Case Report **Open Access**

Anaesthesia management of a Patient with Hypertrophic Obstructive Cardiomyopathy Undergoing c2-c5 Decompression in Cervical Myelopathy

Sayed Makarram Ahmed Bukhari, Atif Nazir

Abstract

Hypertrophic obstructive cardiomyopathy (HOCM) presents unique challenges in perioperative management due to its dynamic left ventricular outflow tract obstruction, diastolic dysfunction, and susceptibility to arrhythmias. This report highlights the anesthetic considerations in an 80-year-old female with HOCM and cervical spondylotic myelopathy undergoing C2-C5 decompressive surgery. The patient, an ASA-III with poorly controlled hypertension, experienced acute hemodynamic instability post-induction, necessitating emergent optimization. Re-attempted surgery under transesophageal echocardiography guidance, invasive hemodynamic monitoring, and tailored anesthetic techniques ensured uneventful completion. This case underscores the critical role of echocardiographic assessment, precise fluid management, and pharmacological strategies in minimizing perioperative risks in HOCM patients. The report aims to contribute to the limited literature on HOCM anesthetic management and to guide clinicians facing similar complexities.

Key words: Cervical Spondylotic Myelopathy, Hypertrophic Obstructive cardiomyopathy (HOCM), Perioperative care

How to cite this Article:
Bukhari SMA, Nazir A. Anaesthesia management of a Patient with Hypertrophic Obstructive Cardiomyopathy Undergoing c2-c5 Decompression in Cervical Myelopathy J Bahria Uni Med Dental Coll. 2026;16(1):202-204 DOI: https://doi.org/10.51985/JBUMDC2025785

INTRODUCTION:

Hypertrophic obstructive cardiomyopathy (HOCM) is a rare disorder characterized by hypertrophy of the left or right ventricle, which may be symmetric or asymmetric and classified as either obstructive or non-obstructive.[1,2] The most common form, subaortic HOCM, involves asymmetric hypertrophy of the interventricular septum, leading to dynamic obstruction of the left ventricular outflow tract (LVOT).[3] The extent and location of hypertrophy vary significantly, influencing the clinical presentation. This autosomal disorder exhibits a male-to-female predominance of 2:1.3, with an annual mortality rate of approximately 3–4% under medical management.[4] Treatment approaches include medical therapy, electrophysiological interventions, percutaneous transluminal septal myocardial ablation, and surgical options.[5] Anesthetic management in HOCM poses unique challenges, particularly in undiagnosed cases. We present the case of a previously undiagnosed HOCM patient undergoing C2-C5 decompressive cervical spine surgery and describe the perioperative anesthetic management in this context.

Case Report

An 80-year-old female with cervical spondylotic myelopathy was scheduled for decompressive surgery (laminectomy, C2-C5) under general anesthesia at Bahria International Hospital, Bahria Town Phase 8, Rawalpindi. The patient was classified as ASA-III, with a history of uncontrolled hypertension despite a quadruple antihypertensive regimen comprising hydralazine 100 mg, irbesartan 300 mg, hydrochlorothiazide 12.5 mg, and lercanidipine 10 mg. She was New York Heart Association (NYHA) Class II and Canadian Cardiovascular Society (CCS) Class I, with no significant limitations on ordinary physical activity. Preoperative echocardiography revealed concentric left ventricular hypertrophy (LVH), a left ventricular end-diastolic dimension (LVEDD) of 46 mm, and an interventricular septal thickness of 12 mm, with a normal ejection fraction (EF) of 60%. No overt left ventricular outflow tract (LVOT) obstruction was reported at rest.

