

Successful emergency transcatheter aortic valve implantation

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Despite the necessity of surgical aortic valve replacement, many patients with symptomatic severe aortic stenosis (AS) cannot undergo surgery because of their severe comorbidities. In these high-risk patients, percutaneous transcatheter aortic valve implantation (TAVI) can be safely accomplished. However, no study has shown that TAVI can be performed for patients with severe AS accompanied by acute decompensated heart failure. In this case report, 1 patient presented a case of severe pulmonary hypertension with decompensated heart failure after diagnosis with severe AS, and was successfully treated via emergency TAVI. Without any invasive treatment, acute decompensated heart failure with severe pulmonary hypertension is common in patients with severe AS, and it can increase mortality rates. In conclusion, TAVI can be considered one of the treatment options for severe AS presented as acute decompensated heart failure patients with pulmonary hypertension.

Keywords: Aortic valve stenosis; Heart failure; Emergency treatment; Heart valve prosthesis implantation

INTRODUCTION

Symptomatic aortic stenosis (AS) usually requires aortic valve replacement (AVR) since the prognosis of medical treatment alone is generally poor. Despite the proven benefits of surgical AVR, many patients with severe AS cannot undergo surgery because of their high morbidity and mortality rates, especially elderly patients. Nowadays, transcatheter aortic valve implantation (TAVI) may be an alternative therapy for high-risk patients and many studies have shown that TAVI could have more favorable outcomes than standard therapies in high-risk patients [1-3].

In patients with severe AS, there is a high prevalence of pulmonary hypertension that can be associated with increased mortality [4,5]. Surgical AVR and TAVI can be effective thera-

pies for these patients and may result in reduced pulmonary artery systolic pressure (PASP) [4]. However, no study has shown that TAVI can be one of the treatment options for patients with severe AS accompanied by acute decompensated heart failure and severe pulmonary hypertension.

We report here in a successful emergency TAVI procedure for a patient with severe AS presented as acute decompensated heart failure.

CASE

An 84-year-old man visited our hospital because of progressive dyspnea upon exertion. His condition could be classified as the New York Heart Association (NYHA) Functional Classification II. He had a medical history of hypertension and old pulmonary tuberculosis. His physical examination showed a pulse rate of 74 beats per minute (bpm) and a blood pressure of 109/68 mmHg. There was systolic murmur at the right upper sternal border. His electrocardiography showed a sinus rhythm with left ventricular (LV) hypertrophy, and his chest X-ray, an enlarged cardiac silhouette with multiple nodules and calcification throughout both

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lungs, which suggested old tuberculosis sequelae (Fig. 1A). There were no abnormal findings from his laboratory examination, except for mild anemia (hemoglobin 11.1 g/dL). His echocardiography showed a degenerative change in his aortic valve with calcification, which suggested severe AS (aortic valve area [AVA], 0.5 cm^2 based on the continuity equation), moderate pulmonary hypertension (right ventricular systolic pressure [RVSP], 60 mmHg), and normal global LV systolic function (ejection fraction [EF], 57%) (Fig. 2A). The pressure curve on his aortic valve was also elevated (peak/mean pressure gradient, 117/74 mmHg) (Fig. 3A). His coronary angiography showed no significant luminal narrowing, except for a minimal mid-right coronary artery lesion (Fig. 4). We performed trans-esophageal echocardiography, which also

showed severe AS ($\text{AVA}=0.64 \text{ cm}^2$ via 2D) and that he was a candidate for the TAVI procedure. However, at that time, he refused any invasive procedure such as AVR or TAVI. He was discharged with maintenance of medical therapy.

Four months after his discharge, he was readmitted because of aggravated dyspnea. In his physical examination, moist rale was heard at both lung fields, and his chest X-ray showed bilateral pulmonary edema (Fig. 1B). His NT-proBNP level was elevated at 14,328 pg/mL. Despite intravenous furosemide therapy, his blood pressure fell to 75/55 mmHg, his heart rate surpassed 100 bpm, and his oxygen saturation fell below 90%. His echocardiography revealed severe AS with significantly decreased LV systolic function (left ventricular ejection fraction [LVEF], from 57% to 34%), further aggra-

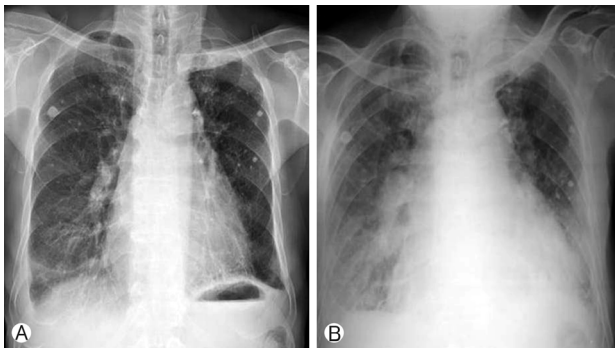


Fig. 1. The patient's chest X-ray. (A) Chest X-ray at when he first visited showed cardiomegaly with multiple nodules and calcification throughout both lung suggesting old tuberculosis. (B) Chest X-ray at 1 day after re-admission showed severe aggravated pulmonary edema.

