ESC European Society of Cardiology

European Heart Journal - Case Reports (2022) **6**, 1–7
https://doi.org/10.1093/ehjcr/ytac277

CASE REPORT
Other

Successful recovery from refractory hypoxia due to right-to-left shunting associated with iatrogenic atrial septal defect after catheter ablation in a patient with a left-ventricular assist device: a case report

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Received 4 November 2021; first decision 31 December 2021; accepted 29 June 2022; online publish-ahead-of-print 22 July 2022

Background Catheter ablation (CA) has been reported to be an effective therapeutic option for ventricular arrhythmias, even in patients with a left-ventricular assist device (LVAD). However, the issues of right-to-left shunting due to iatrogenic atrial septal defect (iASD) associated with procedures for CA have not been well documented. We describe a rare case of refractory hypoxia associated with right-to-left shunting via iASD after CA through the transseptal approach in an LVAD patient.

Case summary A 52-year-old Asian man with a continuous-flow implantable LVAD and progressive right ventricular (RV) dysfunction was admitted because of refractory ventricular tachycardia (VT) and subsequent right heart failure. Since VT could not be controlled by intravenous administration of multiple antiarrhythmic drugs, VT ablation via the transseptal approach was performed. Ventricular tachycardia was terminated to the sinus rhythm after VT ablation; however, hypoxia associated with significant right-to-left shunting across the iASD was detected. Intensive medical management, such as an adjusted mechanical ventilator to increase pulmonary vascular compliance and adjustment of LVAD pump speed, as well as the use of intravenous inotropes to support impaired RV function successfully stabilized the haemodynamic and improved hypoxia for the disappearance of right-to-left shunting. Echocardiography at 7 months after CA showed that the significant iASD and right-to-left shunting had disappeared.

Discussion The evaluation of RV function prior to VT ablation via the transseptal approach is important in the postoperative management of patients with LVAD, because RV dysfunction may cause refractory hypoxia due to iASD.

Keywords Left-ventricular assist device • Ventricular arrhythmia • Ablation • Iatrogenic atrial septal defect • Hypoxia • Case report

ESC Curriculum: 5.6 Ventricular arrhythmia • 6.7 Right heart dysfunction • 7.5 Cardiac surgery

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Handling Editor: Constantinos Bakogiannis
Peer-reviewers: Christoph Clemens Kaufmann; Zaid Iskander
Compliance Editor: Debbie Falconer
Supplementary Material Editor: Ameenathul Mazaya Fawzy

Learning points

- Pre-existing impaired right ventricular function may be responsible for the development of refractory right-to-left shunting after catheter ablation via transseptal left atrial puncture in patients with a left-ventricular assist device (LVAD).
- Right ventricular function may need to be evaluated when planning catheter ablation via transseptal left atrial puncture in patients with an LVAD.
- The intensive medical management could achieve recovery from the refractory right-to-left shunting following iatrogenic atrial septal defect in patients with an LVAD.

Introduction

Ventricular arrhythmias (VAs) are common comorbidities in patients with a left-ventricular assist device (LVAD). Sustained VAs may lead to an increased risk of right ventricular (RV) failure, pump thrombosis, and decreased quality of life.[1–3] Catheter ablation (CA) is the effective treatment for refractory VAs.[4,5] As pre-existing patent foramen ovale (PFO) is known to cause right-to-left shunting in patients with LVAD, iatrogenic atrial septal defect (iASD), caused by a transseptal puncture, may also lead to a refractory right-to-left shunt post-CA. However, the impact of right-to-left shunting via iASD after CA has not been well documented.[6] Here, we describe a rare case of refractory hypoxia associated with right-to-left shunting via iASD after CA through a transseptal approach in an LVAD patient.

