

Haemodynamic collapse immediately after transcatheter aortic valve implantation due to dynamic intraventricular gradient: a case report and review of the literature

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Background

Dynamic intraventricular obstruction after transcatheter aortic valve implantation (TAVI) has been previously reported. There is a risk of haemodynamic collapse in the case of left ventricular outflow tract (LVOT) obstruction due to systolic anterior motion (SAM) of the mitral valve.

Case summary

An 83-year-old woman with aortic stenosis (AS) was referred to our hospital for TAVI. Transthoracic echocardiography revealed a severely calcified aortic valve with a peak velocity of 6.3 m/s across the valve. Acceleration of blood flow (peak velocity 2.6 m/s) at the LVOT due to a septal bulge was also seen. Transfemoral TAVI was performed, and a 29 mm Evolut PRO was implanted under general anaesthesia. After the implantation, a complete atrioventricular block with junctional rhythm developed, and refractory hypotension occurred immediately. Transoesophageal echocardiography revealed LVOT obstruction due to SAM of the mitral valve associated with severe mitral regurgitation (MR), which was not observed preoperatively. Fluid infusion and catecholamine administration were not effective. However, after performing temporary pacing from the right ventricular (RV) apex, the LVOT obstruction and severe MR improved. Her haemodynamics stabilized, and we could complete the procedure. A dual-chamber permanent pacemaker with beta-blocker administration as a longer-term treatment further improved the LVOT obstruction. The patient was finally discharged to a rehabilitation hospital.

Discussion

Alertness and recognition of potential LVOT obstruction after TAVI are important. Pacing from the RV apex, as well as dual-chamber pacing, comprise a less invasive and feasible therapeutic option in such cases.

Keywords

Transcatheter aortic valve implantation • Left ventricular outflow tract obstruction • Systolic anterior motion of the mitral valve • Right ventricular pacing • Case report

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Learning points

- It is important to determine the risk factors, mechanism, and management of dynamic intraventricular obstruction after transcatheter aortic valve implantation (TAVI).
- Pacing from the right ventricular apex might be an effective therapeutic option for acute haemodynamic collapse after TAVI due to left ventricular outflow tract obstruction caused by systolic anterior motion of the mitral valve.
- Dual-chamber pacemaker implantation with beta-blocker administration could further improve haemodynamics and may be an option for longer-term management in such cases.

Introduction

Transcatheter aortic valve implantation (TAVI) has become a promising procedure in high-risk surgical patients with aortic stenosis (AS).¹ Similar to surgical aortic valve replacement (SAVR), dynamic intraventricular obstruction after TAVI has been reported.^{2,3}

Since the haemodynamic profile of such cases is similar to those of patients with hypertrophic cardiomyopathy, alcohol septal ablation (ASA) has been reported as a bail-out therapeutic choice.^{4,5} However, some cases might not be eligible for ASA due to technical issues. We describe the case of a patient who we successfully treated with medication and dual-chamber pacing after recovery from acute haemodynamic collapse by temporary pacing from the right ventricular (RV) apex. We also present a literature review on significant intraventricular obstruction after TAVI.

Timeline

Time	Events
1 month earlier	The patient was admitted to another hospital due to acute heart failure secondary to severe aortic stenosis. Her symptom was improved by oxygen and diuretics, then discharged.
2 weeks earlier	She was referred to our hospital in consideration of transcatheter aortic valve implantation (TAVI).
4 days earlier	Admission to our hospital.
Day 0	TAVI was performed. Haemodynamics collapsed after implantation of Evolut PRO. Severe left ventricular outflow tract obstruction attributable to systolic anterior motion of the mitral valve was observed. Temporary pacing from the right ventricle apex was effective.
Day 1	The patient experienced cardiopulmonary arrest due to self-removal of the temporary pacemaker because of delirium. A temporary pacing lead was inserted again, and return of spontaneous circulation was achieved 40 min from onset.

Continued

Continued

Time	Events
Day 7	Extubation
Day 12	A dual-chamber pacemaker was implanted.
Day 34	The patient was discharged to a rehabilitation hospital due to post-resuscitation encephalopathy.
9 months later	The patient was alive without cardiac symptom although she was in the nursing home because of the progression of cognitive decline and frailty.

