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CASE REPORT

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Eosinophilic myocarditis in dilated cardiomyopathy: a case report

Mengxuan Li[2] and Hong Chen[1*]

Abstract

Background Eosinophilic myocarditis (EM) is a rare cardiac condition that is often difficult to diagnose. Although endocardial myocardial biopsy is considered the gold standard for diagnosis, sampling errors can lead to false-negative results. This case report discusses the diagnosis and treatment of dilated cardiomyopathy in a patient with EM.

Case presentation In this article, we report a case of a 32-year-old female patient diagnosed with dilated cardiomyopathy. EM was strongly suspected based on a progressive increase in eosinophil count, the absence of known allergens or common etiological factors, elevated eosinophil levels in alveolar lavage fluid, and a diagnosis of eosinophilic pneumonia. However, endocardial myocardial biopsy results failed to show definite evidence of myocarditis. Despite the implementation of various therapeutic interventions including pharmacological treatments, electrical defibrillation, endotracheal intubation, and ventilator-assisted breathing, the patient's condition showed minimal improvement. Subsequent initiation of extracorporeal membrane oxygenation and intra-aortic balloon pump support also failed to achieve the anticipated recovery. The patient subsequently underwent heart transplantation, and cardiac tissue samples were sent for pathology examination. The diagnostic report revealed a large number of eosinophils, confirming the diagnosis of EM. After heart transplantation, the patient's vital signs gradually stabilized, and she was discharged in good condition.

Conclusions Endocardial myocardial biopsy plays an important role in diagnosing EM but may yield false-negative results. In this case, heart transplantation provided critical diagnostic information, with the pathology report confirming the presence of eosinophils and supporting the diagnosis of EM.

Keywords Dilated cardiomyopathy, Eosinophilic myocarditis, Fulminant myocarditis, Endocardial myocardial biopsy, Cardiac transplantation

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Background

Dilated cardiomyopathy (DCM) is a heterogeneous cardiac condition typically characterized by ventricular dilation and reduced myocardial contractile function [1]. In this case, the patient was also comorbid with a rare form of eosinophilic myocarditis (EM). EM is a rare form of myocarditis defined by eosinophilic infiltration, often accompanied by eosinophilia [2–4]. This condition can lead to serious cardiac complications, including myocardial necrosis, thrombosis, and endocardial myocardial fibrosis [5]. Although endocardial myocardial biopsy is considered the gold standard for diagnosing EM, it is important to note that sampling errors may lead to negative findings. In this particular case, the initial myocardial biopsy did not provide conclusive evidence of myocarditis. However, the patient later underwent heart transplantation, and cardiac tissue samples were sent for examination. The diagnostic pathology report revealed a substantial presence of eosinophils, which ultimately supported the diagnosis of EM.

Case presentation

The patient, a 32-year-old female, was admitted to the hospital with a history of "recurrent cough, sputum, and shortness of breath for over 5 years, with exacerbations lasting more than 20 days prior to admission". Upon hospitalization, laboratory tests revealed elevated levels of B-type natriuretic peptide precursor (pro-BNP; normal < 125 ng/L) at 1107 ng/L, high-sensitivity troponin T (hs-TnT; normal < 14 ng/L) at 20.0 ng/L, and elevated eosinophil levels (17.1% percentage, 1.74×10^9/L absolute count). Besides, the autoimmune serology results showed the negative ANA, anti-dsDNA and ENA panel.

The MRI report from November 14, 2023 shows that the cardiac MRI with contrast indicates reduced and delayed enhancement of the interventricular septum and anterior wall motion amplitude. A dynamic electrocardiogram (ECG) with heart rate variability analysis on December 3rd, 2023, showed sinus rhythm with a longest R-R interval of 1.232 s and notable ST-T wave changes. Moreover, atrial premature beats were detected at a frequency of 2 times/24 h. A specialized echocardiogram (Fig. 1) performed on December 4, 2023, showed mild left ventricle enlargement and moderately reduced left

