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CASE REPORT
Heart failure

Fulminant eosinophilic myocarditis treated by venoarterial extracorporeal membrane oxygenation and adjunctive immunosuppressive therapy: a case report

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Background Eosinophilic myocarditis can result from drug hypersensitivity or various systemic disorders. Although corticosteroids are effective, treatment strategies are not standardized.

Case summary A 78-year-old male presented with dyspnoea. On admission, he was febrile and hypotensive and had sinus tachycardia with a newly developed right bundle branch block. Laboratory findings showed marked eosinophilia with elevated troponin. The patient rapidly progressed to cardiogenic shock, and venoarterial extracorporeal membrane oxygenation (VA-ECMO) was applied. Eosinophilic myocarditis was suspected, and endomyocardial biopsy confirmed dense myocardial eosinophil infiltration consistent with the diagnosis. Review of history revealed recent cefaclor use for a hand injury as the probable cause. The patient's condition quickly improved after initiation of high-dose intravenous methylprednisolone, allowing weaning from VA-ECMO. However, persistent troponin elevation and myocardial inflammation on fluorodeoxyglucose positron emission tomography (FDG-PET) were observed despite 5 weeks of steroid therapy. Subcutaneous mepolizumab, an anti-interleukin-5 monoclonal antibody, was initiated as an adjunctive immunosuppressive therapy. After seven cycles, troponin levels normalized, cardiac function fully recovered, and follow-up FDG-PET demonstrated a marked reduction in myocardial inflammation.

Discussion We describe a case of fulminant eosinophilic myocarditis that was possibly associated with cefaclor use and successfully managed with VA-ECMO and adjunctive immunosuppressive therapy. Fluorodeoxyglucose positron emission tomography imaging helped detect residual inflammation and assess treatment response. Immunosuppressive therapy allowed a reduction of steroid dose, mitigating the risk of steroid-related side effects.

Keywords Eosinophilic myocarditis • Cardiogenic shock • LAVA-ECMO • Mepolizumab • Case report

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Learning points

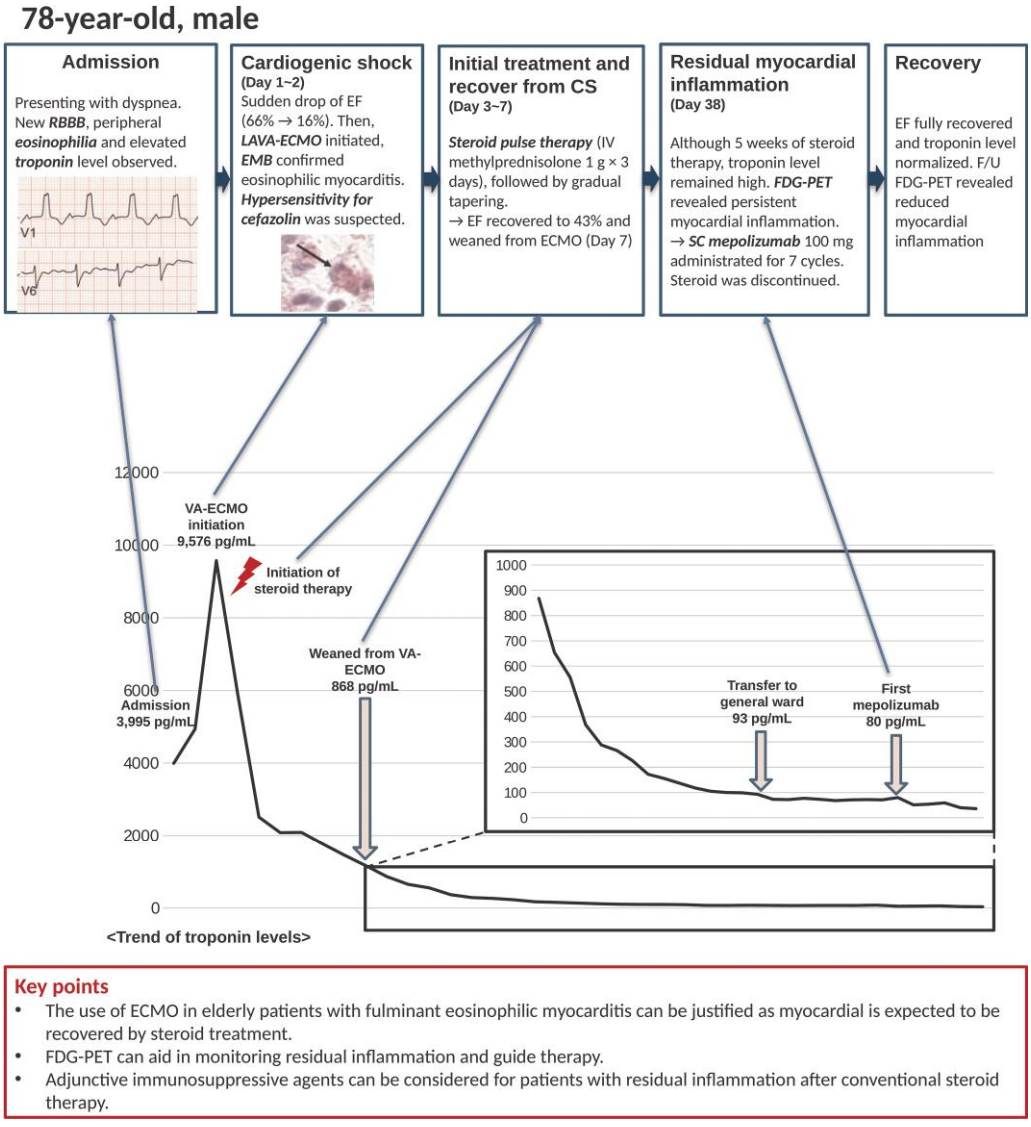
- As eosinophilic myocarditis generally responds well to steroid therapy and has the potential for myocardial recovery, the use of ECMO can be considered despite its inherent risks.
- Fluorodeoxyglucose positron emission tomography may help detect residual inflammation and monitor treatment response in myocarditis, although its role remains debated.
- Adjunctive immunosuppressive agents, such as benralizumab or mepolizumab, can be considered for patients with persistent inflammation after conventional steroid therapy.