Case presentation

An 83-year-old woman was admitted to another hospital due to acute heart failure secondary to severe AS. Her initial condition was improved by medical treatment, however, dyspnoea on exertion (New York Heart Association functional Class II) was still observed. Therefore, she was referred to our hospital in consideration of intervention to AS. At the time of admission, she was haemodynamically stable. She had a systolic murmur of Levine IV/VI at the second left sternal border, clear lung sounds, and no oedema of the lower extremities. Transthoracic echocardiography (TTE) revealed a peak velocity of 6.3 m/s, a peak pressure gradient of 158.8 mmHg, and a mean gradient of 97.1 mmHg. Blood flow acceleration (peak velocity: 2.6 m/s) at the left ventricular outflow tract (LVOT) due to septal bulge was also seen. However, systolic anterior motion (SAM) of the mitral valve was not observed, and mitral regurgitation (MR) was mild ([Supplementary material online, Videos S1 and S2](#)). The left ventricular wall was concentrically hypertrophied with both the septum and posterior wall measured 19 mm. The LV cavity was relatively small, measuring 42 mm at end-diastole, and the ejection fraction was 66%. Electrocardiography showed 58 b.p.m. under normal sinus rhythm ([Figure 1A](#)). Computed tomography showed the annular perimeter at 75.3 mm and an annular area at 424 mm². The basal septum was bulged and led to narrowing of the LVOT ([Figure 2](#)). Coronary angiography revealed no obstruction. We decided to perform TAVI due to the patient's age and frailty (Society of Thoracic Surgeons score of 4.34%, clinical frailty scale of 6). Owing to severe calcification leading to LVOT, a self-expanding valve was selected. Transcatheter aortic valve implantation was performed using a transfemoral

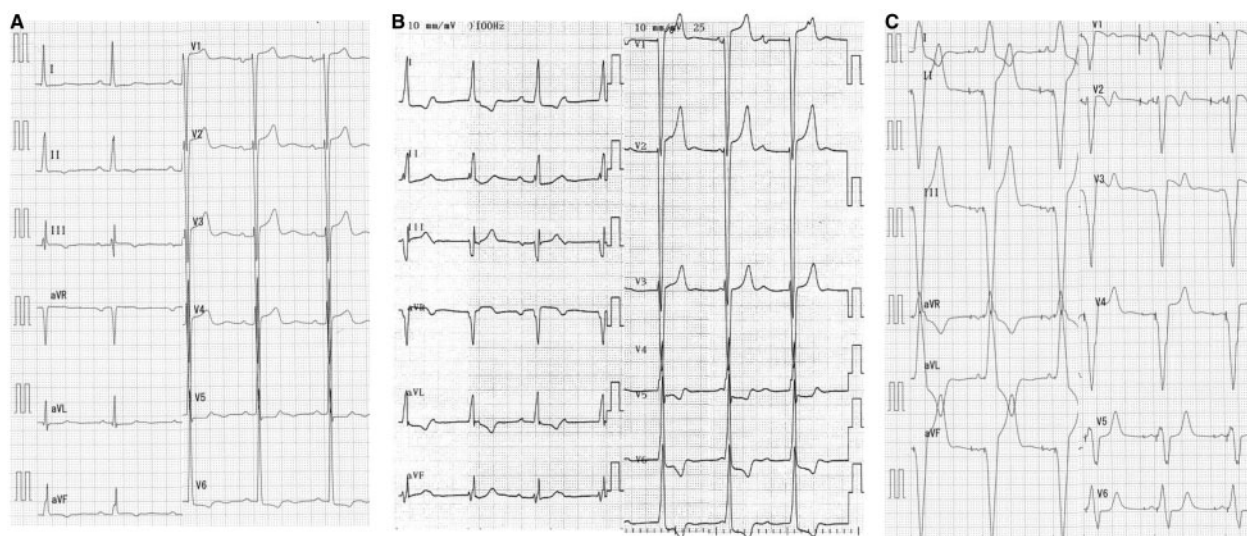


Figure 1 Electrocardiographic findings. (A) Baseline (58 b.p.m.). (B) After implantation of the transcatheter heart valve. Complete atrioventricular block with junctional rhythm (65 b.p.m.). (C) After permanent pacemaker placement (60 b.p.m.).

