Myectomy and mitral repair through the left atrium in hypertrophic obstructive cardiomyopathy: The preferred approach for contemporary surgical candidates?

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Objective: Patients with hypertrophic obstructive cardiomyopathy due to diffuse hypertrophy extending to or below the papillary muscles are poor candidates for alcohol septal ablation and suboptimal candidates for transaortic septal myectomy. In addition, the outflow obstruction is often aggravated by an abnormal mitral valve and subvalvular apparatus.

Methods: We performed transatrial myectomy in 12 patients with diffuse hypertrophy, who were highly symptomatic despite maximal medical therapy. All had at least moderate mitral regurgitation and systolic anterior motion. The anterior mitral leaflet (AML) was detached from commissure to commissure, allowing an easy myectomy through this AML toward the base of the anterior papillary muscle, with mobility fully restored. The abnormal chordae from the septum to the anterior papillary muscle and AML were divided. The continuity of this AML was restored with augmentation using an autologous pericardial patch. The height of the posterior mitral leaflet was reduced and the repair completed using an oversized annuloplasty ring.

Results: The peak intraventricular gradients decreased spectacularly from 98.8 $\pm$ 6.29 to 19.2 $\pm$ 13.4 mm Hg ($P < .001$), and the systolic anterior motion and mitral regurgitation disappeared. One patient died of left ventricular diastolic dysfunction. All other patients left the hospital in New York Heart Association class I or II.

Conclusions: We believe that this technique is preferable for patients with hypertrophic obstructive cardiomyopathy and diffuse hypertrophy extending to the midportion of the left ventricle or beyond. It results in disappearance of outflow tract gradients and allows correction of the mitral valve abnormality. (J Thorac Cardiovasc Surg 2014;147:1833-6)

Transaortic extended surgical septal myectomy (SSM) has generally been accepted as the preferred treatment of hypertrophic obstructive cardiomyopathy (HOCM) in patients with severe drug-refractory symptoms. This technique has been associated with very low mortality, consistent alleviation of outflow gradients and related symptoms, and excellent long-term survival.[1]

However, in recent years, the primary treatment strategy for typical asymmetric septal hypertrophy has, particularly in Europe, shifted toward percutaneous alcohol septal ablation (ASA), despite the suboptimal results in terms of complications, persistent symptoms, and the need for pacemaker implantation.[2] Thus, most contemporary surgical candidates will present with diffuse hypertrophy extending to or below the papillary muscles (PMs), with midcavity muscular obstruction. These patients are poor candidates for ASA but are also suboptimal candidates for the Morrow procedure.

In addition, the outflow obstruction in these patients will often be aggravated by an abnormal mitral valve and subvalvular apparatus. The PMs can be displaced anteroapically or can be attached, or even embedded, in the septal muscular hypertrophy. The posterior mitral leaflet (PML) is often longer with a diminished coaptation–septal distance. This results, together with a narrowed aortic–mitral angle, in systolic anterior motion (SAM) of the mitral valve.[3,4] Although the SAM causes mitral regurgitation (MR), great interindividual difference can occur for comparable degrees of SAM, depending on the geometry of the mitral valve and subvalvular apparatus. The incidence of some degree of MR in patients with HOCM has been reported to be up to 95%,[1] necessitating mitral valve repair in 20% of cases.[4] Centers with a large experience have repeatedly shown a decrease in both SAM and MR with an adequately performed extended SSM.[1,5] However, the persistence of SAM or MR after SSM can be difficult to predict, and treatment remains controversial, because mitral valve repair in the setting of HOCM can be challenging.

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Disclosures: Authors have nothing to disclose with regard to commercial support.

Presented at The 2013 American Association for Thoracic Surgery Mitral Conclave, New York, New York, May 2, 2013.

Received for publication April 29, 2013; revisions received June 24, 2013; accepted for publication July 1, 2013; available ahead of print Aug 28, 2013.

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0022-5223/$36.00

http://dx.doi.org/10.1016/j.jtcvs.2013.07.024

Abbreviations and Acronyms

AML = anterior mitral valve leaflet
ASA = alcohol septal ablation
HOCM = hypertrophic obstructive cardiomyopathy
LVOT = left ventricular outflow tract
MR = mitral regurgitation
PML = posterior mitral leaflet
PM(s) = papillary muscle(s)
SAM = systolic anterior motion
SSM = surgical septal myectomy

METHODS

Patient Selection

From January 2010 to March 2013, we operated on 12 patients with diffuse obstructive hypertrophy. The indications for SSM included drug-refractory class III or IV symptoms (n = 11), an episode of heart failure (n = 1), and a blood pressure decrease of more than 10 mm Hg at peak exercise (n = 3). One patient had undergone previous ASA.

