





# Left atrial veno-arterial extracorporeal membrane oxygenation in post-myocardial infarction ventricular septal defect with cardiogenic shock: a case report

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## Background

Ventricular septal defect (VSD) complicating acute myocardial infarction (MI) represents a life-threatening condition and has a mortality of >90% if left untreated.

## Case summary

A 53-year-old man with a prior medical history of diabetes and hypertension presented with cardiogenic shock secondary to VSD as a mechanical complication of non-reperused inferior MI.

## Discussion

The choice of mechanical support can be difficult in this type of patient. Given the risk of an increased shunt because of veno-arterial extracorporeal membrane oxygenation (VA-ECMO) and the increase in left ventricle (LV) afterload, several measures were taken to plan the best ECMO configuration. Given the absence of any real improvement in the LV and an elevated residual ratio between pulmonary and systemic flow ( $Q_p/Q_s$ ), the final decision was to switch to left atrial VA-ECMO (LAVA-ECMO). The use of LAVA-ECMO improved the patient's haemodynamics and allowed his condition to stabilize; LAVA-ECMO is feasible and may be effective as a mechanical circulatory support (MCS) strategy for patients in cardiogenic shock due to VSD as a mechanical complication of acute MI.

## Keywords

Acute heart failure • Myocardial infarction • Mechanical complication • Ventricular septal defect • Case report

## ESC Curriculum

3.2 Acute coronary syndrome • 6.4 Acute heart failure • 7.1 Haemodynamic instability

## Learning points

- Recognize the limitations and contraindications of different extracorporeal membrane oxygenation (ECMO) configurations.
- Comprehend the advantages of left atrial veno-arterial (LAVA)-ECMO insertion in patients suffering from cardiogenic shock and post-myocardial infarction (MI) ventricular septal defect (VSD).
- Treatment of patients with cardiogenic shock related to MI VSD requires individualized and prompt decision-making.

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## Introduction

Ventricular septal defect (VSD) complicating acute myocardial infarction (MI) represents a life-threatening condition and has a mortality of >90% if left untreated.<sup>1,2</sup>

Veno-arterial extracorporeal membrane oxygenation (VA-ECMO) provides biventricular haemodynamic support in patients with cardiogenic shock. However, VA-ECMO also increases left ventricle (LV) afterload, and in a patient with VSD, it may increase the shunt flow and subsequently the filling pressures. In such patients, the use of VA-ECMO represents an independent risk factor for excess mortality.<sup>3</sup>

The combination of an Impella® microaxial flow catheter (Abiomed, Danvers, MA, USA) and ECMO (ECPeLLA) may provide a solution to this haemodynamic problem, but it conveys a risk of vascular and bleeding complications. Notably, left atrial VA-ECMO (LAVA-ECMO) is another approach to unloading both ventricles while providing haemodynamic support; LAVA-ECMO is used in patients for whom conventional VA-ECMO is contraindicated.<sup>4</sup>

In this case report, we describe the usefulness of LAVA-ECMO as a mechanical circulatory support (MCS) strategy for patients in cardiogenic shock due to VSD as a mechanical complication of acute MI.

