

Septal Myectomy, Papillary Muscle Resection, and Mitral Valve Replacement for Hypertrophic Obstructive Cardiomyopathy: A Case Report

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We report a case of hypertrophic obstructive cardiomyopathy (HOCM) successfully treated with septal myectomy and mitral valve replacement (MVR) combined with a resection of the hypertrophic papillary muscles. The patient, a 74-year-old woman, first underwent the conventional septal myectomy through aortotomy. The papillary muscles revealed a marked hypertrophy, but extended myectomy and precise resection of the hypertrophic papillary muscles were thought to be difficult through the aortotomy. Through the right-sided left atriotomy, MVR and resection of the papillary muscles were additionally performed. The patient was smoothly weaned from the cardiopulmonary bypass, and the postoperative course was uneventful. The outflow pressure gradient was relieved to 0 mm Hg, from 94. The mean pulmonary artery pressure was reduced to 27 mm Hg, from 42. The patient has been doing well in the New York Heart Association (NYHA) functional class between I and II during 45 months of follow-up, without complications related to the use of a prosthetic valve. Septal myectomy is the procedure of choice in the surgical treatment of HOCM for most cases, but some may require additional mitral valve procedures. In patients with marked hypertrophic papillary muscles, MVR and resection of the muscles may be an option of treatment to ensure a relief of the outflow obstruction and to abolish systolic anterior movement in units with limited experience. (*Ann Thorac Cardiovasc Surg* 2008; 14: 258–262)

Key words: hypertrophic obstructive cardiomyopathy, septal myectomy, mitral valve replacement

Introduction

Surgery to relieve obstruction in the left ventricular (LV) outflow tract is required for a few symptomatic patients among those with hypertrophic obstructive car-

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diomyopathy (HOCM). Conventional septal myectomy has been a standard procedure for these patients, but controversy still exists regarding the surgical treatment of choice. Septal myectomy has been reported to be effective for relieving systolic anterior motion (SAM) in the majority of patients with HOCM.¹⁻⁴ Cooley et al., however, advocated that mitral valve replacement (MVR) is more effective for relieving both obstruction of the outflow tract and functional mitral regurgitation relative to septal myectomy for units having limited experience with ventricular septal procedures.^{5,6} In Japan, MVR is often selected as the treatment of choice rather than myectomy because of the shortage of surgical experiences for symptomatic patients with HOCM in each institute. We present a case of HOCM requiring surgi-

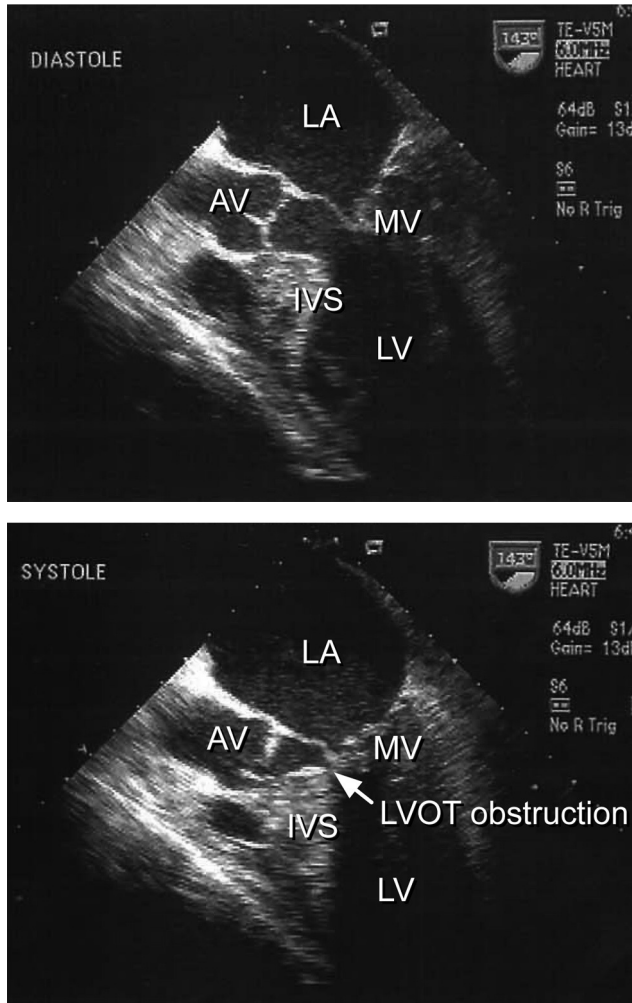


Fig. 1. Transesophageal echocardiogram (TEE) at diastolic (A) and systolic phase (B). TEE exhibited a hypertrophic septum in a maximum thickness of 23 mm, grade 3 mitral regurgitation (MR), and systolic anterior motion (SAM) of the mitral valve (MV). LV, left ventricle; LA, left atrium; MV, mitral valve; AV, aortic valve; LVOT, left ventricular outflow tract; IVS, interventricular septum.

