

Impella as a bridge-to-closure in post-infarction ventricular septal defect: a case series

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Background

Post-infarction ventricular septal defect (PIVSD) is a rare, life-threatening complication of acute myocardial infarction (AMI). Few studies report the use of mechanical circulatory support (MCS) for the treatment of cardiogenic shock in this setting. We describe our experience using a microaxial, transvalvular device (Impella, Abiomed, Danvers, MA, USA) as a bridge-to-closure for PIVSD.

Case summary

We identified 13 patients from two centres with cardiogenic shock due to PIVSD who received an Impella device between January 2016 and February 2022. Nine patients were transferred from another hospital, three with MCS devices [two intra-aortic balloon pumps (IABP), 1 Impella CP]. Eight patients received Impella 5.0, three received Impella 5.5 (one escalated from Impella CP), and two received Impella CP. The median time from AMI to Impella insertion was 5 (3–6) days. Five patients died on Impella support without an attempt to close the ventricular septum (VSD). Seven patients underwent successful VSD closure: six had surgical and one had percutaneous closure. One patient died during attempted percutaneous closure. Time from Impella insertion to VSD closure was 10.5 (7.8–14.0) days. Time from AMI to Impella was 5.0 (2.0–5.3) days in the group that survived to closure, and 6.0 (4.0–7.0) days in those who did not. Thirty-day mortality was 46%.

Discussion

Support with Impella improved clinical stability in most patients, yet multi-system organ failure leading to death occurred in many patients. Patients who survived closure had earlier time from AMI to Impella, underscoring that prompt recognition of PIVSD and initiation of MCS may improve survival to surgical or percutaneous closure.

Keywords

Ventricular septal defect • Cardiogenic shock • Temporary mechanical circulatory support • Acute myocardial infarction • Case series • Ventricular septal rupture

ESC curriculum

3.2 Acute coronary syndrome • 6.4 Acute heart failure • 7.2 Post-cardiac arrest • 7.3 Critically ill cardiac patient

Learning points

- Post-infarction ventricular septal defect (PIVSD) is a rare but highly lethal complication of myocardial infarction.
- Haemodynamic support with a micro-axial transvalvular device placed via the axillary artery may stabilize patients until definitive percutaneous or surgical closure of the PIVSD.
- Prompt recognition of VSD and initiation of MCS may improve survival to surgical or percutaneous closure.

