

Tuberculous meningitis presenting as Acute Ischemic Stroke

Neeraj Kumar Tulara¹, Nilesh Chaudhary²

From ¹Consultant Physician, Department of Medicine, ²Consultant Neurologist, Department of Neurology, Dr L H Hiranandani Hospital, Powai, Mumbai, India

Correspondence to: Dr. Neeraj Kumar Tulara, Department of Medicine, Dr L H Hiranandani Hospital, Hillside Avenue, Powai - 400072, Mumbai, India. E-mail: drntulara@rediffmail.com.

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ABSTRACT

Infection can cause cerebral vasculitis not only by direct invasion of the vessel wall, but by immune complex deposition, or through secondary cryoglobulinemia. Here, we report the case of tuberculous meningitis (TBM) in a middle-aged woman who presented with diabetic ketoacidosis, hyponatremia and accelerated hypertension and after 24 hours had right-sided hemiplegia. Her imaging MRI brain without contrast was suggestive of acute stroke. Hence, she was thrombolysed but she did not show any improvement. Repeat MRI brain with contrast showed tuberculomas and after starting Anti-Tubercular Treatment (ATT), she became alright within 3 days. There was no hemiplegia after 3 days of ATT. So TBM can also present as vasculitis with stroke and it should be kept in mind because recognition of TBM will change the line of treatment and patient can recover within a few days.

Keywords: *Anti tubercular therapy, Tuberculosis meningitis, Vasculitic stroke.*

Tuberculous meningitis (TBM) is a serious form of tuberculosis (TB) affecting the meninges that cover a person's brain and spinal cord. TBM causes significant morbidity and mortality especially if diagnosis and treatment are late. In 2012, there were 8.6 million new cases and 1.3 million deaths due to tuberculosis [1]. TBM occurs in 4% of patients with tuberculosis [2].

Stroke associated with TBM possesses high morbidity and mortality and risk of rapid progression despite appropriate treatment. The treatment includes antitubercular drugs with a duration of 12 to 18 months, though the role of aspirin is controversial, one study demonstrated absolute stroke reduction by 19 % [2].

CASE REPORT

A 50-years-old female, a known diabetic for the last 6 years was admitted in the hospital with complaints of generalized tiredness and episodic confusion. On examination, the patient was fully conscious, oriented and there was no neurological deficit. The pulse rate was 90 beats per minute and blood pressure was 180/90mmHg. There was no pallor, icterus or lymphadenopathy. On further investigations, she was found to have high blood sugars with Diabetic Ketoacidosis, and hyponatremia (124 mmol/l).

The patient was admitted to the hospital and on the second day, she developed sudden onset of aphasia with right hemiparesis as noted by the duty doctor for 1 hour. The National Institutes of Health Stroke Scale (NIHSS) score was 7. Immediate

Magnetic Resonance Imaging (MRI) brain done and her MRI brain Diffusion-weighted imaging (DWI) was suggestive of the left gangliocapsular infarct. So she was thrombolysed with 50 mg IV rtPA. The patient was shifted to ICU for further monitoring and continued on symptomatic treatment.

Post-thrombolysis, the patient was afebrile without any much change in her right hemiparesis. Computed tomography (CT) brain 24 hours post-thrombolysis was not showing any bleed but she had persistent right hemiparesis with aphasia and the patient became confused. Since there was clinical and radiological disparity and did not show expected improvement, contrast-enhanced MRI brain was done. Contrast-enhanced MRI was suggestive of basal exudates with left temporal, left frontal midline cerebellar ring-enhancing lesions on T1 Axial, Flair Axial, and T1 SAGITTAL suggestive of tuberculous meningitis with tuberculomas (Fig. 1 and 2). Fundus examination was done which showed no Papilledema. Cerebrospinal fluid (CSF) examination found that proteins were 190 mg/dl, total cell count 537 cells with lymphocytes (87%), glucose 61mg/dl (serum glucose 180mg/dl), and Adenosine deaminase (ADA) was 13. Her TB PCR, gene expert, and viral markers were negative but X-ray chest was normal.

In view of the MRI and CSF findings, she was started on 5 drug anti-tuberculous drugs adjusted as per her weight (Rifampin, Isoniazid, Ethambutol, Pyrazinamide, and Kanamycin) along with dexamethasone as an anti-inflammatory. The patient showed significant improvement within 48 hours after Anti-Tuberculous therapy (ATT) initiation and now she was able to move her right side with a Motor Power 4/5 in both upper and lower limbs. She

