Case Report

Non-surgical management of an acute decompensated heart failure patient with severe aortic stenosis and concomitant left ventricular outflow tract obstruction

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Article history:
Received 22 May 2021
Revised 20 August 2021
Accepted 10 September 2021

Keywords:
Aortic stenosis
Balloon aortic valvuloplasty
Left ventricular outflow tract obstruction
Transcatheter aortic valve replacement
Percutaneous alcohol septal ablation

A B S T R A C T

Acute decompensated heart failure (ADHF) due to severe aortic stenosis (AS) and concomitant left ventricular outflow tract (LVOT) obstruction is a serious condition. Treatment with medication alone is sometimes difficult, and the efficacy of further interventional strategies has not been fully elucidated. In patients with high surgical risks, combination therapy using transcatheter aortic valve replacement (TAVR) and percutaneous alcohol septal ablation (ASA) is used as a non-surgical intervention. However, this treatment cannot be performed under unstable hemodynamic conditions. This report highlights the utility and efficacy of emergent balloon aortic valvuloplasty (BAV) in controlling heart failure and its potential to serve as a bridge to curative therapy, even in older patients with ADHF due to severe AS with concomitant LVOT obstruction. Furthermore, combination therapy with TAVR and percutaneous ASA could be safely performed after controlling for ADHF using BAV. Non-surgical management is a more feasible treatment option in older patients with ADHF who are at higher risk of complications during surgical intervention.

-Learning objective: Management of acute decompensated heart failure (ADHF) due to severe aortic stenosis (AS) and concomitant left ventricular outflow tract (LVOT) obstruction can be difficult. Despite concomitant LVOT obstruction, emergent balloon aortic valvuloplasty is potentially useful and effective for controlling heart failure and could become a bridge to subsequent combination therapy using transcatheter aortic valve replacement and percutaneous alcohol septal ablation. This sequential non-surgical management is a novel strategy for older patients with high surgical risk and ADHF due to severe AS with concomitant LVOT obstruction.>

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Introduction

The prevalence of aortic stenosis (AS) and simultaneous left ventricular outflow tract (LVOT) obstruction is an unresolved problem [1]. Management is especially difficult in patients with acute decompensated heart failure (ADHF) due to these etiologies. Combination therapy using transcatheter aortic valve replacement (TAVR) and percutaneous alcohol septal ablation (ASA) has been established as an alternative strategy to surgical intervention in patients with high surgical risks due to comorbidities and frailties [2]. However, even non-surgical therapies carry risks for patients with ADHF, as little is known regarding the clinical course and therapeutic strategy in ADHF caused by severe AS and concomitant LVOT obstruction. We describe a case in which an emergent balloon aortic valvuloplasty (BAV) sufficiently controlled severe heart failure and permitted further combined therapy using TAVR and percutaneous ASA in an older patient with severe dementia.
formed consent to publish the case and any accompanying images was obtained from the patient and his family.

