MAGNETIC RESONANCE IMAGING IN INTRACRANIAL TUBERCULOSIS

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Abstract

Tuberculosis (TB) is still a major cause of serious illness in many parts of the world. Intracranially, TB manifests itself variably as meningitis, tuberculoma and tubercular abscess [1]. Although its appearance on MR is not absolutely specific, it is important in the proper clinical setting to recognize the range of possible patterns that can be observed on images [2]. Magnetic Resonance Imaging (MRI) has emerged as a quality imaging tool aiding in the diagnostic evaluation of intracranial TB variably displaying meningeal, parenchymal, osseous and craniovertebral lesions. The MRI characteristics of 18 cases of intracranial TB were reviewed. Results: Multiple lesions occurred with a slightly higher incidence at 61%. In all, 11 patients (61%) presented with meningitis. Meningeal lesions without parenchymal or vascular involvement were seen in 16% of cases. 2 patients had extension of enhancing exudates into the spinal subarachnoid spaces. While 6 patients had focal intra-axial tuberculomas, representing 33% cases, 3 patients presented with infarcts. 1 patient presented with haemorrhagic infarct at right middle cerebral artery territory while two other showed multiple small infarcts. Hydrocephalus was identified in 4 patients and epidural lesions were noted in 2 cases. MRI should be considered as the imaging modality of choice for patients with suspected intracranial TB.

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KEY WORDS : Intracranial tuberculosis; Magnetic resonance imaging; Tubercular meningitis.

Introduction

"The study of TB is well calculated to make a man think regarding the meaning and course of all disease" Sir Robert Philip. The words of Sir Robert Philip cannot ring more true than today, at the dawn of new millennium. Intracranial tubercular lesion is a serious and potentially life threatening manifestation of TB. Indeed, TB involving the central nervous system (CNS) and its covering is a complex and potentially devastating disease. The increasing incidence of this disease in both immunologically normal as well as immunologically susceptible populations makes the subject of intracranial TB one of universal concern. The intracranial lesions commonly appear as meningitis and/or tuberculomas and their sequelae. Clinical response to antituberculous therapy in all forms of neurotuberculosis is excellent if the imaging and laboratory diagnosis is made early before irreversible neurological deficit is established. Towards this end, Magnetic Resonance Imaging (MRI) has emerged as a quality imaging tool aiding in the diagnostic evaluation of intracranial TB variably displaying parenchymal, meningeal, osseous and craniovertebral lesions. The MRI characteristics of 18 proven cases of intracranial TB are reviewed here in this study.

Material and Methods

In this study, MRI characteristics of 18 cases of TB spinal infection were reviewed. These cases were composite group of patients, namely 8 cases from INHS Asvini and 10 cases from

Apollo Hospitals, Chennai. The MRI scans were performed at Bombay Hospitals and Apollo Hospitals, Chennai using 0.5T superconducting unit. All had medical and laboratory proof of CNS tuberculosis. The patients included in the study were those presenting with symptoms of intracranial TB for the first time including headache, mental changes, confusion, lethargy, altered sensorium, and neck rigidity. The patients excluded from the study were primarily those cases who had contraindication to MR imaging such as claustrophobia, cochlear implants and pacemaker. The physical examination and MR imaging studies were performed at presentation and at 6 weeks and 3 months after presentation. The cases were evaluated for location and type of intracranial structure involved by the disease process, and the pattern of meningeal involvement.

The MR examination primarily consisted of the following sequences (a) T1 weighted sagittal images, 400/18 (repetition time msec/echo time msec), with 256x256 matrix, four signals averaged, 28 cm field of view (FOV) and 10 mm section thickness; (b) T1 weighted axial images, 540/15 (repetition time msec/echo time msec), with a 256 x 256 matrix, four signals averaged 28 cm (FOV) and 10 mm section thickness; (c) T2 weighted sagittal spin echo images or fast spin echo images, 2140/80 (repetition time msec/echo time msec), with a 256x256 matrix, one signal averaged, 28 cm FOV and 10 mm section thickness, (d) T1 weighted coronal spin echo images 500 to 600/10-25 (repetition time msec/echo time msec), with a 256x256 matrix, four signal averaged, 28 cm FOV and 10 mm section thickness.

