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Acute Asymptomatic Vasculitis in Patients with Meningitis

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Abstract: ***Tuberculous meningitis (TBM) is the most life - threatening form of extrapulmonary tuberculosis and the most common presentation of neurotuberculosis. TBM patients with infarcts are reported to be three times more fatal than patients without infarct. Contrast enhanced magnetic resonance imaging (MRI) is more sensitive than CT for the detection of ischemic changes, edema secondary to inflammation and basal meningeal inflammation. we reviewed the acute asymptomatic vasculitic infarct (by DWI MRI brain imaging) in 41 patients with tuberculous meningitis encountered during a 2 year period. MRI DWI imaging shows asymptomatic acute vasculitic infarct in 13 patients (31%). Thalamus was the most common site of infarct found in 7 patients (53.84%) followed by basal ganglion in 4 patients (30.76%). One patient had uncal (7.6%) and 1 patient had splenium infarct.***

Keywords: tuberculosis, MRI, infarcts

1. Introduction

Tuberculous meningitis (TBM) is the most life - threatening form of extrapulmonary tuberculosis and the most common presentation of neurotuberculosis. TBM patients with infarcts are reported to be three times more fatal than patients without infarct (7). It is widely believed that cerebral infarction in TBM is due to vasculitis or subsequent intimal proliferation or both with or without superadded thrombosis (17). Since TBM related stroke is rare and has undefined clinical signs, it is difficult for developing countries to diagnose in the clinic and therefore treatment is delayed (8, 9). Other reason why stroke may also be difficult to diagnose in TBM patients include: the stroke in the silent part of the brain, the patient is heavily comatose to notify or display signs or symptoms or the pre - existing weakness due to other lesions may obstruct the clinical picture (10, 11).

Computed tomography (CT) has been a diagnostic aid by identifying exudate in the basal cisterns and basal meningeal inflammation on contrast enhanced scans. However, the effectiveness of CT scans in detecting cerebral ischemia ranges only from 20.5% to 38% (3, 4).

Contrast enhanced magnetic resonance imaging (MRI) is more sensitive than CT for the detection of ischemic changes, edema secondary to inflammation and basal meningeal inflammation. The DWI MRI brain is sensitive in detecting acute infarcts when findings on the T2 - weighted MRI are normal (17).

In this study, we reviewed the acute asymptomatic vasculitic infarct (by DWI MRI brain imaging) in 41 patients with tuberculous meningitis encountered during a 2 year period.

2. Materials and Methods

Total 41 patients with TBM (median age 32, range 23 to 75) without any neurological deficits were included in the study conducted from September 2021 to September 2023 in L. L. R. M. Medical College, Meerut. Patients having focal neurological deficit, seizures, cranial nerve palsies or having contraindications for MR imaging were excluded. Male: female ratio was 1: 1. MR imaging with DWI brain were performed in all the patients.

Diagnostic criteria of tuberculous meningitis

The diagnosis of tuberculous meningitis was based on clinical, cerebrospinal fluid (CSF) and radiological criteria.

Tuberculous meningitis was considered ‘definite’ if acid - fast bacilli were demonstrated in the CSF. It was considered ‘probable’ in patients with one or more of the following: suspected active pulmonary tuberculosis on chest radiography, acid - fast bacilli found in any specimen other than the CSF and clinical evidence of other extra - pulmonary tuberculosis.

Tuberculous meningitis was considered ‘possible’ in patients with at least four of the following: a history of tuberculosis, predominance of lymphocytes in the CSF, duration of illness of >5 days, a ratio of CSF glucose to plasma glucose of <0.5, altered consciousness, turbid CSF or focal neurologic signs (12).
(13).

Definition of infarct

Infarct on magnetic resonance imaging (MRI) was defined as an area of abnormal signal intensity in a vascular distribution without any evidence of mass effect. Infarcts had to be hyperintense to gray matter on both spin density and T2 - weighted images. These lesions also had to be hypointense on T1 - weighted images, with intensities closer to that of CSF (14).

