Advanced Magnetic Resonance Imaging Techniques in Tuberculous Meningitis

Abstract

Background: Tuberculous meningitis (TBM) is a highly devastating manifestation of tuberculosis. So far, the major role of the neuroradiology in the management of TBM has been restricted to diagnosis and follow-up of the complications. This study aimed to establish the use of advanced magnetic resonance imaging (MRI) techniques in the early detection of sequelae of TBM like vasculitis and hydrocephalous. **Materials and Methods:** In this prospective observational study, 30 patients of TBM were recruited during 1 year at a tertiary care health center of northern India and their serial MRI brain was done. Patients were between 18 and 45 years of age. **Results:** Basal/Sylvian exudates were seen in 90% of patients, hydrocephalus was found in 30% of patients and infarcts were found in 27% of patients. No significant difference was found between the mean, mean diffusivity (MD), and mean fractional anisotropy (FA) in frontal white matter, basal ganglia, thalamus, pons of cases and controls. A significant difference was seen between mean cerebral blood flow (CBF) in the region of basal ganglia of cases and controls ($P < 0.05$). No significant difference was seen between mean CBF in frontal white matter, thalamus of cases and controls. Diffusion tensor imaging parameters, MD, and FA were abnormal in the region of infarcts (basal ganglia) in three patients in the first scan, the parameters normalized in one patient (late subacute to chronic infarct in the first scan), and they remained abnormal in two patients. **Conclusion:** Advanced MRI techniques (magnetization transfer imaging) is helpful in visualizing hyperintense thickened meninges in basal cisterns and Sylvian fissures on pre-contrast imaging, and in identifying reduced CBF in the region of basal ganglia.

Keywords: *Arterial spin labeled perfusion magnetic resonance perfusion imaging, diffusion tensor imaging, tuberculous meningitis*

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Introduction

Central nervous system (CNS) tuberculosis accounts for approximately 1% of all cases of tuberculosis, carries a high mortality and a distressing level of neurological morbidity. Tuberculosis remains a worldwide burden, with a large majority of new active tuberculosis cases occurring in underdeveloped and developing countries.[1]

In 80% of new tuberculosis cases, demographic factors such as poverty, crowding, malnutrition, and a compromised immune system play a major role in the worldwide epidemic. Several risk factors for CNS tuberculosis have been identified. Both children and HIV-coinfected patients are at high risk for developing CNS tuberculosis.[2-4] Other risk factors include malnutrition and recent measles in children and alcoholism, malignancies, and the use of immunosuppressive agents in adults.[5-7]

The most serious consequence of tuberculous meningitis (TBM), however, is the development of vasculitis in the vessels of the circle of Willis, the vertebrobasilar system, and the perforating branches of the middle cerebral artery, resulting in infarctions in the distribution of these vessels.[8]

During the early stages of the disease, noncontrast magnetic resonance imaging (MRI) studies usually show little or no evidence of any meningeal abnormality. With disease progression, swelling of the affected subarachnoid spaces occurs with associated mild shortening of T1 and T2 relaxation times in comparison with normal cerebrospinal fluid (CSF). Postcontrast T1-weighted images show abnormal meningeal enhancement, especially in the basal cisterns.

This study was done at a tertiary care center in northern India to establish

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the use of advanced MRI techniques such as arterial spin labeled (ASL) perfusion MR perfusion imaging, magnetization transfer imaging, diffusion-weighted imaging, diffusion tensor imaging (DTI) in the early detection of sequelae of TBM like vasculitis and hydrocephalous.

Diffusion-weighted imaging helps in the early detection of vasculitis. Vasculitis and infarcts have been known to be complications, but not many studies are available in literature about perfusion abnormalities in TBM patients. In this study, ASL perfusion imaging was performed to look for perfusion abnormalities (specifically in the region's most prone to infarction in TBM).

Materials and Methods

Patients of TBM were recruited from the Department of Neurology, and MRI was performed at a tertiary care center in Northern India.