Introduction

Eosinophilic myocarditis, a rare subtype of myocarditis, is characterized by eosinophilic infiltration of the myocardium usually caused by unexpected drug reactions, parasitic infections, or various systemic disorders.[1–3] Although steroid therapy is reported to be effective in treating eosinophilic myocarditis, there is currently no established treatment strategy due to limited evidence.[2,3]

Herein, we report a case of fulminant eosinophilic myocarditis complicated by cardiogenic shock, likely triggered by drug hypersensitivity, that was successfully managed with venoarterial extracorporeal membrane oxygenation (VA-ECMO) and adjunctive immunosuppressive agent on top of steroid therapy.

Summary figure



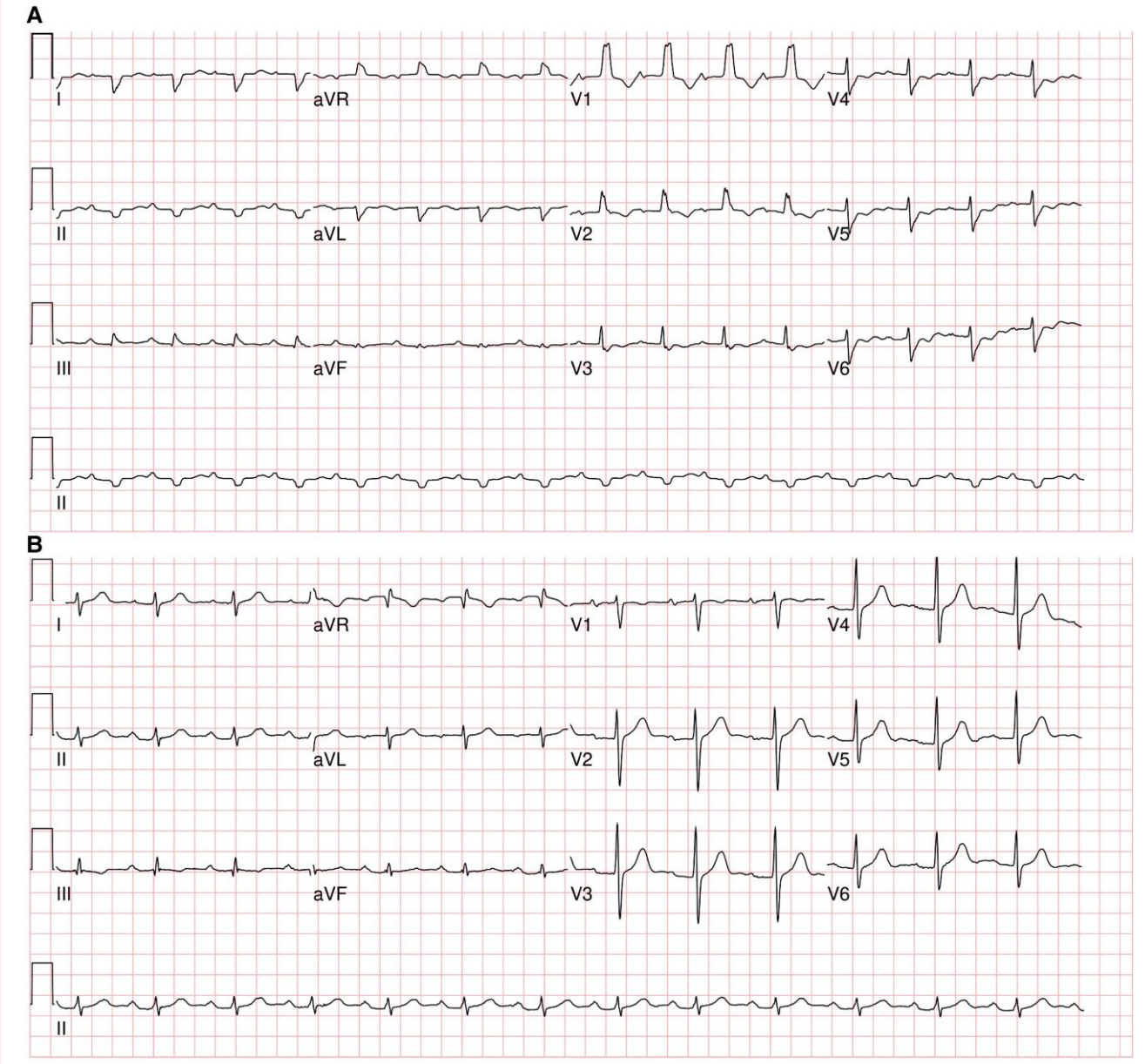


Figure 1 (*A*) Admission ECG showing sinus tachycardia with newly developed right bundle branch block. (*B*) Previous ECG obtained 1 year earlier, demonstrating normal conduction. ECG, electrocardiogram.

Case presentation

A 78-year-old male patient presented to the emergency department with dyspnoea. On arrival, the patient appeared pale and agitated. The patient's blood pressure was 81/51 mmHg, heart rate was 120 beats per minute, respiratory rate was 25 breaths per minute, and body temperature was 39.8 °C. He had recently used cefaclor for a hand injury and resumed it a week ago after a brief interruption due to a rash. At admission, there was no evidence of rash and palpable lymph