3. Results

All the patients had headache as the chief complaint. Twelve patients had vomiting also and 14 patients (34%) presented with classical triad of headache, vomiting and fever. Neurologic presentations on admission include nuchal rigidity (68%) and increased intracranial pressure (45%). Higher mental function, sensory, motor, and cranial nerves examination was normal in all the patients. Patients who

were examined with MRI had abnormal radiologic findings compatible with TBM including hydrocephalus (43%). MRI DWI imaging shows asymptomatic acute vasculitic infarct in 13 patients (31%). Thalamus was the most common site of infarct found in 7 patients (53.84%) followed by basal ganglion in 4 patients (30.76%). One patient had uncal (7.6%) and 1 patient had splenium infarct. (table1).

Table 1

No	Age	Sex	Site of Infarct
1	23	F	Rt. Basal ganglion
2	26	M	Rt. Uncal
3	66	M	Lt. Thalamus
4	75	F	Rt. Thalamus
5	49	M	Lt. Putamen
6	20	F	Lt. Splenium
7	17	F	Lt. Thalamus
8	34	M	Rt. Basal Ganglion
9	38	M	Lt. Thalamus
10	19	M	Lt. Basal Ganglion
11	22	F	Lt. Thalamus
12	48	F	Rt. Thalamus
13	58	M	Rt. Thalamus

4. Discussion

Tuberculous involvement of the central nervous system continues to be significant health problem in many underdeveloped countries. Infection with *Mycobacterium tuberculosis* begins with inhalation of *M. tuberculosis* bacilli, which then spreads through the lymphohematogenous system to the brain and meninges. The tuberculous bacilli are then discharged from these foci directly into the subarachnoid space to cause meningitis. The mycobacteria are deposited in the cerebral tissue where they incite a granulomatous reaction. The granulomas may remain asymptomatic or rupture into the subarachnoid space and leading to the formation of gelatinous exudate predominantly at the basal cisterns. The meningeal inflammatory exudate involves the adventitia, progressively spreads to the entire vessel wall, and leads to necrotizing panarteritis with secondary thrombosis and occlusion. The perforating vessels at the base of the brain, particularly at the origin of the lenticulostriate arteries, are predominantly involved. Most of the exudate is usually produced within the subarachnoid cisterns, thus accounting for the basal ganglia infarction (5). The infarcted areas were demonstrated as hyperintensity regions on T2 - W images and hypointensity on T1 - W images. These findings were more evident on T2 - W images than T1 - W images.

Headache and vomiting are common symptoms in patients with TBM. All patients in this study had headache with vomiting and fever as classical triad in 14 patients. The neurologic symptoms and signs in patients with TBM included drowsiness, meningeal irritation, cranial nerve paresis, seizures, hemiparesis, consciousness alternation, and coma (6). In our patients the meningeal irritation was the most common sign (68%) followed by vomiting. The findings of brain imaging in TBM include hydrocephalus, basilar enhancement, and infarcts, among which the most common abnormality is hydrocephalus. In our patients, hydrpcephalus was found in 43% patients. Cerebral infarction occurs in 15 - 57% of tuberculous meningitis (15). The perforating vessels at the base of the brain, particularly lateral lenticulostriate arteries (branch of M1 segment of middle cerebral artery) and thalamoperforating arteries (branch of P1 segment of posterior cerebral artery), are predominantly involved. Vasculitic infarcts are more evident in MR imaging in patients having focal neurologic deficits or cranial nerve palsies and basal ganglion is the most common site of infarct. However, it is a rare finding in those having no neurologic deficits. In our study, we found acute vasculitic infarct in 13 patients (31%) and the most common site of infarct was thalamus (53.84%) followed by basal ganglion (30.76%). Corticosteroids treatment is recommended for the patients with symptomatic vasculitic infarct. However, early initiation of steroids therapy may prevent development of focal neurologic deficit due to that infarct.