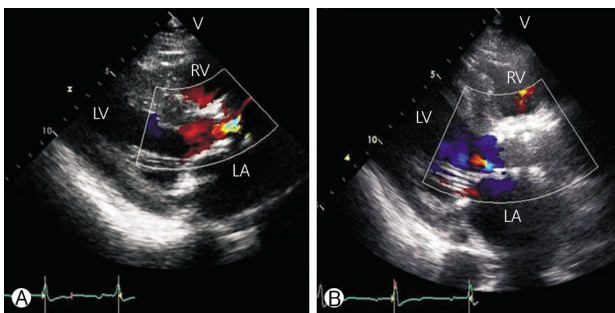


Fig. 2. The patient's 2-D echocardiography. (A) Before TAVI procedure, echocardiography showed a degenerative change of aortic valve with severe calcification. (B) After TAVI procedure, echocardiography revealed good motion of CoreValve and mild paravalvular aortic regurgitation. TAVI, transcatheter aortic valve implantation; LV, left ventricle; RV, right ventricle; LA, left atrium; AV, aortic valve.

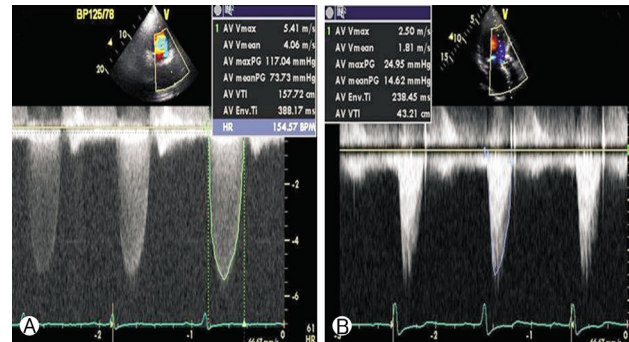


Fig. 3. The patient's pressure curve on aortic valve. (A) Before TAVI procedure, pressure gradient of aortic valve showed elevated (peak/mean pressure gradient=117/74 mmHg). (B) After TAVI procedure, pressure curve at aortic valve showed markedly decreased pressure gradient (peak/mean pressure gradient, 25/15 mmHg). TAVI, transcatheter aortic valve implantation.

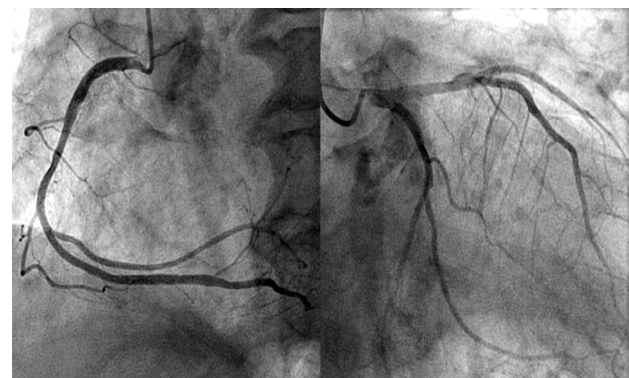


Fig. 4. The patient's coronary angiography showed no significant luminal narrowing except minimal mid-right coronary artery lesion.

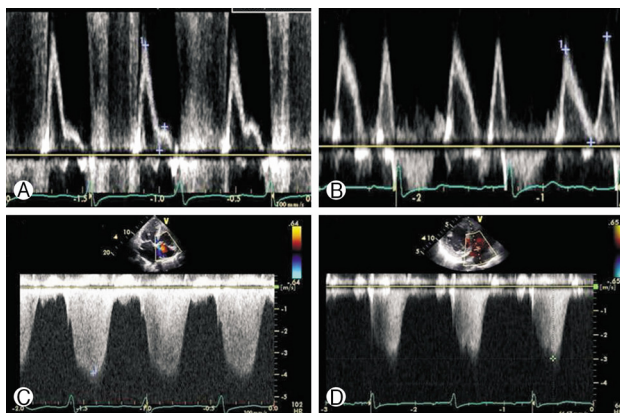


Fig. 5. The patient's Doppler flow study. (A, C) Before TAVI procedure, echocardiography showed diastolic dysfunction and increased RVSP (DT=96 msec, pseudonormalization pattern of LV with elevated filling pressure, $E/E' = 37$, RVSP=86 mmHg). (B, D) After TAVI procedure, echocardiography showed improved diastolic function and reduced RVSP (DT=198 msec, relaxation abnormality of LV filling pattern with elevated LV filling pressure, $E/E' = 23$, RVSP=45 mmHg). TAVI, transcatheter aortic valve implantation; RVSP, right ventricular systolic pressure; DT, deceleration time; LV, left ventricle.

vated pulmonary hypertension (RVSP, from 67 to 86 mmHg), and aggravated diastolic function (deceleration time [DT]: from 131 to 96 msec, and pseudonormalization pattern of his LV filling with elevated LV filling pressure, $E/E' = 37$) (Fig. 5A, 5C). We assumed that his status was aggravated by acute decompensated LV systolic and diastolic function due to severe aortic stenosis, and we decided to perform an emergency TAVI procedure.

