Urgent percutaneous transluminal septal myocardial ablation for left ventricular outflow tract obstruction exacerbated after surgical aortic valve replacement

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Abstract

Percutaneous transluminal septal myocardial ablation (PTSMA) is an established procedure for treating symptomatic hypertrophic obstructive cardiomyopathy. We report a case of urgent PTSMA for treating refractory heart failure due to exacerbated obstruction of the left ventricular outflow tract after surgical aortic valvular replacement to treat severe aortic stenosis.

Introduction

Left ventricular outflow tract (LVOT) obstruction is a recognized complication[1] associated with hypertrophic cardiomyopathy that causes symptoms such as dyspnea on exertion and syncope. Medical treatment is often unable to relieve the symptoms of hypertrophic obstructive cardiomyopathy (HOCM), and surgical treatment with septal myectomy has become the gold standard for such patients[(1)]. Percutaneous transluminal septal myocardial ablation (PTSMA) has emerged as an alternative and has become an established treatment for symptomatic patients with HOCM.

Herein, we describe urgent PTSMA for exacerbated LVOT obstruction after surgical aortic valvular replacement (AVR) in an elderly woman.

Case history / examination:

An 85-year-old woman was referred to us with refractory heart failure after surgical AVR with a 21-mm CROWN PRT (Sorin Group USA Inc., Arvada, CO, USA) and partial septal myomectomy for severe aortic stenosis. Transthoracic echocardiography revealed exacerbated obstruction of the left ventricular outflow tract (LVOT) with a gradient of 200 mmHg and severe mitral regurgitation (MR) with systolic anterior motion (SAM) after surgical AVR (Figure 1a and b). Invasive studies using a PressureWire X Guidewire (Abbott Vascular, Abbott Park, IL, USA) revealed real-time pressure gradients between left ventricular pressure and aortic pressure (Figure 1c). The LVOT pressure gradient was 100 mmHg. No differences were observed between these two values while retracting the pressure wire (Figure 1d and e).

Differential diagnosis, interventions and treatment:

Treatment with the β-blocker cibenzoline and temporal right ventricular pacing did not relieve the heart failure or severe LVOT obstruction. Because the patient was at high risk for surgery, we elected to proceed with urgent PTSMA.

The first to third septal perforator arteries were accessed for ablation (Figure 2a) using a guidewire (Sion Blue, Asahi Intecc USA Inc., Santa Ana, CA, USA), then a 1.2 × 6 and 1.5 × 6-mm Mini Trek balloon

(Abbott) was inflated. Contrast agent was selectively injected distal to the occlusive balloon, and simultaneous transthoracic echocardiography revealed clear delineation of the proximal septum supplied by this vessel (Figure 2, lower panels). Absolute alcohol (6.7 mL) was injected distal to the occlusive balloon over a period of 40 min.

The intraventricular pressure gradient (20 mmHg) and mitral regurgitation induced by SAM were quickly resolved by PTMA. No peri-procedural complications developed. Final coronary angiography after alcohol injection showed occluded target septal arteries and no damage to the left anterior descending artery (LAD; Figure 2b). Peak creatinine kinase and creatinine kinase-MB values were 1,693 and 363 IU/L, respectively.

Outcome and follow-up:

The patient was placed in the cardiac care unit for three days, during which she remained free of malignant arrhythmias. She was discharged on post-procedural day 35 (Figure 3). Echocardiography one month after discharge showed resolution of the mitral regurgitation, SAM and outflow tract obstruction (Figure 2, lower right panels). She has remained free of LVOT obstruction and symptoms for 3 years after PTSMA.

Discussion

The key issue issues in this case are that urgent PTSMA was applied to treat heart failure due to exacerbated LVOT obstruction after surgical AVR, which is an under-recognized cause of postoperative hemodynamic compromise(2).

The impact of a prosthesis–patient mismatch on LVOT thickening cannot be overlooked. Peak prosthetic aortic jet velocity in this patient was <3 m/s.

