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Case report

Myocarditis with multiorgan failure – 135 days fighting for life

Emilia Słabońska¹, Gabriela Mystek¹, Dominika Nicz¹, Katarzyna Lis¹, Monika Otocka¹,
Marek Grochla²

¹Students' Scientific Club, Department of Cardiac Anaesthesiology and Intensive Care, Faculty of
Medical Sciences in Zabrze, Medical University of Silesia, Katowice, Poland

²Department of Cardiac Anaesthesiology and Intensive Care,
Faculty of Medical Sciences in Zabrze, Medical University of Silesia, Katowice, Poland

Address for correspondence:

Emilia Słabońska
Studenckie Koło Naukowe
Klinika Kardiologii i Intensywnej Terapii
ul. M. Curie-Skłodowskiej 9, 41-800 Zabrze
Śląskie Centrum Chorób Serca
tel. +48 32 273 27 31
e-mail: e.slabonska01@gmail.com

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ABSTRACT

Fulminant myocarditis is a rare, life-threatening condition that rapidly progresses to refractory cardiogenic shock requiring mechanical circulatory support. Prolonged extracorporeal life support increases survival but exposes patients to severe hemorrhagic, thromboembolic, and infectious complications. We present a complex case of fulminant myocarditis complicated by prolonged VA-ECMO support, massive transfusions, multidrug-resistant infections, and hepatic abscess formation. A 29-year-old man was admitted with fulminant myocarditis, severe biventricular failure (left ventricular ejection fraction 10%), atrial fibrillation, and combined cardiogenic and septic shock. He required mechanical ventilation, veno-arterial extracorporeal membrane oxygenation (VA-ECMO), intra-aortic balloon pump (IABP), vasoactive support, and renal replacement therapy. Persistent bleeding from the ECMO site resulted in transfusion of 100 units of packed red blood cells over 110 days, complicated by mild transfusion-related acute lung injury. An occipital ischemic stroke without neurological deficits was diagnosed during hospitalization. Following successful VA-ECMO and IABP removal, the patient developed septic shock secondary to a groin wound infection caused by New Delhi metallo- β -lactamase-producing *Klebsiella pneumoniae* and *Pseudomonas aeruginosa*. Subsequently, multiple hepatic lesions suspicious for liver abscesses were identified. Percutaneous drainage yielded vancomycin-resistant *Enterococcus faecium*, prompting targeted antimicrobial therapy and transfer to a specialized hepatology center. Long-term veno-arterial extracorporeal membrane oxygenation serves as an effective bridge to recovery in patients with reversible profound circulatory failure. However, prolonged VA-ECMO support necessitates vigilant surveillance for hemorrhagic, thromboembolic, and infectious complications. A multidisciplinary management strategy is essential for optimizing clinical outcomes and patient prognosis.

KEYWORDS

TRALI, liver abscess, VA-ECMO, multidrug-resistant organisms, massive transfusion, fulminant myocarditis, vancomycin-resistant *Enterococcus faecium*

INTRODUCTION

Fulminant myocarditis is a rare, life-threatening condition that rapidly progresses to refractory cardiogenic shock requiring mechanical circulatory support. Prolonged extracorporeal life support increases survival but exposes patients to severe hemorrhagic, thromboembolic, and infectious complications. We present a complex case of fulminant myocarditis complicated by prolonged VA-ECMO support, massive transfusions, multidrug-resistant infections, and hepatic abscess formation.

CASE REPORT

A 29-year-old man was admitted to the Silesian Center for Heart Diseases in Zabrze in critical condition. He arrived intubated, mechanically ventilated, in severe lactic acidosis, and in combined cardiogenic and septic shock, with acute biventricular heart failure.

Endomyocardial biopsy was not performed because of the patient's critical clinical condition. On the day of admission to the Specialist Cardiac Care Unit, laboratory evaluation revealed a high-sensitivity cardiac troponin I concentration of 14.061 pg/mL, which decreased to 8.115 pg/mL on the following day. Creatine kinase (CK) and creatine kinase-MB (CK-MB) levels were 1.398 U/L and 22.46 ng/mL, respectively. Inflammatory markers were also markedly elevated, with a C-reactive protein (CRP) concentration of 106.78 mg/L on admission and a procalcitonin level of 5.36 ng/mL measured during the subsequent days of hospitalisation.

