CASE REPORT

Development of acute severe right heart failure after transcatheter aortic valve implantation in patient with left ventricle assist device-acquired aortic regurgitation

Sol ventrikül destek cihazı kaynaklı aort yetersizliğinin transkateterik aort kapak replasmanı tedavisi sonrası gelişen akut ciddi sağ kalp yetersizliği

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Summary– A 58-year-old man with a left ventricular assist device (LVAD), which had been implanted 1 year earlier, presented with rest dyspnea. Moderate to severe aortic regurgitation (AR), pre-postcapillary pulmonary hypertension, modarete right ventricular (RV) failure, and low cardiac output were observed at presentation. Transcatheter aortic valve implantation (TAVI) was performed to treat the AR and a self-expandable aortic valve was implanted. Within minutes, hypotension, RV and inferior vena cava dilatation, and left atrial (LA) and left ventricular (LV) collapse occurred and persisted despite LVAD speed reduction. It was observed that severe RV failure had developed and venoarterial extracorporeal membrane oxygenation (VA-ECMO) was applied. Following VA-ECMO treatment, the RV dimensions decreased, and the LA and LV dimensions began to increase, as well as the LVAD flow. Weaning from VA-ECMO was unsuccessful and exitus occurred on the fifth day after TAVI secondary to RV failure. It was surmised that the decrease in blood circulation from the aorta to the LV after treatment of severe AR with TAVI caused an acute increase in the cardiac output and the RV preload. The acute increase in the RV preload led to acute severe right heart failure. It is necessary to prepare the RV to compete with an acute increase in preload before TAVI even when there is only modarete RV failure.

Özet– Bir yıl önce, sol ventrikül destek cihazı (SVDC) implantasyon öyküsü olan 58 yaşında erkek hasta istirahat dispnesi ile başvurdu. Hastanın başvuru sırasında orta-ileri derecede aort yetersizliği (AY), pre-postkapiller pulmoner hipertansiyonu subklinik orta derece sağ ventriküler yetersizliği ve azalmış kardiyak debisi mevcuttu. AY tedavisi için transkatater aort kapak implantasyonu (TAVI) planlandı ve hastaya 'self-expandable' aort kapak takıldı. Hastada dakikalar içersinde hipotansiyon, sağ ventrikül ve inferiyor vena kavada dilatasyon, sol atriyum ve sol ventrikülde kollaps gelişti ve SCDC hızı azaltılmasına rağmen devam etti. SVDC hızının düşürülmesine rağmen kalp boşluklarının boyutlarında herhangi bir değişiklik görülmedi. Hastada ciddi sağ ventrikül yetersizliğinin geliştiğine karar verilerek veno-arteriyal EKMO tedavisi uygulandı. VA-EKMO'dan sonra sağ ventrikül boyutları azaldı, sol atriyum ve sol ventrikül boyutlarında ve SVDC akımında artış izlendi. Hasta sağ ventrikül yetersizliğinin devam etmesi nedeni ile EKMO'dan ayrılmadı ve işlemin 5. gününde hayatını kaybetti. Bu hastada, şiddetli AY'nin TAVI ile tedavisinden sonra aortadan sol ventriküle geri akımının azalmasının, kardiyak debi ve sağ ventrikül ön yükünde akut artışa ve sağ ventrikül ön yükündeki akut artışın da akut ciddi sağ kalp yetmezliğine neden olduğunu düşündük. Hastada orta derece sağ yetersizlik olsa bile, TAVI'den önce, sağ ventrikülü akut ön yük artışını tolere edebilecek şekilde medikal olarak hazırlamak gerekmektedir.