Timeline

3 years prior-	Patient was diagnosed with advanced heart failure due to left main trunk-acute myocardial infarction and had been implanted with a continuous-flow implantable left ventricular assist device (LVAD) (HeartMate Ⅱ®).
6 months prior-	He had undergone aortic root replacement with bioprosthetic aortic valve replacement for secondary annuloaortic ectasia, and device exchange to a HeartMate 3® was performed for pump thrombosis.
4 months prior-	A more recent right heart catheterization (RHC) suggested impaired right ventricular (RV) function.
Admission-	He presented with persistent dyspnoea for a week. The electrocardiogram showed ventricular tachycardia (VT), and chest radiography showed pleural effusion.
Day 14-	He had a VT storm that did not return to a sinus rhythm even after repeated electrical shock.
Day 36-	Catheter ablation was performed to the VT storm via transseptal approach to the left ventricle.
Post-operative day (POD) 0-	Transesophageal echocardiography (TEE) revealed significant right-to-left shunting across the residual iatrogenic atrial septal defect (iASD) after a transseptal puncture, which led to the desaturation and hypoxia.
POD 1-	RHC suggested further deteriorated RV function and right-to-left shunting.
POD 4-	The optimization of pump speed, mechanical ventilator setting, and body fluid level, and the administration of intravenous inotropes for support of RV function resulted in improved hypoxia and weaning off from the mechanical ventilator.
POD 61-	He was discharged and had no persistent VT that could cause hemodynamic instability.
7 months follow-up-	Follow-up TEE did not show apparent right-to-left shunting.

Case presentation

A 52-year-old Asian man had suffered a left main trunk-acute myocardial infarction 3 years prior. Temporary percutaneous mechanical support was initiated, followed by an extracorporeal LVAD (Biofloat™, Nipro, Osaka, Japan; see Supplementary material online, *Figure S1A*), ensuring an adequate physiological flow. However, as his cardiac function did not improve, a continuous-flow implantable LVAD (HeartMate II™, Abbott, Abbott Park, IL, USA; see Supplementary material online, *Figure S1B*) was implanted while waiting for heart transplantation. He had undergone aortic root replacement with bioprosthetic aortic valve replacement (Bentall procedure) for secondary annuloaortic ectasia and device exchange to a HeartMate 3™ (Abbott, Abbott Park, IL, USA; see Supplementary material online, *Figure S1C*) for pump thrombosis 6 months prior. Recent right heart catheterization (RHC) suggested impaired RV function (*Table 1*).

He presented with persistent dyspnoea for a week. His vital signs upon admission included: heart rate, 150 b.p.m.; mean systolic blood pressure, 92 mmHg; and transcutaneous oxygen saturation (SpO_2), 91% (room air). His breath sounds were decreased in the right chest area, and he had lower leg oedema. Electrocardiogram (ECG) showed ventricular tachycardia (VT; *Figure 1A*), and chest radiography showed bilateral pleural effusions with a right-sided predominance (*Figure 2*). The RHC, performed when his heart rhythm temporarily returned to sinus rhythm, showed further RV function deterioration (*Table 1*).

Table 1 Right heart catheterization test findings

	Baseline[a]	Pre-ablation[b]	Post-ablation[b]	At discharge[c]
Pump speed (r.p.m.)	5400	5400	5000	5400
PCWP (mmHg)	12	16	14	3
PAP (mmHg)	24/16 (20)	19/14 (16)	23/18 (20)	16/6 (10)
RVP (mmHg)	22/~14	21/~15	23/~20	15/~6
RAP (mmHg)	13	14	20	6
ABP (mmHg)	89	76	73	83
CO (L/min)	5.82	6.24	3.40	4.81
CI (L/min/m^2)	2.85	3.00	1.66	2.53
PVR (Woods)	1.4	0	1.8	1.5
PAPi	0.6	0.36	0.25	1.67
PCW/RA	0.9	1.1	0.7	0.5
RVSWI (g·m/m^2)	250	75	0	126

r.p.m., revolutions per min; PCWP, pulmonary capillary wedge pressure; PAP, pulmonary artery pressure; RVP, right ventricular pressure; RAP, right atrium pressure; ABP, arterial blood pressure; CO, cardiac output; CI, cardiac index; PVR, pulmonary vascular resistance; PAPi, pulmonary artery pulsatility index; RVSWI, right ventricular stroke work index.
[a]The baseline RV function deteriorated.
[b]Sustained VT and VT ablation further worsened RV function.
[c]RV function improved 60 days after VT ablation; however, impaired RV function remained.