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Introduction

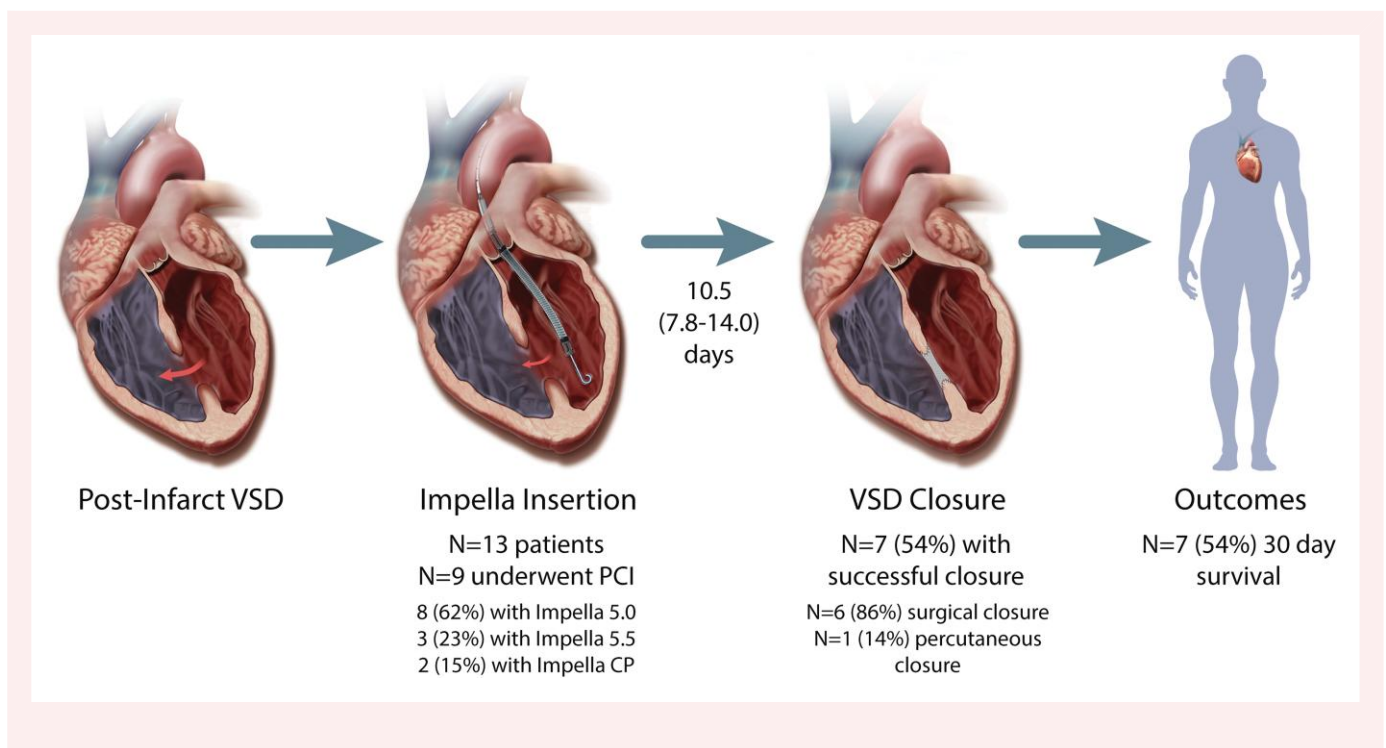
Rupture of the ventricular septum (VSD) is a rare, life-threatening complication of acute myocardial infarction (AMI). In the Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries (GUSTO) registry, 0.2% of the patients had post-infarction VSD (PIVSD), and mortality without surgical repair was 94%.¹ In the contemporary era, post-infarction mechanical complications remain uncommon, but PIVSD is the most frequent with an overall prevalence of 0.21% amongst patients with ST-segment elevation myocardial infarction (STEMI).² Despite advances in the management of AMI, in-hospital mortality remains high at 71% in a contemporary French registry.³ Delaying closure of the PIVSD to allow for scarring of the infarcted tissue is associated with improved surgical outcomes, yet there is no consensus on the optimal timing of surgical or percutaneous closure.^{4,5}

theoretically appealing strategy to support patients with PIVSD until definitive closure.

The low-frequency and high-mortality of PIVSD undermine attempts to collect large case numbers, and the literature reporting Impella use in this setting is limited. Here we describe a contemporary experience from two cardiac centres using Impella as bridge-to-closure, both surgical and percutaneous, in patients with PIVSD.

Summary figure

Sequence of events for post-infarct ventricular septal defect patients included in the study. PCI, percutaneous coronary intervention; VSD, ventricular septal defect. Data presented as median (interquartile range) or *n* (%).



Little data is available to guide the management of patients with PIVSD who are awaiting VSD repair, particularly those with cardiogenic shock (50–60% in American and Japanese surgical registry data, respectively).^{5,6} Left-to-right shunting from PIVSD has been found in computer modelling to decrease cardiac output and increase left ventricle (LV) diastolic volume, pulmonary flow, and pulmonary capillary wedge pressure.⁷ The aim of both medical and device management is to reduce left-to-right shunting and increase forward flow. Historically, the intra-aortic balloon pump (IABP) has been used to support patients as a bridge-to-closure. Advances in mechanical circulatory support (MCS) devices have renewed interest in alternative strategies to the initial management of patients with PIVSD. Computational modelling predicts that microaxial transvalvular devices will decrease pulmonary capillary wedge pressure and flow across the VSD while increasing cardiac output and mean arterial pressure.⁷ In addition to its predicted haemodynamic effects, placement of an Impella 5.0/5.5 (Abiomed, Danvers, MA, USA) in the axillary artery allows for early patient mobilization with few adverse events.⁸ Use of an axillary Impella is thus a

Methods

Study design, setting, and patient population

The electronic medical record was used to identify patients hospitalized at Providence St. Vincent Medical Center and Peace Health Sacred Heart Medical Center between January 2016 and February 2022. All patients with PIVSD who received an Impella device were included in the study. This study was approved by the institutional review boards at both centres, with a waiver of informed consent.

