

Free floating thrombus in the ascending aorta after extracorporeal cardiopulmonary resuscitation: a case series

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Background

Extracorporeal cardiopulmonary resuscitation (ECPR) using venoarterial extracorporeal membrane oxygenation (V-A ECMO) is an advanced resuscitative measure to improve survival in refractory cardiac arrest. Although ECPR allows for organ perfusion during critical interventions, it carries a high-risk of complications, including thrombosis. Thrombus formation within the ECMO circuit and the patient's vasculature is common, yet focal ascending aortic thrombosis following ECPR is rarely reported.

Case summary

We present two case reports of out-of-hospital cardiac arrest managed with ECPR that developed unexpected floating thrombi in the ascending aorta. The first patient, a 45-year-old woman with suspected myocarditis, developed a thrombus that obstructed the left main coronary artery, necessitating surgical thrombus extraction. Despite successful intervention, her cardiac function remained poor, and she required a heart transplant. The second patient, a 46-year-old woman with acute coronary syndrome, had a floating thrombus discovered incidentally. She was managed conservatively but later suffered brain death secondary to cerebral embolism.

Discussion

Ascending aortic thrombosis is an underrecognized complication of ECPR, particularly in patients with non-ejecting hearts. Surgical removal of aortic thrombi, as performed in the first case, may prevent embolic events but lacks standardized guidelines. These cases underscore the need for heightened awareness, early detection, and development of management protocols to mitigate thrombotic risks in ECPR patients. Further studies are warranted to establish treatment strategies for this rare but severe complication.

Keywords

Ascending aorta • Thrombosis • Mechanical support • ECPR • ECMO • Case series

ESC curriculum

7.2 Post-cardiac arrest • 7.1 Haemodynamic instability • 9.1 Aortic disease • 6.4 Acute heart failure

Learning points

- Focal ascending aortic thrombus is a rare but serious complication following ECPR, particularly in non-ejecting hearts.
- Prompt imaging and consideration of surgical intervention may prevent catastrophic embolic events in ECMO patients.
- Consistent guidelines for managing ECMO-related thrombosis are essential to improve outcomes in high-risk cases.

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Introduction

Extracorporeal cardiopulmonary resuscitation (ECPR) is an emerging therapy aimed at improving survival and neurological outcomes in patients with refractory cardiac arrest.¹ By implementing venoarterial extracorporeal membrane oxygenation (V-A ECMO) into conventional resuscitation, ECPR facilitates the restoration of organ perfusion, allowing clinicians time to address the underlying cause of the cardiac arrest.^{2,3} When performed by experienced teams, ECPR has been associated with improved survival rates after out-of-hospital cardiac arrest (OHCA).⁴⁻⁶

Thrombosis is a common complication in patients receiving V-A ECMO support. To minimize the risk of systemic emboli, routine anticoagulation is recommended;⁷ however, achieving the optimal balance between thrombosis prevention and bleeding avoidance remains challenging.⁸

The most common site of thrombosis is within the ECMO circuit, followed by the venous and arterial systems, often due to peri-cannula thrombosis.⁹ In patients with severely impaired left ventricular systolic function and resultant blood stasis, thrombus formation has been reported in the left ventricle,^{10,11} and occasionally in the aortic root.^{12,13}

