CASE REPORT

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Coil Embolization of Septal Branches in Hypertrophic Obstructive Cardiomyopathy

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ABSTRACT

Hypertrophic cardiomyopathy (HCM) has been associated with sudden death in young athletes. On long-term follow up, syncope, chest pain and dyspnea are well known frequent symptoms due to the dynamic left ventricular outflow tract (LVOT) obstruction, mitral regurgitation and diastolic dysfunction. To reduce the LVOT pressure gradient, septal myectomy and alcohol injection have recently been utilized. However, some complications have been reported with alcohol ablation of the septal branches, such as complete atrioventricular block and alcohol spill over to ablate the entire left anterior descending coronary artery. Herein, a case of coil embolization of the septal branches, with successful reduction of the LVOT pressure gradient, is reported in a patient with HOCM. **(Korean Circulation J 2004;34(7):706-710)**

KEY WORDS : Hypertrophic cardiomyopathy ; Ventricular outflow obstruction ; Therapeutic embolization.

Introduction

Hypertrophic cardiomyopathy (HCM) is characterized by left ventricular hypertrophy, typically of a nondilated chamber, without an obvious cause, such as aortic stenosis or systemic hypertension. Syncope, chest pain and dyspnea are well known frequent symptoms due to the dynamic LVOT obstruction, mitral regurgitation and diastolic dysfunction. To reduce the LVOT pressure gradient, septal myectomy and alcohol injection have recently been utilized. The induction of a limited therapeutic infarction, through alcohol ablation of the septal branch, leads to a reduction of the LVOT pressure gradients and associated symptoms. However, some complications have been reported with alcohol ablation of the septal branches, such as complete atrioventricular block and inadvertent spillage of alcohol into a non-target vessel. Herein, a case of coil embolization of septal branches, with successful reduction of LVOT pressure gradient, is reported in a patient with hypertrophic obstructive cardiomyopathy (HOCM).

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Case

A 57-year-old man was referred to our hospital with a history of fainting and a systolic murmur about 3 years ago. He had history of presyncope and symptoms of chest pain and dyspnea on exertion. He had smoked half a pack per day for 40 years. On physical examination, a grade 3/6 mid-systolic murmur was present at the left sternal border and cardiac apex. Echocardiography showed diffuse asymmetric septal hypertrophy and systolic anterior motion (SAM) of the chordae tendinae with flow acceleration at the mid-ventricular level during Valsalva maneuver (peak velocity: 2.9 m/sec). At first, 25 mg of atenolol, twice per day, was prescribed and the patient managed relatively well during his daily life. About 3

years later, he began to complain of dizziness and presyncope, with some chest discomfort. A treadmill exercise test showed positive finding, with ST-segment depression at the inferior and lateral leads during the post-exercise period. An echocardiography showed a significant resting pressure gradient at the level of the left ventricular outflow tract (peak gradient: 73 mmHg) (Figure 1). Coronary angiography showed normal coronary anatomy with three major septal branches with diameters of 1 to 1.5 mm. Therefore, it was decided to ablate the two proximal septal branches by means of transcatheter coil embolization. At first, a 0.014 inch Hi-Torque Whisper® MS (Guidant, Advanced Cardiovascular Systems, Temecula, CA, USA) guide wire was advanced into the target septal branch, and a 3F Renegade™ Hi-Flo microcatheter (Boston Scientific Scimed, Maple Grove, MN, USA) was advanced into the target septal branch along the guide wire. The Tornado® coils (Cook Inc, Bloomington, IN, USA), 0.018 inch, 3 mm in diameter and 2 cm in length, which came loaded in a cylinder, was held firmly against the catheter hub and advanced into the catheter shaft using the stiff end of a 0.014 inch guide wire. Once the coil was inside the catheter, the guide wire and cylinder were removed. The coil was advanced by pushing with the soft tip of a guide wire along the introducer catheter and deployed into the target septal branches. After successful coil embolization of the two septal branches (Figure 2), the pressure gradient of the LVOT disappeared (Figure 3). After the procedure, the peak creatinine kinase level rose to 316 IU/L and the CK-MB to 17 mg/mL 24 hours later. The patient had a transient febrile response, and antibiotics were used for several days. On the follow-up echocardiogram the day after the procedure, a persistent LVOT pressure gradient of 30 mmHg was noted, so 40 mg of verapamil, three times a day, was added to the atenolol upon discharge. A follow up echocardiography was performed 4 months later, which showed no significant in-

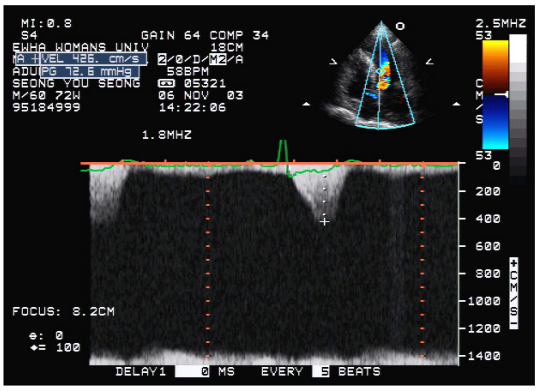

Figure 1. Doppler recordings of the left ventricular outflow tract (LVOT) gradient. It shows significant resting pressure gradient at the level of the LVOT (peak gradient: 73 mmHg).

