soft tissue/collection anterior to it s/o spondylodiscitis. (b) Ax T1W + C images showing peripherally enhancing paravertebral collection on right side. (c) Ax T1W + C images showing peripherally enhancing collection in anterior epidural space compressing the spinal cord.

b) 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 spaces.

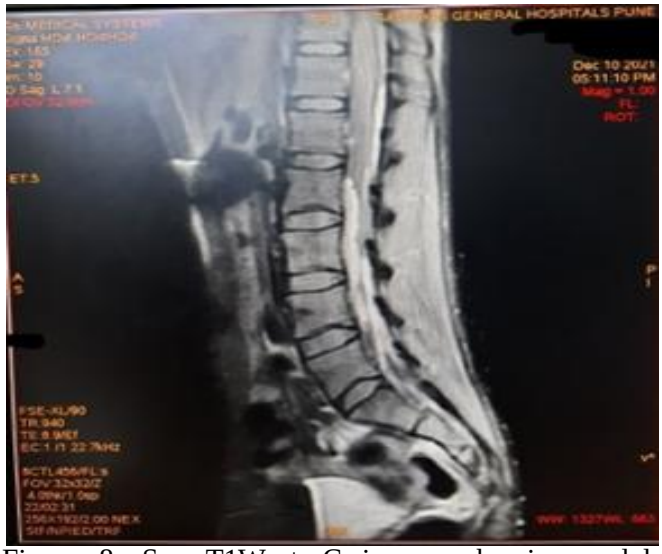


Figure 8: Sag T1W + C images showing nodular intrathecal enhancement filling the subarachnoid space

c) Tuberculous myelitis commonly involves the cervicomedullary segment of the spinal cord. Paraparesis is the most common presenting complaint.Diffuse cord swelling with signal abnormality and diffuse or patchy postcontrast enhancement that may persist for several months is seen on MRI.Intramedullary abscess, cavitation, cord atrophy, and syringomyelia may be associated with poor outcome.

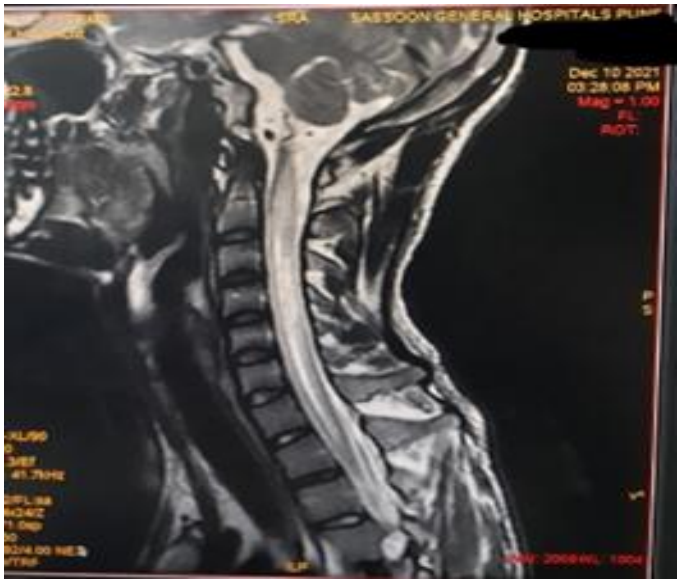


Figure 9: Sag T2 sequence showing long segment hyperintense signal involving cervical cord

Results

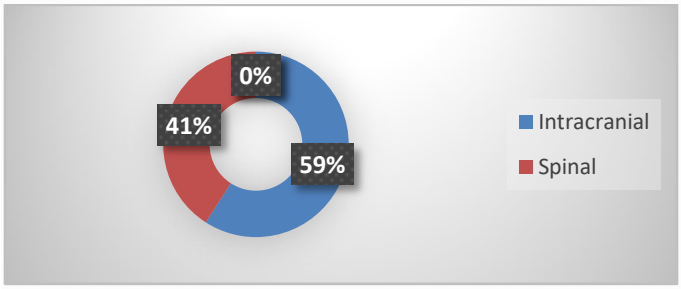
A total of 22 patients were included in the study. There were 12 males (54%) and 10 females (46%) with ages ranging from 13 to 65 years. All the MRI examinations used intravenous contrast.

