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Research Article

Non-Enhanced 3D-TOF-MRA Evaluation in Tuberculous Meningitis and Correlation of Angiographic Abnormalities with Clinical Outcome

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Abstract

Background and Purpose: Neurovascular complications are the main causes of morbidity and mortality in tuberculous meningitis (TBM). However, there is paucity of data available on the spectrum of MRA abnormalities in TBM. We undertook this study to assess the spectrum of abnormalities involving the intracranial arteries in TBM by non-enhanced three- dimensional time-of-flight (3D-TOF) MRA and to determine if there is any relationship between abnormal angiogram and the disease outcome.

Materials and Methods: Twenty-five patients of TBM had clinical, laboratory, MRI and non-enhanced TOF MRA evaluation. Additionally, 11 patients underwent CT scans during follow up. The spectrum of abnormalities in MRA was evaluated. All patients were evaluated for the clinical outcome using the Glasgow outcome score (GOS) at discharge, 3 months and 6 months.

Results: Infarcts were noted in 16 patients, seen predominantly in the distribution of lateral lenticulostriate artery territory (68.75%). 64% patients had abnormal angiograms. MCA was most commonly involved (93.75%) followed by PCA (25%), supraclinoid ICA (18.75%) and ACA (6.25%). Arteries and their segments around the circle of Willis were most commonly involved. The MRA abnormalities include focal stenosis (60%), irregular segmental stenosis (12%), collateral formation (8%) and complete occlusion (4%). Poor outcome (GOS of 1, 2 and 3) was significantly found in patients with abnormal angiograms ($p = 0.014$).

Conclusion: Non-enhanced 3D-TOF-MRA is useful to assess the spectrum of angiographic abnormalities early in the course of illness. It can be used to predict the outcome of the disease in relation to vascular complications.

Keywords: Non-Enhanced; 3D-TOF-MRA; Tuberculous; Angiographic

Abbreviations

3D-TOF: Three-Dimensional Time-of-Flight; Tb: Tuberculosis; TBM: Tuberculous Meningitis; GOS:Glasgow Outcome Score

Introduction

Tuberculosis (TB) is a major global health problem. Geographically, the burden of TB is highest in Asia and Africa. India and China together account for almost 40% of the world's TB cases [1]. Tuberculous involvement of the CNS is an important and serious type of extra-pulmonary involvement, especially in children [2]. CNS TB may take several forms of which TBM is the commonest [3]. The overall mortality rate in TBM is 25% to 35% with long term morbidity ranging from 66% to 88% of survivors [4]. Noninvasive imaging modalities such as CT and MRI are routinely used in the diagnosis of TBM, with MRI offering greater inherent sensitivity and

specificity than CT scan. It also helps in demonstrating the complications of TBM [5]. Vasculitis is a complication that is commonly seen at autopsy in cranial TBM [6,7].

Conventional cerebral angiography was used in the past for most of the studies in TBM [8-12]. Among the non- enhanced MRA techniques that have been available for many years, 3D-TOF-MRA, because of its high sensitivity, faster acquisition times and non-invasiveness, remains the mainstay of intracranial arterial evaluation [7,13,14]. There is paucity of studies evaluating the spectrum of MRA abnormalities in TBM [15-17]. In a study of 20 patients with MRA in TBM by Gupta., *et al*. Gd-DTPA enhanced MRA was abnormal in 50% [15]. Kalita., *et al.* studied the MRA features in TBM but did not evaluate the spectrum of MRA abnormalities [17]. We undertook this study to assess the spectrum of abnormalities involving the intracranial arteries in TBM by non-enhanced 3D-TOF-MRA and to determine if there is any relationship between abnormal angiogram and the disease outcome.

