

Ultrasound Guided Peripheral Nerve Block For Lower Limb Fracture Fixation In A Patient With Hypertrophied Obstructive Cardiomyopathy (HOCM)

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Abstract

The presentation of hypertrophic obstructive cardiomyopathy may vary from an asymptomatic patient to sudden death. The changes in preload, afterload and ventricular contractility may occur due to surgery, anaesthesia and fluid shifts in the perioperative period. This causes worsening of the left ventricular outflow tract obstruction, ultimately causing myocardial ischemia and cardiac dysrhythmias. This case discusses successful anaesthesia management of a 49-year-old male with hypertrophic obstructive cardiomyopathy operated for tibia and fibula open reduction, internal fixation using ultrasound-guided sciatic and femoral nerve block. Transthoracic Echocardiography served as an additional monitoring modality to assess cardiac function intraoperatively. The patient underwent successful surgery with stable hemodynamics and reported excellent pain relief postoperatively.

Keywords: Hypertrophic obstructive cardiomyopathy, Transthoracic Echocardiography, Peripheral nerve block

Introduction

Hypertrophic Obstructive Cardiomyopathy (HOCM), a type of hypertrophic cardiomyopathy is an autosomal dominant disorder affecting 1 in 500 individuals [1, 2]. It is characterised by asymmetrical hypertrophy of the interventricular septum (IVS) occurring in the absence of any underlying cause like aortic stenosis, associated with myocardial fiber disarray. Left ventricular outflow tract (LVOT) obstruction occurs due to the systolic anterior motion of the anterior mitral valve leaflet. It is also associated with mitral regurgitation, diastolic dysfunction (myocardial fibrosis), left atrial enlargement leading to supraventricular arrhythmias and eventually systolic dysfunction.

Symptoms may range from asymptomatic patients to angina, dyspnea, dizziness, syncope, or sudden death occurring at any age [1, 3, 4].

There are a number of treatment modalities for these patients, including pharmacotherapy (beta-blockers and calcium channel blockers) to control symptoms,

implantable cardiac defibrillators to manage malignant arrhythmias, and surgical myectomy and septal ablation to decrease the LVOT obstruction [1].

Changes in preload, afterload, ventricular contractility which occur during the perioperative period can worsen the LVOT obstruction leading to adverse cardiovascular outcomes, such as myocardial ischemia, arrhythmias, and congestive heart failure [1, 5, 6].

Here we report successful management of a patient with HOCM for tibia fibula fixation under ultrasound guided sciatic and femoral nerve block with sedation.

Case report

A 49-year-old, 65 kg male with a known history of hypertension (HTN) presented with pain and swelling over the right lower limb following a bike accident. Radiological investigations confirmed a proximal tibia fibula fracture. There were no associated injuries. He was on Tab metoprolol 25 mg and Tab telmisartan 40 mg OD. Patient denied history of chest pain, dyspnea,

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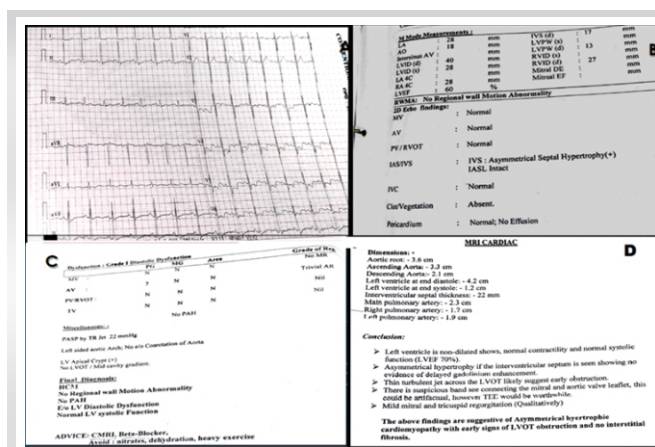
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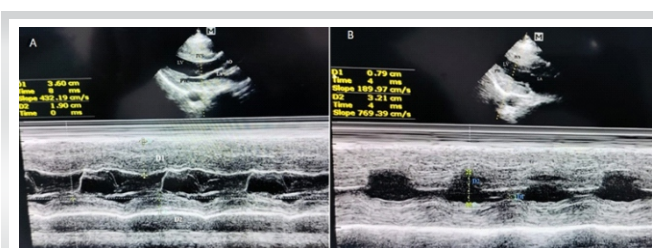
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dizziness or syncope. On examination, he had a pulse rate of 70/min regular, blood pressure of 200/110 mmHg in the right upper limb in supine position. A grade 2 systolic murmur was heard in 3rd ICS on the right sternal border and at the apex. Rest of the systemic examination and airway examination was within normal limits. The ECG showed ST segment depression and T wave inversion in leads V3 to V6. Transthoracic echocardiogram (TTE) revealed hypertrophic cardiomyopathy, asymmetrical septal hypertrophy of the IVS, trivial aortic regurgitation (AR), and evidence of left ventricular diastolic dysfunction with normal systolic function (Fig. 1). Cardiac MRI revealed symmetrical hypertrophic cardiomyopathy with a thin turbulent jet across the LVOT, suggesting early signs of LVOT obstruction without interstitial fibrosis (Fig. 2).