The initial diagnosis was based on clinical features, CSF examination, confirmed by CSF culture, CSF-polymerase chain reaction (PCR), response to anti-tubercular treatment. A detailed history was taken. These patients were subjected to conventional MRI, (with and without contrast), DTI, ASL perfusion MRI, magnetization transfer imaging. A follow-up MR scan was also done using the same protocol in patients, in whom it was considered necessary by the treating physician based on the clinical condition (up to 9 months of the initial scan).

Imaging criteria for different sequences

- T1: For basic anatomy, localization of tuberculomas, demonstrating hydrocephalus
- T2 and fluid attenuated inversion recovery (FLAIR): for looking at focal lesions like tuberculomas, associated infarcts, sulcal hyperintensities associated with meningitis due to thick meningeal exudates. Looking at hydrocephalus and associated periventricular ooze
- Diffusion-weighted imaging: For diagnosing and aging of the various vasculitic infarcts a seen in some patients
- Precontrast magnetization transfer – MT sequences: For demonstrating hyperintense margins of tuberculomas and the thick hyperintense exudates on precontrast MT sequence
- Postcontrast T1 imaging: for demonstrating the tuberculomas, basal meningeal, and leptomeningeal enhancement
- ASL brain perfusion MRI: for demonstrating the values of cerebral blood flow (CBF) in the predefined brain regions and comparing them with values in the age-matched controls to see if any significant difference is seen between the two groups of cases and controls
- DTI: for finding out the mean diffusivity (MD values) and fractional anisotropy (FA values) in predefined brain regions and comparing them with healthy age- and gender-matched controls to see if any difference in seen

between the two groups of CNS tuberculosis and the healthy controls.

To call it altered MD and FA values and reduced CBF values, we compared between the cases and control groups in the respective brain region and if a significant difference was seen between the two groups, then it was labeled as reduced/increased such and such value.

Healthy age- and gender-matched controls were taken for comparing the quantitative data which were: CBF values in ASL brain perfusion imaging, the MD and FA values in DTI imaging.

They were taken such as to emphasize on the points that differences do occur in these parameters in CNS tuberculosis cases as compared to normal age- and gender-matched controls in a bid to say that these parameters are affected and also in which fashion they are affected in brains of patients with CNS tuberculosis.

Follow-up

A total of thirty TBM patients were enrolled in the study. Single follow-up scan was done only in those in which it is deemed relevant/necessary by the physician depending on the clinical condition. This follow-up scan was done anytime up to 9 months from the first scan.

Data analysis

All statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS, version 16.0, SPSS, Inc., Chicago, USA). For comparison, ASL data of 30 age-matched controls were taken from an unpublished MD thesis in our own department. For DTI data for controls was obtained by performing DTI on 15 age-matched volunteers.

MD unit-($\times 10$–3 mm^2/s) and FA values of patients and controls were calculated using Java-based software in regions of frontal white matter, basal ganglia, thalamus, and pons. The means of the values in patient and control groups were compared using independent sample t-test to find out if there was a significant difference between the mean MD and FA values between two groups.

ASL data were analyzed using "Image j" software, CBF values of patients and controls were taken out in regions of frontal white matter basal ganglia, thalamus. The mean CBF values for the patent and control groups were compared using independent sample t-test to find out if there was a significant difference between the two groups.

Results

The present study was conducted in the Department of Radiodiagnosis, at a tertiary care center in northern India.

There were thirty patients included in our study.

Out of these 30, 24 patients had proven CNS tuberculosis by CSF PCR. The remaining six patients had CSF

examination and clinical and radiological evidence in support of CNS Tuberculosis, CSF PCR could not be done in these as regards financial constraints and also enough supporting clinical and radiological evidence was there in these cases for them to be considered CNS tuberculosis.

Table 1 shows the age and sex distribution of patients, the study encompassed a total of thirty patients majority of individuals (67%) were in the age group of 21–30 years the age range was from 18 to 45 years male-to-female ratio was 1:1.

Table 2 shows the meningeal enhancement pattern. The most common pattern of meningeal enhancement was basal and/or Sylvian exudates-(90%). Among 27 patients with exudates-5 had exudates in basal cisterns, 2 had exudates in Sylvian fissures, remaining twenty had exudates in both the locations. About 96% of total exudates (seen on post contrast T1) were visible on pre-contrast MT imaging. Figure 1.

