Acute Right Ventricular Failure Postintubation in a Mitral Stenosis Patient

Sridhar Reddy Musuku, Saroj Pani, John Cagino
Department of Anesthesiology, Albany Medical Center, Albany, NY, USA

Abstract

Mitral stenosis (MS) is prevalent in 0.02-0.2% of the population in developed countries. The pathophysiology of MS results in elevated left atrial pressures and over-time results in pulmonary hypertension (HTN) which ultimately affects the right ventricle. In addition, MS restricts the diastolic filling of the left ventricle. Therefore, during induction patients with MS are limited by their ability to increase cardiac output by increasing stroke volume. Anesthesia goals in severe MS are to avoid sudden changes in heart rate, as well as systemic and pulmonary artery pressures. We report a patient who sustained severe hypotension upon induction and intubation which was resistant to conventional medications. Intraoperative transesophageal echocardiography displayed unique right atrial and right ventricular dilatation. In addition, the leftward inter-ventricular, inter-atrial septal shift and septal bounce were noted as the characteristic findings. Intravenous epinephrine bolus was administered to achieve normo-tension and normal chamber dimensions and interventricular septal position.

Keywords: Acute right ventricular failure, crash on induction, mitral stenosis

Case

This is a patient-consented and approved submission. A 70-year-old male (Height = 180 cm and weight = 104 kg) was scheduled to undergo a mitral valve (MV) replacement and coronary artery bypass grafting. His past medical history included rheumatic fever, coronary artery disease with a single stent in the left anterior descending artery (LAD), hypertension (HTN), diabetes mellitus, insertion of a pacemaker and defibrillator. Preoperative transthoracic echocardiography (TTE) revealed normal biventricular function, severe mitral stenosis (MS) with an area of 0.6 cm², moderate mitral regurgitation, and mild tricuspid regurgitation (TR). Cardiac catheterization revealed a right dominant circulation, patent LAD stent, and 100% right coronary artery (RCA) stenosis. Pulmonary artery (PA) pressures were 75/27 (46) mmHg and the mean MV gradient was 23 mmHg.

A preinduction arterial line, triple lumen central venous access, and a PA catheter were secured under local anesthesia, conscious sedation, and oxygen. Blood pressure preinduction was 114/69 mmHg. Baseline PA pressures were 68/29 mmHg. Norepinephrine and vasopressin infusions were infused to avoid hypotension, and epinephrine was on standby. Induction was accomplished with sufentanil, etomidate, and rocuronium. The patient was mask ventilated, and endotracheal intubation was performed followed by insertion of a transesophageal echocardiography (TEE) probe. Hypotension was observed along with increase in central venous pressures (CVP) and PA pressures. Intermittent epinephrine boluses (10 mcg) were given to restore the mean arterial blood pressure (MAP). The TEE findings postinductions were as follows.

Echocardiographic Findings

A midesophageal (ME) two-dimensional (2D) 4-chamber view revealed a dilated right atrium and right ventricle (RV), a “bi-septal shift,” i.e., left shift of interatrial septum (IAS), and interventricular septum (IVS) [Video 1]. The septal leaflet of the tricuspid valve (TV) was not visualized [Video 1] and a live 3D view also showed a bi-septal shift [Video 1]. An
ME 4-chamber color flow Doppler (CFD) view displayed a moderate TR [Video 1]. An ME long-axis view displayed a narrowed left ventricular outflow tract and dilated RV [Video 1]. After administering epinephrine bolus, ME 4-chamber view revealed a midline IAS and IVS, and septal leaflet of the TV was visualized [Video 2]. Application of CFD displayed a reduced TR [Video 2]. An abnormal IVS motion (bounce) was also a characteristic finding during the TEE examination [Video 2].

The IVS significantly contributes to biventricular function particularly the RV. IVS normally augments RV stroke volume by bulging like a piston into the RV during systole. In this patient, with pulmonary HTN, further augmentation of afterload evidently caused the IVS shift after induction. Compromised RV function was a result of the pressurized pulmonary circulation. RV was at risk of ischemic injury due to stenosed RCA at baseline and intraoperative pressure overload during induction. However, perioperative clinical course of the patient revealed no ischemic injury or dysfunction post-CPB.

Acute RV failure during induction may occur due to airway-related (hypoxia, hypercapnia, high airway pressure), perfusion, or afterload changes. A sudden increase in pulmonary vascular resistance or hypoperfusion of the RV due to systemic hypotension may lead to circulatory collapse. Hypotension after intubation suggests that airway pressure changes may have also contributed to the elevated PVR. Norepinephrine has shown to increase the coronary perfusion and also improve MAP in patients with pulmonary HTN. Vasopressin may also be considered as per literature. Data are limited with regard to inotropes but generally support the use of epinephrine, norepinephrine, dobutamine, and milrinone in RV failure. Even with appropriate pharmacologic choices preinduction, this patient sustained severe hypotension and RV failure.

An acute increase in CVP and echo findings (dilated RA, RV, IVS shift, and acute TR) were indicative of an acute RV failure. However, sharp elevation of CVP alone after induction may be seen in other conditions such as pericardial tamponade or tension pneumothorax. These were not the clinically relevant in this situation. Acute TR which reduced after epinephrine...
was not addressed because of the preoperative TTE findings. In addition, coaptation of the septal leaflet was evident after epinephrine, once the IVS regained its normal position.

Figure 1 is a graphical and schematic representation of CVP, mean PA pressure, MAP, and interventricular septal position.

**Conclusion**

TEE was diagnostic of acute right ventricular failure after intubation. The IVS shift and bouncing motion were characteristic finding. In addition, we learned the effects of epinephrine on reversing the position of IVS and also the acute TR.

**Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

**Financial support and sponsorship**

Nil.

**Conflicts of interest**

There are no conflicts of interest.

**References**

1. Nkomo VT, Gardin JM, Skelton TN, Gottdiener JS, Scott CG, Enriquez-Sarano M, et al. Burden of valvular heart diseases: A population-based study. Lancet 2006;368:1005-11.
2. Frogel J, Galusca D. Anesthetic considerations for patients with advanced valvular heart disease undergoing noncardiac surgery. Anesthesiol Clin 2010;28:67-85.
3. Francois H, Pierre C, Claude T, Denault YA. The right ventricle in cardiac surgery, a perioperative perspective: Anatomy, physiology and assessment. Anesth Analg 2016;7:407-21.
4. Mebazaa A, Karpati P, Renaud E, Algotsson L. Acute right ventricular failure – From pathophysiology to new treatments. Intensive Care Med 2004;30:185-96.
5. Forrest P. Anaesthesia and right ventricular failure. Anaesth Intensive Care 2009;37:370-85.
6. Kwak YL, Lee CS, Park YH, Hong YW. The effect of phenylephrine and norepinephrine in patients with chronic pulmonary hypertension. Anaesthesia 2002;57:9-14.