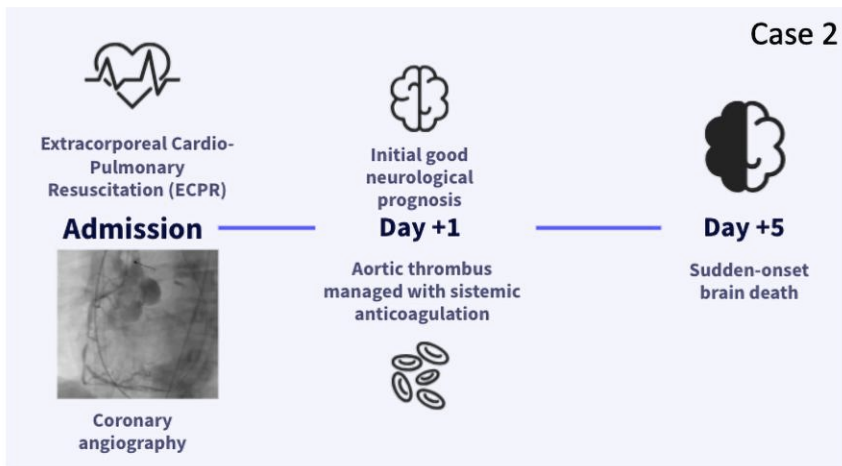
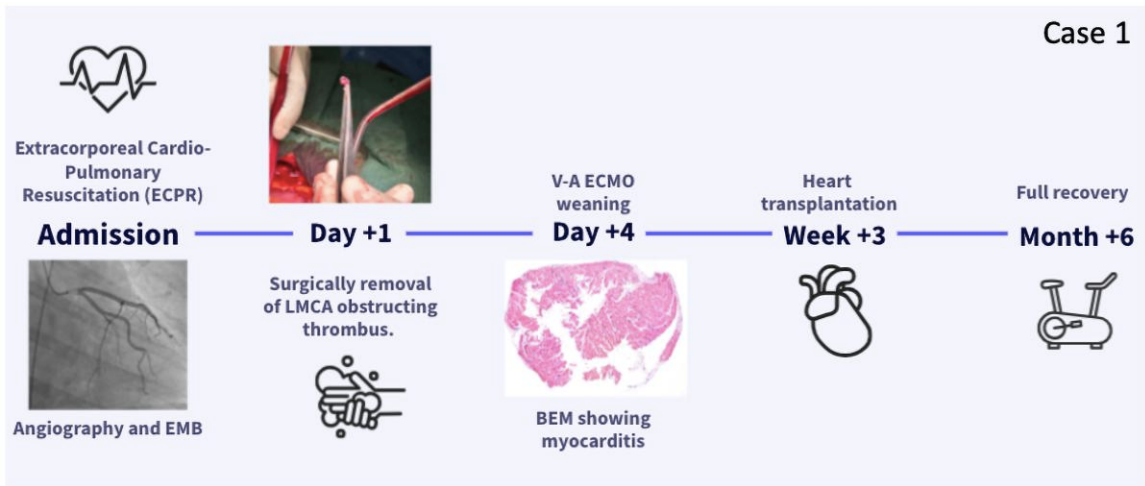
However, evidence regarding the optimal treatment of aortic root thrombosis in ECMO patients remains limited. Additionally, isolated ascending aortic thrombosis without aortic root or left ventricular involvement is, to your knowledge, unprecedented in ECPR cases.

Summary figure

Patient 1

A 45-year-old woman with a history of subclinical hypothyroidism and recent diarrhoea (3 days prior) presented with sudden onset shortness of breath and chest pain, prompting her to seek medical assistance. Upon arrival, paramedics noted poor skin perfusion, with a blood pressure of 85/60 mmHg, heart rate of 128 b.p.m., and oxygen saturation of 88% on room air. A 12-lead electrocardiogram (ECG) showed sinus rhythm at 85 beats per minute (b.p.m.) with a narrow QRS complex, ST-segment elevation in leads aVR and aVL, along with diffuse ST-segment depression in the anterior, inferior and lateral leads (Figure 1A). While preparing for hospital transfer, the patient suffered a cardiac arrest due to ventricular fibrillation. Advanced life support was promptly initiated by the attending medical team. After 30 min without return of spontaneous circulation (ROSC), the medical team contacted our hospital for ECPR. Upon arrival at the hospital 53 min post-cardiac arrest, percutaneous cannulation was performed in the emergency department, and V-A ECMO was initiated, leading to immediate ROSC. The total time from cardiac arrest to ECMO initiation was 65 min. After ECMO cannulation, an endovenous bolus of 5000 units of unfractionated heparin (UFH) was administered.

Invasive coronary angiography was initially interpreted as showing no significant coronary arteries lesions (Figure 1B and [Supplementary material online, Video S1](#)), and an endomyocardial biopsy (EMB) was subsequently performed. Bedside transthoracic echocardiography (TTE) showed increased left ventricular wall thickness (interventricular