He was dependent on circulatory support with an intra-aortic balloon pump (IABP) and veno-arterial ExtraCorporeal Membrane Oxygenation (VA-ECMO), both inserted as life-saving interventions at the referring hospital due to an EF drop to 10%.

Transthoracic echocardiography performed after the initiation of veno-arterial extracorporeal membrane oxygenation (VA-ECMO) and intra-aortic balloon pump (IABP) support demonstrated severe left ventricular systolic dysfunction, with a left ventricular ejection fraction (LVEF) of 15%. The aortic valve opened fully during each systole, while the left ventricular outflow tract velocity-time integral (LVOTVTI) measured 7 cm despite a VA-ECMO flow of 4.5 L/min and IABP counterpulsation at a 1:1 ratio, indicating markedly impaired intrinsic left ventricular stroke volume. In addition, moderate mitral regurgitation, mild-to-moderate tricuspid regurgitation (peak systolic pressure gradient of 38 mmHg), and a small pericardial effusion were identified.

ECMO VA is presented in Figures 1A and 1B. Continuous catecholamine infusions and levosimendan were administered. The patient presented with features of myocarditis and atrial fibrillation, preceded by several days of upper respiratory tract infection, dyspnea, and diarrhea.

Due to multiorgan failure with acute liver and kidney injury (initial laboratory tests revealed ALT > 3.300 U/L, AST 2.619 U/L, and a serum creatinine concentration of 323 µmol/L) renal replacement therapy was initiated with a Cytosorb filter, along with broad-spectrum antibiotic therapy. From the first day of admission, the patient had continuous bleeding from the ECMO cannulation site in the right groin, resulting in numerous transfusions of packed red blood cells (PRBCs). The total number reached more than 100 units over 135 days. Multiple hemostasis evaluations were

performed, none revealing clinically relevant abnormalities. This massive transfusion burden led to mild manifestations of Transfusion-Related Acute Lung Injury (TRALI). Another complication identified on CT imaging was an occipital stroke, though without neurological deficits (Figure 1C).

After improvement of EF to 30%, a decision was made to wean the patient off ECMO, which was successful on the second attempt. Simultaneously, thrombectomy of the right common and superficial femoral arteries was performed. The right groin was sutured, with a Redon drain and a gentamicin-impregnated sponge left in situ. The IABP was removed the following day. Unfortunately, the patient subsequently developed septic shock.

Cultures grew *Klebsiella* NDM and *Pseudomonas aeruginosa*, and targeted antibiotic therapy was initiated, including empirical treatment for NDM (aztreonam + ceftazidime/avibactam + colistin).

Multiple well-demarcated liver lesions appeared, initially suspected to be ischemic in origin. CT described them as diffuse, hypodense, oval, non-enhancing fluid-filled areas (density approx. 8–10 HU), ranging from 1 mm to 3.45 cm in diameter in the right liver lobe. Liver function parameters, including the international normalised ratio (INR), aspartate aminotransferase (AST), alanine aminotransferase (ALT), serum ammonia, albumin, and the overall protein profile, remained within normal limits. Cholelithiasis and choledocholithiasis were excluded. However, jaundice persisted, with total bilirubin levels ranging from 45 to 55 µmol/L and gamma-glutamyl transferase (GGTP) levels exceeding 1.000 U/L.

He was transferred for one day to a collaborating university center, where the largest hepatic lesion was punctured and drained. Culture of the aspirated fluid yielded *Enterococcus faecium* VRE. Targeted antibiotic therapy was initiated (linezolid and oritavancin). Abscesses were suspected (Figure 1D).

Prior to discharge, cardiac magnetic resonance imaging (CMR) was performed to guide further management. The examination demonstrated a dilated left ventricle (LVEDVI 148 mL/m²) with moderately reduced systolic function (LVEF 37%) and mildly impaired right ventricular systolic function (RVEF 43%), accompanied by global left ventricular hypokinesia, most pronounced in the distal interventricular septum and inferior wall. CMR revealed mid-wall late gadolinium enhancement within the interventricular septum, consistent with non-ischemic fibrosis, as well as suspected subepicardial fibrosis in the basal posterior wall, findings compatible with previous inflammatory myocardial injury. In addition, a small subendocardial ischemic scar involving <50% of the myocardial wall thickness was identified in the distal interventricular septum and left

ventricular apex (left anterior descending coronary artery territory); however, its limited extent was considered insufficient to account for the severity of left ventricular systolic dysfunction. Importantly, no myocardial edema or increased T2 signal intensity was detected, and the updated CMR criteria for active myocarditis were therefore not fulfilled, indicating a healed rather than ongoing inflammatory process.

