



Peripheral Nerve Blocks as Anaesthetic Choice for Asymptomatic Severe Aortic Stenosis for Emergency Lower Limb Surgeries: A Case Report

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Abstract

Patients with severe aortic stenosis, whether symptomatic or not possess unique challenge to the anaesthetist . Both general anaesthesia and regional anaesthesia with central neuraxial blockade carry potential risks owing to a fixed cardiac output. Haemodynamic fluctuations associated with anaesthesia can be detrimental in these patients. They are at increased risk for intraoperative and post-operative complications given the severity of aortic stenosis. A clear intraoperative plan should be designed to manage the unique haemodynamics of these patients. We report a case of asymptomatic severe aortic stenosis posted for an urgent lower limb surgery. Ultra sound guided combined popliteal-sciatic and femoral nerve blocks were used as a sole anaesthetic technique. Patient was haemodynamically stable in the intra-operative as well as post operative period. Patient did not require any additional analgesic for about 10 hours post-operatively.

Keywords: *Popliteal sciatic nerve block, Femoral nerve block, Aortic stenosis, Lower limb surgery.*

Introduction

Aortic stenosis may be encountered in as much as 1-4% of adults over 65 years of age .Many of these patients can progress to severe obstruction requiring aortic valve replacement therapies. Patients with asymptomatic aortic stenosis are not traditionally offered surgical aortic valve replacement given the lack of symptoms. However they are at increased risk for intra-operative and post-operative complications. Owing to a fixed cardiac output state in severe aortic stenosis, these patients may not tolerate even modest changes in their haemodynamics associated with central neuraxial blockade or general anaesthesia and can be delirious.

Most of the time, severe asymptomatic aortic

stenosis may be revealed on routine echocardiographic examination in patients presenting for a non-cardiac surgery, posing unique challenges in optimizing the patient. This is especially if the surgery is an urgent one, when the decisions regarding surgery and anaesthesia are to be undertaken rapidly.

Case Report

A 76 year old lady reported at orthopedic casualty with a history of fall while walking and complaints of pain over left ankle. She was unable to stand upright due to pain. She had a history of hypertension and rheumatoid arthritis and was on regular medications. On examination she was moderately built and nourished. (body weight=60

kg, height=145cm) She had no pallor, icterus, cyanosis, clubbing, or lymphadenopathy. She had edema over the left ankle and foot. She did not give any history of chest pain, palpitation, syncope, cough, dyspnoea or fever. She did not have any past surgical history. Her pulse rate was 76/min, regular rhythm. Blood pressure was 140/80 mm of Hg in left upper limb, sitting posture. On cardiovascular examination, auscultation of precordial region revealed a harsh midsystolic ejection murmur of grade 3 intensity in the aortic area; there was no radiation. The murmur was best audible at full expiration and the patient leaning forward. Apex beat was located in the left 5th intercostal space just medial to midclavicular line. All blood investigations were within normal limit. X-ray of the left ankle joint showed a bimalleolar fracture. She was posted for open reduction and internal fixation. On cardiology consultation a 2D Echocardiogram was taken and showed the following findings: Severe aortic stenosis (PPG/MPG =98/54 mm of Hg) EF=84%, mild PAH(PASP 40 mm of Hg) No RWMA.

Considering the urgent nature of surgery, the Orthopedician planned to proceed with the surgery under high risk. Patient and the bystanders were counselled, and after their consent, planned to

proceed with the surgery.

Oral premedications with pantoprazole 40mg and alprazolam 0.25mg were given, and adequate fasting guidelines were followed. Procedure was explained to the patient in detail and ensured her full cooperation. IV access taken with 20G cannula in the right upper limb.

In the operating room monitors were attached which included pulse oximetry, 5 lead ECG and non-invasive blood pressure. After local infiltration, right radial artery cannulated with 20G arterial cannula. Arterial blood pressure transduced to obtain a beat to beat variation in blood pressure. Patient was sedated with intravenous midazolam 1 mg and intravenous fentanyl in an incremental dose upto 1mcg/kg. Patient was positioned in the right lateral decubitus position and left leg slightly flexed. After strict aseptic precautions, a linear high frequency ultrasound probe was used to locate the sciatic nerve in the popliteal fossa. (Hyperintense structure lying superficial to the popliteal artery). After local infiltration of the skin, using "in-plane" technique needle was advanced to the popliteal sciatic nerve.