Left ventricular assist device (LVAD)-acquired aortic regurgitation (AR) is a commonly seen complication after permanent LVAD implantation. The using of continuous-flow LVAD affects 25% to 30% of patients within the first year of implantation.[1] A reduced aortic valve opening, local stasis, high shear stress, inversion of transvalvular gradient, thrombosis, fibrosis, and retraction or fusion of aortic cusps can cause AR.[2,3] In these patients, moderate or severe AR causes recirculation of antegrade blood flow from

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the aorta to the left ventricle (LV) and compromised cardiac output. Treatment is necessary, as it is associated with a poor prognosis.[4] There are various therapies for AR, including noninvasive treatment, percutaneous treatment, and open heart surgery.[1] A noninvasive strategy (adjusting LVAD speed, lowering the blood pressure if it is high) is the first-line treatment. If that fails, percutaneous treatment, such as percutaneous device closure, transcatheter aortic valve implantation (TAVI), or bioprosthetic aortic valve replacement should be performed.[4]

Abbreviations:

AR	*Aortic regurgitation*
LV	*Left ventricle*
LVAD	*Left ventricular assist device*
PVR	*Pulmonary vascular resistance*
RHC	*Right heart catheterization*
RV	*Right ventricle*
TAVI	*Transcatheter aortic valve implantation*
VA-ECMO	*Venoarterial extracorporeal membrane oxygenation*

Presently described is a case of severe right ventricular (RV) heart failure developing after treatment for LVAD-acquired AR with TAVI in a patient with underlying subclinical moderate RV failure.

CASE REPORT

A 58-year-old man with a history of coronary artery bypass graft, ischemic cardiomyopathy for 7 years, and LVAD implantation (HeartMate III; Abbott Vascular, Inc., Santa Clara, CA, USA) about 1 year prior presented with exertional dyspnea ongoing for 3 months, and fatigue and dyspnea at rest for 1 month. The findings from the physical examination, echocardiography, and right heart catheterization (RHC) at pre-LVAD, post- LVAD sixth month, and hospitalization are summarized in Table 1.

Table 1. Details of physical examination, echocardiography, and right heart catheterization pre-LVAD, post-LVAD 6th month, and hospitalization

	Pre-LVAD	Post-LVAD 6th month	Hospitalization
Physical examination			
Rales on lungs	Bilateral basal	No	Bilateral basal
JVD	+	No	+
PTE	+/+	–/–	–/–
Ascites	No	No	No
Echocardiography			
Aortic valve morphology	Normal	Increased echogenicity of cusps	LCC and RCC restricted and malcoaptated
AR	No	Mild	Moderate to severe
RV dilatation	Moderate	Moderate	Moderate
TAPSE (cm)	1.4	1.4	1.2
PSV (cm/sec)	9	8	8
FAC (%)	35	Not confirmed due to bad echogenicity	Not confirmed due to bad echogenicity
IVC (cm)	2.1 (no plethora)	1.9 (no plethora)	2.1 (no plethora)
RHC			
mPAP (mmHg)	47	22	39
PAWP	30	10	25
PVR (Wood)	6.8	2.7	5.6
RAP (mmHg)	15	10	13
RVSWI (mmHgxL/m^2)	605	590	350
CO (L/min)	2.5	4.4	2.5

AR: Aortic regurgitation; CO: Cardiac output; FAC: Fractional area change; IVC: Inferior vena cava; JVD: Jugular venous dilatation; LCC: Left coronary cusp; LVAD: Left ventricular assist device; mPAP: Mean pulmonary arterial pressure; PAWP: Pulmonary arteriolar wedge pressure; PSV: Peak systolic velocity; PTE: Pretibial edema; PVR: Pulmonary vascular resistance; RAP: Right atrial pressure; RCC: Right coronary cusp; RV: Right ventricle; RVSWI: Right ventricular stroke work index; TAPSE: Tricuspid annular systolic excursion.

Pre-LVAD echocardiography had indicated a normal aortic valve, no AR, and mild RV failure. There were no clinical findings of right heart failure in the early post-LVAD follow-up period.

The patient was asymptomatic at the sixth month follow-up visit, and there was no evidence of right or left heart failure on physical examination. Echocardiography showed that the aortic valve did not open in any cycle (Video 1*), a slight increase in valve echogenicity, mild AR (Fig. 1a), and a mild decrease in RV function (Table 1). RHC revealed a decrease in pulmonary pressure and pulmonary vascular resistance (PVR) as well as a decrease in RV function compared with the pre-LVAD findings (Table 1). The LVAD rate was reduced from 4600 rpm to 4000 rpm due to the absence of clinical signs of right or left heart failure.