Materials and Methods

In this two-year prospective study, twenty-five patients of TBM underwent detailed clinical, laboratory (including complete hemogram, serum biochemistry, CSF and HIV evaluation), chest x-ray, MRI and non-enhanced 3D-TOF-MRA evaluation. Additionally, 11 patients underwent CT scans during follow up. The patients were in the age group of 3 to 63 years with the maximum cluster in the 30 to 39 years age group. There were 52% (n = 13) males and 48% (n = 12) females. The diagnosis of TBM was based on clinical, radiological and CSF criteria. The essential criteria included presence of meningitic syndrome comprising headache, vomiting and fever for 2 weeks or more in whom malaria, septic and fungal meningitis were excluded. The supportive criteria included predominant lymphocytic pleocytosis with raised protein, radiological evidence of tuberculosis on CT or MRI scan which included exudate, hydrocephalus, tuberculoma and infarction singly or in combination, evidence of extra CNS tuberculosis and response to anti-tubercular therapy. Presence of the essential criteria and atleast two out of three of the supportive criteria with or without *one or more* of the definitive criteria was considered suggestive of the diagnosis of TBM [18]. Presence of AFB on CSF smear or culture (BACTEC) and positive AFB PCR or IgM antibody in CSF was considered diagnostic of TBM [19]. Patients with other stroke co-morbidities and HIV positive status were excluded from the study. On admission patients were assessed for Glasgow Coma Scale (GCS) scores [20]. Once diagnosed, all the patients were classified into three stages according to clinical severity (Medical Research Council Criteria, 1948) as summarized in on-line table 1 [21]. All patients received standard anti-tubercular therapy. Patients were evaluated for the clinical outcome using the GOS at discharge, 3 months and 6 months [22].

Stage I: Fully conscious and rational with no focal neurological signs.
Stage II: Confused but not comatose or having neurological signs of localization
such as hemiparesis or a single cranial nerve palsy.
Stage III: Comatose or stuporous or having multiple cranial nerve palsies or
complete hemiplegia or paraplegia.

Table 1: Criterion for staging TBM [21].

MRI was performed on a 1.5 T clinical scanner (Achieva *1.5T* Nova Dual*;* Philips Medical Systems*, Best, the* Netherlands) with a neuro-vascular head coil. MRI protocol included multiplanar FSE T2WI (TR,4443 ms/TE,100 ms), SE T1WI (TR,531 ms/TE,13 ms), FLAIR (TR,10000 ms/TE,125 ms), EPI-DWI (TR,2940 ms/ TE,65/ TSE Factor,47/ EPI Factor,47/ b value = 1000 secs/mm^2 and post contrast T1WI with MTC. All patients received intra-venous GD-DTPA (0.1 mmol/kg body weight). Non-enhanced 3D TOF multi slab FFE sequence was used for MRA (TR,23 ms/TE,6.91 ms out of phase/ flip angle, 20°/superior saturation band/ adjunctive techniques : TONE, CHARM, SENSE/acquisition time, 4 minutes 9s/ Matrix: 256 X 512/ Phase direction: LR/FOV,200 x 200 mm^2, slab thickness, 84 mm, slice thickness, 0.82 mm with no gap/ Number of slices : 120). Source and 3D MIP images were evaluated. The MRA was considered abnormal when one or more of the following features were present : (a)occlusion, defined as a total obstruction of a vessel, (b)focal stenosis, defined as a short and regular narrowing of a vessel, (c)irregular segmental stenosis, defined as non-occlusive segmental irregularities of the arterial wall and (d) collateral formation.

Chi square (χ^2) test was used where necessary to compare data and to obtain statistical significance. A p-value of less than 0.05 was considered statistically significant. Spearman's rank correla-

tion coefficient (ρ) was also used where necessary. A value of + 1 was considered suggestive of perfect positive correlation.

Results

The most common presenting symptom was fever (100%). Most common neurological presentation was of meningeal irritation (92%). Evidence of extra cranial TB was found in 44% patients of which 20% had pulmonary TB and 24% had TB of the sites other than the nervous system. Majority of the patients (68%) presented with features of clinical stage III at admission. Cytological analysis was abnormal in all the patients. The most common finding was raised lymphocytes in the CSF (80%).

Abnormal MRI was seen in 96% (n = 24) of patients. The abnormalities detected on MRI were hydrocephalus (80%), infarct (60%), periventricular ooze (52%), cisternal enhancement (48%), cerebral atrophy (28%), parenchymal enhancement (20%), convexity enhancement (20%), tuberculomas (16%), cerebral edema (16%), intraventricular enhancement (16%) and 3rd cranial nerve enhancement (4%) (Figures 1-3). The MRI findings in different clinical stages are summarized in on-line table 2. It was observed that severe hydrocephalus correlated positively with stage III of TBM (by Spearman's rank correlation). Enhancing exudates were noted most commonly with Stage III of TBM. Abnormal enhancement was seen most commonly in perimesencephalic cistern (91.66%) (Figures 1-3). Infarcts were noted in 60% (n = 16) patients. Infarcts were seen predominantly in the distribution of lateral lenticulostriate artery territory (68.75%). DWI was useful in depicting all the infarcts. On-line table 4 summarizes the distribution of infarcts in various arterial territories on CT and MRI.