Device information and management

Impella devices are micro-axial flow pumps placed retrograde across the aortic valve into the LV and aspirate blood from the LV into the ascending aorta. Two types of Impella microaxial flow pumps (Abiomed, Danvers, MA, USA) were used in this study. The Impella 5.0 has a pigtail and uses steel bearings, whereas the 5.5 has ceramic bearings, no pigtail, and a shorter motor.

Both devices are inserted over a wire into the LV through a vascular graft (8–10 French) anastomosed to the axillary artery. After adequate surgical

Table 1 Characteristics of study patients

Age/sex	EF, %	Infarct location	PCI	VSD location	VSD Size, cm	Mechanical support devices	Survived to closure
70/M	70	RCA	Yes	Inferoseptal	1.0	Impella 5.0	Y
63/F	25	LAD	Yes	Inferoseptal	1.0	Impella 5.5	Y
66/F	25	LAD	No	Apical	1.0	Impella 5.0	Y
73/M	65	RCA	Yes	Inferoseptal	2.4	Impella CP → Impella 5.5 + RVAD	Y
57/M	30	LAD	Yes	Apical	2.0	Impella 5.5	Y
72/M	45	LAD	No	Anterior	2.0	Impella 5.0	Y
68/M	30	LAD	No	Anterior	2.0	Impella 5.0	Y
63/M	40	LAD	Yes	Anterior	3.0	Impella 5.0	Y
72/F	65	RCA	Yes	Basal Inferoseptal	N/A	Impella 5.0	N
78/M	60	RCA	No	Basal Inferoseptal	2.15	IABP → Impella 5.0	N
66/M	75	RCA	Yes	Inferoseptal	N/A	Impella 5.0	N
72/M	45	LAD	Yes	Apical	0.9	IABP-Impella CP	N
74/M	25	LAD	Yes	Inferoseptal	N/A	Impella CP	N

EF, ejection fraction; F, female; IABP, intra-aortic balloon pump; LAD, left anterior descending artery; M, male; PCI, percutaneous coronary intervention; RCA, right coronary artery; RVAD, right ventricular assist device; VSD, ventricular septal defect.

hemostasis, unfractionated heparin was administered per hospital protocol. Devices were generally run at the higher ranges of *P*-values (*P* 7–9) to provide maximal reduction in LV pressure and volume. Echocardiography was performed as clinically indicated to ensure pump position and to assess cardiac function. One patient was implanted with a ProtekDuo right ventricular assist device (RVAD) (Liva Nova, London). The ProtekDuo is a dual-lumen cannula inserted via the jugular vein, with the proximal inflow positioned in the right atrium and the distal lumen in the main pulmonary artery. The cannula lumens are attached to the TandemHeart (Liva Nova, London) pump, providing up to 5 L/m of flow.

Data collection and analysis

Data was collected from the electronic medical record, including type and sequence of MCS devices, time from myocardial infarction (MI) to VSD diagnosis, and laboratory and haemodynamic data. Outcome data included bleeding, renal replacement therapy, limb ischaemia, haemolysis, stroke, access site complications, arrhythmia, and survival at 30 days.

Descriptive statistics are presented as median and interquartile range or counts and percentages. Between-group comparisons to test for statistical significance were not performed due to the small sample size and perceived marginal utility of such an analysis.

Results

Of the thirteen patients identified, three were female, and the average age was 70 (66–72) years (Table 1). Nine patients were transferred from another hospital, three with MCS devices (two IABP, one Impella CP). Eight patients received Impella 5.0, three received Impella 5.5 and two patients received an Impella CP (Central Figure). One Impella 5.5 patient with the right coronary artery (RCA) infarct was escalated from CP, with simultaneous placement of a Protek Duo RVAD. Five patients presented with RCA infarction and eight with left anterior descending artery (LAD) infarction, with nine patients receiving percutaneous coronary intervention (PCI) of the culprit artery at the time of presentation. Ten out of thirteen patients had maximal Society for Cardiovascular Angiography and Interventions (SCAI) shock stage D. Of the 10 patients with VSD size measurements available in their charts, the median size was 2.0 cm and ranged from 0.9 to 3.0 cm.

The median time from AMI to VSD diagnosis was 4 (3–5) days and from AMI to Impella insertion was 5 (3–6) days. The average duration

of mechanical support was 12 (10–14) days and initial MCS insertion to VSD closure was 10.5 (7.8–14.0) days. One patient died during attempted percutaneous closure, with seven patients receiving surgical (6) or percutaneous closure (1) and surviving to thirty days. Time of Impella support was 5.0 (7.8–12.0) days in patients who received Impella CP and 12.0 (11.0–14.0) days in those who received Impella 5.0/5.5.

