Low-dose spinal anaesthesia for a parturient with Takayasu's arteritis undergoing emergency caesarean section

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ABSTRACT

Takayasu's arteritis is a rare form of nonspecific obliterative panarteritis of unknown aetiology. Anaesthesia for patients with Takayasu's arteritis is complicated by severe uncontrolled hypertension leading to end-organ dysfunction, stenosis of major blood vessels affecting regional circulation, and difficulties in the monitoring of arterial blood pressure. The anaesthetic approach for parturients with Takayasu's arteritis has not been standardised in the literature, and previous reports have documented the use of general as well as regional anaesthesia. There are few instances in the literature where low-dose spinal anaesthesia alone is used in patients with Takayasu's arteritis undergoing emergency caesarean section. We present a case of the successful management of a parturient with Takayasu's arteritis, who underwent an emergency caesarean section under low-dose spinal anaesthesia.

Keywords: cerebral blood flow, invasive blood pressure, lower segment caesarean section, subarachnoid block, Takayasu's arteritis

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INTRODUCTION

Takayasu's arteritis (TA) is a rare form of nonspecific obliterative panarteritis of unknown aetiology. It affects the aorta and its branches, leading to stenosis, thrombosis and the formation of aneurysms. It predominantly affects women of reproductive age. The anaesthetic approach for parturients with TA has not been standardised in the literature, and the use of general as well as regional anaesthesia has been previously reported.[1-6] There are also few reports on the use of low-dose spinal anaesthesia alone in patients with TA who are undergoing an emergency caesarean section. This case report describes the successful management of a parturient undergoing emergency caesarean section under low-dose spinal anaesthesia.

CASE REPORT

A 21-year-old primigravida (weight 48 kg, height 152 cm), a known case of TA, was scheduled for emergency lower segment caesarean section (LSCS) due to non-progress of labour, oligohydramnios and a poor biophysical profile.

The patient had a one-year history of nonspecific headache, neck pain during swallowing and pain on bending forward. She was a known hypertensive for the same duration, and was controlled on oral amlodepine 10 mg, twice daily. During her first antenatal check-up, she was diagnosed with TA, for which oral prednisolone 10 mg, twice daily, was started. For the optimisation of blood pressure, oral methyldopa 250 mg, twice daily, was started in addition to amlodepine. There was no history of dyspnoea or chest pain on exertion, and the exercise tolerance of the patient was good.

Pre-anaesthetic examination revealed that the patient was alert, oriented and cooperative. Physical examination revealed weak bilateral radial, ulnar, brachial, axillary and carotid artery pulsations. The patient's heart rate was 92 beats/min and the non-invasive blood pressure (NIBP) in both her upper limbs was 180/80 mm Hg (mean arterial pressure [MAP] 113 mmHg). All peripheral pulsations were normally felt in both the lower limbs; however, the NIBP measured 200/90 mmHg (MAP 126 mmHg). The patient's respiratory and neurological examinations showed normal results.

Preoperative investigations of the patient, including a haemogram, renal and liver function tests, coagulation profile and ultrasonography of the kidney-ureter-bladder, were within normal limits. Electrocardiogram (ECG) revealed left ventricular hypertrophy, and carotid Doppler revealed diffuse intimal thickening of the bilateral common carotid arteries, along with fusiform dilatation of the brachiocephalic trunk.

In the operating room, standard monitors were attached to the patient, including a five-lead ECG, an NIBP cuff over the left thigh and a pulse oximeter to the toe. Intravenous access was secured with a 16-G cannula,

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and a right dorsalis pedis arterial line was inserted under local anaesthesia.

The surgery was planned under the subarachnoid block (SAB). Preloading (20 ml/kg) was done using Ringer's lactate solution, and 100 mg hydrocortisone was administered intravenously. Under full aseptic precautions, SAB was administered with 6.5 mg hyperbaric bupivacaine along with 25 µg fentanyl at lumbar 4–5 interspace, using a 25-G spinal (sprotte) needle. While positioning (left lateral) for SAB, flexion of the neck was avoided. The patient was then placed in the supine position and oxygen (5 l/min) was administered via a face mask. A small pillow was placed under the patient's head, and left lateral tilt was maintained. Surgery was only commenced after the achievement of T6 dermatomal sensory block.

After the administration of SAB, a transient fall in the patient's blood pressure (128/78 mmHg [MAP 94 mmHg]) was treated by increasing the rate of administration of crystalloid and colloid, and by raising the foot end of the operation table. MAP was corrected to 100 mmHg. Following the delivery of the baby, oxytocin infusion was started (10 units in 500 ml of crystalloid at a rate of 8–10 drops/min). After about 4–5 minutes, the patient's blood pressure suddenly dropped to 118/72 mmHg (MAP 87 mmHg). An injection of 6 mg ephedrine was administered to the patient, and the table was turned to a 15° Trendelenberg position. The MAP was corrected to a value of above 100 mmHg immediately, as recorded by the continuous monitoring of invasive blood pressure (IBP), and the patient did not have any symptoms of poor cerebral perfusion, as evidenced by continuous verbal communications. No further fall of the MAP was noted, and the rest of the procedure was uneventful.