The diagnosis of fulminant myocarditis was established on the basis of the clinical presentation, markedly elevated cardiac biomarkers, acute severe biventricular systolic dysfunction following a recent viral-like illness, and CMR findings consistent with prior inflammatory myocardial injury. Follow-up transthoracic echocardiography demonstrated further improvement in left ventricular systolic function, with recovery of the LVEF to 45%. As the patient remained in good cardiovascular and respiratory condition, he was transferred to a tertiary hepatology center for further management. The chronological course of hospitalization and the key therapeutic interventions are summarized in Figure 2.

DISCUSSION

According to the recommendations of the ECMO Therapy Group of the Polish Society of Anaesthesiology and Intensive Care, haemorrhagic and infectious complications are among the most common and most serious adverse events associated with venoarterial extracorporeal membrane oxygenation (VA-ECMO) therapy [1].

Studies indicate that massive bleeding from ECMO cannulation sites typically requires around 17 units of PRBCs [2].

The current Extracorporeal Life Support Organization (ELSO) guidelines also emphasise the need to individualise anticoagulation with unfractionated heparin and to perform regular monitoring of coagulation parameters, including activated clotting time (ACT), activated partial thromboplastin time (aPTT), anti-factor Xa activity, platelet count, and fibrinogen concentration. In the event of significant bleeding, the guidelines recommend temporarily reducing or discontinuing anticoagulation, correcting haemostatic abnormalities, and addressing the underlying cause of bleeding while closely monitoring the ECMO circuit for thrombus formation.

Another major concern during prolonged VA-ECMO support is the risk of infection. Current ELSO guidelines do not recommend routine prolonged prophylactic antibiotic therapy. Instead, strict adherence to aseptic technique during cannulation and cannula care, daily assessment for signs of infection, prompt collection of microbiological specimens when infection is suspected, and early initiation of targeted antimicrobial therapy are strongly recommended. In the present case, the

development of multiple liver abscesses represented a severe infectious complication that required prolonged antimicrobial treatment and multidisciplinary management [1,3].

The present case supports previous observations regarding patients requiring prolonged VA-ECMO support. Berger et al. [4] demonstrated that, despite the high mortality associated with the acute phase of cardiogenic shock, patients successfully weaned from VA-ECMO may achieve favourable long-term outcomes. However, these patients require close long-term follow-up because of the risk of late complications related to both the underlying disease and extracorporeal support. In their cohort, the mean duration of VA-ECMO support was approximately 11 days, and longer support was associated with a higher incidence of complications and a more complex clinical course. Similarly, in our patient, prolonged VA-ECMO therapy was complicated by both thromboembolic events (ischaemic stroke) and severe infectious complications, underscoring the importance of comprehensive daily assessment and the individualisation of anticoagulation and antimicrobial therapy in accordance with current ELSO recommendations.

CONCLUSIONS

As this case illustrates, prolonged circulatory support with VA-ECMO may facilitate recovery from profound cardiogenic shock, even in patients with fulminant myocarditis. However, prolonged extracorporeal support is associated with a substantial risk of both haemorrhagic and infectious complications. Successful management therefore requires individualised anticoagulation, early recognition and prompt treatment of complications, and close collaboration within a multidisciplinary team. This case also underscores the importance of actively investigating potential sources of infection in patients receiving prolonged VA-ECMO support who present with persistent signs of infection, as early diagnosis and appropriate treatment may significantly improve clinical outcomes.