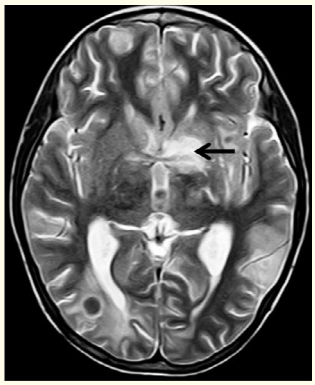
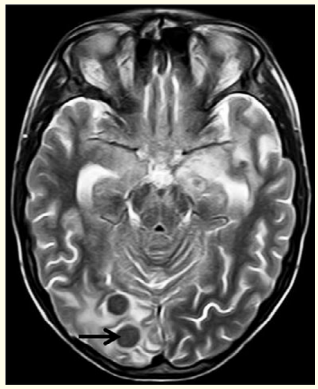


Figure 1A



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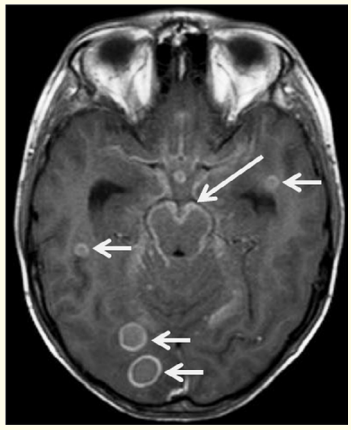


Figure 1C

Figure 1D

Figure 1E

Figure 1: Patient 5: Admission MR images (A to D) and follow up CT image (E) of a fifteen- year-old boy presenting with Stage III TBM. A and B, T2-weighted images show T2 hyperintense left basal ganglia infarct (arrow in A) and T2 hypointense tuberculoma (arrow in B). C, Post contrast T1-weighted image shows basal cistern enhancement (long arrow), ring enhancement of tuberculomas (small arrows) and mild dilatation of temporal horn of lateral ventricles. D, 3D-TOF-MRA axial MIP image shows severe focal stenosis of M1 segment of bilateral MCAs (short arrow), irregular segmental stenosis of P1 and P2 segments of left PCA (long arrow) and irregular segmental stenosis P2 segment of right PCA (broken arrow). E, Follow up axial CT image shows infarct (arrow) in left basal ganglia and caudate head (third day). Clinical outcome at the end of follow up was poor (severe disability).

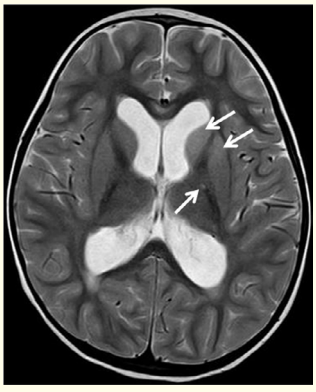
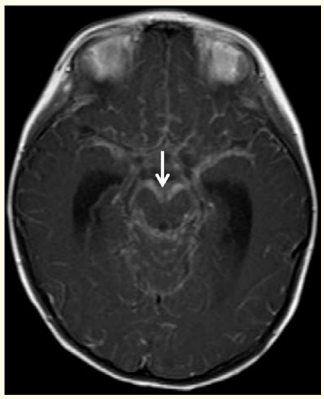


Figure 2A



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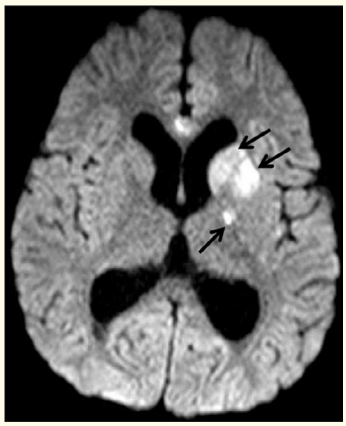

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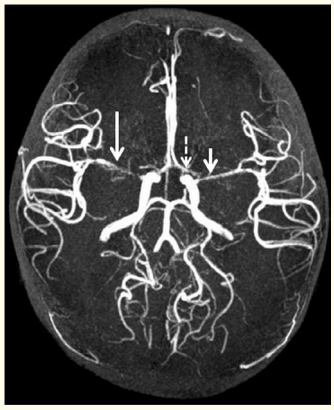

