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Case Report

Impella-Assisted Left Ventricular Decompression for ECMO-Induced Ventricular Distension during Transcatheter Aortic Valve Implantation: A Case Report

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Abstract

Objective: Although transcatheter aortic valve implantation (TAVI) is a less invasive alternative for patients with severe aortic stenosis (AS) who are at high risk, severe complications may occur and occasionally require mechanical circulatory support.

Case Presentation: An 89-year-old male with symptomatic, extremely severe AS underwent TAVI through the femoral approach. After rapid pacing during balloon aortic valvuloplasty (BAV), he experienced ventricular fibrillation (VF) that did not respond to direct current (DC) defibrillation. Veno-arterial extracorporeal membrane oxygenation (VA-ECMO) was initiated. VF persisted despite repeated DC shocks. After BAV, the heavily calcified aortic valve remained fixed in an open position, resulting in acute aortic regurgitation (AR). Initiating the VA-ECMO led to direct inflow of ECMO output into the left ventricle, causing a rapid increase in left ventricular (LV) end-diastolic pressure and severe LV distension. Even after valve deployment under ECMO support, transesophageal echocardiography revealed severe acute mitral regurgitation (MR) due to leaflet separation. Massive frothy sputum from the endotracheal tube indicated fulminant pulmonary edema. An Impella CP was inserted across the implanted TAVI valve through the femoral approach to decompress the left ventricle. Successful defibrillation was achieved after Impella activation. During intensive postoperative care, the acute pulmonary edema improved markedly. The Impella was removed on postoperative day (POD) 1, and VA-ECMO was discontinued on POD 2. The patient was discharged from the intensive care unit on POD 7.

Conclusion: Impella-mediated LV unloading was highly effective for acute circulatory collapse caused by acute AR and secondary MR during TAVI.

Keywords:

balloon aortic valvuloplasty, extracorporeal membrane oxygenation, mitral regurgitation, aortic regurgitation, acute pulmonary edema

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Introduction

The most significant advantage of transcatheter aortic valve implantation (TAVI) is its ability to provide an aortic valve replacement for elderly patients with severe aortic stenosis (AS) who are at high risk, without requiring

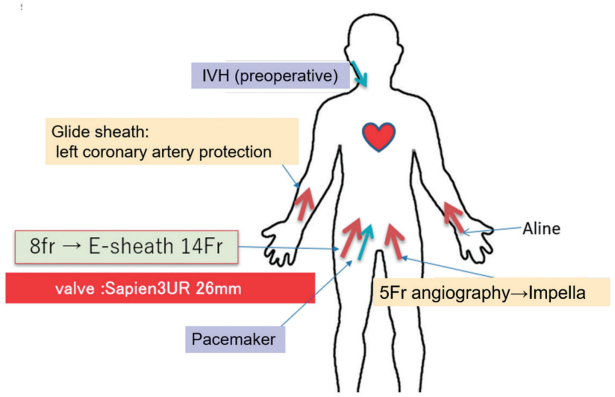

Fig. 1 The TAVI access route strategy. IVH: intraventricular hemorrhage; TAVI: transcatheter aortic valve implantation

a thoracotomy or extracorporeal circulation. Severe complications may occur, necessitating mechanical circulatory support to maintain hemodynamics. However, subsequent initiation of veno-arterial extracorporeal membrane oxygenation (VA-ECMO) can result in adverse events.

The present case study emphasizes the crucial role of Impella-mediated left ventricular (LV) unloading in rapidly stabilizing acute circulatory collapse during TAVI, particularly when ECMO-induced LV distention occurs.

Case Presentation

An 89-year-old male with a medical history of hypertension and knee osteoarthritis exhibited symptoms of exertional dyspnea when walking indoors. Therefore, he was referred to our hospital for further evaluation, leading to the diagnosis of aortic valve stenosis. His daily activities were confined to indoor walking. Considering the patient's age and overall health status, TAVI was opted as the treatment modality.

Transthoracic echocardiography (TTE) revealed no wall motion abnormalities, and the ejection fraction (EF) was preserved at 62%. The aortic valve showed severe calcification on both sides of the tricuspid valve, resulting in restricted leaflet opening. The maximum transvalvular velocity was 5.27 m/s, the mean pressure gradient was 77.8 mmHg, and the aortic valve area was 0.43 cm^2. These findings indicated an extremely severe AS. Conversely, no other significant valvular diseases were identified.

A detailed analysis by computed tomography revealed no evidence of aortic dissection or aneurysmal changes. Moreover, the access route used was within the normal parameters. A thorough evaluation of the aortic root disclosed severe calcification in all 3 leaflets. Measurements showed an aortic angulation of 34°, an annular area of 520 mm^2, a circumference of 80.1 mm, a left coronary artery height of 11.4 mm, and a right coronary artery height of 21.5 mm. Calcification was also detected in the coronary arteries.

