Case Report

A Presumed Mechanism of Mitral Regurgitation after Left Ventriculoplasty

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After isolated left ventriculoplasty, the mechanism of mitral regurgitation (MR) remains unclear. A 68-year-old male with ischemic cardiomyopathy presented with a new onset of severe MR after left ventriculoplasty without a mitral procedure. He needed a second operation for heart failure because of the MR. We speculate about its mechanism and express caution about the procedure. (Ann Thorac Cardiovasc Surg 2010; 16: 139–141)

Key words: left ventriculoplasty, mitral regurgitation

Introduction

Methods of surgical left ventriculoplasty for a severely remodeled left ventricle (LV) with diffuse akinetic areas have evolved to reduce LV wall stress. The recurrence of mitral regurgitation (MR) has been reported to relate to a severely remodeled LV and decreased systolic function, but its true mechanism after isolated left ventriculoplasty remains unclear. We experienced one case of a new onset of acute MR after left ventriculoplasty without a mitral procedure and speculate about its mechanism.

Case

A 68-year-old male with ischemic cardiomyopathy (ICM) was admitted to our hospital for coronary artery bypass grafting (CABG) and left ventriculoplasty. Eleven years earlier he had undergone percutaneous coronary angioplasty (PCI) to the proximal anterior left descending artery (LAD) and the left circumflex artery. Upon recent admission, he presented with heart failure, and a coronary angiography showed total occlusion of #4 (AV, atrioventricular branch), #12, and #13; 90% stenosis of #4 (PD, posterior descending artery) and #7; and a 75% stenosis of #11. There was no restenosis at the previous PCI lesion. Preoperative transthoracic echocardiography (TTE) showed severely remodeled LV; the LV end-diastolic dimension (LVDd) was 75 mm, and the LV end-systolic dimension (LVDs) was 66 mm. The LV wall motion was akinesis or severe hypokinesis, except for the basal area. Mitral tethering was mild, and the MR was trivial. The LV end-diastolic volume index (LVEDVI) and the LV end-systolic volume index (LVESVI) were 164 ml/m^2 and 108 ml/m^2 in quantitative gated single photon emission computed tomography (QGS-SPECT), and the LV ejection fraction was 26%. Delayed enhanced magnetic resonance imaging (DE-MRI) showed diffuse LV subendocardial DE except for the basal posteroinferior area.

The patient underwent overlapping left ventriculoplasty (OLVP) concomitant with CABG. The LAD was bypassed by the left internal thoracic artery, and the #4 PD and the two #14s were bypassed by using saphenous vein grafts from the ascending aorta. We made a longitudinal LV incision of 8 cm on the anterior wall along the LAD and determined the suture line with our original 72-ml cone-shaped sizer for the OLVP. The cardiopulmonary bypass (CPB) and aortic cross clamp (AXC) times were 231 and 131 minutes, respectively. The early postoperative course was uneventful, but a grade 2 MR was detected by TTE a few days after the operation and

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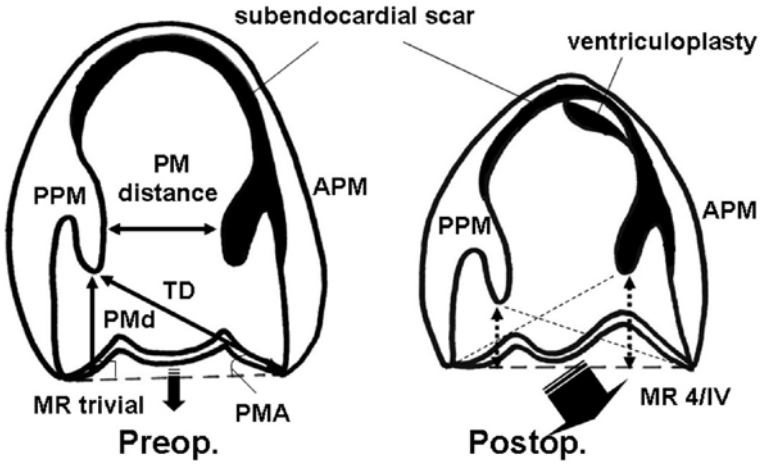

Fig. 1. Pre- and postoperative morphology of submitral apparatus.
APM is more displaced to the apex than PPM postoperatively.
PM, papillary muscle; PPM, posterior papillary muscle; APM, anterior papillary muscle; TD, tethering distance; PMd, papillary muscle depth; PMA, papillary muscle angle; MR, mitral regurgitation.

increased to grade 3 in two weeks. The MR jet was from the central to the lateral portion (Fig. 1). MR progressed to grade 4 in four weeks after surgery, and the patient presented with pulmonary hypertension (systolic pulmonary artery pressure of 50 mmHg) and shortness of breath.