Figure 2D

Figure 2E

Figure 1: PPatient 6: Admission MR images of a four-year-old child presenting with Stage III TBM. A, T2-weighted MR image shows subtle hyperintense signal in the left basal ganglia, caudate head and internal capsule(arrows). B, Diffusion weighted image shows restricted diffusion in left basal ganglia, internal capsule and caudate head (arrows), suggestive of infarct. C, Post contrast T1-weighted image shows basal cistern enhancement (arrow) and prominent lateral ventricles. D and E, 3D-TOF-MRA axial MIP images (with different angulations) show severe focal stenosis of proximal M1 segment of right MCA (long arrow in D), irregular segmental stenosis of distal M1 segment of right MCA (long arrow in E), irregular segmental stenosis of M1 segment of left MCA (short arrow in E) and focal stenosis of A1 segment of left ACA (broken short arrow in E). Clinical outcome at the end of follow-up was poor (severe disability).

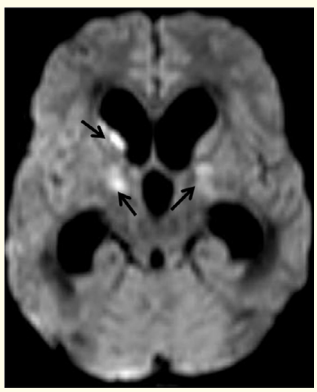

Figure 3A

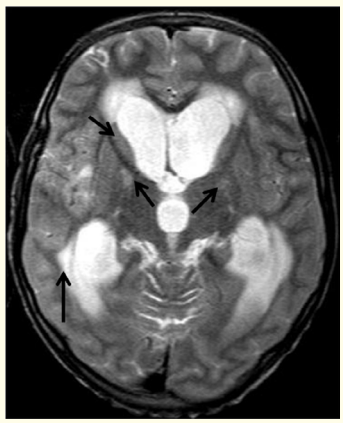

Figure 3B

Figure 3C

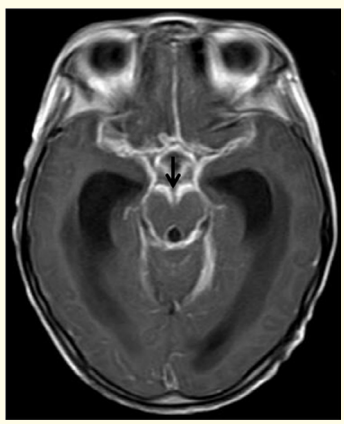

Figure 3D

Figure 3E

Figure 3: Patient 12: Admission MR images of a ten-year-old child presenting with Stage III TBM. A, T2-weighted image shows hydrocephalus with periventricular ooze (long arrows), bilateral internal capsule (genu) and right caudate head hyper-intensities (short arrows). B, Diffusion weighted image shows areas of restricted diffusion involving right caudate head and bilateral internal capsule (genu) regions (arrows) suggestive of infarcts. C, Post contrast T1-weighed image shows basal cistern enhancement (arrow). D and E, 3D-TOF-MRA axial (D) and coronal (E) MIP images show focal stenosis of proximal M1 segment of right MCA (short solid arrow in D), irregular segmental stenosis of M1 segment of left MCA and distal M1 segment of right MCA(long arrows in E), irregular segmental stenosis of M2 segment of bilateral MCA (broken arrows in D), focal stenosis of P3 segment of left PCA (long solid arrow in D) and focal stenosis of A1 segment of bilateral ACAs (dotted arrows in D and E). Patient succumbed to his illness after 15 days.

MRI Findings	Number and Percentage (%) in the 3 clinical stages		
	Stage I (n = 1)	Stage II (n = 7)	Stage III (n = 17)
Normal Scans	1 (100)	0 (0)	0 (0)
Hydrocephalus	0 (0)	5 (71)	15 (88)
Infarct	0 (0)	4 (57)	12 (70)
Periventricular white matter changes	0 (0)	2 (28)	11 (64)
Cisternal enhancement	0 (0)	1 (14)	11 (64)
Intra ventricular enhancement	0 (0)	0 (0)	4 (23)
Parenchymal enhancement	0 (0)	0 (0)	5 (29)
Convexity enhancement	0 (0)	0 (0)	5 (29)
Tuberculomas	0 (0)	0 (0)	4 (23)
Cerebral oedema	0 (0)	1 (14)	3 (17)
Cerebral atrophy	0 (0)	0 (0)	7 (41)
Cranial nerve enhancement	0 (0)	0 (0)	1 (5)

Table 2: MRI findings in 25 patients with TBM presenting in different clinical stages.

