



## Post Caesarean Sagittal Sinus Thrombosis after Spinal Anaesthesia: A Case Report

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**Abstract:** Central venous thrombosis, although rare, is a recognized cause of puerperium stroke. We present a case of successfully managed sagittal sinus thrombosis (SST) developed in a parturient after Caesarean delivery under spinal anaesthesia.

Cerebral venous sinus thrombosis is a rare disorder with an incidence of 3:1,000,000, and pregnancy, being a recognized hypercoagulable state, is known to increase the risk of CVST. The incidence of CVST, as a complication of pregnancy, is 1:10,000 to 1:20,000 deliveries during the third trimester of pregnancy and the immediate postpartum period<sup>1,2</sup>.

The association of CVST with pregnancy and puerperium remains a recognized cause of maternal mortality and morbidity in developing countries<sup>3,4</sup> and represents the main cause of puerperium stroke<sup>4,5</sup>. We report a case of sagittal sinus thrombosis occurring after spinal anaesthesia for caesarean section.

**Case Report:** A previously healthy 23 yr old, ASA I, gravida 1, para 1 parturient was admitted to the hospital at 37 weeks gestation for gestational hypertension associated with uterine contractions. Her past medical history was not significant. Before pregnancy she was taking no medication other than oral contraceptives. During pregnancy the patient suffered from a pyelonephritis and presented with an episode of threatened preterm labour at 32 weeks gestation. Initial physical examination revealed cervical effacement. Her blood pressure was 140/90 mmHg and urinalysis showed 2+ proteinuria. The patient was diagnosed as a case of mild pre-eclampsia. Routine blood tests were within normal limits.

She presented with non progression of labour with foetal distress. The patient did not receive any antihypertensive medication for her progressive hypertension. Considering the non progression of labour, spinal anaesthesia was planned for emergent caesarean section. Under aseptic conditions technique was performed successfully on the first attempt with a 25G pencil point spinal needle (Top spinal needle, Malaysia) was introduced into the L3–L4 interspace and 2.0 mL of 0.5% hyperbaric bupivacaine mixed with 10 µg fentanyl was administered. There was no hemodynamic compromise and the patient delivered a healthy female infant with apgar scores of 7/9/9.

Surgery was performed uneventfully within one hour. In the evening, the patient complained of an intense headache, altered sensorium, associated with nausea and vomiting and she developed two generalized grand mal seizures. Convulsions stopped after administration of thiopentone 50 mg, fosphenytoin 250 mg and valproic acid 400 mg iv and the patient was intubated transferred to the intensive care unit kept on mechanical ventilation electively for 4 hours. Second day morning, the patient was weaned and extubated as she found to be afebrile, normotensive and had no abnormal neurological signs. However, on the third postpartum day, she developed paresthesia, progressing from the left hand to the face over the distribution of the facial nerve, associated with acute left hemiparesis, blurred vision, and somnolence. Fundoscopic examination revealed mild bilateral papilledema.

Brain computed tomography (CT) after contrast injection, revealed bilateral fluid filled lateral ventricles mild hyperintensity on CSF in basal cistern on T2(flair) and T1W1 that represent mild intraventricular bleed and subarachnoid haemorrhage. Brain magnetic resonance imaging (MRI-venography) showed thrombosis in anterior half of superior sagittal sinus with prominent adjacent cortical veins these findings (Images).

