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Case Report

Anaesthesia Section

Anaesthetic Management in a Patient with Severe Left Ventricular Dysfunction Undergoing Lumbar Laminectomy and Spinal Fixation: A Case Report

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ABSTRACT

Severe Left Ventricular Systolic Dysfunction (LVSD), in addition to serious valve heart disease, presents a daunting challenge for anaesthesiologists in non-cardiac surgery. We present the case of a 75-year-old woman scheduled for L3 laminectomy and spinal fixation due to lumbar canal stenosis. She had an Ejection Fraction (EF) of 20%. On pre-operative assessment, severe LVSD with valvular lesions was noted. Anaesthesia was induced with etomidate, fentanyl, and vecuronium to minimise cardiac stress and maintain stable blood flow. Arterial and central venous lines were placed for continuous monitoring. Anaesthesia was maintained with a mixture of oxygen, air, and sevoflurane. Infusions of noradrenaline and dobutamine were adjusted to maintain stable blood pressure. The patient was positioned with care to avoid venous compression and to maintain preload. Fluid administration was performed very cautiously to avoid overload. She remained stable throughout the procedure and was extubated uneventfully. Later, she was transferred to the intensive care unit for close monitoring during recovery. This case illustrates that careful anaesthetic planning, close monitoring, appropriate fluid management, and proactive support are of paramount importance in high-risk cardiac patients for spine surgery, since even slight changes in blood flow can lead to major complications.

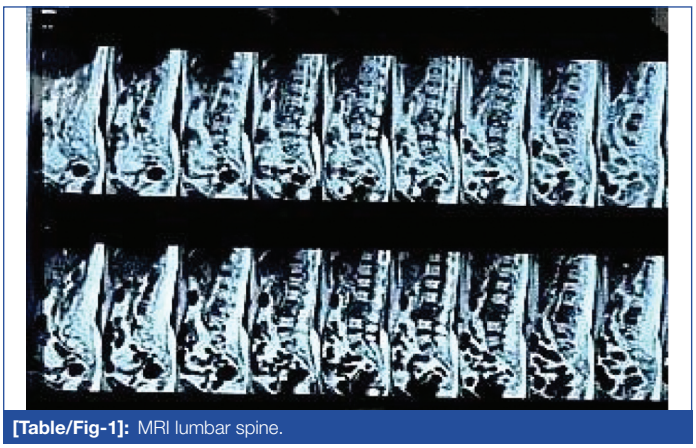
Keywords: Cardiomyopathy, Etomidate, Haemodynamic monitoring, Inotropes, Prone position

CASE REPORT

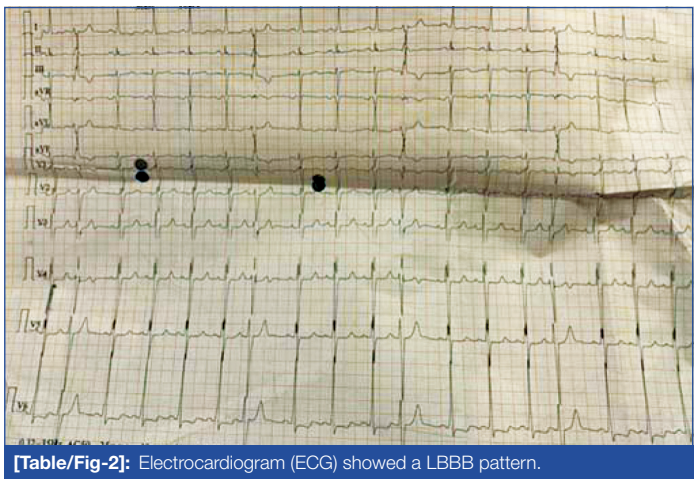
A 75-year-old female was posted under neurosurgery for L3 laminectomy and spinal fixation due to progressive difficulty in walking following a fall five months ago. She was average build, weighing 54 kg and standing 164 cm tall. The patient gave a history of dyspnoea on exertion with the performance of activities of daily living, without any chest pain or orthopnoea, corresponding to New York Heart Association Class III dyspnoea. She was a known case of hypertension for 10 years on regular medications, type 2 diabetes mellitus for two years controlled by insulin, and ischaemic heart disease for 15 years with a history of Left Bundle Branch Block (LBBB). Her regular medications included sustained-release ranolazine 500 mg once daily, torsemide 10 mg once daily, and amlodipine 5 mg once daily. Magnetic Resonance Imaging (MRI) of the lumbar spine showed a compression fracture of L3 with retropulsion of fracture fragments, causing spinal canal stenosis [Table/Fig-1].