approach under general anaesthesia, and a 29-mm Evolut PRO (Medtronic, Minneapolis, MN, USA) was implanted after balloon dilatation. Immediately after implantation, a complete atrioventricular (AV) block with junctional rhythm (over 60 b.p.m.) developed (Figure 1B). Shortly after, her haemodynamics collapsed and refractory hypotension occurred. Fluid infusion (over 500 ml) and intravenous administration of dopamine (5 µg/kg/min) were not effective. Bolus infusion of norepinephrine and phenylephrine slightly increased the patient's blood pressure only temporarily. Transoesophageal echocardiography demonstrated normal functioning of the implanted valve and revealed LVOT obstruction attributable to SAM of the mitral valve associated with severe MR (Figure 3, Supplementary material online, Videos S3 and S4). However, after performing temporary pacing from the RV apex at 80 b.p.m., which was higher than the junctional rhythm, the LVOT obstruction and MR decreased. Thereafter, the haemodynamics stabilized. The patient was successfully extubated and transferred to the intensive care unit.

There, hypotension was reproducibly induced by interrupting the pacing. Transthoracic echocardiography revealed SAM of the mitral valve under junctional own beat, which was improved by RV pacing (Figure 4, Supplementary material online, Videos S5 and S6). When accidental self-removal of the pacing lead due to delirium occurred, the haemodynamics deteriorated, although junctional rhythm was observed and led to cardiopulmonary arrest. A temporal pacing lead was reinserted emergently while performing resuscitation; thereafter, a return of spontaneous circulation was achieved.

Since severe obstruction was still observed (peak velocity 6.8 m/s) under RV pacing, a beta-blocker (bisoprolol) was administered. However, significant acceleration (peak velocity 3.5 m/s) remained, and the complete AV block was persistent. Therefore, a dual-chamber permanent pacemaker was implanted. The permanent RV lead was placed at the apex because it showed better haemodynamic

effects than those of septal pacing (Supplementary material online, S7). After adjustment of AV synchrony and medication (bisoprolol: 5 mg, enalapril: 5 mg, and amlodipine: 2.5 mg), TTE revealed mild MR with no evidence of SAM, and the LVOT obstruction was almost resolved. The patient developed post-resuscitation encephalopathy, therefore she was finally discharged to a rehabilitation hospital 34 days after TAVI. At the 9-month follow-up, she had no cardiac symptoms although she was in the nursing home because of the progression of cognitive decline and frailty.

Discussion

Dynamic intraventricular obstruction has been reported to occur in approximately 15% of patients undergoing SAVR for AS.⁶ Its predictive factors were small LV diameter, asymmetrical hypertrophy, high ejection fraction, and high valve gradients.⁷ In contrast to hypertrophic obstructive cardiomyopathy, wherein obstruction is usually located in the LVOT, systolic obstruction after aortic valve replacement tends to be seen in the mid-ventricle because most cases involve concentric hypertrophy.^{6,7} However, asymmetrical hypertrophy, which is also seen in patients with AS and a septal bulge as in our case, might induce LVOT obstruction after TAVI. Dynamic LVOT obstruction is a complex phenomenon arising from the interplay of subaortic hypertrophy, LV ejection fraction, venturi effects, and anterior positioning of the mitral valve apparatus. It is associated with SAM of the anterior mitral leaflet, which causes significant MR. The treatment strategy usually focuses on maintaining LV filling pressure, slowing the heart rate to increase the diastolic filling time, and decreasing inotropy. Conservative therapies often improve the haemodynamics in patients with mid-ventricular obstruction.^{2,8,9} However, since LVOT obstruction can further deteriorate the haemodynamics, some cases reportedly require bail-out treatment (Table 1).^{3-5,10-13}

