Successful Coronary Stenting of the Left Anterior Descending Artery at the Branching Site of the Targeted Septal Perforator Immediately after Percutaneous Transluminal Septal Myocardial Ablation in Hypertrophic Obstructive Cardiomyopathy

Tsuyoshi HONDA, Tomohiro SAKAMOTO, Shinzo MIYAMOTO, Seigo SUGIYAMA, Michihiro YOSHIMURA and Hisao OGAWA

Abstract

We report a case of simultaneous percutaneous treatment of hypertrophic obstructive cardiomyopathy (HOCM) and coronary artery disease. Cardiac catheterization revealed a left ventricular outflow tract pressure gradient (LVOTPG) of 130 mmHg and a significant left anterior descending artery (LAD) stenosis at the site of the 1st major septal branch. The LVOTPG was eliminated by injection of ethanol into the branch. Subsequently, a coronary stent was implanted in the LAD. A coil stent was selected due to possibility of repeat septal ablation in the future. Simultaneous treatment of HOCM and LAD stenosis is considered safe and effective using a coil stent.

Case Report

A 75-year-old Japanese man visited the outpatient clinic of the Cardiovascular Medicine Department of the Kumamoto University Hospital because of dyspnea on exertion and recurrent syncope. The results of M-mode and two-dimensional echocardiography were consistent with typical hypertrophic obstructive cardiomyopathy (HOCM): septal thickness of 15.2 mm; normal posterior wall; mid-systolic closure of the aortic valve (Fig. 1, panel A); systolic anterior movement of the anterior mitral leaflet (Fig. 1, panel B); and obstruction of the sub-aortic valvular area by the septal myocardium (Fig. 1, panels C and D). His electrocardiogram (ECG) showed a left-ventricular hypertrophic pattern. He was wait-listed for admission after his first visit to our hospital and went home after the examination. On the way home, he became unconscious and was rushed to the emergency outpatient unit of our institute by ambulance. On arrival, he was fully conscious and his ECG showed normal sinus rhythm without ischemic ST-T changes.

He was admitted to our ward and underwent cardiac catheterization including coronary angiography (CAG) to assess the severity of HOCM and to determine the most effective therapeutic strategy. Left heart catheterization revealed a left ventricular outflow tract pressure gradient (LVOTPG)
Figure 1. M-mode and two-dimensional echocardiography showed the following changes consistent with typical hypertrophic obstructive cardiomyopathy (HOCM): mid-systolic closure of the aortic valve (panel A); systolic anterior movement of the anterior mitral leaf (panel B); a septal thickness of 15.2 mm; normal posterior wall; and obstruction of the sub-aortic valvular area by the septal myocardium (panels C and D).

Figure 2. Left ventricular outflow tract pressure gradient (LVOTPG) before (left panel) and after (right panel) percutaneous transluminal septal myocardial ablation (PTSMA). The PTSMA procedure completely eliminated the 130 mmHg LVOTPG.
of 130 mmHg (Fig. 2, left panel). The LVOTPG was increased by the Valsalva maneuver as well as by post-extra-systolic potentiation (Brockenbrough phenomenon). In addition to left ventricular hypertrophy, he had several coronary risk factors including hypertension, smoking and hypercholesterolemia. CAG showed a 90% stenosis of the left anterior descending coronary artery (LAD) at the origin of the 1st major septal perforator (Fig. 3, panel A). Percutaneous transluminal septal myocardium ablation (PTSMA) was chosen to treat his symptoms that were due to the elevated LVOTPG caused by HOCM. Significant reversible ischemia was also documented in the anteroseptal myocardium by a rest thallium-201 myocardial scintigram and some of his symptoms were thought to be due to myocardial ischemia. Therefore, coronary angioplasty of the LAD was performed simultaneously with PTSMA.

