Archives of Anesthesiology and Critical Care (In Press); x(x): xx-xx.

Available online at http://aacc.tums.ac.ir

Case Report

Anesthetic Management of a Patient with Severe Multiple Valvular Heart Disease for Emergency Major Non-Cardiac Surgery: A Case Report

Hannah May R. Ona*, Carlo D. Monteblanco, Vidal A. Esguerra

Department of Anesthesiology, Philippine General Hospital, University of the Philippines, Manila, Philippines.

ARTICLE INFO

Article history:
Received 29 April 2026
Revised 20 May 2026
Accepted 03 June 2026

Keywords:
Heart valve diseases;
Intestinal obstruction;
Laparotomy;
Anesthesia;
Case report

ABSTRACT

Valvular heart disease is a significant clinical factor associated with elevated perioperative cardiovascular risk. Current literature offers limited guidance on managing severe valvular heart disease in non-cardiac emergency surgeries. In this report, a 61-year-old male with sigmoid adenocarcinoma presents with hemodynamic instability due to partial gut obstruction, complicated by severe aortic and mitral stenoses from rheumatic heart disease. Echocardiography, electrocardiography, and blood tests were employed for preoperative planning. Emergency open abdominal surgery was done under combined regional and general anesthesia, employing techniques that maintained adequate preload and avoided heart rate extremes. Patient tolerated the procedure and the immediate postoperative course. In this case, anesthetic management incorporating effective fluid management, careful hemodynamic monitoring, and maintaining cardiac output is crucial in preventing acute cardiac decompensation.

Introduction

Valvular heart disease (VHD) is a major clinical predictor of increased perioperative cardiovascular risk [1]. Particularly, severe obstructive VHD, such as aortic (AS) or mitral stenosis (MS), can precipitate cardiac decompensation related to undesirable hemodynamics in response to anesthetic agents, surgical stress, perioperative fluid shifts, and intraoperative blood loss. Globally, VHD is most commonly caused by rheumatic heart disease (RHD), which often involves multiple valve issues (stenosis or regurgitation of two or more valves) and mixed VHD (combined stenotic and regurgitant lesions on the same valve) [2]. Despite the prevalence of RHD, limited data exist in current literature regarding clinical decision-making in patients with severe VHD, particularly with concomitant severe AS and MS, as the majority of existing reports focus on single-valve disease in the context of non-cardiac surgery and far less in acute emergency settings.

Case Report

The patient was a 61-year-old, 50-kg Filipino male diagnosed with sigmoid adenocarcinoma stage IIIB, with a three-year history of gradually increasing abdominal girth, vomiting, weight loss, and decreasing stool caliber. He was also known to have hypertension and RHD resulting in heart failure (HF), which accounted for his baseline exertional dyspnea, three-pillow orthopnea, and bipedal edema. He was maintained on the following oral medications: phenoxymethylpenicillin, atorvastatin, enalapril, spironolactone, and empagliflozin. He had no history of previous hospitalizations or surgery. The rest of his ancillary history was unremarkable save for paternal hypertension. The patient arrived at the

The authors declare no conflicts of interest.
*Corresponding author.
E-mail address: hrona@up.edu.ph
DOI:

emergency department (ED) complaining of severe generalized abdominal pain. He was hypotensive, tachycardic, and tachypneic. He exhibited a systolic murmur at the right parasternal border, second intercostal space, and a holosystolic murmur at the apex with axillary radiation. He also had abdominal distension with diffuse direct and rebound tenderness. Since partial gut obstruction from the sigmoid mass was considered, emergency surgery was deemed necessary. Fluid resuscitation with plain normal saline solution was done at the ED, improving blood pressure (BP) and heart rate (HR). Intravenous (IV) cefoxitin was administered before surgery.