Characteristics of patients who survived closure (*n* = 8) vs. those who did not (*n* = 5) are shown in Table 2. Survivors were younger (67 vs. 72 years old) and more often male (*n* = 6). Six of the eight patients (75%) who survived to closure presented with LAD infarcts. Survivors had shorter time from AMI to VSD diagnosis (3.5 vs. 5.0 days), shorter time from AMI to initial MCS (5.0 vs. 6.0 days), and longer total Impella time (14.0 vs. 7.0 days).

Thirty-day mortality in this cohort was 46% (*n* = 7), with non-survivors developing multi-system organ failure leading to withdrawal of care. Only one patient received renal replacement therapy concomitant with Impella and did not experience renal recovery. Importantly, no neurological complications were observed during Impella support. Serious access site complications were rare and included one access site haematoma and one arterial thrombotic occlusion incidentally noted and treated by right subclavian thrombectomy at the time of Impella 5.0 removal. Another patient required surgical embolectomy for non-access site-related arterial thrombosis. However, all three patients survived and were discharged home after successful VSD closure. Two patients required transfusion for non-surgical site bleeding, with one patient requiring surgical intervention for oropharyngeal bleeding from intubation. There was no clinical evidence of intravascular haemolysis in any patient nor in the patients (*N* = 9) who had routine monitoring of serum lactate dehydrogenase.

Discussion

In this case series from two centres, we report the feasibility of using transvalvular, microaxial flow devices (Impella) to support patients with PIVSD until percutaneous or surgical VSD closure. The median MCS support time on Impella was nearly two weeks, facilitated by the insertion of the device into the axillary artery in the majority of patients. Most of the patients were transferred from a level one shock centre. We observed a trend for earlier diagnosis and MCS support

Table 2 Characteristics of patients who survived to closure vs. non-survivors

Variable	Survivors (N = 8) ^a	Non-Survivors (N = 5)
Age, years	67 (63–70.5)	72 (72–74)
Sex, male	6 (75%)	4 (80%)
EF, %	35 (29–50)	60 (45–65)
Peak lactate, mmol/L	3.2 (2.8–4.4)	4.5 (2.4–8.1)
Interval from AMI to VSD diagnosis, days	3.5 (1.8–4.0)	5.0 (4.0–7.0)
Interval from AMI to initial MCS insertion, days	5.0 (2.0–5.3)	6.0 (4.0–7.0)
Interval from initial MCS insertion to VSD closure, days	10.5 (7.8–14.0)	N/A
Total Impella time, days	14.0 (12.0–14.5)	7.0 (5.0–10.0)

Data presented as median (interquartile range) or *n* (%).

AMI, acute myocardial infarction; EF, ejection fraction; MCS, mechanical circulatory support; VSD, ventricular septal defect.

^aSurvivors defined as patients who survived attempted VSD closure.

in patients with PIVSD who survived closure. Of patients who underwent successful closure, all survived for 30-days.

Ventricular septal defects are a rare complication of AMI associated with high morbidity and mortality, even in the contemporary era. An increased prevalence of PIVSD was observed during the COVID-19 pandemic due to delays in the presentation and treatment of AMI.^{9–11}

Ideally, percutaneous or surgical closure of the PIVSD should be delayed to allow for fibrosis of the newly infarcted tissue.^{12,13} Though there are no randomized trial data supporting the optimal procedural timing or method of closure, a contemporary registry suggests that in-hospital mortality is lower with surgical PIVSD repair compared to percutaneous closure.¹⁴ In addition, based on US registry data, a delay of at least 7 days is associated with lower surgical mortality.¹⁵ Patients who develop cardiogenic shock while awaiting closure are often managed with inotropes and IABP, though there is no consensus on the optimal choice of mechanical support for patients who fail this strategy.¹²