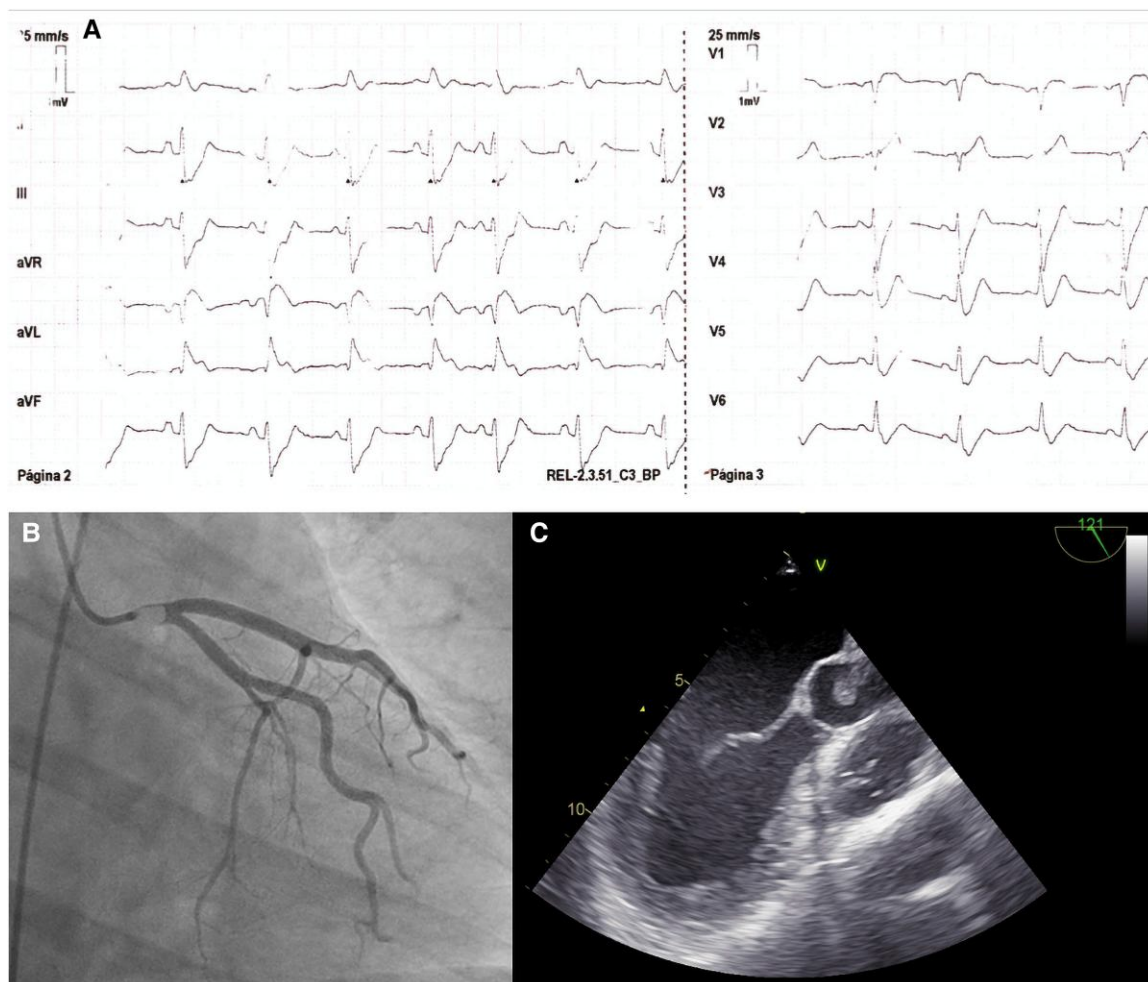


Figure 1 (A): 12-lead electrocardiogram showing sinus rhythm at 85 beats per minute with a narrow QRS complex, ST-segment elevation in leads aVR and aVL, along with diffuse ST-segment depression in the anterior, inferior and lateral leads. (B): Coronary angiogram showing a contrast filling defect in the proximal part of the left main coronary artery. (C): Transoesophageal echocardiography revealing a free-floating mass on the aortic face of the left coronary leaflet of the aortic valve, extending into the ascending aorta with a maximum diameter of 15×20 mm. (Case 1).

septum diameter 23 mm; posterior wall diameter 19 mm), left ventricular ejection fraction (LVEF) of $<5\%$, non-opening of the aortic valve, and no evidence of pericardial effusion or other mechanical complications. No regional wall motion abnormalities were observed at this point. In light of these findings, dobutamine was initiated at a dose of up to 10 mcg/kg/min to increase cardiac output, and an intra-aortic balloon pump (IABP) was implanted to assist with ventricular unloading.

Prior to coronary angiography, the activated partial thromboplastin time (aPTT) was profoundly elevated, with no clotting detected (aPTT ratio >10), and no UFH was administered during the procedure. In the following hours, UFH was titrated to achieve an aPTT of 60 to 70 s, and an activated clotting time (ACT) of 180 to 200 s. Suspecting fulminant myocarditis, methylprednisolone 1 g was administered. Ultrasensitive troponin I was 2473 ng/L at admission, with a peak value $>125\,000$ ng/L at 24 h. Creatine kinase peaked at 9794 U/L at 24 h. NT-proBNP was 186 pg/mL at admission, rising to a peak of 12014 pg/mL on day 6.