At the 10th month post-LVAD, the patient presented with exertional dyspnea. Echocardiography indicated that the aortic valve did not open in any cycle. A coaptation defect was present (Fig. 1b) as well as moderate AR (Fig. 1c) in both systole and diastole (Fig. 1d), an increase in LV and RV diameters, and a decrease in RV function compared with the control measurements at 6 months. The device speed was increased from 4000 rpm to 4600 rpm due to the increase in LV size and exertional dyspnea.

Despite increasing the rate of the device, the patient was hospitalized because of increased dyspnea. There were no overt right heart failure findings on physical examination. Transthoracic and transesophageal echocardiography showed no aortic valve opening, retraction in the non-coronary cusp and left coronary cusp (Video 2*), and moderate-to-severe AR (both in diastole and systole) from this region (Video 3*). It was determined that the AR increased when the device speed was increased (Fig. 2a-d). RHC during hospitalization revealed that the pulmonary pressure and PVR had increased, while the cardiac output and RV functions had decreased compared with the sixth month catheter findings. The echocardiographic and RHC findings demonstrated moderate RV dysfunction (Table 1). It was thought that the patient's signs and symptoms were due to AR because of an increase in an AR jet and the dyspnea complaints developed when the LVAD rate was increased. A noninvasive strategy to treat the AR by decreasing the LVAD rate was attempted, but this strategy failed because it caused a decrease in the mean blood pressure to <60 mmHg and

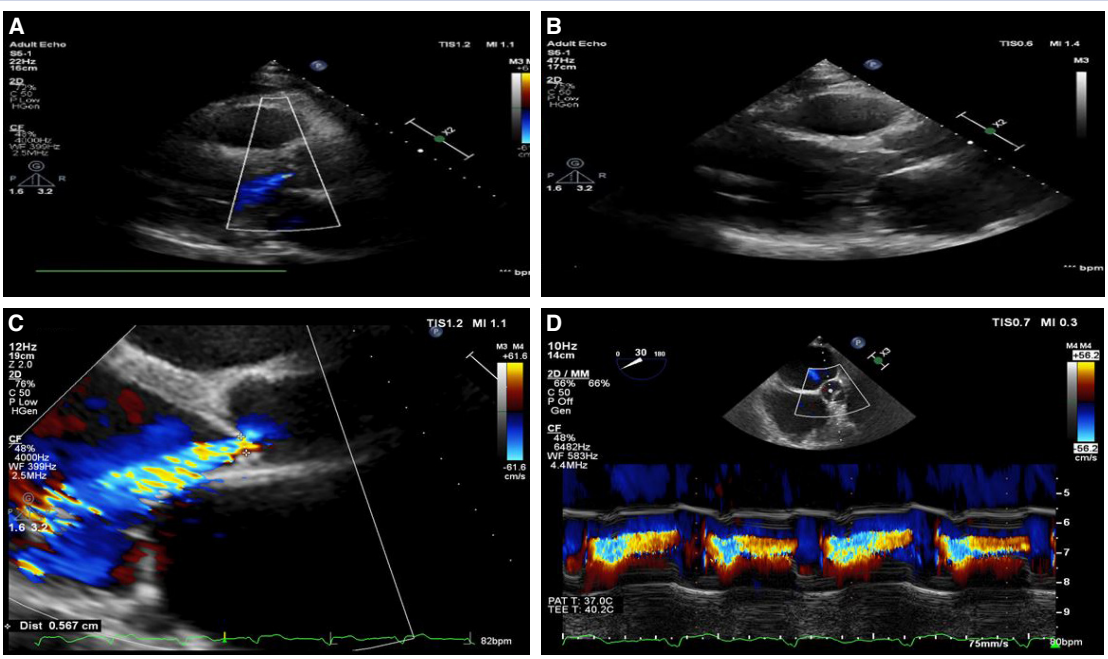

Figure 1. **(A)** Transthoracic echocardiography (TTE) results illustrating mild aortic regurgitation (AR) at the sixth month control visit; **(B)** TTE image revealing a coaptation defect at the 10th month follow-up; **(C)** TTE showing moderate-to-severe AR at the 10th month control visit. The vena contracta measured 5.6 mm; **(D)** Color M-mode echocardiography showed that aortic regurgitation was present during all of the diastole and mid to end systole.