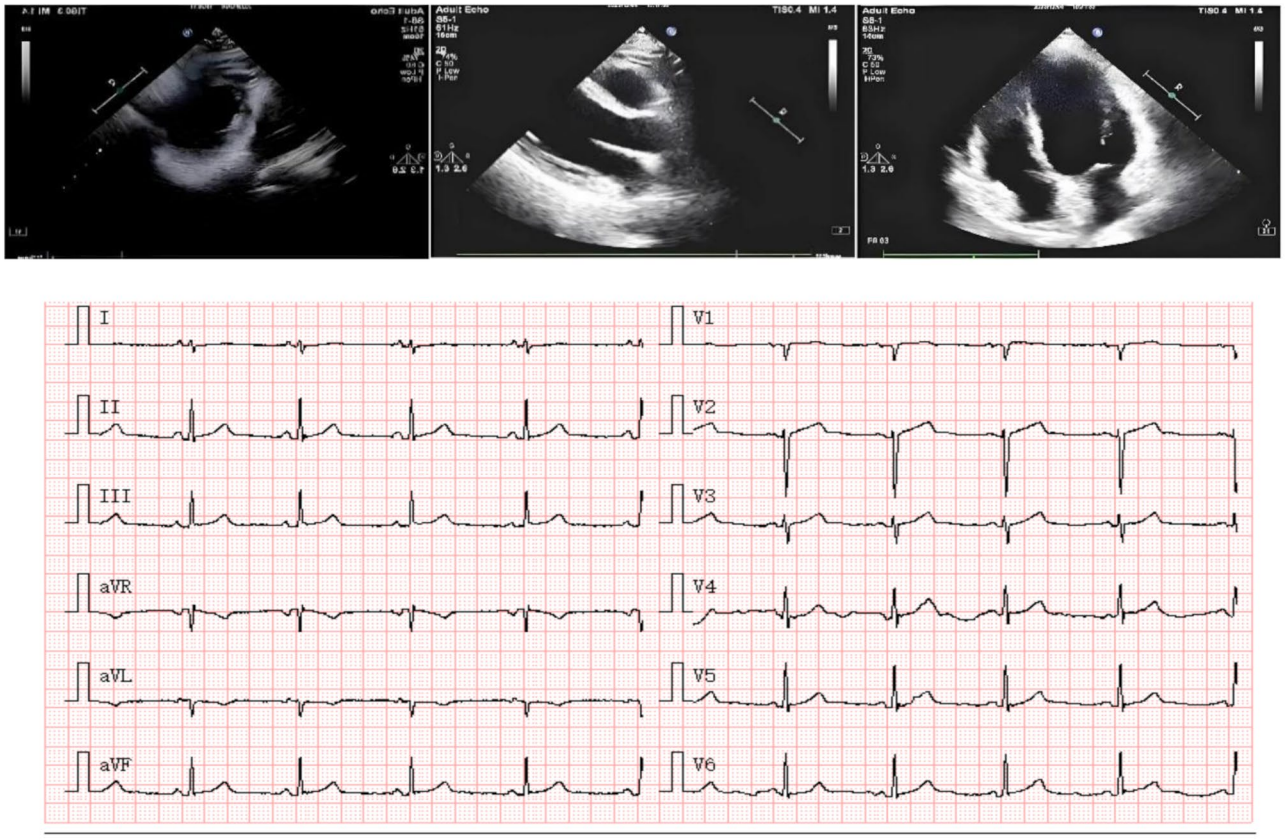

Fig. 1 Specialist echocardiography revealed a slight enlargement of the left ventricle accompanied by moderately reduced left ventricular systolic function (top). Electrocardiography (ECG) image of her fist admission (bottom)

ventricular (LV) systolic function, the specific value of ejection fraction (EF) was 49% (Fig. 1).

