Anesthetic management of a multiple and mixed cardiac valvular disease for noncardiac surgery: a case report

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A thorough knowledge of pathophysiology of multiple and mixed valvular heart disease is a requirement for a stable perioperative care. We present anesthetic management of a 62-year male patient with bivalvular mixed lesions with gastric adenocarcinoma for gastrectomy. Fixed-output cardiac state, anemia, maintenance of hemodynamic goals for longer duration, and postoperative analgesia were the anesthetic concerns. Hemodynamic aberrations can be inevitable in such scenario. Proper understanding of the lesions, optimization, preparation, and planning for anticipation of adverse hemodynamic events plays a crucial role for expecting a better clinical outcome.

Keywords:
anesthetic challenges, analgesia, fixed cardiac output, hemodynamic goals, multiple valvular cardiac disease

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Introduction

Valvular heart disease (VHD) can present as multiple or mixed-spectrum lesions in healthcare settings. A thorough understanding of the pathophysiology of VHD is essential for planning anesthesia and perioperative care. We describe a male patient with mitral and aortic valvular mixed lesions secondary to Rheumatic heart diseaese with adenocarcinoma stomach posted for gastrectomy.

Anesthetic concerns were fixed cardiac-output state, longer-duration surgery and difficult airway challenging the maintenance of hemodynamic goals, analgesia, blood loss with preexisting severe anemia, and added challenge of managing with biohazard state.

Case presentation

A 62-year male with complaints of melena and easy fatiguability was diagnosed to have adenocarcinoma of the stomach and posted for laparoscopic distal gastrectomy. He was a known case of rheumatic fever on penicillin prophylaxis since teenage. He was diagnosed to have valvular disease following syncopal attacks 7 years back and was on treatment with Pencillin G, diuretics, and Angiotensin-converting enzyme inhibitors. He experienced breathlessness on exertion (New York Heart Association 2), with effort tolerance of two metabolic equivalents.

He was a chronic smoker with past history of pulmonary tuberculosis and had completed the course of anti-tubercular treatment. He was hepatitis B antigen positive.

On examination, the airway was difficult with Mallampatti grade 3 and restricted neck extension.

Preoperative hemoglobin was 6.5 g/dl and was optimized with two units of packed red blood cells and hemoglobin improved to 9.3 g/dl. Chest radiograph showed cardiomegaly. Transthoracic echocardiography revealed dilated left atrium with moderate mitral stenosis and mild mitral regurgitation with calcified posterior mitral leaflet and mitral valve area –1.6 cm^2 with mitral valve peak pressure gradient/mean pressure gradient –24/10 mmHg. There was moderate aortic stenosis with moderate aortic regurgitation with aortic valve area –1.6 cm^2 and AV PPG/MPG –63/38 mmHg, there was mild pulmonary artery hypertension of 48 mmHg with ejection fraction of 60%.

The patient was accepted under American Heart Association 4 with intermediate cardiac risk. Physiological changes like decreased preload, sympathetic stimulation with carbon dioxide, and peritoneal inflation with laparoscopy could adversely have concerns in patients with valvular heart diseases, so a change in surgical plan to laparotomy was done. A

written informed high-risk consent was taken. Clexane 40 mg subcutaneous was started 12 h prior to the surgery.