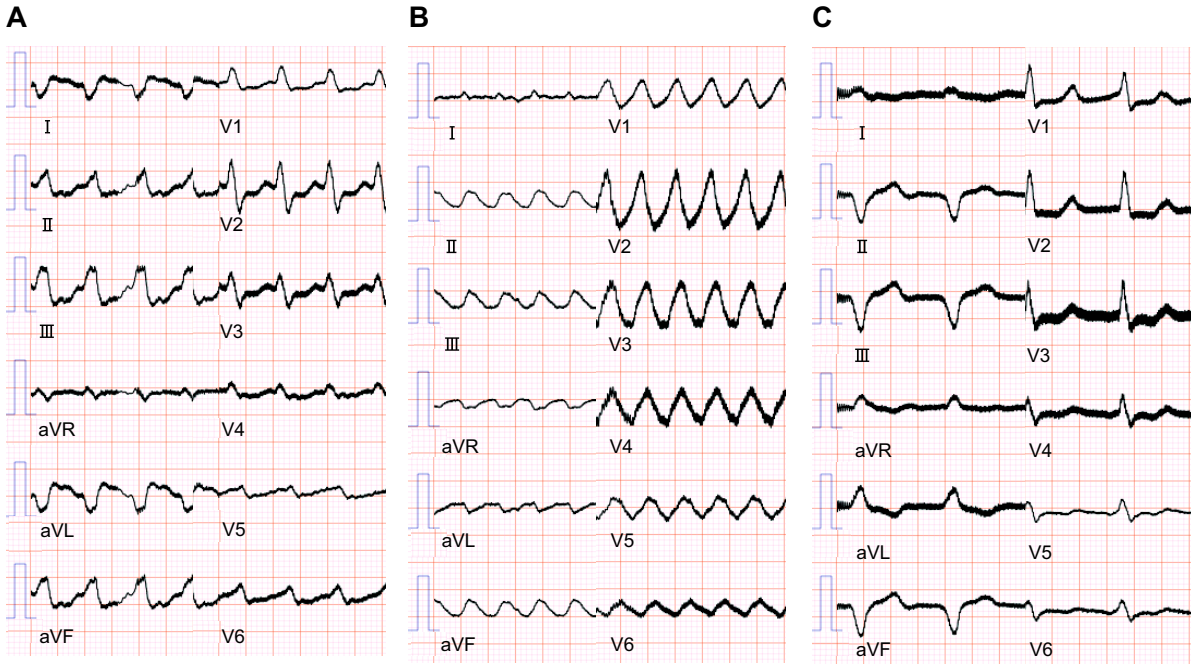

Figure 1 Electrocardiographic findings. (*A*) An electrocardiogram on the day of admission, which showed a sustained ventricular tachycardia of 150 b.p.m. (*B*) Uncontrolled ventricular tachycardia, which led to the decision to perform ventricular tachycardia ablation. (*C*) An electrocardiogram wherein ventricular tachycardia was terminated by the ventricular tachycardia ablation. Unfortunately, clear electrocardiogram findings were not obtained because the left-ventricular assist device interfered with other electrical devices.

Subsequently, he had a VT storm which could not be terminated by electrical shock (*Figure 1B*). Preoperative transoesophageal echocardiography (TEE) did not show an apparent PFO or ASD.

Catheter ablation was performed under general anaesthesia. Access to the left ventricle (LV) was limited for the transseptal approach, as the patient had undergone a prior Bentall operation. Air-trap filters were attached to all peripheral venous lines to prevent paradoxical air embolization. All procedures were performed using the CARTO™ electroanatomic mapping system (Biosense Webster, Diamond Bar, CA, USA). According to the ECG with a

