"MRI in Evaluation of Intracranial Tuberculosis"

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Aims & Objectives :-

• To determine MR imaging criteria for the diagnosis of suspected intra cranial tuberculosis.

• To differentiate tuberculomas into various MR morphologic types.

• To analyse the importance of MRI in the diagnosis of varied presentations of tuberculosis of the brain.

- Inclusion criteria :-
- *Both male and female of any age group.*

All patients who have compatible clinical features along with diagnostic investigation findings suggesting CNS TB.

Exclusion criteria : -

> Implanted electric and electronic devices are a relative contraindication to the magnetic resonance imaging, and in particular:

- heart pacemakers (especially older types)
- insulin pumps
- *implanted hearing aids*
- neurostimulators
- intracranial metal clips
- metallic bodies in the eye

Metal hip replacements(old type), sutures or foreign bodies in other sites are relative contraindications to the MRI because they obscure the visualization of normal anatomy due to artifact effect.

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I. Introduction

Tuberculosis (TB) is a devastating disease caused by *Mycobacterium tuberculosis* with over eight million deaths reported annually worldwide from direct or indirect consequences of the disease ^[1]. Central nervous system (CNS) involvement is the most devastating form of the disease and is an important cause of morbidity and mortality in affected individuals, with permanent sequelae occurring in the majority of the victims^[2] It comprises 10% of all tuberculosis cases and 20% of TB cases in immunocompromised patients ^[3,4]. In almost all cases, the causative organism is *M. tuberculosis*. The most common mode of spread of the infection to the CNS is via the hematological route, usually from a pulmonary focus. ^[5]. Once the organism gains access to the CNS, it incites a strong granulomatous inflammatory response, the effects of which can usually be readily appreciated on magnetic resonance imaging (MRI), thus enabling radiological diagnosis and timely institution of treatment.

Tuberculomas are the most common variety of intracranial parenchymal tuberculosis. Tuberculomas can present as solitary or multiple tuberculomas and can also occur with or without meningitis ^{[4].} The cornerstone of CNS TB diagnosis and associated problems is imaging. The modality of choice is commonly contrast-enhanced magnetic resonance imaging (MRI).

Meningeal tuberculosis	Parenchymal tuberculosis
Leptomeningitis	Tuberculoma
Pachymeningitis	Infarct
	Abscess
	Encephalitis
	Encephalopathy

Patterns of central nervous system tuberculosis

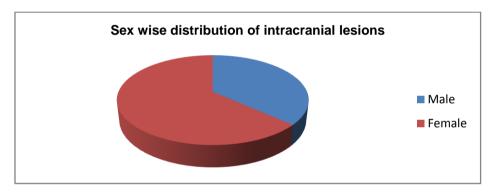
II. Material and Methods

Study was conducted in GCS medical college, Ahmedabad and includes 30 patients over a period of 24 months from JANUARY 2021 – DECEMBER 2022. MRI was done using 1.5 tesla GE signal explorer machine including standard scan protocol.

III. Results

A 1.5-T scanner (GE) was used for examination. The number of lesions, their localizations, dimensions, signal characteristics, and contrast enhancement patterns were all recorded.

The findings of this study revealed that the majority of patients were female, with 19 (63.34%), and male with 11 (36.6%). According to age group, the majority of patients 20 (66.67%) were 15–55 years of age.



Percentage of distribution of intracranial disease .

Tuberculomas are the most common variety of intracranial parenchymal tuberculosis (Patient Number:19).

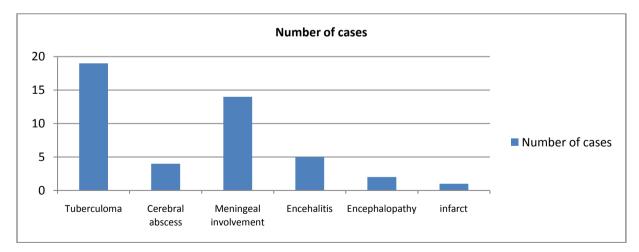


Table	no:1
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Features of Parenchymal lesions and associated abnormalities.