Case report

A 79-year-old man with a history of hypertension, chronic obstructive pulmonary disease, and severe dementia (Mini-Mental State Examination score 15) presented to our institution with New York Heart Association (NYHA) class IV ADHF. The patient’s hemodynamic condition was unstable; blood pressure and heart rate were 95/55 mmHg and 98 beats/min, respectively. Physical examination revealed a systolic ejection murmur with Levine grade III/VI and bilateral inspiratory crackles. Electrocardiography showed sinus rhythm without conduction disorders (Fig. 1A). Chest radiography revealed pulmonary congestion with pleural effusion (Fig. 1B). Transthoracic echocardiography (TTE) showed a normal left ventricular ejection fraction of 59% with severe asymmetric septal hypertrophy and severe AS with a degenerated valve, a mean aortic valve pressure gradient (PG) of 75 mmHg, and a peak velocity of 5.5 m/s. The systolic tricuspid regurgitation PG was 45 mmHg, which indicated pulmonary hypertension. The patient was diagnosed with ADHF with cardiogenic shock due to severe AS. The patient then received dobutamine (3 μg/kg/min) and noninvasive positive pressure ventilator support. However, intravenous diuretics were ineffective, and heart failure was not sufficiently controlled. TTE revealed systolic anterior motion of the mitral valve, suggesting concomitant LVOT obstruction (Fig. 1C-F). Surgical aortic valve replacement (SAVR) and concomitant myectomy is the standard curative therapy for severe AS and concomitant LVOT obstruction; however, the perioperative mortality rate in this case was estimated to be extremely high, with a Society of Thoracic Surgeons’ database risk of 21.9% [3]. Non-surgical intervention of TAVR and percutaneous ASA would be desired; however, even these interventions were believed to be challenging in this ADHF case. Our heart team elected to perform an emergent BAV as a palliative therapy that could serve as a bridge to TAVR. Retrograde BAV was performed using a 20-mm INOUE balloon (TORAY, Tokyo, Japan). After three-time valvuloplasty without rapid ventricular pacing, the peak simultaneous pressure of the aorta and left ventricle decreased from 130 to 66 mmHg; the residual pressure was mainly caused by LVOT obstruction due to severe asymmetric septal hypertrophy (Fig. 1G). Discontinuation of dobutamine usage and subsequent intravenous fluid therapy sufficiently enabled control of the LVOT obstruction. Furthermore, we started to administer bisoprolol 0.625 mg/day after the procedure and added up to a maximum of 2.5 mg/day, which led to the reduction of the residual peak LVOT-PG to 15 mmHg (Fig. 1H). Moreover, we administered a single antiplatelet therapy with aspirin 100 mg/day. Although subsequent curative therapy was planned, the patient had difficulty with long hospitalization due to severe dementia and delirium. He was temporally discharged with a NYHA class II condition after undergoing computed tomography angiography (CTA) for TAVR.

Two weeks later, the patient was readmitted with progressive dyspnea due to deteriorated NYHA class III heart failure. TTE suggested recurrent severe AS with a peak velocity of 5.1 m/s and transaortic mean PG of 59 mmHg without significant LVOT obstruction (Fig. 2A). After the administration of dual antiplatelet therapy with aspirin 100 mg/day and clopidogrel 75 mg/day, TAVR

Fig. 1. Emergent balloon aortic valvuloplasty for severe aortic stenosis with acute decompensated heart failure. (A) Electrocardiography showed sinus rhythm without conduction disorders. (B) Chest radiography showed pulmonary congestion with bilateral pleural effusion. (C-E) Transthoracic echocardiography revealed a severely calcified aortic valve and concomitant asymmetric left ventricular hypertrophy (yellow dotted line) with systolic anterior motion of the mitral valve (yellow arrow). (F) The transaortic pressure gradient before emergent balloon aortic valvuloplasty (BAV). (G1 and G2) The aorta-left ventricular pressure gradient measured by pressure wire pre-BAV (G1) and post-BAV (G2). (H) The transaortic pressure gradient after BAV. LA, left atrium; LV, left ventricle.
was performed via the femoral artery under local anesthesia, and an Edwards SAPIEN-3 aortic prosthesis (Edwards Life Science, Irvine, CA, USA) was used to improve access to the coronary arteries in consideration of percutaneous ASA when the LVOT obstruction progressed. The prosthetic valve was implanted with 1cc inflation based on the CTA findings (Fig. 2B-E). The peak LVOT-PG was 45 mmHg immediately after TAVR but decreased to approximately 15 mmHg after infusion therapy (Fig. 2F). After optimal medications with bisoprolol, which was increased up to 5 mg/day, and introducing cibenzoline 150 mg/day, the patient was discharged.

One month after TAVR, the patient developed recurrent exertional dyspnea. TTE showed no evidence of valve prosthesis dysfunction and demonstrated significant LVOT obstruction with a peak PG of 109 mmHg caused by dehydration (Fig. 3A). We performed percutaneous ASA to improve this dynamically deteriorated LVOT obstruction. After the adjustment of body fluids, percutaneous ASA was performed for the first septal artery with 1.5-ml absolute ethanol and a 2.0-mm over-the-wire balloon (EmergeTMOTW, Boston Scientific Corporation, St Paul, MN, USA) (Fig. 3B and C). The residual peak LVOT-PG decreased to 5 mmHg even after nitroglycerin loading (Fig. 3D). The patient was discharged without any disabilities or pacemaker implantation. No further hospitalization was required after a follow-up period of more than 6 months.