Direct mechanical unloading of the LV with a microaxial, transvalvular pump simultaneously reduces left ventricular wall stress and myocardial oxygen consumption while increasing cardiac output and aortic pressure. Computational simulation of VSD physiology with Impella further predicts decreased flow through the VSD.⁷ Early concerns about the use of microaxial pumps in the setting of PIVSD included the possibility of ingestion of necrotic material leading to pump failure and neurologic events, as well as the possibility of causing right-to-left shunting.¹⁶ Notably, we did not observe echocardiographic or clinical evidence of right-to-left shunting in our cohort. Further, there were no significant access site complications or MCS-related major bleeding in our cohort, likely related to the use of surgically implanted Impella 5.0 or 5.5 devices. Finally, the use of upper extremity cannulation facilitates mobilization of the patient, which can reduce the risk of complications during longer durations of MCS support.¹⁷

In our cohort of patients with PIVSD, Impella improved clinical stability, yet 30-day mortality remained high (47%), underscoring the importance of early recognition, monitoring, and treatment to reduce progression to later stages of cardiogenic shock. In this series, we observed that survivors were younger and had shorter time from AMI to VSD and time from AMI to MCS. Additionally, survivors had a lower ejection fraction (35% vs. 60%) before MCS initiation than non-survivors, though the interpretation of EF is confounded by varying

degrees of VSD shunt flow and inotropic support. Non-survivors developed multi-system organ failure (*N* = 4) or other complications of prolonged hospitalization and cardiogenic shock, such as right ventricular failure (*N* = 1).

Limitations

There are limitations to this study, most notably the small sample size which precludes statistical hypothesis testing and robust conclusions. Though we present the largest case series reported in the literature to date, PIVSD remains a rare condition, making it difficult to gather data. For example, there was a lack of complete haemodynamic data (pre- and post-MCS) recorded in the medical record for most patients in our cohort. There is also the possibility of significant selection and survivor bias in this population, as is evident in most cardiogenic shock literature. Outcomes from observational, retrospective data for a high-mortality condition such as post-infarction VSD, including our study, may be limited due to confounding from patient selection and the natural history of the disease.

Conclusion

In patients with PIVSD, Impella is a safe and feasible bridge-to-closure strategy, but morbidity and mortality remain high. Early identification of patients, use of MCS, and agreed-upon treatment algorithms could increase survival for this rare, highly vulnerable patient population.

Lead author biography



Dr. Abraham is a cardiologist with the Providence Heart Institute, and the division chief of Advanced Heart Failure. He earned his undergraduate degree at Rhodes College and after completing a Rotary International Scholarship at the University of New South Wales (Sydney, Australia), he completed his medical degree, residency, and fellowship in cardiovascular medicine at Johns Hopkins. His research interests include advanced heart failure, cardiogenic shock, temporary mechanical circulatory support, and remote haemodynamic monitoring.

Dr. Abraham is a member of the Cardiogenic Shock Working Group and co-founder of Hemodynamic Frontiers of Heart Failure (HF2). He is an active investigator in clinical trials for all stages of heart failure.

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Consent: The institutional review boards at both institutions approved a waiver of informed consent and waiver of HIPAA authorization under expedited review for this minimal risk, retrospective research project, because the following criteria were met: (i) (a) An adequate plan to protect the identifiers from improper use and disclosure; (b) An adequate plan to destroy the identifiers at the earliest opportunity consistent with conduct of the research, unless there is a health or research justification for retaining the identifiers or such retention is otherwise required by law; and (c) Adequate written assurances that the protected health information will not be reused or disclosed to any other person or entity, except as required by law,

for authorized oversight of the research study, or for other research for which the use or disclosure of protected health information would be permitted by this subpart; (ii) The research could not practicably be conducted without the waiver or alteration; and (iii) The research could not practicably be conducted without access to and use of the protected health information. Furthermore, patients had already received care as part of the standard of care for their medical condition, the study consisted only of a review of medical record data and involved no new interventions or interactions with patients, and patients reviewed may no longer be seen at the care facilities involved in this study and/or may have been lost to follow up. There were no direct risks or benefits to patients involved in the research, and protections are in place to ensure confidentiality of patient information is maintained.

Conflict of interest: S.J., K.S., E.K., A.V.: None. J.A.: Speaker's bureau, Abbott, Abiomed; Consultant—Abbott, Abiomed; Steering committee—Abbott, Abiomed.

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Data availability

To protect the privacy of patients in this study, the data for this project cannot be shared publicly due to the presence of patient-identifying information.

