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Case Report

Hypoxemia After Percutaneous Mitral Valve Replacement: Management

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PERCUTANEOUS MITRAL VALVULOPLASTY is a well-established procedure for the treatment of mitral stenosis.[1] More recently, percutaneous mitral valve repair and replacement (PMVR) procedures have been performed for both mitral stenosis and regurgitation, and offer a less invasive approach for high risk patients.[1–7] The typical approach to PMVR is transapical through the left ventricular apex as it provides a more direct delivery of the valve.[8,9] However, the mitral valve also can be repaired or replaced via a trans-septal approach.[3–7,10,11] One complication of trans-septal procedures is a residual iatrogenic atrial septal defect (iASD).[3–12]

Percutaneous approaches to valve treatment may be preferred in patients with significant pulmonary hypertension (PHTN).[12,13] Outcomes depend on the etiology, severity, and reversibility of the PHTN.[12,13] Classification of PHTN is important for determining short- and long-term therapeutic plans. Pulmonary hypertension is classified into 5 groups: 1) pulmonary artery (reactive) hypertension; 2) pulmonary venous hypertension due to left heart dysfunction; 3) pulmonary hypertension related to pulmonary parenchymal disease and hypoxia; 4) pulmonary hypertension related to thromboembolic disease; and 5) pulmonary hypertension related to systemic disorders affecting the vasculature directly.[13–16] Alternatively, PHTN can be described as precapillary (arterial), parenchymal (arterial), or postcapillary (venous). Category 2 or postcapillary pulmonary venous hypertension can result from mitral valve dysfunction.[13–15] Left untreated, patients with pulmonary venous hypertension can develop a mixed PHTN to include reactive precapillary pulmonary artery hypertension.[12,14,15]

The authors present a case of an elderly patient with severe pulmonary hypertension who underwent percutaneous trans-septal mitral valve replacement for mixed prosthetic valve stenosis/regurgitation. The case was complicated by an iASD and acute post-procedural hypoxemia.

Case Presentation

The patient is an 86-year-old female with a history of coronary artery disease, myocardial infarction, congestive heart failure, and a prior mitral valve replacement. She presented with prosthetic mitral valve stenosis (MS) and moderate mitral regurgitation (MR) (Fig 1; Video Clip 1). The planned procedure was a transcatheter, valve-in-valve replacement via a trans-septal approach.

After placement of intravenous and intra-arterial catheters, general anesthesia was administered, a pulmonary artery catheter was placed, and a transesophageal echocardiogram (TEE) exam was performed. The initial pulmonary artery catheter data showed severe PHTN (Table 1). The TEE exam showed moderate mitral regurgitation, severe mitral stenosis. The peak and mean transmittal gradients were 34 and 23 mmHg, respectively, and the mitral valve area was < 1.0 cm^2.

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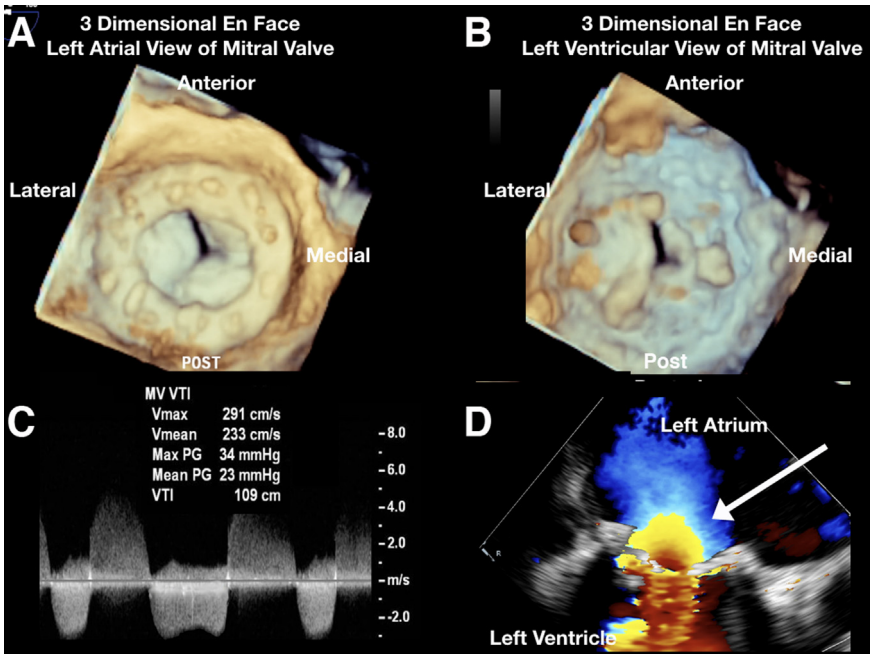