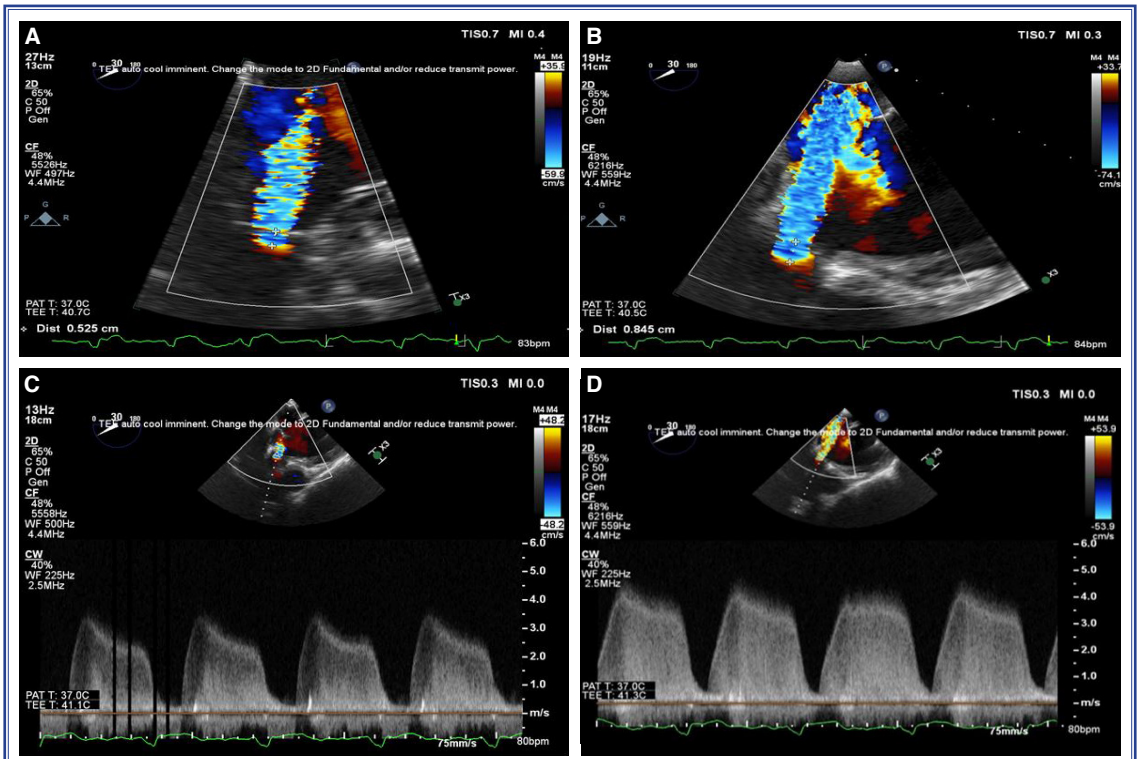

Figure 2. **(A)** Transthoracic echocardiography (TTE) transgastric view showing the proximal isovelocity surface area (PISA) of aortic regurgitation (AR) at a left ventricular assist device (LVAD) speed of 3600 rpm; **(B)** TEE transgastric view of PISA of AR at an LVAD speed of 4600 rpm; **(C)** Continuous wave Doppler image of the AR at an LVAD speed of 3600 rpm; **(D)** Continuous wave Doppler image of the AR at an LVAD speed of 4600 rpm.

an increase in fatigue symptoms in the patient. The heart team decided to perform TAVI to treat the AR because open heart surgery was regarded as high risk.