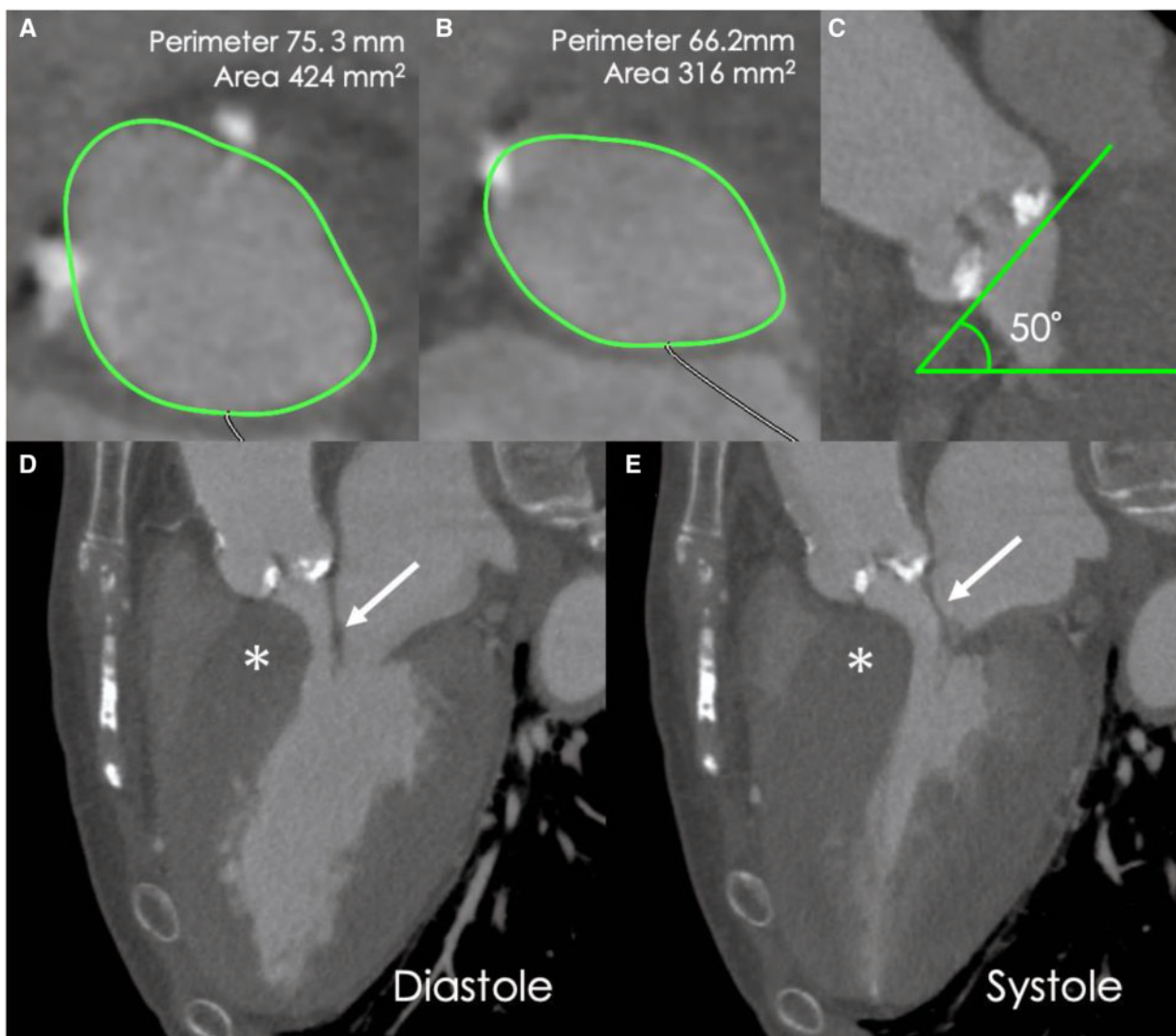


Figure 2 Pre-procedural cardiac computed tomography images. (A) Annular assessment. (B) Left ventricular outflow tract assessment. (C) The angulation of the aorta was 50°. (D, E) Long-axis computed tomography images of the diastolic and systolic phases showing interventricular septal bulge (*). Asymmetric septal hypertrophy or systolic anterior motion of the anterior mitral leaflet are not observed. Anterior mitral leaflet (arrow).