## Summary figure

## Case presentation

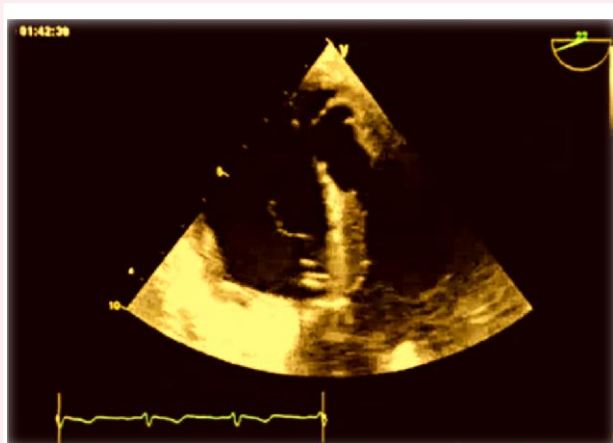
A 53-year-old man presented to a secondary medical institution with non-reperfused inferior MI 36 h after the beginning of chest pain. His systemic blood pressure was 75/35 mmHg. On physical examination, he presented with a 5/6 holosystolic murmur at the fourth intercostal space along the left sternal border. The patient was known to have poorly controlled diabetes and hypertension. He was then transferred to our tertiary-care institution for further management. The electrocardiogram showed Q waves in the inferior leads. His laboratory profile demonstrated elevated concentrations of high-sensitivity troponin I of 2566 ng/dL (normal value < 0.250 ng/mL), lactate 3.1 mmol/L (normal value < 1 mmol/L), creatinine 2.4 mg/dL (normal value < 1.0 mg/dL), and N-terminal pro-hormone brain natriuretic peptide 8618 pg/mL (normal value < 125 pg/mL). A bedside transthoracic echocardiogram (TTE) demonstrated a complex posterobasal VSD involving a torrential shunt from the LV to the right ventricle (RV) with a maximum diameter of 29 mm (Figure 1). A computed tomography (CT) scan further characterized the lesion (Figure 2).

Coronary angiography was performed, showing no significant stenosis in the left coronary system and a total thrombotic occlusion in the mid-segment of the right coronary artery (Figure 3), with an LV end-diastolic pressure of 25 mmHg. Right heart catheterization was also performed and revealed elevated right heart filling pressure [right atrial (RA) pressure of 18 mmHg], pulmonary capillary wedge pressure

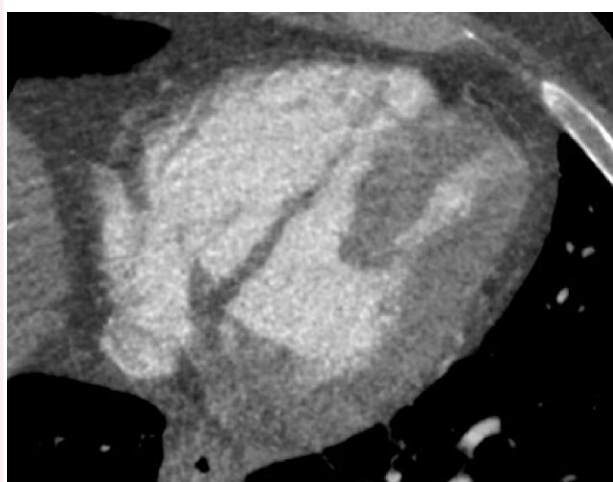
	Clinical progression	Systemic arterial pressure (mmHg)	PCWP (mmHg)	Right atrial pressure (mmHg)	Norepinephrine ( $\mu\text{g}/\text{kg}/\text{min}$ )	Qp/Qs	VTI LVOT (cm)	VSD gradient (mmHg)	ECMO flow (litres)
Day 1	Arrival to our institution in cardiogenic shock (75/35 mmHg). TTE showing a complex posterobasal VSD. IABP insertion, temporal haemodynamic improvement.	90/40 (57) with IABP	28	18	0.2	2.54	17	40	NA
Day 2	Haemodynamic deterioration.	80/40 (53)	32	25	0.4	9.81	11	49	NA
Day 2	VA-ECMO cannulation. Qp/Qs and PCWP increased.	102/77 (85)	26	12	0.01	3.83	7	66	3.2
Day 2	Switch to LAVA-ECMO. Qp/Qs and PCWP decreased.	98/78 (84)	18	10	0.01	2.15	13	37	3.2
Day 3	Haemodynamic improvement.	110/70 (83)	16	10	0	2.1	12	35	3.2
Day 4	Ischaemic stroke. Local thrombus aspiration performed and alteplase (5 mg) administered.	105/78 (87)	16	10	0	2.05	NA	NA	3.2
Day 5	Motor aphasia. Continued with haemodynamic improvement.	110/85 (93)	17	10	0	2.04	NA	NA	2.9
Day 6	Haemorrhagic transformation of the stroke with catastrophic consequences.	105/78 (87)	16	11	0	2.04	NA	NA	2.7

Haemodynamic characteristics with IABP, VA-ECMO, and LAVA-ECMO. Several haemodynamic measures improved after the initiation of LAVA-ECMO, such as significant decreases in the PCWP, Qp/Qs ratio, and VSD gradient. Notice the slight increase in the Qp/Qs ratio and VSD gradient when the classic VA-ECMO configuration was placed.