24 h after admission, TTE showed a reduction in ventricular wall thickness, with partial LVEF recovery, and a segmental contractility deficit of the anterior and lateral walls, findings that were not suggestive of myocarditis. Following the development of regional wall motion abnormalities,

the coronary angiogram was retrospectively reviewed, and a contrast filling defect in the proximal part of the left main coronary artery was noted (Figure 1B). Suspecting a space-occupying lesion in the aorta, transoesophageal echocardiography (TEE) was performed, revealing a free-floating mass on the aortic face of the left coronary leaflet of the aortic valve, extending into the ascending aorta with a maximum diameter of 15×20 mm (Figure 1C). Given the possibility of a thrombus or an underlying tumour lesion obstructing the left main coronary artery, an emergency sternotomy was performed for surgical removal of the mass (Figure 2) and definitive diagnosis. After the intervention the patient showed clinical improvement, being weaned off V-A ECMO 4 days later and subsequently extubated on day 8 without any neurological deficits.

The EMB revealed myocarditis, and pathological examination confirmed the surgically extracted mass to be a thrombus (Figure 3). Intravenous methylprednisolone was maintained at a dose of 1 mg/kg for 3 days. Due to persistent ventricular dysfunction and dependence on inotropic support, the patient required a heart transplant, which was performed without immediate complications. At 6 months of follow-up, the patient was alive and had fully recovered.

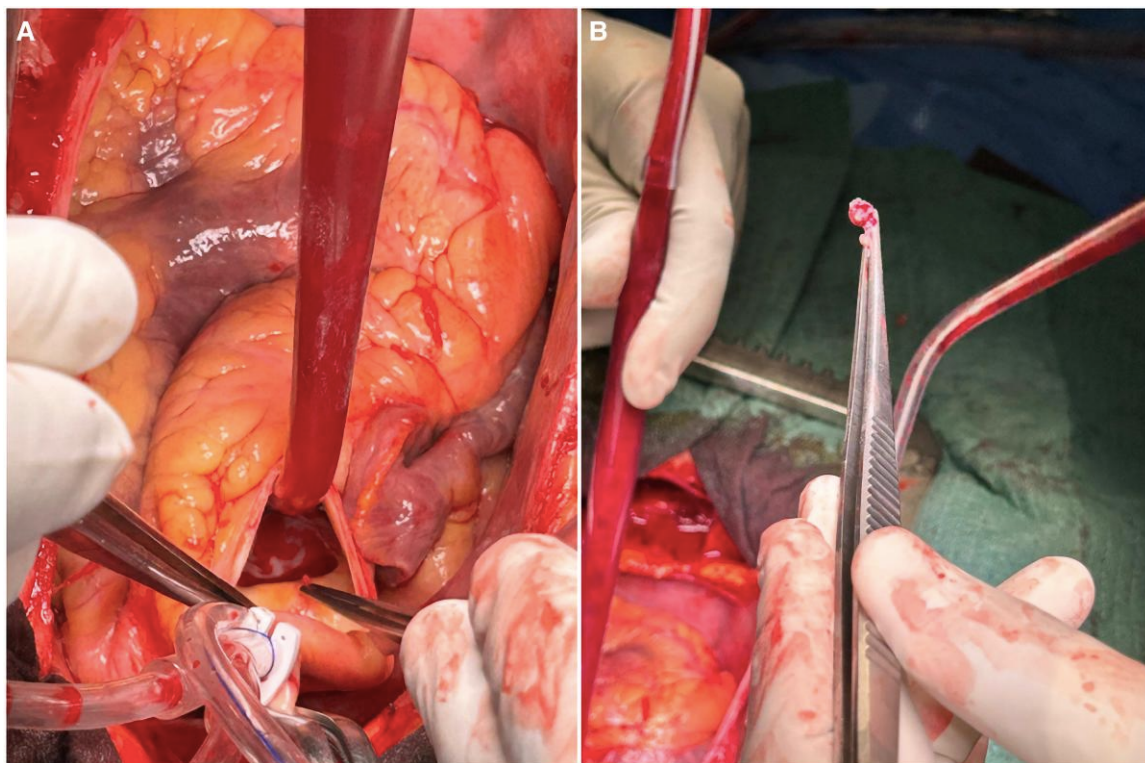


Figure 2 Surgical removal of the aortic thrombus. (Case 1).