The initial surgical plan was to perform a trephine distal loop colostomy. Preoperative blood investigations revealed anemia, elevated white blood cell count, severe hyponatremia, mild hypochloremia, mild hypoalbuminemia, and elevated prothrombin time. Respiratory alkalosis with secondary metabolic alkalosis was notable on arterial blood gas (ABG). Electrocardiography (ECG) showed sinus tachycardia and left ventricular (LV) hypertrophy. Chest radiography revealed multichambered cardiomegaly. Abdominal radiography demonstrated small bowel ileus. Echocardiography obtained a year prior to admission showed severe AS with moderate aortic regurgitation (aortic valve area [AVA] of 0.56 cm^2, peak systolic velocity [Vmax] of 5.4 m/s, and mean pressure gradient [ΔP] of 74.3 mmHg), severe mitral regurgitation (MR) with moderate MS (mitral valve area [MVA] of 2.08 cm^2), dilated left atrium (LA), concentric LV hypertrophy, high pulmonary hypertension probability, and ejection fraction (EF) of 64%. Cardiac point-of-care ultrasound (POCUS) on admission revealed an EF of 25-30%, LV wall thickening with global hypokinesia, LA enlargement, thickening of aortic cusps and anterior mitral valve leaflet with commissural fusion, and severe MS (MVA of 0.98 cm^2).

The patient had an American Society of Anesthesiologists (ASA) Physical Status classification of 4E for severe valve dysfunction and severely reduced EF for an emergency procedure. In the operating room, baseline vital signs were a BP of 125/60, an HR of 95 bpm, an RR of 20 cpm, and 100% oxygen saturation. The ECG displayed a normal sinus rhythm. Ultrasound-guided bilateral quadratus lumborum (QL) block was performed using isobaric bupivacaine 0.25% with epinephrine 1:400,000 and dexamethasone 4 mg (20 mL per side) with the patient in the lateral decubitus position. A second IV access was secured, and an arterial line was established. Airway topicalization was done using lidocaine 10% spray. After preoxygenation, general anesthesia was induced using IV etomidate 15 mg (0.3 mg/kg), rocuronium 60 mg (1.2 mg/kg), and fentanyl 100 mcg (2 mcg/kg). During intubation under videolaryngoscopy, HR remained at 90–99 bpm, BP at 90–100/50–60, and oxygen saturation at 100%, with no noted arrhythmias. Anesthesia was maintained with sevoflurane 2 vol% at 1:1 oxygen:air. Pressure-controlled ventilation was employed in relation to intra-abdominal and intrathoracic pressure changes, maintaining a tidal volume of 8 mL/kg. Crystalloids were prudently given while monitoring pulse pressure variation (PPV), blood loss, and urine output.

Low-dose norepinephrine infusion was utilized to help maintain systemic vascular resistance (SVR) and keep the mean arterial pressure (MAP) > 65 mmHg. Intraoperative hemodynamic status remained stable, with an HR of 80–90 bpm, MAP > 65 mmHg, PPV < 13%, end-tidal CO_2 ($EtCO_2$) of 35–38 mmHg, no arrhythmias, and no desaturations. Based on intraoperative findings of purulent ascites, ruptured appendicitis, and an obstructing sigmoid mass without bowel perforation, the surgical plan was converted to laparotomy with abscess evacuation, appendectomy, and proximal transverse loop colostomy. Estimated blood loss was 30 mL. Prior to the end of surgery, point-of-care testing (POCT) showed metabolic acidosis with a lactate of 1.09 mmol/L. Urine output was 0.9 mL/kg/h. On emergence, the patient was weaned off norepinephrine, extubated, and transferred to the post-anesthesia care unit (PACU). Postoperative pain was managed with IV paracetamol, ketorolac, tramadol, and the QL block. The patient remained hemodynamically stable with adequate pain control in the PACU. Postoperatively, laboratory examinations revealed mild anemia with a normal platelet count and normal serum sodium and chloride levels but still with hypoalbuminemia. He was cleared for forward transfer on postoperative day 2.

Discussion

Multiple VHD in low-to-middle-income countries is primarily caused by RHD, which is a sequela of pharyngitis or other GAS infections. The pathogenesis of RHD involves molecular mimicry, in which the M protein is the most implicated GAS antigen that activates an aberrant immune response due to the similarities between its α-helical structure and that of other proteins, such as those in cardiac valves [3]. The combination of mitral and aortic valve lesions is a common pattern in RHD; however, the concurrence of AS and MS specifically occurs least frequently compared to other bivalvular patterns [4–6]. In our patient, the glaring pathology was the coexistence of severe AS and MS with reduced EF. The patient's mitral valve lesion appeared to progress from regurgitation to stenosis as a product of commissural fusion from persistent valvulitis [7]. Valve hemodynamics of severe AS includes AVA ≤1.0 cm2, aortic Vmax ≥4 m/s, and mean ΔP ≥40 mm Hg, while severe MS can be defined by a MVA ≤1.5 cm^2 and diastolic pressure half-time ≥150 ms [8]. Concomitant

severe AS and MS is extremely poorly tolerated, largely due to the greater compromise in cardiac output compared to either lesion in isolation, since the effects of impaired LV diastolic filling due to MS and obstructed LV outflow due to AS are compounded [9].