IABP, intra-aortic balloon pump; TTE, transthoracic echocardiogram; PCWP, pulmonary capillary wedge pressure; Qp/Qs, ratio of the pulmonary cardiac output to the systemic cardiac output; LVOT, left ventricular outflow tract, VTI, velocity-time integral; VSD, ventricular septal defect; VA-ECMO, veno-arterial extracorporeal membrane oxygenation; LAVA-ECMO, left atrial VA-ECMO.



**Figure 1** Transoesophageal echocardiogram. Deep transgastric two-dimensional view of the complex post-infarction posterobasal ventricular septal defect.



**Figure 2** Computed tomography showing a complex posterobasal ventricular septal defect.

(PCWP) of 28 mmHg, and a Qp/Qs ratio of 3.91. An intra-aortic balloon pump (IABP) was inserted to support the LV.

The patient was given norepinephrine (0.2 µg/kg/min), and the IABP was maintained at a ratio of 1:1 for 36 h with no respiratory compromise. Afterwards, the patient started to exhibit obtundation, increased lactate concentration to 4.1 mmol/L, and a Qp/Qs ratio of 9.81. In an emergency Heart Team meeting, it was decided to escalate treatment to VA-ECMO to maintain the best theoretical haemodynamic profile for at least 14 days, after which surgical correction of the VSD would be performed.

Given the risk of an increased shunt because of VA-ECMO and the increase in LV afterload, several measures were taken to plan the best ECMO configuration. The procedure was performed with superficial sedation without general anaesthesia to avoid the risk of further haemodynamic compromise and the need for respiratory support. A left arterial 17 Fr femoral ECMO cannula was inserted in a standard

fashion. Right femoral venous access was obtained. A TTE was used to measure the haemodynamic profile for multiple configurations (Summary figure). A 25 Fr multifenestrated ECMO cannula was placed in the cavoatrial junction, and invasive haemodynamics 20 min later showed a Qp/Qs ratio of 3.83, PCWP of 26 mmHg, and RA pressure of 12 mmHg.

Given the absence of any real improvement in the LV and an elevated residual Qp/Qs ratio, the final decision was to switch to LAVA-ECMO. With intracardiac echocardiogram guidance (AcuNav 8 Fr, Siemens), a transseptal puncture was performed, and a hydrophilic 0.35 mm Terumo Guide Wire passed through a patent foramen ovale (PFO). A multipurpose 6 Fr catheter and a 0.35 mm Amplatz Extra Stiff wire were advanced to the left upper pulmonary vein. A 25 Fr multifenestrated cannula was then sent across the interatrial septum via the PFO without any impediment into the left atrium (LA) (Figure 4). Biatrial and biventricular unloading was possible and involved side holes in both the LA and right cavities. Invasive haemodynamics showed a Qp/Qs ratio of 2.15, PCWP of 18 mmHg, and RA pressure of 10 mmHg; 4 h after the procedure, the patient was weaned from all vasopressor support, giving only furosemide, dexmedetomidine, and anticoagulation with heparin, and maintained without intubation.

The patient showed haemodynamic and renal improvement over the next 48 h and a normalization of creatinine levels (2.4–0.71 mg/dL) and did not require further vasopressor support. A new TTE demonstrated an improvement in LV and RV functions.

