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CASE REPORT

Anesthetic management of a patient with Takayasu's arteritis undergoing total laparoscopic hysterectomy

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ABSTRACT

Takayasu's arteritis (TA) is a rare, chronic progressive panendarteritis involving the aorta and its main branches, resulting in ischemia and persistent hypertension. Hypertension affects the anesthetic management where the main goal is to control the hemodynamics and prevent end organ damage. We present one case of diagnosed TA with hypertension and multiple cardiac morphological abnormalities posted for total laparoscopic hysterectomy.

Key words: Takayasu arteritis; Total laparoscopic hysterectomy

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INTRODUCTION

Takayasu's arteritis (TA) is a rare disease characterized by a focal stenotic process involving the aorta and the proximal segments of its main branches.[1] The affected vessel appears very thick walled, shortened, and rigid, with marked perivascular sclerosis and adhesion to surrounding tissues.[2] The disease is prominent in young women, has an onset before 40 years of age, with tuberculosis as a known culprit. This disease is also referred as pulseless disease, aortic arch syndrome, young female arteritis, and idiopathic aortitis.[3] We present a case report of a diagnosed TA with hypertension and multiple cardiac morphological abnormalities posted for total laparoscopic hysterectomy, which was successfully managed by us by undertaking a precisely tailored induction and maintenance protocol for general anesthesia (GA). Patient was discharged home after full recovery.

CASE REPORT

A 44-year-old female (79 kg, 168 cm) with medical history of hypothyroidism (since 5 y), TA, hypertension (both since 2 y) as well as fibroid uterus and bilateral ovarian polyp, was posted for total laparoscopic hysterectomy in our hospital. Concomitant medications included azathioprine 50 mg, prednisolone 10 mg, amlodipine 10 mg, aspirin (EcospirinTM, Usv Ltd.) 75 mg and levothyroxine sodium (EltroxinTM, Concordia International) 50 μg.

She revealed a history of recurrent left upper arm pain and numbness starting 2 y back which was now controlled with above medications. There was

no history of syncope or seizures. The patient's blood pressure (BP)was 150/80 mmHg in the right upper limb; however, her left brachial artery and radial artery were not palpable. Chest radiography demonstrated enlargement of the cardiac shadow. Twelve-lead electrocardiography revealed only left ventricular hypertrophy. Echocardiography revealed trivial mitral regurgitation, trivial tricuspid regurgitation with an ejection fraction of 45%.

Contrast angiography done 2 y back showed stenosis of the left subclavian artery, left axillary artery and brachial artery. There was no involvement of the coronary arteries, and carotid Doppler was normal. All other blood investigations were unremarkable. On the day of operation, all the routine medications were given orally 4 h preoperatively with sips of water. Standard monitoring was done along with invasive right radial arterial BP. Baseline heart rate was 80 beats/min and BP in the right radial artery was 150/80 mmHg. Electrocardiogram (leads II and V5), end tidal carbon dioxide ($EtCO_2$), body temperature, and peripheral artery oxygen saturation were monitored throughout anesthesia. The infusion was initiated with dexmedetomidine 0.5 μg/kg/h for 10 min, along with inj hydrocortisone 100 mg, inj magnesium 1 gm, inj lignocaine 1mg /kg followed by standard general anesthesia (GA) with inj propofol 120 mg, fentanyl 150 μg and morphine 5 mg. After confirmation of loss of consciousness, rocuronium (50 mg) was administered intravenously and oral intubation was performed. The lungs were ventilated with oxygen (2 L/min) and air (2 L/min) and anesthesia was maintained with sevoflurane 1.5% and dexmedetomidine infusion. We prevented fluctuations in baseline BP to avoid end organ damage, and maintained EtCO2 between 35 and 40 mmHg during anesthesia. Hemodynamics and other vital parameters were stable during pneumoperitoneum and the duration of the surgery was 188 min. Total blood loss was 200 ml and IV fluids were replaced adequately with KABILYTETM (Multiple Electrolytes Injection Type 1 USP) 1.5 L). We used infiltrative analgesia by administration of 10 ml of 0.5% bupivacaine to the surgical port sites. The patient was extubated after recovery of neuromuscular blockade and adequate spontaneous breathing. Postoperative pain was managed with inj paracetamol and inj tramadol IV. The patient was shifted to the post-op surgical ICU for observation and was discharged from hospital subsequently on day 4 from ward without any complications.

DISCUSSION

TA is a rare disease characterized by a focal stenotic process involving the aorta and the proximal segments of its main branches.[1] The affected vessel appears very thick walled, shortened, and rigid, with marked perivascular sclerosis and adhesion to surrounding tissues.[2] The disease is prominent in young women, has an onset before 40 years of age, with tuberculosis as a known culprit. This disease is also referred as pulseless disease, aortic arch syndrome, young female arteritis, and idiopathic aortitis.[3] The major clinical finding is loss of palpable pulses in the upper limbs and neck. The unsuspected ischemia in vital regional vascular beds may be associated with high anesthetic risks for these patients. Hypertension, the major complication affecting anesthetic management in patients with TA, is commonly renovascular, and is the most common cause of renovascular hypertension in India.[4] TA is of 4 types;[5] type I disease involves the aortic arch and its main branches, type II lesions are restricted to descending thoracic and abdominal aorta, type III patients show features of both type I and type II, type IV patients additionally have involvement of the pulmonary artery. This patient belonged to type 1 TA anatomically and to grade 2b, as per Ishikawa classification system for acute pancreatitis.

Since monitoring cardiovascular status and function is extremely important in these cases and may be complicated if peripheral pulses are non-palpable in any extremity, therefore right radial arterial monitoring was done in this patient as left hand was the affected one. Perioperative steroid replacement (hydrocortisone 100 mg) was given to prevent the occurrence of hypotensive crisis, as these patients are on long term steroid replacement therapy, e.g. prednisolone 10 mg (Wysolone™ 10 mg Tablet DT, Pfizer).[6]

In order to prevent end organ damage due to uncontrolled blood BP and because of cardiac involvement (left ventricular hypertrophy, mitral and tricuspid regurgitation) in this patient, the cardioprotective drugs were kept ready. Antihypertensive drugs and thyroid replacement were continued until the morning of surgery. Slow infusions of dexmedetomidine[7] and lignocaine were initiated as a pre-induction sequence to facilitate a stress free induction and intubation. Magnesium was infused as premedication to prolong the neuromuscular blocking effects, blunting of stress response and decrease opioid requirements. BP was not allowed to go beyond 150 mmHg systolic and maximum recorded heart rate was 80/min. Postoperative pain relief is also very important to prevent stress related complications. Analgesics were used judiciously by local infiltration of port sites plus

paracetamol and tramadol IV. We deliberately avoided diclofenac for analgesia to prevent hypertension and renal complications.

CONCLUSION

Takayasu's arteritis is a progressive disorder with varied clinical features indicative of vascular involvement, end organ damage making general anesthesia for laparoscopic surgery particularly challenging. Patients of Takayasu's arteritis can be managed successfully under GA with pre-induction measures preventing stress response to intubation and minimizing the hemodynamic alterations associated with this disease during and after surgery.

Conflict of interest: Nil

Authors' contribution: All authors took part in conduct of this case report and manuscript preparation.

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