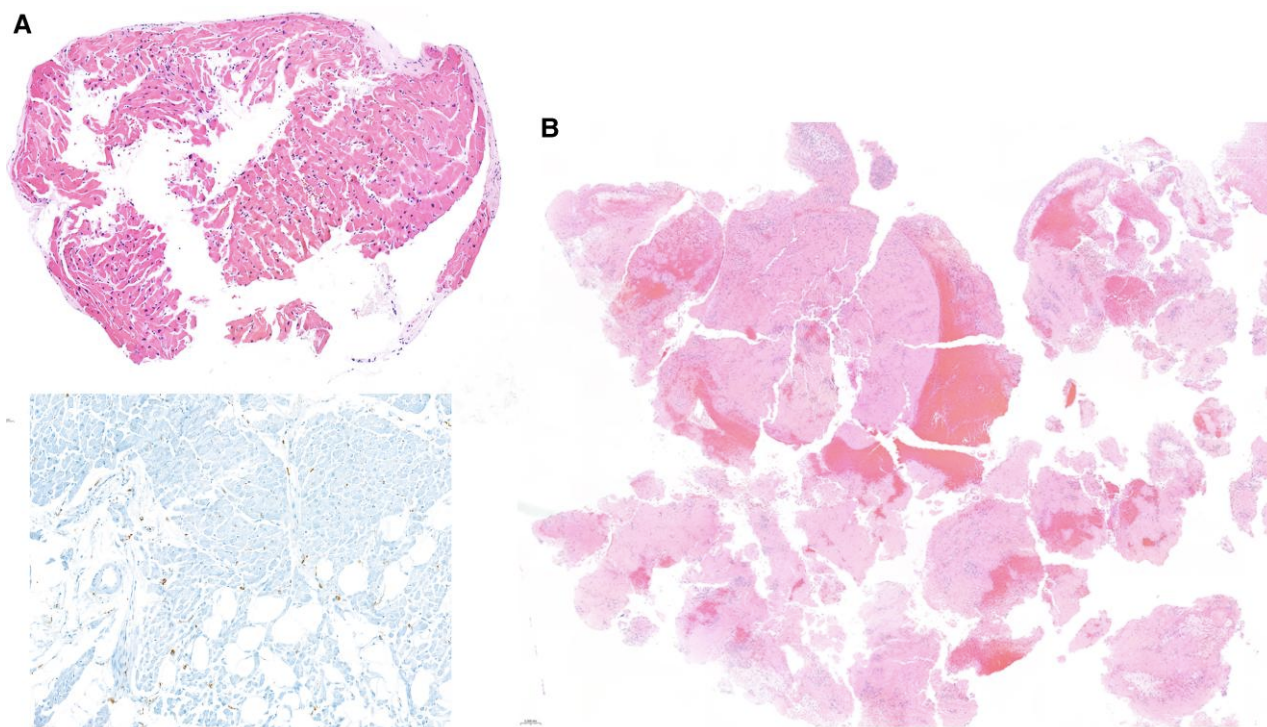


Figure 3 (A): histological findings compatible with myocarditis: lymphohistiocytic inflammation without giant cells (17 histiocytes per mm^2 and 9 T lymphocytes per mm^2), and without signs of necrosis. (B): Fibrino-leukocytic tissue compatible with thrombus, associated with minimal fibromyxoid fragments. (Case 1).

Patient 2

A 46-year-old woman with a history of tobacco smoking, hypertension, and dyslipidemia suddenly experienced dizziness while driving. After stopping the vehicle, she lost consciousness and went into cardiac arrest. Cardiopulmonary resuscitation was initiated by paramedics 8 min later, with an initial shockable rhythm. After 20 min without ROSC, the medical team contacted our hospital, and the decision was made to transfer for ECPR. The total low-flow time until V-A ECMO initiation was 52 min. The ECG showed sinus rhythm at 100 b.p.m., a wide QRS alternating between right and left bundle branch block morphology; additionally, there were non-specific repolarisation abnormalities and frequent ventricular extrasystoles (Figure 4A).

Suspecting acute coronary syndrome as the cause of the cardiac arrest, intravenous acetylsalicylic acid (450 mg) was administered, and the patient was transferred to the catheterisation laboratory. Coronary angiography revealed acute thrombosis of the left main trunk extending to the left anterior descending (LAD) artery and the left circumflex artery, which was treated with effective thrombus aspiration (see [Supplementary material online, Video S2](#)). Additionally, two-vessel coronary artery disease was identified, with a 95% stenosis in the mid segment of the LAD, treated with the implantation of a drug-eluting stent, and 80% stenosis in the mid segment of the right coronary artery. Furthermore, a central filling defect surrounded by contrast and measuring 10 × 15 mm, was identified within the ascending aorta 10 mm cranially from the sinotubular junction (Figure 4B), deemed compatible with a floating thrombus. Although emergent surgical removal of the

thrombus was considered, the surgical risk was deemed prohibitive due to the patient's haemodynamic instability in the post-arrest setting, and a conservative approach was preferred.