Throughout this period, EM was strongly suspected due to a progressive increase in eosinophil count, significant eosinophilic infiltration in alveolar lavage fluid, and a diagnosis of eosinophilic pneumonia. A consultation with the respiratory department subsequently confirmed the diagnosis of eosinophilic pneumonia. On February 22, 2024, the cardiac MRI images of this patient showed an enlarged heart mainly with the right atrium and left ventricle enlargement, the motion amplitude weakened. A few punctate and linear low-signal perfusion defect areas can be seen in the basal part of the ventricular septum and part of the free wall myocardium of the left ventricle, and there is a small amount of pericardial effusion (Fig. 2). The steroid regimen was carefully adjusted according to disease progression and treatment response. Methylprednisolone was initiated at 40 mg oral daily on November 18 for suspected eosinophilic myocarditis, then reduced to 30 mg daily on November 29 after eosinophil counts normalized (decreasing from 17.1 to 0.5%). During the second hospitalization for fulminant myocarditis, high-dose IV methylprednisolone (500 mg daily) was started on June 1 for cardiogenic shock. Tapering began on June 4 as cardiac biomarkers improved (myoglobin decreasing from 947.20 to 302.60 ng/mL, CK-MB mass from 42.20 to 15.89 ng/mL, and hs-TnT from 4293.0 to 1763.0 ng/L), with further dose reduction on June 6 when biomarkers showed continued decline (myoglobin 62.80 ng/mL, CK-MB mass 2.03 ng/mL, and hs-TnT 1800.0 ng/L). On December 6, 2023, the patient underwent a myocardial biopsy, but the results failed to provide definite evidence of myocarditis (Fig. 3).

On June 1, 2024, the patient was readmitted for the evaluation of myocardial markers. During the period before this admission, the patient took Entresto, metoprolol succinate tablets, spironolactone, furosemide tablets, and dapagliflozin tablets for long-term control of the disease. However, she developed cough and sputum again, mainly white frosting sputum and occasionally a small amount of yellow pus sputum. After lying flat, the cough got worse, and the patient enjoyed a high pillow position at night. One week ago, the patient began to develop a fever at night, with a maximum body weight of 38.2°C, which could be relieved by self-administration of "ibuprofen", but the fever returned the next night, accompanied by night sweats throughout the body, occasional body aches, and no abdominal distension and abdominal pain. There was no nausea, vomiting or edema of both lower limbs. The laboratory results revealed a myoglobin level (normal < 72 ng/mL) of 127.50 ng/ml, creatine kinase isoenzyme MB mass (CK-MB mass; normal < 5 ng/mL) at 63.95ng/ml, and high-sensitivity troponin T at 1367.0ng/L. Shortly after admission, the patient developed a malignant arrhythmia, necessitating urgent interventions, including medication administration, electrode fibrillation, tracheal intubation, and assisted ventilation via a ventilator. Despite these aggressive interventions, the patient's condition did not show significant improvement.

On June 2, 2024, extracorporeal membrane oxygenation (ECMO) was initiated to support cardiac circulation. On June 4, 2024, the patient continued to experience persistent ventricular tachycardia (Fig. 4), undetectable blood pressure, and unstable vital signs despite optimal medical management and high-dose vasoactive medications during ECMO support. Subsequently, the patient was diagnosed with fulminant myocarditis (FM), and an intra-aortic balloon pump (IABP) was implemented.

On June 7, 2024, specialist echocardiography (Fig. 5) revealed LV enlargement, with a normal-sized right atrium, normal interventricular septum, and posterior wall thickness, the EF value was 18%. However, there was severely reduced coherence in LV wall motion and a lack of overall wall movement coordination. By June 12, 2024, the patient's hemodynamic status showed slight improvement, leading to a gradual reduction in ECMO support intensity, with plans for imminent ECMO weaning.