Concomitant septal myectomy in high-risk patients undergoing SAVR has been recommended.¹⁴ However, surgical resection might not be feasible for patients undergoing TAVI due to patient comorbidities and frailty. Currently, ASA has been reported to be a bail-out therapeutic choice to manage severe intraventricular gradient after TAVI.^{4,5,10–12} Alcohol septal ablation is less invasive than surgical resection. However, up to 20% of patients might not have an appropriate septal artery.¹⁵ Moreover, ASA requires experienced operators and equipment. Dual-chamber pacing is reported as a therapeutic alternative to hypertrophic obstructive cardiomyopathy and is less invasive than myectomy or ASA.¹⁶ A possible mechanism for its therapeutic effect has been suggested that initiation of electrical impulse at the RV apex alters the systolic contraction sequence of the basal septum. Although the pacing is not considered as the primary therapy for obstruction, cohort studies have indicated that pacing

effectively reduces the LVOT gradient and symptoms.^{16,17} In fact, RV apex pacing was essential in managing the acute haemodynamic collapse in our case. In addition, a dual-chamber pacemaker was also effective in reducing the residual LVOT gradient. A previous paper reported that disordered AV synchrony following TAVI caused intraventricular obstruction.⁹ Increasing the LV filling pressure by maintaining AV synchrony is important to reduce the gradient. Thus, dual-chamber pacing might be considered if there is no haemodynamic improvement with a single RV pacing.

Based on our literature review, one article has already reported the effectiveness of pacing in LVOT obstruction after TAVI.¹³ However, we concluded that our case was valuable as the first report to clearly demonstrate the crucial difference in outcomes with and without pacing (Figure 4, Supplementary material online, Videos S5 and S6).

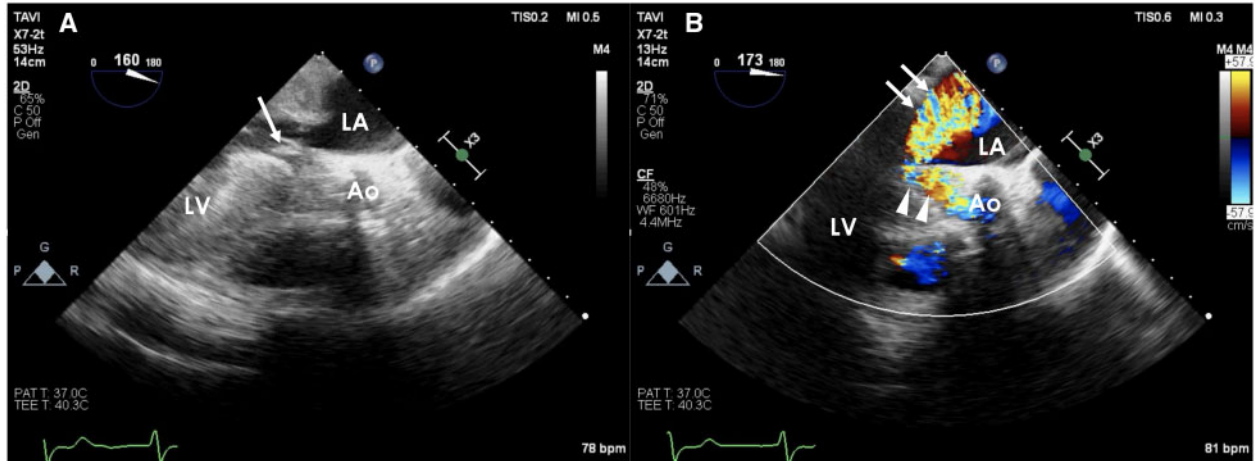


Figure 3 Intraoperative transoesophageal echocardiography after transcatheter aortic valve implantation. (A) Left ventricular outflow tract obstruction with systolic anterior motion of the anterior mitral valve leaflet (arrow). (B) Severe mitral regurgitation (arrow) and turbulent flow of the left ventricular outflow tract (arrowhead). The mitral regurgitation jet is blowing towards the posterior side.