Transaortic valve implantation was performed according to the local standard protocol under general anesthesia. A preprocedural multislice computed tomography image demonstrated an effective perimeter from the annulus of 24 mm, and no calcification on the valve cusps, annulus or commissures. Transesophageal echocardiography was performed during the procedure (Fig. 3a). To avoid paravalvular AR, an oversized (no. 34), self-expandable Evolut R valve (Medtronic Inc., Minneapolis, MN, USA) implantation was performed (Fig. 3b, c). Just before the deployment of the valve, LVAD output was reduced to provide a stable intraannular valve position. After deployment of the valve, the previously reduced LVAD output parameters were returned to preprocedural levels. The severity of AR was observed to decrease, but residual mild AR persisted (Fig. 3d). Valve-in-valve implantation was planned for the residual AR. However, hypotension was observed within 10 minutes after the implantation of the first valve. Echocardiography recorded shortly thereafter showed that the RV and inferior vena cava were highly dilated, the left atrium (LA) had shrunk, and that the LV had collapsed (Fig. 4a). A reduction in the LVAD flow was attempted, but the severe suction in the LV and dilatation of RV did not improve. Based on these findings, it was decided that acute right heart failure had developed in this patient who previously had only subclinical moderate right heart failure. Venoarterial extra corporeal membranous oxygenation (VA-ECMO) was applied to treat the acute RV failure. It was determined that the RV dimensions decreased and the LA and LV dimensions returned to pre-procedure sizes after VA-ECMO (Fig. 4b). Cardiac resuscitation was performed for 20 minutes before the VA-ECMO implantation.

VA-ECMO weaning was unsuccessful on the third day post TAVI as a result of continued RV failure (Fig. 4c, d). Exitus occurred on the fifth day secondary to multiorgan failure.

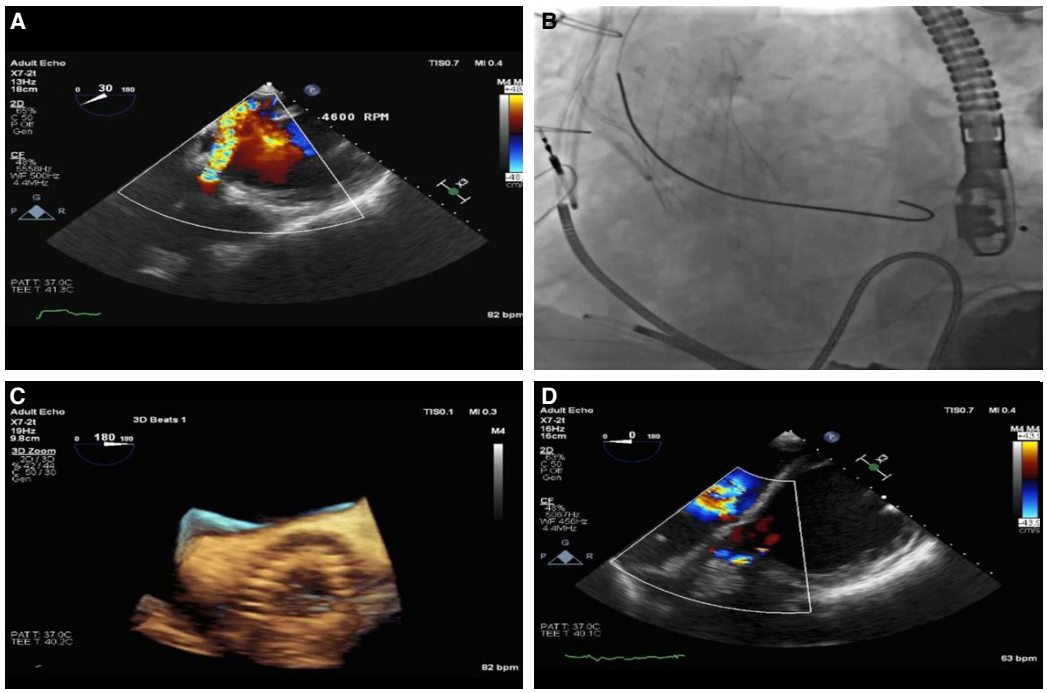

Figure 3. **(A)** Transthoracic echocardiography (TTE) transgastric view demonstrating aortic regurgitation during the procedure before the aortic valve deployment; **(B)** Fluoroscopic view after aortic valve deployment; **(C)** 3D-transesophageal view of the aortic valve after deployment; **(D)** Transgastric view of mild paravalvular aortic regurgitation after deployment.