Forty days after the first operation, the patient underwent a reoperation. It consisted of mitral annuloplasty (MAP) with a 30-mm semirigid total ring, papillary muscle approximation (PMA), and papillary muscle suspension (PMS). All procedures were performed by a superior transseptal approach. Although the mitral leaflet was normal, the anterior papillary muscle (PM) was entirely scarred and slightly tethered to the apex compared to the posterior PM. PMA joined the PM side-by-side by two pledgeted mattress sutures of 3-0 prolene through the mitral annulus. PMS fixed the distance between the approximated PM heads and the mitral annulus by a CV-4 expanded polytetrafluoroethylene (ePTFE) suture,[1] which was passed through the anterior portion of the MAP ring. The CPB and AXC times were 179 and 75 minutes, respectively. There was no MR on the intraoperative transesophageal echocardiography or the postoperative TTE. Although the patient needed an AAI pacemaker implantation as a result of sinus bradycardia after surgery, he recovered to New York Heart Association (NYHA) class I and was discharged on foot a month later. This case report was approved by the institutional review board, and informed consent to use medical records was obtained from the patient.

Discussion

Our surgical strategy for ICM with significant MR is to perform OLVP when the LVDd is larger than 65 mm and mitral complex reconstruction (MCR) that consists of MAP, PMA, and PMS when the distance between the PMs is greater than 30 mm. In this case, the LVDd was severely increased to 75 mm without significant functional MR. Mitral tethering was mild, and the PM distance was only 18 mm before surgery. Therefore we performed only left ventriculoplasty (OLVP) and CABG. Significant MR emerged just after surgery, and a second operation for MCR was necessary to relieve the patient's symptoms.

Table 1 and Fig. 1 show the change of the mitral complex geometry before and after the first operation.[2] The parameters in the 2-chamber view of TTE, which indicates posterior PM tethering, decreased more apparently than those in the 4-chamber view. This imbalanced improvement of mitral tethering may have caused the postoperative MR.

We consider two possible hypotheses for this pathology. First, the distortion of PMs by the left ventriculoplasty changed the subvalvular mitral apparatus and caused a new onset of MR. Second, the heterogeneity of the myocardial viability contributed to the postoperative PM function.

(1) We have never experienced such a significant new MR after left ventriculoplasty without MCR. Therefore we cannot confirm the first hypothesis, but we consider

Table 1. Echocardiographic findings

	Preop.		Postop.	
	4-chamber (APM)	2-chamber (PPM)	4-chamber (APM)	2-chamber (PPM)
Coaptation height, mm	9	11	11	7
Tenting area, cm^2	1.8	1.8	2.4	1.7
Tethering distance, mm	36	36	32	33
Papillary muscle depth, mm	28	29	24	19
Papillary muscle angle, °	45	50	38	35
Papillary muscle distance, mm	21		26	
LVDd, mm	75		62	
LVDs, mm	66		55	
Mitral regurgitation	0/4		4/4	

APM, anterior papillary muscle; PPM, posterior papillary muscle; LVDd, left ventricular end-diastolic dimension; LVDs, left ventricular end-systolic dimension.

that three-dimensional PM distortion can be made by any left ventriculoplasty procedures, including the Dor operation, and it may contribute to an early recurrence of MR after surgery.[3,4)]

(2) The preoperative DE-MRI showed a diffuse LV subendocardial DE, except for the basal posteroinferior area, and the wall thickening of that area was improved in QGS-SPECT. That of the anterolateral area did not significantly increase after surgery, which supports the second hypothesis.

Conclusion

We must consider a new MR after left ventriculoplasty even if the mitral tethering is mild and MR is not significant preoperatively.

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