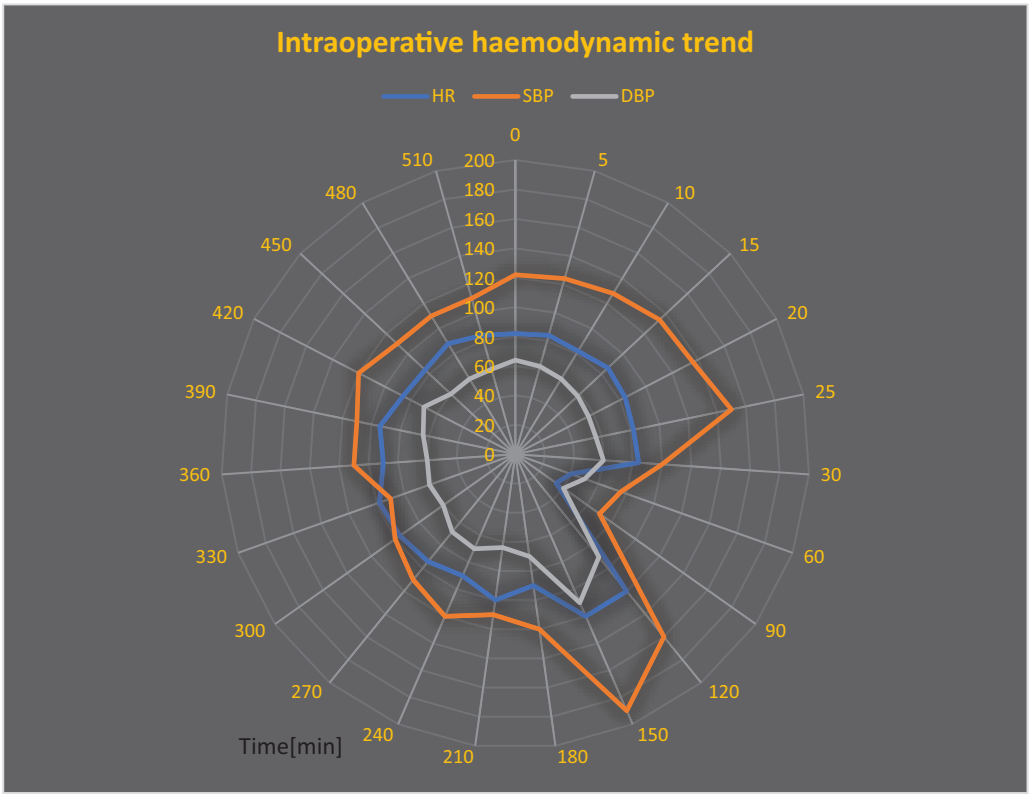
General anesthesia with thoracic epidural with invasive monitoring was planned. Emergency cardiac drugs and defibrillator were prepared.

Baseline blood pressure and heart rate were 130/80 mmHg and 86/min, respectively. His SpO_2 was 96% while breathing room air and ECG displayed normal sinus rhythm. Under universal precautions, a 16 G intravenous access was secured and thoracic epidural was placed in sitting position at T5–T6 level. Left radial arterial line and ultrasound-guided right internal jugular vein triple-lumen central catheter were secured.

Antibiotics Piperacillin and Tazobactam combination 4.5 g and Metronidazole 500 mg intravenous were given before induction.

Intravenous midazolam 1 mg was given and 15% lignocaine oral spray was done to reduce the intubation stress response. After preoxygenation, coinduction was done with sevoflurane 2% and titrated doses of fentanyl up to 5 μg/kg. Intubation was done under C-MAC guidance with succinylcholine 1.5 mg/kg. Postintubation, there was an episode of hemodynamic instability manifesting as hypotension of 78/44 mmHg, which was treated with Ringer lactate 200 ml and intravenous phenylephrine 100 μg, but it was followed by sudden severe sinus bradycardia of 35 bpm, unresponsive to atropine 0.6 mg, therefore, a bolus of intravenous adrenaline 100 μg was administered. This resulted in hypertension of 200/100 mmHg and tachycardia of 120 bpm, which was rapidly treated with aliquots of esmolol and metoprolol and stabilized. Maintenance of anesthesia was done with O_2 : air : sevoflurane, cisatracurium, and fentanyl 0.5 μg/kg. A low dose of noradrenaline infusion 0.05–0.1 μg/kg/min was required throughout the surgery. Titrated doses of metoprolol and esmolol were used to maintain the heart rate of 80–90 bpm and blood pressure in the range of 120/70 mmHg intraoperatively (Fig. 1). Fluids were judiciously given with a colloid and two pints of PRBCs and fresh frozen plasma were administered, monitoring pulse-pressure variations, central venous pressure, and urine output. Intraoperative hemodynamic status was stable. The patient was extubated and shifted to the ICU for monitoring, and postoperative analgesia was managed with fentanyl infusion 1 μg/kg/h and paracetamol 1 g

Figure 1



Intraoperative hemodynamic trend.