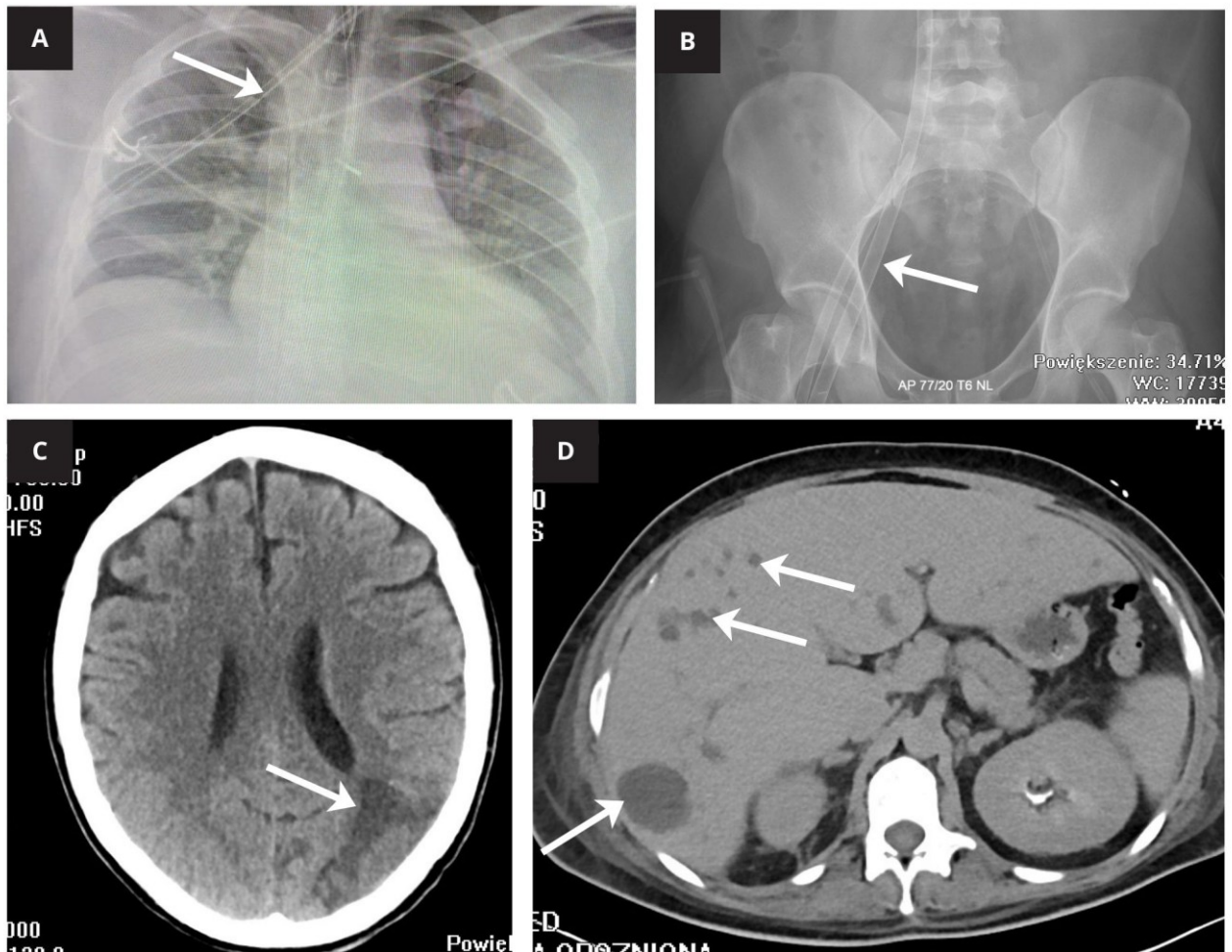


Fig. 1. (A) Anteroposterior chest radiograph demonstrating cardiomegaly and the course of the ECMO cannula within the mediastinum (arrow). The endotracheal tube is in situ, and bilateral atelectatic and inflammatory pulmonary opacities are present. **(B)** Pelvic radiograph showing the course of the venous ECMO cannula inserted via the right femoral vein and extending into the inferior vena cava (arrow). **(C)** Axial non-contrast computed tomography (CT) of the head demonstrating a hypodense lesion in the left occipital lobe, consistent with an acute ischaemic infarction in the territory of the left posterior cerebral artery (arrow). **(D)** Axial contrast-enhanced CT of the abdomen demonstrating multiple hypodense lesions with peripheral rim enhancement throughout the hepatic parenchyma, consistent with multiple liver abscesses (arrows).

