CASE REPORT

Anesthetic management of Takayasu’s arteritis for cesarean section

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ABSTRACT

Takayasu’s arteritis (TA), also called pulseless disease, aortic arch syndrome, occlusive thromboaortopathy, or aortic arteritis, is a chronic vasculitis mainly involving the aorta and/or its main branches, such as the brachiocephalic, carotid, subclavian, vertebral, renal, coronary and pulmonary arteries. Major challenges for anesthesia in patients with TA involve severe uncontrolled hypertension, end-organ dysfunction, stenosis of major blood vessels, and difficulties in monitoring arterial blood pressure. The cardiovascular complications attributed to the disease can be seriously enhanced during pregnancy. We present successful anesthetic management of emergency cesarean section under general anesthesia in a parturient with long-standing Takayasu’s disease with renovascular hypertension complicated by eclampsia.

Key words: Takayasu’s disease; Arteritis; Hypertension; Anesthesia, General

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INTRODUCTION

Takayasu’s arteritis (TA) is a rare, inflammatory pan-endarteritis causing thrombosis occlusion and aneurysm of major arteries.1 Autoimmune mechanisms and infections like tuberculosis have been suggested as etiology of TA.2 Almost 80% are women in reproductive age.3 Pre-existing hypertension with TA may complicate the mother and fetus in the form of pre-eclampsia and intrauterine growth retardation respectively.

Various case-reports suggest the use of regional anesthesia for parturients with TA but there is limited literature on general anesthesia for cesarean section. We report successful outcome of an eclamptic parturient with type-III TA undergoing emergency cesarean section.

CASE REPORT

20-year-old, 150 cm, 37 weeks, primigravida came for emergency cesarean section with hypertension, giddiness and two recent episodes of convulsions treated with magnesium-sulphate.

Patient gave ten years past history of giddiness and claudication in right lower limb and left upper limb. Angiography revealed occlusion of right renal and both subclavian arteries with significant narrowing of descending thoracic aorta and right common carotid artery. Coronary angiography appeared normal. She was diagnosed with TA and underwent right renal angioplasty with descending thoracic aortoplasty two years back. She was started on regular methotrexate, prednisolone, atenolol and amlodipine. Patient had received treatment for pulmonary Koch’s six months back. Her hemogram, liver and renal functions, electrolytes, coagulation profile, ECG, 2D-echo and fundoscopy, all were within normal limits.

On examination, patient was delirious, and rowdy with facial puffiness. Her pulse was 84/min in left dorsalis pedis artery, blood pressure (BP) 160/100 mmHg in left popliteal artery and respiratory rate 24/min. Right carotid and right femoral arteries were feeble; bilateral radial and brachial arteries were not palpable. Bruit was heard on right carotid artery and subclavian arteries. Her muscle tone, and
superficial and deep tendon reflexes were normal. Ultrasonography recorded normal foetal heart rate of 140 beats/min.

In the operating room, standard monitoring was initiated with a five lead ECG, pulse oximeter on left toe and non-invasive BP by sphygmomanometer on left calf. Left lateral tilt was maintained throughout. Intravenous access was secured with an 18G cannula on the left forearm. We opted for general anesthesia (GA) considering patient’s neurological status. After pre-oxygenation, premedication with glycopyrrolate 0.2 mg and hydrocortisone 200 mg was given IV. Rapid-sequence induction was performed with thiopentone sodium 5 mg/kg followed by rocuronium 0.9 mg/kg to aid intubation. A pillow was placed below the head to avoid hyperextension of head during laryngoscopy. Patient was maintained on a combination of O2 and N2O (50-50%) and sevoflurane (0.6 to 0.8 MAC). Fentanyl was administered in graded doses (30+20+50 µg). After the delivery of baby, oxytocin infusion was started (10 U in 500 ml Ringer’s solution) at 8-10 drops/min. After five minutes of oxytocin infusion, the patient developed hypotension, whereas the systolic blood pressure dropped to 90 mmHg but responded to inj. ephedrine 6 mg. The total blood loss was around 800 ml and was replaced by crystalloid solutions. Patient was not reversed and extubated because of pre-operative convulsions, delirium and seizures aggravation by underlying TA (Type-III). Hence we chose to administer GA. To avoid reduction of blood flow due to stretching of carotid arteries, neck extension was prevented by placing a pillow beneath patient’s head. Aorto-caval compression was prevented by left lateral tilt. Nitroglycerin infusion was kept ready to attenuate pressor response during intubation thereby protecting against cerebral hemorrhage. Since the patient was on chronic corticosteroid therapy, additional pre-operative steroids were given to prevent Addisonian crisis.

