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Case Report

Acquired discrete subaortic stenosis late after mitral valve replacement



Jagdish C. Mohan^{*}, Madhu Shukla, Vishwas Mohan, Arvind Sethi

Fortis Institute of Cardiac Sciences, Fortis Hospital, Shalimar Bagh, New Delhi 88, India

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ABSTRACT

Although acquired left ventricular outflow obstruction has been reported in a variety of conditions, there are scant reports of its occurrence following mitral valve replacement (MVR). This study describes two female patients, who developed severe discrete subaortic stenosis, five years following MVR. In both cases, the mitral valve was replaced by a porcine Carpentier-Edwards 27-mm bioprosthesis with preservation of mitral valve leaflets. The risk of very late left ventricular outflow tract obstruction after bio-prosthetic MVR with preservation of subvalvular apparatus needs to be kept in mind in symptomatic patients.

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1. Introduction

In patients, who undergo mitral valve replacement (MVR) with a high- or low-profile prosthesis, left ventricular outflow tract (LVOT) obstruction is a well-recognized but infrequent postoperative complication.^{1–9} The LVOT obstruction can be transient or dynamic^{1–6} or may be due to projecting preserved exuberant anterior mitral leaflet⁷ or can be fixed due to development of discrete subaortic stenosis (DSS).^{8,9} Transient obstruction can occur immediately after MVR, if the LV cavity is small, anterior basal septum is thick, aorto-mitral angle is steep and/or excessive inotropes are used in presence of under-filled ventricle.^{1–6} Fixed DSS has been described in a few cases late after MVR due to development of an abnormal tissue ridge or membrane secondary to flow disturbance caused by

abnormally oriented prosthetic struts.^{8,9} Systolic contact between interventricular septum and prosthetic struts may lead to subendocardial remodeling resulting in a fibromuscular membranous ridge in susceptible subjects. Herein, we report two cases of very late LVOT obstruction after MVR with a bio-prosthesis and preservation of the native posterior mitral valve leaflet.

2. Case report 1

In February 2010, a 28-year-old woman underwent MVR due to severe mitral stenosis and 2+ mitral regurgitation complicated by cardiac failure. Preoperative echocardiography showed a normally functioning aortic valve with no LVOT gradient. She underwent implantation of a 27-mm porcine mitral valve

^{*} Corresponding author.

E-mail address: a51hauzkhas@gmail.com (J.C. Mohan).

Abbreviations: LV, left ventricle; RV, right ventricle; AO, aortic root; LA, left atrium; MVO, mitral valve orifice; LVOT, left ventricular outflow tract.

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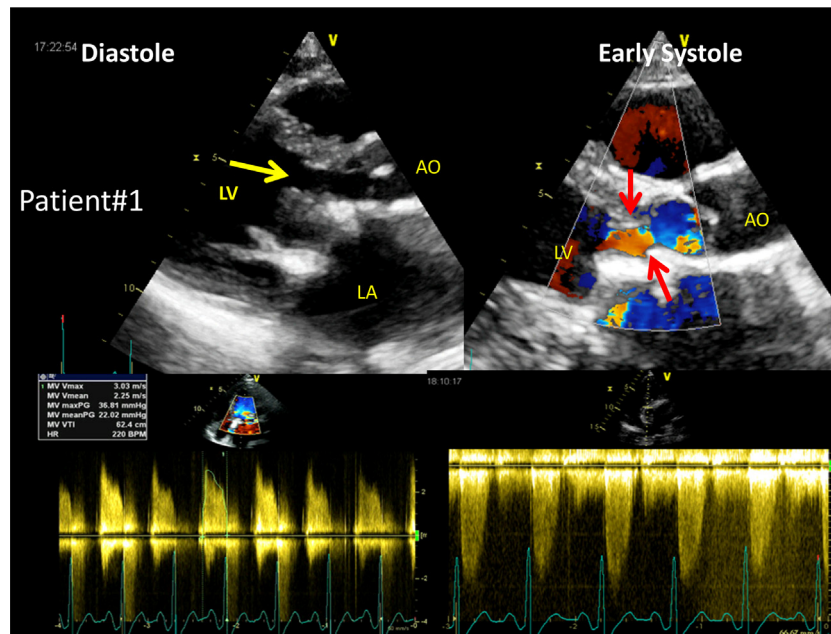


