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Case Report Open Access

Cerebral Venous Thrombosis: A Complicated Anaesthetic Scenario for Caesarean Section

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Abstract

Cerebral venous thrombosis is a rare cerebrovascular disease and causes 0.5% of all strokes. Headache is one of the usual symptoms along with nausea, vomiting and seizures which are lesser common. Focal neurological deficits may also occur. Both genetic and acquired conditions may predispose to cerebral venous thrombosis. Management of a caesarean section in this situation poses significant anaesthetic challenges in view of anticoagulation, haemodynamic stability, and neurological outcomes. We present a case of a 30 year old lady who was primigravida associated with cerebral venous thrombosis posted for caesarean section.

Keywords: Cerebral venous thrombosis; Spinal anaesthesia; Casarean section

Introduction

Cerebral venous thrombosis (CVT) is an uncommon cerebrovascular disease that can occur at any age and is responsible for 0.5% of all strokes [1]. Isolated headache without focal neurological findings or papilledema occurs in approximately 25% of patients with CVT which leads to confusion in its clinical diagnosis [2]. Focal or generalized seizures can also occur in these patients [3]. Comorbid conditions (eg: thrombophilias, inflammatory bowel disease), transient physiological changes (egpregnancy, dehydration), medications such as oral contraceptives, substance abuse, and events such as head trauma are some predisposing conditions in addition to Virchow's triad [3]. Both genetic and acquired prothrombotic conditions can contribute to CVT [4].

We aimed to find out the safety of spinal anaesthesia as an alternative to general anaesthesia for caesarean section in haemodynamically stable patients with timely recognized cerebral venous thrombosis.

Case Report

A primigravida of 30 years old at 37 weeks of gestation was admitted to our hospital for elective caesarean section (CS). Prior exposure to anaesthesia was a diagnostic laparoscopy for endometriosis two years earlier. She had regular antenatal check-ups during the first two trimesters which were uneventful. However in the third trimester she complained of headache which was continuous in nature, unilateral and lasted for five to six days. She had no complaints of altered consciousness, altered vision, nausea, vomiting or seizures. As per advice by the neurologist she underwent an MRI which showed a right transverse sinus and superior sagittal sinus thrombosis (Figures 1 and 2).



Figure 1: (MRI Transverse)-MRI Angiogram in transverse plane showing right transverse sinus thrombosis.

Accordingly she was started on enoxaparin (low molecular weight heparin) (LMWH) 60 mg subcutaneous (SC) twice daily. Three days prior to CS, enoxaparin was changed to unfractionated heparin 5000 units SC twice daily.

On the day of surgery after confirming her fasting status and laboratory investigations (Hb-12.4 g%, Plt-252000 c/mm³, TLC-8000 c/mm³, APTT-22.9 seconds, INR-1.24) she was shifted into the operating room. Standard ASA monitoring was done along with real time arterial blood pressure monitoring. Spinal anaesthesia was given using 10 mg of hyperbaric bupivacaine via a 26 G Quinke Babcock needle. Vitals were monitored throughout the procedure which was stable.

A healthy male baby was born with an Apgar score of 9/10. The surgery lasted approximately one hour and was uneventful. Post-surgery strict monitoring for any neurological signs like headache, change in consciousness level was done. However there were no such complaints. Enoxaparin was restarted 24 hours after surgery.



Figure 2: (MRI Saggital)-MRI Angiogram in sagittal plane showing superior sagittal sinus thrombosis.

Discussion

The association of CVT with pregnancy and puerperium is known [5]. This complication is also common in India, with a prevalence of 4.5/1000 obstetric admissions [6]. The thrombus, once formed, enlarges and may cause venous congestion leading to increase in intracranial pressure. If left untreated, it may cause ischemia [7].

Cerebral venous thrombosis can cause increased venous and capillary pressure. This can decrease cerebral perfusion and may cause cytotoxic oedema and ischaemic injury. Vasogenic edema may result due to disruption of the blood-brain barrier, and capillary rupture causes parenchymal haemorrhage [8].

Cerebral venous thrombosis may also cause decrease in cerebrospinal fluid absorption, and cause an increase in the ICT. Consequently, this may cause cytotoxic oedema and ischaemic vascular injury and cause pulmonary haemorrhage [8].

The transverse sinus is most commonly affected followed by superior sagittal sinus and straight sinus [9]. The thrombosis occurs in more than one site and often affects cortical, jugular and internal cerebral vein [9]. Headache, focal deficits, seizures, loss of consciousness may be presenting symptoms. Raised intracranial pressure (ICP) may cause cranial nerve palsies too.

The STROKE journal suggests that for patients with CVT, initial anticoagulation with adjusted-dose UFH (unfractionated heparin) or weight-based LMWH is reasonable, followed by vitamin K antagonists. Our patient also received LMWH.

However one must keep in mind that anticoagulation is always associated with an increased risk for extracranial bleeding complications [10].

CVT patients have usually been managed by general anaesthesia (GA). But GA itself carries a risk as it may lead to raised ICP due to laryngoscopy and positive pressure ventilation may cause a decrease in cerebral venous drainage [11].

Large volumes of drug injected into the epidural space for epidural anaesthesia can also increase ICT [11]. Although CVT is a known complication of spinal anaesthesia, if dehydration and hypotension is prevented this can be avoided. Considering that our patient was haemodynamically stable, her coagulation profile within normal limits and with no features suggestive of raised ICP, we decided to give spinal anaesthesia.

Additionally, the patient would be awake for any neurological evaluation and to experience child birth.

Conclusion

Spinal anaesthesia is a safe alternative to GA for CS in patients having stable hemodynamic and neurological parameters with timely recognised CVT and timely cessation of anticoagulants.

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