A dose of 20ml of local anaesthetic (10ml of 0.25% levobupivacaine and 10 ml of 1 % lignocaine with adrenaline) was injected around the sciatic nerve.

Figure 1: Sciatic nerve as seen as hyperechoic structure lying superficial to the popliteal artery



Then the patient was positioned supine. After strict aseptic technique, the left femoral nerve was located below the left inguinal crease using the same high frequency linear ultrasound probe. Femoral block was given in an "in-plane

"technique using the same needle A dose of 20ml of local anaesthetic was injected around the femoral nerve.(10ml of 0.25% levobupivacaine and 10 ml of 1 % lignocaine with adrenaline)

Figure 2: Femoral nerve seen lateral to the pulsatile femoral artery in ultrasonography



A waiting period of 20 minutes given for the local anaesthetic to act. After ensuring adequate anaesthesia surgery was started.

Total operating time was about 80 minutes .Total blood loss was about 200ml and the same was replaced with crystalloids. Patient's haemodynamics remained stable during the entire peri-operative period. No additional analgesics were required during the intra-operative period.

Discussion

Aortic valve maintains anterograde flow of blood to the aorta when open and prevents retrograde flow of blood into the left ventricle when closed. Reduction in aortic valve area in aortic stenosis causes a resistance to the flow of blood across the aortic valve. This causes a pressure overload in the left ventricle leading to the development of an aortic trans-valvular gradient as the aortic valve stenosis worsens.

Left ventricle begins to hypertrophy by increasing the left ventricular muscle mass to overcome this pressure gradient, so that the ratio of pressure to ventricular thickness remains constant. This will result in reduced left ventricular compliance, an increase in oxygen demand-supply ratio which can result in angina pectoris, even when there is absence of coronary artery disease and finally left ventricular systolic dysfunction.

Most common acquired cause of aortic valve stenosis is calcification of degenerative aortic valve. Presence of a bicuspid aortic valve also predisposes to the development of aortic valve stenosis.

Table 1 Echocardiographic Grading of Aortic stenosis

	Mild	Moderate	Severe
Peak velocity(m/s)	<3.0	3.0 – 4.0	>= 4.0
Mean gradient(mm of Hg)	<20	20 - 40	>= 40
Aortic valve area (cm2)	>1.5	1.0 – 1.5	<1.0

Aortic stenosis is clinically characterized by angina pectoris, syncope or dyspnoea on exertion. Clinical signs of aortic stenosis include a low volume anacrotic pulse (pulsus parvus et tardus), a palpable thrill in carotid artery (carotid shudder), a heaving apex beat which is normal in location, a palpable systolic thrill in aortic area which is best felt in full expiration with the patient sitting and leaning forward and the thrill may be radiating to the carotid artery, an ejection click on auscultation and a harsh mid systolic ejection murmur with direction of selective propagation towards carotid, best audible in full expiration with the patient sitting and leaning forward and with the diaphragm of the stethoscope (crescendo-decrescendo murmur – saw tooth like).

In patients with aortic stenosis, the aortic kick contributes to 40% of total cardiac output instead of the usual 20%; so the maintenance of normal sinus rhythm is of paramount importance and any arrhythmia developing in the perioperative period can be detrimental.

Also peri-operative hypoxia, hypercarbia or tachycardia can worsen the pulmonary hypertension associated with severe aortic stenosis. So in severe aortic stenosis with severe pulmonary artery hypertension, there is high risk for administering both general anaesthesia and central neuraxial blockade.

General anaesthesia itself increases the stress response in the patient which can lead to haemodynamic instability. This will be poorly tolerated by a patient with severe aortic stenosis.

Central neuraxial blockade can lead to hypotension and bradycardia, which may be poorly tolerated by the patient.

Hence for this patient we planned to proceed with ultra-sound guided femoral nerve block combined with popliteal sciatic nerve block.

Femoral nerve supplies the skin over the antero-medial aspect of the thigh and knee, medial border of the leg and medial malleolus (via saphenous nerve).

Sciatic nerve supplies cutaneous innervation to the

posterior aspect of the thigh and the entire foot and leg below the knee except a thin medial strip (saphenous nerve).

Conclusion

Ultra sound guided combined femoral nerve and popliteal sciatic nerve block is the best option for patients with severe aortic stenosis presenting for emergency lower limb surgeries; where general anaesthesia as well as central neuraxial blockade poses threats of haemodynamic fluctuations.

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