Table 3 shows appearance of lesions on T2-weighted imaging and enhancement pattern: Three types of lesions were seen in our patients as described in Table 3. All the 16 patients with tuberculomas showed lesions with hypointense center with hyperintense rim (T2), and ring enhancement. The next most commonly seen lesions were T2 hyperintense and showing nodular enhancement. These were additional lesions. Least commonly seen lesions were those with T2 hyperintense center and showing ring enhancement. Figure 2.

In addition, 29 patients had normal angiography (time of flight). One patient had narrowing in the left proximal MCA (middle cerebral artery) this patient had basal and bilateral sylvian exudates. Among complications, hydrocephalus was seen in 30%[9] of our patients. It was communicating type in all 9 of them. Infarcts were seen in 27% ($n = 8$) of patients. About 63% of total infarcts ($n = 8$) were in basal ganglia. About 37% of total infracts ($n = 3$) were in the thalamus. Three patients had infarcts (in basal ganglia) showing diffusion restriction (acute and early subacute phase) rest infarcts showed no diffusion restriction (late subacute to the chronic stage.)

Arterial spin-labeled perfusion magnetic resonance imaging data

Table 4 shows the comparison of means of CBF in frontal white matter, basal ganglia, and thalamus. No significant difference was seen between mean CBF in frontal white matter or thalamus of cases and controls; however, a significant difference was seen between mean CBF in the region of basal ganglia of cases and controls ($P < 0.05$). Figure 3.

Diffusion tensor imaging data

Table 5 shows comparison of means of MD and FA in the region of frontal white matter, basal ganglia, thalamus, and pons. No significant difference was found between the mean MD and mean FA in frontal white matter, basal gangli, thalamus or pons of cases and controls. Figure 4.

Follow-up imaging

A follow-up scan was done in 13 patients. Eleven had basal/Sylvian exudates at the first scan, ten of which resolved at the follow-up scan (91%). Seven patients had tuberculomas on first scan, they completely resolved in three patients (43%), whereas four patients (57%) had remaining tuberculomas on follow-up study [Table 6].
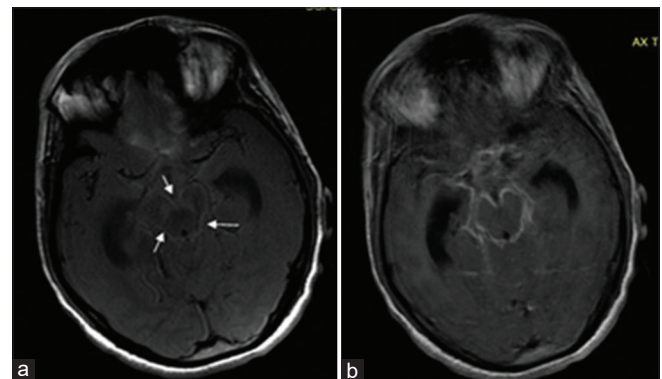

Figure 1: (a) hyperintense basal exudates on magnetization transfer sequence. (b) Postcontrast enhancement in these same locations confirming the exudates
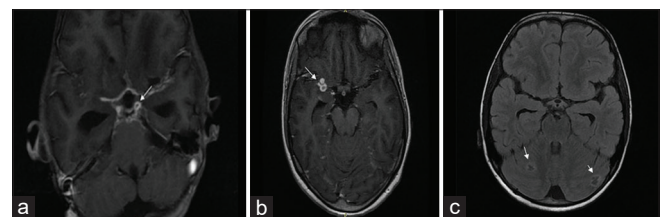

Figure 2: (a and b) exudates and tuberculoma in subarachnoid space. (c) Tuberculomas in bilateral cerebellar hemispheres
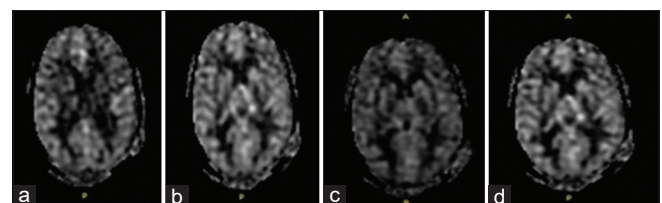

