Section: General Medicine

Case Report

ARTERY OF PERCHERON INFARCT IN A CASE OF TUBERCULOUS MENINGITIS WITH TUBERCULOMA: A RARE PRESENTATION

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Abstract

Vasculitic infarcts are a known complication of tuberculous meningitis and an important cause of morbidity in these patients. Most of these infarcts are located in the basal ganglia and thalamus. Here we describe a 15-year-old girl who presented with fever, headache, and vomiting for 2 weeks followed by dizziness and a confusional state for 2 days. She had an Artery of Percheron (AOP) infarct and intracranial tuberculomas on MRI while tuberculous meningitis was confirmed from CSF findings. AOP infarct as a complication of tuberculous meningitis is an uncommon phenomenon, with only three cases reported in literature, so far. Diagnosis of AOP infarct can be challenging because of the diverse, often nonspecific clinical manifestations, paucity of focal neuro deficits, and normal appearance on preliminary neuroimaging.

INTRODUCTION

Tuberculous meningitis is a serious disease caused by Mycobacterium tuberculosis associated with significant morbidity and mortality. The complications of this disease include hydrocephalus, stroke, hyponatremia, raised intracranial pressure, seizure, and tuberculomas.[1] Strokes contribute significantly to the residual disability in these patients. The area of preference for infarcts in these patients includes the heads of the caudate nuclei, the anteromedial thalami, the anterior limbs of the internal capsules, and the genus of the internal capsules, the so-called tubercular zone.[2] Our patient presented with symptoms of chronic meningitis followed by acute disorientation. MRI revealed an Artery of Percheron (AOP) infarct along with tuberculomas. AOP infarct is an extremely rare complication of tuberculous meningitis, being reported only in three cases.[3–5] Suspicion of this uncommon infarct is important because clinically the patients usually do not have focal neurologic deficits commonly associated with strokes.

CASE REPORT

A 15 year old girl was admitted with complaints of holocranial dull headache and low grade fever for last 2 weeks. She had recurrent bouts of vomiting intermittently during this period. Two days prior to admission, she felt dizzy followed later by confusional state. Examination revealed normal vitals. She was drowsy and disoriented to time, place and person. Neck rigidity was present. There was no obvious paucity of limb movements. Planters were bilaterally flexor.

CT head did not reveal any abnormality. MRI Brain showed T2 FLAIR hyperintensities with diffusion restriction in bilateral thalami, consistent with Artery of Percheron (AOP) infarct. [Figure 1A and 1B]. Contrast study revealed multiple enhancing nodular lesions in bilateral cerebral and cerebellar hemispheres, some of which were pial-based. [Figure 2A, 2B, 3A and 3B]

CSF study revealed lymphocytic pleocytosis with raised protein and low sugar. [Cell Count: 90 Neutrophils: 30% Lymphocytes: 70%, Protein: 110, Sugar: 40]. CSF Tb PCR was positive.

Routine blood examination and electrolytes were within normal limits. Chest X-Ray was unremarkable.

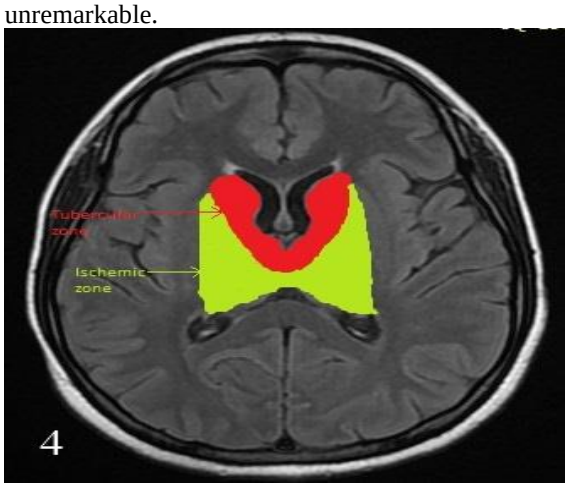

Figure 4

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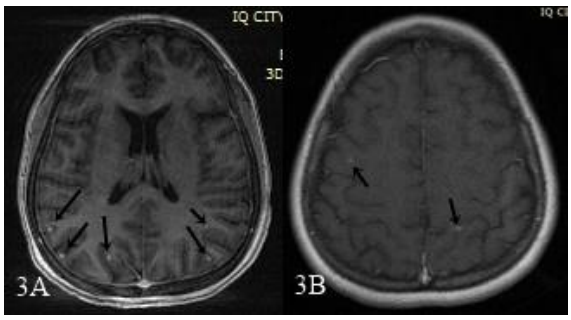

