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Case Report

Successful use of unilateral spinal block after failed epidural block in a patient with severe aortic stenosis

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Introduction

General anesthesia (GA) is the preferred technique for patients with severe aortic stenosis (AS) undergoing noncardiac surgery.[1] Spinal anesthesia (SA) is fraught with dangers in these patients because of unpredictable and rapid fall in systemic vascular resistance (SVR), resulting in low diastolic blood pressure leading to decrease in coronary perfusion pressure; this precipitates myocardial ischemia, rhythm disturbance, ventricular dysfunction, and vicious cycle of worsening hypotension.[2] In these patients when GA poses major risk because of associated respiratory comorbid conditions, combined spinal epidural (CSE) and graded epidural anesthesia (EA) have been employed; however, the risk of significant hypotension from profound vasodilation exists in spinal technique.[3,4] EA causes gradual decrease in SVR induced by slow peripheral sympathetic blockade which can be managed by vasopressors but failed/patchy epidural block can be a concern.[5,6] Unilateral spinal block (USB) can be employed as a bailout anesthetic technique for lower limb surgery in select patients, where graded EA had failed and GA is contraindicated.

Case report

An 85-year-old female patient presented for left hip arthroplasty following an injury. Her past medical history included severe AS, angina, hypertension, and pulmonary tuberculosis in New York Heart Association Functional Class III. A preoperative echocardiogram revealed a critical AS with aortic valve area $<0.8\ cm^2$, peak aortic transvalvular gradient of 120 mmHg, an ejection fraction (EF) of 60%, and left ventricular hypertrophy (LVH), with mild aortic and mitral regurgitation. The patient had refused coronary angiography and surgical management of aortic valve disease in the past.

Preoperative chest radiograph showed fibrosis in both upper zones and bilateral pleural effusion. CT scan confirmed nodulofibrotic lesions with features of bilateral pleural

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effusion and thickening, along with adjacent subsegmental atelectasis. Her medications included low molecular weight heparin (LMWH), ecosprin, losartan, atrovastatin, indapamide, rifampicin, ethambutol, and pyrizinamide.

The patient was 160 cm in height and weighed 48 kg. Preoperative blood pressure (BP) and heart rate (HR) were 158/90 mmHg and 88 bpm respectively and Mallampati class III air way. The patient had mild scoliosis. She had grade 4/6 ejection systolic murmur radiating to carotids. Auscultation of lungs was notable for bronchial breath sounds in right upper lobe and decreased air entry base of lungs. Her breath holding time and SPO_2 on room air was 15 s and 88–90% respectively. The Arterial blood gases revealed pH 7.37, PaO_2 – 76 mmHg, $PaCO_2$ – 37.2 mmHg, and HCO_3 – 23 mEq. Forced vital capacity (FVC) and forced expiratory volume in 1 s (FEV1) were 1.28 L (50% of predicted normal) and 0.89 L (41.9% of predicted normal) respectively, and FEV1/FVC was 72.04%, indicating moderate restrictive ventilatory defect.

The electrocardiogram (ECG) revealed HR of 92 bpm, left axis deviation, and features of LVH. Laboratory values included a hemoglobin of 10.2 g/dl and total serum proteins 6.4 g/dl with normal liver and renal function tests. Tab losartan and LMWH were omitted, 12 h prior to surgery. The patient had normal coagulation profile, and antibiotic prophylaxis against infective endocarditis was provided on morning of surgery.

