Tubercular Meningitis and Cortical Venous Thrombosis: A Rare but Potentially Lethal Combination - A Case Report and Literature Review

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INTRODUCTION:

Tubercular meningitis is a severe form of meningitis caused by tuberculosis bacteria. It is a rare condition, but in India, it is a significant public health concern, as the country has a high incidence of tuberculosis. This disease affects the protective layers surrounding the brain and spinal cord, leading to inflammation and the formation of blood clots in the veins of the brain. These clots can lead to a condition called cortical venous thrombosis, which can be life-threatening. In this case, an 18-year-old man in India has been experiencing symptoms of tubercular meningitis complicated by cortical venous thrombosis. He has been suffering from a severe headache, fever, and neck stiffness, which are typical symptoms of meningitis, and are signs of confusion and disorientation.

We immediately started treatment for this patient’s condition, which included a combination of antibiotics to fight the tuberculosis bacteria and anticoagulant medication to prevent further blood clots from forming. We also monitored his intracranial pressure to prevent any further complications. These symptoms are concerning as they indicate a worsening of his condition, and we closely monitored him to prevent any further complications.

In India, tuberculosis is a significant public health concern, and the country has implemented several programs to control the spread of the disease like the National Tuberculosis elimination program. These include early detection, treatment, and prevention strategies such as vaccination and contact tracing.

CASE PRESENTATION:

An 18-year-old male presented to the emergency department with a history of fever and headache for the past 1 month. The headache was moderate to severe in intensity, generalized, and not relieved by analgesics. He also had episodes of vomiting for the last two days. There was no history of seizures or focal neurological deficits. There was no significant medical or family history.

On examination, the patient was conscious and oriented to time, place, and person. His vital signs were stable. Neurological examination revealed neck stiffness and positive Kernig’s sign. The rest of the examination was unremarkable.

A diagnostic workup revealed a low hemoglobin level of 10.6 g/dl, a high ESR level of 98 mm/hour, and a CRP level of 45 mg/L. MRI of the brain showed diffuse leptomeningeal enhancement with evidence of cortical venous thrombosis in the right cerebral hemisphere. Cerebrospinal fluid analysis showed a high
protein level of 180 mg/dl, a low glucose level of 22 mg/dl, and lymphocytic pleocytosis with a total of 600 cells/mm3, including 70% lymphocytes. The chest X-ray showed Hilus tightening classical of Tuberculosis (Figure 1) and computed tomography (CT) scan shows bilateral multiple granuloma in the upper air field (Figure2). The HIV serology was non-reactive. The Cartridge-based nucleic acid amplification (CBNAAT) was positive for Mycobacterium Tuberculosis. The patient was admitted with a provisional diagnosis of tubercular meningitis and started on empirical antituberculous therapy, consisting of isoniazid, rifampicin, pyrazinamide, and ethambutol. He was also started on dexamethasone and antiemetics.

On the third day of admission, the patient developed a worsening headache and altered sensorium. A repeat neurological examination showed a Glasgow Coma Scale (GCS) score of 11/15, with no focal neurological deficits. Magnetic resonance venography (MRV) was done, which showed diffuse meningeal enhancement and thrombosis of the superior sagittal sinus and bilateral ethmoid sinus. (Figure 3). and section filling defect of Superior sagittal sinus is seen with bilateral sigmoid sinus suggestive of cortical venous thrombosis (CVT). (Figure 4). The patient was diagnosed with tubercular meningitis complicated by cortical venous thrombosis. The antituberculous therapy was continued, and he was started on anticoagulation therapy with low molecular weight heparin (LMWH). The dexamethasone dose was increased, and the antiemetic regimen was modified.

Over the next few days, the patient’s symptoms improved after 2 weeks of therapy. His headaches subsided, and the fever abated. The visual acuity in the right eye improved, and the power in the right upper and lower limbs increased to 4/5. The repeat MRI of the brain showed a significant reduction in the size of the thrombus and an improvement in the leptomeningeal enhancement. However, the patient developed complications during his hospital stay. He developed acute kidney injury due to hypotension, which required hemodialysis.

DISCUSSION:

Tubercular meningitis (TBM) is a severe form of meningitis caused by Mycobacterium tuberculosis that affects the membranes surrounding the brain and spinal cord. It is a common form of central nervous system (CNS) tuberculosis and has a high mortality rate. Cortical venous thrombosis (CVT) is a rare but severe complication of TBM that can lead to further neurological damage and death. This case report discusses an 18-year-old man in India who presented with symptoms of TBM and was later diagnosed with CVT.

The patient was started on standard antitubercular therapy of Rifampin, Isoniazid, Pyrazinamide, Ethambutol, and supportive care. Including intravenous fluids and antipyretics. However, his clinical condition continued to deteriorate, and he developed focal neurological deficits. A magnetic resonance imaging (MRI) scan of the brain was performed, which revealed the presence of multiple infarcts in the right cerebral hemisphere and thrombosis of the superior sagittal sinus, and bilateral sigmoid sinus consistent with CVT.

