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Fatal tuberculous meningovasculitis

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Abstract— Tuberculous meningitis usually results in ischemic stroke, and rarely in hemorrhagic infarcts. We report a medicat history of a patient presenting a fatal outcome of tuberculous meningovasculitis. Brain MRI showed corticosubcortical T2-Flair and Diffusion weighted hyperintense lesions within frontal lobe, temporal lobe and right cerebella hemisphere, with contrast enhancement.

Key words: Vasculitis, Meningoencephalitis, Mycobacteria tuberculosis, MRI, PCR

INTRODUCTION

Tuberculous meningitis (TBM) usually results in ischemic stroke by arteritis. The clinical picture of strokes in TBM is polymorphous. Diffusion weighted imaging (DWI) provides information regarding tissue ischemia at an early stage allowing rapid treatment. We report in this paper, a case report of a patient presenting a fatal tuberculous meningovasculitis.

Case report:

A 45-year-old woman, previously healthy, admitted to the hospital with a 15 days history of worsening pancranial headache, confusion and fever with peaks at 40˚ C. She also complained of photophobia, nausea and recurrent vomiting. The neurologic symptoms progress after 8 days to increasing weakness of the lower limbs. On neurological examination, the patient was somnolent and disorientated, with bilateral sixth cranial nerve palsy and neck stiffness. Kernig's sign was positive. The patient was noted to have flaccid paraparesis (motor grade 4/5) with T8 sensory level. The deep tendon reflexes were present on the upper limbs and absent on the lower limbs. The plantar responses were extensor. The cerebrospinal fluid was noted to be clear colorless and contained 10 RBCs/mm^3, 560 WBCs/mm^3 (96% lymphocytes), glucose 78 mg/dL (serum level, 229 mg/dL), protein 230 mg/dL. Contrast brain and spine MRI was unremarkable, but electroencephalogram showed diffuse slowing. Laboratory tests revealed hyponatremia of 125 mmol/l(normal 136-144) and CRP of 25 mg/l (normal <7). Chest computed tomography scans showed bilateral basal alveolar syndrome with airbronchogram (figure 1) and real Time Mycobacteria tuberculosis PCR analysis performed on CSF was strongly positive. The patient was treated with 4 major antituberculosis and corticosteroids. Two days later, the patient presented sudden alteration of consciousness status immersing the patient in coma (GCS = 7). The patient was admitted in intensive care unit, where, performed brain MRI showed corticosubcortical T2-Flair and Diffusion weighted hyperintense lesions within frontal lobe (figure 2), temporal lobe (figure 3) and right cerebella hemisphere (figure 4), with contrast enhancement. The whole was suggestive of disseminated cerebral vasculitis complicating tuberculosis infection. Unfortunately, despite a heavy treatment, the patient went into deep coma and passed away by cardiac arrest, 5 days after her intensive care unit admission.

Ischemic involvement of small and medium sized vessels at the base of the brain is a common complication in tuberculous meningitis (TBM).[1]TBM usually results in ischemic stroke but rarely it may result in hemorrhagic infarcts that are attributed to both arterial and venous thrombosis.[2] Stroke in TBM occurs in 15–57% of patients. Many factors simultaneously play a role in its occurrence: stage of meningitis, hypertension, hydrocephalus, and exudates.[3]Most infarcts in TBM are as a result of hemodynamic hypoperfusion due to a variable combination of vasospasm, intimal proliferation and thrombosis of cerebral blood vessel walls [1]. It is regarded as a poor prognostic predictor of TBM.[2]
The pathophysiological mechanisms implicated in cerebral vasculitis are diverse and can include: a direct pathogenic effect of the infectious agent on the vessels, role of cytokines (tumor necrosis factor, vascular endothelial growth factor and matrix metaloproteineases) in damaging the blood brain barrier, immunological involvement via the induction of antigen expression on endothelial cells and the formation of immune complexes.[1] In late stage, organization of basal exudates may strangulate the vessels leading to vascular narrowing and focal weakness.[3]The common pathological changes in cranial blood vessels in tuberculous meningitis are arteritis: Infiltrative, proliferative, necrotizing or fibrinoid necrosis. Vascular involvement starts in adventitia and progressively encroaches to involve the entire vessel wall constituting panarteritis tuberculosa.[3] The uncommon complications include aneurismal dilatation, mycotic aneurysm, thrombus formation and venous sinus thrombosis.[2] these different pathological changes probably depend on the type, virulence of Myobacterium tuberculosis and on the host immune response to the infection.[2]

Most of the strokes in TBM are small, multiple, bilateral and located in the basal ganglia especially the 'tubercu-

lar zone' which comprises of the caudate, anterior thalamus, anterior limb and genu of the internal capsule, corresponding to the deep sylvian region.[1]These are attributed to the involvement of medial striate, thalamotuberal and thalamostriate arteries which are embedded in exudates and likely to be stretched by a coexistent hydrocephalus. Cortical stroke can also occur due to the involvement of proximal portion of the middle, anterior and posterior cerebral arteries as well as the supraclinoid portion of the internal carotid and basilar arteries.[2] Small vessel occlusion is common in the early stage of TBM resulting in monoplegia whereas middle cerebral or internal carotid arterial territory infarcts are common in advance stage resulting in hemi or quadriplegia.[2] Vertebrobasilar territory strokes earlier have been reported to be rare but recently 20% of strokes in TBM have been reported in vertebrobasilar territory based on MRI study.[2]Arteries traversing the sulci of the vertex are usually unaffected.[3]