A detailed description of the procedural steps has been mentioned: The procedure was initiated under general anesthesia. **Fig. 1** demonstrates the TAVI access route strategy.

Because coronary angiography (CAG) revealed a low takeoff of the left coronary artery, coronary artery protection was performed. Initially, a guidewire (Guideplus 2 EL, JL3.5; Nipro, Osaka, Japan) was placed in the left anterior descending coronary artery; however, it dislodged during the balloon advancement step of balloon aortic valvuloplasty (BAV). Therefore, the coronary artery protection was modified to the left circumflex artery (LCx) (**Fig. 2A**).

In this case, BAV was performed because of extensive calcification in all 3 leaflets, which was considered to increase the risk of inadequate valve expansion or malposition despite the use of a balloon-expandable valve.

BAV was attempted using a Z-MED 20-mm balloon (B. Braun, Melsungen, Germany) under rapid ventricular pacing at 180 bpm using a temporary external pacemaker (**Fig. 2B**). However, the balloon position was unstable, and rapid pacing persisted for a prolonged period, causing ventricular fibrillation (VF). Despite multiple direct current (DC) shocks, the VF persisted. Therefore, VA-ECMO was established through the left femoral artery and vein under ongoing chest compressions (8 min after the onset of VF). Defibrillation was still unsuccessful, and the VF remained refractory. We decided to perform the procedure with valve placement under ECMO support.

A large amount of frothy, blood-tinged sputum (~2000 mL) was suctioned from the endotracheal tube. Mechanical ventilation was temporarily halted, and a high positive end-expiratory pressure of 20 cm H_2O was applied. Considering that bleeding could occur from the bronchial or intercostal arteries, aortography was performed; however, no active extravasation was detected. The procedure was resumed, and BAV was repeated, followed by the deployment of a Sapien 3 Ultra Resilia 26-mm valve (Edwards Lifesciences, Irvine, CA, USA) (**Fig. 2C**). Postdeployment DC shock also failed to terminate the VF.

Transesophageal echocardiography performed under VA-ECMO support revealed marked separation of the anterior and posterior mitral leaflets (A2/P2), with Doppler findings indicating severe mitral regurgitation (MR); however, accurate quantification was limited by concurrent VF. The findings were indicative of acute pulmonary edema, suggesting the need of LV decompression. Therefore, an Impella CP (Abiomed, Danvers, MA, USA) device was placed across the newly implanted TAVI valve

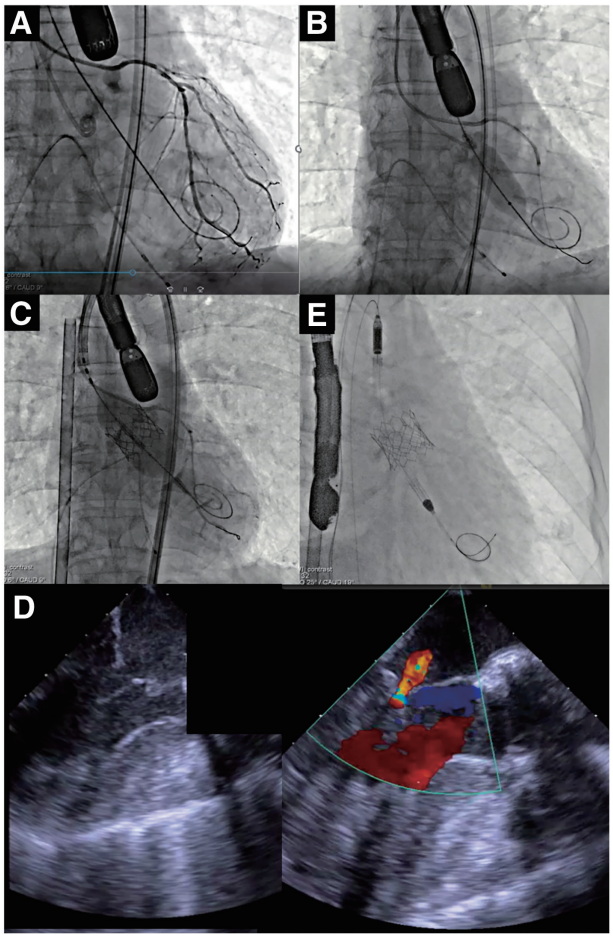

Fig. 2 Procedural findings. (**A**) The procedure was performed to protect the left coronary artery, which was identified as low. The initial placement of the guidewire was in the anterior descending artery. However, due to its subsequent extrusion, the protection was modified to the circumflex artery. (**B**) Balloon aortic valvuloplasty was attempted under rapid pacing; however, slippage occurred, and sufficient expansion could not be achieved. (**C**) The transcatheter aortic valve implantation procedure was continued, and a Sapien 3 Ultra Resilia 26-mm valve was implanted. (**D**) Transesophageal echocardiography revealed separation of the anterior and posterior mitral leaflets (A2/P2). (**E**) Subsequently, the Impella CP was implanted