Q8H. Epidural activation was not done as the patient required noradrenaline infusion 48 h postoperatively. The patient was shifted to the ward after 48 h of monitoring in the ICU without any complications and did not require further transfusions.

Discussion

The prevalence of VHD is 2.5% in industrialized countries. The risk of perioperative outcomes is increased with associated aortic stenosis [1], cardiac failure, and arrhythmias. Thorough knowledge of pathophysiology, preoperative optimization, and monitoring can give a favorable clinical outcome [2].

Multiple VHD constitutes both stenotic and regurgitant lesions on more than or equal to two cardiac valves, whereas mixed VHD are stenotic and regurgitant lesions on the same valve. Rheumatic heart fever is the predominant cause in 51% and degenerative pathogenesis in others (41%). Other less frequent causes are infective endocarditis, radiation therapy, inflammatory diseases, and drug-induced VHD.

Multiple and mixed VHD have clinical and hemodynamic implications. It depends on the complex role of several factors like specific combination of VHD, severity of each lesion, the loading conditions, and the ventricular systolic and diastolic function.

Reduction in cardiac output is greater with combined aortic and mitral stenosis than isolated stenotic lesions on these valves. Aortic and mitral pressure gradients can be lower and can lead to underestimation of the severity of stenotic lesions on these valves [3].

The proximal valve disease can mask the severity of the distal valve disease. Predominant clinical features are caused by the hemodynamically more severe lesion, if similar degrees of valvular severity are assumed, symptoms are related to the disease of the upstream valve.

Symptoms of cardiac failure are present due to hemodynamic effects on cardiac chambers in multivalvular diseases [4].

Moderate-to-severe AS is associated with anemia because of acquired coagulopathy, gastrointestinal bleeding due to angiodysplasia (Heyde's syndrome) [5], and additionally added to this was ongoing bleeding from adenocarcinoma of the stomach in our case.

Bivalvular stenotic lesions were considered dominant in our case, so the goals were to maintain the heart rate of 80–90 bpm as higher heart rates are allowed in patients with aortic insufficiency [5] and sinus rhythm, optimizing the contractility, maintaining the preload and afterload. Maintenance of 'cardiac output' was significant in our case. Hemodynamic instability in our patient postintubation can be reasoned due to the combined effect of phenylephrine, fentanyl, and succinylcholine from delayed onset of action of drugs due to stenotic lesions or from suppression of sympathetic stimulation. Our patient required low-dose noradrenaline support to maintain the afterload intraoperatively and postoperatively. Laparoscopic procedures, which increase the intraabdominal pressures, are at high risk for perioperative cardiac adverse events [6]. So a change in surgical plan to do laparotomy was made in our case after discussion with surgeons. Anemia was another factor in our case increasing the chances of adverse events and it was corrected promptly preoperatively and intraoperatively. C-MAC helped to prevent the hemodynamic perturbance by avoiding multiple attempts for intubation. Maintaining the cardiac goals for longer duration of time was a challenge.

Although the guidelines are clear for single-valve disease, there is no common recommendation for management of multiple VHDs [7].

Use of cardiac monitoring and consideration of regional anesthesia like paravertebral blocks or erector spinae block can add further benefits in management of patients with VHD.

A meticulous planning, interdepartmental optimization, and discovering the dominant lesions are crucial to plan anesthesia management for a multiple and mixed VHD case for noncardiac surgery.

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Conflicts of interest

There are no conflicts of interest.

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