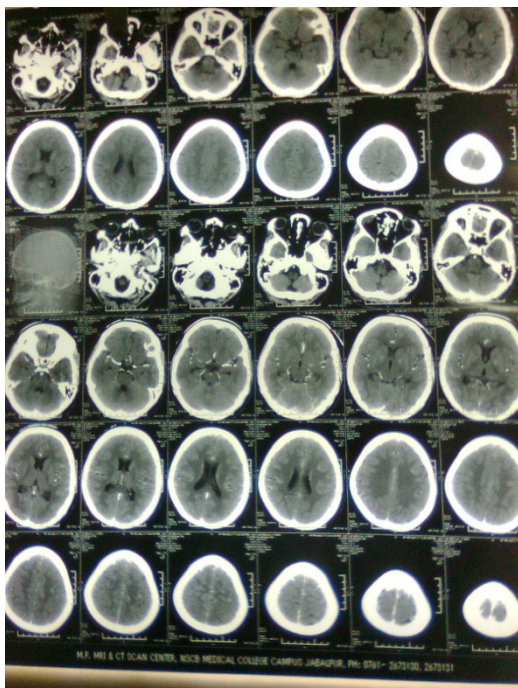


Figure 1: CT Head

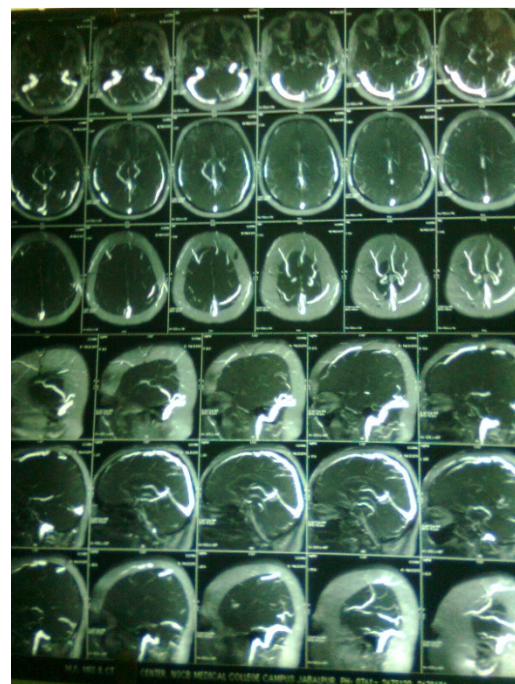


Figure 2: MRI Head

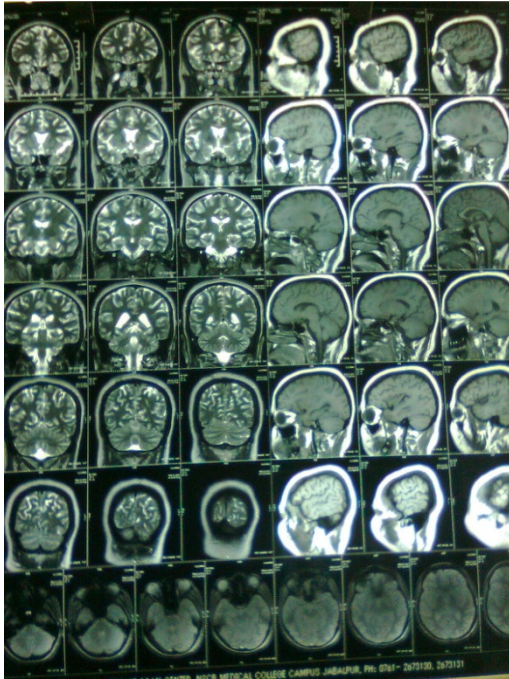


Figure 3: MRI Image 2

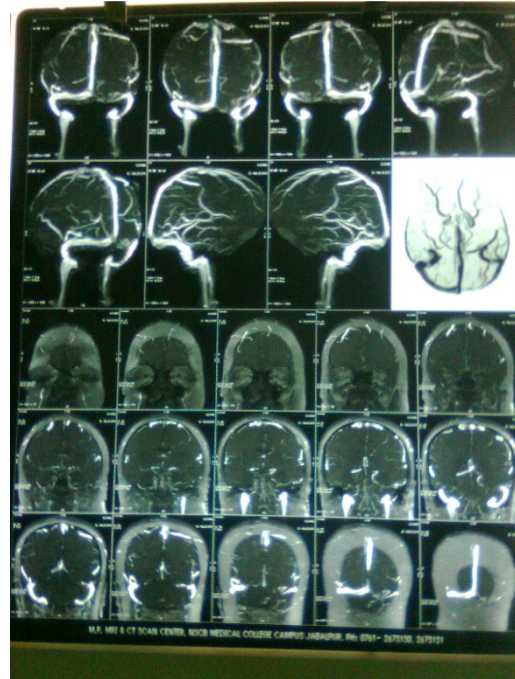


Figure 4: MRV Image

Treatment consisted of anticoagulation initiated with inj. heparin (500 IU/kg/day) iv and maintained with low molecular weight heparin (enoxaparin). Mannitol, fosphenytoin (fosolin), levcitarem (levipril), paracetamol, ranitidine, furosemide, dexamethasone and aravone were also included in the treatment. During the next three days, the patient's arterial blood pressure varied between 130/60 mmHg and 140/90 mmHg.

Initial investigations were normal platelet counts and bleeding time (1.5min), clotting time (4.5min), prothombin time (16 sec) but raised FDP level (1600 ng/ml) and D-dimer level (2.5). Neither hyperhomocysteinemia nor any anticardiolipin and antiphospholipid antibodies were detected.

On day 6<sup>th</sup> postpartum, tablet folic acid (folvite) and tablet nicoumalone (acitrom) 1mg OD were also added after neurophysician opinion. Patient allowed starting oral feeds 10<sup>th</sup> day onwards. Complete motor recovery was gained on day 14<sup>th</sup> and on next day 15<sup>th</sup> patient was discharged without any neurological deficit. The patient was advised to avoid oral contraceptives and continue her antiepileptic medications and oral anticoagulants until the three-month follow-up visit.

