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Case Report

Anaesthesia Section

Anaesthetic Management of Multivalvular Surgery Complicated by Submitral Aneurysm and Pulmonary Hypertension: A Case Report

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ABSTRACT

Submitral Aneurysm (SMA) is a rare cardiac condition that presents with perioperative anaesthetic complications, especially with comorbidities such as Pulmonary Hypertension (PH) and severe valvular disease. The case reported is a 21-year-old female with a medical illness history of rheumatic heart disease, severe mitral and Tricuspid Regurgitation (TR), PH, and basal SMA. She was admitted with palpitations, dyspnoea, and cough. Preoperative assessment revealed ventricular dysfunction and pulmonary disease. She was to undergo general anaesthesia for Mitral Valve Replacement (MVR), tricuspid valve repair, and SMA repair. Anaesthesia management included maintaining cardiac output, preserving right ventricular function, and controlling pulmonary pressures. Invasive monitoring and intraoperative echocardiography were used. Postoperative intensive care unit management was carried out, and ventilatory support was weaned on the 2nd postoperative day. The postoperative course was uneventful, with enhanced functional capacity and satisfactory echocardiographic parameters at follow-up. The case highlights the complexity of anaesthetic care for multivalvular heart disease with SMA and PH. It underscores the need for personalised treatment, precise monitoring, and multidisciplinary care for successful management.

Keywords: Cardiac anaesthesia, Cardiac surgery, Mitral regurgitation, Tricuspid regurgitation

CASE REPORT

A 21-year-old female, weighing 43 kg and with a height of 160 cm {Body Mass Index (BMI) 16.8 kg/m^2} presented with a one-month history of palpitations, worsening exertional dyspnoea, and non-productive cough. She had a known history of rheumatic heart disease for 17 years. She did not complain of chest pain, syncope, or fever. At presentation, the patient was tachycardic with a heart rate of 108 beats/min, blood pressure of 96/60 mmHg, respiratory rate of 24 breaths/min, and oxygen saturation of 94% on room air. Electrocardiography revealed sinus tachycardia without ischaemic changes. Functional status was New York Heart Association class IV, with dyspnea even at rest. On examination, the patient was dyspneic at rest. Her dyspnea corresponded to modified Medical Research Council grade 4. Jugular venous pressure was elevated, and bilateral pitting pedal oedema was present. Cardiovascular examination revealed a pansystolic murmur at the apex radiating to the axilla. Abdominal examination revealed mild splenomegaly. Respiratory examination revealed bibasilar crepitus. Airway examination was within normal limits with good mouth opening, Mallampati class II, normal neck movement, and satisfactory thyromental distance.

Laboratory findings revealed haemoglobin of 11.1 g/dL, absolute leucocyte count of 16,300/mm^3, and platelet count of 1.85×10^5/mm^3. Liver and renal function tests were normal. High-resolution thoracic computed tomography revealed cardiomegaly, pulmonary oedema, and fibrous bands in the right upper, middle, and lower lobes and left upper lobe. Echocardiography was indicative of an SMA, severe Mitral Regurgitation (MR), severe TR, severe PH, global left ventricular hypokinesia, and a Left Ventricular Ejection Fraction (LVEF) of 42%.

Quantitative echocardiographic assessment demonstrated severe PH, with an estimated pulmonary artery systolic pressure of approximately 72 mmHg and a right ventricular systolic pressure of approximately 75 mmHg. Right ventricular function was moderately impaired with a tricuspid annular plane systolic excursion of 14 mm and mild right ventricular dilatation. The SMA measured approximately 4.2×3.6 cm and was located along the posterior mitral annulus adjacent to the posterior mitral leaflet. The aneurysm communicated with the left ventricle through a neck measuring approximately 1.2 cm in width. No intraluminal thrombus was visualised within the aneurysmal sac on echocardiographic assessment. The patient was classified as American Society of Anaesthesiologists (ASA) physical status IV.

She was scheduled for MVR, tricuspid valve repair, and SMA repair. For preoperative optimisation, the following was done: Cautious use of diuretics with intravenous furosemide 20 mg twice a day to relieve pulmonary congestion; Continued metoprolol 12.5 mg once a day; and maintain euvolaemia. Supplemental oxygen was administered to achieve an oxygen saturation above 96%. The patient's packed red blood cells were cross-matched to anticipate intraoperative blood loss and possible haemodilution during Cardiopulmonary Bypass (CPB). The standard ASA monitoring devices were used intraoperatively. The induction of anaesthesia was with intravenous fentanyl 100 µg, midazolam 2 mg, and propofol 70 mg, using vecuronium 6 mg for paralysis prior to intubating the patient with a 7.5 mm cuffed endotracheal tube. Vascular access was achieved with a 7 Fr right internal jugular central line and a 20-gauge radial arterial line.

