Magnetic resonance imaging of central nervous system tuberculosis: An experience of 50 cases from Central India

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ABSTRACT
Objective: Central Nervous System Tuberculosis (CNSTB) continues to be a common problem world-wide and particularly in India. With the widespread availability of Magnetic Resonance Imaging (MRI), it is increasingly used for the diagnosis and follow up of CNSTB. This study presents a review of 50 cases of CNSTB seen in central India.
Materials and Methods: Between 2006 and 2009, 50 cases of CNSTB (28 Spinal, 22 Cerebral) were diagnosed and followed up. MRI was performed using either a 0.2 T or 1.5 T unit using a standard protocol. Contrast study was needed in 32 cases. The diagnosis was later confirmed either by histopathological or microbiological examination in most cases and by a positive treatment response in some cases. All patients underwent routine radiograph of the chest to look for associated pulmonary tuberculosis, haematological tests and in some cases CT scan guided aspiration / biopsy and CSF analysis.
Results: There were 20 male and 30 female patients with age range of 4 to 65 years. Most common spinal lesion noted was the typical paradiscal involvement followed by skip lesion and posterior element lesion. The lesion was extradural in 19 and intradural in 3 cases. The most common cerebral lesion was meningitis followed by tuberculomas, abscess and obstructive hydrocephalus. Response to treatment was assessed using MRI in 30 cases.
Conclusion: MRI has revolutionized the imaging of CNSTB. The diagnosis can be made with reasonable certainty, obviating the need for an invasive procedure both for diagnosis and treatment. This was confirmed by the resolution of brain and spinal cord pathological changes after anti-tubercular chemotherapy. MRI also allows for monitoring the response to treatment of patients with CNSTB. However in some cases confirmation of diagnosis by needle aspiration and histopathological analysis may be indicated when the imaging findings are not conclusive.
Key words: magnetic resonance imaging, central nervous system, tuberculosis.

INTRODUCTION
Co-existence of tuberculus bacili and homosapiens is present since time immemorial. This disease was recognised as “yakshma” in humans in the year of 1600 BC by Charaka and Sushrsrutha. This disease is also mentioned in Rig Veda and Atharva Veda during 3000 – 1800 BC.

Tuberculosis (TB) is a granulomatous devastating infectious disease caused by Mycobacterium tubercle bacillus. It is a major public health issue globally more so in developing countries like India, Pakistan & South Africa. Of late, significant resurgence is seen worldwide and various factors like HIV, poverty, increased population etc. are responsible for this resurgence. Due to TB, every year 8 million people die in the world.

Tubercular involvement of the brain and spine is commonly seen producing neurological disorders especially in developing countries. The incidence of CNS TB is directly proportional to the tuberculous infection in the population (around 5%). This infectious disease involves the central nervous system via hematogenous spread from the primary focus, usually from lung. It involves the brain parenchyma, meninges, spinal cord, bone coverings of the spinal column and brain. CNS TB has high mortality and morbidity. Hence early diagnosis is
necessary for appropriate treatment.

It is difficult to diagnose CNS TB clinically. The other routine investigations like culture, immunological tests of the tissue and bio fluid take long period to get the results. Imaging plays a major role in early diagnosis. This helps to institute early proper treatment which reduces morbidity and mortality. With a widespread availability of Magnetic Resonance Imaging (MRI), its use is increasing for the diagnosis and follow up of CNS TB. MRI is more sensitive than CT in diagnosis and to determine the extent of involvement of parenchyma, meninges, bones, ligaments and soft tissue. Recent advances in MRI techniques like magnetisation transfer imaging, diffusion imaging, proton magnetic resonance spectroscopy helps in better tissue characterisation and in assessing response to treatment. This paper presents review of 50 cases of CNS tuberculosis seen at Wockhardt Hospital in Central India. 60% cases were followed up after medical and surgical treatment for 1 year to see the response of treatment.