Intraoperative hemodynamic goals in AS include avoiding preload reduction, systemic arterial hypotension, and extremes in heart rate and maintaining high-normal SVR, adequate contractility, and sinus rhythm to minimize myocardial oxygen demand and increase coronary perfusion time [10]. Similarly, hypotension, tachycardia, and arrhythmias are also avoided in MS. Promoting LV diastolic filling is essential in left heart stenotic lesions; vigilance for ECG changes, particularly atrial fibrillation, and maintenance of sinus rhythm, ideally with normal HR, are paramount for adequate atrial contraction and increased diastolic filling time. Since AS is considered preload-dependent, volume should be enough to fill the left atrium; however, fluids must be loaded with caution, especially with coexisting MS, due to potential drastic elevations in LA and pulmonary pressures that could induce right ventricular failure and pulmonary edema [11].

Further hemodynamic collapse can result from the volume-depleted state in PGO, contributed by emesis, reduced oral intake, and third spacing. Vomiting causes potassium, chloride, and hydrogen ion losses, often resulting in metabolic alkalosis. Decreased appetite and poor solute intake can cause hyponatremia and worsen dehydration. Increased intraluminal pressure can impair venous drainage and absorptive capacity, leading to bowel wall edema and ascites as fluids shift out of the intravascular space [12]. As such, preoperatively, our patient was administered crystalloids for fluid resuscitation, a nasogastric tube was inserted for gastrointestinal decompression, and started on broad-spectrum antibiotics for mitigating bacterial overgrowth.

ASA standard monitors (pulse oximeter, non-invasive BP monitor, ECG, capnograph, and temperature probe) [13] were employed intraoperatively. Since LA enlargement and high pulmonary arterial pressure in the setting of severe VHD were present, preventing further exacerbation of pulmonary hypertension was intended by avoiding hypoxemia, hypercarbia, and hypothermia. Invasive arterial BP monitoring was also established prior to anesthesia induction for real-time detection of changes in HR and BP, enabling careful titration of anesthetics. It also allowed for immediate assessment of PPV, which best predicts fluid responsiveness at >13%, and collection of blood samples for POCT. Severe VHD may be a limitation to PPV use in hemodynamic management; however, PPV has been shown to have a moderate predictive ability in goal-directed fluid therapy in valvular stenosis cases [14]. Moreover, dynamic variables similar to PPV have been successfully utilized in optimizing preload and cardiac output in the context of concurrent left-sided stenotic lesions [15]. In our case, IV fluids were provided judiciously, maintaining PPV <13%. Metabolic acidosis is often multifactorial. Here, it was likely contributed by the fluid shifts that tend to occur in open-cavity surgeries. Lactate, an important marker of cellular perfusion, was found to be <2 mmol/L, beyond which it suggests significant tissue hypoperfusion and predicts poorer prognosis [16].

For the anesthesia induction, etomidate was the chosen sedative. It is rapid and cardiostable, having minimal effects on HR and MAP and preserving SVR, central venous pressure, pulmonary artery pressure, and stroke volume [17]. As such, it is favorable in RSI in the critically ill [18]. Ketamine is another option in hemodynamically unstable patients due to its cardiovascular-stimulating properties; however, its negative inotropic effect may prevail in catecholamine-depleted states and in severe heart failure, resulting in cardiac output-related hemodynamic instability [19]. Although adrenal suppression is a concern, cortisol levels and the incidence of adverse events in patients given etomidate and in those given other common RSI drugs, such as propofol, midazolam, and ketamine, have not been found to be significantly different between the two groups [20].

To ensure optimal blunting of sympathetic response to intubation, opioids are administered as adjuncts to etomidate, which does not have analgesic effects [17]. Such a combination has safely attenuated sympathetic stimulation while preventing cardiovascular collapse during induction in patients with HF [21]. Topicalizing the airway also helps minimize the sympathetic surge from airway manipulation.