nodes. Electrocardiogram (ECG) showed a newly developed right bundle branch block (*Figure 1A*), compared to the ECG taken 1 year ago (*Figure 1B*). Chest radiography revealed pulmonary congestion and cardiomegaly. Laboratory findings showed leukocytosis with peripheral eosinophilia (total eosinophil count: 1330/mcL) and elevated troponin-T (3995 pg/mL). Arterial lactate level was 2.1 mmol/L (*Table 1*). Transthoracic echocardiography (TTE) revealed a normal left ventricular (LV) ejection fraction (EF) of 66%, with wall thickening of 16 mm.

After 12 h, lactate increased to 6.7 mmol/L and troponin-T level to 9576 pg/mL with clinical deterioration. The patient's blood pressure dropped to 71/50 mmHg, and inotropic agent was initiated. Follow-up TTE revealed that the LVEF was depressed to 16%, and the right ventricular (RV) fractional area change (FAC) decreased to 13% (see Supplementary material *S1*). As cardiogenic shock progressed rapidly, VA-ECMO was implemented. Because his pulse pressure was substantially diminished, and LV distention was expected after VA-ECMO

initiation, transseptal cannulation was simultaneously performed. Left atrial pressure was 15 mmHg. Coronary angiography revealed no significant stenoses. With suspicion of acute myocarditis, endomyocardial biopsy (EMB) was performed to identify the specific subtype. Histological examination revealed eosinophilic myocardial infiltration consistent with eosinophilic myocarditis (*Figure 2*).

