Case Report

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Anesthetic Management of Severe Mitral Stenosis with Left Atrial Clot in a Known Case of Hypothyroidism with Undetected Obstructive Sleep Apnoea Posted for Mitral Valve Replacement

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Abstract

A 55 year old, diabetic, hypertensive female patient diagnosed with rheumatic heart disease and severe mitral stenosis was posted for mitral valve repair surgery. She was a known case of hypothyroidism with seizure disorder. On 2D echo, she had a severely dilated left atrium and a moderate degree of pulmonary hypertension (RVSP 48 mm Hg) She was found to have a left atrial clot on the Trans Esophageal Echocardiography (TEE) performed prior to surgery. On clinical examination, the patient was obese and a candidate for difficult airway. Her pulse was irregularly irregular suggestive of atrial fibrillation which was confirmed on ECG. Patient was taken up for surgery under high-risk with all surgical and anesthetic concerns explained to the family. Although there were no events intraoperatively, patient was difficult to wean off the ventilator postoperatively with one episode of cardiac arrest during a weaning trial. Patient was successfully revived after CPCR as per AHA guidelines. After an elective tracheostomy was performed, various causes of the sudden ventilator dependency that were ruled out included cor pulmonale owing to pulmonary hypertension, an undetected preoperative obstructive sleep apnoea, embolization of the clot to the brain, and chronic hypothyroidism leading to respiratory muscle weakness.

Keywords: Mitral stenosis; Obesity; Hypothyroidism; Obstructive sleep apnoea; Pulmonary hypertension, Cor pulmonale, Respiratory failure.

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Introduction

Mitral stenosis is one of the most common cardiac anomalies associated with rheumatic heart disease.[1] The normal area of the mitral valve is 4–6 cm^2 with severe mitral stenosis detected when mitral valve area is lesser than 1 cm.[2] Symptoms of mitral stenosis vary with the severity of disease. Milder forms of the disease often tend to go unnoticed since patients are often asymptomatic till the degree of stenosis increases. With progression of the disease, patients start complaining of breathlessness with minimal exertion or even at rest. The decrease in mitral valve surface area reduces left ventricular filling, causes stasis of blood in the left atrium and increase in left atrial pressures which eventually lead to left

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atrial dilatation and enlargement which can often be seen on chest radiogram as straightening of the left heart border. The increase in left atrial pressures cause a subsequent increase in pulmonary vascular resistance leading to pulmonary hypertension and pulmonary oedema in severe cases. The stasis of blood in the left atrium can lead to the formation of a left atrial thrombus. In cases that are associated with atrial fibrillation, there is always a risk of embolization of this thrombus into the pulmonary or cerebral circulation.

The goal of anesthetic management in such cases is to achieve rhythm control and prevent tachycardia, hypoxia and hypotension intraoperatively along with judicious use of intravenous fluids.

Patients with obesity and increased body mass index[2] (normal BMI 18.5–24.9 kg/m^2) pose a further challenge to case management. Apart from being difficult airway candidates,[3,4] such patients may have an undetected obstructive sleep apnoea which often complicates the postoperative period. Obstructive sleep apnoea is a chronic disorder leading to partial or complete collapse of the upper airway during sleep with a consequent reduction in airflow. Postoperatively, these patients tend to have increased propensity for airway collapse impairing gas exchange and respiratory function.[5]

Long standing hypothyroidism when combined with the above comorbidities increases the perioperative risk of surgery.[7,8] When the hypothyroidism is not well controlled it leads to a number of complications, mainly by decreasing the body's metabolism of induction agents and causing respiratory muscle weakness hence making postoperative weaning from ventilator difficult.[9]

Case Report

A 55 year old female patient with RHD and severe mitral stenosis was posted for mitral valve replacement. She was a known case of hypertension, diabetes, hypothyroidism and seizure disorder for the last 10 years, on medication for all of the above.

She gave a history of previous hospitalization one month ago, for heart failure and pulmonary oedema wherein she was detected with severe mitral stenosis following rheumatic heart disease. She had since been started on anticoagulation therapy which had now been switched from Warfarin to unfractioned Heparin for the surgery.

She continued to have breathlessness and fatigue on minimal exertion post the hospitalization and hence decided to undergo the surgery as advised. On clinical examination, patient was obese with BMI of 30 kg/m.[2] She had a Mallampati score of three and a thyromental distance of 2 finger breadths and a short neck, making her a difficult airway candidate. Her pulse was irregularly irregular pattern which was suggestive of atrial fibrillation. Her heart rate and blood pressure were within normal limits.

The patient's routine blood work was within normal limits with TSH value of 9.9 mcg/dl. Echocardiography of the patient confirmed the diagnosis of atrial fibrillation with a controlled rate. The 2d echo showed severe dilatation of the left atrium with severe pulmonary artery hypertension (48 mm Hg). A Trans oesophageal Echocardiography (TEE) revealed a left atrial clot (3 × 3.8 cm).

