

## Transcranial Doppler as a Bedside Evaluation Tool in Tubercular Meningitis: Case Report and Review of Literature

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**Abstract:** Transcranial Doppler (TCD) is an emerging modality for bedside evaluation of cerebral hemodynamics. It provides an easy, convenient and non-invasive modality for the assessment of intracranial blood flows and pressure. Role of TCD in evaluation and management of vasculopathy related to tubercular meningitis (TBM) has shown contrasting results in previous studies. We describe the TCD findings in a patient of TBM with extensive basal exudates, which were reversible after treatment with antitubercular therapy (ATT) and steroids.

**Keywords:** Transcranial doppler; Tubercular meningitis; TBM vasculopathy; Vasculitic infarcts; Steroids

### 1. INTRODUCTION

Vasculopathy is a well-recognized and frequently catastrophic complication of Tubercular Meningitis (TBM).<sup>1</sup> The hallmark feature is development of basal exudates with cranial nerve palsies or cerebral infarctions.<sup>2</sup> Extensive damage to cerebral vessels causes widespread infarctions, including brainstem involvement.<sup>3,4</sup> Transcranial doppler ultrasonography (TCD) provides a safe, efficacious, non-invasive bedside method of evaluation of arteries at the base of the brain.<sup>5</sup> Critically ill patients with advanced disease may require frequent close monitoring of cerebral hemodynamics, including intracranial blood flows and pressures. Repetitive evaluations with Magnetic Resonance Imaging (MRI) or Lumbar puncture may be difficult and thus TCD may play an important role in the management of these patients. Here we report a case of TBM with vasculopathy who showed elevated blood flow velocities in the Posterior (PCA), Middle (MCA) and Anterior Cerebral Arteries (ACA). These changes were reversible after treatment with ATT and steroids.

### 2. CASE HISTORY

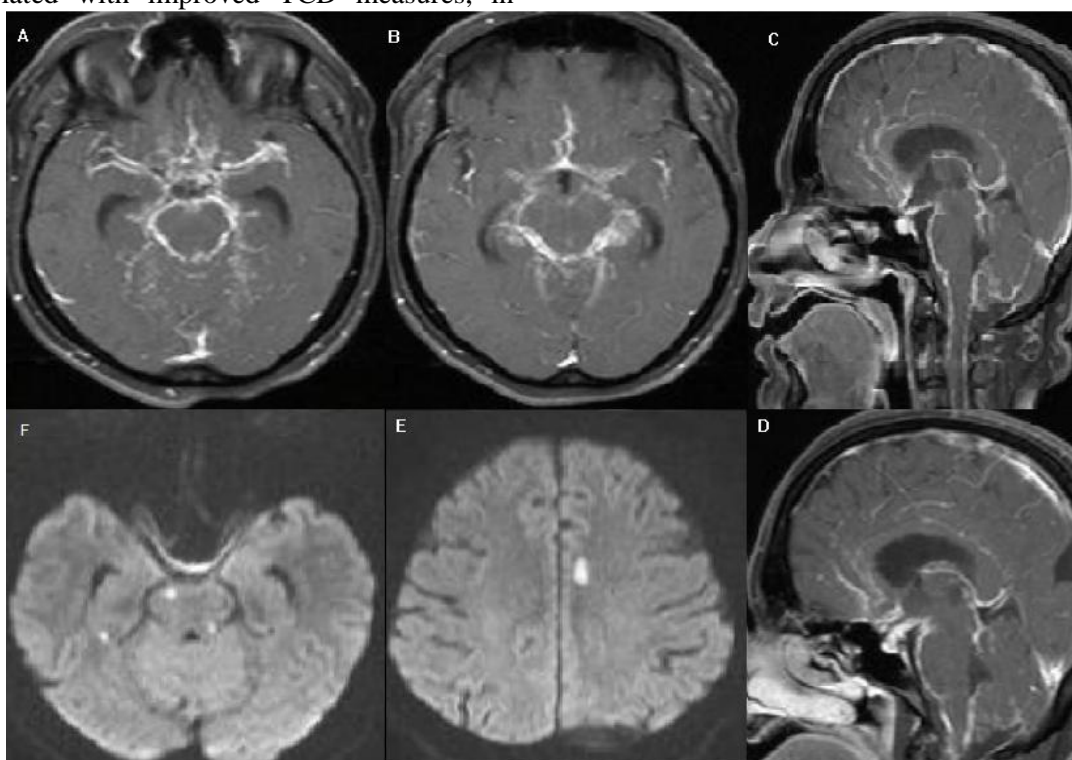
43-year-old lady, without comorbidities, complained of intermittent fever associated with holocranial throbbing headache for 3 months and irrelevant speech for 5 days prior to presentation. She was drowsy, arousable to