Discussion

In the present report, we describe our experience of managing an older patient who presented with ADHF and cardiogenic shock with severe AS and concomitant LVOT obstruction, which was successfully treated using a non-surgical strategy: emergent BAV and subsequent combined therapy using TAVR and percutaneous ASA. LVOT obstruction is caused by hypertrophic cardiomyopathy, sigmoid septum, post-mitral valve surgery, and left ventricular hypertrophy [4]. The incidence of concomitant LVOT obstruction with severe AS is reported to vary from 1% to 4%, and LVOT obstruction in severe AS is considered clinically challenging [3].

The precise evaluation of the severity of LVOT obstruction can be difficult in patients with severe AS [5]. Although the Valsalva maneuver and nitroglycerin loading test, which can reduce left ventricular preload and afterload, are useful for diagnosing the presence of LVOT obstruction, severe AS makes these tests difficult owing to the risk of hemodynamic collapse. The aortic valves should be prioritized in cases with confirmed severe AS; however, additional interventions for potential LVOT obstruction in certain severe AS remain controversial. Emergent TAVR is challenging, particularly in ADHF with cardiogenic shock because TAVR before precisely evaluating the aortic valves and arterial access using CTA is difficult [6]. Although BAV has a poor long-term prognosis and some limitations, including aortic regurgitation, transient deterioration of LVOT obstruction, and recurrent recoiled-AS [7], in our case, emergent BAV was effective in controlling heart failure and made subsequent curative therapy feasible. We also observed that the alleviation of left ventricular afterload due to interventions dramatically deteriorated LVOT obstruction. To prevent this deterioration of LVOT obstruction potentially leading to suicide left ventricle phenomenon, appropriate management, including intravenous fluid therapy and decreasing inotropy with beta-blocker pre-procedure in cases without ADHF, were reportedly useful [8]. Although management would be difficult in the ADHF conditions such as our case, stopping the intravenous use of dobutamine just
before the procedure to AS might be crucial. Thus, BAV may be effective and useful for the diagnosis of concomitant LVOT obstruction, we should pay careful attention to this dramatic hemodynamic change to prevent catastrophic complications.

SAVR and concomitant septal myectomy is the curative therapy for severe AS with LVOT obstruction [3]. Our patient had some limitations for surgery due to the control of ADHF, severe dementia, and frailty. Combination therapy using TAVR and percutaneous ASA is an alternative strategy for patients with high surgical risks [2]. Whether TAVR or percutaneous ASA should be performed first remains unclear. TAVR prior to percutaneous ASA has several concerns, including the above-mentioned sudden reduction of left ventricular afterload, which can worsen preexisting LVOT obstruction [1]. Moreover, interference of the implanted TAVR valve may result in difficulty engaging the catheter at the orifice of the coronary artery during percutaneous ASA. With regard to valve selection, balloon-expandable Edwards SAPIEN-3 valves are suitable for TAVR owing to their low pop-up phenomenon frequency and their accessibility to the coronary arteries due to shorter stent length, larger struts, and lower position of the inner skirt [9]. Conversely, percutaneous ASA prior to TAVR is effective in reducing septal thickness and facilitating transcatheter valve implantation [10]. However, percutaneous ASA prior to TAVR might be unsuitable for patients with ADHF since percutaneous ASA intentionally leads to myocardial infarction. The adequate duration for safely performing percutaneous ASA after ADHF remains unclear, particularly in patients with severe AS.

In summary, we encountered a case of ADHF with severe AS and LVOT obstruction that was successfully treated with emergent BAV and subsequent combined therapy using TAVR followed by percutaneous ASA. This sequential non-surgical strategy could be feasible and effective, particularly in older patients with higher surgical risks. Further investigations into the management of ADHF with severe AS and LVOT obstruction are warranted.

**Declaration of Competing Interest**

The authors declare that there are no conflicts of interest.

**Acknowledgments**

None.

**Funding**

None.

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