We performed the emergency TAVI procedure under general anesthesia. We punctured the right common femoral artery (CFA) using the standard percutaneous access techniques for vascular access in CoreValve delivery. Under rapid pacing with a temporary pacemaker, we successfully inserted and inflated a 25 mm balloon (Z-med, NuMed Inc., Hopkinton, NY, USA) at the aortic valve. Then we inserted a 29 mm CoreValve through the right CFA sheath and deployed it at the aortic valve under fluoroscopic and intra-operative transthoracic echocardiogram guidance to prevent a size mismatch. The immediate post-procedural transthoracic echocardiography showed good CoreValve motion and mild paravalvular aortic regurgitation (Fig. 2B). We performed follow-up transthoracic echocardiography 5 days after the procedure, and it showed that the patient's global LV systolic function had normalized (LVEF, from 34% to 58%), his RVSP had

decreased (from 86 to 45 mmHg), and his diastolic function had improved from a restrictive physiology to an abnormal relaxation pattern (E/E' , from 32 to 23) (Fig. 5B, 5D). The pressure curve on his aortic valve significantly decreased compared to the pre-procedural findings (peak/mean pressure gradient, 25/15 mmHg) (Fig. 3B).

The patient's dyspnea was relieved after the procedure, and his chest X-ray showed marked improvement. He was discharged 5 days after the procedure, and his 3-month follow-up echocardiography showed no significant interval change from his immediate post-procedural echocardiographic findings. After 10 months, he visited our outpatient clinic without any symptom or major cardiovascular adverse event.

DISCUSSION

AS is one of the most common diseases of heart valves among elderly patients. Since the median survival time after the appearance of symptoms related to AS, such as angina, dyspnea or syncope, is only 2 to 3 years, patients with symptomatic AS should be treated with AVR [6]. However, for high-risk patients with severe comorbidities, the "gold-standard" treatment with conventional surgery cannot be the most effective treatment option. Since 2002, when TAVI was first performed [7], it has been used throughout the world as an alternative treatment option for patients with high surgical risks. A randomized trial with 358 patients with AS who were not suitable candidates for surgery demonstrated that TAVI significantly reduced the rates of death from any cause, repeat hospitalization, and cardiac symptoms, as compared with standard therapy [3].

Pulmonary hypertension is not uncommon among severe AS patients. Ben-Dor et al. [4] had reported that in a select study group of high-risk patients with severe AS for whom TAVI was considered, 34% had severe pulmonary hypertension defined as $PASP \geq 60$ mmHg. As expected, these patients had higher prevalence of NYHA III-IV, enlarged cardiac chambers and decreased LV function [8]. There is a positive correlation between the level of $PASP$ and LV end-diastolic pressure, which is also independently correlated with pulmonary hypertension. Some studies have shown that severe pulmonary hypertension could be one of the risk factors in patients with AS undergoing AVR [4,5,8]. Miceli et al. [8] had

reported that patients with elevated PASP levels had a 400% higher risk of in-hospital mortality and a 10.1% absolute risk of death compared with those with normal PASP.

In our case report, the patient's age was over 80 and his logistic European System for Cardiac Operative Risk Evaluation was 13.5%. He also had elevated RVSP that suggested pulmonary hypertension. When he first visited our hospital, he was a high-risk patient for surgical AVR and a good candidate for TAVI, but he refused any invasive procedure. After a few months, his status was worsened by his decompensated heart function, as expected, and surgical AVR became a more risky treatment option for him. Although RVSP may decrease soon after AVR, once pulmonary hypertension has developed in patients with severe AS, the process may not be reversible, and this may explain why late outcomes are worse in those with higher RVSP [5]. Furthermore, a significant and sustained decrease in pulmonary pressure up to 1 year after TAVI has been reported, which is comparable to that seen with surgical valve replacement [4]. We had no choice but to perform the TAVI procedure in this desperate situation.

Acute decompensated heart failure is common in patients with severe AS who have had no invasive treatment such as AVR or TAVI. If acute decompensated heart function with severe pulmonary hypertension due to severe AS occurs, surgery can increase the mortality rate. In conclusion, TAVI can be considered one of the treatment options for patients with severe AS who present acute decompensated heart failure with pulmonary hypertension.

CONFLICT OF INTEREST

There was no potential conflict of interest relevant to this article.

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