Bedside TTE showed a severely depressed LVEF, prompting the implantation of an IABP. Double antiplatelet therapy with acetylsalicylic acid and cangrelor was administered, and intravenous heparin was titrated to achieve an aPTT of 60 to 70 s, and an ACT of 180 to 200 s. Over the following hours the patient's hemodynamics stabilized with the use of vasopressors and inotropes, and serum lactate levels normalized. Neurological assessment showed that brainstem reflexes were still preserved. A non-contrast computed tomography scan excluded intracranial bleeding, and an electroencephalogram performed 48 h after admission demonstrated cortical reactivity to all stimuli. On day 5 post-admission, just as preparations for mechanical support weaning were underway, the patient suddenly developed bilateral fixed mydriasis and loss of brainstem reflexes. Transcranial Doppler ultrasound revealed reverberant blood flow in both middle cerebral arteries and the vertebral artery, leading to a diagnosis of brain death. In accordance with the patient's previously expressed wishes and with the family's consent, the patient became an organ donor, donating the liver and both kidneys.

Discussion

The application of ECPR as a rescue strategy for refractory cardiac arrest is steadily increasing globally. In 2022, ECPR was provided to 2069

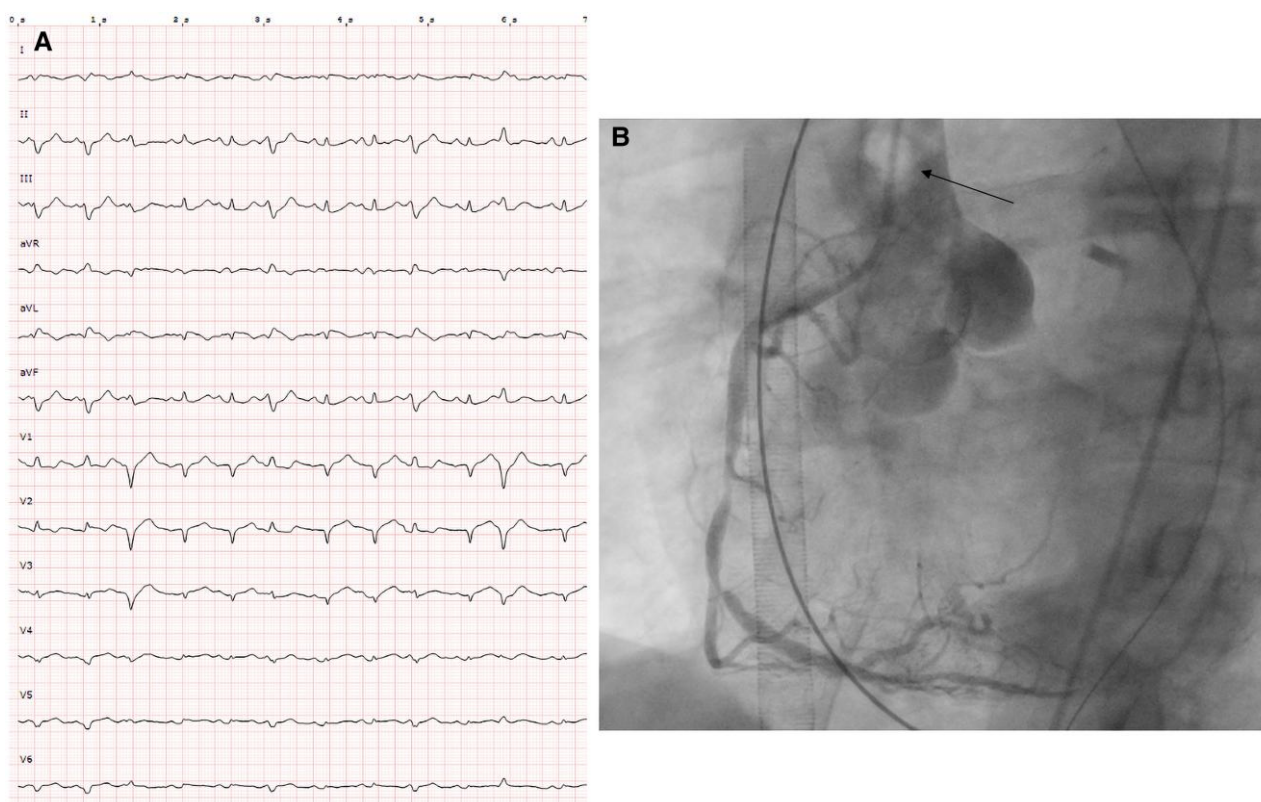


Figure 4 (A): 12-lead electrocardiogram showing sinus rhythm at 100 b.p.m., a wide QRS alternating between right and left bundle branch block morphology; additionally, non-specific repolarization abnormalities and frequent ventricular extrasystoles can be noted. (B): Central filling defect surrounded by contrast and measuring 10 × 15 mm within the ascending aorta 10 mm cranially from the sinotubular junction, deemed compatible with a floating thrombus. (Case 2).