Figure 3: (a-d) CBF map at the level of basal ganglia, thalamus in one patient

Table 1: Age and sex distribution

Age (years)	Number of patients, n (%)	Males, n (%)	Females, n (%)
11-20	3 (10)	1	2
21-30	20 (67)	10	10
31-40	6 (20)	3	3
41-50	1 (3)	1	0
Total	30	15 (50)	15 (50)

Table 2: Meningeal enhancement pattern

Parameters	Basal/sylvian exudates	Leptomeningitis over cerebral convexities	Both
Number of patients, *n* (%)	15 (50)	3 (10)	12 (40)

Table 3: Appearance of lesions on T2-weighted imaging, and enhancement pattern

Parameters	Hyperintense with nodular enhancement	Hypointense center with hyperintense rim, ring enhancement	Hyperintense center, ring enhancement
Number of patients, *n* (%)	6 (37)	16 (100)	2 (13)

Table 4: Comparison of means of cerebral blood flow in frontal white matter, basal ganglia, and thalamus

Location	Parameter	Patients	Controls	*P*
Frontal white matter	CBF (ml/100 g/min)	19.18±3.6	19.67±4	0.36 (>0.05)
Basal ganglia	CBF (ml/100 g/min)	43.13±8	49.8±4.5	0.002 (<0.05)
Thalamus	CBF (ml/100 g/min)	43.9±5	43.7±4	0.15 (>0.05)

CBF: Cerebral blood flow

Table 5: Comparison of means of mean diffusivity and fractional anisotropy in region of frontal white matterbasal ganglia, thalamus, and pons

Location	Parameter	Patients	Controls	*P*
Frontal white matter	Mean MD ($\times 10^{-3}$ mm^2/s)	0.855±0.081	0.835±0.091	0.085 (>0.05)
	Mean FA	0.305±0.05	0.297±0.064	0.08 (>0.05)
Basal ganglia	Mean MD ($\times 10^{-3}$ mm^2/s)	0.816±0.10	0.797±0.05	0.39 (>0.05)
	Mean FA	0.178±0.03	0.176±0.03	0.7 (>0.05)
Thalamus	Mean MD ($\times 10^{-3}$ mm^2/s)	0.833±0.08	0.814±0.08	0.8 (>0.05)
	Mean FA	0.226±0.07	0.237±0.08	0.3 (>0.05)
Pons	Mean MD ($\times 10^{-3}$ mm^2/s)	0.793±0.12	0.738±0.14	0.07 (>0.05)
	Mean FA	0.552±0.11	0.569±0.09	0.4 (>0.05)

MD: Mean diffusivity, FA: Fractional anisotropy
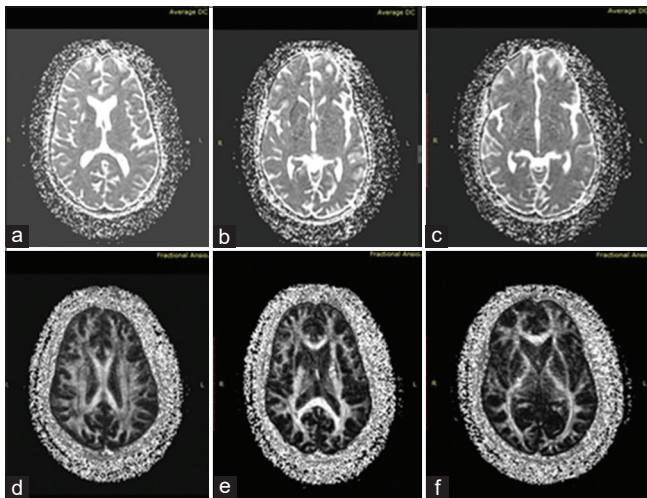

Figure 4: (a-f) MD and FA maps of a patient at the level of basal ganglia and thalamus

Changes in cerebral blood flow and diffusion tensor imaging parameters on follow-up scan

Five patients had reduced CBF (in basal ganglia-4, thalamus-1)-4 of them also had infarcts (basal ganglia-3, thalamus-1) in the first scan. Four out of those (80%) CBF restored to normal. 1 had persistent low CBF in the region of basal ganglia (had acute/early subacute infarct in basal ganglia on first scan). The basal and Sylvian exudates remained unresolved in this patient on follow-up scan. Follow-up scan was done 3 months after first scan in this patient.