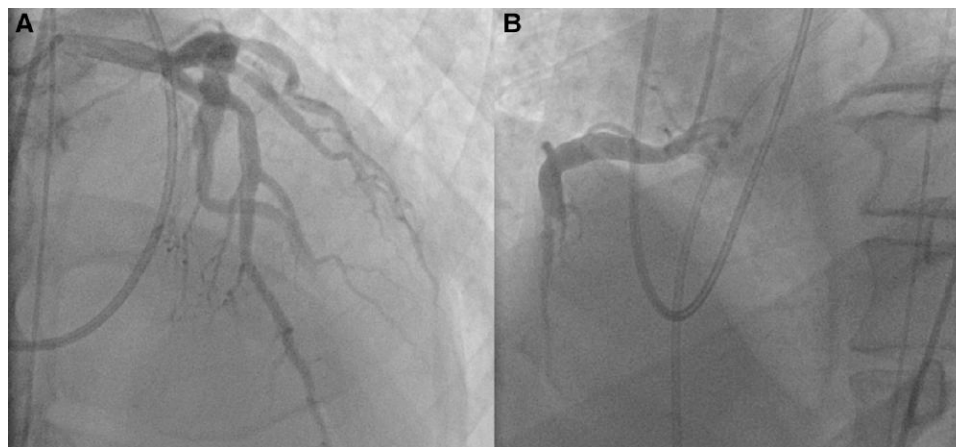
Unfortunately, on Day 4 of LAVA-ECMO, the patient developed an ischaemic stroke identified in the hyperacute phase while physical examination was made; this was characterized by motor aphasia and hemiparesis. Local thrombus aspiration was performed, and local alteplase (5 mg) was administered 90 min after the stroke onset. Nevertheless, the neurological damage resulted in permanent motor aphasia. A transoesophageal echocardiogram (TEE) was performed, showing no evidence of thrombus formation in the LAVA-ECMO cannula and the left atrial appendage. On Day 6, the patient experienced haemorrhagic transformation of the stroke with catastrophic consequences. At this point, it was decided to stop the circulatory support given the impossibility of stopping the anticoagulation and the severity of the haemorrhage. The patient passed away on Day 6 of the hospitalization.

## Discussion

Here, we have demonstrated the feasibility and possible benefit of LAVA-ECMO in a patient with cardiogenic shock with post-MI VSD, and after a review of the literature, this is the first case report using this ECMO configuration for this pathology.

There is no standard MCS configuration for patients presenting with cardiogenic shock and acute post-MI VSD; VA-ECMO can be used to support both ventricles, but its use increases the shunt flow through VSD by increasing the afterload to the LV. The use of two univentricular devices (such as Impella) can be considered, but its use for this purpose remains off-label. In cases involving conventional VA-ECMO, it is important to consider EPELLA or the recent LAVA-ECMO configuration, which provides a viable percutaneous option to unload both ventricles without the need for additional vascular access but having the necessity to perform a transseptal puncture in order to place the drainage cannula through the interatrial septum. Some forms of MCS are not readily available in every emergency setting and in many developing countries due to their high cost, which is the case with Impella.

The LAVA-ECMO was first described in 2018 for three patients with biventricular cardiogenic shock.<sup>5</sup> Its use has been reported more recently in a case involving cardiogenic shock and acute aortic valve regurgitation.<sup>4</sup> In our patient, after a multidisciplinary team consultation, it was decided to support the patient with VA-ECMO in order to have



**Figure 3** Coronary angiography. (A) Left coronary system without significant stenosis. (B) Right coronary artery with thrombotic occlusion in the middle segment.



**Figure 4** Chest X-ray of the left atrial veno-arterial extracorporeal membrane oxygenation cannula at the end of the procedure.

the chance to perform a surgical repair with a better haemodynamic profile, considering also that the myocardial tissue was not optimal for surgery. At the beginning, the conventional VA-ECMO increased the shunt, so we decided to move to LAVA-ECMO; then, we documented rapid improvement in invasively measured haemodynamics after cannulation and noticed how the shunt flow was decreased with this configuration as compared with conventional VA-ECMO; the property of LAVA-ECMO to unload both ventricles while giving complete haemodynamic support let us decrease the shunt as seen in the Summary figure.