adult patients in the Extracorporeal Life Support Organization registry, with a reported survival to hospital discharge of 30%.¹⁴ Nevertheless, ECMO-associated complications, such as cannulation site complications, brain and coronary hypoxia, bleeding, and thromboembolism, are more prevalent in patients undergoing ECPR.¹⁵

Aortic root thrombosis is a rare complication of V-A ECMO and its prevalence has not been systematically documented in literature. However, its occurrence has been increasingly reported in case studies, particularly in patients with non-ejecting hearts. A case series involving six patients with cardiogenic shock treated with V-A ECMO (including two who underwent ECPR) described thrombosis of the aortic face of the valve leaflets, sometimes extending into the aortic root or left ventricle.¹³ However, to our knowledge, this is the first reported cases series of isolated ascending aortic thrombosis in ECPR patients without associated aortic root or left ventricular involvement.

Several pathophysiological factors unique to ECPR may contribute to susceptibility to aortic thrombosis during this therapy. First, prolonged blood stasis in the aortic root and ascending aorta during extended periods of cardiac arrest can increase the risk of thrombus formation in these regions. Additionally, during the first hours of ECMO support, the high flow rates required to achieve haemodynamic stability can contribute to endothelial damage and turbulence, further predisposing to thrombosis.¹⁶ Another relevant factor is the delayed initiation of therapeutic anticoagulation following ECPR due to the high-risk of bleeding associated with this procedure, which often forces to initiate anticoagulation cautiously or at a lower dose, increasing the risk of thrombotic complications.¹⁷ Together, these conditions create a particularly thrombogenic environment, increasing the risk of rare complications like ascending aortic thrombosis, as observed in our cases.

To reduce the risk of these complications, a prevention algorithm could include protocolized early imaging for thrombus detection, adjustment of ECMO flow rates after achieving multiorgan stabilisation to minimize turbulence, and dynamic management of anticoagulation using advanced monitoring tools, with targets adjusted to a higher anticoagulation range to mitigate thrombotic risk. Additionally, ventricular unloading techniques, such as pharmacologic inotropes or mechanical venting, may help reduce stasis in the aortic root.

Evidence regarding the optimal treatment for aortic thrombosis after V-A ECMO remains scarce. Invasive management, such as surgical venting of the left chambers, has been reported, while conservative strategies include anticoagulation and ventricular unloading via increased inotropic support, or the insertion of a percutaneous device if the former is insufficient. However, surgical removal of an aortic root thrombus has not been described.

We have presented two dramatic cases of young patients who suffered OHCA, rescued with ECPR, who developed a floating thrombus in the ascending aorta despite adequate anticoagulation. In the first case, involving a patient with myocarditis as the cause of cardiac arrest, the thrombus caused occlusion of the left main coronary artery. A successful surgical thrombus extraction was performed; however, the patient suffered an extensive myocardial infarction and was later referred for a heart transplant. In the second case, with acute coronary syndrome as the cause of cardiac arrest, a floating thrombus in the ascending aorta was incidentally discovered. A conservative approach was adopted; however, the patient subsequently suffered sudden brain death, presumably due to cerebral thromboembolism, and became an organ donor. In conclusion, our case series highlights focal ascending aortic thrombosis as a rare but serious complication of ECPR, emphasising the need for vigilance and proactive management. Treatment strategies remain unclear, with surgical removal yielding better outcomes than conservative management in our cases. These findings underline the importance of standardized protocols for early detection and management, as well as further research to improve outcomes in this high-risk population.

Lead author biography



Marco Tomasino is a medical doctor from Italy (University of Palermo). Since 2020, he has been training as a cardiologist in Vall d'Hebron Hospital (Barcelona, Spain). He has a special interest in acute cardiovascular care, adult congenital heart disease, and electrophysiology.

Supplementary material

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

Author contributions

Marco Tomasino (Project administration [lead], Writing—original draft [lead], Writing—review & editing [lead]), Maria Vidal-Burdeus (Conceptualisation [equal], Project administration [equal]), Aitor Uribarri (Conceptualisation [equal], Supervision [equal]), Francisco González-Santorum (Conceptualisation [supporting], Data curation [equal], Validation [equal]), Jordi Riera (Supervision [lead], Validation [lead]), María Alejandra Gabaldón (Investigation [equal]), Neiser Palmer (Validation [supporting]), and Ignacio Ferreira-González (Data curation [lead], Supervision [lead], Validation [lead]).

Consent: The authors confirm that consent was obtained from all the patients in accordance with COPE guidelines.

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Data availability

All data underlying this article are available in the article.