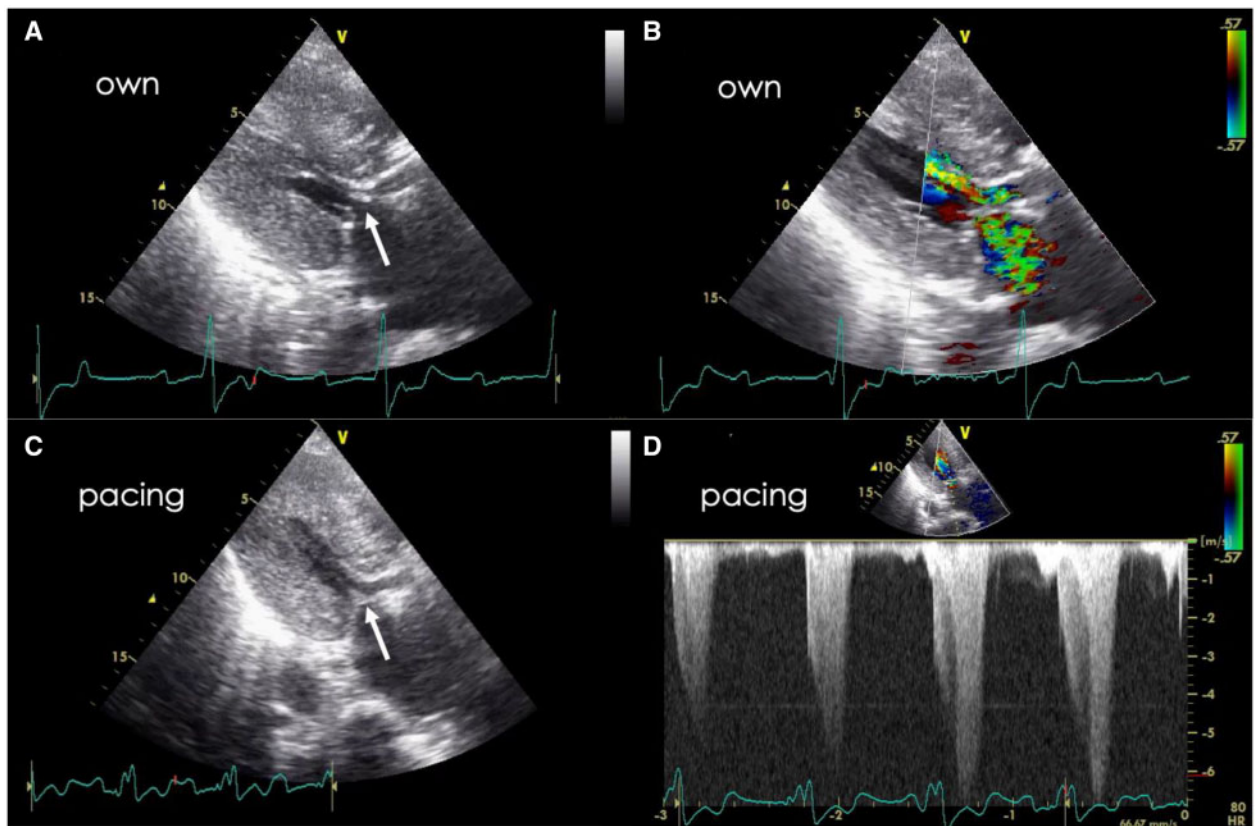


Figure 4 Transthoracic echocardiography after transcatheter aortic valve implantation. (A, B) Under own junctional rhythm. Left ventricular outflow tract obstruction with systolic anterior motion of the mitral valve (arrow). The anterior mitral leaflet is thickened. (C) Systolic anterior motion of the anterior mitral leaflet (arrow) and left ventricular outflow tract obstruction are decreased by right ventricular pacing. (D) The peak flow velocity and pressure gradient are 6.8 m/s and 185 mmHg, respectively, even under right ventricular pacing.

Table 1 Summary of published case reports of significant intraventricular obstruction after TAVI