MATERIALS AND METHODS
This is a review of 50 cases of CNS TB which were diagnosed and treated and followed up for 1 year at our Wockhardt hospital, NAGPUR, INDIA. The study period was of 3 years from 2006-2009. All patients underwent an MRI scan on .2 TESLA SIGMA UNIT (GE WIPRO) and 1.5 TESLA ACHIVA (PHILLIPS). All cerebral cases underwent T1,T2W& FLAIR images in sagittal, axial & coronal planes.18 cases underwent T1W and Fat supressed T1 images in sagittal,axial and coronal planes after the administration of Gadopentetate dimeglumine at a dose of 0.1 mmol/kg (GDTPA). 2-3 mm images where also obtained for small lesions like tuberculomas. In 3 cases, post contrast Multi-Voxal Proton MR Spectroscopy was carried out for further evaluation. Spectroscopy measures where obtained on short, intermediate & long TE. For spinal cases, T1,T2 W images were obtained in sagittal & axial plane along with STIR coronal images. Gadolinium enhanced T1W & fat supressed T1 images were obtained in 12 cases in all three planes. All patients underwent chest x-ray, routine haemogram, PCR & Mantoux test.

14 patient’s who had equivocal findings and had diagnostic dilemma underwent CT guided aspiration of soft tissue mass/collection or FNAC, biopsy of vertebral body and CSF analysis for confirmation of diagnosis.

30/50 cases were followed up for 1 year after CNS TB diagnosis. 16 cases of confirmed CNS TB were treated conservatively while 14 cases underwent surgical treatment & followed by ATT. 6 spinal tuberculosis and 10 cerebral tuberculosis cases were managed conservatively. 12 spinal cases were operated since they had developed neurological deficit. 2 patients of cerebral tuberculosis underwent surgery because of diagnostic dilemma. All these follow up cases were evaluated on T1 & T2 W sequences & pattern of altered signal intensity was observed. Post contrast Fat suppressed T1 & T1 W images were correlated with pre contrast images. A pattern of enhancement & regression of lesion was recorded.

Follow up MRI examination was done with an interval of 3-6 months after initiation of treatment.

RESULTS
A total of 50 patients with CNS meningitis were included (20 were males(40%) and 30 females(60%)). The mean age of the patients was 39 years (range 4-65 years). Of the total 50 patients, 21 patients had positive history of pulmonary tuberculosis, four patients had tubercular adenitis. Five patients had a positive family history of tuberculosis. Chest X-ray was positive for tuberculosis in 21 patients. 28 patients were diagnosed with Spinal tuberculosis and 22 with cerebral tuberculosis. Most common spinal lesion noted was the typical paradiscal involvement followed by skip lesion...
and posterior element lesion. The most common cerebral lesion was meningitis followed by tuberculomas, abscess and obstructive hydrocephalus. The details of the cerebral and spinal tuberculosis are listed in table-1. Laboratory investigations of the patients have been listed in table-2.

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**DISCUSSION**

The findings seen on MRI in our study of 50 cases were similar to that of reports documented in the literature. We encountered more pronounced lesions on MRI which may be due to delayed patient presentation. Delayed presentation is possibly due to poor socio-economic status and paucity of MRI facility in Central India. Some rare cases were seen in our study. They were entire spinal axis involvement, facetal cyst, reactivation of calcified cerebral tuberculoma after four years inspite of ATT treatment, and simultaneous involvement of Spine and Brain.

The most common manifestation of CNS Tuberculosis across all age groups is meningitis and intracranial tuberculomas. The tuberculosis meningitis is seen as hematogenous spread from primary focus usually the lungs. It can also be seen secondary to direct extension from infected CSF or rich focus rupture. Early diagnosis of meningitis helps to reduce morbidity and mortality.

The most common appearance of tuberculous meningitis on CT is obliteration of basal cisterns by iso or mildly hypodense exudates. On MRI, the typical imaging findings are meningeal

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**Table.1 Distribution of Spinal and cerebral tuberculosis**

<table>
<thead>
<tr>
<th>Type of CNS-TB</th>
<th>Cerebral tuberculosis (n=22)</th>
<th>Spinal tuberculosis (n=28)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subtype</td>
<td>Meningitis 8</td>
<td>Extradural - discal, paradiscal, posterior appendages 17</td>
</tr>
<tr>
<td></td>
<td>Tuberculomas 6</td>
<td>Skip lesions 4</td>
</tr>
<tr>
<td></td>
<td>Abscess 3</td>
<td>Intramedullary 3</td>
</tr>
<tr>
<td></td>
<td>Hydrocephaulus 3</td>
<td>Combined 2</td>
</tr>
<tr>
<td></td>
<td>Infarction 2</td>
<td>Arachnoiditis 2</td>
</tr>
</tbody>
</table>