Figure 3

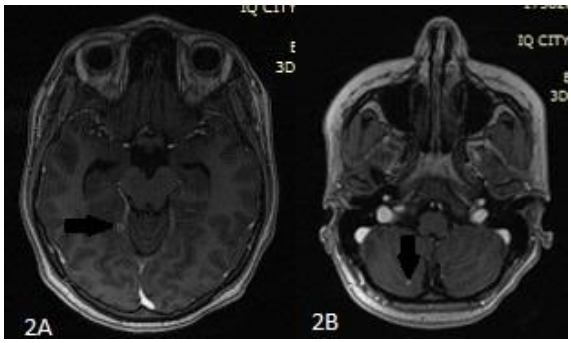

Figure 2

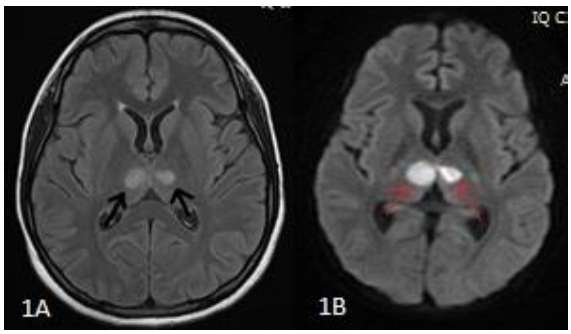

Figure 1

The patient was treated with antituberculous drugs (rifampicin, isoniazid, pyrazinamide and streptomycin) along with steroids. She improved with the treatment and was discharged after 2 weeks. She was doing well on her follow-up after one month.

DISCUSSION

Cerebral infarction is a known complication of tuberculous meningitis occurring in 15–57% of tuberculous meningitis patients, mainly during stage three of the illness.[6]

Most of the strokes in TBM are multiple and bilateral. The area of predilection of infarcts has been named "TB zone" (tubercular zone) as the region supplied by medial striate, thalamotuberal and thalamoperforating arteries; that is, the heads of the caudate nuclei, the anteromedial thalami, the anterior limbs of the internal capsules and the genus of the internal capsules. Similarly, an "IS" zone (ischemic zone) was defined as the region supplied by the lateral striate, anterior choroidal and thalamogeniculate arteries; that is, the lenticular nuclei, the posterolateral thalami, and the posterior limbs of the internal capsules. [Figure 4] E-Y. Hsieh et al showed that infarctions caused by TBM occurred predominantly in the TB zone (75%), followed by the IS zone (11.1%), the cerebral cortex (5.5%), subcortical white matter (2.8%).[2]

Mei-Ling Sharon Tai et al proposed the vascular supply classification and observed that this was more accurate than the classification of “TB zone” vs “ischemic zone as proposed by E-Y. Hsieh et al. They found that cerebral infarction in TBM involved especially the perforators and terminal cortical branches, rather than “TB zone” versus “ischemic zone”.[7] The mechanism of infarction is likely, vasculitis and intimal proliferation, while the role of thrombosis is uncertain.[8]

In our case, the patient had areas of restricted diffusion in bilateral thalamus corresponding to the territory of Artery of Percheron (AOP). AOP is an uncommon anatomic variant, in which a single dominant thalamoperforating artery supplies the bilateral medial thalamus with variable contribution to the rostral midbrain. Occlusion results in a characteristic pattern of ischemia: bilateral paramedian thalamic infarcts with or without midbrain involvement. AOP infarcts may give rise to a wide array of diverse clinical manifestations like altered mental status, memory impairment, psychosis, coma, dysarthria, aphasia, and oculomotor dysfunction. AOP infarct should be kept as a differential diagnosis in patients presenting with acute onset of altered sensorium with a normal CT scan. In the absence of lateralizing focal neurologic deficits and clinical syndromes unlike most other strokes, the diagnosis of such infarcts may be missed or delayed.[9–11]

Thrombosis of internal cerebral veins may also result in bilateral thalamic infarcts mimicking AOP infarct. Susceptibility-weighted image (SWI) with phase imaging can differentiate between the two.[12]

Our patient presented with altered mental status without any focal deficit which was caused by AOP infarct. Evidently this infarct was the complication of tuberculous meningitis, as proven by the CSF findings. In addition the patient also had multiple tuberculomas in brain which is also another complication of tuberculous meningitis.

To the best of our knowledge AOP occlusion leading to bilateral thalamic infarcts has been reported in only three cases of tuberculous meningoencephalitis.[3–5]

The learning points in this case were

1. AOP infarcts can occur as a rare complication of TBM
2. We must keep a high suspicion of AOP infarcts in such patients because of the diverse and often nonspecific clinical features, paucity of focal deficits and clinical syndromes and normal initial neuroimaging.

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CONCLUSION

Artery of Percheron infarct is an extremely rare ischemic complication of tuberculous meningitis, which may present with altered mental status in these patients. Physicians must be aware of this condition as early diagnosis is challenging and important in the management of these patients.

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