Anaesthesia was achieved through inhalation of isoflurane gas mixed with air. It was supplemented once during this period with analgesic boluses (50 µg) of fentanyl intermittently plus additional use of vecuronium boluses (1 mg). Ventilation followed a lung-protective approach, using tidal volumes of approximately 6-7 mL/kg and a rate of 14-16 breaths/min, with a positive end-expiratory pressure of approximately 4 cm H_2O. To avoid hypercarbia due to elevated end-tidal CO_2 concentrations, mechanical ventilation rates were adjusted as needed to maintain an end-tidal CO_2 level between 35 mmHg and 38 mmHg. Fraction of inspired oxygen was regulated between 0.5 and 0.6 to maintain arterial oxygen saturation >98% while avoiding hypoxia and/or further increases in pulmonary vascular resistance.

The surgery was performed using CPB with systemic heparinisation (300 IU/kg) and cold blood cardioplegia for cardioplegic arrest. The total time on CPB was approximately 128 minutes, with aortic cross-clamp time of 92 minutes. During surgery, the patient required light inotropic support from norepinephrine (0.05-0.1 µg/kg/min) and milrinone (0.3-0.5 µg/kg/min) to maintain haemodynamic stability. The mitral valve was replaced with a mechanical prosthesis; the tricuspid valve was corrected with an annuloplasty ring; and the SMA was successfully removed and repaired. After surgical repair and replacement of the valve, Transesophageal Echocardiography (TEE) showed that the mechanical mitral prosthesis was appropriately seated, with no paravalvular leak; trace TR; and that the SMA repair was intact. There was an increase in the strength of the muscle contraction in the ventricle. Weaning from the CPB was assisted by gradual rewarming, preload optimisation, and right-sided heart support with milrinone and norepinephrine to decrease pulmonary vascular resistance. After weaning from the bypass, the patient had stable haemodynamics and good urine output.

After surgery, the patient was moved into the intensive care unit, where she was intubated and mechanically ventilated, with sedation maintained using midazolam at 0.02-0.05 mg/kg/h and analgesia via fentanyl at 1-2 µg/kg/h. The inotropic medications were slowly taken off over 24 hours. The patient was extubated without complications on postoperative day 2, after meeting the usual extubation criteria. The patient had stable haemodynamics, was on 2 L/min of oxygen via nasal cannula, and was alert and oriented. On postoperative day four, she was moved to a regular nursing unit, where she was discharged on postoperative day 10. She was stable with normal vital signs and discharged on anticoagulation therapy, beta-blockers, and diuretics.

During both her one- and three-month follow-up visits, the patient did not complain of shortness of breath, difficulty keeping up with physical activity, or irregular heartbeats. A follow-up echocardiogram showed a well-functioning prosthetic mitral valve, minimal to mild residual TR, and an LVEF of 50% to 52% without residual aneurysms or signs of heart failure.

DISCUSSION

Anaesthetic care of valvular heart disease, particularly of rheumatic aetiology, is complicated because of the complex interrelationship between compromised cardiac performance, PH, and systemic circulation haemodynamic compromise. Anaesthesia requires careful preplanning and technique to avoid complications and obtain optimal benefit [1,2]. In the present case, the coexistence of severe MR, severe TR, PH, and a large SMA created significant perioperative challenges. The anaesthetic goals were to maintain forward cardiac output, prevent increases in pulmonary vascular resistance, and preserve right ventricular function.