The cardiologist advised him to continue the antihypertensive medication and avoid exertion, nitrates and dehydration. Additionally serial monitoring of blood pressure, ECG perioperatively to prevent arrhythmias and heart failure was advised.

Patient was considered ASA III E. Preoperatively high risk consent was explained. Patient was kept nil by mouth 6 h for solids and was allowed clear fluids upto 2 h prior to surgery. An 18 gauge IV line was secured in the left upper limb. Antibiotic prophylaxis with ceftriaxone 1 gm was administered after test dose. Defibrillator was checked and kept ready along with resuscitative and antiarrhythmic drugs. Patient was sedated with inj Fentanyl 1 mcg/kg and inj Midazolam 1 mg. The patient was given lateral position for the sciatic nerve block by the subgluteal approach. Ultrasound (Samsung sonoace R7) and peripheral nerve stimulator (B Braun) (0.3-0.5 MA), was used to locate the sciatic nerve using the 10 cm echogenic needle. The drug administered was 12.5 ml of 0.75% ropivacaine + 12.5 ml of 2% lignocaine with adrenaline [7]. Similarly USG-guided



femoral nerve block was administered using 9 ml of 1% lignocaine with adrenaline + 9 ml of 0.375% ropivacaine mainly for sensory anaesthesia. Intravenous dexamethasone 8mg was administered. Dexmedetomidine IV infusion at the rate of 0.5 ug/kg/min was administered for sedation throughout the procedure. Complete sensori-motor anaesthesia was achieved after 35 min of giving the block. Tourniquet was inflated for 1.5 h and the procedure lasted for 2 h. During the procedure 1200 ml of Ringer Lactate solution was administered. There were no haemodynamic fluctuations. Intraoperative hemodynamics remained in the range of HR 65-74/min, SBP 150-180 mmHg, and DBP 60-90 mmHg.

Focused TTE was performed at the following time points, after onset of block, after tourniquet inflation and after tourniquet deflation to provide real-time assessment of cardiac function and guide intraoperative fluid management decisions (Fig. 2). Postoperatively the VAS scores were <4 for the first 24 h. The patient was mobilised on postoperative day one and could be discharged on day two.

Discussion

Perioperative goals in case of HOCM are adequate preload, maintain SVR, avoid sympathetic activation, avoid tachycardia, maintain sinus rhythm and mean arterial blood pressure at >65 to 70 mmHg to maintain coronary perfusion pressure [1, 4, 5, 6, 8]. The use of general anaesthesia can cause severe sympathetic stimulation during intubation whereas neuraxial anaesthesia carries the risk of decrease in SVR which can worsen the LVOT obstruction [3, 8, 9].

Regional anaesthesia (RA) in the form of peripheral nerve block (PNB) is an attractive and effective option in patient with HOCM posted for lower limb surgery. Besides minimising the intraoperative haemodynamic fluctuations, it offers the advantage of postoperative pain relief, less nausea vomiting, early oral feeding and early rehabilitation [4, 5]. Previous authors have described use of plexus blocks and peripheral nerve blocks for hip fracture fixation and knee arthroscopy respectively [4, 5]. Some authors have performed nephrolithotripsy under low dose spinal with haemodynamic monitoring [8]. In our case, below knee

surgery with thigh tourniquet was to be performed under sciatic and femoral nerve block, where major fluid shifts were not expected, we decided against invasive monitoring. Dexmedetomidine a selective alpha-2 adrenergic agonist, through its central inhibition of norepinephrine release at the locus coeruleus in the pons, indirectly reduces the sympathetic response during surgery thus reinforcing our perioperative goal [5]. Literature mentions use of IV fluid bolus and alpha-adrenergic agonist (phenylephrine) are the preferred first line of treatment for hypotension in patients with HOCM [2, 8, 9]. In our case there were no major haemodynamic fluctuations requiring the above interventions.

Transthoracic echocardiography serves as a valuable intraoperative tool to assess hemodynamic disturbances caused by hypovolemia, increased LVOT obstruction, systolic anterior motion (SAM) of the mitral valve, or left ventricular systolic dysfunction [10] (Fig. 2). In our case the TTE confirmed that there was no worsening of LVOT

obstruction. The decreased left ventricular internal diameter in diastole prompted us to give fluid bolus. The lower postoperative pain scores helped in enhanced recovery after surgery.

Conclusion

Familiarity with the intricate pathophysiology of hypertrophic obstructive cardiomyopathy (HOCM) is pivotal for safe and effective perioperative care. The successful surgical outcome in this case was made possible by the coordinated efforts of the anesthesia, cardiology, and surgical teams—underscoring the importance of a comprehensive, multidisciplinary approach. As the pathophysiological understanding of HOCM continues to advance, the anaesthesiologist's role remains central in devising and executing meticulous perioperative strategies aimed at minimising risk and ensuring haemodynamic stability.

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Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his/her consent for his/her images and other clinical information to be reported in the Journal. The patient understands that his/her name and initials will not be published, and due efforts will be made to conceal his/her identity, but anonymity cannot be guaranteed.

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