Induction of anesthesia with propofol, nalbuphine, and cis-atracurium resulted in profound hypotension, refractory to multiple boluses of epinephrine (2.5 µg). Peak airway pressures were elevated (28–29 cmH2O), and pink frothy sputum was observed, indicating acute pulmonary edema. Bedside echocardiography confirmed severe obstructive cardiomyopathy with systolic anterior motion (SAM) of the mitral valve, moderate mitral regurgitation, and a small left ventricular cavity, consistent with dynamic LVOT obstruction.(Fig 1) The surgery was aborted, and the patient was stabilized with beta-blocker therapy (bisoprolol 2.5 mg BID) and fluid optimization.

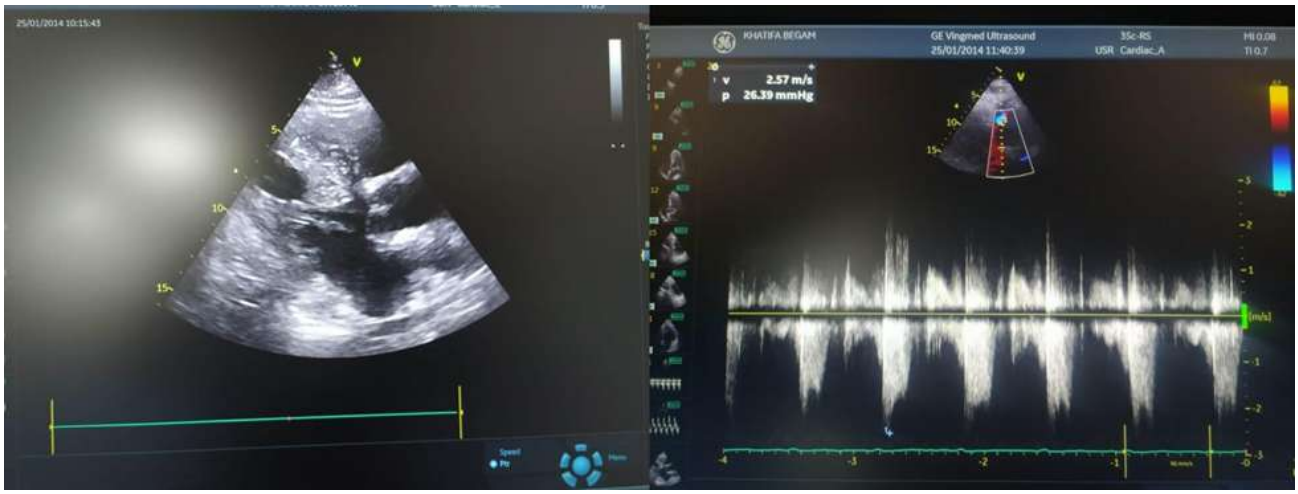
On the subsequent day, surgery was reattempted with a revised anesthetic plan to address hemodynamic challenges. Preoperative anxiolysis with midazolam was administered to reduce anxiety and facilitate smooth induction. Invasive

Sayed Makarram Ahmed Bukhari
Postgraduate Resident, Department of Anesthesiology
Bahria International Hospital, Rawalpindi
Email: makarrombukhari@gmail.com

Atif Nazir
Chief Resident, Department of Anesthesiology
Bahria International Hospital, Rawalpindi
Email: atifnazir656@gmail.com

Received: 23-10-2025
Accepted: 20-11-2025 1st Revision: 26-10-2025

Figure 1. Echocardiogram showing increased IVS thickness, SAM & LV outflow acceleration



monitoring included transesophageal echocardiography (TEE) to assess real-time ventricular function and loading conditions, along with placement of an arterial line in the left brachial artery and a 7.0Fr central venous catheter in the right femoral vein. Anesthesia was induced with low-dose propofol and fentanyl to blunt the sympathetic response to laryngoscopy, while cis-atracurium was used for neuromuscular blockade due to its minimal cardiovascular effects.Maintenance was achieved with sevoflurane (0.5 MAC), nitrous oxide (0.5 MAC), and dexmedetomidine (0.3µg/kg/hr) to ensure hemodynamic stability.Dynamic LVOT obstruction was monitored throughout the procedure using TEE, which guided adjustments in preload and afterload. Phenylephrine was used to maintain systemic vascular resistance (SVR), while care was taken to avoid hypovolemia or agents that could increase contractility. Glyceryl trinitrate was used sparingly to manage blood pressure without exacerbating obstruction. The surgical procedure proceeded uneventfully, and the patient was extubated in a deep plane and transitioned to an i-gel airway to minimize postoperative stress.

