CASE CONFERENCE

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CASE 1—2016
Problem-Solving in Transcatheter Aortic Valve Replacement: Cardiovascular Collapse, Myocardial Stunning, and Mitral Regurgitation

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TRANSCATHETER AORTIC VALVE replacement (TAVR) is a minimally invasive approach to aortic valve replacement that has disseminated rapidly worldwide.[1] During TAVR, short periods of rapid ventricular pacing (RVP) commonly are applied to facilitate precise positioning of the valve prosthesis before deployment.[2,3] Although RVP typically is brief and well tolerated, it can precipitate myocardial ischemia with ventricular stunning and consequent hemodynamic lability.[1–5] Besides temporary myocardial stunning, there are multiple possible etiologies for cardiovascular collapse during TAVR, including pericardial tamponade, acute aortic regurgitation, coronary occlusion, arterial rupture, and acute aortic dissection.[6–7] The rapid diagnosis and management of cardiovascular collapse during TAVR often are facilitated by expert transesophageal echocardiography (TEE) in the setting of a multidisciplinary cardiac team.[7,8] Furthermore, systolic anterior motion of the mitral valve with left ventricular outflow tract obstruction and significant mitral regurgitation can also complicate aortic valve replacement.[9–11] In this case conference, the integrated management of cardiovascular collapse during TAVR complicated by ventricular stunning and subsequent dynamic left ventricular outflow tract obstruction is described.

CASE PRESENTATION*

A 91-year-old female with severe aortic stenosis and congestive heart failure presented for transaortic TAVR with a 23-mm Edwards-Sapien prosthesis. Her concomitant coronary artery disease had been managed successfully medically after recent coronary stenting. Her preoperative transthoracic echocardiogram was significant for severe aortic stenosis with a calculated aortic valve area equal to 0.78 cm^2, a left ventricular ejection fraction of 75%, and mild mitral regurgitation. Furthermore, there was global left ventricular hypertrophy with mild diastolic dysfunction. Although there was a septal knuckle with a diameter of 1.5 cm (normal 0.6-1.1 cm), there was no left ventricular outflow tract obstruction.

On arrival to the hybrid operating room, the patient was awake, alert, and normotensive. After placement of standard monitors, a right radial arterial catheter was placed for continuous blood pressure monitoring. The intravenous induction of general anesthesia and subsequent endotracheal intubation were uneventful. Anesthesia was maintained with titrated isoflurane and remifentanil infusion. Neuromuscular blockade was achieved with intermittent vecuronium and a phenylephrine infusion was titrated to maintain adequate systemic vascular resistance. Central venous access was secured under ultrasound guidance. An oximetric pulmonary artery catheter also was floated via the right internal jugular vein; correct placement was confirmed both by pressure transduction and TEE. Furthermore, comprehensive TEE confirmed the presence of severe aortic stenosis, with hyperdynamic left ventricular systolic function, left ventricular hypertrophy, septal knuckle, mild central mitral regurgitation, and normal right ventricular systolic function (Fig 1). Left ventricular end-diastolic diameter was measured at 4.4 cm (normal 4.2-5.5 cm) and left ventricular posterior wall diameter was 1.7 cm (normal 0.7-1.1 cm). Detailed three-dimensional imaging of the aortic annulus also confirmed adequate dimensions for a balloon-inflatable 23-mm Edwards-Sapien prosthesis.[3,8,11]

After the placement of both venous and arterial femoral catheters, a right ventricular pacing wire was placed via the right femoral vein under fluoroscopic and echocardiographic

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1053-0770/2602-0033$36.00/0
http://dx.doi.org/10.1053/j.jvca.2015.03.015

Key words: transcatheter aortic valve replacement, cardiovascular collapse, myocardial stunning, mitral regurgitation, transesophageal echocardiography, systolic anterior motion, dynamic, left ventricular outflow tract obstruction, intra-aortic balloon counterpulsation

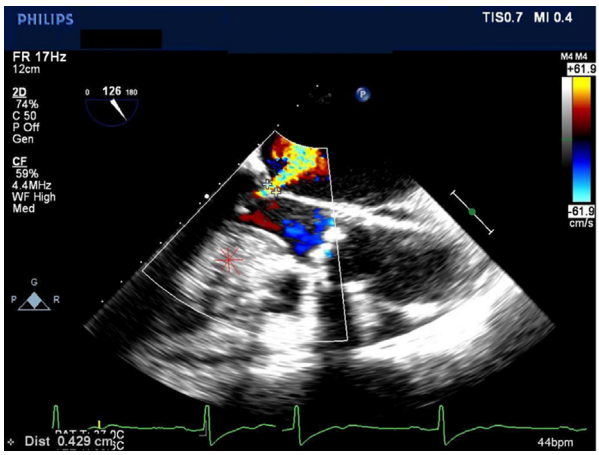

Fig 1. Midesophageal aortic valve long-axis view that depicts a calcified and stenotic aortic valve with a prominent septal knuckle (red star) and mild mitral regurgitation.

guidance. After minimally invasive partial sternotomy, the ascending aorta was exposed and cannulated with a 7-French sheath via the Seldinger technique.[12] After adequate heparinization, an Edwards-Sapien delivery system was placed through the 7-French sheath and positioned in the ascending aorta. A 22-mm balloon was aligned across the native aortic valve under fluoroscopic and TEE guidance. After initiation of RVP, balloon valvuloplasty of the native aortic valve was performed. Rapid termination of RVP was followed by prompt hemodynamic recovery. There was new mild aortic regurgitation detected on surveillance TEE.[7,8] Thereafter, a 23-mm Edwards-Sapien prosthesis was positioned precisely across the aortic valve under fluoroscopic and TEE guidance. After initiation of RVP for a mean arterial pressure of 40 mmHg, the valve prosthesis was deployed in a standard fashion.