ECG and pulse oximeter monitors were attached in Operation Theater. Under local anesthesia, 16 gauge cannula was inserted for intravenous (IV) access; fentanyl 20 μg IV was given. Under local anesthesia, central venous cannulation in right internal jugular vein and radial artery catheter for invasive hemodynamic monitoring was established. Central venous access was also used perioperatively for administering vasopressors. Oxygen @4–6 L/min was provided with face mask. Lactated Ringer's solution (300 ml) was infused and continued under central venous pressure monitoring. A continuous infusion of inj. norepinephrine (NE) was started @0.025 μg/kg/min through central venous route. An 18-gauge Tuohy epidural needle was inserted at L3–4 spinal interspace. A 20-gauge epidural catheter was inserted 4 cm into the epidural space. 2 ml of bupivacaine 0.5% plain was injected into the epidural catheter. After reassessment of BP, HR, ECG ST segment monitoring and sensory level, a second dose of 3 ml of bupivacaine 0.5% was given after 10 min. A L1 level of sensory anesthesia was obtained in nonoperative limb (right) with mild loss of sensation in operative limb (left). A third dose of 3 ml of bupivacaine 0.5% was repeated after 10 min. A T8 sensory level of anesthesia was obtained in nonoperative limb but no improvement of anesthesia was appreciated in operative limb. The patient remained comfortable. After a further wait of 15 min, it was decided to give unilateral SA. The patient was turned into semi-reclining position with support; 1.5 ml of 0.5% bupivacaine (7.5 mg) (hyperbaric) was injected into subarachnoid space by 25 G-Quinke needle after confirmation of CSF. The patient was placed in left lateral position for 5 min, and then shifted to supine position. At this time, her blood pressure decreased to 84/48 mmHg and her HR decreased to 60 bpm. She denied any complaint of breathlessness or chest pain. Her CVP was 8 mmHg. She was given bolus of 300 ml of Ringer lactate and inj. phenylephrine in aliquots of 50 μg to raise blood pressure. Her NE infusion was increased to 0.05 μg/kg/min. Her blood pressure increased to 110/68 mmHg and HR stabilized at 78 bpm within 3 min. Her CVP increased to 11 mmHg. A T8 sensory level of anesthesia was achieved in operative limb and the patient was positioned for surgery. Urinary bladder was catheterized. The entire operative procedure lasted for 1 h and 10 min. Light sedation was provided with additional 30 μg of fentanyl and 1 mg of midazolam (Table 1).

The patient was transfused with 1 unit of packed red blood cells and 1200 ml of Ringer's lactate solution. Estimated blood loss and urine output were 350 ml and 400 ml respectively.

The patient was shifted to intensive care unit. Epidural catheter was removed. She was given maintenance fluid @100 ml/h of Ringer's lactate and intravenous tramadol hydrochloride for pain relief in postoperative period. NE infusion was gradually tapered off in next 12 h.

There was no complaint of postdural puncture headache or cardiac event. She had an uneventful postoperative recovery and was discharged after 5 days.

Discussion

There is a significantly higher incidence of preoperative mortality and nonfatal myocardial infarction in patients with AS than in patients without AS (14% vs 2%, $P < 0.05$); also, this rate is substantially higher in patients with severe AS compared with moderate AS (31% vs 11%, $P = 0.04$).[7]

Opioid-based GA is preferred in these patients to avoid the detrimental effects of volatile agents on sinus automaticity, myocardial contractility, preload, and SVR.[8] Our patient had critical AS, she was symptomatic for tuberculosis, and had clinico-radiologic evidence of compromised respiratory status. The opioid-based techniques require large doses of narcotic agents to blunt the hemodynamic response at induction and for maintenance of anesthetic depth, but the associated respiratory depression necessitates the requirement of postoperative ventilation which could have resulted into added morbidity in our patient with compromised respiratory status; hence, GA was avoided and regional technique was contemplated. Irrespective of the anesthetic technique, goals during anesthesia in patients with severe AS are maintenance of normal sinus rhythm, adequate vascular volume, and higher SVR with aggressive use of α-adrenergic agents.[9]

Ho et al. studied clinical outcome in 22 asymptomatic patients with AS undergoing hypotensive EA for total hip replacement. In their study, aortic valve area ranged from 0.9 to 1.8 cm^2 and peak gradient from 12 to 64 mmHg. Systolic BP was maintained at 60–100 mmHg and HR varied between 70 ± 11 bpm, and the mean duration of hypotension was 91 min. Total crystalloid administered was 1695 ml (range: 900–4000 ml) and mean estimated blood loss was 234 ml (range: 100–1500). They reported absence of complications in terms of death, myocardial infarction, cerebro-vascular accident, or pulmonary embolism with hypotensive EA in patients with noncritical AS.[4]

In our patient, graded EA was selected because of more gradual onset of peripheral sympathetic nervous system blockade which could be managed easily by vasopressors. We started the infusion of reduced dose of NE under invasive

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