Patient Characteristics

The mean patient age was 53.0 ± 10.5 years (range, 34-61), and 83% were men. All 12 patients had diffuse hypertrophy extending below the level of the PMs, with an average peak left ventricular outflow tract (LVOT) gradient of 98.8 ± 6.29 mm Hg and a mean septal thickness of 25 ± 6 mm. Most patients (92%) had moderate to severe MR (mean 2.6 ± 0.8) and SAM. The color flow jet of the MR was either central or posteriorly directed, suggesting the SAM was responsible for the MR.

Surgical Data

All patients underwent surgery under normothermia, with a mean cardiopulmonary bypass time of 243 ± 88 minutes. Intermittent antegrade warm blood cardioplegia was administered every 15 minutes for 3 or more minutes. The mean aortic crossclamping time was 148 ± 30 minutes.

The mitral valve was exposed through a transatrial septal approach. The anterior mitral valve leaflet (AML) was detached from the annulus from commissure to commissure, providing excellent exposure of the septum to the apex of the heart. SSM was then performed, starting 5 mm under the aortic annulus just in the middle of the insertion of the AML. It was prolonged horizontally to the left to the limit of the outflow tract, with a depth of at least 1.5 cm less than the preoperatively measured depth. The myectomy was taken downward to the base of the anterior PMs over the same thickness, preferably in 1 block, using the combination of a no. 11 bladed knife and a stitch cutter, which allowed for easy appreciation of the resection depth. The mean depth of this muscle resection was 11.7 ± 2.89 mm. Starting the resection at the midpoint of the AML insertion prevents injury to the bundle of His, which is situated in the vicinity of the mitral posteromedial commissure. The anterior PM embedded in the hypertrophy was carefully mobilized, and abnormal chordae from the septum toward the anterior PM and AML were divided (Figure 1). The AML was reconstructed with an autologous untreated pericardial patch, generously sized in breadth and height to account for the stitching space, using a running locked 5-0 polypropylene suture (Pronova, Johnson & Johnson, New Brunswick, NJ). If the height of the PML was more than 2 cm (92% of cases), the PML was incised 5 mm from, and parallel to, the posterior annulus and resutured, reducing the height to approximately 1 cm. The height of the "new" AML was measured, and a corresponding complete and semirigid ring (Physio II, Edwards LifeSciences, Irvine, Calif) was implanted, carefully avoiding undersizing (mean size, 34.0 ± 1.6).

Statistical Analysis

Statistical analyses were performed using Statistica statistical software (StatSoft, Tulsa, Okla). The results for the continuous data are reported as the mean ± standard deviation or median and range, as appropriate. Paired *t* tests were used to compare continuous parameters, and discontinuous data were compared using the Fisher exact test. $P < .05$ was considered statistically significant.

RESULTS

Intraoperative transesophageal echocardiography showed a reduction in the interventricular thickness from 25 ± 6 mm to 15 ± 3 mm ($P < .001$). There was no or trivial MR (grade, 0.71 ± 0.49), with a mean coaptation length of 9.2 ± 0.7 mm and a posterior shift of the coaptation site, resulting in the complete disappearance of the SAM in all patients. Transthoracic echocardiography at discharge showed a spectacular alleviation of the peak intraventricular gradients (98.8 ± 6.29 vs 19.2 ± 13.4 mm Hg, $P < .001$).

Patch augmentation of the AML is time-consuming; however, the longer aortic crossclamping time (mean, 148 ± 30 minutes) did not result in greater postoperative troponin T levels, despite the hypertrophy. In addition, the long aortic crossclamping time did not correspond to the ischemic time in our practice, because we have chosen to optimize the myocardial protection in these hypertrophic hearts by delivering warm blood cardioplegia for 3 to 4 minutes every 15 minutes. However, after unclamping the aorta, the suboptimal diastolic function required a precise optimization of the preload and, therefore, a longer reperfusion time.

In 1 patient, severe pulmonary hypertension persisted (preoperative systolic pulmonary artery pressure, 80 mm Hg), with subsequent pulmonary edema immediately after weaning from cardiopulmonary bypass, necessitating the use of extracorporeal membrane oxygenation. He eventually died of left ventricular diastolic dysfunction and subsequent right ventricular failure. The other 11 patients were discharged after a median hospital stay of 15 days, and after 4 weeks, all were in New York Heart Association class I or II. One patient required a pacemaker for complete atrioventricular block (right bundle branch block was present after previous ASA).

DISCUSSION

The technique of leaflet extension (through the aortic valve) and transmitral myectomy have been previously described[6-8]; however, we have used a combination of transmitral myectomy with the classic Carpentier techniques of mitral valve repair.

Diffuse Hypertrophy With Embedded Papillary Muscles

Despite the excellent long-term results and low complication rate of SSM, the use of ASA has continued to increase, especially in Europe. Therefore, contemporary