Effect of Progressive Left Ventricular Dilatation on Degree of Mitral Regurgitation Secondary to Mitral Valve Prolapse

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Described herein is a 71-year-old man who at age 61 was found by echocardiogram to have severe mitral regurgitation (MR) from mitral valve prolapse. During the subsequent 9 years the MR progressively lessened as his left ventricular cavity dilated and his ejection fraction progressively fell such that just before orthotopic heart transplantation the degree of MR was no longer severe, and the prolapse of the mitral leaflets had disappeared. This report describes this unique patient. © **2019 Published by Elsevier Inc. (Am J Cardiol 2019;123:1887−1888)**

To our knowledge, the resolution of severe mitral regurgitation (MR) from mitral valve prolapse (MVP) as the cardiac output progressively fell and the left ventricular cavity progressively dilated has not been described. Such is the purpose of this report.

Case description

A 71-year-old white man, who was born in December 1946, was told when in his 40s that he had a "heart murmur" from MVP. Because of the precordial murmur an echocardiogram was done when he was 61 years old (April 2008), and it confirmed MVP with marked leaflet thickening, severe leaflet prolapse, and severe MR; additionally, the left ventricular size and function were normal. The tricuspid valve also had evidence of prolapse. Thereafter, he was asymptomatic and working out regularly with a trainer until age 70 (October 2016), when experiencing an upper respiratory infection, he also noted exertional dyspnea, orthopnea, and lower leg edema. Examination in January 2017 disclosed no precordial murmur; echocardiogram showed the left ventricular ejection fraction to be 20% (Figure 1). The thickened mitral leaflets were tented toward the left ventricular wall without prolapse and there was moderate MR. The electrocardiogram showed atrial fibrillation, ventricular premature complexes, left ventricular hypertrophy with strain, and prolonged Q-T interval. Cardiac catheterization disclosed angiographically normal coronary arteries and the following pressures (in mm Hg): mean pulmonary artery wedge 26; right ventricle 35/3, mean right atrium 6; left ventricle 95/17, and aorta 105/75. The cardiac index (Fick) was 1.3 L/min/m^2. The left ventricular end-diastolic dimension was 7.1 cm, and its systolic diameter, 6.4 cm. His B-type natriuretic peptide was 1440 pg/ml. He was placed for the first time on full heart failure medications.

Repeat echocardiogram in November 2017 showed the left ventricular ejection fraction to be 10% and the MR was only of mild degree and no mitral prolapse was seen. An intracardiac defibrillator was inserted and the atrioventricular node ablated. Heart failure medications and apixaban were continued. Because of lack of improvement from either the medications or devices, orthotopic heart transplant was performed in June 2018. The explanted heart weighed 620 g. The epicardial coronary arteries were devoid of any narrowing. The myocardium was devoid of grossly visible lesions. The mitral valve leaflets were classic for MVP (Barlow syndrome type), and the tricuspid valve also had evidence of prolapse (Figure 2).

When contacted in December 2018, he was asymptomatic and back to work!

Comments

Described herein is a patient with classic MVP known to be present for at least 3 decades. Several years before heart transplantation when the left ventricular function was normal the echocardiogram showed severe MR. With time, the left ventricular ejection fraction and cardiac output progressively fell, and the left ventricular cavity progressively dilated such that just before heart transplantation there was no precordial murmur, no mitral valve prolapse, and only mild MR by echocardiogram. As the left ventricular cavity dilated, the mitral chordae were pulled laterally preventing the mitral leaflets from prolapsing into the left atrial cavity. Current guidelines support mitral valve repair before the left ventricular ejection fraction falls below 60% to avoid the inevitable consequence of LV failure.[1] We are not aware

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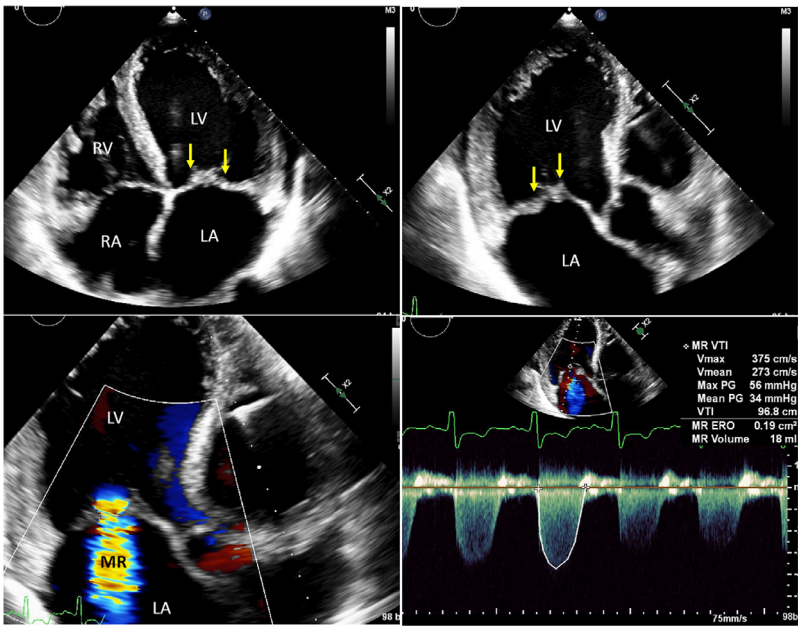

Figure 1. *Top left:* Apical 4-chamber view at end-systole showing 4-chamber dilation with thickened mitral leaflets (arrows) that never prolapsed into the left atrium (LA). The LA is bowed toward the right atrium (RA) consist with high LA pressure. RV = right ventricle. *Top right:* Apical long-axis view showing thickened mitral leaflets (arrows) without prolapse. *Bottom left:* Apical long-axis view of centrally directed mitral regurgitation (MR) jet. *Bottom right:* Continuous wave doppler of the MR jet showing a low peak velocity − 3.75 m/s, suggesting very elevated LA pressure. The calculated EROA was 0.19 cm^2, a value suggesting only mild MR.

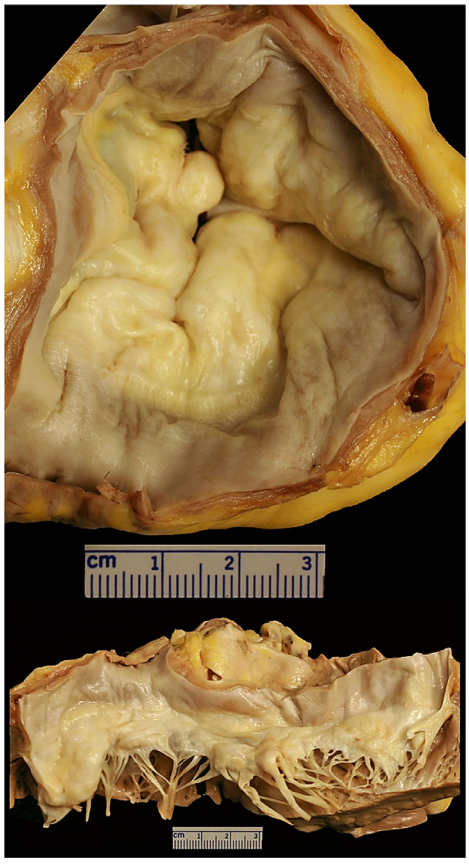

Figure 2. Photograph of the mitral valve, typical of mitral valve prolapse, in the explanted native heart. *Top*: View from the left atrium. *Bottom*: View of the opened mitral valve.

of a similar published report describing the resolution of MVP and severe reduction in MR from classic MVP with progressive worsening of left ventricular function.

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