A Successful Case of Percutaneous Transluminal Septal Myocardial Ablation for Mitral Regurgitation that Emerged Following Mitral Valve Repair Surgery

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Abstract

A 70-year-old man complaining of exertional dyspnea was referred to our hospital for recurrence of mitral regurgitation (MR) after mitral valve repair. Echocardiography revealed the presence of a bulging subaortic septum with flow acceleration in the left ventricular outflow tract (LVOT) and systolic anterior motion of the mitral valve with moderate MR, consistent with LVOT obstruction. Since medical therapy was not sufficiently effective, we performed percutaneous transluminal septal myocardial ablation (PTSMA). After the procedure, the MR resolved, with a reduced LVOT pressure gradient. The patient’s symptoms improved. PTSMA was effective for the treatment of MR that emerged after mitral valve repair.

Key words: percutaneous transluminal septal myocardial ablation, mitral valve repair, mitral regurgitation, left ventricular outflow tract obstruction, bulging subaortic septum, echocardiography

Introduction

Mitral valve repair is considered to be the first-line therapy for mitral regurgitation (MR). The advantages of mitral valve repair over replacement include reduced operative risks, less possibility for heart failure and superior long-term survival (1). However, mitral valve reoperation is needed in cases of recurrent MR, and this problem remains a clinically unresolved issue (2). We herein report the case of a patient with MR that emerged after mitral valve repair who successfully avoided reoperation by receiving percutaneous transluminal septal myocardial ablation (PTSMA), although PTSMA is commonly used in patients with hypertrophic obstructive cardiomyopathy (3).

Case Report

A 70-year-old man with a history of essential hypertension developed dyspnea on exertion (NYHA class II) and was admitted to another hospital. Echocardiography showed severe MR accompanied by prolapse of the middle scallop and a bulging subaortic septum (Fig. 1). The left ventricle (LV) was not dilated (LV end-diastolic/end-systolic diameter=53/30 mm) and the LV contractions were preserved. Mitral valve repair was performed for symptomatic MR, and the sliding leaflet technique was used to prevent postoperative systolic anterior motion of the mitral valve (SAM). Transesophageal echocardiography performed immediately after the mitral valve repair revealed only mild MR without SAM. However, after the surgery, moderate MR appeared along with a reduction in the LV cavity (LV end-diastolic/end-systolic diameter=42/22 mm). The blood flow through the left ventricular outflow tract (LVOT) was accelerated, with a peak pressure gradient of 64 mmHg and SAM. Since the patient’s symptoms were not relieved by the mitral valve repair, and instead became even more severe, he was referred to our hospital.
At that time, the patient experienced dyspnea even while walking short distances (NYHA class III). The blood pressure was 110/55 mmHg, and the pulse rate was 66 beats per minute. A chest X-ray obtained on admission did not show pulmonary congestion. An electrocardiogram disclosed a sinus rhythm with high voltage in the precordial leads. Echocardiography demonstrated mild concentric LV hypertrophy with a wall thickness of 11 mm and a bulging subaortic septum (proximal septum=15 mm) with a normal systolic function (Fig. 2). There was no prolapse; however, moderate MR was observed due to SAM. The blood flow was accelerated in the LVOT where the subaortic septum was bulging, with a peak pressure gradient of 125 mmHg (Fig. 3). Cardiac catheterization showed no coronary artery stenosis, with stable systemic hemodynamics and a preserved cardiac output. LV angiography demonstrated mild MR (Sellers classification 2/4). Simultaneous pressure measurement of both the LV and aorta revealed a small difference in peak systolic pressure between the LV and aorta; however, the pressure difference dramatically increased to 100-120 mmHg following premature ventricular contractions (PVCs), indicating a positive Brockenbrough-Braunwald sign (Fig. 4). We determined that the dyspnea was due to MR secondary to LVOT obstruction emerging after the mitral valve repair. When we treated the patient with a beta-blocker, neither his symptoms nor LVOT obstruction improved, leading us to consider repeated cardiac surgery; however, the patient declined this procedure. Therefore, we decided to perform PTSMA since the mechanism underlying the patient’s functional MR was similar to that of obstructive hypertrophic cardiomyopathy.

With attention to the possibility for atrioventricular block, ethanol was slowly injected into the major septal branch of the left anterior descending coronary artery. After PTSMA, the difference in peak systolic pressure between the LV and aorta was reduced to 3.4 mmHg, even after PVC provocation. Post-PTSMA LV angiography showed no MR.

Echocardiography also revealed an improvement of the LVOT obstruction (peak pressure gradient, 12 mmHg) and MR (mild) with disappearance of SAM (Fig. 5). Since then, the patient has been free from symptoms (NYHA class I) and was discharged eight days after PTSMA without any complications.