Fig. 1 – Upper panels show 2D-echocardiographic transthoracic parasternal long axis views in diastole (yellow arrow) along with narrow LVOT and turbulent jet (red arrows) in early systole. Left lower panel shows a mean trans-mitral diastolic gradient of 22 mmHg. Right lower panel shows peak velocity of 5.1 m/s across the LVOT.

bioprosthesis (Edwards Lifesciences, S.A.; Horw, Switzerland) with preservation of the native posterior mitral valve leaflet and its subvalvular apparatus. The postoperative course was smooth, and the patient was discharged on medical therapy. She became symptomatic with exertional dyspnea in July 2015. She progressively worsened over next three months. At presentation, her physical examination revealed pulse rate of 102/minute, supine blood pressure of 100/80 mmHg, distended jugular veins, hepatomegaly, basal rales, a loud apical pansystolic murmur with a prominent basal ejection systolic murmur. A 12-lead electrocardiogram showed normal sinus rhythm, sinus tachycardia and left atrial overload. Hematological and biochemical parameters were unremarkable. Plain chest skiagraphy showed enlarged cardiac silhouette, pulmonary venous congestion and a small right-sided pleural effusion. Her trans-thoracic echocardiogram revealed enlarged left atrium, normal left ventricular ejection fraction, asymmetric septal hypertrophy, anteriorly oriented mitral prosthesis, moderate tricuspid regurgitation, dilated inferior vena cava and estimated pulmonary systolic pressure of 86 mmHg. The leaflets of mitral bioprosthesis were thickened, two of the leaflets were immobile while the one leaflet was flail and prolapsed into the left atrium during systole causing severe mitral regurgitation along with a narrow antegrade jet during diastole (vena contracta = 7 mm). On Doppler examination, mean trans-mitral diastolic gradient was 22 mmHg, and peak systolic gradient was 244 mmHg (Fig. 1).

The LVOT was narrow in systole and diastole with reduced distance between the struts of the mitral bioprosthesis and the anterior septum. Color Doppler evaluation showed flow turbulence and continuous-wave Doppler examination revealed a peak LVOT gradient of 104 mmHg (mean 65 mm). There was no aortic regurgitation. Careful examination of the

LVOT revealed a membrane attached to the ventricular surface of the bioprosthetic strut and the left side of the interventricular septum (Figs. 2 and 3). Multi-planar 3D reconstruction of the left ventricle in short axis view showed narrow elliptical LVOT and a prominent ridge of tissue attached to the anterior aspect of the struts and protruding into the LVOT.

The patient is awaiting MVR and reconstruction of the LVOT.

3. Case report 2

A 38-year old female was evaluated for complaints of fatigue and class II dyspnea in September 2010. She had undergone MVR with a #27 Carpentier-Edwards bioprosthesis with preservation of posterior leaflet, five and a half years back for calcific mitral stenosis. At the time of operation, detailed echocardiogram showed normal aortic valve and the LVOT. At presentation, her physical examination showed an average built lady, pulse rate 78 BPM, supine blood pressure of 120/86 mmHg and no evidence of heart failure. Precordial examination revealed normal heart sounds, a basal ejection systolic murmur and a high-pitched early diastolic murmur along left sternal border. A 12-lead electrocardiogram showed sinus rhythm and non-specific ST-T changes. Plain chest skiagram was unremarkable. Her routine biochemistry was normal.