Number of lesions	Single	11	36%
	Multiple	19	63%
Location of lesion	Supratentorial	17	56.67%
	Infratentorial	6	20%

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	Both	7	23.3%
Size of lesion	<2.5 cm	18	60%
	>2.5 cm	12	40%
Features of edema and mass	Present	21	70%
effect			
	Absent	9	30%
Associated abnormality	No abnormality	13	43.3%
	Hydrocephalus	3	10%
	Meningitis	12	40%
	Hydrocephalus and meningitis	1	3%
	Infarction	1	3%

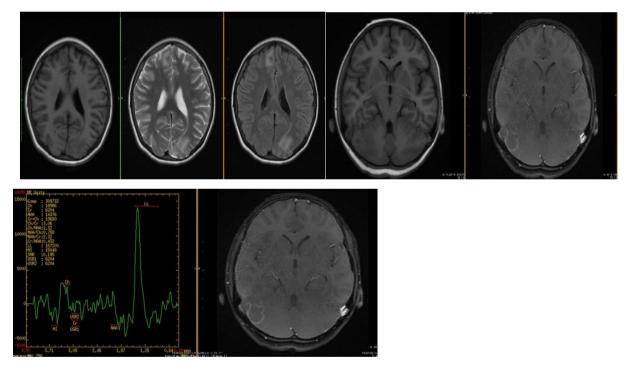
 Table no : 2

 Table showing the types and associations of meningeal lesions

Combination lesions	Number	Percentage
Meningeal lesions only	4	13%
Meningeal and parenchymal	8	26.67%
Meningeal and vascular	1	3.33%
Meningeal, parenchymal and	1	3.33%
vascular		

Case:1

A female patient of age 14 years presented to GCS hospital with chief complain of fever, chills, cough and weight loss for 3 months and diagnosed with sputum positive tubercular bacilli.



IV. Findings :

Presence of few well-defined altered signal intensity lesions are noted in right parietal region along the falx , cortical and subcortical region of right frontal and left parieto-occipital and right cerebellum, which appears isointense on T1W and hypointense on T2W and FLAIR images, largest of size 20×18 mm in right cerebellar region.

On contrast study, the lesions show peripheral enhancement.

On MRI spectroscopy, lipid lactate peak is seen.

Mild perilesional edema is noted as evident by T2W and FLAIR hyperintensity.

Finding raise p/o Multiple Tuberculoma.

Description :

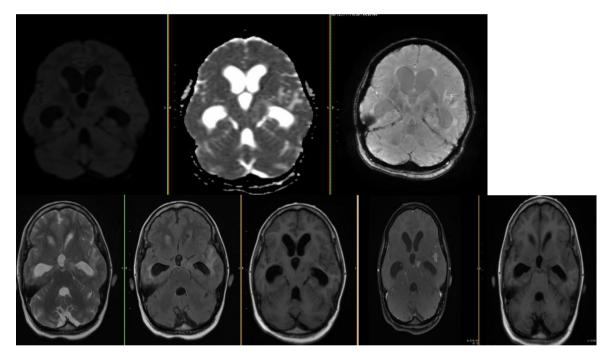
Parenchymal tuberculomas are the most common form of intracranial parenchymal tuberculosis. They occur due to conglomeration and coalescence of tubercular microgranulomas, which tend to occur at the grey-white matter junction due to arrest of the hematogenously spread microbes caused by a reduction in 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 ^[6]. 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 occur in four stages – non caseating granuloma, caseating granuloma, caseating granuloma with central liquefaction, and calcified granuloma.

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.

Case : 2

A female patient of age 30 years presented with neck rigidity, headache, semiconscious and past history of pulmonary tuberculosis 10 years before.



Findings :

Presence of multiple small well defined peripherally enhancing lesions are noted in left sylvian cistern, interpeduncular cistern, bilateral crural cistern, bilateral ambient cistern, qudrigeminal cistern, superior cerebellar cistern, pre pontine cistern, bilateral foramen of luschka - magendie and retrocerebellar space. Mild edema is seen in cortical and subcortical region of left anterior temporal lobe along the sylvian fissure. There is dilatation of both lateral ventricle, 3rd ventricle, 4th ventricle, cisterna magna with mild surrounding oozing of CSF.

Above findings raise p/o TB Meningitis with communicating hydrocephalus.

Description :

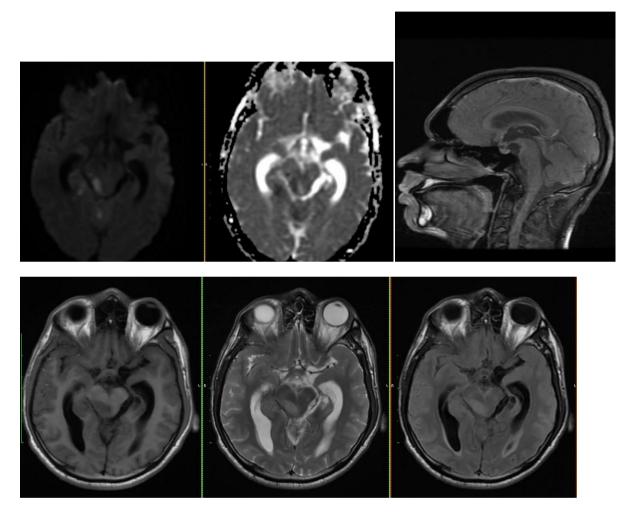
In most patients, TB involving the leptomeninges is thought to spread by haematogenous dissemination from a primary source outside the CNS such as the lung or gastrointestinal tract. The meningitis process may affect the cranial cerebrospinal fluid (CSF) pathway, the spinal subarachnoid pathway, or both¹⁷ Hydrocephalus may result from blockage of the basal subarachnoid cisterns by the dense basal exudate, or narrowing of the aqueduct and third ventricle by tuberculomas.