Table 1 **Initial laboratory findings**

Test	Result	Reference value
Haemoglobin, g/dL	14.6	13.0–17.4
White blood cell count, /mcL	11 290	4000–10 800
Neutrophils, *n* (%)	8300 (73.5)	1700–7000 (39–74)
Eosinophils, *n* (%)	1330 (11.8)	0–500 (0–7)
C-reactive protein, mg/L	138.3	0–8
Creatinine, mg/dL	1.06	0.68–1.19
Aspartate aminotransferase, IU/L	286	13–34
Alanine aminotransferase, IU/L	286	5–46
Troponin-T, pg/mL	3995	0–14
CK, IU/L	4827	44–245
CK-MB, ng/mL	87.6	0–5
NT-proBNP, pg/mL	18 563	0–486
Arterial lactate, mmol/L	2.1	0.5–1.6
Arterial pH	7.488	7.35–7.45
Arterial pCO2, mm Hg[a]	23.4	35–45
Arterial pO2, mm Hg[a]	74.7	83–108

CK, creatine kinase; CK-MB, creatine kinase-myocardial band; NT-proBNP, N-terminal prohormone of brain-type natriuretic peptide
[a]Results under an oxygen supply of 6 L/min via a nasal cannula.

Genetic testing, including rearrangement of *PDGFR*, *JAK2*, and *BCR-ABL1*, showed no abnormalities. Autoimmune serologies, including antineutrophil cytoplasmic antibodies (ANCA) targeting myeloperoxidase (MPO) and proteinase 3 (PR3), were negative. Serological tests for parasitic infections were negative. The patient's total immunoglobulin E level was 2237 kU/L (reference: <100 kU/L in adults). Finally, based on the recent history of cefaclor use and a previous episode of rash, hypersensitivity to cefaclor was identified as the most likely cause.

Steroid pulse therapy with intravenous methylprednisolone 1 g/day was administered for 3 days. Consequently, LVEF improved to 43% and RV FAC to 31%, and wall thickness reduced to 12 mm (see Supplementary material *S2*), allowing successful weaning from VA-ECMO. At that time, troponin-T level was 868 pg/mL. Cardiac magnetic resonance (CMR) imaging performed after weaning from VA-ECMO revealed subtle diffuse patchy and linear subepicardial-to-mesocardial late gadolinium enhancement (LGE) in the LV myocardium with elevated tissue mapping parameters, consistent with acute myocarditis (see Supplementary material *S3*).

As steroid therapy gradually tapered to 30 mg of oral prednisolone over 5 weeks, troponin-T level remained elevated (80 pg/mL). To assess the residual myocardial inflammation, cardiac positron emission tomography (PET) using F-18 fluoro-2-deoxyglucose (FDG) was performed, which revealed persistent inflammatory activity in the myocardium (*Figures 3A* and *B*). Furthermore, he developed paresthaesia in both feet, which was suggestive of steroid-induced neuropathy. Following consultation with an allergist, subcutaneous mepolizumab (100 mg monthly) was initiated to treat the remaining myocardial inflammation while allowing reduction of the steroid dose (*Table 2*). Steroid therapy was discontinued after five cycles of mepolizumab treatment. After seven monthly cycles of subcutaneous mepolizumab along with guideline-directed medical therapy