Table 2: Distribution of cases according to age

Age	Number of cases	Percentage
13-25 years	8	36%
26-50 years	8	36%
51-65 years	6	28%

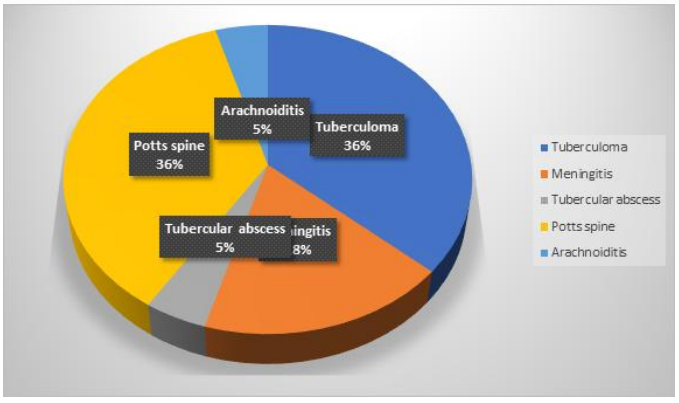
Intracranial manifestations (59%) were more common than spinal manifestations (41%).

Graph 1:



Tuberculomas in various stages was the most common finding seen in approximately36% cases. Meningitis accounted to approximately 18% cases. Tubercular abscess was seen in 5% of the cases.

Graph 2:



3 out of 4 patients with TB meningitis developed complications in the form of vasculitic infarcts (13%) and hydrocephalus (4%).

Conclusion

CNS tuberculosis has both intracranial and spinal manifestations. These can show various imaging appearances, including meningitis, tuberculoma, abscess, vasculitic infarcts, hydrocephalus and calcified granu Loma. In addition, the radiologic manifestations of this disease are not always typical and can sometimes may be mistaken with other lesions such as brain tumors, pyogenic abscess, toxoplasmosis or NCC. 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.

References

1. Sanei Taheri M, Karimi MA, Haghighatkhah H, Pourghorban R, Samadian M, DelavarKasmaei H. Central nervous system tuberculosis: an imaging-focused review of a reemerging disease. Radiol Res Pract. 2015; 2015:202806. doi:10.1155/2015/202806

2. Bernaerts A., Vanhoenacker F. M., Parizel P. M., et al. Tuberculosis of the central nervous system: overview of neuroradiological findings. European Radiology. 2003;13(8):1876–1890. doi: 10.1007/s00330-002-1608-7

3. Rock R. B., Olin M., Baker C. A., Molitor T. W., Peterson P. K. Central nervous system tuberculosis: pathogenesis and clinical aspects. Clinical Microbiology Reviews. 2008;21(2):243–261. doi: 10.1128/cmr.00042-07.

4. Trivedi R, Saksena S, Gupta RK. Magnetic resonance imaging in central nervous system tuberculosis. Indian J Radiol Imaging. 2009;19(4):256-265. doi:10.4103/0971-3026.57205

5. Khatri GD, Krishnan V, Antil N, Saigal G. Magnetic resonance imaging spectrum of intracranial tubercular lesions: one disease, many faces. Pol J Radiol. 2018;83: e524-e535. Published 2018 Dec 29. doi:10.5114/pjr.2018.81408

6. Desai N, Krishnan R, Rukmangadachar L. Central Nervous System Tuberculosis Presenting with Multiple Ring-Enhancing Lesions: A Diagnostic Challenge.Cureus.2022;14(2):e21819. Published 2022 Feb 1. doi:10.7759/cureus.21819

7. Bom Anji JB, Gupta N, Gulati P, Das CJ. Imaging in tuberculosis. Cold Spring HarbPerspect Med. 2015;5(6): a017814. Published 2015 Jan 20. doi:10.1101/cshPerspect. a017814