DTI parameters (MD and FA) were abnormal in three patients in basal ganglia region-all three had infarcts (one patient had increased MD reduced FA-in region of late subacute/chronic infarct, two patients had reduced MD reduced FA-in region of acute/early subacute infarct)-the parameters normalized in the former patient (the one with late subacute/chronic infarct in first scan)-in the follow-up scan performed 5 months after first scan, overall infarct size decreased and now occupied a very small portion of basal ganglia while in the latter two patients (acute/early subacute infarct in first scan)-in the follow-up scan performed 3 months after the first scan the MD increased and FA remained low as the infarct became chronic. These changes are consistent with the evolution of infarct [Table 7].

Discussion

In this study, MRI findings of thirty patients of TBM along with DTI parameters and ASL perfusion data (CBF) were analyzed. Follow-up imaging of 13 patients was done between 3 and 7 months. The ASL perfusion data and the DTI

Table 6: Imaging findings in 13 patients on follow-up scan done between 3 and 7 months

Parameters	Total patients with follow up	Resolution of basal/sylvian exudates (n=11)	Resolution of tuberculomas (n=7)	Resolution of CBF abnormality (n=5)	Resolution of DTI abnormality (n=3)
Number of patients	13	10/11	Complete - 3/7	Resolved - 4/5	Resolved - 1/3
			Incomplete - 4/7	Persisted - 1/5	Persisted - 2/3

CBF: Cerebral blood flow, DTI: Diffusion tensor imaging

Table 7: Cerebral blood flow and diffusion tensor imaging parameters on follow-up scan

Parameters	1st scan	2nd scan
Reduced CBF	5 patients	4 resolved
		1 persistent
Reduced MD, reduced FA	2 patients (acute/early subacute infarct in basal ganglia)	2 patients
		Increased MD
		Reduced FA
Increased MD, reduced FA	1 patient (chronic infarct in basal ganglia)	1 patient
		Normal MD
		Normal FA

CBF: Cerebral blood flow, MD: Mean diffusivity, FA: Fractional anisotropy

data of patient group ($n = 30$) were compared with control group ($n = 35$ for ASL data and $n = 15$ for DTI data-age matched). For the 13 patients with follow-up comparison of findings of first and second scans was also done.

Gupta and Kumar[9] have discussed in their review article that the common sites for exudates in TBM are interpeduncular fossa, pontine cistern, peri-mesencephalic cistern, and suprasellar cistern and Sylvian fissures. The involvement of sulci over the convexities is less common in TBM patients. In our study, exudates in the above-mentioned locations were seen in 90% of patients ($n = 27/30$). In our study, enhancement of the leptomeninges in the sulci overlying the cerebral convexities was seen in 50% of patients ($n = 15$). Our findings are consistent with the study of Gupta et al.,[10] in which basal meningeal involvement was seen in 84.6% of patients (22 out of 26) and leptomeningitis was seen in 46% of patients.

Exudates (basal/sylvian) were visible on pre-contrast MT imaging in 96% of patients in our study. These findings are consistent with the previous studies. Gupta et al.[11] demonstrated hyperintense thickened meninges in basal and sylvian cisterns on pre-contrast MT images in all the 18 patients of TBM in their study. A possible reason for the hyperintensity of meninges on precontrast MT images is that the exudates are composed of cellular infiltrate, degenerated and partly caseated fibrin, tubercles, and bacilli. These are the probable reasons for differential MT ratio between brain parenchyma and inflamed meninges, and hence its visibility on precontrast MT-SE images (lower transfer of magnetization in inflamed meninges as compared to surrounding brain parenchyma). The space between pia and arachnoid in the region of CSF

cisterns favors lodgment of inflammatory tissue.[12] Hence, they are hyperintense on MT imaging as compared to thin meningeal inflammation over cerebral convexity sulci, which are not well visualized on precontrast MT imaging.