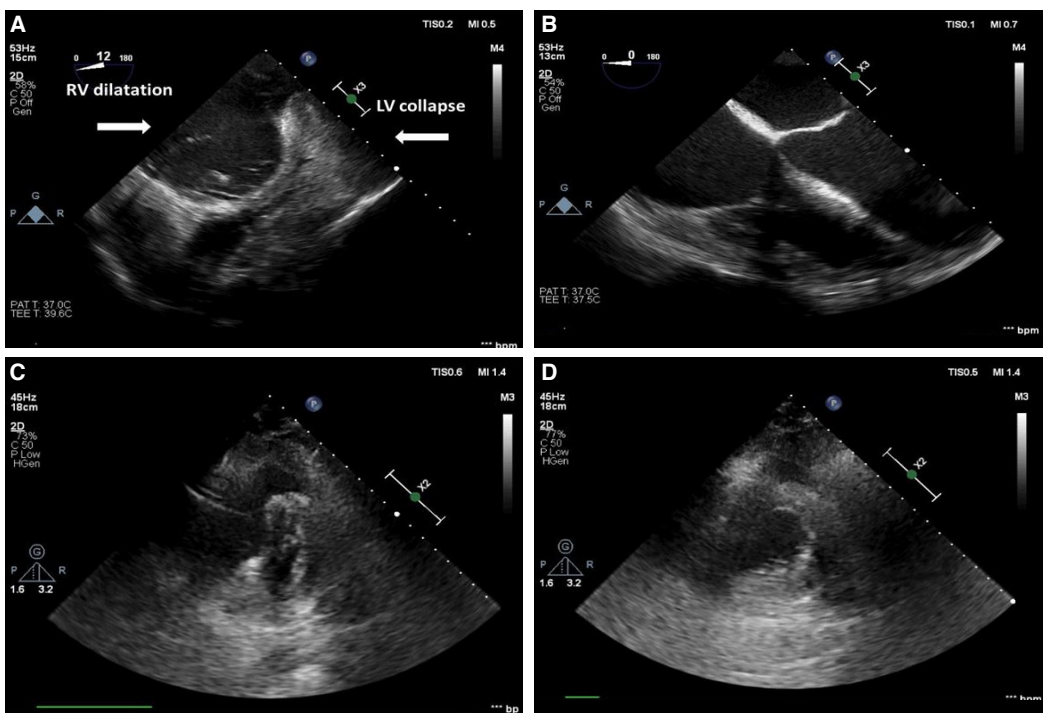

Figure 4. **(A)** Transgastric view illustrating that the left ventricle (LV) had collapsed and the right ventricle (RV) had dilated 10 minutes after deployment; **(B)** Mid-esophageal view shows that the LV and RV dimensions returned to preprocedural levels; **(C)** An image of the LV collapse when the extracorporeal membrane oxygenation (ECMO) flow decreased; **(D)** The LV collapse decreased when the ECMO flow increased.

DISCUSSION

Moderate or severe LVAD-acquired AR increases the symptoms of heart failure and is associated with a worse prognosis and increased support duration.4 A continuous closed aortic valve, nonpulsatile blood flow, and inversion of the transvalvular gradient are risk factors for AR.[2,3] In this case, during the patient's follow-up, it was observed that the aortic valve was continuously closed though the device settings were adjusted to keep a mean blood pressure between 60–80 mmHg and to allow the aortic valve to open intermittently. The continuously closed aortic valve may have caused cusp retraction and a gradual increase in AR during follow-up.

Invasive treatment should be considered in severely symptomatic patients who are refractory to medical and/or device management therapy. In patients who have a high surgical risk or contraindication for redo-sternotomy, minimally invasive procedures like TAVI or a percutaneous device closure can be used as an alternative to surgery.[1] In this case, percutaneous treatment was planned because the medical/device treatment failed and we selected TAVI because percutaneous device closure was not appropriate due to an eccentricity of the malcoaptation zone.

There are anecdotal reports of the use of TAVI to treat patients with LVAD-acquired AR. TAVI has been reported to be successful in decreasing symptoms in these patients.[1] In most cases, a self-expandable prosthesis is used rather than a balloon-expandable device in pure AR because there is potential risk of annular rupture due to the requirement of oversizing of prosthetic valve to achieve anchoring to the noncalcified aortic valve.[5] In this case, we used a no. 34. self-expandable Evolut R prosthetic valve for a 24-mm aortic annulus in order to prevent valve migration.