verbal stimuli and was able to follow simple commands. Vitals were stable and she had neck stiffness. Cranial nerves, eye, motor and sensory examination were grossly normal. Cerebellar examination was remarkable for limb ataxia. Blood investigations showed high Erythrocyte Sedimentation Rate (ESR) of 42. Complete blood counts, electrolytes, renal and liver functions were normal. A Magnetic Resonance imaging of the brain showed T2/ FLAIR hyperintensities in bilateral brainstem, right temporal lobe and left centrum semi-ovale with diffusion restriction, suggestive of acute infarcts. (Figure 1) A post-contrast study revealed basal exudates with pachymeningeal enhancement, consistent with tubercular meningitis. A lumbar puncture showed high opening pressure of 30 cm of water with Cerebro-spinal fluid (CSF) analysis showing 45 cells (70% lymphocytes) with high protein- 800 mg/dL and CSF sugar of 45 mg/dL. CSF gram stain, culture, KOH mount, testing for cryptococcal antigen and India ink preparation were negative. A CSF meningo-encephalitis panel, including testing for neuro viral infections was negative. CSF testing for tuberculosis with Polymerase chain reaction (TB-PCR) was positive. A bedside TCD study, using a 2 MHz probe, at the time of presentation showed high mean and peak systolic velocities in the bilateral Middle cerebral artery (Right – 120 cm/sec, left- 138 cm/sec) and bilateral posterior cerebral artery (right P1 – 109 cm/sec,

left P1 – 157 cm/sec). Anterior cerebral artery (ACA) velocities were 82.8 cm/sec on the right and 119 cm/sec on the left. (Figure 2)

Corresponding PI values in Right MCA was 0.63, in left MCA was 0.71, in right ACA - 1.04 and in left ACA was 1.43. She was initiated on anti-tubercular therapy along with intravenous steroids. She showed symptomatic improvement in sensorium and headache after 3 days of initiation of steroids. On day 12, repeat MRI brain showed resolution of basal exudates. This correlated with improved TCD measures, in

terms of reduced peak systolic velocity in the right MCA to 100 cm/sec, in left MCA to 79.7 cm/sec, in right ACA to 50.8, left ACA to 102, in right PCA-P1 to 77 and left PCA-P1 to 68.1 cm/sec. Similar results were seen in the peak velocities of Basilar artery. (Figure 2) Mean flow velocities in all intracranial vessels showed a similar decreasing trend after treatment with steroids. The PI values did not show any significant reduction in the Middle or a posterior cerebral artery, however, a decrease in PI values was noted in the Basilar artery.



**Figure1:** MRI brain with contrast showing extensive meningeal enhancement and basal exudates (a,b: axial cuts and c,d: sagittal cuts). Diffusion weighted images showing acute vasculitic infarcts in the bilateral midbrain, right temporal region and left cerebral cortex.