**Discussion:** This report highlights the diagnostic challenge and management of a central venous sinus thrombosis (CVST) presenting in the postpartum period. Following an uneventful delivery facilitated with spinal analgesia, the patient experienced on evening postpartum the gradual onset of an atypical headache with focal neurological signs including left arm and face paresthesia, acute left hemiparesis, blurred vision, altered consciousness, and generalized grand mal seizures.



In the absence of seizures or other neurological signs in the puerperium, it is difficult to distinguish CVST-induced headache from spinal headache associated with regional anaesthesia. The diagnosis of sagittal sinus thrombosis was made by neuroimaging. Magnetic resonance imaging and magnetic resonance angiography are considered the most sensitive diagnostic tests, disclosing the diagnosis in 90% of cases.

Treatment with heparin must begin immediately upon confirmation of the diagnosis, even in the presence of a hemorrhagic infarct<sup>1,6</sup>. Intravenous anticoagulation should be continued until remission of the acute stage of the disease, i.e., normal consciousness, improvement of headache and resolution of focal neurological deficits. Subsequent therapy should be converted from parenteral to oral anticoagulation for a period of three to six months. Patients with hereditary thrombophilia should be treated for a longer period (6–12 months).

Women who have suffered from a CVST while taking oral contraceptives should be counselled about alternative methods of contraception<sup>1</sup>. Long-term follow-up of patients who have experienced pregnancy-related CVST is important, since recurrence, although rare, is possible within the first 12 months<sup>1</sup>. Subsequent pregnancy is not contraindicated, although prophylactic low-dose anticoagulation should be considered, while recognizing that the risk-benefits of this therapy during pregnancy have not been established. Counselling about symptoms suggestive of CVST recurrence and neurological surveillance during pregnancy is strongly recommended. Regional anaesthesia or analgesia for labour or Caesarean delivery in women with a history of CVST history is not contraindicated, while prophylactic anticoagulation is recommended in the postpartum period<sup>1</sup>.

While the pathogenesis of puerperal CVST has not been clearly elucidated, it may be related to three aspects characterizing Virchow's classic triad: 1) stasis of intracerebral blood flow (especially in the valveless sagittal sinus); 2) vascular endothelial damage due to fluctuations in intracranial pressure (especially during delivery); and 3) the hypercoagulable state associated with dehydration and physiologic anaemia of pregnancy<sup>7</sup>.

Specific blood coagulation disorders such as antithrombin III deficiency, protein C, or protein S have also been identified as a particularly important origin of CVST; however, these tests must be carefully interpreted in pregnancy<sup>3</sup>. Our patient's finding of a low functional protein S at 50% (normal range 60–115%) can be a spurious one in pregnancy. It has been shown that free protein S levels fall significantly between the first and second trimester of normal pregnancy, without significant change in function. Therefore, we need to postpone protein S evaluation for hypercoagulability until at least six weeks postpartum<sup>8</sup>.

The complex aetiology may be associated with the following factors: hereditary thrombophilia, anticardiolipin antibodies, hyperhomocysteinemia, Caesarean delivery, pregnancy-related hypertension, and the use of oral contraceptives which may present a risk factor in 10% of cases<sup>1,6</sup>. Common inherited dispositions of thrombophilia include mainly the factor V Leiden mutation (15–17% of cases) and the prothrombin-gene-mutation 20210GA (10–12% of cases), whereas antithrombin III, protein C- and protein S-deficiency are found in only 2–6% of



cases<sup>1</sup>. In this patient, preeclampsia, decrease in protein S (even though commonly observed during pregnancy), and the use of oral contraceptives, were possible CVST-triggering factors<sup>8</sup>.

Implications of spinal anaesthesia in the pathogenesis of this entity remain controversial. Suggested mechanisms included chemical arachnoiditis and changes in intracranial pressure. Direct implications of the anaesthetic technique were considered doubtful<sup>9</sup>.

In our case, considering the rapid progression of labour, a spinal analgesia technique was chosen and performed uneventfully. No significant hemodynamic disorders were observed during delivery but after anaesthesia. Furthermore, the postpartum headache was atypical, with an unclear postural character. Therefore, leakage of CSF and important changes in intracranial pressure seem unlikely contributing factors. However, a cause effect association is difficult to demonstrate in rare events such as CVST concomitant with spinal anaesthesia, and the question is still under debate<sup>10</sup>.

The patient should remain under clinical follow-up and the diagnosis of CVST has to be considered. Early intervention with systemic heparinization is critical when this diagnosis is confirmed.

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