High-risk consent was obtained preoperatively and the patient along with her relatives was explained the risk of anesthesia and surgery owing to her comorbid conditions and the risk- associated with embolization of the clot. The patient was instructed to take her antithyroid and antiepileptic medications as per schedule and avoid all hypoglycemic agents before surgery.

Preoperative vitals were within normal limits, random blood sugar was normal and lungs were clear. A crash cart with all emergency drugs, defibrillator and transcutaneous pacing wires were kept on standby. Calculated infusions of ionotropic and vasopressor agents were made for intraoperative hemodynamic management. Prior to induction, a large bore IV cannula was secured. An arterial line connected to a transducer was inserted in the Rt femoral Artery to monitor fluctuations in blood pressure throughout the surgery. A central venous catheter was secured in the Rt internal jugular vein for monitoring fluid status with calibrated guidewire insertion to avoid arrhythmias and clot embolization.

Patient was preoxygenated using 4 litres of oxygen. Premedication was administered with Inj Midazolam 5 mg and Inj Fentanyl 200 ug intravenously in titrated doses. General anesthesia was administered using Inj Etomidate 20 mg IV slowly over 30 mins to avoid a sudden fall in blood pressure. Succinylcholine 75 mg was given as muscle relaxant of choice and Train of Four (TOF) count was monitored. Patient was intubated after the count reached 0.

Inj Noradrenaline (3/50) was started at a low dose postinduction through the CVC to maintain a target MAP of 70 mm Hg and BP was monitored continuously *via* the arterial line using a transducer.

The patient was kept on an infusion of Inj Atracurium, Midazolam and Fentanyl to maintain the plane of anesthesia along with Sevoflurane as inhalational agent of choice. Postoperatively, the patient was kept intubated and ventilated with ionotropic support overnight. The patient's respiratory efforts remained unsatisfactory the next day due to which weaning from the ventilator was delayed. Extubation was performed 2 days postoperatively after patient had regained motor power and responded to commands satisfactorily. Postextubation, the patient complained of breathlessness and was unable to maintain saturation in spite of supplemental oxygenation.

Bipap ventilation was started following which the patient exhibited decreasing levels of consciousness with serial ABGs showing an increasing trend of hypercapnia. Patient was then electively intubated and put back onventilatory support. Over the next several days, ventilatory settings were changed based on the serial ABGs of the patient and weaning was attempted gradually.

After several unsuccessful weaning attempts over the next 7 days, the patient was scheduled for an elective tracheostomy owing to prolonged need for intubation. Post the tracheostomy, the patient remained on ventilatory support. Weaning attempts were unsuccessful with the patient developing tachycardia and hypoxia if taken off ventilator during one such weaning trial, the patient went into cardiac arrest. The patient was revived successfully after 2 cycles of high quality CPCR as per AHA guidelines. Weaning attempts were then witheld for a few days. Patient was hemodynamically stable postresuscitation and showed no signs of organ damage.

An Indirect laryngoscopy (IDL) performed by the ENT team of doctors showed mobile and functional vocal cords ruling out vocal cord lesion/palsy as a cause of her respiratory distress. On retesting her blood samples, her TSH had increased to 12 mcd/dl. The patient's medications were reviewed by the medicine team and her dose of Thyroxine was increased.

Weaning attempts were restarted after a few days and the patient was given rigorous chest physiotherapy and a high protein diet to increase respiratory muscle strength. Eventually, the patient showed improved ABGs with an acceptable oxygen saturation with 6 litres of oxygen *via t*-piece which was then gradually changed to room air. Serially improving blood gas reports and a better level of consciousness made the patient a candidate for discharge. The patient was sent home after being under observation for a week, with an elective tracheostomy closure planned after two weeks.

Discussion

After discussion with the respiratory medicine team, it was concluded that there could be 4 possible reasons for the acute onset of respiratory distress in this patient–

1. Cor pulmonale owing to pulmonary hypertension:

   Long standing pulmonary hypertension could have lead to remodeling of pulmonary vasculature and increased pulmonary vascular resistance which could have caused dilatation and hypertrophy of the right ventricle.[10]

2. Preexisting, undetected OSA leading to respiratory fatigue and consequent respiratory failure. Respiratory failure, leading to decreased ventilation and accumulation of carbon dioxide leads to hypoxia and hypercarbia which decreases levels of consciousness complicating the weaning process postoperatively.[11]

3. An embolism of a clot to the brain leading to ischemic injury of the respiratory centre or loss of the gag reflex.[12,13]

4. Severe hypothyroidism could be a possible cause of the respiratory failure. Decrease in serum thyroid levels can affect the respiratory system by either decreasing the response to hypoxia/hypercapnea or by causing weakness of the diaphragm and respiratory muscles leading to accumulation of carbon dioxide and consequently leading to hypoxia and decreasing level of consciousness.[14–18]

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