Postoperatively, analgesia was maintained with an injection of tramadol and paracetamol suppositories. Injections of 50 mg hydrocortisone every eight hours were administered intravenously for the first two postoperative days, followed by 10 mg oral prenisolone twice daily. The patient was uneventfully discharged along with a healthy baby on the tenth postoperative day.

DISCUSSION

TA is a form of granulomatous vasculitis of the aorta and its major branches. In 1908, Mikito Takayasu first described TA in a patient with a wreath-like coronary anastomosis of retinal vasculature surrounding the papilla.[7] The disease is prominent in women of childbearing age, with a preocclusive phase of rheumatic or systemic symptoms and segmental arterial involvement.[1]

Four types of patients with TA can be identified.[8] Type I involves the aortic arch and its main branches. Lesions in Type II are restricted to the descending thoracic and abdominal aortas. Patients with Type III show features of both Types I and II, and patients with Type IV show additional involvement of the pulmonary artery. Our patient was categorised as Type I, with involvement of the carotids and the brachiocephalic trunk.

Anaesthesia in patients with TA is complicated by severe uncontrolled hypertension leading to end-organ dysfunction, stenosis of major blood vessels affecting regional circulation, and difficulties in monitoring blood pressure. A major cause of hypertension is renovascular, but it can also result from an abnormal function of the carotid and aortic sinus baroreceptors and/or reduced elasticity, and a marked narrowing of the aorta and major arteries.[9]

The anaesthetic goal in patients with TA is the maintenance of blood pressure during the perioperative periods. There are some advantages and disadvantages of both general and regional anaesthesia. Tracheal intubation, extubation and inadequate depth under general anaesthesia result in considerable fluctuations in blood pressure that can lead to cerebral haemorrhage, rupture of aneurysms and cardiac dysfunction in patients with TA. Regional anaesthesia, on the other hand, can lead to hypotension, thus inducing cerebral ischaemia or infarction. However, regional anaesthesia in conscious patients is the easiest way to monitor cerebral function.[3-5] There are many case reports that describe successful surgery under general as well as regional anaesthesia in patients with TA.[1-6] However, there is a scarcity of literature reporting on the use of low-dose SAB alone for emergency caesarean section in patients with TA, which prompted this case report. A dose of 6.5 mg hyperbaric bupivacaine along with 25 µg fentanyl was administered, as larger doses of local anaesthetics are associated with higher levels of autonomic block and consequently, more severe hypotension. It is also a known fact that doses of intrathecal bupivacaine between 5 mg and 7 mg are sufficient to provide effective anaesthesia for caesarean sections.[10]

Our aim was to maintain the MAP in the range of 100–120 mmHg, so that there was less than 20% reduction from the preoperative values. As patients with TA may not tolerate acute decrease, preloading with 20 ml/kg of Ringer's lactate was performed. This is because

diffuse arteritis result in stenotic and non-compliant vessels, which interfere with compensatory mechanisms to increase blood pressure.[5]

A pillow was placed under the patient's head in order to prevent the extension of the neck, which might have reduced carotid blood flow by stretching the arteries, and a left lateral tilt was maintained to prevent aortocaval compression. The administration of epidural anaesthesia was avoided, as the procedure would have taken a longer time than SAB, and it was not possible to wait for a longer duration in view of the urgent nature of the surgery.

Electroencephalography monitoring, transcranial Doppler and continuous measurements of jugular venous oxygen saturation (JVO_2) have been used to monitor intraoperative cerebral haemodynamics, and these techniques have been found to be of clinical value. It has been observed that the mean velocities of cerebral blood flow (CBF) and JVO_2 start to decrease in proportion to the reduction in MAP.[11] It is also well known that the curve for autoregulation (CBF vs. MAP) shifts to the right for chronically hypertensive patients; that is, the lowest blood pressure at which autoregulation of CBF functions is higher in these patients than that in normotensive patients. Therefore, MAP is very important for maintaining a stable CBF and protecting the brain from any damage. It was not possible to perform any of specialised neurological monitoring for cerebral haemodynamics in our emergency operating room. Therefore, we aimed to maintain the MAP within a range of 100–120 mmHg. Although there were two episodes of transient hypotension, it was corrected almost instantaneously. This rapid correction was possible only because of the continuous monitoring of IBP, and all these efforts enabled recovery without any neurological deficits.

Accurate blood pressure monitoring, which is always a cause for concern for the anaesthetist, is even more crucial in patients with TA. There have been some controversies regarding the use of invasive arterial lines in patients with TA.[12,13] In our patient, the lower limb vessels were unaffected, and hence, we selected the dorsalis paedis artery for cannulation, without any adverse consequences. None of the previous reports have made a serious effort to standardise the monitoring of IBP in patients with TA. In our opinion, the monitoring of beat-to-beat variations in blood pressure in unaffected vessels helps in taking adequate proactive measures to correct the MAP intraoperatively. In situations where neurological monitoring is not possible, the monitoring of IBP should be made mandatory, as the MAP only guides the anaesthesiologist regarding the maintenance of cerebral perfusion.

In conclusion, the choice of the anaesthesia technique to be used should be tailored according to the presentation of patients with TA. SAB can be safely performed in patients with Type I TA, provided adequate preloading is done, a low dose of local anaesthetic is selected and MAP is maintained at the preoperative level.

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