TBM related stroke most commonly manifests with insidious focal neurological deficit, acute confusional state, meningeal syndrome, headaches, cranial nerve paralysis, coma and seizures. The other neurological manifestations depend on the location of infarctions. A large stroke may result in raised intracranial pressure.[2] Tuberculous cerebral vasculitis should be included in the differential diagnosis of any neurological deterioration arising during the course of tuberculous meningitis.[1]

Diffusion weighted imaging (DWI) provides information regarding tissue ischemia at an early stage (within the first hour after stroke) as compared to conventional magnetic resonance imaging (MRI).[4] DWI provided information not available on conventional T2 weighted imaging in terms of the multiplicity of lesions and detection of clinically unrelated area in a significant number of patients.[4]
MRA classically visualizes segmental narrowing, parietal irregularities and, sometimes, obstructions. Leptomeningeal vessels over the convexities of the brain may be stretched as a result of internal hydrocephalus or brain swelling. Sometimes luxury perfusion with early draining veins may be seen. Although the cerebral vasculature can be visualized by MRA, direct angiography remains the gold standard for imaging the vascular lumen.[1]

Corticosteroids should be prescribed in combination with antituberculous drugs to treat tuberculous meningitis.[1]This regimen were thought to reduce mortality and morbidity but their role in reducing strokes has not been proven. Aspirin also reduces mortality and its role in reducing stroke in TBM needs further studies.[2]

Conflict of interest The authors to have no conflict on interest

Acknowledgment

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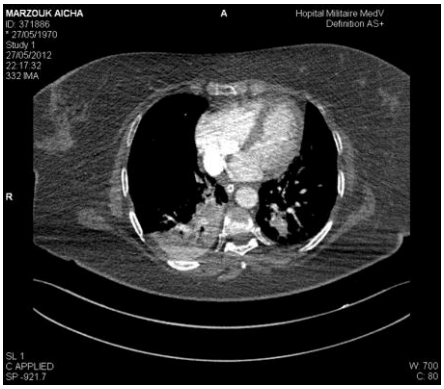

Figure 1: Chest computed tomography scans showing bilateral basal alveolar syndrome.

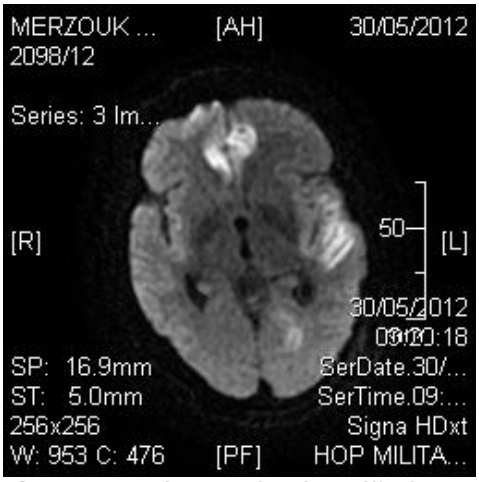

Figure 2: Brain MRI showing Diffusion-weighted hyper-intense lesions at the internal part of frontal lobe.

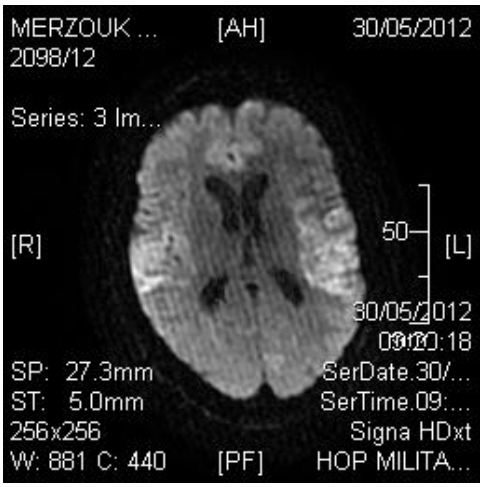

Figure 3: Brain MRI showing Diffusion-weighted hyper-intense lesions in both temporal lobes.

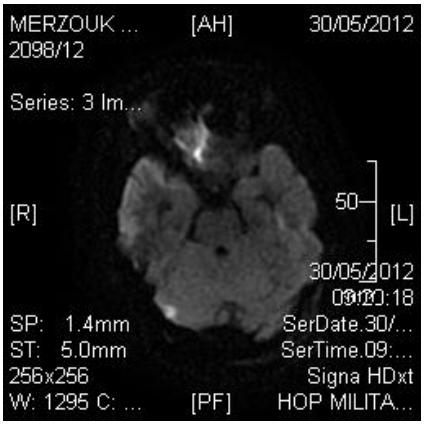

Figure 4: Brain MRI showing Diffusion-weighted hyper-intense lesions interesting the right cerebellar hemi-sphere

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