For paralysis, rocuronium has been recommended in patients with faster baseline HRs due to its potential vagolytic effect [22]. Succinylcholine can increase the risk of postoperative pulmonary complications [23] and potentially aggravate muscle injury in statin users [24] such as our patient.

Maintenance of anesthesia was safely achieved with sevoflurane. In patients with VHD, sevoflurane may offer a more notable attenuation of myocardial injury and inflammatory response than propofol-based anesthesia [25].

Major open abdominal surgeries are often associated with severe postoperative pain, which can increase opioid use and instigate chronic pain. While epidural analgesia as part of multimodal analgesia is effective in reducing surgical stress response, providing cutaneous and visceral pain relief, and decreasing postoperative ileus risk, its sympathetic blockade can cause significant hypotension from local anesthetic-induced SVR reduction [26]. Such a response is notably deleterious in VHD, although neuraxial techniques with incremental dosing have been used successfully [27]. Abdominal wall blocks offer an alternative method of affording pain control with the

advantage of producing a more limited effect on the sympathetic tone. QL block is performed by administering local anesthetic adjacent to the anterolateral aspect of the QL muscle and its fascia to provide analgesia for abdominal incisional and visceral pain. Compared to the more traditional transversus abdominis plane block, the QL block is superior in terms of dermatomal coverage, pain control duration, and opioid-sparing effect [28].

Conclusions drawn from this case are limited in their generalizability, given the relatively low strength of evidence of case reports. Case series or interventional studies are likely beneficial in demonstrating the safety and efficacy of such management in this cardiac patient population.

Conclusion

Managing severe multiple VHD in the setting of emergency non-cardiac procedures is uniquely complex due to the compounded challenges involved in the pathology of the surgical diagnosis and that of the underlying heart disease. Careful attention to hemodynamics and its response to surgical stress and anesthetic management is critical to avoid acute cardiac decompensation. An individualized, goal-directed anesthetic approach—prioritizing a cardiostable induction and maintenance of anesthesia, noninvasive and invasive monitoring strategies, and reliable multimodal analgesia—is essential to optimize outcomes in such a high-risk population.

References

[1] Eagle KA, Berger PB, Calkins H, Chaitman BR, Ewy GA, et al. ACC/AHA guideline update for perioperative cardiovascular evaluation for noncardiac surgery—executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Update the 1996 Guidelines on Perioperative Cardiovascular Evaluation for Noncardiac Surgery). J Am Coll Cardiol. 2002; 39(3):542-53.

[2] Unger P, Pibarot P, Tribouilloy C, Lancellotti P, Maisano F, et al. Multiple and Mixed Valvular Heart Diseases. Circ Cardiovasc Imaging. 2018; 11(8):e007862.

[3] Zhuang S, Guo D, Yu D. A mini review of the pathogenesis of acute rheumatic fever and rheumatic heart disease. Front Cell Infect Microbiol. 2025; 15:1447149.

[4] Laudari S, Subramanyam G. A study of spectrum of rheumatic heart disease in a tertiary care hospital in Central Nepal. Int J Cardiol Heart Vasc. 2017; 15:26-30.

[5] Manjunath CN, Srinivas P, Ravindranath KS, Dhanalakshmi C. Incidence and patterns of valvular heart disease in a tertiary care high-volume cardiac center: a single center experience. Indian Heart J. 2014; 66(3):320-6.

[6] Basnet K, Bhattarai SR, Shah S, Joshi A, Sah SK, et al. Rheumatic Heart Disease among Patients with Valvular Heart Disease Admitted to the In-patient Department of a Tertiary Care Centre: A Descriptive Cross-sectional Study. JNMA J Nepal Med Assoc. 2022; 60(249):419-24.

[7] Marijon E, Mirabel M, Celermajer DS, Jouven X. Rheumatic heart disease. Lancet. 2012; 379(9819):953-64.

[8] Otto CM, Nishimura RA, Bonow RO, Carabello BA, Erwin JP 3rd, et al. 2020 ACC/AHA Guideline for the Management of Patients With Valvular Heart Disease: Executive Summary: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Circulation. 2021; 143(10):e784.

[9] Unger P, Lancellotti P, de Cannière D. The clinical challenge of concomitant aortic and mitral valve stenosis. Acta Cardiol. 2016; 71(1):3-6.