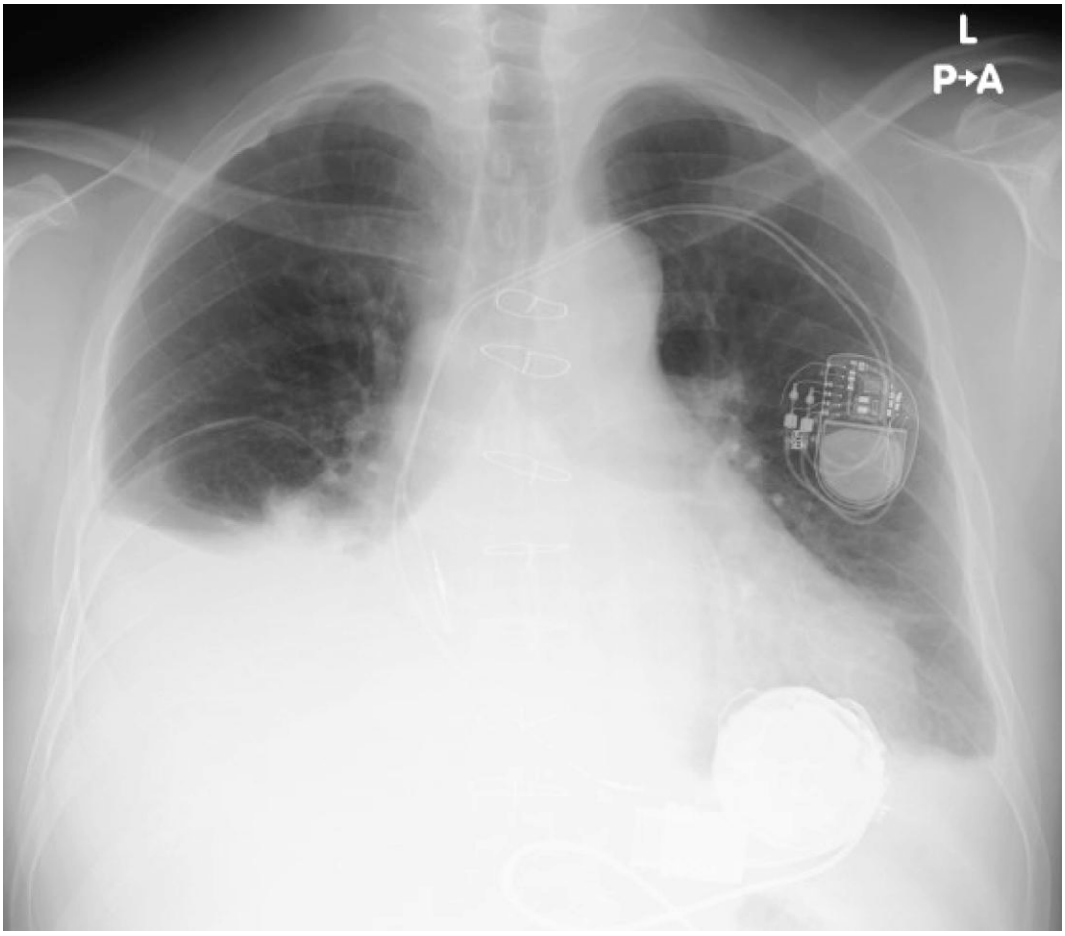

Figure 2 Chest radiograph findings. The figure shows a chest radiograph with bilateral pleural effusion on the day of admission.

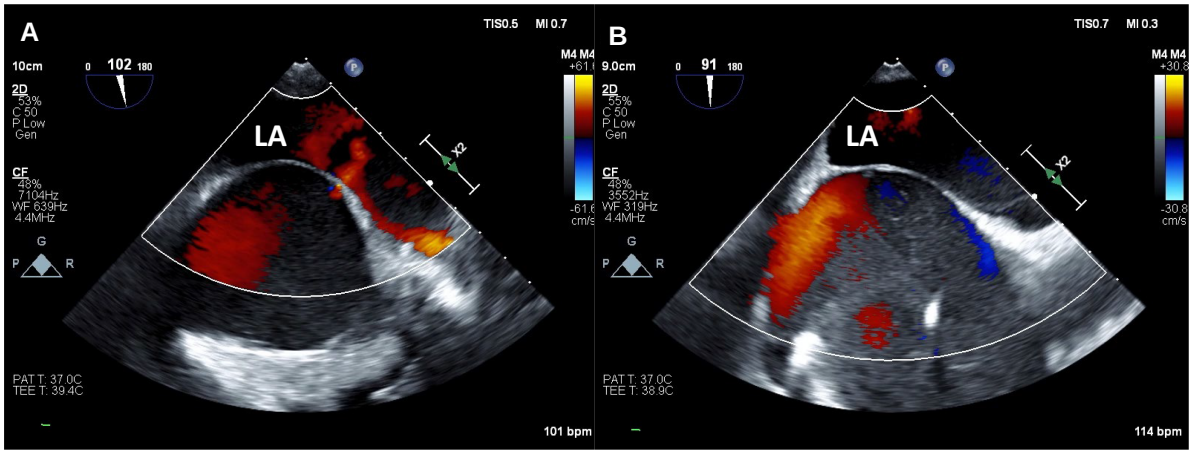

Figure 3 Transoesophageal echocardiographic findings. (*A*) The iatrogenic atrial septal defect immediately after the transseptal puncture for ventricular tachycardia ablation. The size of the defect was 2.0 mm, and the pressure gradient between the right and left atria was 6 mmHg. Re-occlusion of the iatrogenic atrial septal defect with the ablation sheath improved hypoxia. A right-to-left shunting was thought to have contributed to the hypoxia. (*B*) A follow-up transoesophageal echocardiography after 6 months, which did not show apparent right-to-left shunting.LA, left atrium; TEE, transoesophageal echocardiography.