**Table.2 Laboratory investigation reports of patients with CNS TB**

<table>
<thead>
<tr>
<th>Investigation</th>
<th>Reports</th>
<th>Number of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>CSF analysis</td>
<td>Low glucose concentration, Elevated protein</td>
<td>14</td>
</tr>
<tr>
<td>CSF culture</td>
<td>AFB +ve</td>
<td>2</td>
</tr>
<tr>
<td>WBC count</td>
<td>Lymphocytosis</td>
<td>25</td>
</tr>
<tr>
<td>Immunoglobulin</td>
<td>IgG, IgM, PCR +ve</td>
<td>28</td>
</tr>
<tr>
<td>Mantoux reaction</td>
<td>Positive</td>
<td>5</td>
</tr>
<tr>
<td>HIV test</td>
<td>Positive</td>
<td>4</td>
</tr>
<tr>
<td>CT – biopsy/aspiration</td>
<td>Positive</td>
<td>8</td>
</tr>
</tbody>
</table>
Due to obstruction to CSF flow by inflammatory exudates in the basal cisterns, communicating hydrocephalus is the most common complication of CNS TB. This is caused by the obstruction leading to increased intracranial pressure.

Tuberculomas often appear as multiple tiny (2 mm) T2 hyperintense foci. They show homogenous enhancement on gadolinium enhanced T1 images. In our study, we had 6 cases of tuberculomas, one treated case of CNS TB showed reactivation of lesion after 4 years. Quantum MT & MR spectroscopy are showing promising results for further evaluation of tuberculomas & response to treatment. In our study, we had carried out an MRI with DWI and ADC maps to assess the extent of edema and to differentiate between cerebral abscess and tuberculoma based on diffusion characteristics.
enhancement which is more pronounced in basal cisterns, cerebral convexity sulci and sylvian fissure. These changes are better delineated on gadolinium-enhanced MRI than contrast-enhanced CT\textsuperscript{1,2}. Extension of infection into the ventricles may be seen in the form of intense enhancement along the ventricle margins and/or choroid plexus enhancement. With appropriate treatment, these appearances resolve quickly. The radiologic resolution may be delayed if exudates are thick. This appearance is nonspecific and such appearance maybe seen in other infections, inflammatory diseases such as sarcoidosis, rheumatoid arthritis, primary and secondary neoplasm. In our study, we had 8 cases (16\%) of cerebral meningitis (Figure 1).

The diagnosis of tuberculoma is often difficult. Brain tuberculomas make 5-8\% of intracranial masses in developing countries. Tuberculomas can be seen within the brain parenchyma, although most commonly seen over the convexity of frontal and parietal lobes. Tuberculoma and meningitis can co-exist, but not a consistent finding\textsuperscript{4}. On MRI, non casseating tuberculoma is hypointense related to parenchyma on T1 images and hyperintense on flair and T2 images. It shows homogenous enhancement on gadolinium contrast study. Caseating tuberculomas having solid centre, appear iso to hypointense on T1 and T2 images. Varying amount of perilesional edema is seen which appears hyperintense on T2 and flair images. Caseating tuberculomas with a liquid centre show central hypointensity on T1 and hyperintensity on T2 surrounded by hypointense margins which represent capsule. Gadolinium enhanced MR shows rim enhancement. With appropriate treatment, tuberculomas can resolve totally. Calcification is seen in one-fourth cases and is best identified on CT\textsuperscript{1}.

CNS Miliary tuberculosis is rarely associated with tuberculosis meningitis. Miliary tuberculomas appear as multiple tiny (2 mm) T2 hyperintense foci. It shows homogenous enhancement on gadolinium enhanced T1 images\textsuperscript{1}. In our study, we had 6 cases of tuberculoma, one treated case of CNS TB showed reactivation of lesion after 4 years (Figures 2 & 3). Quantum MT & MR spectroscopy are showing promising results for further evaluation of tuberculomas & response to treatment. In our study, we had carried out Post contrast Multivoxal MR Spectroscopy in three cases which showed elevated lactate peak with diminished NAA & Cholin.

A rare complication of CNS TB is cerebral abscess formation. They are large in size & more often multiloculated. On MRI, they mimic liquid centered caseating tuberculomas. Intense surrounding edema is also seen with cerebral abscess. They are better evaluated on diffusion imaging and proton MR spectroscopy.