During the pre-anaesthesia evaluation, she was conscious and oriented, with a blood pressure of 138/76 mmHg, a heart rate of 72 beats per minute, and an SpO_2 of 98% on room air. Airway assessment revealed a Mallampati class II, with adequate mouth opening and full neck range of motion. Systemic examination revealed normal heart sounds, clear bilateral lung fields, and no pedal oedema. Electrocardiogram (ECG) showed a LBBB pattern [Table/Fig-2]. Echocardiography demonstrated an EF of 20%, suggestive of severe LVSD with global left ventricular hypokinesia, dilated left atrium and ventricle, grade III diastolic dysfunction, severe Mitral Regurgitation (MR), moderate Aortic Regurgitation (AR), and mild Tricuspid Regurgitation (TR) with an estimated right ventricular systolic pressure more than 20 mmHg and raised right atrial pressure. Laboratory investigations showed haemoglobin 12.3 g/dL, total leukocyte count 7,600/mm^3, platelet count 180×10^3/µL and normal renal, liver, and electrolyte profiles. Based on her systemic comorbidities, the patient was classified as American Society of Anaesthesiologists (ASA) physical status III.

Antihypertensive medications were continued on the morning of surgery, and diuretics were withheld. She was kept nil per os



[Table/Fig-1]: MRI lumbar spine.



[Table/Fig-2]: Electrocardiogram (ECG) showed a LBBB pattern.

for solids for six hours and two hours for liquids, and informed high-risk consent was obtained. In the operating room, standard ASA monitoring was established, including ECG, non-invasive blood pressure, pulse oximetry, capnography, and temperature monitoring.

General Anaesthesia (GA) was induced following adequate preoxygenation, using intravenous lidocaine 60 mg, fentanyl 85 µg, etomidate 8 mg, and vecuronium 6 mg to facilitate endotracheal intubation. The trachea was intubated atraumatically with a 7.0 mm cuffed endotracheal tube using a video laryngoscope. After establishing anaesthesia and attaining haemodynamic stability, invasive lines were secured. This included a 20-G right radial arterial line for continuous blood pressure monitoring and a 7 Fr right internal jugular central venous catheter for monitoring Central Venous Pressure (CVP) and administration of inotropes. For maintenance of anaesthesia, an oxygen-air-sevoflurane mixture was used. Haemodynamics were supported with a dobutamine infusion of 5 µg/kg/min and a noradrenaline infusion of 0.05 µg/kg/min, titrated to effect based upon blood pressure and heart rate. Haemodynamic parameters are depicted in [Table/Fig-3].

Stage of surgery	Heart Rate (beats/min)	Blood pressure (mmHg)	MAC (Sevoflurane)	Haemodynamic events	Management
Baseline (Pre-induction)	72	138/76	–	Haemodynamically stable	Standard monitoring established
Post-induction	68-74	120/70	0.8	Mild decrease in BP following induction	Dobutamine infusion continued at 5 µg/kg/min, noradrenaline started at 0.05 µg/kg/min
Post-intubation	70-78	126/72	0.9	No significant tachycardia or hypertension	Adequate anaesthetic depth maintained
After prone positioning	68-75	118/70	0.9-1.0	Transient fall in BP	Noradrenaline titrated to maintain MAP
During surgical dissection	65-75	110-120 /68-72	0.8-1.0	Stable haemodynamics	Inotrope and vasopressor infusions continued
Mid-surgery	64-72	112/70	0.8-0.9	No hypotension or hypertension	Fluids administered judiciously
End of surgery	70-76	124/74	0.6-0.7	Haemodynamically stable	Gradual reduction of anaesthetic concentration
Post-reversal (Pre-extubation)	72-78	128/76	–	No adverse events	Inotropic and vasopressor support was tapered.

[Table/Fig-3]: Haemodynamic parameters at various time-points.

The patient was then positioned prone with adequate padding to avoid pressure-related complications. The surgery was then started. Fluids were administered judiciously, and approximately 350 mL of Ringer's Lactate was administered during the 150-minute surgery. Blood loss was around 150mL, and an equal amount of packed red blood cells was replaced. Packed red blood cells were transfused to maintain adequate oxygen-carrying capacity and haemodynamic stability in view of severe LVSD and high cardiac risk. At the end of the surgery, the patient was carefully positioned supine, and after confirming adequate recovery on train-of-four monitoring ratio >0.9, reversal of neuromuscular blockade was performed with neostigmine 2.5 mg and glycopyrrolate 0.5 mg. After complete reversal of neuromuscular blockade, the patient was extubated and transferred to the ICU for 24 hours of observation. She was discharged on post-operative day 7. At the three-month follow-up, she was clinically stable.

DISCUSSION

The present case involved a patient with severe LVSD with an EF of approximately 20%, associated valvular heart disease, and LBBB, who required lumbar spine fixation in the prone position. These clinical findings indicated markedly reduced cardiac reserve with a high-risk of perioperative haemodynamic instability, making anaesthetic management particularly challenging. Anaesthetic management of patients with severe LVSD and valvular heart disease undergoing non-cardiac surgery is challenging. Poor myocardial contractility, altered volume status, and position-related haemodynamic changes combine to present peculiar challenges in the case of prone spine surgery [1,2]. Severe LVSD and valvular lesions in this patient demanded a well-planned and meticulously executed anaesthetic technique to maintain stable haemodynamics throughout the perioperative period. The principal anaesthetic challenges included preventing myocardial depression, maintaining adequate coronary perfusion pressure, avoiding sudden reductions in preload or systemic vascular resistance, and preventing arrhythmias in the presence of conduction abnormalities such as LBBB. Similar high-risk cardiac profiles have been reported in the literature, such as the case described by El Quafi K et al., where a patient with an EF of 25% undergoing hip fracture surgery required meticulous perioperative planning and invasive monitoring to prevent haemodynamic instability [3].