Results

18 patients with clinical findings and positive cerebrospinal fluid analyses for TB meningitis were evaluated with MRI before and after Gd-DTPA enhancement (0.1 mmol/kg), using 0.5T superconducting unit and the images were retrospectively analyzed. Multiple lesions occurred with a slightly higher incidence at 61% (Table-1). In all, 11 patients (61%) presented with meningitis (Ta-

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ble-2) and MRI in these cases showed diffuse, thick, meningeal enhancement at the basal cisterns. Meningeal lesions without parenchymal or vascular involvement were seen in 16% of cases while the combination of meningeal and parenchymal lesions was as common as 27% (Table-3). 7 of the 11 patients presented with acute meningitis, of which 2 also had arachnoiditis while the remaining 4 patients showed clinical features of chronic meningitis. 2 patients had extension of enhancing exudates into the spinal subarachnoid spaces (Table-4). While 6 patients had focal intraxial tuberculomas representing 33% cases, 3 patients presented with infarcts (Table-2). Infarcts, haemorrhagic [1] or bland [2], were detected in 3 patients; most were in the basal ganglia and internal capsules. Large middle cerebral arterial territory infarcts were seen in only one case. Hydrocephalus was identified in 4 patients and epidural lesions were noted in 2 cases (Table-2).

TABLE 1

Table showing the overall pattern of intracranial lesions

Location	Number	Percentage
Single lesions	7	39%
Multiple lesions	11	61%

TABLE 2

Table showing the type of intracranial involvement

Location	Number	Percentage
Meningeal	11	61%
Tuberculoma	6	33%
Infarct	3	16%
Hydrocephalus	4	22%
Epidural	2	11%

TABLE 3

Table showing the types and associations of meningeal lesions

Combination Lesion	Number	Percentage
Meningeal lesions only	3	16%
Meningeal and parenchymal	5	27%
Meningeal and vascular	2	11%
Meningeal, parenchymal and vascular	1	05%

TABLE 4

Table showing the distribution of meningeal lesions

Vertebral lesion	Number	Percentage
Basal cistern only	7	39%
Basal cistern and spinal extension	2	11%
Basal cistern and ependymal involvement	2	11%

Without Gd-DTPA enhancement, the MR images were generally insensitive to detection of active meningeal inflammation and granulomas. The signal intensity of granulomas was usually isointense to gray matter on both T1 and T2 weighted images, whether they were associated with diffuse meningitis or presented as localized tuberculoma(s). A few granulomas showed focal hypointensity on T2-weighted images. On Gd-DTPA enhanced T1-weighted images, abnormal meningeal enhancement indicating active inflammation was conspicuous and the granulomas often appeared

as conglomerated ring-enhancing nodules, which seems to be characteristic of granulomas. Thin rim enhancement around the suprasellar cisterns was observed in 9 out of 11 patients with meningitic sequelae.

Discussion

TB involving the CNS and its coverings is a complex and potentially devastating disease. The incidence of TB meningitis in the world has increased during the last few years. This has been attributed to the emergence of factors such as the spread among the homeless, a growing number of cases in patients with acquired immuno deficiency syndrome, the expanding population and the emergence of drug resistant TB bacillus [3,4]. Significantly, the presence of TB elsewhere in the body favours the diagnosis although its absence does not exclude it.

Mycobacterium tuberculosis infection of the CNS, results in a granulomatous inflammatory reaction that involves the meninges and/or cerebral parenchyma and often concomitantly the spinal cord. The disease therefore manifests itself in the CNS as meningitis, tuberculoma and tubercular abscess [5]. While cranial tuberculous meningitis (CTBM) is often a disease of childhood, tuberculomas and spinal TB are invariably an adult manifestation. The great majority of patients with neurotuberculosis are diagnosed and treated early because of characteristic clinical, imaging and CSF findings.