The MR leads to volume overload of the left ventricle, while TR leads to volume overload of the right ventricle. Regurgitant volume adds to left ventricular end-diastolic volume in MR, potentially deleterious to left ventricular function and reduced ejection fraction. In TR, right ventricular volume loading can be deleterious to right ventricular function and elevate systemic venous pressure. Atrioventricular synchrony must be maintained during surgery because atrial fibrillation increases volume load and reduces cardiac output. The treatment includes the administration of inotropes, such as milrinone, to enhance myocardial contractility, and pulmonary vasodilators, such as sildenafil, to reduce right ventricular afterload [3]. In the present case, milrinone and norepinephrine were used intraoperatively to support right ventricular function and maintain systemic perfusion during weaning from CPB. Similar pharmacologic strategies have been described by Kalbande JV et al., who reported the use of dobutamine and noradrenaline following repair of a congenital SMA with severe MR [4]. Likewise, Sun P described a patient with severe valvular disease and PH in whom aggressive haemodynamic support and early institution of CPB were required to prevent rapid circulatory deterioration [5]. These reports highlight the importance of targeted inotropic support and maintenance of ventricular function in patients with combined valvular lesions and SMA [4,5].

PH is challenging to control using anaesthetics because it compromises the right ventricular function. Anaesthetic drugs and methods that elevate pulmonary vascular resistance can cause acute right heart failure. To avoid this complication, anaesthetic care is planned preoperatively maintain or optimise preload while reducing afterload and ensure maximum right ventricular perfusion. Selective lowering of pulmonary vascular resistance can be achieved with inhalational anaesthetics, such as nitric oxide, or with intravenous prostacyclins, without affecting systemic vascular tone. To detect early decompensation, right ventricular function can also be monitored invasively using echocardiography and invasive haemodynamic monitoring [6,7]. In our patient, ventilation was carefully managed with controlled tidal volumes, minimal positive end expiratory pressure, avoidance of hypercarbia, and maintenance of adequate oxygenation to prevent increases in pulmonary vascular resistance. In the current case the patient's low BMI (16.8 kg/m^2) was also considered in the preoperative risk assessment. Sun et al. also highlighted the use of inhaled nitric oxide to selectively reduce pulmonary vascular resistance during surgery, while maintaining systemic vascular tone. In contrast, in the present case, pulmonary vasodilation was achieved pharmacologically with milrinone, without the need for inhaled nitric oxide [5].

Induction of anaesthesia in patients with life-threatening valvular heart disease requires drugs capable of opposing adverse haemodynamic changes. Propofol, although extremely favoured, causes vasodilatation and hypotension and hence should be titrated with great care. Opioids such as fentanyl cause analgesia and suppress sympathetic activity. Muscle relaxants such as vecuronium are used for intubation and surgical exposure on a weight- and renal function-corrected basis. Maintenance of anaesthesia is best achieved through the application of inhalation anaesthetic drugs such as isoflurane or sevoflurane supplemented with intravenous opioids as an adjunct for maintenance of anaesthesia to a proper comfort level. Monitoring must be closely performed to diagnose and treat perioperative complications [2,8]. In the present case, induction with fentanyl, midazolam, propofol, and vecuronium followed by maintenance with isoflurane and intermittent fentanyl provided stable intraoperative haemodynamics. Butiyani P and Kapuriya J described the use of etomidate-based induction in a patient with severe MR and markedly reduced ejection fraction undergoing laparoscopic cholecystectomy, emphasising the importance of maintaining stable haemodynamics in the setting of compromised ventricular function. Kalbande JV et al., reported a morphine-based anaesthetic technique with titrated propofol and sevoflurane in a patient undergoing surgical repair of SMA, demonstrating that opioid-based anaesthesia can provide favourable haemodynamic stability in such high-risk patients [4,9].

Ongoing intra-arterial blood pressure monitoring using invasive arterial lines and fluid resuscitation using central venous catheters to enable tracking of right atrial pressure complements basic ASA monitoring. TEE provides real-time cardiac anatomical and functional assessment and is particularly valuable for evaluating valve function and ventricular performance during cardiac surgery. During intraoperative care, special attention is given to maintaining optimal cardiac output, careful fluid management, and avoiding arrhythmias. Postoperatively, these patients require close monitoring in the intensive care unit, with careful titration of inotropes, fluid balance management, and early initiation of anticoagulation in patients receiving mechanical prosthetic valves [10-12]. Kalbande JV et al., highlighted the crucial role of TEE in identifying aneurysmal anatomy and guiding surgical repair. Wani Z and Sharma M described an elderly patient with a multilocular SMA compressing the right pulmonary artery, in whom complex

intraoperative findings and postoperative arrhythmias contributed to a high-risk clinical course. Compared with these cases, the present patient demonstrated a favourable perioperative recovery with stable haemodynamics, successful aneurysm repair, and early postoperative recovery [4,12].