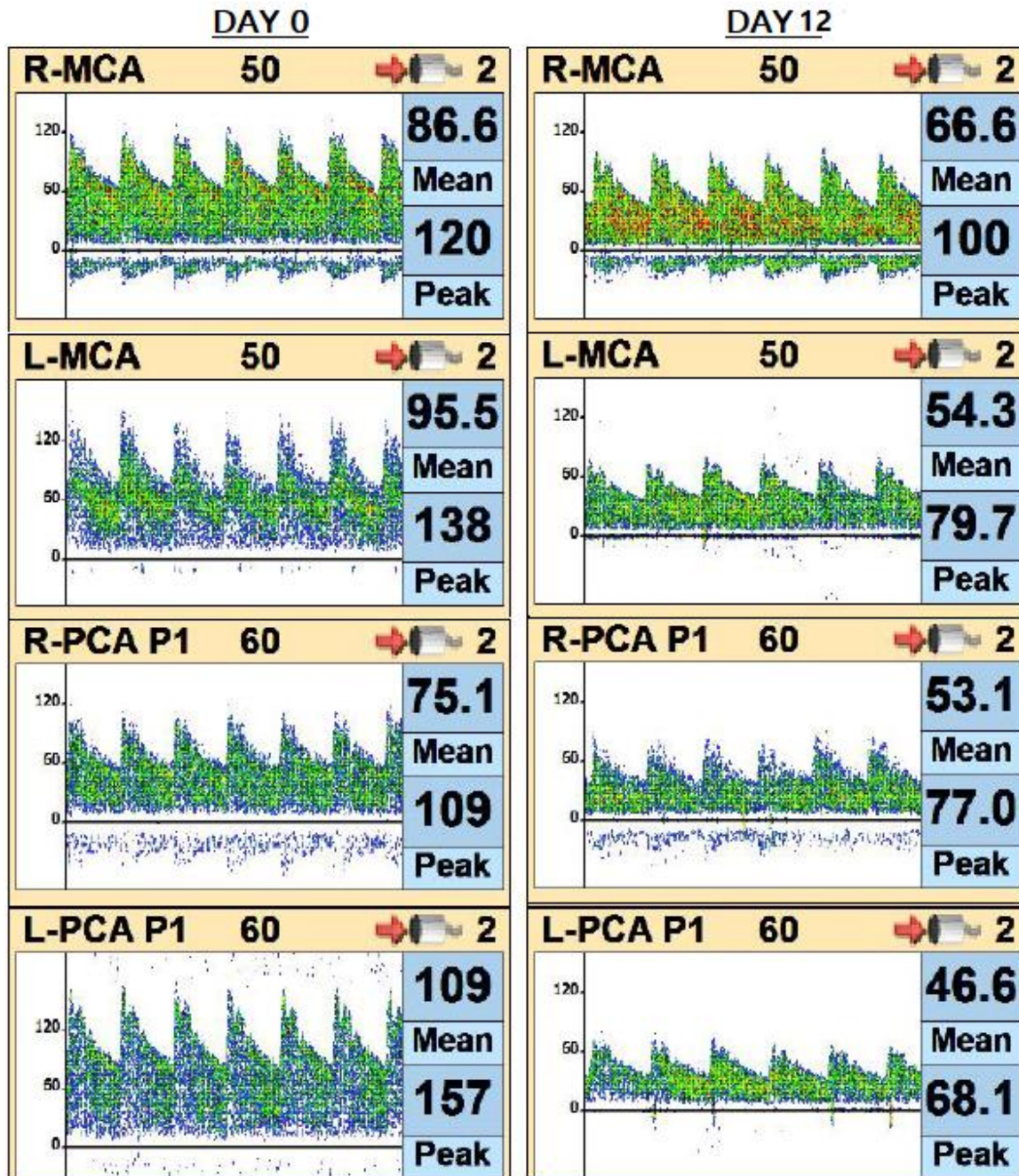
### 3. DISCUSSION

TCD is an inexpensive and sensitive measure of intracranial blood flow velocities, which helps in identifying areas of arterial stenosis. In addition, it also helps in monitoring the management of raised intracranial pressures (ICP) in TBM patients. Tai et. al. correlated TCD findings in 36 patients of TBM with Computed Tomography or Magnetic Resonance angiography (CTA/ MRA). 80% of patients having TCD criteria for vasculopathy, showed focal narrowing of major intracranial arteries on either CTA/MRA.<sup>6</sup> Van-Toorn et. al. used TCD to study blood flow velocities and found high velocities in basal cerebral arteries in 14 children diagnosed with TBM.<sup>7</sup> These high values persisted for 7 days suggesting the cause

of elevated velocities to be vasculitis rather than functional vasospasm. Further, in children with non-communicating hydrocephalus, a decline in PI values was noted post CSF diversion procedure.

However, similar results were not seen in children with communicating hydrocephalus, who showed a poor correlation between PI values and ICP. In another study, children having TBM with hydrocephalus with vasculopathy and cerebral infarcts, CSF diversion procedures failed to decrease the PI values. Post-operative PI values in these patients were significantly higher as compared to children having TBM with hydrocephalus but without infarcts.<sup>8</sup>





**Figure 2:** Transcranial doppler findings on Day 0 (before starting treatment) and Day 12 (after 12 days of therapy with ATT and steroids).

In our patient, initial TCD assessment at the time of presentation showed high peak systolic and mean flow velocities in the insonated intracranial vessels, which correlated with dense basal exudates and pachymeningeal enhancement on MR imaging. Based on the criteria proposed by various authors,<sup>6,9-11</sup> TCD values indicated Left PCA vasculopathy (MFV > 85 cm/sec) with >50% stenosis (PSV > 145 cm/sec) and <50% narrowing in right PCA (PSV 100-145 cm/sec). After treatment with steroids, the basal exudates settled as shown in repeat MRI, and TCD showed reduced peak and

mean flow velocities, signifying reduced obstruction to blood flow in the tested intracranial vessels.

To conclude, patients with TBM-vasculopathy develop cerebral hemodynamic alterations due to inflammatory lesions in the subarachnoid space leading to steno-occlusive disease of the cerebral vessels. This can be assessed via a bedside Transcranial doppler, which helps in identifying vascular narrowing, diagnosing vasculopathy and monitoring the response to therapy.

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