Transluminal Percutaneous Septal Myocardial Ablation in a Patient With Hypertrophic Obstructive Cardiomyopathy

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Reduction of septal mass by inducing septal infarction using catheter techniques is a new therapy for hypertrophic obstructive cardiomyopathy (HOCM). We report a case of severe HOCM that was dramatically improved by this non-surgical treatment. A 60-year-old woman with HOCM had suffered dyspnea (NYHA class III) with syncopal attack despite medical treatment. Left heart catheterization showed a resting pressure gradient across the left ventricular outflow tract (156 mmHg). Two proximal septal branches of the anterior descending coronary artery were catheterized with a balloon catheter by the usual percutaneous coronary angioplasty techniques and were completely blocked by injection of absolute alcohol. The pressure gradient decreased to 26 mmHg after the procedure. Symptoms were markedly improved (NYHA class I) without any medical treatment. The reduced pressure gradient was maintained at the 3-month follow-up catheterization (36 mmHg). Further long-term follow-up is needed, but this treatment would seem to be a promising technique for reducing pressure gradient in symptomatic patients with HOCM. (Jpn Circ J 1998; 62: 537 – 540)

Key Words: Hypertrophic obstructive cardiomyopathy; Septal infarction; Percutaneous myocardial ablation

Some patients with hypertrophic cardiomyopathy who have evidence of left ventricular outflow tract (LVOT) obstruction suffer symptoms of angina, dyspnea, and syncope as a consequence. Medical treatment1–3 or dual-chamber pacing4,5 can reduce LVOT obstruction and diminish symptoms. Patients who do not respond to these treatments require surgical resection of the affected portion of the interventricular septum6–8. However, surgery requires extracorporeal circulation and is associated with a moderate surgical risk of at least 5%.6,8 Sigwart9 was the first to attempt a non-surgical technique to reduce septal mass by inducing septal infarction using catheter techniques. He reported that inducing septal necrosis by injecting alcohol into the first major septal artery decreased the degree of LVOT obstruction and improved clinical symptoms in patients with severe hypertrophic obstructive cardiomyopathy (HOCM). Knight et al.10 have reported the efficacy of this novel technique in immediate and short-term follow-up results. We report here a case of severe HOCM that was dramatically improved by this non-surgical treatment.

Case Report

A 60-year-old woman with HOCM who, despite medical treatment, had suffered dyspnea on effort for approximately 10 years experienced exacerbation of symptoms (class III, New York Heart Association classification) with syncope attack and was admitted to our hospital. She was found to have a 4/6 ejection murmur. Electrocardiography revealed pronounced left-ventricular (LV) hypertrophy with strain, and echocardiography showed asymmetrical septal hypertrophy, systolic anterior movement of the mitral valve, and slight mitral incompetence. The resting pressure gradient across the LVOT assessed by Doppler echocardiography was more than 100 mmHg.

Left heart catheterization was carried out through the right radial artery under local anesthesia. Continuous pullback pressure recording from the LV apex up to the aorta using a 6F pigtail catheter revealed a systolic pressure gradient of 150 mmHg in the LVOT, ie 266 mmHg in systole at the midzone and apex in LV and 116/74 mmHg in the ascending aorta. There was no abnormality of the aortic valve. No provocative maneuver, such as dobutamine infusion, was attempted. Left ventriculography also showed a hyperkinetic contraction pattern with systolic obstruction in LVOT. Coronary angiography demonstrated no significant obstructive lesions.

To reduce this severe resting pressure gradient, transluminal percutaneous septal myocardial ablation was considered to be indicated because the patient rejected any surgical treatment, including permanent pacemaker implantation. After adequate explanation of the procedure, including its possible risks, informed consent was obtained from the patient and her family. The ethics committee in our institution approved the use of this new treatment for this patient.

A 8F percutaneous transluminal coronary angioplasty (PTCA) guiding catheter (Judkins’ type) was inserted into the left coronary artery through the right femoral artery and a 6F pigtail catheter was inserted into the apical left ventricle through the right brachial artery (Fig 1A). A resting pressure gradient of 156 mmHg was measured simultane
A temporary pacing catheter was placed at the right ventricle. The first major septal branch of the left anterior descending coronary artery (LAD) was identified and catheterized with a 2-mm over-the-wire system balloon catheter. After balloon inflation at 2 atm, the guidewire was withdrawn and a test injection of contrast medium was made through the catheter to confirm that the cannulated vessel supplied the correct area of the myocardium. After intravenous administration of diamorphine (5 mg), 2 ml of absolute alcohol was slowly injected through the lumen of the balloon catheter (Fig 1C) and left in situ for 5 min with the balloon remaining inflated. The patient did not complain of chest pain. Angiography after deflation of the balloon showed occlusion of the septal branch; however, a gradient of 132 mmHg still remained (Fig 1D). At the second trial, another smaller proximal septal branch was catheterized with the same balloon (Fig 1E). The gradient fell to 63 mmHg with balloon inflation only (Fig 1F). Subsequently, 5 ml of alcohol was injected through the lumen of the inflated balloon (Fig 1G). The patient reported slight chest discomfort only at the start of the alcohol injection into the second septal branch. The gradient decreased to 26 mmHg after the injection (Fig 1H). The septal branch was blocked completely, however no flow disturbance was seen in the LAD (Fig 1I). Temporary pacing for complete atrio-ventricular (AV) block was not required and there was no ventricular arrhythmia, except for some simple ventricular premature beats during the procedure. LV end-diastolic pressure decreased from 17 mmHg to 13 mmHg following the procedure. Echocardiography just after the procedure showed a high echoic area in the proximal septum, probably because of the contrast and alcohol injection. Creatine kinase activity rose to a maximum of 1923 IU (MB isozyme 196 IU) after the procedure. Left catheterization was repeated the next day. The left coronary angiogram showed complete blockage of both septal