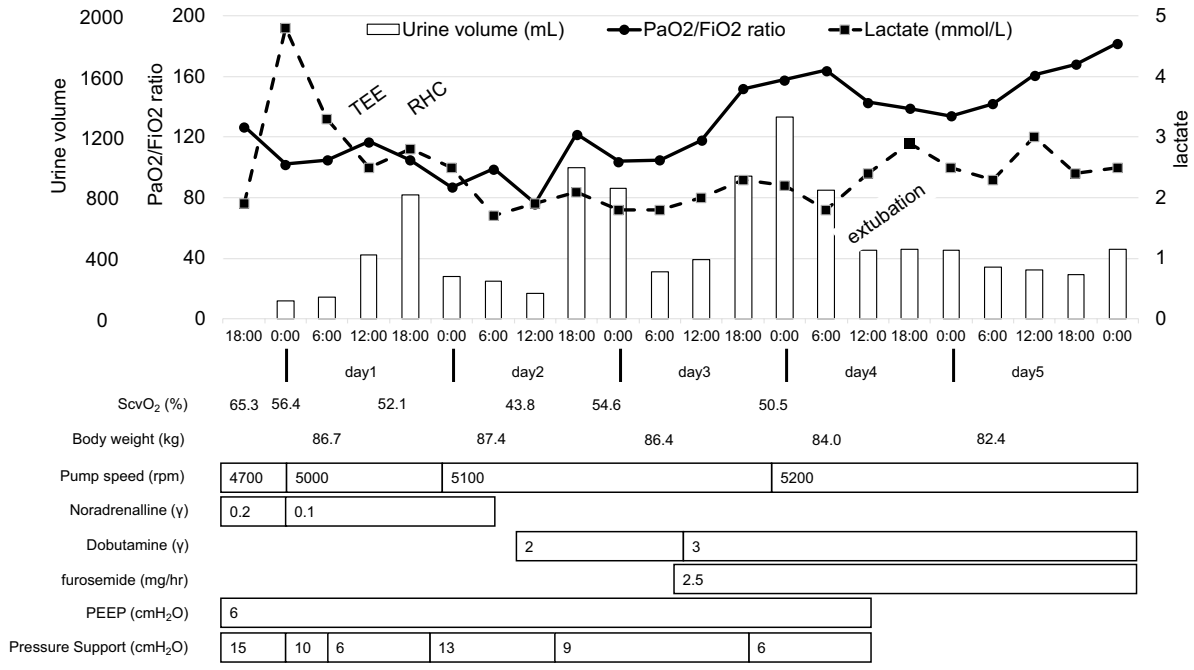

Figure 4 The clinical course. The figure shows the clinical course in the acute phase after ventricular tachycardia ablation. Apparent desaturation was detected immediately after the ablation. On postoperative Day 2, further deterioration of the saturation and systemic circulation developed. Therapeutic interventions, including changes in pump speed and ventilator settings, administration of an intravenous inotrope, and optimized fluid levels using intravenous diuretics, resulted in improved systemic circulation and saturation. FiO_2, fraction of inspiratory oxygen; PaO_2 partial pressure of atrial oxygen; PEEP, positive end-expiratory pressure; RHC, right heart catheterization; $ScvO_2$, central venous oxygen saturation; TEE, transoesophageal echocardiography; VT, ventricular tachycardia.

positive R wave in V1, the clinical VT origin was possibly from LV, which could not be terminated. Right ventricular mapping showed that the VT circuit was rotating around the RV outflow tract. Creating a linear block between the pulmonary and tricuspid valves thus terminated the VT (*Figure 1C*).