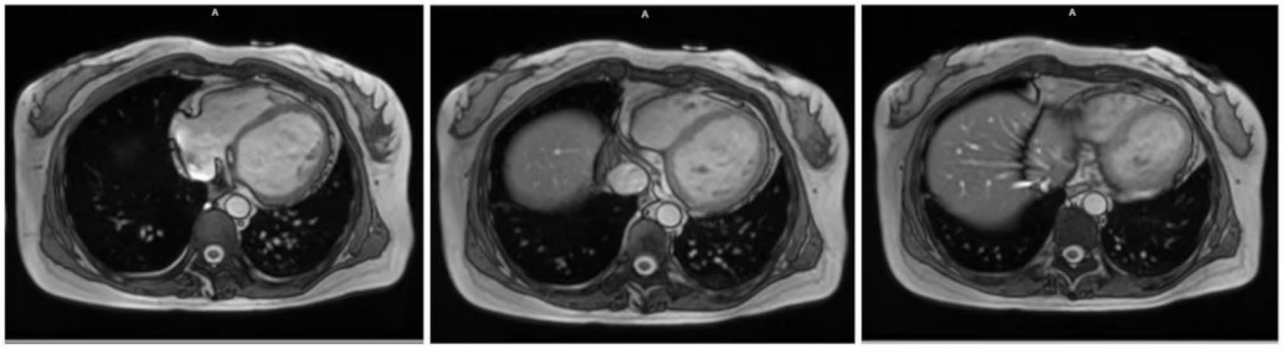

Fig. 2 Cardiac MRI images exhibited an enlarged heart mainly with the right atrium and left ventricle enlargement, a few punctate and linear low-signal perfusion defect areas can be seen in the basal part of the ventricular septum and part of the free wall myocardium of the left ventricle, and there is a small amount of pericardial effusion

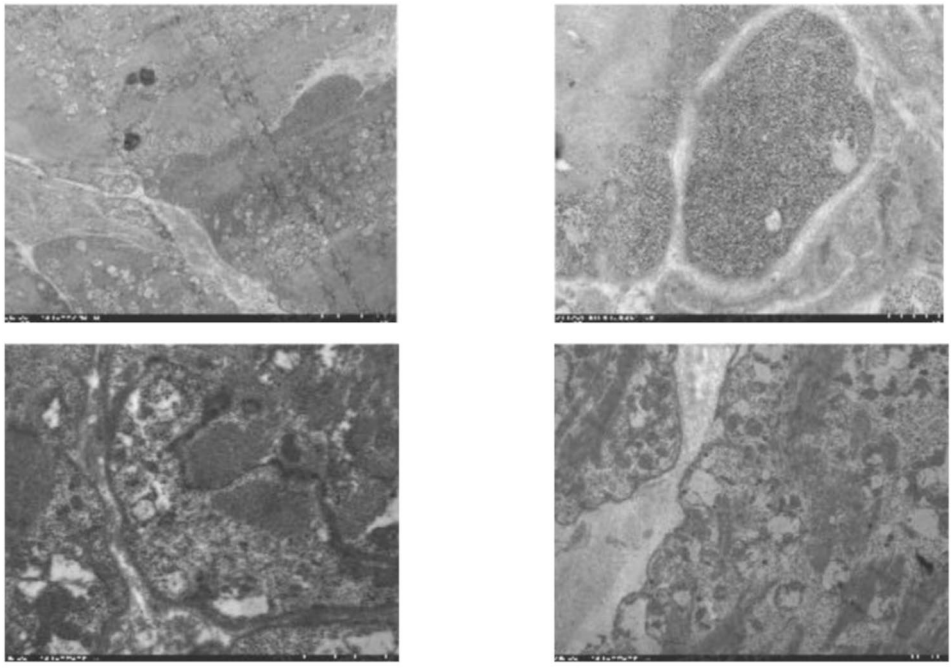

Fig. 3 Cardiac biopsy report at first admission (top line) and after heart transplantation (bottom line). Top line: The myocardial tissue sections showed scattered lymphocyte infiltration, mild fibrosis, and some myocardial cells with branching. Based on the tissue morphology, special staining, and electron microscopy, there is no definitive evidence of myocarditis. However, glycogen storage disease cannot be ruled out. Please consider the clinical context in conjunction with these findings. Bottom line: The findings are consistent with myocarditis with post-myocardial infarction changes. Eosinophilic myocarditis cannot be ruled out