DISCUSSION

Hypertrophic obstructive cardiomyopathy (HOCM) is characterized by asymmetric septal hypertrophy and dynamic left ventricular outflow tract (LVOT) obstruction, often exacerbated by specific physiological triggers. These include reduced preload, increased contractility, and decreased afterload, all of which contribute to a worsening intraventricular gradient. The perioperative period poses significant risks for these patients due to the interplay of hemodynamic fluctuations, anesthetic agents, and surgical stress.

The pathophysiology of HOCM is marked by diastolic dysfunction, as impaired ventricular relaxation reduces cardiac compliance. Additionally, systolic anterior motion (SAM) of the mitral valve leaflet can further exacerbate LVOT obstruction and mitral regurgitation. These features complicate the perioperative management, as seen in this case, where the patient developed acute pulmonary edema and refractory hypotension following anesthesia induction. SAM, unmasked post-induction, highlighted the importance of understanding latent versus overt LVOT obstruction, especially in previously undiagnosed cases.[6,7]

In managing such patients, the primary anesthetic objectives are to maintain sinus rhythm, optimize preload, sustain afterload, and avoid increases in myocardial contractility. Beta-blockers, like bisoprolol administered in this case, are fundamental for reducing heart rate and mitigating the severity of LVOT obstruction. Volume optimization through judicious fluid administration is crucial to maintain ventricular filling and prevent hypovolemia.

The intraoperative use of transesophageal echocardiography (TEE) was instrumental in this case. It enabled dynamic monitoring of SAM, LVOT obstruction severity, and ventricular filling conditions, allowing timely adjustments in fluid and pharmacologic therapy. TEE is considered the gold standard for intraoperative assessment in HOCM and is particularly valuable in guiding management decisions.

The anesthetic strategy employed included the use of agents with minimal negative inotropic effects, such as sevoflurane, nitrous oxide, and dexmedetomidine, alongside careful titration of phenylephrine to maintain systemic vascular resistance (SVR).[8] These agents were chosen to avoid exacerbating LVOT obstruction while ensuring hemodynamic stability. Opioids and sedatives were administered to suppress catecholamine surges, reducing sympathetic outflow and associated risks of tachycardia.

Furthermore, rapid administration of induction agents and vasodilators must be avoided in HOCM due to their propensity to precipitate hemodynamic collapse. Instead, slow, controlled administration of induction agents, as performed in this case, mitigates such risks.[9]

This case underscores the importance of preoperative optimization, the utility of advanced intraoperative monitoring, and a multidisciplinary approach in managing HOCM patients undergoing non-cardiac surgery.[10] By tailoring anesthetic and pharmacologic strategies, clinicians can minimize perioperative complications and achieve favorable outcomes even in complex cases.

CONCLUSION

Hypertrophic obstructive cardiomyopathy presents significant anesthetic challenges due to its complex hemodynamic profile and dynamic LVOT obstruction. This case highlights the pivotal role of preoperative optimization, intraoperative echocardiographic guidance, and tailored anesthetic techniques in ensuring favorable surgical outcomes. Advanced monitoring with TEE and the judicious use of beta-blockers, vasopressors, and volatile anesthetics contributed to the safe and successful management of this patient. This report adds to the limited literature on HOCM management in non-cardiac surgical settings and provides a framework for perioperative care in similar high-risk cases.