On termination of RVP, the patient developed acute heart block with bradycardia and persistent hypotension. Despite ventricular pacing and aggressive vasopressor administration, complete cardiovascular collapse ensued. Diagnostic TEE showed adequate valve position and function and ruled out pericardial tamponade, acute aortic dissection, and annular rupture. There was global biventricular hypokinesis, acute mitral annular dilation, and torrential central mitral regurgitation, consistent with ventricular stunning (Fig 2). Aggressive cardiopulmonary resuscitation ensued with brief chest compressions and ongoing bolus intravenous epinephrine. Prompt aortography confirmed patency of the right and left coronary ostia. Although preparations were made for immediate cardiopulmonary bypass, there was return and steady recovery of spontaneous circulation.

Given the ventricular stunning and severe mitral regurgitation, the cardiac team proceeded with percutaneous placement of an intra-aortic balloon pump via the right femoral artery to maximize coronary perfusion and decrease left ventricular afterload.[13] The tip of the balloon catheter was positioned about 3-5 cm distal to the origin of the left subclavian artery by both fluoroscopic and echocardiographic guidance. Despite inotropic support and intra-aortic balloon counterpulsation, there was failure to achieve complete hemodynamic recovery, heralded by low cardiac output, acute pulmonary hypertension, and systemic hypotension with a mean arterial pressure of 50 mmHg. Further echocardiographic evaluation at this point in the resuscitation found mild paravalvular aortic regurgitation, improving ventricular systolic function, left ventricular outflow tract obstruction (LVOT) due to systolic anterior motion of the anterior mitral leaflet (SAM), and severe eccentric mitral regurgitation (Fig 3).

Given these new echocardiographic findings, maximal therapy for relief of the LVOT obstruction commenced. The inotropic support and intra-aortic balloon counterpulsation both were discontinued. Furthermore, aggressive intravascular volume resuscitation and titrated pharmacologic support of systemic vascular resistance with norepinephrine and vasopressin were instituted. The management maneuvers also were guided by serial echocardiographic assessment of the LVOT, SAM, and mitral regurgitation. With this comprehensive management, the patient experienced complete hemodynamic and ventricular

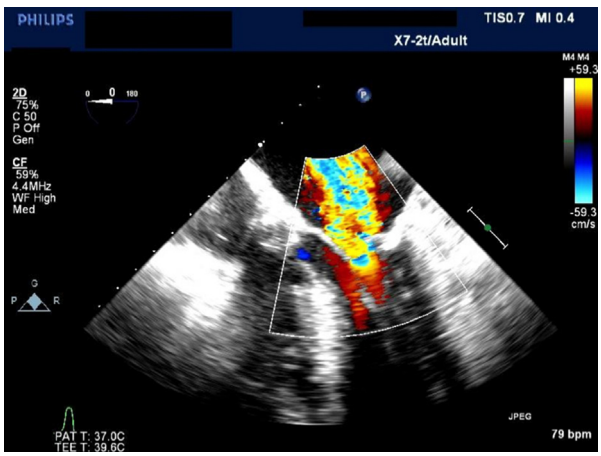

Fig 2. Modified midesophageal 4-chamber view immediately after valve deployment that shows severe central mitral regurgitation and no obstruction of the left ventricular outflow tract.

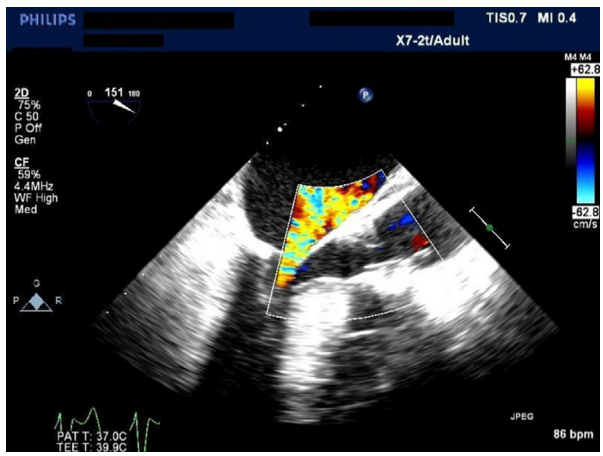

Fig 3. Midesophageal aortic valve long-axis view after return of spontaneous circulation that demonstrates left ventricular outflow tract obstruction and eccentric mitral regurgitation.