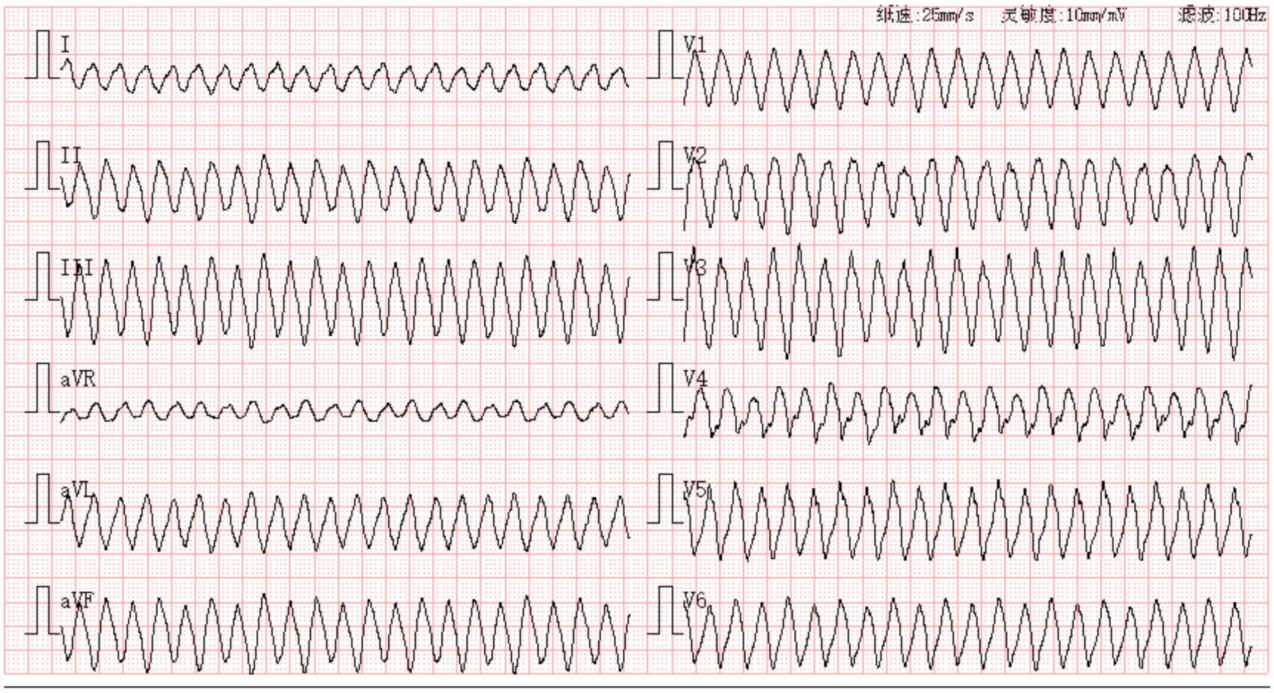

Fig. 4 ECG demonstrated ventricular tachycardia

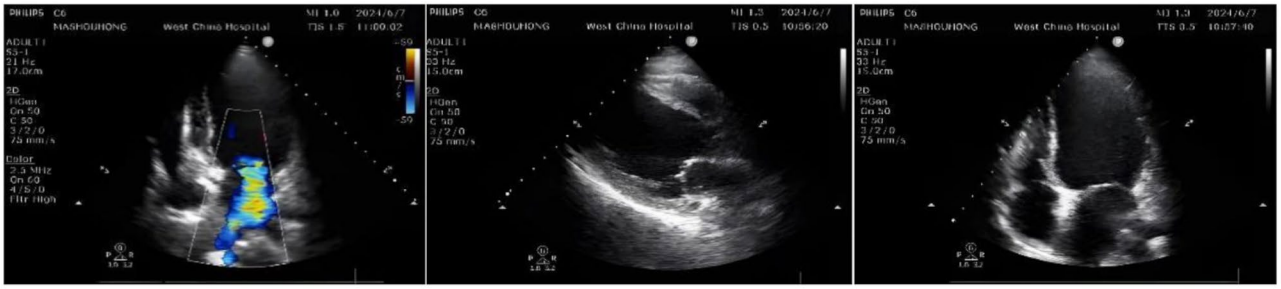

Fig. 5 Specialist echocardiography revealed left ventricular enlargement, normal right atrial size, normal interventricular septum and posterior wall thickness of the left ventricle, severely reduced left ventricular wall motion coherence, lack of overall wall movement coordination, severely reduced left ventricular systolic function measurements, no obvious abnormalities in the visual lumen of the aorta and pulmonary artery, and unclear display of the left and right coronary arteries

However, on June 13, 2024, following a reduction in ECMO support, the patient experienced paroxysmal tachycardia and a marginal increase in lactate levels, indicating poor cardiac function and complicating the removal of ECMO. Given these developments, heart transplantation was considered as a potential therapeutic option. Myoglobin levels were re-evaluated on June 14, 2024, demonstrating an increase to 427.50 ng/ml, with creatine kinase isoenzyme MB mass at 9.27 ng/ml, B-type natriuretic peptide precursor at 2612 ng/L, and high-sensitivity troponin T at 687.2 ng/L.