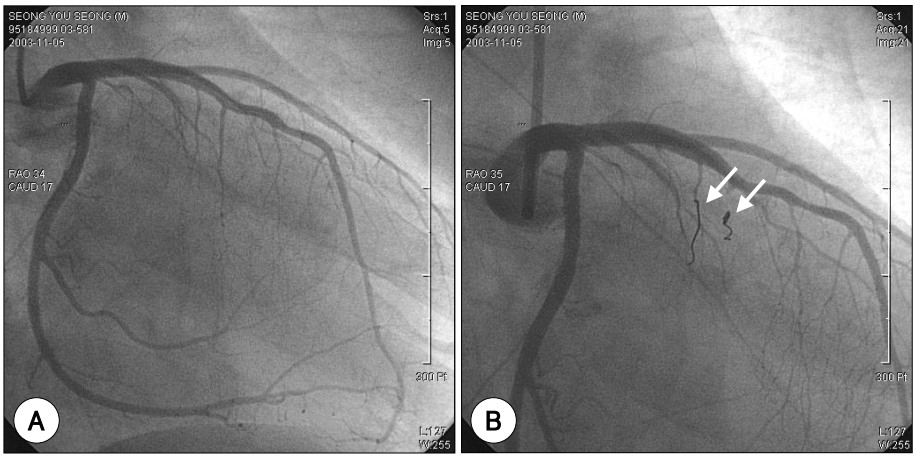

Figure 2. A: right anterior oblique view of the left coronary artery with several septal branches before the coil embolization. B: after the coil embolization, the 1st and 2nd septal branches (arrows) do not fill with contrast.

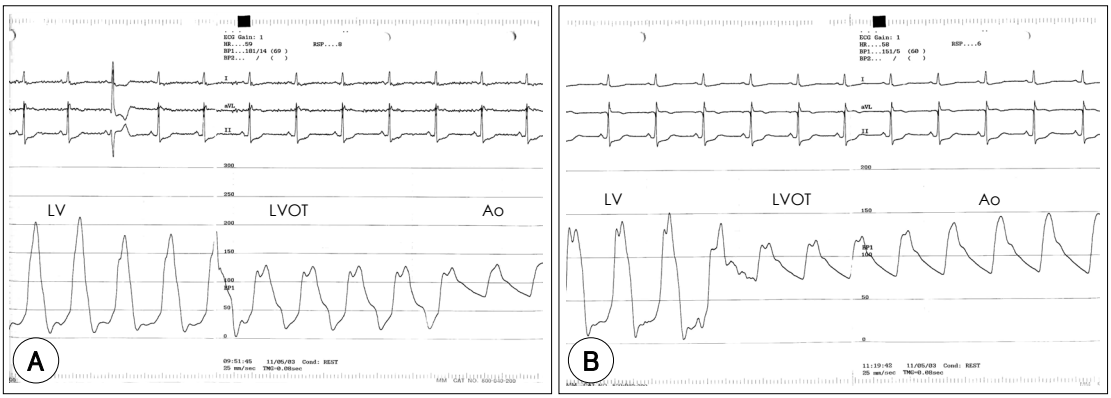

Figure 3. A: pressure curves of the left ventricle (LV), left ventricular outflow tract (LVOT) and aorta (Ao) before septal branch occlusion, showing a resting pressure gradient of 70 mmHg. B: after septal branch occlusion, the peak pressure gradient of the LVOT disappeared.

terval changes in the septal thickness. The LVOT pressure gradient was reduced at rest (PG: 30 mmHg), but with a significant pressure gradient present during Valsalva maneuver (PG: 63mmHg). The patient is doing well without dyspnea, presyncope or dizziness.

Discussion

Hypertrophic cardiomyopathy (HCM) is a common genetic abnormality that can occur in as many as 1 in 500 persons.[1] The cardinal feature is inappropriate myocardial hypertrophy, with no obvious cause of the hypertrophy (e.g. aortic stenosis or systemic hypertension), frequently with asymmetrical involvement of the septum, accompanied by a hyperdynamic systolic function.[1]

Park et al[2] has reported the features of HCM in Korea patients. The types of HCM were septal hypertrophy (61%), apical hypertrophy (33%) and midventricular hypertrophy (6%). Patients with septal hypertrophy were subdivided into non-obstructive (30%) and obstructive types (31%).