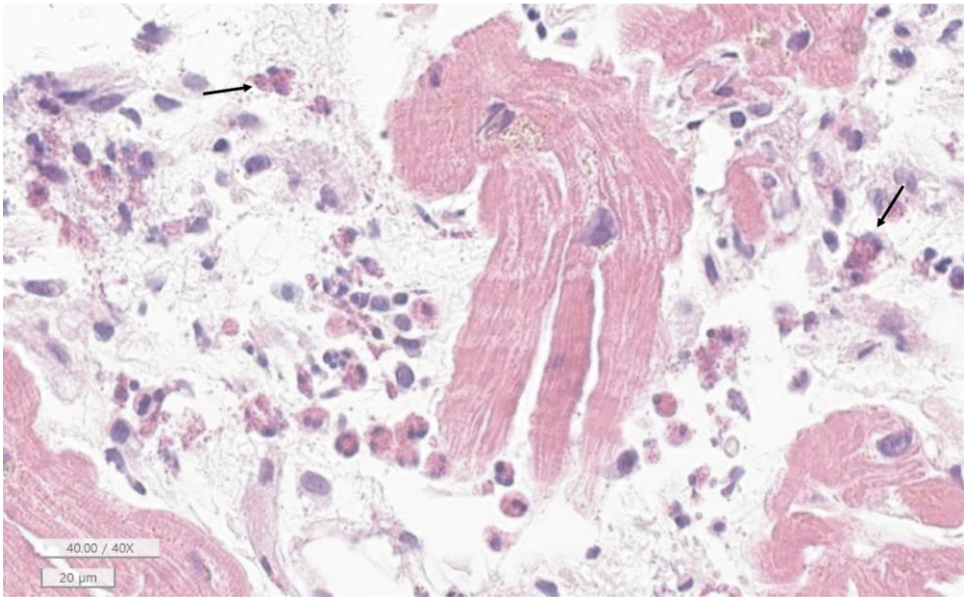


Figure 2 Endomyocardial biopsy specimen showing eosinophilic infiltration (arrows) within the myocardium.