On June 19, 2024, an allogeneic heart transplant was successfully performed. Postoperatively, the patient was transferred to the intensive care unit in a sedated state with ventilator-assisted support. Cardiovascular and respiratory function remained stable, and vital signs were consistently monitored. Subsequent examination of the cardiac tissue revealed a significant presence of eosinophils, and eosinophilic myocarditis cannot be ruled out (Fig. 3). The diagnosis of EM relies on histopathological evidence of eosinophilic infiltration in the myocardium, supported by the following features such as inflammatory infiltrates (≥ 10 eosinophils per high-power field in affected areas), myocyte damage (evidence of necrosis, degranulation of eosinophils, and eosinophil-derived cytotoxic proteins) and absence of other causes (exclusion of infections, vasculitis, or drug hypersensitivity) [4, 5].

By June 26, 2024, the patient's condition stabilized, with vital signs showing a heart rate of 80 beats/min, oxygen saturation at 100%, and blood pressure at 124/70 mmHg. The patient successfully passed voluntary respiratory testing, managed airway secretions well, and underwent successful airway leak testing. Consequently, tracheal extubation was performed, and non-invasive ventilation was initiated.

Following extubation, the patient's vital signs remained relatively stable. On June 27, 2024, the patient regained consciousness, displayed mild fatigue, and was able to comply with medical instructions. Finally, on July 24, 2024, the patient was discharged from the hospital in good condition.

Conclusions

Current research suggests that eosinophil cationic protein and major basic protein play key roles in mediating myocardial damage in EM by activating myocardial mast cells, leading to myocardial fibrosis [4, 6, 7]. Although endocardial myocardial biopsy is considered the gold standard for diagnosing EM, it is important to note that sampling errors can lead to false-negative results. In suspected cases of myocarditis, biopsy findings have shown an incidence rate of only 0.1% [8], indicating its low sensitivity and specificity [3, 9]. In this particular case, the myocardial biopsy failed to provide conclusive evidence of myocarditis, while this patient exhibited a significant eosinophilic infiltration in alveolar lavage fluid and a diagnosis of eosinophilic pneumonia, hence the EM was highly suspected. Cardiac tissue examined after heart transplantation showed a large number of eosinophils, and then the EM diagnosis was finally confirmed.

The diagnosis of EM can be challenging, endomyocardial biopsy is definitive for the diagnosis of EM and shows inflammatory infiltration with nests of eosinophils, additionally, some imaging diagnostic tools also play an important role in identifying this condition when biopsy findings are equivocal. Among these, cardiac magnetic resonance imaging (CMR) and positron emission tomography (PET) scans are particularly valuable. CMR is a powerful non-invasive imaging modality that can provide critical insights into myocardial inflammation and damage, including myocardial edema, which is a hallmark of EM. EM shows a patchy or diffuse non-ischaemic subendocardial pattern of late gadolinium enhancement (LGE), but can also less frequently demonstrate mid-wall and subepicardial LGE [10]. PET scans, particularly those using fluorodeoxyglucose (FDG), can assess metabolic activity in the myocardium, which is especially useful for detecting inflammation associated with eosinophilic infiltration. FDG-PET can visualize areas of increased

glucose metabolism that correspond to inflammatory activity in the myocardium, this feature can help confirm the diagnosis and the treatment effect of EM when combined with other imaging modalities [11].