Conflicts of Interest: Nil

Source of Funding: Nil

Acknowledgement: Nil

Authors Contribution:
Sayed Makarram Ahmed Bukhari: Substantial contribution to design of study, acquisition of data, Initial manuscript drafting
Atif Nazir: vSubstantial contribution to design of study, acquisition of data, Critical Review and final version of the manuscript

REFERENCES

1. Ommen SR, Mital S, Burke MA, Day SM, Deswal A, Elliott P, Evanovich LL, Hung J, Joglar JA, Kantor P, Kimmelstiel C, Kittleson M, Link MS, Maron MS, Martinez MW, Miyake CY, Schaff HV, Semsarian C, Sorajja P. 2020 AHA/ACC guideline for the diagnosis and treatment of patients with hypertrophic cardiomyopathy: executive summary: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Circulation. 2020;142(25):e533–e557. doi:10.1161/CIR.0000000000000938.
2. Elliott PM, Anastasakis A, Borger MA, Borggrefe M, Cecchi F, Charron P, Hagege AA, Lafont A, Limongelli G, Mahrholdt H, McKenna WJ, Mogensen J, Nihoyannopoulos P, Nistri S, Pieper PG, Pieske B, Rapezzi C, Rutten FH, Tillmanns C, Watkins H. 2014 ESC guidelines on diagnosis and management of hypertrophic cardiomyopathy: the Task Force for the Diagnosis and Management of Hypertrophic Cardiomyopathy of the European Society of Cardiology (ESC). Eur Heart J. 2014;35(39):2733–79. DOI:https://doi.org/10.1093/eurheartj/ehu284
3. Maron BJ, Maron MS. Hypertrophic cardiomyopathy. Lancet. 2013;381(9862):242–55. DOI:https://doi.org/10.1016/S0140-6736(12)60397-3
4. Lebowitz S, Kowalewski M, Raffa GM, Chu D, Greco M, Gandolfo C, Mignosa C, Lorusso R, Suwalski P, Pilato M. Review of contemporary invasive treatment approaches and critical appraisal of guidelines on hypertrophic obstructive cardiomyopathy: state-of-the-art review. J Clin Med. 2022;11(12):3405. DOI:https://doi.org/10.3390/jcm11123405
5. Maurizi N, Antiochos P, Owens A, Lakdawala N, Saberi S, Russell MW, et al. Long-term outcomes after septal reduction therapies in obstructive hypertrophic cardiomyopathy: insights from the SHARE Registry. Circulation. 2024 Oct 22;150(17):1377–90. doi:10.1161/CIRCULATIONAHA.124.069378.
6. Basit H, Alahmadi MH, Rout P, et al. Hypertrophic cardiomyopathy [Internet]. Updated 2024 Jun 7. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2025 Jan –. Available from: https://www.ncbi.nlm.nih.gov/books/NBK430788/
7. Chou CJ, Lai YC, Ou SY, Chen LC, Huang JH, Chen YW, Wu CY. Unexpected systolic anterior motion of the mitral valve-related hypoxemia during transurethral resection of the prostate under spinal anesthesia: a case report. BMC Anesthesiol. 2022;22(1):207. doi:10.1186/s12871-022-01754-x.
8. Ahmed A, Zaidi RA, Hoda MQ, Ullah H. Anesthetic management of a patient with hypertrophic obstructive cardiomyopathy undergoing modified radical mastectomy. Middle East J Anaesthesiol. 2010;20(5):739-742.
9. Cunningham JJ, Braun AS, Hussey P, Momaya A, Kukreja P. Regional anesthesia for arthroscopic knee repair in a patient with hypertrophic obstructive cardiomyopathy (HOCM) under monitored anesthesia care with dexmedetomidine infusion. Cureus. 2024;16(2):e53862. DOI:https://doi.org/10.7759/cureus.53862
10. Bhave A, Mohan G, Couture L, Sharma G, Yirerong J. Multidisciplinary approach to management of hypertrophic cardiomyopathy with severe left ventricular outflow obstruction in pregnancy. JACC Case Rep. 2023;27:102057. DOI:https://doi.org/10.1016/j.jaccas.2023.102057