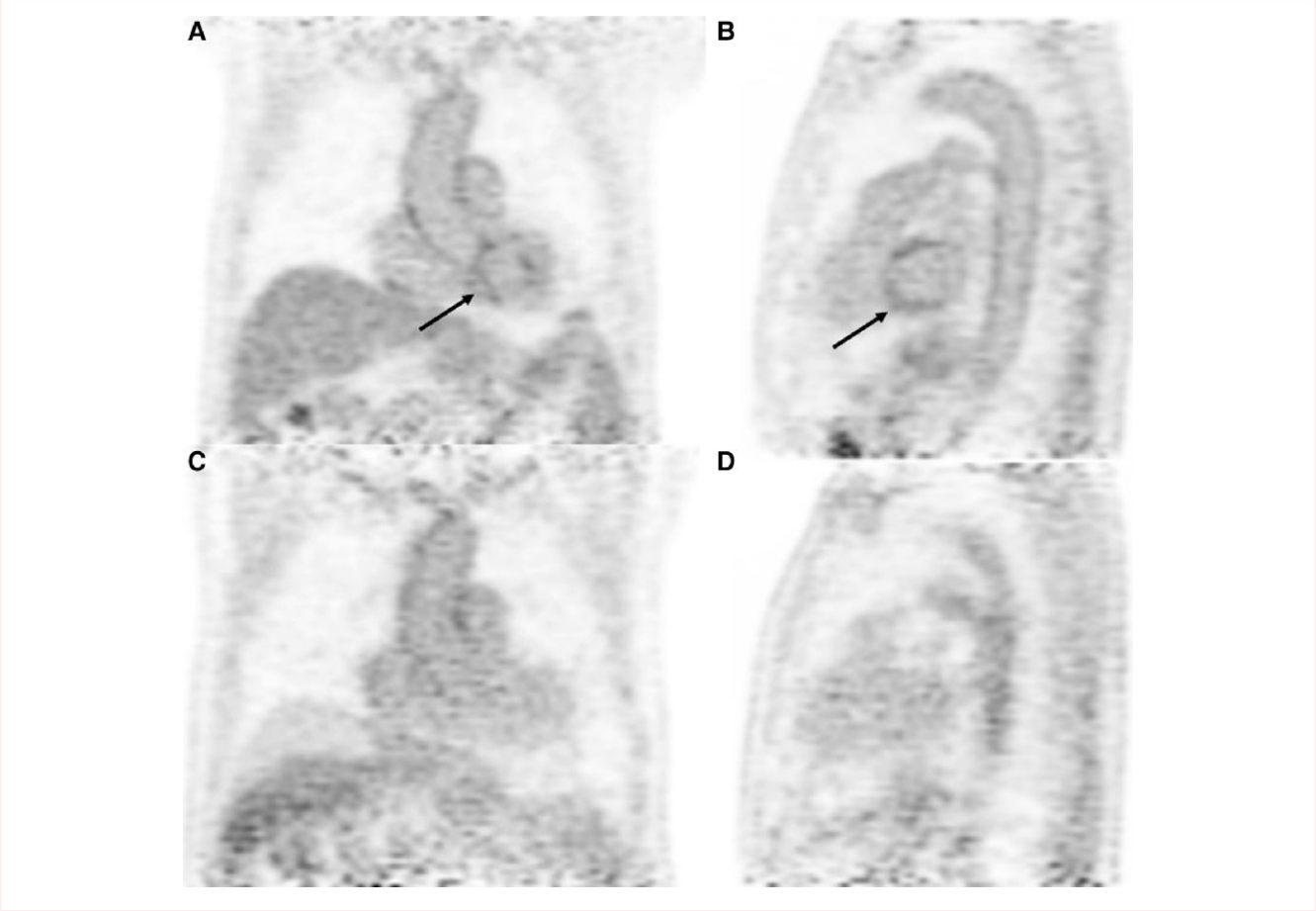


Figure 3 Serial cardiac FDG-PET images. (*A*: coronal; *B*: sagittal) Initial PET scan demonstrating increased FDG uptake in the left ventricular myocardium (arrows), consistent with residual myocardial inflammation. (*C*: coronal; *D*: sagittal) Follow-up PET scan after adjunctive immunosuppressive therapy showing marked reduction of myocardial FDG uptake. FDG, fluoro-2-deoxyglucose; PET, positron emission tomography.

for heart failure, troponin-T level was normalized and he denied symptoms of heart failure. A follow-up TTE revealed normal biventricular systolic function (LVEF, 75%; RV FAC, 36%) with normal wall thickness (10 mm), and diastolic function was preserved (relaxation abnormality pattern) (see Supplementary materials *S4* and *S5*). NT-proBNP declined markedly from 18 563 pg/mL at admission to 82 pg/mL at follow-up. Tests confirmed the absence of restrictive physiology and complete haemodynamic recovery. Follow-up FDG-PET imaging showed decreased FDG uptake in the myocardium, indicating an improvement in myocardial inflammation (*Figures 3C and D*). Follow-up CMR demonstrated resolution of LGE and normalization of tissue mapping parameters, corroborating the clinical and functional recovery (see Supplementary material *S6*).

Discussion

Acute myocarditis is a myocardial disease with inflammatory process caused by various aetiologies.[1,4,5] It is diagnosed based on the clinical presentation, ECG, biomarkers (e.g. troponin), cardiac imaging (e.g. TTE, CMR), and invasive diagnostic tools (e.g. EMB).[1,4,5]

Among patients with eosinophilic myocarditis, approximately 20% present with acute heart failure or cardiogenic shock.[2] In the pooled data of case reports involving 179 histologically proven eosinophilic myocarditis cases, 43% experienced arrhythmic events in the acute phase, including cardiac arrest; 17% required temporary MCS devices; and 22% died during hospitalization.[2]

In the present case, the patient exhibited typical features of eosinophilic myocarditis, including fever, dyspnoea, ECG change, peripheral eosinophilia, and elevated troponin levels. Early recognition of these findings is essential for determining whether temporary MCS needs to be implemented, because MCS carries risks such as vascular injury and bleeding especially in older patients. In this case, the potential reversibility of eosinophilic myocarditis supported the decision to use temporary MCS.[6–8]

Progressive LV dysfunction, worsening hypoxia, and reduced pulse pressure prompted the use of VA-ECMO with preemptive transseptal cannulation [left atrial venoarterial extracorporeal