Kamra et al.[13] have also shown similar results. In their study on evaluating infectious meningitis with MT imaging, they included 100 patients with meningitis, 65 of which were of TBM. In all patients with TBM, abnormal meninges were visible on precontrast T1-weighted MT images as mild-to-moderate hyperintense signal around the brain stem in the basal cisterns, consistent with thickened pia-arachnoid, which showed enhancement after the administration of contrast material.

In our study, we found tuberculomas in 16 patients (53%). They were parenchymal in seven patients, in subarachnoid spaces in one patient, eight patients had lesions in both the locations. Gupta and Kumar[9] have described in their review article that the common site for tuberculomas in CNS tuberculosis is parenchymal (include cerebral hemispheres, basal ganglia, cerebellum, and brainstem). The ventricular system and meninges are rarely involved. However, in our study, we found that lesions in subarachnoid spaces are not that uncommon in tuberculous basal meningitis.

We found in our study that T2 hypointense lesions (with hyperintense rim) and showing ring-type enhancement were the most common type of lesions found in all 16 patients who had tuberculomas. Wasay et al.[14] have similar results as our study, in their study on 100 patients with intracranial tuberculomas. They found that an isointense or hypointense core with a hyperintense rim on T2-weighted and FLAIR images was the most common type of lesion. The core hypointensity of lesions on these images was related to caseation necrosis and a large number of cells.

Hydrocephalus and vasculitis are known complications of TBM. The adhesive exudates obstruct CSF leading to hydrocephalus. Obliterative vasculitis can cause infarction.[10] In our study, hydrocephalus was present in 30% of patients (9/30). All these were communicating type; none of the patients had obstructive hydrocephalus. Gupta and Kumar[9] have described in their article that in TBM hydrocephalus develops commonly as a result of blockage of the basal cisterns by the inflammatory exudates (communicating type). Obstructive hydrocephalus can develop occasionally due to the mass effect of a focal

parenchymal lesion or entrapment of the ventricle by granulomatous ependymitis.

In a study on 102 CNS tuberculosis patients by Wasay et al.[15] hydrocephalus was found in 37% of patients. Anderson et al.[16] have demonstrated hydrocephalus in 42% of cases in their study on 104 patients with TBM. Our findings are consistent with the two above-mentioned studies.

In their study on 26 patients with TBM Gupta et al.[10] found the presence of hydrocephalus in as many as 73% (19 patients). It was communicating in 17 patients and obstructive type in 2 of the 19 patients due to blockage of aqueduct by tuberculomas. In another study by Sobri et al.[17] on 42 TBM patients hydrocephalus was found in 62% (26 out of 42) patients. Thus, the incidence of hydrocephalus in TBM varies in different studies, and probably its occurrence depends on the severity of disease.

We found infarcts in 8 (27%) of our patients. Five patients had infarcts in the basal ganglia (63% of all infarcts), whereas the remaining 3 had small infarcts in the thalamus. Three patients had infarcts in basal ganglia which showed diffusion restriction (acute/early subacute stage). Infarcts in rest 5 did not show diffusion restriction (late subacute/ chronic stage).

The locations of infarcts in our patients were consistent with the results of the study of Gupta et al.[10] Twelve out of 14 patients with infarcts in their study were in the region of basal ganglia, internal capsule. Two patients had large infarcts in anterior and middle cerebral artery territory. In our study however, we did not see any large territory infarcts. Gupta and Kumar et al.[9] have discussed in their article about the same location of infarcts in TBM patients. This was attributed by the authors to the involvement of lenticulostriate arteries by the inflammatory exudates. Involvement of the large vascular territory such as the middle cerebral artery may also be encountered, although rare.

In our study, MR angiographic abnormalities were found in only one patient. None of our patients had large vascular territory infarcts. Kalita et al.[18] have performed a study on MRA in TBM patients including 67 TBM patients, 40 of which had infarcts, MRA-abnormality was found in 34 patients, most commonly the middle cerebral artery (MCA) was involved. In their study on 26 TBM patients, Gupta et al.[10] found that MRA revealed focal arterial narrowing in ten patients, the vessels commonly affected being the terminal segment of the internal carotid artery and the proximal segments of the middle and anterior cerebral arteries. One patient also had a small aneurysm of the proximal middle cerebral artery. Therefore it can be said that the percentage of patients with infarcts and angiographic abnormalities in various studies vary largely with the severity of disease.