	Age (years)	Sex	Pre TTE	Implanted valve	Onset	Symptoms	TTE findings	Treatment
Suh et al. ²	82	F	AVPG(p) 121 mmHg MVO (PG 12 mmHg)	23 mm Sapien (TF)	1 day later	Hypotension	MVO	Fluid Beta-blocker
Takeda et al. ¹³	91	M	AVPG(p) 152 mmHg Septal bulge	26 mm Sapien (TA)	Immediately	Not mentioned	LVOTO (PG 50 mmHg) SAM of the mitral valve MR severe	Beta-blocker Cibenzoline Pacemaker*
Soraja et al. ⁴	89	M	AVPG(m) 65 mmHg	26 mm Sapien (TA)	1 month later	Exertional dyspnoea	LVOTO (PG 55 mmHg) SAM of the mitral valve	Beta-blocker ASA*
Gerckens et al. ¹¹	88	F	AVPG(p) 115 mmHg	23 mm Sapien (TF)	12 h later	Hemodynamic collapse	MVO (PG 108 mmHg) SAM of the mitral valve MR moderate	Fluid, Pacemaker Beta-blocker Emergent ASA*
Krishnaswamy et al. ¹⁰	91	F	AVPG(m) 58 mmHg ASH	23 mm Sapien (TF)	Immediately	Hemodynamic collapse	LVOTO (PG 120 mmHg) SAM of the mitral valve MR severe	Emergent ASA*
Alfonso et al. ⁸	79	F	LVOTO (PG 12.5 mmHg) AVPG(m) 82 mmHg	26 mm CoreValve (TF)	Immediately	Asymptomatic	MVO (PG 120 mmHg)	Asymptomatic
Ibrahim et al. ⁹	81	F	AVPG(p) 109 mmHg	29 mm CoreValve (DA)	Immediately	Not mentioned	MVO	Induced by sinus arrest →spontaneous restoration
Leya et al. ³	87	F	AVPG(m) 45 mmHg Sigmoid septum	26 mm CoreValve (TF)	Immediately after BAV	Hemodynamic collapse	LVOTO+MVO SAM of the mitral valve MR severe	AV pacemaker Beta-blocker Valve in valve (26 mm CoreValve)* ASA (ad hoc)*
Yanagiuchi et al. ¹²	83	M	ASH, SAM of the mitral valve	26 mm Sapien 3 (TF)	Immediately	Hypotension	LVOTO (PG 121 mmHg)	ASA (ad hoc)*
Kitahara et al. ⁵	86	F	AVPG(p) 64 mmHg ASH, SAM of the mitral valve → Disappeared after beta-blocker initiation	23 mm Sapien 3 (TF)	15 min later	Hemodynamic collapse	LVOTO PG >50 mmHg SAM of the mitral valve MR severe	Fluid Beta-blocker infusion Emergent ASA*

ASA, alcohol septal ablation; ASH, asymmetrical septal hypertrophy; AVPG, aortic valve pressure gradient; BAV, balloon aortic valvuloplasty; DA, direct aortic; LVOTO, left ventricular outflow tract obstruction; (m), mean; MR, mitral regurgitation; MVO, mid-ventricular obstruction; (p), peak; PG, pressure gradient; SAM, systolic anterior motion; TA, trans aortic; TAVI, transcatheter aortic valve implantation; TF, transfemoral; TTE, transthoracic echocardiography.

*Most effective treatment.

Leya *et al.*³ reported a case in which severe LVOT obstruction was successfully managed by implanting a second self-expandable valve ('valve in valve'). Retaining the radial force at the LVOT might be a solution for LVOT obstruction. However, no report has compared balloon-expandable and self-expandable prosthetic valves. In our review, LVOT obstruction developed regardless of valve type (Table 1). From another perspective, prophylactic MitraClip (Abbott, Davis, CA, USA) implantation prior to TAVI was reported to be useful in preventing exacerbation of LVOT obstruction due to SAM.¹⁸ Further studies are warranted to clarify the effectiveness of such novel approaches.

Alertness and recognition of possible LVOT obstruction after TAVI are important. Pacing from the RV apex and dual-chamber pacing comprise a less invasive and feasible therapeutic option in such cases.

Lead author biography



Nana Endo, MD, is a senior resident of cardiology in Tokyo Women's Medical University. She graduated from Tokyo Women's Medical University in 2014. She finished junior resident program in 2016. After completing resident program of emergency medicine, she started her career as cardiologist since 2017. Membership: Japanese Circulation Society, Japanese Association of Cardiovascular Intervention and Therapeutics.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

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Slide sets: A fully edited slide set detailing these cases and suitable for local presentation is available online as [Supplementary data](#).

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: H.O. and J.Y. report that they belong to the division (Clinical Research division for Cardiovascular Catheter

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