Infarcts were seen predominantly in stage III of TBM and showed positive correlation by Spearman's rank correlation. 16 out of 25 (64%) patients had abnormal MRA appearances (Figures 1-3). Abnormal MRA was seen predominantly in stage III of TBM and showed positive correlation by Spearman's rank correlation. Abnormal angiogram was seen frequently in patients with positive imaging findings like hydrocephalus, infarcts and abnormal post contrast enhancement. MCA was most commonly involved (93.75%). Other arteries that were involved include PCA (25%), supraclinoid ICA (18.75%) and ACA (6.25%). Arteries and their segments around the circle of Willis were most commonly involved. The arterial segments found abnormal on angiogram (n = 16) are summarized in on-line table 3. The abnormalities seen in the arteries on MRA, in order of frequency, are focal stenosis (60%, n = 15), irregular segmental stenosis (12%, n = 3), collateral formation (8%, n = 2) and occlusion (4%, n = 1). The most common finding was focal stenosis (60%) (Figures 1-3). Collateral formations seen in the two patients were in the form ofcluster of thin vessels in the region of basal ganglia. Correlation of clinical outcome with 25 angiograms at the end of follow up (assessed by GOS) is summarized in on-line table 5. Poor outcome (GOS of 1, 2 and 3) was significantly associated with abnormal angiogram (p = 0.014). Predictive value of angiogram for poor outcome was calculated. Sensitivity, specificity, positive predictive value and negative predictive value were found to be 0.813, 0.778, 0.867 and 0.70 respectively. Of the 11 patients who underwent additional CT scan for follow up, 90.9% (n = 10) patients had imaging abnormalities.

Arterial Segment	Number of patients	Percentage (%)
Middle Cerebral Artery	15	93.75
M1 Unilateral	5	31.25
M1 Bilateral	5	31.25
M2 Unilateral	5	31.25
M2 Bilateral	1	6.25
Anterior Cerebral Artery	1	6.25
A1 Unilateral	0	0
A1 Bilateral	1	6.25
A2 Unilateral	0	0
A2 Bilateral	1	6.25
Posterior Cerebral Artery	4	25
P1 Unilatral	1	6.25
P1 Bilateral	2	12.5
P2 Unilateral	0	0
P2 Bilateral	1	6.25
P3 Unilateral	1	6.25
Supraclinoid ICA	3	18.75
Unilateral	0	0
Bilateral	3	18.75

Table 3: Arterial segments found abnormal on angiogram (n = 16).

Arterial Territory	Number of Patients	Percentage (%)
Middle Cerebral Artery	8	50
Anterior Cerebral Artery	8	50
Posterior Cerebral Artery	2	12.5
Lateral Lenticulostriate Artery	11	68.75
Medial Lenticulostriate Artery	2	12.5
Thallamoperforating Artery	2	12.5
Anterior Choroidal Artery	3	18.75
Posterior Choroidal Artery	1	6.25

Table 4: Topographic distribution of infarcts on MRI and CT in arterial territories (n = 16).

Clinical Outcome	Number of patients	Angiogram	
		Normal (n = 9)	Abnormal (n = 16)
Poor (GOS 1,2,3)	15	2	13
Good (GOS 4,5)	10	7	3

Table 5: Clinical outcome correlated with 25 angiograms at the end of follow up (assessed by GOS).

GOS: Glasgow Outcome Score.

Discussion

TB is an infectious disease caused by the bacillus *Mycobacterium tuberculosis* and ranks as the second leading cause of death from an infectious disease worldwide, after the human immunodeficiency virus (HIV) [1]. TBM is the most common form of CNS involvement in TB [3]. Autopsy studies have shown that vasculitis is a common complication in cranial TBM [6]. The adventitial layer of small and medium-sized vessels develop changes similar to those of the adjacent tuberculous exudates. The intima of the vessels may eventually be affected or eroded by fibrinoid-hyaline degeneration. In later stages, the lumen of the vessel may get completely occluded by reactive subendothelial cellular proliferation [23]. Ischemic cerebral infarction resulting from the vascular occlusion is common sequelae of tuberculous arteritis. The MCAs and lenticulostriate arteries are most commonly affected [15,23].