In our study, we performed DTI on all patients, and compared MD and FA values in frontal white matter, basal ganglia, thalamus and pons-with 15 age-matched controls. We did not find any significant difference in these parameters between our patients and controls.

Abnormal DTI parameters (as compared to controls) were seen in regions of infarcts decreased MD with reduced FA in basal ganglia in three patients with acute/early subacute infarcts. Increased MD with reduced FA in was seen in three patients with late subacute/chronic infarcts (in basal ganglia in two patients, in thalamus in one patient). Malik et al.[19] have performed DTI on neonates with meningitis-compared to controls significantly decreased FA values were observed in periventricular white matter.

The MD and FA values in rest of patients (those without infarcts) were in the normal range. Trivedi et al.[20] have described similar changes in MD and FA values in infarct/ stroke-in their review article on DTI. In the transition from acute to subacute to chronic stroke, the MD first decreases in the acute phase and then renormalizes and subsequently increases.[20] On the other hand, anisotropy declines and remains low in chronic infarcts.

Ward et al.[21] have also shown on their study in infants with severe hypoxic ischemic encephalopathy that the MD and FA values in white matter, basal ganglia, and thalamus obtained in the 1st week were significantly less than controls, the MD values increased (pseudonormalized) while the FA values remained low on serial imaging.

We performed ASL perfusion imaging and CBF values were taken out in frontal white matter, basal ganglia and thalamus. Significant difference was found between the CBF in cases and controls in region of basal ganglia in our study ($P < 0.05$). Five of our patients had infarcts in the basal ganglia, all five had basal/sylvian exudates (4-both basal and sylvian, 1-sylvian). However, decreased CBF values were found in 11 other patients who did not have infarcts in the basal ganglia. All 11 of these patients had basal and sylvian exudates.

In their review article on stroke in TBM, Misra et al.[22] have described that most of the strokes in TBM are located in and around the basal ganglia (caudate, anterior thalamus, anterior limb, and genu of the internal capsule.) They have attributed the location of infarcts to the involvement of medial striate, thalamotuberal and thalamostriate arteries which are embedded in exudates and likely to be stretched by a hydrocephalus if present.

The location of infarcts mentioned are consistent with our study; however, the fact that we found reduced CBF in 11 other patients can be attributed to the involvement of perforators embedded in exudates as all 11 of these patients had basal/sylvian exudates.

Follow-up imaging was done in 13 patients-11 of these had basal and/or sylvian exudates in the first study, on follow-up

imaging exudates resolved in ten patients. Seven patients out of 13 had tuberculomas at initial study, three of these patients showed complete resolution of tuberculomas rest 4 still had some persisting lesions (incomplete resolution) on the follow-up study.

Among the 13 patients, five patients had reduced CBF (in basal ganglia-4, thalamus-1)-four of them also had infarcts (basal ganglia-3, thalamus-1) in first scan. All five had basal and/or sylvian exudates. In the follow-up, four out of those five (80%) CBF restored to normal. The exudates in these four also resolved on follow-up. One had persistent low CBF in region of basal ganglia (had basal ganglia infarct initially). Basal and sylvian exudates did not resolve in this patient.

DTI parameters (MD and FA) were abnormal in three patients in basal ganglia region, all three had infarcts (one patient had increased MD and reduced FA-subacute/chronic infarct, two patients had reduced MD reduced FA-acute/ early subacute infarct). The parameters normalized in the former patient-overall infarct size decreased and now occupied a very small portion of basal ganglia, whereas in the latter two patients, MD increased and FA remained low as the infarcts became chronic. These changes are consistent with the evolution of infarct.

Conclusion

Advanced MRI techniques (magnetization transfer imaging) is helpful in visualizing hyperintense thickened meninges in basal cisterns and Sylvian fissures on pre-contrast imaging, and in identifying reduced CBF in the region of basal ganglia. Limitation of our study-in the available time period of 1.5 years limited number (n = 30) of patients could be included and more extended follow-up was not possible.

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Conflicts of interest

There are no conflicts of interest.

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