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## Case Report

### Cortical venous thrombosis – a rare complication of tuberculous meningitis

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**ABSTRACT:** Occlusion of the central veins and sinuses occurs owing to thrombus, thrombophlebitis, or tumours. Cerebral venous thrombosis (CVT) is less frequent than arterial thrombosis, but can produce a cascade of sequelae and may be fatal. The usual predisposing factors for CVT include infections, pregnancy and puerperium, hypercoagulable states, etc. Many infectious causes are related to CVT, but a review of literature showed only few cases related to tuberculosis. Vascular changes consisting of arterial narrowing with or without occlusion are frequently seen at autopsy in cases of tuberculous meningitis. We report one such case of tuberculous meningitis where the patient developed cortical venous thrombosis after 5 days of illness. She was treated empirically, initially, till confirmation of the diagnosis and later was put on antitubercular drugs along with prednisolone therapy and anticoagulation, which led to complete recovery. Thus, if treated aggressively and in time, tuberculous meningitis and its complications are completely curable.

**KEY WORDS:** *Tuberculous meningitis; Cortical venous thrombosis; Magnetic resonance venogram; Rare complication of TBM*

#### INTRODUCTION

Cerebral venous thrombosis (CVT) i.e. thrombosis of the intracranial veins and sinuses is a rare type of cerebrovascular disease that affects about 5 people per million and accounts for 0.5% of all strokes. Several disorders can cause, or predispose patients to CVT and these include all medical, surgical, and gynecological causes of deep vein thrombosis in the legs, genetic and acquired prothrombotic disorders, cancer, haematological diseases, vasculitis and other inflammatory systemic disorders, pregnancy and puerperium, infections, as well as several local causes such as brain tumours, arteriovenous malformations, head trauma, CNS infections, and infections of the ear, sinus, mouth, face, or neck.<sup>1-3</sup> The current gold standard to diagnose CVT is the combination of MRI to visualise the thrombosed vessel and magnetic resonance venography to detect the non-

visualisation of the same vessel.<sup>4-6</sup> The diagnosis of tuberculous meningitis is based on CSF picture supported by polymerase chain reaction.

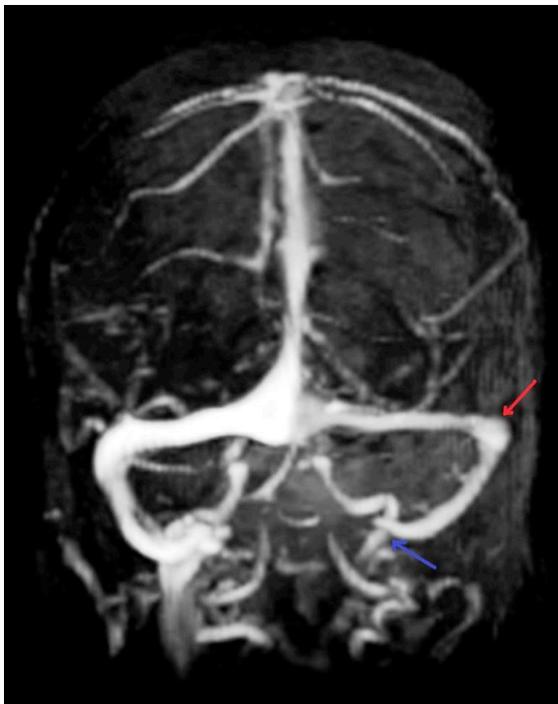
#### CASE DETAILS

A 36-year-old Hindu female patient was admitted to our hospital with complaints of high-grade continuous fever with generalized headache (throbbing type) along with projectile vomiting 2-3 episodes/day for 1 day prior to admission to the hospital. The patient was a healthy individual with no addictions. Family and social history were not contributory. She had mixed dietary habits. On admission, she was vitally stable except that she was febrile (Oral temperature = 101°F). On neurological examination, her higher functions were normal. She had ptosis on the right side along with severe retro-orbital pain. Tone, power and deep tendon reflexes were normal in all four limbs. Neck stiffness was present along with positive Kernig's sign. Plantar response was equivocal in both the lower limbs. She was started on standard empirical treatment of pyogenic meningitis viz. injection dexamethasone 24mg IV stat dose along with injectable ceftriaxone 2gm IV 12 hourly and