TIMELINE OF EVENTS

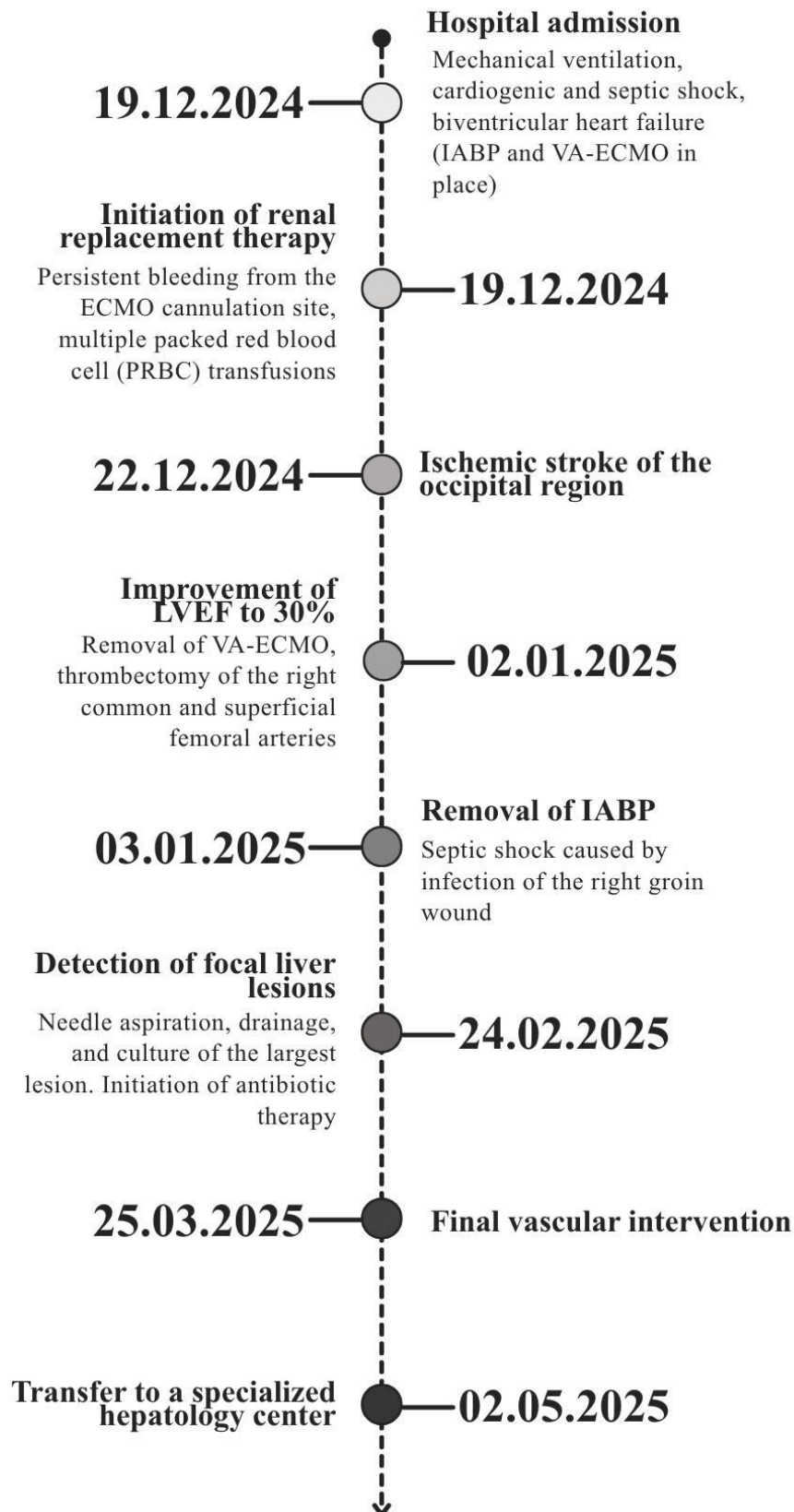


Fig. 2. Chronological timeline highlighting the key moments in the patient's treatment, from admission to the Silesian Centre for Heart Diseases in Zabrze up to the transport to a specialized hepatology centre

Conflict of interest

The authors declare no conflicts of interest.

Authors' contribution

Study design – M. Grochla, G. Mysłek, D. Nicz

Manuscript preparation – E. Słabońska, G. Mysłek, D. Nicz, K. Lis, M. Otocka

Literature research – E. Słabońska, G. Mysłek, D. Nicz, K. Lis, M. Otocka

Final approval of the version to be published – M. Grochla, E. Słabońska, G. Mysłek, D. Nicz, K. Lis, M. Otocka

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