**Discussion**

**Recurrence of MR after mitral valve repair**

Mitral valve repair is an established procedure that provides excellent long-term clinical outcomes to the levels observed in the general population. However, cases of recurrent MR after mitral valve repair have been reported. In one report, reoperation was performed in 6.2% of patients during 20 years of follow-up (2). Progressive degeneration of the mitral valve (i.e., leaflet prolapse or thickening) and suture...
dehiscence are considered to be the main mechanisms of MR recurrence after mitral valve repair (4). In the present case, transthoracic and transesophageal echocardiography showed neither of these findings. Since LVOT obstruction due to a bulging subaortic septum was induced by provocation, the LVOT obstruction was thought to be the cause of the MR recurrence. After PTSMA, both the MR and dyspnea on exertion were corrected. Recurrent MR secondary to LVOT obstruction was confirmed as the cause of the patient’s dyspnea.
before PTSMA after PTSMA

![Diagram showing echocardiography before and after PTSMA.](image)

**Figure 5.** Echocardiography before and after PTSMA. Before PTSMA, a bulging subaortic septum was present (*). This led to LVOT flow acceleration accompanied by SAM (red triangle), resulting in moderate MR (A and B). After PTSMA, the SAM resolved and the MR became milder, with an LVOT peak pressure gradient of 12 mmHg (C and D).

**The LVOT obstruction emerged after mitral valve repair**

In the present case, two mechanisms were considered to be the causes of LVOT obstruction. One mechanism is LV anatomical changes, and the other is a bulging subaortic septum.

Anatomical changes in the form of anterior displacement of the mitral valve coaptation point after mitral valve repair may produce LVOT obstruction (5). In patients with relatively elongated posterior leaflets, reductions in the mitral annulus diameter or LV volume after mitral valve repair cause an anterior shift of the coaptation point closer to the interventricular septum, thereby producing SAM (6). SAM causes failure of normal coaptation of the leaflets in mid to late systole, which leads to MR (7). The occurrence of SAM after mitral valve repair has been described as being a cause of recurrent MR, with an incidence ranging from 1% to 2% (8-10). Not all patients with SAM develop significant MR. Schwammenthal et al. found that a greater degree of MR was induced by SAM if the mobility of the posterior leaflet was limited (11). In this case, the sliding leaflet technique, which reduces the height of the posterior leaflet, was performed to prevent SAM and LVOT obstruction (12). However, this technique did not prevent SAM. The restricted mobility of the posterior leaflet due to mitral valve repair could have caused significant MR induced by SAM.

Another possible mechanism of LVOT obstruction is a bulging subaortic septum, often observed in elderly atherosclerotic hearts due to aortic dilatation and fibrotic changes of the interventricular septum (13, 14). This condition usually follows a benign time course; however, in some cases, LVOT obstruction with hemodynamic changes leads to acute decompensated heart failure (15). Civelek et al. reported the incidence of recurrent MR due to LVOT obstruction to be 1.4% in patients with a septal bulge observed intraoperatively. The authors successfully prevented postoperative LVOT obstruction and MR by surgically resecting the septal bulge (16). However, in our case, we did not observe MR immediately after mitral valve repair, and the condition occurred late after surgery. The large LV cavity, which remained just after the mitral valve repair, may have masked the LVOT obstruction and MR. In the late period after surgery, the LV cavity became smaller, with a reduction in the volume overload following mitral valve repair. The size of the LV cavity after surgery was smaller than expected, which may be due to the patient’s history of hypertension. We assumed that the smaller LV cavity contributed to the changes in the geometry of the papillary muscles and the development of LVOT obstruction and MR. However, this assessment was difficult to make intraoperatively.

No known cause of recurrent MR after mitral valve repair is found in approximately 3% of patients (4). The present case emphasizes the importance of LVOT obstruction as a cause of MR.

**Percutaneous procedure**

In recent years, minimally invasive treatment has become popular in the treatment of valvular heart disease. Honěk and colleagues reported a similar case which showed that PTSMA can be used to manage post-mitral valve repair LVOT obstruction with a bulging subaortic septum noted prior to surgery (manuscript in preparation). They observed recurrent MR after mitral valve repair with the implantation of an annuloplasty ring and neochordae. PTSMA was effective in preventing reoperation, as observed in our case. However, their operation did not use the sliding plasty technique, and the recurrent MR could have been avoided by using this technique in the original repair.

In contrast, our patient underwent mitral valve repair with implantation of an annuloplasty ring and sliding plasty of the posterior leaflet to prevent SAM. In addition to the re-
port by Honěk et al., we showed that PTSMA is an effective treatment, regardless of the previous operative procedure, in patients with recurrent MR after mitral valve repair due to a bulging subaortic septum. Compared to surgical resection, PTSMA can achieve similar results less invasively for the treatment of recurrent MR due to LVOT obstruction with a bulging subaortic septum. A growing number of elderly patients receive mitral valve repair due to advances in perioperative management. Since the incidence of a bulging subaortic septum and the risk for reoperation is relatively high in such patients, PTSMA is therefore considered to be an important therapeutic option.

PTSMA is effective for the treatment of recurrent MR due to LVOT obstruction in patients with a bulging subaortic septum after mitral valve repair. If a patient complains of dyspnea with recurrence of MR after mitral valve repair, then the presence of LVOT obstruction should be considered.

The authors state that they have no Conflict of Interest (COI).

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