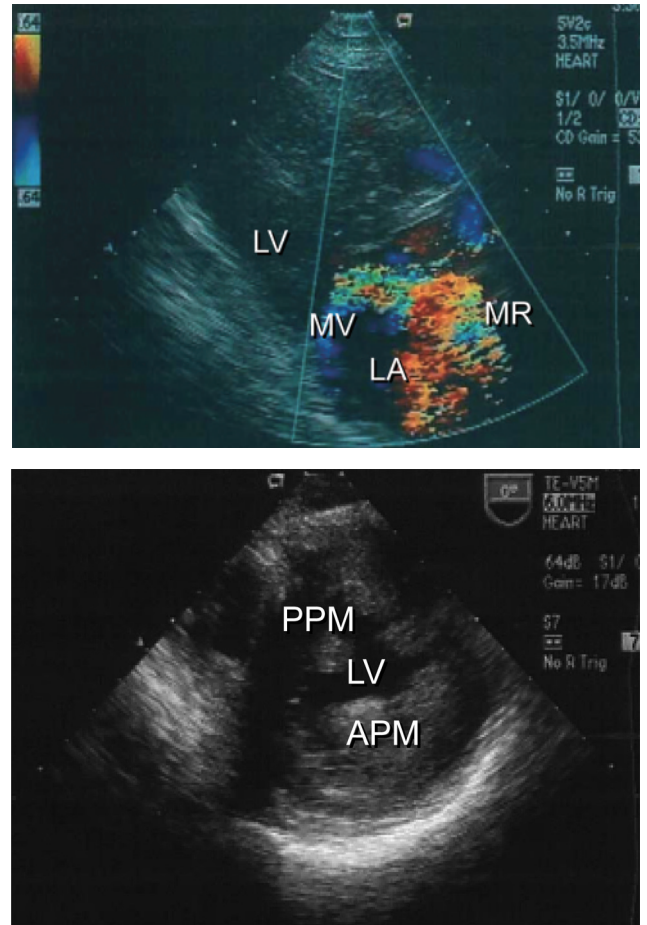


Fig. 2. Echocardiogram at systolic phase showing grade 3 mitral regurgitation (MR) (A) and transesophageal echocardiogram (TEE) showing hypertrophic papillary muscles (B). LV, left ventricle; LA, left atrium; MV, mitral valve; MR, mitral regurgitation; APM, anterior papillary muscles; PPM, posterior papillary muscles.

cal treatment, which is relatively rare in Japan, to relieve obstruction in the LV outflow tract and functional mitral regurgitation. Since the papillary muscles revealed a marked hypertrophy through the aortotomy view, MVR and resection of the papillary muscles were performed in addition to septal myectomy in this case.

Case Report

A 74-year-old woman was diagnosed as having HOCM

in August 2000, when the pressure gradient of the LV outflow tract was 60 mm Hg. The patient recurrently showed the symptoms of heart failure and was indicated for surgery. However, she had refused surgical treatment and received only medical treatment with β -blocker. In February 2003, the patient was admitted to our hospital again as a result of heart failure.

On admission, an echocardiogram (UCG) revealed a markedly elevated right ventricular (RV) pressure, hypertrophic interventricular septum in a maximum thickness of 23 mm, grade 3 mitral regurgitation (MR), and systolic anterior motion (SAM) of the mitral valve (Figs. 1 and 2). LV diastolic dimension (LVDD), LV systolic dimension (LVDs), left atrial dimension (LAD),

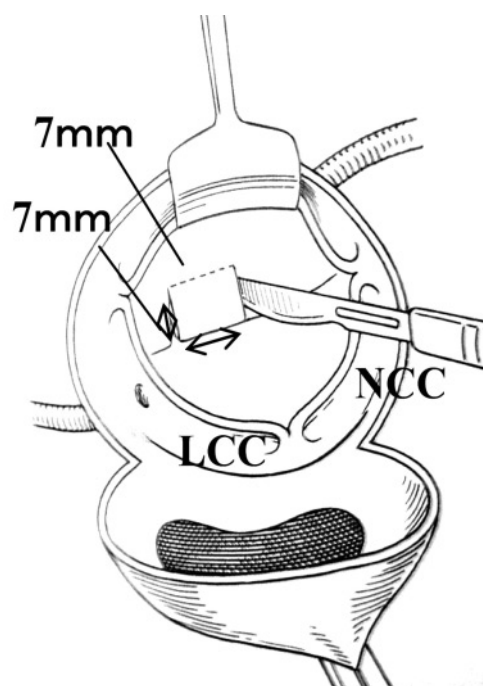


Fig. 3. Schema of septal myectomy through the aortotomy view. The total resection of the septal bulge measured 7 mm wide, 7 mm deep, and 12 mm long. NCC, noncoronary cusp; LCC, left coronary cusp.