References

- Crenshaw BS, Granger CB, Birnbaum Y, Pieper KS, Morris DC, Kleiman NS, et al. Risk factors, angiographic patterns, and outcomes in patients with ventricular septal defect complicating acute myocardial infarction. *Circulation* 2000;**101**:27–32.
- Elbadawi A, Elgendy IY, Mahmoud K, Barakat AF, Mentias A, Mohamed AH, et al. Temporal trends and outcomes of mechanical complications in patients with acute myocardial infarction. *JACC Cardiovasc Interv* 2019;**12**:1825–1836.
- de Grandsaigne HT, Bouisset F, Porterie J, Biendel C, Marcheix B, Lairez O, et al. Incidence, management, and prognosis of post-ischaemic ventricular septal defect: insights from a 12-year tertiary centre experience. *Front Cardiovasc Med* 2022;**9**:1066308.
- Honey M, Belcher JR, Hasan M, Gibbons JR. Successful early repair of acquired ventricular septal defect after myocardial infarction. *Br Heart J* 1967;**29**:453–456.
- Arnautakis GJ, Zhao Y, George TJ, Sciortino CM, McCarthy PM, Conte JV. Surgical repair of ventricular septal defect after myocardial infarction: outcomes from the society of thoracic surgeons national database. *Ann Thorac Surg* 2012;**94**:436–444.
- Sakaguchi G, Miyata H, Motomura N, Ueki C, Fukuchi E, Yamamoto H, et al. Surgical repair of post-infarction ventricular septal defect—findings from a Japanese national database. *Circ J* 2019;**83**:2229–2235.
- Pahuja M, Schrage B, Westermann D, Basir MB, Garan AR, Burkhoff D. Hemodynamic effects of mechanical circulatory support devices in ventricular septal defect. *Circ Heart Fail* 2019;**12**:e005981.
- Abraham J, Wang L, Kumar V, Kirker EB, Spinelli KJ. Axillary transvalvular microaxial pump as extended bridge to transcatheter aortic valve replacement in cardiogenic shock with severe aortic stenosis. *J Heart Lung Transplant* 2022;**41**:434–437.
- Abraham MD*, Saltarocchi S, Vignaroli W, Chourda E, Vinciguerra M, Romiti S, et al. Increased incidence of postmyocardial infarction ventricular septal defects during Covid-19 pandemic: a case series. *J Card Surg* 2022;**37**:1759–1763.
- Aldujeli A, Hamadeh A, Briedis K, Tecson KM, Rutland J, Krivickas Z, et al. Delays in presentation in patients with acute myocardial infarction during the COVID-19 pandemic. *Cardiol Res* 2020;**11**:386–391.
- Gluckman TJ, Wilson MA, Chiu ST, Penny BW, Chepuri VB, Waggoner JW, et al. Case rates, treatment approaches, and outcomes in acute myocardial infarction during the coronavirus disease 2019 pandemic. *JAMA Cardiol* 2020;**5**:1419–1424.
- Heckle MR, Brooksbank JA, Agarwal MA, Ibebuogu UN. Outcomes of early versus delayed transcatheter closure of post-myocardial infarction ventricular septal defect. *Cardiovasc Revascularization Med Mol Interv* 2020;**21**:1093–1096.
- Dimagli A, Guida G, Sinha S, Dixon L, Fudulu D, Gemelli M, et al. Surgical outcomes of post-infarct ventricular septal defect repair: insights from the UK national adult cardiac surgery audit database. *J Card Surg* 2022;**37**:843–852.
- Giblett JP, Matetic A, Jenkins D, Ng CY, Venuraju S, MacCarthy T, et al. Post-infarction ventricular septal defect: percutaneous or surgical management in the UK national registry. *Eur Heart J* 2022;**43**:5020–5032.
- Jones BM, Kapadia SR, Smedira NG, Robich M, Tuzcu EM, Menon V, et al. Ventricular septal rupture complicating acute myocardial infarction: a contemporary review. *Eur Heart J* 2014;**35**:2060–2068.
- Meyns B, Vanermen H, Vanhaecke J, Sergeant P, Daenen W, Flameng W. Hemopump fails as bridge to transplantation in postinfarction ventricular septal defect. *J Heart Lung Transplant* 1994;**13**:1133–1137.
- Zaky M, Nordan T, Kapur NK, Vest AR, DeNofrio D, Chen FY, et al. Impella 5.5 support beyond 50 days as bridge to heart transplant in end-stage heart failure patients. *ASAIO J* 2023;**69**:e158–e162.