Trans-thoracic echocardiogram revealed thickened left ventricular walls, anteriorly oriented mitral prosthesis projecting into the LVOT, which showed flow turbulence during systole on color Doppler examination (Fig. 4). Continuous-wave Doppler examination showed a peak gradient of 65 mmHg across the LVOT (mean 38 mmHg) and moderate

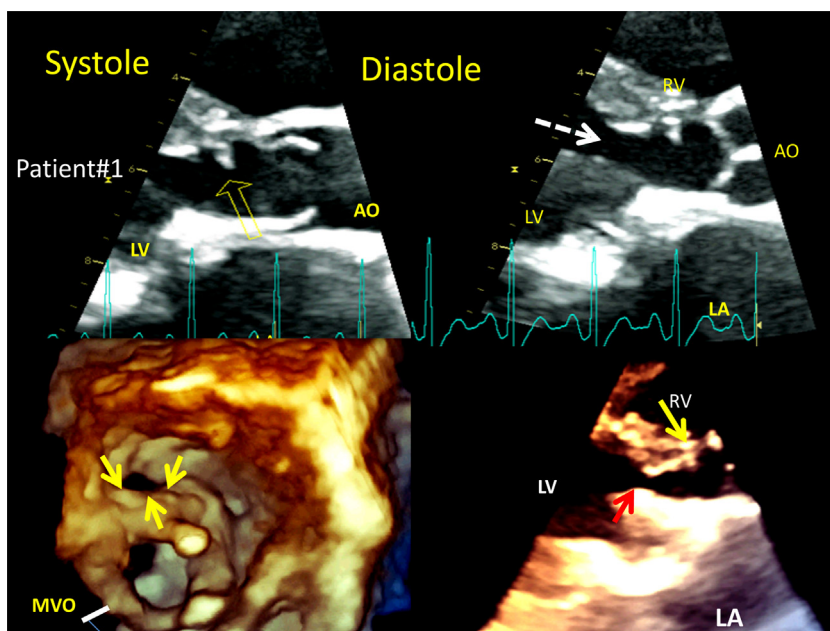


Fig. 2 – Upper panels show LVOT in parasternal long axis views. Yellow hollow arrow points toward the membrane. The lower panels show the shape and the size of the LVOT during systole in 3D reconstruction (left lower, yellow solid arrows) and two levels of obstruction in tandem in LVOT (right lower image, red arrow and yellow arrow). Persistent non-closure of the mitral orifice (MVO) during systole is due to the flail leaflet.

aortic regurgitation. The aortic valve showed no structural abnormality. Multiple views showed localized thickening of the anterior interventricular septum at the contact point of the strut during systole (Fig. 5). The leaflets of the mitral bioprosthesis were functioning normally and there was a mean trans-mitral gradient of 4 mmHg with no regurgitation.

The need for reoperation was discussed with the patient along with the possibility of another operation, as and when the mitral bioprosthesis shows deterioration. She preferred to be on conservative treatment and has been followed for five years with some deterioration in symptoms and LVOT gradients (peak gradient –72 mmHg).