The GA was preferred due to the need for prone positioning, airway protection, controlled ventilation, and complete immobility during spinal fixation surgery. Regional anaesthesia could cause significant sympathetic blockade, leading to hypotension and reduced preload, which may be poorly tolerated in patients with severe LVSD and significant valvular disease. GA allowed better haemodynamic control with invasive monitoring and titrated inotropic and vasopressor support. The issues in anaesthesia management for such patients include maintaining a balance between myocardial oxygen supply and demand, avoiding sudden changes in preload and afterload, maintaining a sinus rhythm, and preventing myocardial depression. Etomidate, known for its stable cardiovascular profile, was chosen for induction. It induces anaesthesia very smoothly, with minimal effect on contractility, even in patients with low cardiac reserve. Fentanyl was added as an opioid supplement to blunt sympathetic responses to intubation and surgical stress. A low dosage of a volatile agent should be used to provide an adequate dose of anaesthetic without serious cardiac depression [4,5]. In the present case, anaesthetic depth was maintained with sevoflurane at a Minimum Alveolar Concentration (MAC) range of approximately 0.8-1.0, which allowed adequate anaesthesia while minimising myocardial depression. Comparable anaesthetic approaches using cardiostable induction agents such as etomidate with opioid supplementation have also been reported by Mittal AK et al., and Kumar M et al., emphasising the importance of minimising myocardial depression in patients with severely reduced EF [6,7].

In our patient, intraoperative heart rate was maintained between 65-78 beats per minute, and blood pressure was maintained within 110-128/68-76 mmHg with the support of dobutamine and noradrenaline infusions, thereby preventing episodes of significant hypotension or hypertension. While dobutamine increases the contractility and stroke volume, noradrenaline maintains the coronary perfusion pressure and SVR. The titration of these drugs together is an art of balancing to ensure perfusion without undue tachycardia or a rise in afterload. Continuous invasive arterial pressure monitoring is essential, as it provides direct feedback on these changes. The

placement of a central venous catheter facilitates the estimation of volume trends and provides a safe route for inotropic infusion. While interpreting CVP, clinical correlation remains important in patients with LVSD [8,9]. Kumar M et al., similarly highlighted the role of inotropic optimisation with dobutamine to improve ventricular function in patients with severely reduced EF undergoing major surgery [7].

Fluid therapy presents another significant obstacle. Underfilling is associated with hypotension and inadequate cardiac output, whereas overloading causes pulmonary congestion and decompensation. The most appropriate strategy is a restrictive, goal-directed fluid approach guided by invasive monitoring. Small incremental boluses of crystalloid, guided by haemodynamic response, maintain optimal preload without overloading the ventricle. Blood loss replacement should be guided by clinical parameters and perfusion indices rather than fixed volumes. Prone positioning has a significant effect on the haemodynamics. Abdominal and thoracic compression may result in reduced venous return and diminished cardiac output, which these patients poorly tolerate. Haemodynamic monitoring must not be interrupted during repositioning. Changes in inotrope or vasopressor support should be implemented promptly after turning the patient [10,11]. Consistent with this approach, intraoperative fluid administration in the present case was restricted to approximately 350 mL of crystalloid during the 150-minute procedure, and haemodynamic stability was maintained even after prone positioning. Dynamic monitoring and careful preload optimisation have also been emphasised by Mittal AK et al., who demonstrated that careful control of systemic vascular resistance and fluid balance helps maintain forward cardiac output and prevent pulmonary congestion in patients with reduced EF [6].

Maintaining the heart rate within an optimal range is another very important factor. Excessive tachycardia reduces diastolic filling time and compromises myocardial perfusion, while marked bradycardia decreases cardiac output in patients with fixed stroke volume. Myocardial ischemia and arrhythmias can be minimised by gentle titration of the depth of anaesthesia, avoidance of excessive sympathetic stimulation, and correction of electrolyte abnormalities. Refractory hypotension during induction, low cardiac output following prone positioning, arrhythmias related to myocardial irritability, and maintaining the delicate balance between preload and afterload are recurring challenges. Careful titration of anaesthetic agents and continuous haemodynamic monitoring in the present case helped prevent significant arrhythmias or ischemic episodes despite severe LVSD [12].

CONCLUSION(S)

Prudent planning, close monitoring, and active intraoperative management are required for the anaesthetic management of patients with severe LVSD and valvular heart disease undergoing major spine surgery. The successful outcome in this case depends on careful medication selection, guided and limited fluid therapy, prompt administration of inotropes and vasopressors, and careful placement to maintain haemodynamic stability. A properly balanced anaesthetic strategy should be employed that minimises myocardial depression yet allows for adequate anaesthesia, along with strict post-operative surveillance to prevent decompensation and allow successful recovery of these high-risk cardiac patients.

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