Table 2 Timeline of clinical course and treatment

Date	Clinical event	Steroid dose[a]	Mepolizumab
2024-04-16	Presented with cardiogenic shock	—	—
2024-04-17	VA-ECMO initiation[b]; EMB performed (*Figure 2*)	—	—
2024-04-18~20	Steroid pulse therapy	Intravenous MP 1000 mg	—
2024-04-21~27	Intravenous steroid maintenance	Intravenous MP 62.5 mg	—
2024-04-22	Weaned from VA-ECMO	Dose maintained	—
2024-04-28~05-04	Transition to oral steroid	Oral PD 40 mg	—
2024-05-02	Cardiac MRI (see Supplementary material *S3*)	Dose maintained	—
2024-05-05~13	Oral steroid taper	Oral PD 35 mg	—
2024-05-14~22	Oral steroid taper	Oral PD 30 mg	—
2024-05-23	1st mepolizumab; FDG-PET (*Figures 3A and B*)	Dose maintained	Subcutaneous 100 mg
2024-05-24~30	Oral steroid taper	Oral PD 25 mg	—
2024-05-31~	Oral steroid taper	Oral PD 20 mg	—
2024-08-22	Follow-up FDG-PET (*Figures 3C and D*)	—	—
2024-09-19	5th mepolizumab; discontinued steroid therapy	Stopped	Subcutaneous 100 mg
2024-11-14	7th (final) mepolizumab	—	Subcutaneous 100 mg
2024-12-03	Follow-up cardiac MRI (see Supplementary material *S6*)	—	—

EMB, endomyocardial biopsy; FDG-PET, fluorodeoxyglucose-positron emission tomography; MP, methylprednisolone; MRI, magnetic resonance imaging; PD, prednisolone; VA-ECMO, venoarterial extracorporeal membrane oxygenation
[a]Steroid doses are presented as daily dose.
[b]VA-ECMO was initiated with 2400 revolutions per minute and 3.6 litres per minute.

membrane oxygenation (LAVA-ECMO)] to prevent LV distension. Although routine transseptal cannulation is not recommended, LAVA-ECMO may benefit patients in whom LV distension is anticipated just after VA-ECMO initiation.[9] Although microaxial flow pumps can also support LV function and unloading, the device cannot provide biventricular support; therefore, it is not an appropriate option for this patient.[8]

Although high-dose steroid therapy is considered the first-line strategy and eosinophilic myocarditis generally responds well to steroid therapy, dosing and duration are not standardized and the criteria for initiating adjunctive therapy remain unclear.[1–3] In this case, despite initial haemodynamic improvement with initial methylprednisolone treatment, troponin-T remained elevated and FDG-PET suggested ongoing residual myocardial inflammation after 5 weeks, indicating a suboptimal response to steroid therapy maintenance. Although the role of FDG-PET imaging in myocarditis remains unclear, unlike in cardiac sarcoidosis, it may aid in assessing treatment response.[1,4,10] Furthermore, the development of steroid-induced peripheral neuropathy contributed to the initiation of mepolizumab because immunosuppressive therapy not only enhances the anti-inflammatory process in the myocardium but may also facilitate the reduction of steroid use, thereby mitigating the risk of steroid-related side effects. Although benralizumab—a potent interleukin-5 receptor blocking agent—is considered for eosinophilic myocarditis, mepolizumab, which targets interleukin-5 itself, was administered as benralizumab was not available at our institution.[5,11]

In conclusion, given the potential for myocardial recovery, VA-ECMO can be considered even in older patients with eosinophilic myocarditis. Furthermore, the addition of immunosuppressive agents to conventional steroid therapy may be considered to control residual myocardial inflammation detected by multimodal assessments.[1,3,10] Finally, further studies are warranted to establish optimal treatment strategies for eosinophilic myocarditis and to define appropriate methods for assessing treatment response.

Lead author biography

Dong Woo Suh, MD, is a cardiology fellow at Severance Hospital in Seoul, Korea. Dong Woo Suh, MD, is trained in internal medicine and is currently a clinical fellow in cardiology at Severance Hospital, Seoul, Korea. His main interests are interventional cardiology and critical care medicine.

Supplementary material

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

Author contributions

Dong Woo Suh [Data curation, Investigation, Writing—original draft (lead)], Sanghyup Lee (Conceptualization, Supervision, Writing—review & editing [lead], Writing—original draft [supporting]), Chan Joo Lee [Resources, Writing—review & editing

(supporting)], and Kyung Hee Park [Resources, Writing—review & editing (supporting)]

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Conflict of interest. The authors declare that they have no conflict of interest.

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Data availability

The data that support the findings of this case report are available on reasonable request from the corresponding author.

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