Fig 1. (A) Coronary angiogram before procedure. (B) Preprocedural pressure gradient was 156 mmHg measured simultaneously. (C) Alcohol was injected into the most major septal branch of the LAD. (D) The gradient of 132 mmHg remained. (E) Another smaller proximal septal branch was occluded with the inflated balloon. (F) The gradient decreased to 63 mmHg. (G) Alcohol was injected into this septal branch. (H) The gradient decreased to 26 mmHg. (I) Postprocedural angiogram showed both septal branches completely blocked.

Fig 2. Magnetic resonance imaging show asymmetrical septal hypertrophy before procedure (A) and the septal thinning with a complex change in intensity (B) at systole.

Fig 3. The 3-month follow-up angiogram shows both septal branches remaining blocked (A). The pressure gradient was 36 mmHg (B).
branches. However, just after contrast injection, the patient experienced ventricular fibrillation requiring 100-J and 200-J DC cardioversions for conversion to regular sinus rhythm. The pressure gradient was 43 mmHg, as assessed by pull-back of a pigtail catheter from the apical ventricle to the aorta. There were no adverse sequelae and the patient was discharged 12 days after the intervention.

The patient experienced considerable improvement in symptoms and on resumption of normal activities her dyspnea reduced to class I without any medical treatment. Two months later echocardiography revealed reduced thickness and contraction of the proximal septum and reduced systolic anterior movement of the mitral valve. Magnetic resonance imaging also revealed septal thinning with a complex change in intensity (Fig 2). Follow-up left heart catheterization and angiography were carried out 3 months after the procedure. It revealed that the two septal branches were completely blocked and simultaneously the pressure gradient was 36 mmHg (Fig 3). There was no change in these findings compared with immediate results after the procedure.

Discussion

It has been reported that obstruction of the LVOT occurs in a significant number of patients with HOCM, causing symptoms of angina, dyspnea, and syncope. Intravascular hemolysis can be seen in patients with severe obstruction. Established treatments for relief of symptoms in HOCM are negative inotropic drugs, dual-chamber pacing, and surgical resection. Beta-blockers and other negative inotropic drugs such as verapamil and disopyramide can reduce LVOT obstruction and diminish symptoms. However, many patients remain symptomatic despite taking these drugs. Dual-chamber pacing can also reduce the obstruction both by altering left ventricular excitation and by optimizing the timing of arterial contraction. This therapy has been recommended to patients unresponsive to medical treatment before considering surgery. Patients who do not respond to pacing require surgical intervention. The majority of patients who undergo surgical resection derive long-term symptomatic benefit without significant impairment of left ventricular function. However, surgery requires extracorporeal circulation and is associated with a moderate surgical risk of at least 5%.8

A non-surgical technique for achieving a reduction in septal mass by inducing septal infarction using catheter techniques has been reported by Sigwart. The first septal branches of the LAD supply the myocardium of the proximal interventricular septum, the area of the myocardium whose abnormal structure and function are responsible for the production of LVOT obstruction in HOCM. The induction of permanent septal necrosis by alcohol injection into the septal branches was shown to be effective in reducing the pressure gradients in 3 patients with severe HOCM. Knight et al have reported the efficacy of this novel technique in immediate and short-term follow-up results in the first consecutive 18 patients.

We treated a female patient with severe HOCM by this novel non-surgical technique. The pressure gradient across LVOT decreased markedly from 156 mmHg to 26 mmHg and the patient experienced a dramatic improvement in symptoms after this procedure. Three months after the procedure, the patient remained free of symptoms and left catheterization showed no significant change in the pressure gradient. Thus, this treatment should be considered a promising technique for LVOT gradient reduction in symptomatic patients with HOCM.

Nevertheless, we believe that this non-surgical therapy has several limitations. One problem is identification of the target septal vessel. In a minority of patients, the proximal interventricular septum is supplied by a number of small septal branches rather than one larger first septal artery. In our patient, the pressure gradient could not be greatly reduced simply by blocking the most major proximal septal artery, and alcohol injection in another small but more proximal septal branch was needed. Thus, it is important to recognize that in such patients alcohol injection may be required in more than one branch. Myocardial contrast echocardiography may be effective in identifying the target septal vessel.

The second problem is arrhythmias caused by myocardial ablation. It has been reported that about 50% of patients develop complete AV block and that some require a permanent pacemaker. Preprocedural placement of a temporary pacing catheter is thought to be essential. Ventricular fibrillation rarely occurs during the procedure but may occur afterwards as a result of irritability induced by myocardial necrosis. Our patient developed ventricular fibrillation the day after coronary angiography. Prophylactic administration of intravenous lidocaine should be considered. Careful monitoring is required for the first few days to control arrhythmias.

Finally, long-term follow-up is necessary to determine the effectiveness of this therapy. Several reports have already shown its efficacy in the immediate and mid-term. If clinical and hemodynamic improvement is maintained in the longer term then non-surgical septal reduction could be the most effective and the least invasive treatment in symptomatic patients who have HOCM despite receiving medical treatment. Careful evaluations will be required in many cases to compare this therapy with other established forms of therapy, such as pacing and surgery.

References