At MRI, although the meningeal inflammation or basal exudate may not be readily apparent on the pre-contrast MR images, post-contrast T1-weighted MR images show diffuse meningeal enhancement, mainly at the basal cisterns.^[8,9,10] With extension into the ventricular system, abnormal

enhancement of the ependymal lining of the ventricles and the choroid plexi may be seen.

Case : 3

A male patient of age 20 years presented with chief complain of left upper and lower limb weakness, disorientation, diminished response, slurred speech and history of weight loss and night sweats. The lesion was possibly suspected to be tubercular and further evaluation is confirmed by CSF study.



Findings :

Presence of few small diffusion restricted areas are noted in thalamus , midbrain , pons , splenium of corpus callosum , cerebellum ,middle cerebral peduncle on right side, which appears hypointense on T1W and hyper on T2W & FLAIR images.

On FLAIR images there are few sulci hyperintensity noted in bilateral parieto-occipital region.

On post contrast study shows homogenous thickened meningeal and pial enhancement along with bilateral cerebral hemispheres and brainstem

Bilateral lateral ventricle and 3rd ventricle appears to be mildly dilated.

Above findings suggest Meningitis with secondary Vasculitis induced Infarcts

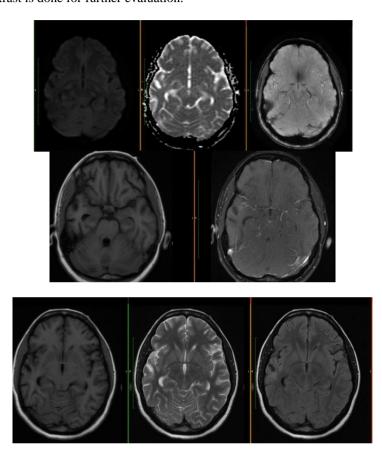
Description :

Vascular complications are also relatively common. Vasculitis and vascular occlusions are detected in 20-41% of the imaged cases of CNS TB. Tubercular infection shows a predilection to involve the small and medium sized vessels. It predominantly involves the lenticulostriate and thalamoperforating vessels and subsequently produces infarcts in these vascular territories involving the bilateral gangliothalamic regions, which have been referred to as the 'medial tuberculosis zone' .^{[11].}

DWI is of particular value in the detection of vascular complications such as infarcts ^{[12].}

Case : 4

A lady of 30 years presented to GCS with chief complain of neck rigidity, fever, disorientation for 5 days. Patient is on AKT for pulmonary tuberculosis. MRI Brain with contrast is done for further evaluation.



Findings :

Presence of altered signal intensity lesion is noted involving cortical and subcortical region of right parietal and temporal lobes which appears hypointense on T1WI and hyperintense on T2WI and FLAIR images, with adjacent thickening of meninges. On post-contrast study, it shows mild gyriform, leptomeningeal and pachymeningeal enhancement. On DWI, it does not show diffusion restriction. On SWAN, it does not show blooming. Mild dilatation of bilateral lateral ventricles and 3rd ventricle is noted, p/o borderline hydrocephalus.

Above findings raise p/o Meningo-encephalitis.

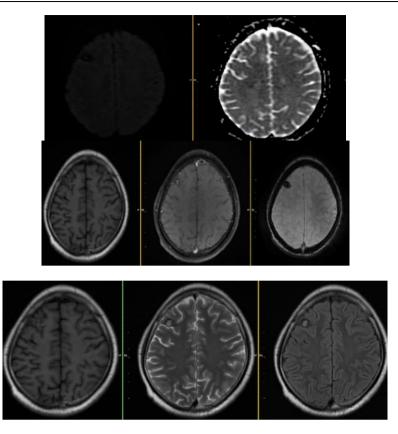
Description :

Tubercular encephalitis can occur with or without associated meningitis. Encephalitis refers to involvement of a focal area of brain parenchyma in the infective process, which is identified on MRI as swelling and altered signal intensity of the involved gyri. The gyri appear hypointense on T1W and hyperintense on T2W scans compared to normal parenchyma and show patchy post-contrast enhancement ^[13]. Pathologically it is composed of tubercular microgranulomata with scarce tubercular bacilli and without the associated caseous necrosis.