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injection vancomycin 1gm IV 12 hourly till the CSF report was awaited and lumbar puncture was done immediately after ruling out raised intracranial tension. She had no papilledema. Her CSF report was: TLC-120/mm<sup>3</sup>; polymorphs 34%, lymphocytes 66%, CSF protein 450mg/dl and CSF sugar 60mg/dl that favored the diagnosis of tuberculous meningitis.

MRI-Brain was done which showed meningeal enhancement after gadolinium contrast studies that were suggestive of meningitis. She was started on antitubercular treatment viz. Isoniazid, Rifampicin, Pyrazinamide along with Streptomycin and oral Prednisolone, and other supportive treatment. Meanwhile, the CSF TB-PCR report turned out to be positive which confirmed our diagnosis. While on treatment, after 3 days, the patient again started complaining of diplopia and headache and so keeping in mind the picture of CVT, Magnetic resonance venogram (**Figure 1**) was done which was suggestive of CVT in right sigmoid sinus and right-sided internal jugular vein. The patient was started on injectable enoxaparin for 5 days, which was then overlapped with warfarin from second day of starting enoxaparin. The patient became asymptomatic within the next 24 hours and was discharged in a haemodynamically stable condition and was advised to continue warfarin for 6 months with regular monitoring along with antitubercular treatment and tapering dose of prednisolone.



**Figure 1: Red-Arrow showing right sigmoid sinus thrombosis and Blue-Arrow showing thrombosis of right internal jugular vein**

## DISCUSSION

Tuberculous meningitis, i.e. meningitis due to infection with *Mycobacterium tuberculosis*, is more common in those from countries where tuberculosis is common, but is also encountered in those with immunosuppressed states such as HIV infection. Stroke-like manifestations consequent to cerebral venous thrombosis have also been observed with tuberculous meningitis<sup>7</sup>

Tuberculosis is associated with CVT in very few patients reported in the literature. Of the reported cases, two had disseminated TB with no involvement of central nervous system,<sup>8</sup> one had only pulmonary disease, and another had chronic granulomatous meningitis.<sup>9</sup> To our knowledge, there are only 4 reported cases of CVT secondary to tuberculosis<sup>7</sup> and this is the fifth such case reported till now.

*Mycobacterium tuberculosis* infects one third of the world population and kills almost 3 million people each year.<sup>10</sup> The pathophysiologic process to explain the relation between TB and CVT includes: (1) injury to endothelium, (2) alterations in normal blood flow, and (3) alterations in the blood coagulability. Blood stasis occurs because intracranial sinus is a low-pressure system without valves. Hypercoagulable state occurs in patients with TB, because they show increased platelet aggregability,<sup>11</sup> Sarode et al found significant hyper aggregation of platelets in 88% patients with intestinal TB. Endothelium injury in intracranial veins may be consequence of the same mechanism, which occurs in arterial vessels because of TB. Arteries running through the subarachnoid space may show obliterative endarteritis with inflammatory infiltrates in their walls, and marked intimal thickening. Another theory suggests that a transition G→A at position 20210 is associated with elevated prothrombin concentration and thrombosis. People with mutation in this gene and presence of factor V Leiden mutation may increase 5 - 10 fold risk of venous thrombosis.<sup>12</sup> However, both mutations when alone are not high risk factors for venous thrombosis, so lifetime anticoagulation is controversial.<sup>12</sup> In our case, it was still not possible to establish a connection between CVT and tuberculosis.

## CONCLUSION

Cerebral venous thrombosis is a rare disease that can occur at any age. Its occurrence in tuberculous meningitis is rare and so its clinical picture is routinely confused and falsely attributed to tuberculous meningitis. A very high index of suspicion is required to correctly diagnose the complication as its timely aggressive treatment results in an excellent outcome for the patient in the form of complete recovery.

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