and estimated RV systolic pressure were 46.4 mm, 22.1 mm, 52.1 mm, and 65 mm Hg, respectively. The left ventriculography (LVG) also showed grade 3 MR, ejection fraction of 85%, and a markedly hypertrophied ventricular septum. The pressure data (mm Hg) were as follows: PCWP (23), PA 71/26 (42), RV 70/4, RA (6), LV 190/6, AO 96/60 (PCWP, pulmonary capillary wedge pressure; PA, pulmonary artery; RA, right atrium; AO, aorta). Since the patient realized the deterioration of symptoms as the pressure gradient of the LV outflow tract was increased to 94 mm Hg, she finally consented to surgical treatment.

The operation was performed on February 25, 2003. A written informed consent was obtained before the operation after a full explanation. The conventional septal myectomy was first undertaken as a standard procedure through aortotomy under cardiopulmonary bypass. External pressure was slightly placed on the RV and the septum to push the septal bulge into aortotomy view (Fig. 3). A 4 mm incision toward the left coronary cusp (LCC) and a 3 mm incision toward the noncoronary cusp (NCC) were made from the center of the hypertrophied septum. A total resection of the sep-

tal bulge measured 7 mm wide, 7 mm deep, and 12 mm long. After myectomy, an observation of the LV cavity revealed more-markedly hypertrophied papillary muscles than had been expected preoperatively, which might reduce the LV diastolic volume. Moreover, the papillary muscles were thought to cause a redirected blood flow that would catch the mitral valve leaflets leading to the persistent SAM. An extended myectomy of the septal bulge below the mitral leaflet tips and a precise resection of hypertrophied papillary muscles were thought to be difficult through the aortotomy. Thus MVR and the resection of the hypertrophied papillary muscles were additionally performed through the right-sided left atriotomy to ensure the relief of SAM. A declamping of the aorta to observe the LV outflow tract was performed not before MVR, but after completing it and the resection of the hypertrophied papillary muscles. The time of operation, cardiopulmonary bypass, and aortic cross-clamping were 240, 163, and 114 min, respectively. The patient was smoothly weaned from the cardiopulmonary bypass, and the postoperative course was uneventful.

The pressure data measured on the 4th postoperative day were as follows: PCWP (16), PA 49/16 (27), RV 44/0, RA (0), LV 157/0, and AO 162/64 (mm Hg). The outflow pressure gradient was relieved to 0 mm Hg, from 94. The mean pulmonary artery pressure was also reduced, to 27 mm Hg, from 42. Postoperative LVDD, LVDs, and LAD measured by UCG were 36.7 mm, 21.7 mm, and 35.1 mm, respectively. There was no deterioration of cardiac function resulting from the resection of hypertrophied papillary muscles in the postoperative course. The patient was discharged from the hospital on foot on the 37th postoperative day without heart block. She has been doing well in the New York Heart Association (NYHA) functional class between I and II during 45 months of the follow-up period. There have been no complications related to the use of a prosthetic valve, such as valve failure, embolism, or warfarin-induced serious hemorrhaging.

Figure 4 shows histological findings of the resected papillary muscles. There were transformed and/or concentrated nuclei in the myocardial cells and physaliform nucleus, which were consistent with the findings of hypertrophic myocardium. Microscopic examination of the resected mitral valve leaflets showed myxoid degeneration and hyalinization, which supported in part the validity of MVR.

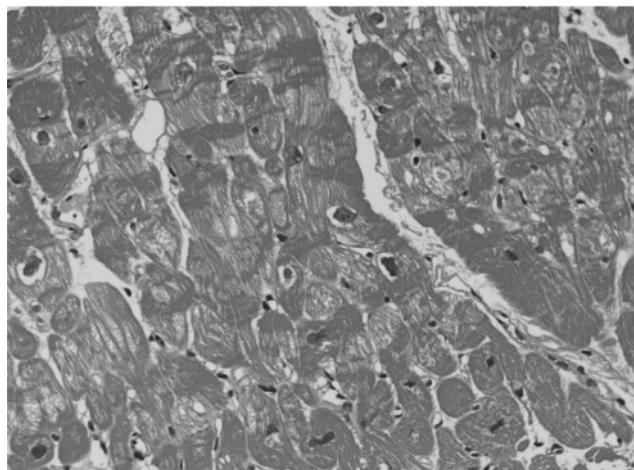


Fig. 4. Histological findings of the resected papillary muscles stained with hematoxylin and eosin ($\times 200$).