through the right femoral artery (85 min after VF onset) (**Fig. 2E**). After Impella CP activation, DC shock successfully restored the sinus rhythm. The procedure was then completed, and the patient was transferred to the intensive care unit (ICU). The total operative time was 4 h 0 min, and the duration of anesthesia was 4 h 50 min. An immediate postoperative chest radiography revealed marked pulmonary congestion (**Fig. 3A**).

Postoperatively, during treatment in the ICU, the patient's acute pulmonary edema improved markedly (**Fig. 3B** and **3C**). The mean pulmonary artery pressure decreased from >20 mmHg to ~15 mmHg, and catecholamine support was no longer required, thus allowing the removal of the Impella on postoperative day (POD) 1. Although catecholamines were again required temporarily after Impella removal, they were gradually weaned as the patient's blood pressure stabilized. ECMO was discontinued on POD 2, extubation was achieved on POD 4, and the patient was transferred out of the ICU on POD 7. Postoperative TTE revealed an EF of 63% with no asynergy. The maximum transvalvular velocity was 2.09 m/s, the mean pressure gradient was 8.7 mmHg, and the aortic valve area was 1.44 cm^2, confirming the resolution of AS. Furthermore, aortic regurgitation (AR) was trivial, and MR remained mild to moderate, with no deterioration in valvular function. The postoperative course was favorable, and the patient regained ambulation on the ward. He was then transferred to a rehabilitation facility on POD 30.

Discussion

In this case study, the occurrence of acute AR during TAVI resulted in acute MR and subsequent acute pulmonary edema. The implementation of LV unloading using an Impella device demonstrated remarkable efficacy in managing these complications.

Previous case reports describe the use of Impella for circulatory collapse due to worsening AR or MR after TAVI,[1] as well as reports of acute AR after BAV that progressed to cardiogenic shock.[2] A similar report also mentions the intraoperative use of Impella during TAVI in situations such as immediate post-TAVI cardiogenic shock, coronary complications, and concomitant high-risk percutaneous coronary intervention.[3] However, no reports have described intraoperative Impella implantation for acute hemodynamic collapse caused by combined AR and MR during TAVI, with rapid stabilization as observed in our patient. Hence, the present case is a rare and an informative case.

In our patient, spontaneous cardiac activity failed to recover after rapid pacing, resulting in subsequent VF. Two factors may account for this progression. First, prolonged rapid pacing caused a significant reduction in cardiac output. Second, the catheter placed in the LCx for coronary protection inadvertently became wedged, causing iatrogenic myocardial ischemia (**Fig. 2A**). These factors may have markedly impaired coronary perfusion, ultimately precipitating VF.

Although the use of VA-ECMO in the setting of AR is generally contraindicated because it markedly increases LV afterload, its initiation was deemed unavoidable in this case to maintain vital organ perfusion.[4]

In this case, balloon expansion was unstable because of repeated sliding, and localized mechanical stress during