After removing the catheter sheath from the left atrium (LA), SpO_2 decreased to 86% under mechanical ventilating support with an 80% fraction of inhalation oxygen (FiO_2). Arterial blood gas analysis revealed partial pressure of atrial oxygen (PaO_2) was 60.2 mmHg, and the PaO_2/FiO_2 (*P/F*) ratio was 75 upon 80% oxygenation. Transoesophageal echocardiography revealed significant right-to-left shunting across the residual iASD (*Figure 3A*). *Figure 4* shows the clinical course in the intensive care unit after VT ablation. The pump speed was lowered from 5400 to 4700 r.p.m. to prevent exacerbation of right-to-left shunt due to decreased LA pressure. The mean arterial blood pressure (mABP) was maintained above 70–80 mmHg to prevent LV collapse due to a relatively high afterload. Additionally, positive end-expiratory pressure (PEEP) and inspiratory pressure support were kept in a minimal setting to maintain low intrathoracic pressure. However, the blood gas lactate level rose to 4.8 mmol/L, probably due to inadequate systemic circulation; hence, the pump speed had to be increased to 5000 r.p.m. However, after increasing the pump speed, *P/F* ratio and central venous oxygen saturation ($ScvO_2$) decreased paradoxically (*P/F* ratio, 105.5; $ScvO_2$, 52.1%). On postoperative day (POD) 1, sedation for TEE resulted in respiratory depression and low mABP, necessitating an increase in inspiratory pressure to 13 cm H_2O and pump speed of 5100 r.p.m. The RHC showed further RV function deterioration (*Table 1*). The ratio of right-to-left shunting was 10%, and the quantity of pulmonary blood flow/quantity of systemic blood flow ratio (Q_p/Q_s) was 0.82. Intravenous inotrope dobutamine and diuretics were administered to support RV function and optimize fluid status. The patient was extubated on POD 4. The RHC performed at POD 60 showed no right-to-left shunting and improved RV dysfunction (*Table 1*). The patient was discharged at POD 61 and had no persistent VT that could cause haemodynamic instability. Follow-up TEE at 7 months after CA showed no obvious right-to-left shunt (*Figure 3B*), and fast VT with haemodynamic instability did not recur after CA. Heart transplantation was performed 1.2 years after CA. Iatrogenic ASD was evaluated during transplantation and found to be closed.

Discussion

A systematic review has summarized some procedural complications of VT ablation in patients with LVAD.[6] Although PFO reportedly enhances right-to-left shunting-induced hypoxia among patients with LVAD,[7] hypoxia following right-to-left shunting from iASD, after the transseptal puncture, has never been reported. The predicted mechanism of hypoxia following right-to-left shunting in the present patient is as follows (*Figure 5*).[7–9] Firstly, LV unloading by LVAD reduces the LA pressure. Secondly, positive pressure ventilation reduces pulmonary vascular compliance and increases intrathoracic pressures, leading to elevated RV afterload and right