The patient was discharged from the hospital after two weeks and was advised to continue antitubercular therapy and oral anticoagulant therapy for six months.

The pathogenesis of CVT in TBM is thought to be due to a combination of factors, including direct invasion of the cerebral veins by the Mycobacterium tuberculosis, the inflammatory response to the infection, and a procoagulant state induced by the infection.

A review of literature showed the only 6 cases of cortical venous thrombosis which was due to tubercular meningitis and no evidence of TB anywhere else which is summed up in Table 1. Out of them except one all of them had fever and headache, and meningeal enhancement and infarcts on MRI of the brain. MRI is the imaging modality of choice, as it can detect both thrombosis and infarcts associated with CVT. The treatment of CVT in TBM is based on anticoagulant therapy, which is known to improve outcomes. However, the optimal duration of anticoagulant therapy in these patients is not well defined.

The timely diagnosis and prompt initiation of antitubercular therapy are essential for improving outcomes. CVT is a rare but severe complication of TBM that requires a high index of suspicion for early diagnosis. Anticoagulant therapy is the treatment of choice for CVT in TBM, and the duration of therapy should be
individualized based on the patient’s clinical course and imaging findings. Further studies are needed to define the optimal management strategies for patients with TBM complicated by CVT.

REFERENCES:


Table 1: Data showing previously reported cases of cortical venous thrombosis complicating Tuberculous meningitis with MRI+MRV findings and presenting complaints.6-11

<table>
<thead>
<tr>
<th>S. No</th>
<th>Author name/Year</th>
<th>Age/sex</th>
<th>Presenting complaints</th>
<th>Radiological findings on MRI + MRV</th>
<th>Outcome</th>
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<tbody>
<tr>
<td>1.6</td>
<td>Elkeslassy et al. 1997</td>
<td>48 year/female</td>
<td>Fever, headache – 2 months Drowsiness – 10 days</td>
<td>Superficial cortical venous thrombosis</td>
<td>Survived</td>
</tr>
<tr>
<td>S. No</td>
<td>Author name/Year</td>
<td>Age/sex</td>
<td>Presenting complaints</td>
<td>Radiological findings on MRI + MRV</td>
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<td>2.7</td>
<td>Ranjith et al/2015</td>
<td>1 month/ female</td>
<td>Fever – 1-month Headache, drowsiness – 8 days Seizures – 1 day</td>
<td>Vasculitic infarcts; bilateral moya-moya pattern of arteriopathy; thrombosis of the left transverse sinus.</td>
<td>Survived</td>
</tr>
<tr>
<td>3.8</td>
<td>Finsterer et al/2000</td>
<td>61 year/ male</td>
<td>Fever, headache, multiple cranial nerve palsies – 1-month</td>
<td>Superior sagittal sinus thrombosis; the patient had disseminated tuberculosis (TBM + Pulmonary)</td>
<td>Survived</td>
</tr>
<tr>
<td>4.9</td>
<td>Ramdasi et al/2015</td>
<td>20 year/ female</td>
<td>Fever, headache, vomiting – 4 days</td>
<td>Thrombosis of the left transverse and sigmoid sinus</td>
<td>Died</td>
</tr>
<tr>
<td>5.10</td>
<td>Verma et al/2013</td>
<td>26 years/ Male</td>
<td>Fever – 4 months Headache – 1-month Diplopia – 15 days Altered sensorium with seizures – 2 days</td>
<td>Non communicating hydrocephalus with leptomeningeal enhancement. Superior and inferior sagittal sinus narrowing with increased collaterals.</td>
<td>Survived</td>
</tr>
<tr>
<td>6.11</td>
<td>Guenifi et al/2016</td>
<td>Case series: 3 women (33,31,31 years)</td>
<td>Different for all</td>
<td>Sagittal sinus (2 cases), lateral sinus (2 cases) and transverse sinus (1 case) thrombosis</td>
<td>All survived</td>
</tr>
<tr>
<td>7</td>
<td>Present case</td>
<td>18 years/Male</td>
<td>Fever, Headache – 1 month Altered sensorium- 3 days</td>
<td>Superior sagittal sinus, Bilateral sigmoid sinus</td>
<td>Survived</td>
</tr>
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</table>

Figure Legends
**Figure 1:** X-ray of the chest PA view showing hilus tightening

**Figure 2:** CT Scan of the lungs consistent with Tuberculosis and showing Bilateral chest infiltrates in the Upper lung fields

**Figure 3:** MR venography showing filling defects of Superior Sagittal sinus (Blue arrows) in the Sagittal section.

**Figure 4:** MR venography in coronal section filling defect of Superior sagittal sinus is seen with bilateral sigmoid sinus suggestive of cortical venous thrombosis (CVT).

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