Patients with obstructive HCM typically complain of dyspnea, angina, presyncope and/or syncope on exertion. Patients with non-obstructive HCM rarely present with these symptoms, and usually the symptoms are milder. The clinical course of HCM is very variable. At present, the risk factors for sudden death are considered to be young age, syncope, a malignant family history, myocardial ischemia, sustained ventricular tachycardia on electrophysiological testing, ventricular tachycardia on ambulatory monitoring and massive hypertrophy with outflow obstruction.[1)3)4)]

Medically, beta blockers and other negative inotropic drugs, such as verapamil and disopyramide, can reduce the LVOT obstruction and diminish the symptoms.[5)6)]

In some patients, medical therapy ultimately proves insufficient for control of the symptoms. A myectomy for symptomatic HOCM that is unresponsive to medical therapy has been successfully carried out for more than three decades.[3] The procedure consists of excising a portion of the hypertrophied septum, mobilization of the papillary muscles and readaptation of the mitral valve subvalvular apparatus.[5] The operative procedure is complicated by a high mortality rate of 1.6 to 10.0%, with the possibility of perioperative complications, such as the emergence of a ventricular septal defect, total atrioventricular block or cerebral embolism, particularly in connection with an intraoperative myectomy.[7]

The beneficial effects of a myectomy led to the concept of nonsurgical septal myocardial reduction therapy (NSRT).[5] The induction of a limited therapeutic infarction, through alcohol ablation of the septal branch, leads to a reduction in the LVOT pressure gradients and associated symptoms.[8] This is presumably due to a re-

modeling process comparable to that occurring after acute myocardial infarction. The data so far published in the literature show a favorable short-term outcome after NSRT, with reduction in the LVOT gradient, with symptomatic improvement seen in approximately 80% of patients. Data concerning the long-term results are scarce. NSRT remains an investigational clinical procedure as the long-term results remain to be established.[9)]

Chang et al[10)] have recently reported that an LVOT gradient ≥25 mmHg after NSRT, in the catheterization laboratory, and a peak CK <1300 U/L were the only independent predictors of an unsatisfactory outcome. The number of injected arteries, the volume of ethanol injected and the speed of injection (bolus vs. slow injection) are associated with large infarcts.[11)] In contrast, Boekstegers et al[12)] reported that small septal infarctions (mean CK 413±193 U/L) were sufficient to result in a sustained decrease in the LVOT obstruction and an improvement in the symptoms. The incidence of complications, such as complete heart block with necessary permanent pacemaker implantation, seems to diminish with minimization of the infarct size. To minimize the amount of ethanol required and, correspondingly, the size of the septal infarction, identification of the culprit septal branch appears to be crucially important. Recently, intra-procedural myocardial contrast echocardiography (MCE) has been suggested for defining the extent and localization of the induced septal necrosis more precisely than pressure guidance.[8)]

The septal damage caused by 96% ethanol instillation, followed by myocardial shrinkage and scar formation, may potentially be arrhythmogenic and give rise to life threatening ventricular arrhythmias.[13)] Baseline conduction abnormalities have been suggested as risks factor for the development of complete heart block. Female gender, number of injected septal arteries and bolus injection of ethanol may also contribute to the development of complete heart block.[14)] With experience and technique modification, such as the use of myocardial contrast echocardiography and slow injection of alcohol, the incidence of complete heart block that requires a permanent pacemaker can be decreased.[14)] Left or right ventricular free wall infarctions, due to inadvertent spillage of alcohol into a non-target vessel, have been reported.[6)]

To avoid several serious complications that can be induced by unintended alcohol spillage, the method of coil embolization for septal branch ablation was introduced by us. Coil embolization has been used to occlude the small vessels, such as an intracranial aneurysm, arteriovenous malformation and patent ductus arteriosus. Coronary angiography was performed and the first and second septal branches ablated with coils. The pressure gradient across the LVOT was eliminated just after this procedure, after which the patient experienced a dramatic improvement in symptoms. The dramatic decrease in the LVOT pressure gradient after NSRT in the catheterization laboratory is a good prognostic factor. However, the peak CK level (316 IU/L) was relatively low, so the infarcted lesion may be expected to be small. The eliminated LVOT pressure gradient might have been caused by the myocardial contractile dysfunction induced by the septal infarction. Four months after the procedure, the patient remained free of symptoms, and the follow up echocardiography showed a mild persistent LVOT pressure gradient at rest (PG: 30 mmHg). Several limitations must be considered in this report. No myocardial contrast echocardiography was performed in this study, which may have been useful in identifying the target septal vessels.[8)] One day after the coil embolization, a follow up echocardiography was performed, but no significant reduction in the pressure gradient was obtained, so verapamil and atenolol was prescribed to the patient, and maintained. Although the patient felt symptomatic improvement, septal branch ablation with a coil is considered a palliative and not a curative procedure in this case. A follow-up coronary angiography may be required to see if collateral vessels have developed, and if feasible, embolization of the third septal branch may be attempted after the contrast echocardiography. The possible advantage of coil embolization may be the absence of complications, such as complete AV block or alcohol spill over, which have frequently been reported in alcohol

ablation.

So far, coil embolization of the septal branch has not been reported as effective in the treatment of hypertrophic obstructive cardiomyopathy, and more experiences and long-term follow-up data are required to determine the effectiveness of this therapy.

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