Several anaesthetic challenges are encountered in patients with SMA and severe valvular disease. These include maintaining forward cardiac output in the presence of significant regurgitant lesions, preventing increases in pulmonary vascular resistance in patients with PH, preserving right ventricular function, minimising the risk of arrhythmias due to atrial or ventricular dilatation, and the potential for sudden haemodynamic collapse during induction or CPB weaning. The presence of an SMA further complicates surgical and anaesthetic management due to distortion of the mitral annulus and risk of rupture or thromboembolism [4].

Alternative anaesthetic strategies have been described in the literature. Etomidate-based induction has been preferred due to its minimal cardiovascular depression, particularly in patients with severely reduced ventricular function. Similarly, inhaled nitric oxide or prostacyclin analogues may be used for selective pulmonary vasodilation in cases of severe PH. In the present case, these strategies were not employed because the patient maintained acceptable ventricular function and pulmonary haemodynamics with the chosen anaesthetic technique and pharmacologic support. Therefore, a balanced anaesthetic technique using opioid based induction, volatile anaesthetic maintenance, and targeted inotropic support was considered appropriate and resulted in a favourable perioperative outcome [5,7,13].

[Table/Fig-1] provides a comparative overview of anaesthetic and surgical strategies in managing valvular pathology and SMA across reported cases [4,5,9,12].

Authors	Case description	Anaesthetic/surgical management	Key takeaways
Current case	21/F with severe MR/TR, PH, SMA. Underwent MVR, tricuspid repair, and SMA excision	Fentanyl, midazolam, propofol, and vecuronium for induction; isoflurane and fentanyl for maintenance. Milrinone and norepinephrine were used for right ventricular support	-Avoid an increase in peripheral vascular resistance -Use inotropes judiciously -Early extubation favoured
Sun P [5]	54/F with severe mitral stenosis/ TR, PH, and haemothorax. Emergency thoracotomy and double valve surgery.	Etomidate, sufentanil, and rocuronium induction; Sevoflurane and remifentanil maintenance. Inhaled nitric oxide and early CPB.	- Etomidate preferred - Inhaled Nitric oxide helps reduce peripheral vascular resistance - Early CPB avoided deterioration
Butiyani P and Kapuriya J [9]	65/M with severe MR, LVEF 20-25%. Underwent laparoscopic cholecystectomy.	Etomidate, fentanyl, vecuronium induction; Sevoflurane maintenance.	- Severe MR managed safely with precautions - Low-pressure pneumoperitoneum is essential - Regional block reduced opioid need
Kalbande JV et al., [4]	27/M with congenital SMA and severe MR. Underwent aneurysmorrhaphy and MVR.	Morphine, titrated propofol, vecuronium; Sevoflurane, TEE guided surgery. Dobutamine and noradrenaline post-CPB.	- TEE is crucial for diagnosis and intraoperative guidance - The morphine-based technique is haemodynamically stable - Postoperative anticoagulation needs monitoring
Wani Z and Sharma M [12]	89/F with long-standing dyspnea, SMA compressing the right pulmonary artery. Underwent surgical repair with a double pericardial patch and bypass.	Surgical complications included LAD injury, bypass grafting, and refractory arrhythmias.	- Elderly with SMA poses a high surgical risk - Post-op arrhythmias can be fatal - Multilocular SMA with thrombus adds complexity

[Table/Fig-1]: Anaesthetic and surgical management of valvular pathology and SMA [4,5,9,12].
MR: Mitral regurgitation; TR: Tricuspid regurgitation; PH: Pulmonary hypertension; SMA: Sub-mitral aneurysm; MVR: Mitral valve replacement; LVEF: Left ventricular ejection fraction; CPB: Cardiopulmonary bypass; NO: Nitric oxide; TEE: Transesophageal echocardiography; LAD: Left anterior descending artery.

CONCLUSION(S)

This case highlights the successful anaesthetic management of a young patient with SMA and severe MR, TR, and PH. Careful preoperative optimisation, vigilant intraoperative monitoring, and a balanced anaesthetic technique helped maintain stable haemodynamics and prevent worsening of PH. Particular attention was directed toward preserving right ventricular function, avoiding increases in pulmonary vascular resistance, and ensuring adequate systemic perfusion. TEE and judicious inotropic support played an important role in guiding surgical repair and facilitating safe separation from CPB. This case emphasises that meticulous perioperative planning and multidisciplinary coordination are essential for achieving favourable outcomes in complex valvular heart disease with SMA.

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