On CT, cerebral abscess show hypodensity, perilesional edema, mass effect, and ring enhancement on contrast study. It is difficult to differentiate cerebral abscess on imaging from caseating tuberculomas and pyogenic abscess, since all the three conditions show ring enhancement on contrast study. In our study, 3 cases of cerebral abscess were present (Figure 4).

Tuberculous cerebritis very rarely occurs\textsuperscript{3}. In our study, one case of Cerebritis was seen normal CT 4 year ago.

Communicating hydrocephalus is the most common complication of CNSTB. This is caused due to obstruction to CSF flow by inflammatory exudates in the basal cisterns. Rarely, noncommunicating hydrocephalus maybe seen secondary to mass effect by tuberculomas producing obstruction to CSF flow. In plain CT and MRI, initial presentation maybe of mild hydrocephalus. In cases of obstructive hydrocephalus under pressure, surgical intervention may be required. Chronic hydrocephalus can lead to atrophy of brain parenchyma. We encountered 2 cases of noncommunicating hydrocephalus and 1 case of obstructive hydrocephalus (Figure 5).

Vasculitis is also common
complication of CNS TB. This may be caused by direct invasion of the vessels by Mycobacteria or from extension of adjacent arachnoiditis. Infarction is common (>50% of patients) in the acute phase and results from vasculitis mainly involving pontine perforator, lenticulostriate and thalamoperforator arteries. Small infarcts are common in the basal ganglia and brainstem. These infarcts can cause mental retardation, stroke or blindness. Ischemic infarcts are seen in 20-40% of patients on CT and are seen as areas of hypodensities. On plain MRI, infarcts appear hypointense on T1 & hyperintense on T2 W images. Acute infarct shows diffusion restriction on diffusion imaging. Contrast enhanced MR Angiography is useful in delineating vascular abnormality. MRI is more sensitive than CT in demonstrating infarcts. We encountered one case of acute infarct involving thalamus showing diffusion restriction. Second case of infarct was seen in periventricular deep white matter on left side (Figure 6). Involvement of cranial nerves is seen in 17-70% cases. 2nd, 3rd, 4th & 7th nerve are commonly involved. We did not encounter any case of cranial nerve involvement.

Two distinct patterns of spinal tuberculosis are seen: (A) Spondylodiscitis which is characterized by destruction of two or more contiguous vertebrae, apposing vertebral end plates, intervening discs with pre or paravertebral soft tissue mass component/ collection. (B) Atypical pattern is of spondylitis without disc involvement. This atypical pattern is showing increase in frequency. Affection of the spine is the most common spinal infection and is called as Pott's Spine. Roughly 50% cases seen in India of osteoarticular tuberculous occur in spine. All ages may be affected but more
common in adults, especially in immunocompromised individuals. Most lesions occur in or below the mid dorsal spine. Cervical and upper dorsal affection is uncommon. The spine infection is due to hematogenous spread via venous plexus of Baston.

Infection begins from the anterior part of the vertebral body and extends into the adjacent end plates. Subsequently there is erosion of end plates and disc resulting into reduction in disc space. Subligamentous extension beneath the anterior longitudinal ligament is a common feature. Involvement of posterior elements can be seen. In the lumbar region, psoas abscess can extend into the groin along the ileo-psoas muscle. Fan shaped calcification within the abscess is virtually diagnostic of Pott's Spine. If untreated, then Pott's Spine results into collapse of vertebra & anterior wedging of vertebra with resultant Kyphosis and Gibbus's formation. There is no or little reactive sclerosis or periosteal reaction. These features helps to distinguish tubercular spondylo-discitis from pyogenic infection of spine. MRI is a modality of choice in the diagnosis and assessment of tubercular spondylo-discitis because of its sensitivity to delineate soft tissue abnormalities, its multiplanar capability and early detection of marrow abnormalities. CT is better than MRI in demonstrating bony fragments. Presence
Spinal Tuberculous Meningitis:
The pathophysiology of spinal meningitis is similar to that of cerebral TBM. During primary infection, a submeningeal tubercle gets formed which ruptures into the subarachnoid space and elicits mediators delayed hypersensitivity. Non-enhanced MRI findings are CSF loculation, obliteration of the spinal subarachnoid spaces, ill-defined spinal cord in cervicothoracic region and matting of nerve roots in lumbar region. Postcontrast MRI shows nodular, thick, linear intradural enhancement which can completely fill the subarachnoid space. Chronic spinal tuberculous meningitis may not show enhancement and can lead to arachnoiditis. The complication of arachnoiditis is Syringomyelia and is seen as spinal cord cavitation which shows CSF signal intensity pattern on T1, T2 and Flair sequences. There is no enhancement seen on contrast study.