References

1. Suverein MM, Delnoij TSR, Lorusso R, Brandon Bravo Bruinsma GJ, Otterspoor L, Elzo Kraemer CV, et al. Early extracorporeal CPR for refractory out-of-hospital cardiac arrest. *N Engl J Med* 2023;**388**:299–309.
2. Abrams D, MacLaren G, Lorusso R, Price S, Yannopoulos D, Vercaemst L, et al. Extracorporeal cardiopulmonary resuscitation in adults: evidence and implications. *Intensive Care Med* 2022;**48**:1–15.
3. Holmberg MJ, Granfeldt A, Guerguerian AM, Sandroni C, Hsu CH, Gardner RM, et al. Extracorporeal cardiopulmonary resuscitation for cardiac arrest: an updated systematic review. *Resuscitation* 2023;**182**:109665.
4. Bartos JA, Grunau B, Carlson C, Duval S, Ripeckyj A, Kalra R, et al. Improved survival with extracorporeal cardiopulmonary resuscitation despite progressive metabolic derangement associated with prolonged resuscitation. *Circulation* 2020;**141**:877–886.
5. Belohlavek J, Smalцова J, Rob D, Franek O, Smid O, Pokorna M, et al. Effect of intra-arrest transport, extracorporeal cardiopulmonary resuscitation, and immediate invasive assessment and treatment on functional neurologic outcome in refractory out-of-hospital cardiac arrest. *JAMA* 2022;**327**:737.
6. Martínez-Martínez M, Vidal-Burdeus M, Riera J, Uribarri A, Gallart E, Milà L, et al. Outcomes of an extracorporeal cardiopulmonary resuscitation (ECPR) program for in- and out-of-hospital cardiac arrest in a tertiary hospital in Spain. *Med Intensiva (English Edition)* 2024;**48**:565–574.
7. McMichael ABV, Ryerson LM, Ratano D, Fan E, Faraoni D, Annich GM. 2021 ELSO adult and pediatric anticoagulation guidelines. *ASAIO J* 2022;**68**:303–310.

8. Olson SR, Murphree CR, Zonies D, Meyer AD, Mccarty OJT, Deloughery TG, et al. Thrombosis and bleeding in extracorporeal membrane oxygenation (ECMO) without anticoagulation: a systematic review. *ASAIO J* 2021;**67**:290–296.
9. Staessens S, Moussa MD, Pierache A, Rauch A, Rousse N, Boulleaux E, et al. Thrombus formation during ECMO: insights from a detailed histological analysis of thrombus composition. *J Thromb Haemost* 2022;**20**:2058–2069.
10. Alhussein M, Moayedi Y, Posada JD, Ross H, Hickey E, Rao V, et al. Ventricular thrombosis post-venoarterial extracorporeal membrane oxygenation. *Circ Heart Fail* 2017;**10**: e003757.
11. Weber C, Deppe AC, Sabashnikov A, Slottosch I, Kuhn E, Eghbalzadeh K, et al. Left ventricular thrombus formation in patients undergoing femoral veno-arterial extracorporeal membrane oxygenation. *Perfusion* 2018;**33**:283–288.
12. Nishihara T, Kudamatsu N, Hamada T, Nakata Y, Yamamoto W, Nandate H, et al. A case report of thrombotic complete obstruction of the ascending aorta as a complication of venoarterial extracorporeal membrane oxygenation support: steps to prevent thrombosis. *J Cardiothorac Surg* 2020;**15**:185.
13. Hireche-Chikaoui H, Grüber MR, Bloch A, Windecker S, Bloechlinger S, Hunziker L. Nonejecting hearts on femoral veno-arterial extracorporeal membrane oxygenation: aortic root blood stasis and thrombus formation—a case series and review of the literature. *Crit Care Med* 2018;**46**:e459–e464.
14. Tonna JE, Boonstra PS, MacLaren G, Paden M, Brodie D, Anders M, et al. Extracorporeal life support organization registry international report 2022: 100,000 survivors. *ASAIO J* 2024;**70**:131–143.
15. Inoue A, Hifumi T, Sakamoto T, Kuroda Y. Extracorporeal cardiopulmonary resuscitation for out-of-hospital cardiac arrest in adult patients. *J Am Heart Assoc* 2020;**9**: e015291.
16. Nessler N, Gouin-Thibaut I, Parasido A, Flécher E, Mansour A. Early endothelial injury in cardiogenic shock patients on venoarterial ECMO. *Intensive Care Med* 2024;**50**:1929–1930.
17. Gutierrez A, Kalra R, Chang KY, Steiner ME, Marquez AM, Alexy T, et al. Bleeding and thrombosis in patients with out-of-hospital ventricular tachycardia/ventricular fibrillation arrest treated with extracorporeal cardiopulmonary resuscitation. *J Am Heart Assoc* 2024;**13**:e034516.