Most of the studies in TBM that focused on associated vasculitis were done using conventional angiography [8-11]. Lehrer described the prominent angiographic features in TBM comprising of increased sweep of the pericallosal artery, narrowing of the supraclinoid portion of the ICA and narrowing or occlusion of smaller or medium sized arteries with scanty collaterals [8]. Mathew., *et al.* described cerebral angiographic findings in 10 cases of TBM. Narrowing and occlusion of intracranial arteries were the major findings, the most common sites of abnormalities being the supraclinoid portion of the ICA and proximal portion of the ACA and MCA. Hydrocephalic pattern and delayed circulation were the other features. In this report of 10 patients with TBM who underwent angiography, 8 had hemiparesis and neurological deficit was more permanent in this group [9]. In a clinico - angiographic analysis of 63 patients with TBM by Dalal, angiography was normal in 57% of patients and partial stenosis of the terminal segment of the

ICA was seen in 14% [10]. Very few studies were done with MRA in TBM although MRA is being increasingly used in routine clinical practice for neurological illnesses. It is more so in the context of the spectrum of MRA abnormalities seen in vasculitis secondary to TBM. Gupta., *et al.* studied 26 patients with TBM by MRI and performed MRA in 20 of them. Gadolinium - DTPA- enhanced MRA revealed focal arterial narrowing in 10 patients, the vessels commonly involved being the terminal segments of ICA and the proximal segments of the MCA and ACA. One patient also had a small aneurysm of the proximal MCA [15]. They did not evaluate the full spectrum of MRA abnormalities and their co-relation with clinical outcome. Unlike them, we used non-enhanced 3D-TOF-MRA for our study. Kalita., *et al.* studied the MRA features in sixty-seven patients. Half the patients with TBM had MRA abnormality involving both anterior and posterior circulations and 61.8% of them had corresponding infarcts. However, they did not evaluate the spectrum of MRA abnormalities [17]. In the present study, 16 out of 25 (64%) patients had abnormal angiograms. Abnormal angiogram was seen predominantly in stage III of TBM and showed positive correlation by Spearman's rank correlation (ρ = 1). Abnormal angiogram was seen frequently in patients with positive imaging findings like hydrocephalus, infarcts and abnormal post contrast enhancement (Figures 1-3). Most significant correlation was with hydrocephalus (p = 0.01). This was also reported by Kalita., *et al.* in their study. In their series, 69.5% patients with hydrocephalous had MRA abnormalities, whereas only 40.9% patients without hydrocephalous had MRA abnormalities.[17]In the present study, MCA was most commonly involved (93.75%). Other arteries that were frequently involved include PCA (25%), supraclinoid ICA (18.75%) and ACA (6.25%). Arteries and their segments around the circle of Willis were most commonly involved. The MRA abnormalities seen in the arteries werefocal stenosis (60%, n = 15), irregular segmental stenosis (12%, n = 3), collateral formation (8%, n = 2) and occlusion (4%, n = 1). The most common finding was focal stenosis (60%). Gupta., *et al*. also reported that focal narrowing was the most common abnormality in their study [15]. Collateral formation seen in the two patients were in the form ofcluster of thin vessels in the region of basal ganglia. Mathew., *et al*. found three types of collaterals in their study-the net-like cluster of thin vessels in the region of basal ganglia and base of the brain, transdural external-internal carotid anastomosis and increased cortical anastomosis with altered architecture of the arrangement. "Early veins" were also seen in their study [9]. The less number of collaterals in our study compared to conventional angiography may be explained by the inherent difficulty of non-enhanced TOF MRA to detect collaterals, especially in slow flow situations.