Early detection of acute vasculitic infarct in TBM patients having no neurological deficit may be one of the indication to start steroid to prevent development of serious complications like hemiparesis or cranial nerve palsies.

In conclusion, the present study documents that MRI Brain DWI imaging is helpful in detecting asymptomatic acute infarct in sizable number of tuberculous meningitis patients.

References

[1] Kennedy DH, Fallon RJ. Tuberculous meningitis. JAMA 1979; 241: 264–8

[2] Thwaites G, Chau TT, Mai NT, Drobniewski F, McAdam K, Farrar J, Tuberculous meningitis. J Neurol Neurosurg Psychiatry 2000; 68: 289–99

[3] Wallace RC, Burton EM, Barrett FF, Leggiadro RJ, Gerald BE, Lasater OE. Intracranial tuberculosis in children: CT appearance and clinical outcome. Pediatr Radiol 1991; 21: 241–6

[4] Gupta RK, Gupta S, Singh D, Sharma B, Kohil A, Gujral RB. MR imaging and angiography in tuberculous meningitis. Neuroradiology 1994; 6: 87–92

[5] Leung AN, Muller L, Pineda PR, FitzGerald JM. Primary tuberculosis in childhood: Radiographic manifestations. Radiology 1992; 182: 87–91

[6] Alarcón F, Escalante L, Perez Y, Banda H, Chacon G, Duenas G. Tuberculous meningitis: Short course of chemotherapy. Arch Neurol S1990; 47: 1313–7

[7] Anuradha HK, Garg RK, Agarwal A, et al. Predictors of stroke in patients of tuberculous meningitis and its effect on the outcome. J Med.2010; 103: 671–8.

[8] Deiva K, Sothratanak S, Husson B, et al. Febrile brain stroke and tuberculous meningitis: Persisting threat in nonendemic countries. Neuropediatrics.2010; 41: 273–5.

[9] Sheu J, Yuan R, Yang C. Predictors for outcome and treatment delay in patients with tuberculous meningitis. Amer J Med Sci.2009; 338.

[10] Pasticci MB, Paciaroni M, Floridi P, et al. Stroke in patients with tuberculous meningitis in a low TB endemic country: An increasing medical emergency? New Microbiologica.2013; 36: 193 - 8.

[11] Misra UK, Kalita J, Maurya PK. Stroke in tuberculous meningitis. J Neur Sci.2011; 303: 22–30

[12] Thwaites GE, Chau TTH, Stepniewska K, Phu NH, Chuong LV, Sinh DX, et al. Diagnosis of adult tuberculous meningitis by use of clinical and laboratory features. Lancet 2002; 360: 1287–92
[13] Kalita J, Mishra UK. Outcome in tuberculous meningitis at 6 and 12 months: a multiple regression analysis. Int J Tuberc Lung Dis 1999; 3: 261 - 5.
[14] Longstreth WT Jr, Dulberg C, Manolio TA, Lewis MR, Beauchamp NJ Jr, O'Leary D, et al. Incidence, manifestations, and predictors of brain infarcts defined by serial cranial magnetic resonance imaging in the elderly: the Cardiovascular Health Study. Stroke 2002; 33: 2376–82.
[15] Mishra, U. K., Kalita, J. & Maurya, P. K. Stroke in tuberculous meningitis. J Neurol Sci.15303, 22 - 30 (2011).
[16] Wasay M, Farooq S, Khowaja ZA, Bawa ZA, Ali SM, Awan S, et al. Cerebral infarction and tuberculoma in central nervous system tuberculosis: Frequency and prognostic implications. J Neurol Neurosurg Psychiatry 2014; 85: 1260 - 4.
[17] Del Brutto OH. Infections and stroke in: Ginsberg MD, Bogousslavsky J. editors. Cerebrovascular disease. Pathophysiology, diagnosis, and management. Blackwell Science: 1998. P.1628 - 46.