Case : 5

A 42 years old patient presented with Generalized toni clonic seizures and pt is K/C/O Tuberculoma and has completed AKT for 3 months.

MRI brain was advised for further evaluation.



Findings :

Presence of well defined lobulated lesion of size 10 x 10 mm is noted in right frontal region, which appears iso to hyperintense on T1W, T2W and FLAIR images with peripheral hypointense rim. On DWI, it does not show diffusion restriction. On SWAN, it shows blooming within. On Post contrast study, it does not show any abnormal enhancement.

Above findings raise p/o Calcified tuberculoma involving right frontal region.

Description :

The MRI features of individual tuberculomas depend on two factors, namely, the presence or absence of caseation and the solid or cystic nature of its centre. Broadly, three combinations frequently occur: (a) non-caseating granuloma, (b) caseating granuloma with solid centre and (c) caseating granuloma with central liquefaction. Non caseating granulomas are hypointense to brain on T1 weighted images, hyperintense on T2 weighted images and display homogenous enhancement on post gadolinium T1 weighted images, isointense to hypointense on T2 weightage images and display rim enhancement on post gadolinium T1 weighted images. Caseating granulomas with central liquefaction are hypointense to brain on T1 weighted images, hyperintense on T2 weightage images and display rim enhancement on post gadolinium T1 weighted images. Caseating on T2 weighted images with central liquefaction are hypointense to brain on T1 weighted images, hyperintense on T2 weighted images with a peripheral hypointense rim representing the tuberculomas capsule and display rim enhancement on post gadolinium T1 weighted images hyperintense on T2 weighted images with a peripheral hypointense rim representing the tuberculomas capsule and display rim enhancement on post gadolinium T1 weighted images [1].

V. Discussion

Tuberculoma, a common manifestation of TB, presents as one or more space-occupying lesions and usually causes seizures and focal signs. Clinical response to antitubercular treatment (ATT) in all forms of neurotuberculosis is excellent if the diagnosis is made early before irreversible neurological deficit is established. It is therefore critical that an early diagnosis with aggressive therapy be the focus of management strategy in any given case. The MRI appearances of the parenchymal involvement are evident as single or multiple tuberculomas or uncommonly manifest as miliary lesions. Significantly, they can occur with or without meningitis.

There are six important phenomena, which often aid in the cross-sectional imaging evaluation in a given case of intracranial tubercular lesion ^{[14].} These general rules comprise: (1) different lesions in a single patient may be in different stages of evolution from one another at the time of initial diagnosis; (2) different lesions in the same patient may respond to medical therapy differently from one another (i.e. resolve at different rates); (3) similar appearing lesions in different patients may respond to medical therapy differently; (4) different meningeal and parenchymal lesions in the same patient may respond to medical treatment differently, with the meningeal lesions lagging behind the resolution of the parenchymal lesions; (5) larger centrally caseous lesions in any location in general, take longer to resolve than smaller non caseous lesions; (6) the larger the lesion is initially, the greater is the likelihood of permanent sequelae such as calcification and encephalomalacia.

The utility of MRI is excellent, in as much a modality it identifies disease and the location of the pathology, displays the extent of involvement, distinguishes it often from other etiologies, guides in biopsy/drainage procedures and provides insight into the appropriate mode of treatment (medical v/s surgical). Furthermore, MRI offers the advantage of possessing a high contrast resolution, the capacity to perform direct multiplanar imaging, the ability to accurately depict parenchymal and meningeal lesions.

Healing is identified by the variable appearances of regression in areas of thin sheet like meningeal enhancement. However, persistent enhancement may be present despite successful ongoing ATT at sites with thick exudates and at regions such as suprasellar and perimesencephalic regions [1]. The other findings signifying response to therapy include, progressive reduction in perilesional edema, regression in size and disappearance of the tuberculoma, appearance of non enhancing, residual encephalomalacia with or without calcification.

VI. Conclusion

Intracranial tuberculosis is a single disease entity with "many faces" on imaging appearance and can hence produce a wide spectrum of patterns in the brain, including tubercular meningitis, tuberculoma, tubercular cerebritis, abscess, and encephalopathy. When response of patient at 3 and 6 months is evaluated, there is significant reduction in perilesional edema is noted, however the number and size of lesion remains same in most of the patients. Careful evaluation of the MRI, appropriate use of modified techniques, and recent advances along with adequate clinico-radiological correlation can enable accurate and timely diagnosis by the radiologist and hence early institution of treatment. This is particularly important in view of the high incidence of permanent neurological sequelae in patients with delayed initiation of treatment.

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