[10] Samarendra P, Mangione MP. Aortic stenosis and perioperative risk with noncardiac surgery. J Am Coll Cardiol. 2015; 65(3):295-302.

[11] Frogel J, Galusca D. Anesthetic considerations for patients with advanced valvular heart disease undergoing noncardiac surgery. Anesthesiol Clin. 2010; 28(1):67-85.

[12] Jackson P, Vigiola Cruz M. Intestinal Obstruction: Evaluation and Management. Am Fam Physician. 2018; 98(6):362-7.

[13] Thompson JP, Mahajan RP. Monitoring the monitors--beyond risk management. Br J Anaesth. 2006; 97(1):1-3.

[14] Høiseth LØ, Hoff IE, Hagen OA, Landsverk SA, Kirkebøen KA. Dynamic variables and fluid responsiveness in patients for aortic stenosis surgery. Acta Anaesthesiol Scand. 2014; 58(7):826-34.

[15] Ng JH, Leonard I. Managing multiple valvular disease in spine surgery: A case report. JCA Adv. 2025; 2(2):100097.

[16] Riaz S, Mir AW, Sidiq S, Gurcoo SA, Shah MA, et al. Perioperative serum lactate as a predictor of post-operative length of hospital stay and in-hospital mortality in patients undergoing major emergency abdominal surgeries. Asian J Med Sci. 2024; 15(5):16-21.

[17] Valk BI, Struys MMRF. Etomidate and its Analogs: A Review of Pharmacokinetics and Pharmacodynamics. Clin Pharmacokinet. 2021; 60(10):1253-69.

[18] Pillay L, Hardcastle T. Collective Review of the Status of Rapid Sequence Intubation Drugs of Choice in Trauma in Low- and Middle-Income Settings (Prehospital, Emergency Department and Operating Room Setting). World J Surg. 2017; 41(5):1184-92.

[19] Bovill JG. Intravenous anesthesia for the patient with left ventricular dysfunction. Semin Cardiothorac Vasc Anesth. 2006; 10(1):43-8.
[20] Xia Z, Kamra K, Dong J, Harp KA, Xiong Y, et al. Comparison of efficacy and safety of etomidate with other anesthesia induction drugs for patients undergoing cardiac surgery: A systematic review and meta-analysis of randomized controlled trials. Heliyon. 2024; 10(22):e38274.
[21] Stockham RJ, Stanley TH, Pace NL, King K, Groen F, et al. Induction of anesthesia with fentanyl or fentanyl plus etomidate in high-risk patients. J Cardiothorac Anesth. 1987; 1(1):19-23.
[22] Virmani S, Tempe DK, Datt V, Tomar AS, Banerjee A, et al. Effect of muscle relaxants on heart rate, arterial pressure, intubation conditions and onset of neuromuscular block in patients undergoing valve surgery. Ann Card Anaesth. 2006; 9(1):37-43.
[23] Schaefer MS, Hammer M, Santer P, Grabitz SD, Patrocinio M, et al. Succinylcholine and postoperative pulmonary complications: a retrospective cohort study using registry data from two hospital networks. Br J Anaesth. 2020; 125(4):629-36.
[24] Turan A, Mendoza ML, Gupta S, You J, Gottlieb A, et al. Consequences of succinylcholine administration to patients using statins. Anesthesiology. 2011; 115(1):28-35.
[25] Yang XL, Wang D, Zhang GY, Guo XL. Comparison of the myocardial protective effect of sevoflurane versus propofol in patients undergoing heart valve replacement surgery with cardiopulmonary bypass. BMC Anesthesiol. 2017; 17(1):37.
[26] Nimmo SM, Foo ITH, Paterson HM. Enhanced recovery after surgery: Pain management. J Surg Oncol. 2017; 116(5):583-91.
[27] Brown J, Morgan-Hughes NJ. Aortic stenosis and non-cardiac surgery. Contin Educ Anaesth Crit Care Pain. 2005; 5(1):1–4.
[28] Liu X, Song T, Chen X, Zhang J, Shan C, et al. Quadratus lumborum block versus transversus abdominis plane block for postoperative analgesia in patients undergoing abdominal surgeries: a systematic review and meta-analysis of randomized controlled trials. BMC Anesthesiol. 2020; 20(1):53.