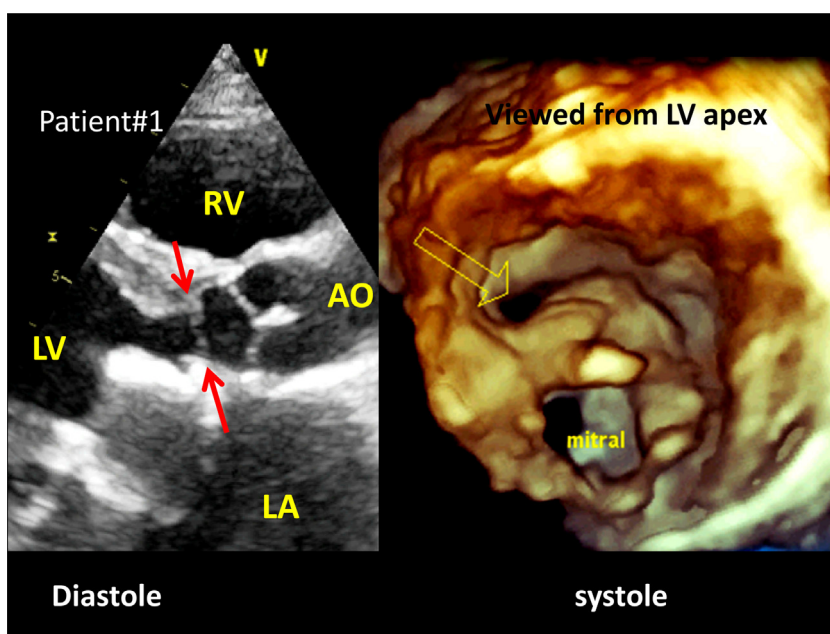


Fig. 3 – Visualization of the membrane during diastole (red arrows) in parasternal long axis view and the crescent opening of the LVOT during systole in 3D plane (right image). Note exuberant growth of tissue on the bioprosthetic strut facing the LVOT.

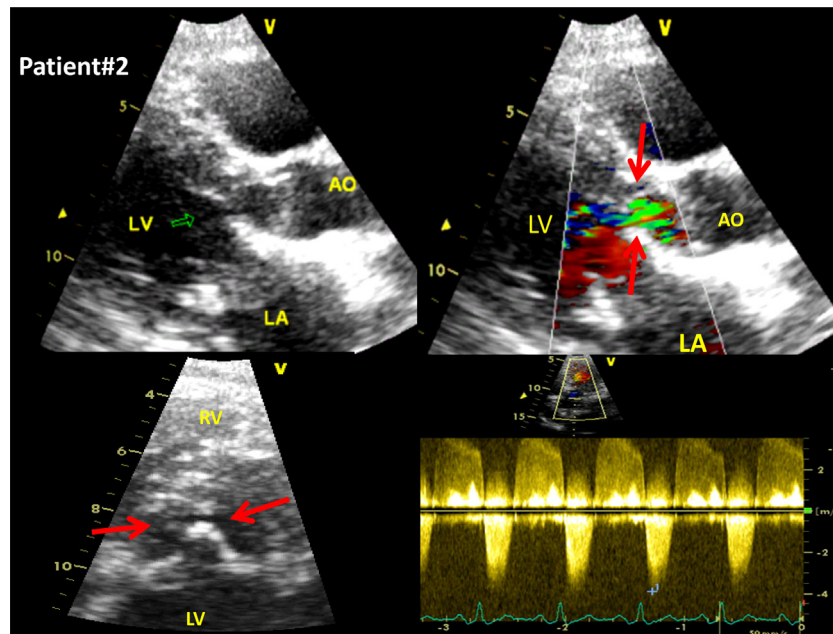


Fig. 4 – Trans-thoracic 2D echocardiographic parasternal long axis view focusing on the LVOT. Note narrow LVOT with color flow turbulence during systole in upper two images. The lower left image in short axis shows abnormally placed bioprosthetic strut juxtaposed to localized thickening of the anterior interventricular septum (red arrows). Right lower image shows spectrum of systolic and diastolic flow across the LVOT with a peak gradient of 65 mmHg on continuous-Doppler examination.

4. Discussion

Transient or dynamic obstruction of the LVOT after MVR is well-recognized. Transient obstruction can occur immediately after MVR if the LV cavity is small, anterior basal septum is thick, aorto-mitral angle is steep and/or excessive inotropes are used in presence of under-filled ventricle.¹⁻⁶ In some

patients with mild LVOT obstruction due to the high-profile prosthesis and its abnormal position, the obstruction may be worsened by preload reduction secondary to atrial fibrillation and diuretics or by hyper-contractile cardiac function as a result of inotropic therapy.