Fig 1. Two-dimensional (2D) and three-dimensional (3D) images of the stenotic bioprosthetic mitral valve. A and B are 3D en-face views of the bioprosthetic mitral valve from the perspective of the left atrium (A) and left ventricle (B). (C) Continuous-wave Doppler assessment of transmitral flows showing a peak and mean gradient of 34 and 23 mmHg, respectively. (D) A 2D color Doppler flow across the stenotic valve from the midesophageal two-chamber window. The white arrow points to the flow convergence consistent with stenosis.

The left ventricular systolic function was depressed mildly. There was moderate to severe tricuspid regurgitation and moderate right ventricular systolic dysfunction (Video Clip 2).

The procedure was performed via the right and left femoral veins. A pacing wire was placed in the left femoral vein and advanced into the right ventricle. Through the right femoral vein, after puncture and dilation of the interatrial septum (IAS) using an 8-mm diameter, 4-cm long balloon, a 9-French Agilis sheath (St Jude Medical, St Paul, MN) was advanced across the IAS (Fig 2). Subsequently, the Agilis catheter was replaced with a 16-French delivery sheath placed in the inferior vena cava (IVC) just below the right atrium. Through this device, a 14-mm diameter balloon catheter was placed across the IAS and through the stenotic mitral bioprosthesis (Fig 2). During rapid ventricular pacing, a balloon valvuloplasty was performed, reducing the peak transvalvular gradient from 34 to 9 mmHg. Subsequently, a 26-mm Sapien 3 valve (Edwards Lifesciences, Irvine, CA) was advanced via the IVC, through the right atrium and IAS and into the mitral position, the latter during rapid ventricular pacing. The valve was postdilated once. Postprocedural imaging showed a few paravalvular regurgitant jets, altogether resulting in mild MR. The final transvalvular peak and mean gradients were 8 (1.4 m/s) and 4 mmHg. The prosthetic mitral valve area was 2.5 cm^2 (Fig 3; Video Clip 3). The delivery system and pacing wire were removed. Of note, there was now a left to right shunt across the IAS with a peak velocity of 1 m/s (Fig 4A). The defect was 5 × 4 mm or 0.25 cm^2 area measured using three-dimensional planimetry. Throughout the procedure the patient was hemodynamically stable (heart rate 70-72 beats/min; systemic blood pressure 130/70-150/70 mmHg) with minimal vasoactive support (Table 1). Post procedure, the left ventricular ejection fraction was interpreted as normal, and the right ventricular systolic function was reduced moderately. With the completion of the procedure and continued hemodynamic stability, the dose of norepinephrine was reduced.

Approximately 20 minutes after the procedure ended, while still under general anesthesia, the peripheral oxygen saturation (SaO_2) value decreased from 97% to 81% and then to 65% (Table 1). The results of an arterial blood gas analysis were pH 7.34, pCO_2 45 mmHg, pO_2 40 mmHg. During this time while the heart rate (70-75 beats/min) and systemic blood pressure (130/60-130/70 mmHg) appeared unchanged, there were small elevations in the pulmonary artery and central venous pressures (CVPs) (Table 1). TEE exam revealed no significant changes in valvular or ventricular functions. There was, however, a significant right-to-left shunt across the IAS with a velocity of 1 m/s (Fig 4B).

In order to increase systemic vascular resistance (SVR) and reduce pulmonary vascular resistance (PVR), increases in anesthetic depth, fraction of inspired oxygen (F_IO_2), the norepinephrine dose, and minute ventilation were performed. Despite this, SaO_2 continued to be low. Management discussions included medical management versus placement of an IAS occluding device. The former included administration of inhaled nitric oxide or inhaled prostacyclin. Neither of these options was available immediately. The patient already was receiving an intravenous pulmonary vascular dilator (nitroglycerin [NTG]) and a systemic vascular constrictor (norepinephrine). Milrinone was available immediately and, with the SaO_2 continuing to decline from 81%, it was decided to administer it via the inhaled route. Given the severity and urgency of the hypoxemia, it was determined to administer 4 mg milrinone (1.0 mg/5 mL) by nebulizer consistent with prior literature.[17–21] Within 10-15 minutes SaO_2 increased and this effect persisted despite a decrease in systemic blood pressure

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