Intramedullary Spinal Tuberculoma and Abscess:
Intra spinal tuberculomas accounts for 2-5% of spinal tuberculosis. Intramedullary spinal tuberculoma (IMT) is still rare and the incidence documented is 2 in 1,00,000 cases of all tuberculosis. In 1828, first case of intraspinal tuberculoma was reported by Albercrombie. Intramedullary tuberculosis is almost always secondary to pulmonary tuberculosis. Some cases may present only with isolated extra pulmonary form. It is also commonly seen in immunocompromised patients. On MRI, the IMT shows spinal cord swelling which regresses after specific antitubercular treatment. Gadolinium enhanced MRI is mandatory since tuberculomas are evolving granulomas. In the initial phase of tuberculoma, one may see chronic granulomatous inflammation with formation of giant cells, which homogeneously enhances with contrast due to break down of blood-brain barrier. Collagen subsequently gets deposited along the capsule of the tuberculoma and

of bony fragment in the intra and/or extraspinal soft tissue is characteristic of Tubercular Spondylitis. MRI findings of tuberculous spondylitis may be non specific and consists of low signal intensity on T1 W images and heterogenous signal intensities on Flair and T2 W images. Intra osseous abscess show low signal intensities on T1 and very high signal intensities on T2 and STIR images within the vertebral body. In chronic vertebral tuberculosis, signal intensity pattern is variable.

Gadolinium enhanced study better delineates epidural abscess, myelitis, paravertebral masses, cord and nerve root compromise. Abscess and granulomatous tissue show peripheral enhancement while low intensity area represent necrotic tissue. These changes are best seen on Fat Suppressed T1 sequences. Atleast in 75 % cases, disc shows enhancement on gadolinium enhanced study similar to that of pyogenic discitis. One may see disc preservation, normal signal intensity and lack of enhancement. Reported false negative rate with gallium scan is around 70 % while of isotope bone scan is 35 %.

We recorded 17 cases of Spinal Tuberculosis, which showed discal, paradiscal and posterior appendageal involvement. 4 cases showed skip lesions (Figures 7 & 8).

Intraspinal Tuberculosis:
Intraspinal tuberculosis can originate by three ways. 1) Primary tubercular lesion 2) Secondary from intra cranial tubercular meningitis 3) Extension from tubercular-discitis.

Tuberculous lepto-meningitis may be associated with involvement of the spinal cord and nerve root. It has been designated as radiculo-myelitis. Wadia and Dastur in a series of 70 cases of spinal tuberculosis found radiculo-myelopathy associated with spinal meningitis as its first clinical presentation.
the contents of the lesion get transformed into caseous material. On contrast MRI, the initial homogeneous enhancement lesion becomes rim enhanced lesion at this stage. On appropriate chemotherapy this lesion shows regression in size and may disappear completely. Residual gliosis may be seen as hyperintense signal on T2 W images.

Three cases of intramedullary tuberculosis were found in our study. One case showed Myelitis while two showed tuberculoma. Both the cases of intramedullary tuberculomas showed total disappearance of altered signals and cord swelling after 9 months of appropriate chemotherapy. Before treatment, both the cases showed significant long segment cord oedema. On post contrast study, tuberculomas showed rim enhancing lesion with central necrotic hypointense zone (Figure 9).

**Intramedullary Spinal Abscess**:

Spinal intramedullary abscess is still rare and is extremely rare in comparison to brain abscess. The first case of spinal intramedullary abscess was documented by Har19,18. On non contrast MRI, it shows enlarged hyperintense cord with hypointense nodule on T2 images. On post contrast study, it shows enhancing nodular lesion showing central hypointense necrotic tissue. Intramedullary tuberculomas presenting as cord abscess are more common in Asian countries. In the past, venous infarct complicated by bacterial infection was thought to be the cause of intramedullary abscess. Similar appearance can be seen with intramedullary metastasis or hydatid cyst.