It has recently been shown that DSS can also develop in adulthood after acquired heart diseases. Khoshnevis et al.⁸ reported three patients with rheumatic heart disease who

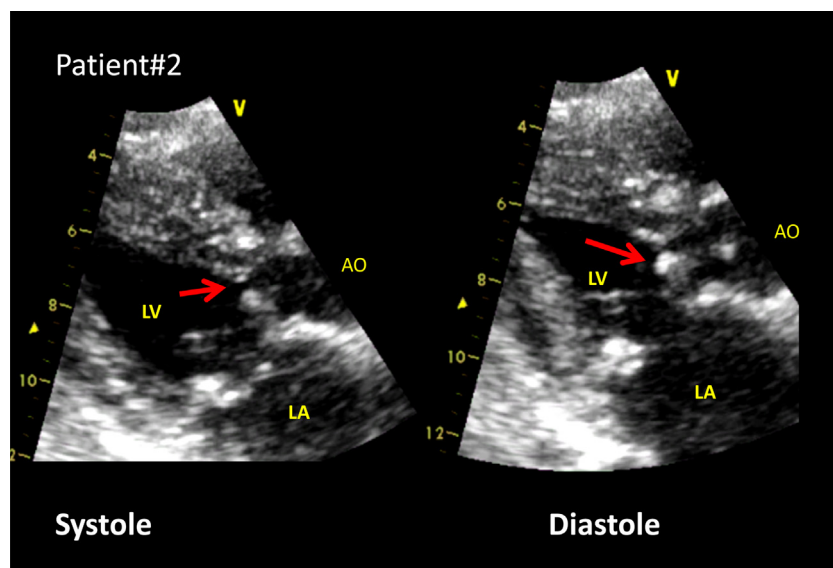


Fig. 5 – 2D echocardiographic parasternal long axis views showing narrow LVOT (red arrow) both during systole and diastole due to abnormally projecting bioprosthetic strut. Note markedly thickened anterior interventricular septum. The bioprosthetic leaflets show normal mobility.

developed severe stenosis by a subaortic membrane many years after the prosthetic replacement of the mitral valve. Patel et al.⁹ described a patient, who developed DSS four years after combined aortic and MVRs. Oliver et al.¹⁰ reported three patients with rheumatic mitral valve stenosis who developed DSS. One of them had a previous mitral commissurotomy performed six years before the subaortic membrane was diagnosed. The other two patients were diagnosed with severe DSS and rheumatic mitral stenosis at 35 and 64 years of age, respectively. In all the three patients, a subaortic membrane was confirmed and removed during the operation. Both of our patients had rheumatic mitral stenosis. Mitral stenosis is characterized by a small left ventricular cavity and possibly an unfavorable aorto-mitral angle; the factors, which may predispose to increased shear stress in the LVOT and consequent abnormal tissue growth.

In most cases of postoperative LVOT obstruction such as ours, obstruction results from protrusion of a high-profile prosthetic valve into the LVOT or from abnormal subvalvular positioning of the prosthesis in a small LV cavity. If the prosthesis is not oriented properly, a strut or tissue overgrowth may obstruct the outflow tract. The cause of the mitral valve improper positioning may have been due to the over preservation of the posterior mitral valve leaflet leading to abnormal position of the bioprosthesis. Anti-anatomical positioning of prosthesis results in continuing turbulence produced by abnormal strut into the LVOT, which can result in permanent deposits of fibrous tissue and fixed subaortic obstruction.⁸

It has been suggested that abnormal fluid dynamic forces at the LVOT level can cause septal shear stress.^{10,11} Different morphologic alterations can cause changes in fluid dynamic factors that increase septal shear stress. Both retrospective and prospective studies of children developing DSS have found an increased mitral-aortic fibrous distance, a steeper aorto-septal angle and a narrower LVOT.^{6,9} Minor morphologic changes in the LVOT can produce marked modifications in dynamic forces and septal shear stress. These morphologic changes act both in the onset and in the progression of the subaortic obstruction. Although progression of DSS obstruction can be very rapid in infants and small children at vulnerable periods of development, our two cases show that the obstruction progresses much more slowly during adult life.

Conflicts of interest

The authors have none to declare.

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