The decision to go with LAVA-ECMO instead of ECPPELLA was made because both therapeutic options have not been described for this kind of case, and to our consideration, the risk of vascular and other complications would increase using ECPPELLA compared with LAVA-ECMO.

The placement of LAVA-ECMO is not technically challenging in centres that perform transeptal procedures and can be applied through a PFO without any further septostomy, as proved in this case; most of the time, the PFO is located in the anterosuperior portion of the interatrial septum but has many variants,<sup>6</sup> and the size and stiffness of the ECMO cannula enabled it to be located and maintained in a proper position regardless of the PFO anatomy; LAVA-ECMO is also cheaper and does not need any further device or surgical strategy to unload both ventricles.

It is important to have close monitoring of the drainage cannula with an echocardiogram confirming no evidence of thrombus formation and checking that the left atrial appendage is clean, paying special attention to optimal anticoagulation parameters.

Posterior VSD cases are challenging due to their adverse and complex morphology (usually large and with serpentine intramyocardial tissue). Furthermore, their dissection paths may reach the free wall of the LV, the RV function is usually worse, and they are associated with a worse prognosis. A high index of suspicion should be maintained and prompt an initial echocardiogram. Additionally, cardiac CT with 3D imaging can provide a more complete anatomical assessment, favouring better pre-procedural planning.<sup>7</sup>

Even with the unfortunate death of the patient, the use of LAVA-ECMO in this pathology resulted in an important clinical improvement by allowing the withdrawal of vasopressors and avoiding intubation of the patient. The mere fact of suffering from this serious mechanical complication exposes the patient to the risk of sudden death, cerebral vascular events, and other vascular complications. The strategy of maintaining circulatory support in this type of patient allows the surgeon to perform his intervention in a better haemodynamic context, and the tissue to be operated on is in better condition for surgical closure. The daily review of this type of case and detailed monitoring of the haemodynamic status and the LAVA-ECMO drainage cannula as well as the left atrial appendage, in addition to close contact with the Heart Team, would allow these patients to undergo surgery in the best possible haemodynamic state.

The optimal management of post-MI VSD is a subject of debate. While surgery is the recommended approach, percutaneous treatment may also be appropriate in certain patients.<sup>3</sup> Surgical repair carries a high risk of death, particularly in patients experiencing cardiogenic shock, which is the only scenario where early surgical intervention is preferable to delayed surgery. On the other hand, a delay of >2 weeks

in surgical repair gives the necrotic tissue time to mature, making the procedure easier and reducing its mortality.<sup>7</sup> Both interventional therapy and surgical therapy improve the survival of post-MI VSD, and while surgical repair decreases hospital mortality, it also increases incident stroke, pneumonia, and renal replacement therapy.<sup>7</sup>

## Conclusion

The LAVA-ECMO is feasible and may be effective as an MCS strategy for patients in cardiogenic shock due to VSD as a mechanical complication of acute MI.

## Lead author biography



Gian-Manuel Jiménez-Rodríguez, MD, MSc, graduated from the National Autonomous University of Mexico. He completed his training in Cardiology, Intensive Cardiovascular Care, Interventional Cardiology, and Congenital Heart Diseases at the National Institute of Cardiology ‘Ignacio Chavez’, Mexico. After his interventional cardiology training in Mexico, he spent 1 year of structural heart fellowship at the University Medical Centre Utrecht, The Netherlands. He is currently working as an interventional cardiologist at the National Institute of Cardiology ‘Ignacio Chavez’.

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**Consent:** The patient reported in this case is deceased. 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’s relative in line with COPE guidance.

**Conflict of interest:** None declared.

**Funding:** None declared.

## Data availability

The data underlying this article will be shared on reasonable request to the corresponding author.

**Declaration:** All the authors mentioned above have corroborated their authorization to the corresponding author to be responsible for the upcoming comments and possible modifications to this manuscript. The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors’ institution and patient consent where appropriate.

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