A prospective study has shown that flow velocity is abnormal in 14% of patients at rest after AVR for aortic stenosis and that it can be provoked or worsened by ventricular unloading or inotropic stimulation. The mechanism of a dynamic intraventricular pressure gradient in the setting of a hypertrophic left ventricle might include systolic cavity obliteration or outflow tract obstruction caused by SAM. The role of cavity squeezing rather than SAM is the fundamental mechanism of abnormal flow velocity after AVR for aortic stenosis.

Afterload is increased in patients with aortic stenosis, and the ventricle is often small and hypertrophic. Valve replacement induces a dramatic decrease in afterload that might further decrease left ventricular volume and increase fiber shortening. A combination of these factors might lead to cavity squeezing, which in turn will increase systolic flow velocity(3).

Percutaneous transluminal septal myocardial ablation was initially reported in 1995 as a novel technique for the nonsurgical reduction of septal hypertrophy in patients with hypertrophic cardiomyopathy (HCM)(4). Surgical myectomy remains the gold standard of treatment for symptomatic patients who have LVOT obstruction that is refractory to medical therapy, as it is safe and effective over the long-term. However, PTSMA in expert hands can effectively reduce the gradient in selected patients with low procedural complication rates(5).

Both myectomy and PTSMA reduce LVOT obstruction and significantly improve New York Heart Association functional class in patients with HCM. However, each type of therapy has advantages and disadvantages that must be counterbalanced when deciding how to treat LVOT obstruction.

One case report has described successful PTSMA for heart failure due to significant LVOT obstruction that manifested after AVR(6). Although PTSMA relieved LVOT obstruction and symptoms during the acute phase, modest recurrence was confirmed six months later. In contrast LVOT obstruction and symptoms have not recurred in our patient during 3 years of follow-up after PTSMA.

Both case reports underline the need for PTSMA options with low procedural morbidity to treat LVOT obstruction, particularly for highly symptomatic patients who are contraindicated for surgery.

Conclusion: Urgent PTSMA might be a safe option for treating heart failure due to exacerbated LVOT obstruction after surgical AVR.

Conflicts of interest

None declared.

Author Contributions:

Yoshitaka Sasahira, MD; coronary intervention, drafting of the manuscript

Ryotaro Yamada, MD; coronary intervention, revising the manuscript

Naofumi Doi, MD; coronary intervention, revising the manuscript

Shiro Uemura, MD; coronary intervention, final approval of the manuscript submitted

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Figure legends:

Figure 1. Baseline transthoracic echocardiography and pressure wire findings.

Transthoracic echocardiography revealed exacerbated left ventricular outflow tract (LVOT) obstruction and severe mitral regurgitation (MR) with systolic anterior motion (SAM) after surgical AVR (Figure 1a and 1b).

Invasive pressure wire shows real-time pressure gradients between left ventricular (green line) and aortic (red line) pressure (Figure 1c). Pressure gradient LVOT was 100 mmHg. These gradients did not differ during pressure wire retraction (Figure 1d and e).

Figure 2. Percutaneous transluminal septal myocardial ablation.

The first to third septal perforator arteries were accessed for ablation (Figure 2a). Contrast agent was selectively injected distal to occlusive balloon, and its effects were monitored by simultaneous transthoracic echocardiography. Gradient in LVOT was reduced to from 229 to 20 mmHg immediately after alcohol was

injected, without evident malignant arrhythmias (lower panels). Final coronary angiography after alcohol injection shows occluded target septal arteries and no injury to LAD (Figure 2b). Echocardiography at one month of follow-up shows obvious resolution of mitral regurgitation, SAM and outflow tract obstruction (lower right panels).

Figure 3. Post-procedural progress.

Patient remained free of malignant arrhythmias for three days in coronary care unit. She was discharged from hospital on post-procedure day 35 with pleural effusion and laboratory data improvement.