A 7 Fr Judkins-type guiding catheter (Heartrail, JL4.0, Terumo, Tokyo, Japan) was cannulated into the left coronary artery. A 5 Fr multipurpose catheter was placed into the apex of the left ventricle for continuous monitoring of the LVOTPG. First, a 0.014” guidewire (Neo’s Rinato®, Asahi Intecc, Inc., Nagoya, Japan) was inserted into the target septal branch and then, using a double wire technique, another 0.014” guide wire was inserted into the LAD. After occlusion of the septal branch with an over-the-wire type, 2.0-mm diameter balloon catheter (Stormer®, Medtronic, Inc., Minneapolis, MN), contrast media was infused (Levovist®, Schering AG, Berlin, Germany) through the wire lumen of the catheter and myocardial contrast echocardiography (MCE) was performed to determine whether the targeted septal branch perfused the left-ventricular outflow tract. After confirming perfusion of the left ventricular outflow tract by MCE, absolute ethanol was slowly injected through the wire lumen of the balloon catheter while the balloon was inflated. The injection was performed after the patient was anesthetized by intravenous diamorphine. Because of the
Figure 4. Left ventricular outflow tract pressure gradient (LVOTPG) before (panel A), and 7 days (panel B), 6 months (panel E) and 1 year (panel F) after percutaneous transluminal septal myocardial ablation (PTSMA). Mitral regurgitation (MR) before (panel C), and at 7 days (panel D), 6 months (panel G) and 1 year (panel H) after PTSMA. The effect of PTSMA persisted for one year and the mitral regurgitant flow gradually improved after PTSMA.
bifurcation of the septal branch, wiring and ballooning were done twice for each branch. Ethanol was injected into each branch during two separate inflations (1 ml and 2 ml, respectively) with the balloon inflation maintained for 10 minutes after each injection. After completion of the PTSMA procedure, CAG of the LCA revealed abrupt disruption of the septal branch (Fig. 3, panel B) and simultaneous pressure recording from the aorta and left ventricle showed no LVOTPG (Fig. 2, right panel). CAG showed that the lesion morphology of the LAD stenosis remained unchanged after the septal inflation and ethanol injection.

After the PTSMA procedure, intravascular ultrasound was performed to determine the reference lumen diameter of the LAD stenosis. Then a 3.5-mm diameter coil coronary stent (S670®, Medtronic, Inc., Minneapolis, MN) was directly implanted in the LAD at the site of the disrupted septal branch. The lesion was successfully dilated to completely eliminate the diameter reduction and the stent was positioned appropriately (Fig. 3, panel C).

Although the creatine kinase peak was 2,008 IU/l with an MB fraction of 340 IU/l, the progress after PTSMA was favorable and a complete block did not occur during the peri-procedural period.

Discussion

PTSMA is now recognized as a standard therapeutic strategy for symptomatic HOCM because it is minimally invasive and highly effective. Given the relatively high age distribution of HOCM patients treated with this therapy, it is quite likely that many of these patients will also have atherosclerotic disorders such as coronary artery disease. In fact, there are some reports of cases of HOCM treated with both PTSMA and coronary angioplasty (2–4).

In this report, we present a case of HOCM treated simultaneously with PTSMA and coronary stent implantation. The LVOTPG was 130 mmHg before the procedure and the patient had experienced recurrent syncope despite medical therapy with carvedilol (10 mg/day). It is likely that medical therapy alone was not effective enough to relieve the symptom; therefore, we selected PTSMA as a treatment for this HOCM case. The PTSMA procedure was performed just before the angioplasty and eliminated the LVOTPG. A stent was implanted in the stenotic LAD at the site of origin of the septal branch that was used for the PTSMA. We selected a coil stent to allow for easy accessibility to the septal branch vessels in the event that an additional PTSMA procedure might be necessary in the future.

It was reported that the septal branch could be occluded with a covered stent placed in the LAD instead of ethanol ablation in patients with HOCM (5). In HOCM cases like the present case, where the LAD stenosis occurs at the site of the targeted septal perforators, this method could be used as an alternative to ethanol injection. However, it was also reported that covered stent implantation resulted in the ultimate failure to reduce the LVOTPG due to collateral formation (6). Therefore, the therapeutic strategy must be carefully evaluated in HOCM cases complicated by CAD in the LAD at the site of the targeted septal perforators.

The beneficial effect of PTSMA persisted for one year in the present case, because the LVOTPG measured by a continuous-wave Doppler probe positioned at the cardiac apex was reduced to 28 mmHg at 6 months and 11 mmHg at 1 year after PTSMA, and the mitral regurgitant flow gradually improved after PTSMA (Fig. 4, panel A–H).

Conclusions

This case report illustrates successful coronary stenting of the LAD after PTSMA in HOCM. The stent was implanted in the LAD at the branching site of the septal perforators that were targeted for the ablation procedure. We suggest that implantation of a coil stent may be the best choice for treatment of the LAD stenosis to allow for repetition of the PTSMA procedure in the future.

References