The primary treatment of cord abscess is appropriate anti tubercular chemotherapy. The response to chemotherapy is usually good. In case of no clinical response to ATT, deterioration of the neurological status or nonspecific neuroimaging features and paradoxical increase in size of the lesion even after ATT surgical intervention is the answer19.

**Arachnoiditis**:

Arachnoid adhesions usually develop secondary to spinal infections, commonest being tuberculosis or surgery. MRI is the study of choice for evaluation of arachnoiditis. T1W image may reveal an indistinct or absent cord outline due to the increase in the signal intensity of the surrounding CSF which is the result of elevated CSF protein content, presence of inflammatory exudate, or formation of adhesions over spinal cord surface. T2 weighted MRI may demonstrate CSF loculation and obliteration of the subarachnoid space or irregularly thickened, clumped nerve roots. With more severe arachnoiditis, progression of nerve root clumping and leptomeningeal adhesions may lead to angular defects in the dural sac. Peripheral adhesions of the nerve roots to the walls of the thecal sac produce so called Featureless or Empty sac appearance. The most common pattern of enhancement is a smooth, linear layer of enhancement outlining the surface.
of the cord and nerve roots. The second most common pattern is nodular pattern with discrete foci of enhancement seen along the surface of the cord and nerve roots. The least common pattern consists of diffuse intradural enhancement that completely fills the subarachnoid space. In our study, 2 cases of Arachnoiditis were seen. (Figure 10). Rare late complications of intraspinal infections are Cord Atrophy & Syrinx formation.

**Therapeutic Response assessment:**

MRI is the modality of choice for documenting the response to appropriate chemotherapy in CNSTB. The duration of medical treatment is largely empirical. Little data is available in the literature. Followup imaging after treatment may show decrease in the lesion size within a period of 3 to 4 months. Total resolution of lesion is seen within one year. Rarely, we may see paradoxical increase in intracranial tuberculomas or appearance of new lesion during Antitubercular treatment.

Magnetization transfer imaging is better than conventional spin echo sequences for imaging abnormal meninges. Meninges appear hyperintense on precontrast T1W MT images and show further enhancement on contrastenhanced T1W MT images. MT ratio quantification also helps in predicting the etiology of the meningitis. Appearance of inflamed meninges on precontrast T1W MT images with low MTR is specific for Tubercular meningitis. This helps in differentiating TBM from other non tuberculous chronic meningeal infections.

For the differentiation of Tuberculomas, in vivo proton MR spectroscopy and Quantitative MT imaging are helpfull. Cellular components of the lesions appear brighter and relatively specific for the disease on MT T1W imaging. Lesion deliniation is better on T1W MT imaging in comparison to conventional SE imaging. This inturn helps in improved assessment of the disease load.

On T1W Magnitzation Tranfer imaging the solid center appears hypointense surrounded by a hyperintense rim. MTR's value from the rim and the core are reported as 23.8 +/- 1.76 and 24.2 +/- 3.1, respectively.

MTR quantification from the rim of the abscess is helpfull in the differentiating Tuberculous abscess from pyogenic abscesses. M.Tuberculosis bacilli contains high lipids and are probably responsible for the significantly lower MTR values in the rim of tuberculous abscess (19.89 +/- 1.55) compared with pyogenic abscesses (24.81 +/- 0.03).

Diffusion weighted imaging (DWI) shows diffusion restriction in tuberculomas with liquid necrosis. There is no such diffusion restriction in lesions with solid caseation. Restricted diffusion is seen in T2 hypointense lymphoma. This may help to differentiate lymphoma from tuberculoma.

Tuberculous abscess shows restricted diffusion on DWI with low apparent diffusion coefficient (ADC).
values. This is probably as a result of the presence of intact inflammatory cells in the pus.

CONCLUSION

MRI has revolutionised the imaging of CNSTB. The diagnosis of CNS TB can be made with reasonable certainty by MRI, obviating the need for an invasive procedure both for diagnosis and treatment. This was confirmed by the resolution of brain and spinal cord pathological changes after anti-tubercular chemotherapy. MRI also allows for monitoring the response to treatment of patients with CNSTB. The conventional imaging & post contrast study supplemented by advanced MR techniques like Spectroscopy, Diffusion MRI, Diffusion Tenser MRI helps in improved tissue characterisation, better management and treatment response. However, in some cases confirmation of diagnosis by needle aspiration and histopathological analysis may be indicated when the imaging findings are not conclusive.

REFERENCES