Paravalvular AR, inadequate sealing, valve embolization, annular rupture, and conduction disturbances are well-known potential complications of TAVI.[6] Reported complications of TAVI in patients with LVAD-acquired AR include valve migration to the LV apex and periprocedural death (range from 31% to 70%) mostly as a result of RV failure, and it has been noted that high pulmonary arterial pressure is a risk factor for RV failure after TAVI, as in our patient.[5,7,8] In our case, there was moderate RV dysfunction and dilatation, as well as pulmonary hypertension, but no clinically overt RV failure before the TAVI procedure. Nonetheless, severe RV failure and collapse developed shortly after prosthetic valve deployment. Severe LVAD-acquired AR causes a decrease in the cardiac output and the RV preload, and correction of AR increases both the cardiac output and the RV preload. Although there is not enough information in the literature, we recommend that medical treatments such as milrinone, nitric oxide, and phosphodiesterase inhibitors be used before the procedure to reduce the risk of RV failure after TAVI (especially in patients with pulmonary hypertension). In addition, excess fluid treatment should be avoided during the procedure.

Conclusion

The cause of severe RV failure in this patient appears to have been an acute increase in the RV preload when blood circulation from the aorta to the LV decreased after the TAVI procedure. We concluded that, even when there is only mild or moderate RV failure, it is necessary to decrease the RV afterload before the procedure to compete with an acute increase in preload after TAVI.

*Supplementary video file associated with this article can be found in the online version of the journal.

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Authorship contributions: Concept: O.N.; Design: B.Z; Supervision: K.C.; Materials: B.Z.; Data collection: G.E; Literature seach: D.C.; Writing: B.Z.; Critical revision: O.N., K.C.

REFERENCES

1. Bouabdallaoui N, El-Hamamsy I, Pham M, Giraldeau G, Parent MC, Carrier M, et al. Aortic regurgitation in patients with a left ventricular assist device: A contemporary review. J Heart Lung Transplant 2018;37:1289–97. [CrossRef]
2. Cowger J, Pagani FD, Haft JW, Romano MA, Aaronson KD, Kolias TJ. The development of aortic insufficiency in left ventricular assist device-supported patients. Circ Heart Fail 2010;3:668–74. [CrossRef]
3. Hatano M, Kinugawa K, Shiga T, Kato N, Endo M, Hisagi M, et al. Less frequent opening of the aortic valve and a continuous flow pump are risk factors for postoperative onset of

aortic insufficiency in patients with a left ventricular assist device. Circ J 2011;75:1147–55. [CrossRef]

4. Truby LK, Garan AR, Givens RC, Wayda B, Takeda K, Yuzefpolskaya M, et al. Aortic Insufficiency During Contemporary Left Ventricular Assist Device Support: Analysis of the INTERMACS Registry. JACC Heart Fail 2018;6:951–60.
5. Phan K, Haswell JM, Xu J, Assem Y, Mick SL, Kapadia SR, et al. Percutaneous Transcatheter Interventions for Aortic Insufficiency in Continuous-Flow Left Ventricular Assist Device Patients: A Systematic Review and Meta-Analysis. ASAIO J 2017;63:117–22. [CrossRef]
6. Neragi-Miandoab S, Michler RE. A review of most relevant complications of transcatheter aortic valve implantation. ISRN Cardiol 2013;2013:956252. [CrossRef]
7. Ong BH, Chiam PT, Sim DK, Tan TE. Post-implantation transcatheter aortic valve migration in a left ventricular assist device patient with severe aortic insufficiency. Eur Heart J 2014;351616. [CrossRef]
8. Retzer EM, Sayer GT, Fedson SE, Nathan S, Jeevanandam V, Friant J, et al. Predictors of survival following trans-catheter aortic valve closure for left ventricular assist device associated aortic insufficiency. Catheter Cardiovasc Interv 2016;87:971–9. [CrossRef]

Keywords: Aortic regurgitation; aortic valve implantation; right ventricular failure; transcatheter; ventricular assist device.

Anahtar sözcükler: Aort yetersizliği; aort kapak replasmanı; sağ ventrikül yetersizliği; transkateterik; sol ventrikül destek cihazı.