Contrary to regional anesthesia which allows cerebral function monitoring in awake patient, GA mandates mean arterial pressure (MAP) monitoring as an indicator of stable cerebral blood flow. Clinical usefulness has been found with transcranial Doppler, jugular venous oxygen monitoring and electroencephalogram in detecting cerebral hypoperfusion in TA. We did not have neurological monitoring in our OR and therefore aimed to maintain the MAP within 100–120 mmHg. Invasive BP monitoring was not attempted because of the risk of arterial thrombosis following

DISCUSSION

TA is a chronic panarteritis involving the aorta and its main branches as well as coronary and pulmonary arteries, thereby progressing to vital organ ischemia. First described in 1908 by Japanese ophthalmologists, Mikito Takayasu and Onishi, TA is most commonly seen in oriental countries with an incidence of 2.6/million/year and a male:female ratio of 1:9. Its etiology is unknown, although its association with HLABw52 gene, autoimmunity, reproductive hormones and infections has been suggested. Isolated cases of TA co-existing with latent and active tuberculosis have been described, which was the probable cause of TA in our patient.

Four types of TA are identified:

- Type I Involves the aortic arch and its main branches
- Type II Involves descending thoracic and abdominal aorta
- Type III Has features of both type I and II
- Type IV Additional involvement of pulmonary artery

Anesthesia in TA is complicated by uncontrolled hypertension leading to end organ dysfunction, stenosis of major blood vessels affecting regional circulation, and difficulties in monitoring BP. Hence pre-operative assessment should aim to understand distribution of arteritis and degree of vital organ involvement.

Although anesthesia with subarachnoid block, epidural block or combined spinal-epidural has been used successfully in parturients with TA, there are limited reports on the use of GA in TA for emergency cesarean section. Regardless of the approach, goal should be to maintain adequate arterial perfusion pressures.

Our patient was severely eclamptic with renovascular hypertension, altered sensorium and seizures aggravated by underlying TA (Type-III). Hence we chose to administer GA. To avoid reduction of blood flow due to stretching of carotid arteries, neck extension was prevented by placing a pillow beneath patient’s head. Aorto-caval compression was prevented by left lateral tilt. Nitroglycerin infusion was kept ready to attenuate pressor response during intubation thereby protecting against cerebral hemorrhage. Since the patient was on chronic corticosteroid therapy, additional pre-operative steroids were given to prevent Addisonian crisis.
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cannulation. We preferred manual BP measurement over automatic measurement to avoid compromise in capillary perfusion due to over-inflation of cuff. The only palpable left dorsalis pedis artery was used to record BP and measure oxygen saturation. Several case reports state that BP generally remains unaffected in these patients undergoing surgery, regardless of how it is measured. Although, vasopressors should be avoided because of pre-existing compromised organ perfusion, we used a single bolus of ephedrine to combat hypotension following the delivery of the baby. Hyperventilation was avoided to prevent reduction in cerebral perfusion.

Patient was closely monitored for 24 h as GA might lead to hypertensive episodes causing cerebral haemorrhage or infarction and cardiac decomposition. Fortunately, she had an uncomplicated recovery without any convulsions or neurological deficit.

The choice of anesthetic technique depends upon the stage and severity of arteritis. In GA, the aim is to maintain the blood pressure to the patient's pre-operative level for successful outcome. Oscillometric method of non-invasive BP and pulse oximetry can also provide reliable monitoring where invasive BP or Doppler is not feasible.

Authors' Contribution:
SSN: Conduct of the case, writing the case report
AU: Conduct of the case, assisted writing the case report
ND: Conduct of case, Manuscript editing

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