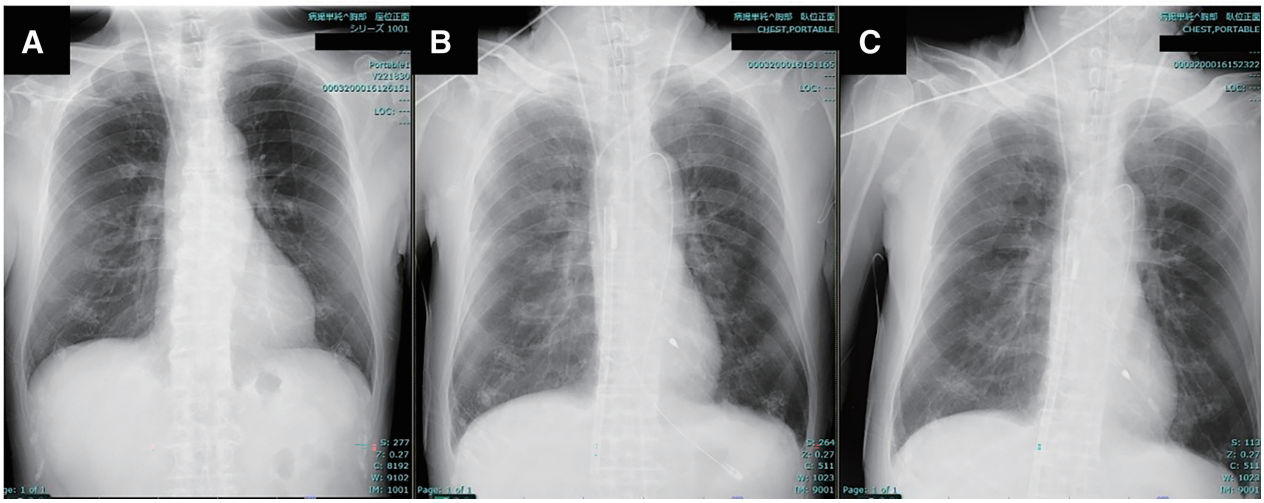

Fig. 3 A comprehensive evaluation of the X-ray findings revealed a significant improvement in the patient's pulmonary edema. (**A**) Preoperative. (**B**) Immediately after surgery. (**C**) Postoperative day 1.

dilation may have caused partial damage to the highly calcified valve leaflets. After BAV, the heavily calcified aortic valve remained fixed in an open position, leading to acute AR despite incomplete dilation. The balloon size for predilatation should be carefully selected. In retrospect, the use of a smaller balloon (e.g., 18 mm) may have mitigated mechanical stress on the heavily calcified leaflets and minimized the risk of leaflet disruption. Therefore, careful selection of balloon size is particularly important in patients with extremely severe calcification.[5)]

This AR was caused by leaflet disruption from BAV, which occurs in approximately 1%–2% of cases.[6)] Under these conditions, the initiation of VA-ECMO resulted in direct inflow of ECMO output into the left ventricle, causing a rapid and marked increase in LV end-diastolic pressure and the immediate onset of pulmonary edema. Moreover, the worsening AR combined with persistent VF virtually abolished effective LV ejection, leading to the rapid accumulation of blood within the left ventricle. Then, the LV overdistension caused papillary muscle tethering, resulting in separation of the A2 and P2 scallops and the development of acute MR, which further exacerbated pulmonary edema. The LV overdistension itself was also considered a contributing factor to the persistence of VF. Considering these conditions, LV decompression was deemed essential. Consequently, we inserted an Impella CP device across the deployed transcatheter aortic valve. LV unloading with the Impella rapidly relieved the ECMO-induced LV overdistension, thereby enabling successful defibrillation and restoration of sinus rhythm. Thereafter, the pulmonary edema improved markedly. In this case of acute hemodynamic collapse during TAVI, LV decompression using an Impella device was highly effective.

Conversely, inserting an Impella device across a TAVI valve carries a potential risk of mechanical interaction between the 2 devices, including possible valve displacement, structural damage, or systemic embolization due to Impella–valve contact.[7)] Moreover, the procedure is technically demanding. Although only a limited number of cases describing Impella insertion through a TAVI valve have been reported, they indicate that the approach is feasible.[1)] However, no established guidelines currently exist for the concomitant use of TAVI valves and Impella devices, and further clinical research is required. In the present case, the absence of TAVI valve damage may be attributable to the relatively short duration of Impella support, which lasted only 1 day. Importantly, even in the absence of overt structural damage, subtle leaflet injury associated with Impella insertion across transcatheter heart valves cannot be definitively excluded. Such injury may affect mid- to long-term valve durability; thus, careful follow-up is warranted.

Conclusion

A case was presented in which refractory VF caused by reduced coronary perfusion during TAVI required the initiation of ECMO. BAV resulted in the fixation of the heavily calcified aortic valve in an open position, thereby allowing VA-ECMO flow to pass directly into the left ventricle. This resulted in a marked increase in LV end-diastolic pressure, causing the abrupt onset of pulmonary edema and acute MR.

Rapid LV decompression using an Impella device contributed significantly to hemodynamic stabilization, thus enabling the successful restoration of sinus rhythm and resolution of pulmonary edema. Therefore, Impella

may be a valuable therapeutic choice for acute circulatory collapse during the perioperative period of TAVI.

Declarations

Ethics approval and consent to participate

Ethical approval was waived because this study is a single case report and does not involve research on human subjects.

Consent for publication

Written informed consent for publication of this case report and any accompanying images was obtained from the patient.

Data availability

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

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Author contributions

Conception and design: K. Matsumoto
Acquisition and analysis of data: K. Matsumoto and Y. Takeuchi
Manuscript drafting: K. Matsumoto and K. Okamoto
Interpretation of data and critical revision for important intellectual content: all authors
Final approval of the version to be published: all authors
Accountability for all aspects of the work: all authors.

Disclosure statement

All authors declare that there are no conflicts of interest in relation to the manuscript.

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