Patients with abnormal angiograms in our study either had infarcts on admission or later developed infarcts in the course of illness. 11 out of 25 patients were followed up by CT. 7 patients with abnormal angiograms showed infarcts on follow up CT. In our study, infarcts were seen predominantly in the distribution of lateral lenticulostriate artery territory (68.75%). Rojas-Echeverri., *et al.* studied 24 patients with DSA and found that the lateral striate territory was most commonly affected. Their rate of infarcts in TBM (66.6%) was higher than those from autopsy. Infarcts in their study were seen in 82% of those with abnormal and 54% of those with normal DSA. Kalita., *et al.* found that even though about half of their patients had infarction, they did not have corresponding MRA abnormalities [17]. Jinkins's report on intracranial TB included 14 patients with TBM, 7 of whom underwent angiography. Admission CT revealed hydrocephalus in 35.7% patients and infarcts in 28.5%. Arteritis was observed in 85.7% patients on angiogram. Late CT revealed an additional patient with an infarct [4].

We correlated the angiographic abnormalities with the clinical outcome of the patient which was assessed with GOS [22]. Poor outcome (GOS of 1, 2 and 3) was significantly associated with abnormal angiogram (p = 0.014). Sensitivity, specificity, positive predictive value and negative predictive value of abnormal angiogram for poor outcome in our study were found to be 0.813, 0.778, 0.867 and 0.70 respectively. In the DSA study by Rojas-Echeverri., *et al*. 7 of the 24 patients (29.1%) died, 4 of 11 with abnormal angiograms and 3 of 13 with normal DSA. They found the predictive values of abnormal angiography for infarcts. Sensitivity, specificity, positive predictive value and negative predictive value were 0.56, 0.75, 0.82 and 0.46 respectively. Mortality was not related to abnormal angiography but functional outcome was. They concluded angiography is justified in TBM only in specific clinical trials designed to assess new therapeutic modalities against infarcts [12]. Kalita., *et al*. followed up 62 of 67 patients, and reported that at 6 months, 13 patients died, 12 had poor recovery, nine partial recovery, and 28 had complete recovery which was not related to baseline MRA abnormality (P = 0.63). These differences in result of the two stud-

ies from the present study may be due to difference in sample size, stage of meningitis at presentation, duration of illness and response to anti-tubercular treatment. Other prognostic markers in TBM include clinical indices like level of consciousness, stage of meningitis, BCG vaccination, CSF findings and evidence of raised intracranial pressure, radiological findings such as hydrocephalus, infarction, severity of exudate and tuberculoma, and EEG findings like motor and somatosensory evoked potentials [19].

The limitation of this study is related to the widely noted fact that MRA overestimates the stenosis compared to conventional angiography [24-26]. We, therefore, did not try to quantify the percentage of stenosis in our study and rather used practically reproducible descriptive terms to define the abnormalities. Other pitfalls of MRA include difficulty to visualize slow blood flow and lack of sensitivity in the detection of small-vessel diseases [25,27,28] Additionally, the use of intravenous paramagnetic contrast to increase the signal of blood in vessels may allow better definition of small vessels [25]. Contrast enhanced MRA was avoided in this study for reasons including ease of examination by non-enhanced MRA, avoidance of excess contrast dose and cost effectiveness.

Clinically silent infarcts are seen in one-third of the patients of TBM [30]. MRI follow-up with inclusion of DWI may therefore be a better method for such study. The patients with MRA abnormalities need close follow-up and this may be a potential area of research for therapeutic intervention such as antiplatelet and corticosteroids [17]. In a randomized controlled trial on 118 patients with TBM, patients receiving aspirin had significantly lower mortality (21.7% vs. 43.4%) and insignificantly lower frequency of infarction compared to those without aspirin (24.2% vs. 43.3%) [31]. Gujjar., *et al.* suggested that HHH therapy (Hypervolemia - Hypertension - Hemodilution) along with steroid can probably be used to improve motor deficits and altered mental status due to focal cerebral ischemia in the acute stage. They suggested that further studies with additional monitoring (for example with continuous transcranial-doppler, MRI or CT angiographic studies and better hemodynamic monitoring) are indicated to explore its efficacy [29]. This study partially fulfills this need.

Conclusion

In conclusion, non-enhanced 3D-TOF-MRA is useful to assess the spectrum of angiographic abnormalities early in the course of illness. Vascular abnormalities are seen predominantly in the vessels around the circle of Willis and common abnormality is focal stenosis. Non-enhanced 3D-TOF-MRA can be used to predict the outcome of the disease in relation to vascular complications. The vascular complications are seen predominantly in stage III of TBM. The presence of vascular abnormalities suggest a poor outcome. Follow up MRA with MRI is a potentially important application in the management of TBM.

Acknowledgments

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