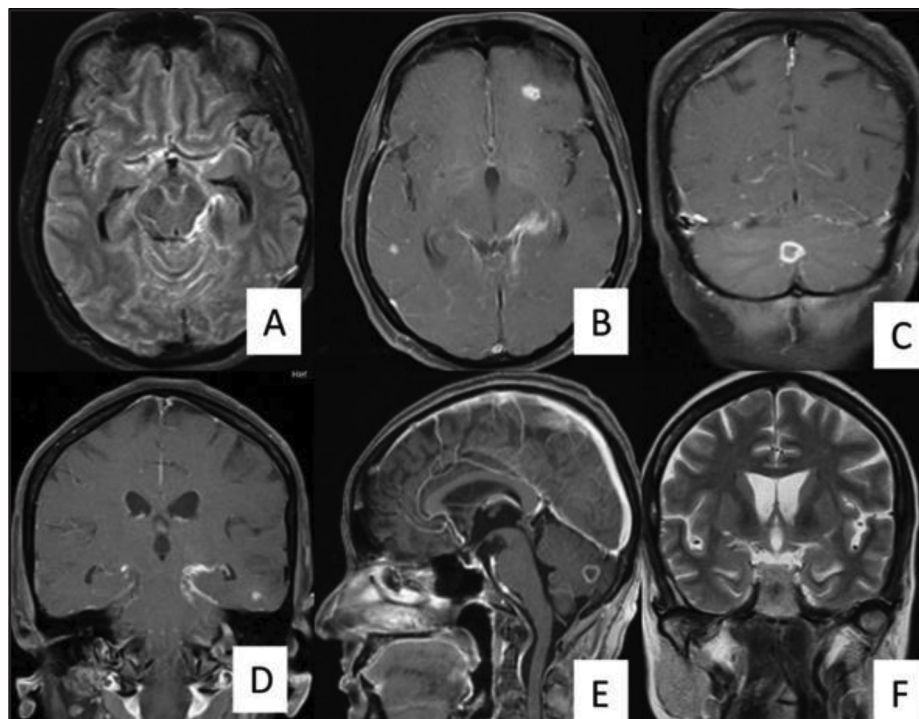


Figure1: (a) Tuberculous meningitis showing basal exudates; (b) Left frontal granuloma with basal exudates; (c and e) cerebellar vermis tuberculoma; (d) Left temporal granuloma; (f) T2 sequence

was fully oriented to Time, Place and Person and was mobilized. She continued the same ATT. Her sugar level and BP were under control on oral medications and discharged on ATT. The patient was persistently on follow-up, showed full recovery and at present is on maintenance ATT.

DISCUSSION

Tuberculous meningitis (TBM) is a complicated form of extrapulmonary tuberculosis (10% of all tuberculosis cases) [3,4,5]. It clinically manifests as subacute to chronic meningitis

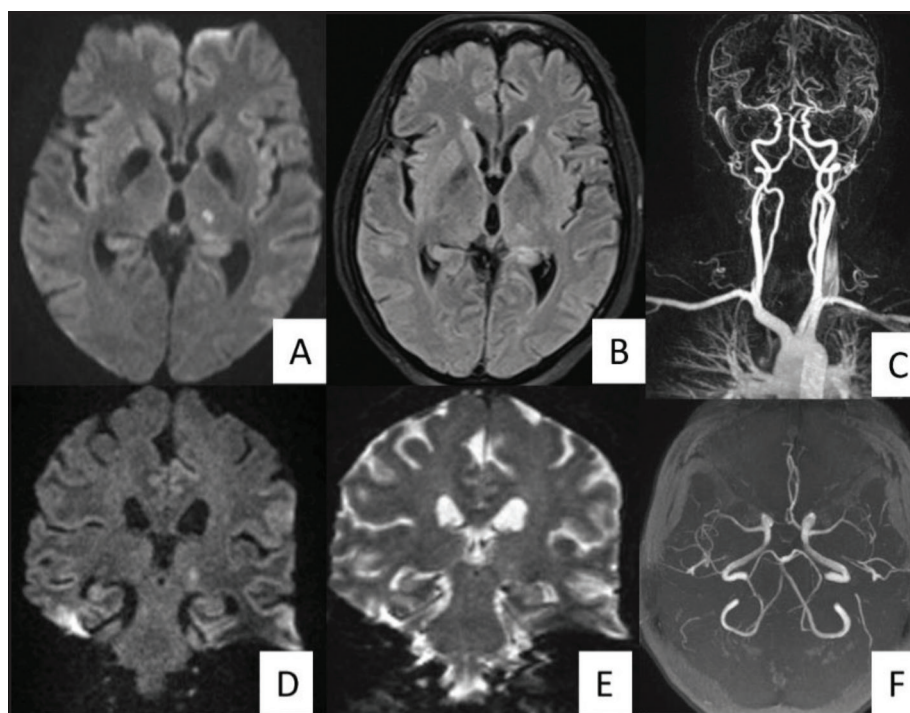


Figure 2: (a) DWI Axial brain acute infarct left thalamus; (b) FLAIR; (c) MR angiogram extracranial; (d) MRI DWI; (e) ADC; (f) MR angiogram intracranial

with fever headache and altered mentation [1,2,6,7]. Risk factor includes retroviral infection, diabetes mellitus, malignancy and chronic kidney disease[5]. Complications of tuberculous meningitis infections include hydrocephalous and secondary tuberculous vasculitis manifesting as stroke (20% of TBM). Stroke associated with TBM has high morbidity and mortality and risk of rapid progression despite appropriate treatment. Stroke associated with TBM associated vasculopathy (vasculitis) are mainly ischemic involving basal ganglia region and middle cerebral arterial territory but it can also involve posterior circulation (basilar artery) mainly due to the perforators and terminal cortical branches involvement [8]. Mechanisms of vasculopathy in TBM which manifest as stroke includes basal exudates associated inflammation, spasm and inflammation-related thrombosis, progressive hydrocephalous with vascular stretching, necrotizing pan arteritis with or without thrombosis secondary to lymphohistiocytic, neurophilic or mixed exudates, and angiopathy of perforating vessels[1,5,6]. Treatment includes antitubercular drugs for a duration of 12 to 18 months, though the role of aspirin is controversial, one study demonstrated absolute stroke reduction by 19% [2]. Adjuvant dexamethasone for four weeks works as an anti-inflammatory and helps to minimize complications of TBM like hydrocephalous and vasculopathy related to tuberculous meningitis [1].

Our case was unique as the presentation was acute ischemic stroke brain DWI suggestive of restriction without other evidence of meningitis but subsequent contrast MRI demonstrated exudates. We used r-tpA in this case which helped her in early recovery from an acute ischemic stroke due to vasculopathy.

CONCLUSION

Tuberculous meningitis can present as acute ischemic stroke due to tuberculosis vasculitis. r-tpA in acute stroke setting in tuberculosis vasculitis can be beneficial

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