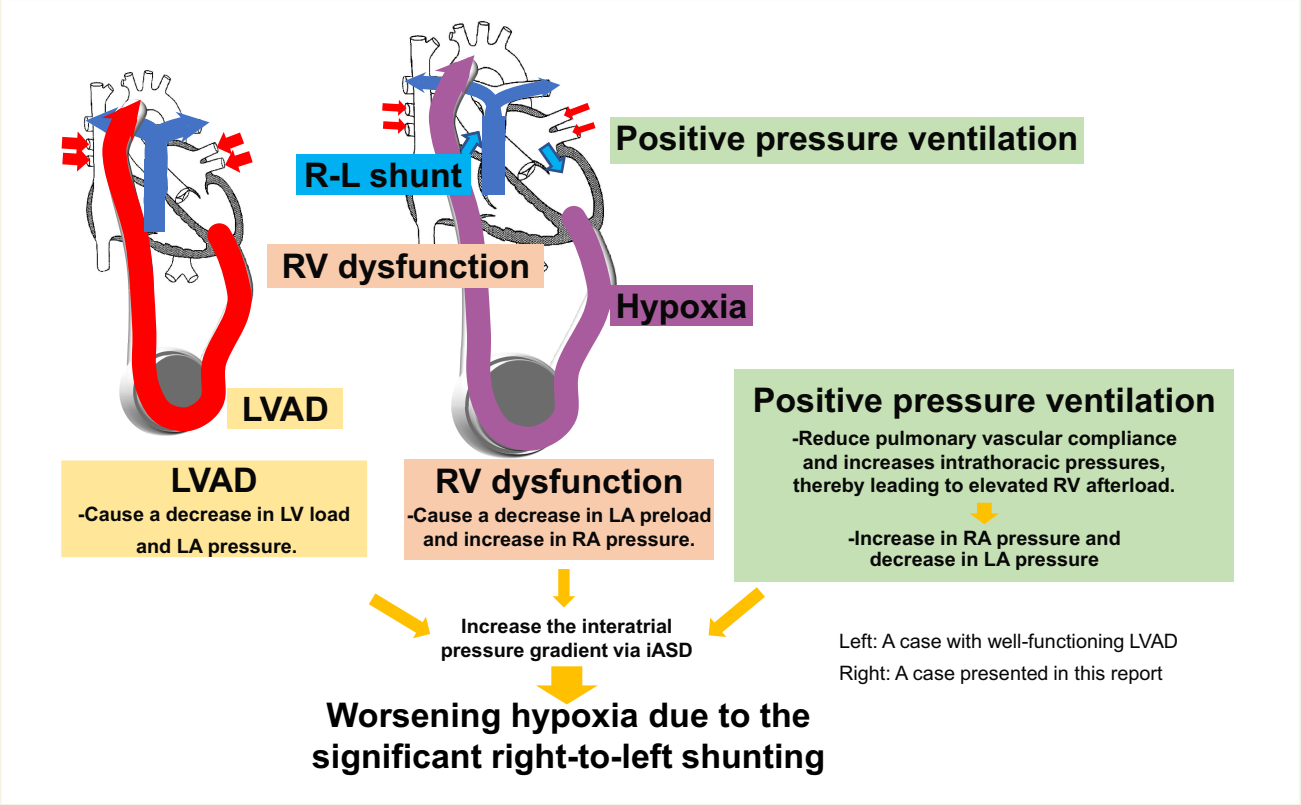

Figure 5 Predicted haemodynamic change. iASD, iatrogenic atrial septal defect; LA, left atrium; LVAD, left-ventricular assist device; RA, right atrium; R-L shunt, right-to-left shunt; RV, right ventricle.

atrial pressure, resulting in an additional decrease of LA pressure. A pre-existing RV dysfunction might have caused a further decrease in LA pre-load and increase in right atrium pressure, and a subsequent increase the interatrial pressure gradient via iASD, which might have resulted in severe hypoxia owing to the significant right-to-left shunting. Therefore, RV function should be evaluated before transseptal puncture to estimate hypoxia risk. In the present patient, the pump speed was set as low as possible to reduce LV/LA unloading, the PEEP/inspiratory pressure was kept to a minimum to decrease intrathoracic pressure, and the body fluid level was adjusted, which resulted in improved hypoxia and weaning off from the mechanical ventilator. Reportedly, iASD after ablation is small enough to close spontaneously in patients with LVAD[6] and no-LVAD.[10] In patients with LVAD, who have significant right-to-left shunting during the early postoperative period, spontaneous closure of iASD may be expected by optimizing the interatrial pressure gradient.

Implantable LVAD has been indicated for patients with end-stage heart failure, who are candidates for heart transplantation (bridge to transplantation) or who need but are ineligible for heart transplantation (destination therapy). Due to the global shortage of donors, the support period for implantable LVAD support has been prolonged. Donor shortage is particularly serious in Japan, where the waiting period for heart transplantation may be ≥5 years. Since prolonged LVAD support may lead to increased complications such as right heart failure and refractory arrhythmias, the present case may provide valuable insight to determine a strategy for patients requiring long-term implantable LVAD support.

Conclusions

In conclusion, the present case suggests that pre-existing impaired RV function may be responsible for the development of right-to-left shunting post-CA, and implies that RV function should be evaluated when planning CA via the transseptal puncture in patients with LVAD.

Lead author biography

Shotaro Komeyama graduated with an MD degree in 2014 from the Gifu University School of Medicine, Japan. He has been working as a cardiovascular fellow at the Department of Transplant Medicine, National Cerebral and Cardiovascular Center, Japan, since 2020. His principal field of interest is heart transplantation, mechanical circulatory support, and ventricular assist devices.

Supplementary material

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

Acknowledgements

The authors thank Kengo Kusano, MD, PhD, for determining the therapeutic strategy for ventricular arrhythmias, and also Yasumasa Tsukamoto, MD, PhD, for his valuable advice for demonstrating the haemodynamic of patients.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The patient provided written informed consent for the anonymized publication of his findings in line with COPE guidelines.

Conflict of interest: None declared.

Funding: This work was supported by a Japan Heart Foundation Research Grant and by the JSPS KAKENHI (grant number JP19K09256) to T.W.

Data availability

Although the data evaluated in this report are not available in a public repository, data will be made available to other researchers upon reasonable request.

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