TB meningitis usually results from haematogenous seeding of the CNS from a primary pulmonary source of infection. Small tubercular lesions (Rich foci) initially develop in the CNS either in the meninges, the brain, or spinal cord and become active after a quiescent period. CSF seeding from the rupture of a pial or subependymal granuloma causes TB meningitis. TB meningitis is a disease which responds well to treatment when diagnosed and treated early. CNS involvement has frequently been found secondary to TB elsewhere in the body, particularly the lungs. Initially, the meningitic process can mimic a flu-like syndrome, followed rapidly by the development of profound neurologic deficits. The disease may present subtly as headache and mental changes or acutely as confusion, lethargy, altered sensorium, and neck rigidity. Typically, the disease evolves over 1 or 2 weeks longer than bacterial meningitis. Paresis of cranial nerves (ocular nerves in particular) is a frequent finding, and the involvement of cerebral arteries may produce focal ischemia. Hydrocephalus is common. Severe sequelae may result if cases of CTBM are subject to delayed onset of treatment. If unrecognized, TB meningitis is uniformly fatal. This disease responds to chemother-

apy. However, neurologic sequelae are documented in 25% of treated cases, in most of which the diagnosis has been delayed [6].

MR imaging features in CTBM depend largely on the stage of the disease process at which MRI is performed [1]. The early lesions of CTBM show little or no abnormality and can be missed on conventional spin echo images especially if contrast scans are not performed. The lesions are often picked up at this stage as subtle findings, by applying unique plain MRI sequences, such as FLAIR or specific contrast MRI sequences as MTC. Later the more obvious CTBM lesions display shortened T1 and T2 weighted images [7]. MRI of the brain often demonstrates a characteristic pattern of abnormal meningeal enhancement after the administration of intravenous gadolinium, typically at basal cisterns [8,9]. Fig. 1 is a post gadolinium axial T1 image exhibiting enhancing, exudative meningeal lesions of the basal cisterns and the Circle of Willis polygon along with a post gadolinium sagittal T1 image showing nonhomogeneous enhancement of the basal cisterns and a small focal enhancing granuloma at tectum. It has been well known that besides the basal cistern, enhancing lesions are also observed at meninges within the sulci over the cerebral convexities and at Sylvian fissure [10]. In Fig. 2, post gadolinium coronal T1 image shows diffuse enhancement of the suprasellar cistern with extension to both the Sylvian cisterns. Other sites where abnormal enhancement may be observed is at the choroid plexus or at the ependymal lining of the ventricular system [3,8]. Abnormal enhancement of these sites may be present even after a full course of antitubercular treatment on follow-up contrast MRI scans. The long term sequelae of tuberculous meningitis are infarcts, ependymitis, meningeal calcification or ependymal calcification and focal areas of atrophy. The more common complications of CTBM include cranial nerve entrapment, hydrocephalus and ischemic infarcts. Hydrocephalus can be communicating due to basal exudate or noncommunicating due to obstruction of cerebral aqueduct or fourth ventricular foramina. Communicating hydrocephalus is the most common complication and is secondary to obstruction of the CSF flow caused by meningeal exudates in the basal cistern. However, large parenchymal tuberculomas obstructing CSF flow pathway is not uncommon [11]. Remarkably, the incidence of hydrocephalus increases with the duration of illness [12]. Ischemic infarct as a complication of CTBM has a reported frequency of 21% to 38% in studies which employed CT as the imaging tool [13]. The infarcts are often haemorrhagic and commonly seen at basal

Fig. 1a & b : Tubercular meningitis : A) Axial T1 image after intravenous gadolinium shows enhancing, exudative meningeal lesions involving the basal cisterns and the Circle of Willis polygon. B) Mid sagittal T1 image after intravenous gadolinium shows nonhomogeneous enhancement of the basal cisterns and a small focal enhancing granuloma at tectum.

Fig. 2 : Tubercular meningitis : A) Coronal T1 image after intravenous gadolinium shows diffuse enhancement of the suprasellar cistern with extension to both the Sylvian cisterns.

Fig. 3a & b : Tubercular meningitis with arteritis causing hemorrhagic infarct : A) Axial T2 image shows infarct involving the right MCA territory. B) Post gadolinium axial T1 image shows enhancement within the infarct with associated patchy arterial enhancement due to slow flow associated with arteritis.

ganglia and internal capsule regions [8]. Fig. 3 is an illustrative case with axial T2 image showing infarct of the right, middle cerebral artery (MCA) territory,

while post gadolinium axial T1 image shows enhancement within the infarct with associated patchy arterial enhancement at Sylvian fissure due to slow flow associated with arteritis.