There were transformed and/or concentrated nuclei in the myocardial cells and physaliform nucleus, which were consistent with the findings of hypertrophic myocardium.

Discussion

Surgical treatment for HOCM is among the more technically challenging of cardiac operations for acquired disease.⁷⁾ The conventional septal myectomy has been a standard procedure to relieve SAM and obstruction of the outflow tract; however, it is often difficult to accomplish because of the limited exposure of the septal bulge through the aortotomy view. Part of the septal bulge cannot be easily seen, which may cause imprecision in the extent of myectomy leading to an inadequately small resection with persistent obstruction, or to a large resection resulting in iatrogenic ventricular septal defect or complete heart block.⁷⁾

In the present case, the conventional septal myectomy was first undertaken to relieve the obstruction of LV outflow through aortotomy. Because the papillary muscles revealed a marked hypertrophy through the aortotomy view, MVR and a resection of the papillary muscles were additionally performed to abolish SAM. In general, the purpose of septal myectomy is not to enlarge LV outflow tract anatomically, but to relieve SAM of the mitral valve, the main cause of obstruction in most patients with HOCM. Although septal myectomy has been reported to be effective for most patients with HOCM, MVR may be required under some circumstances, as follows: (i) organic changes are present in the mitral valve leaflets or subvalvular

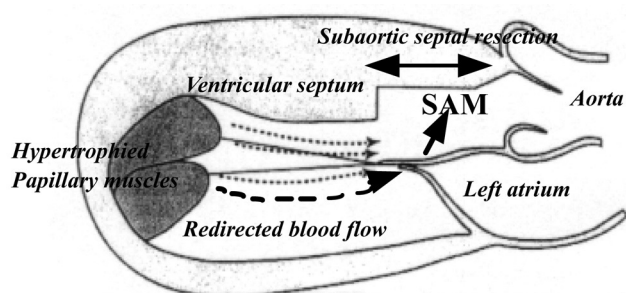


Fig. 5. Schema of the feasible flow mechanism after subaortic septal resection.

Blood flow is still redirected by the remaining septal bulge so that it comes from a posterior direction. It may catch the mitral valve and lead to systolic anterior motion (SAM), resulting in the obstruction of the outflow tract.

tissues; (ii) isolated myectomy is thought to be inadequate to relieve SAM; (iii) in a few cases in which only SAM cannot account for the obstruction of LV outflow tract in the presence of hypertrophied myocardium.^{5,7)} In the presence of extremely hypertrophied papillary muscles, as in our case, a resection of the subaortic septum may not be enough to relieve SAM of the mitral valve. The impact of the resection of the subaortic septum on the blood flow is limited to only the tips of the mitral leaflets; thus flow is still redirected by the remaining septal bulge so that it comes from a posterior direction (Fig. 5).⁷⁾ It may catch the mitral valve leaflets and still lead to SAM, resulting in an obstruction of the LV outflow.

Recently, a modification of the septal myectomy, termed extended myectomy, mobilization and partial excision of the papillary muscles, has been performed at centers with extensive experience.^{7,8)} A partial excision of papillary muscles is thought to contribute to redirecting flow anteriorly, away from valve leaflets, thus relieving SAM. However, this procedure requires advanced technical skills, and the determination of the extent of myectomy would be difficult especially in units having limited experience with septal procedures. Although there are many drawbacks to using a prosthetic valve in the treatment, MVR may assure the relief of SAM and mitral regurgitation. It was reported that mitral valve procedure in addition to septal myectomy was identified as a risk factor related to the adverse influence on postoperative survival.⁹⁾ But it is the most important procedure to relieve SAM in the

surgical treatment of HOCM; thus additional mitral valve procedure would be necessary when isolated septal myectomy is thought to be inadequate. A resection of the papillary muscles may influence postoperative cardiac function, but in this case it offered an enlargement of the LV cavity and satisfactory late results. Conventional septal myectomy was first attempted in the present case, but it remains to be determined whether only MVR is effective for relieving obstruction in such cases of HOCM.

In summary, we report a case of HOCM successfully treated with septal myectomy and MVR combined with a resection of the hypertrophic papillary muscles. The outflow pressure gradient was relieved, to 0 mm Hg, from 94. The patient was discharged from the hospital on foot without heart block and has been doing well in NYHA functional class between I and II for 45 months after surgery. When isolated septal myectomy is thought to be inadequate to relieve SAM, additional MVR may be a practical option of treatment at centers with limited surgical experience.

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