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. In adults, tuberculomas are predominantly in the frontal and parietal lobes of the supratentorial compartment, whereas in children, they are located in the infratentorial compartment. 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. Caseating granulomas with solid centre appear hypointense to isointense to brain on 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 weighted images with a peripheral hypointense rim representing the tuberculomas capsule and display rim enhancement on post gadolinium T1 weighted images [1,14]. Moderate perilesional edema is commonly associated with all the three types of parenchymal lesions. Fig. 4 illustrates this with an axial T2 image showing an ill-defined hyperintense mass lesion at the grey white matter junction of the left occipital lobe with surrounding oedema. An axial post gadolinium T1 image after intravenous shows a focal homogeneously enhancing lesion anteriorly abutting a small ring-enhancing lesion posteriorly.

Tubercular abscess is an uncommon feature of CNS TB. They can be multiple or single often resembling pyogenic abscess. Typical abscess has a capsule,

which has a thin, smooth, regular enhancing wall with moderate perilesional edema. Fig. 5 is a tubercular abscess seen in post gadolinium axial and parasagittal T1 images a rim enhancing left cerebellar lesion surrounding a hypointense central core, close to the left sigmoid sinus. Occasionally, intracranial TB manifests as an epidural mass. The pointers to the diagnosis are the peripheral rim enhancement, the central areas of necrosis, the presence of associated meningeal or parenchymal lesions and the chronicity of condition. Fig. 6 is a biopsy proven tubercular epidural lesion involving the left parietal region, seen as an elliptical, enhancing epidural lesion on left parasagittal T1 image and a coronal T1 image after intravenous gadolinium.

There are six important phenomena, which often aid in the cross-sectional imaging evaluation in a given case of intracranial tuberculoma [15]. These general rules comprise : (1) different lesions in a single patient may be in different stages of evolution

Fig. 4a & b : Tuberculoma : A) Axial T2 image shows an ill-defined hyperintense mass lesion located at the grey white matter junction at the left occipital lobe with surrounding oedema. B) Post gadolinium axial T1 image shows a focal homogeneously enhancing lesion anteriorly abutting a small ring-enhancing lesion posteriorly.

Fig. 5a & b : Tubercular abscess : A) Axial T1 image after intravenous dye infusion shows a cerebellar tubercular abscess close to the sigmoid sinus exhibiting smooth, rim enhancement of its wall surrounding a hypointense central core. B) Left parasagittal T1 image after intravenous gadolinium shows uniform rim enhancement of wall of the abscess.

Fig. 6a & b : Tubercular epidural lesion (proven by biopsy) A) Left parasagittal T1 image after intravenous gadolinium shows peripheral meningeal enhancement of an elliptical, epidural lesion at the left parietal region. B) Coronal T1 image after intravenous gadolinium shows the enhancing epidural lesion.

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 vs 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, the facility of early detection of bone marrow lesions. Besides its role in diagnosis, MRI is also a useful imaging tool for follow up of intracranial TB cases. 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.

While TB meningitis cannot be differentiated from other meningitides on the basis of MR findings, intraparenchymal tuberculomas show characteristic T2 shortening not found in most other space-occupying lesions. This is a useful aid in the appropriate clinical setting, where tuberculoma is suspected. The differential diagnosis of CTBM on MRI includes, infections, non-infectious inflammatory diseases and neoplastic involvement. Broadly, the infection group encompasses non-tuberculous bacteria, fungus and virus, while non-infectious inflammatory diseases are due to sarcoidosis and rheumatoid arthritis and neoplastic diseases can be primary or secondaries.

Early in the twentieth century, VA Moore had fittingly explicated that "as a destroyer of man, TB has no equal" [16]. Not surprisingly, this notion is relevant at the dawn of the new century. To conclude, MRI should be considered to be the imaging modality of choice for patients with suspected intracranial tuberculosis. Promising technical refinements in MRI are underway across the globe and clearly, these advances in technology hold the solutions to the mysteries of intracranial TB in the future.

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