The primary treatment for EM focuses on addressing the underlying etiology; however, due to the often-unknown cause in many cases, corticosteroid therapy is frequently employed as a first-line treatment. For instance, a 55-year-old patient with acute necrotizing EM showed significant improvement following early corticosteroid treatment [12]. In another case, a patient with EM triggered by eosinophilia induced by anti-tuberculosis medications demonstrated considerable enhancement in prognosis after receiving high-dose corticosteroid therapy [13]. In this report, the patient received high-dose hormonal treatment in combination with immunoglobulin therapy. Subsequent follow-up assessments revealed a significant reduction in myocardial markers.

The clinical manifestations of EM are diverse. Notably, patients presenting with chest pain as the initial symptom are at risk of being misdiagnosed with myocardial infarction. EM can manifest either as acute FM or chronic restrictive cardiomyopathy [14]. It is well recognized within the medical community that EM is intrinsically linked to a range of disease conditions or physiological states, contributing to 11.5% of cases of FM and 18.1% of cases of non-FM [15]. FM is a rapidly progressive inflammatory myocardial disorder, often accompanied by severe hemodynamic disturbances leading to heart failure. As the most severe subtype of myocarditis, FM carries an in-hospital mortality rate as high as 40-80%, even with pharmacologic and mechanical interventions [16]. Early initiation of immunomodulatory therapy, including high-dose glucocorticoids, adequate immunoglobulin administration, and antiviral treatment with neuraminidase inhibitors, is critical for managing FM [17]. For FM patients with concurrent heart failure and cardiogenic shock, high-dose glucocorticoids are currently recommended both domestically and internationally, as they help suppress the immune response and reduce myocardial inflammation and edema [18]. The ECG between DCM and FM could vary. In terms of voltage changes, DCM often shows high voltage in left chest leads and low voltage in limb leads, while FM is mainly characterized by low voltage of the QRS complex. Regarding ST-T changes, DCM mostly presents with ST-segment depression and T-wave inversion, whereas in FM, ST-segment elevation or depression and T-wave inversion can occur. When it comes to conduction blocks, DCM is dominated by intraventricular or bundle-branch block, while atrioventricular block and bundle-branch block are more frequently seen in FM. In terms of arrhythmias, DCM has diverse types, while frequent premature beats and short-burst ventricular tachycardia are more common in FM.

Gammaglobulin, as an immunomodulator, not only clears the virus but also regulates the immune response, reducing inflammation and cytokine secretion. This approach can shorten the disease course and improve patient outcomes. Recent literature from both domestic and international researchers has documented significant success with the combination of high-dose glucocorticoids and adequate gammaglobulin in the early stages of FM [19, 20]. In patients with FM, life support therapy should be initiated as early as possible to alleviate cardiac burden, improve cardiac function, and reduce mortality [21]. Studies have shown that early application of positive-pressure respiratory ventilation and IABP can effectively reduce LV load, improve LV function, shorten hospitalization, and improve survival. Notably, patients receiving IABP therapy exhibited a 29.5% reduction in in-hospital mortality risk compared to those who did not receive this intervention [22]. Encouragingly, ECMO support in FM cases has resulted in discharge survival rates ranging from 60–100% [23], substantially reducing FM-associated mortality. Madershahian et al. [24] posited that combining IABP and ECMO may synergistically benefit patients by using the advantages of both techniques: IABP improves coronary blood supply, reduces LV afterload, and enhances perfusion, while ECMO provides essential cardiac support. This combined approach is hypothesized to yield a synergistic effect, as supported by existing theoretical frameworks. In the case of our patient, sustained ventricular tachycardia, unmeasurable blood pressure, and unstable vital signs persisted despite the administration of high-dose vasoactive agents for hemodynamic stabilization. Consequently, a concurrent implementation of IABP and ECMO was initiated as a critical life support intervention. Among current surgical treatments, heart transplantation remains the primary therapeutic option for DCM. In China, the annual number of cardiac transplants exceeds 300, with a notable success rate of 90–98%. Post-transplantation, recipients commonly enjoy an expected survival of approximately 13 years, aligning with advanced global standards in cardiac care.

The diagnosis between EM and DCM can be tricky sometimes, EM is characterized by eosinophilic infiltration of the myocardium, arising from various causes, including allergic reactions, infections, or hematological disorders, while DCM is characterized by ventricular dilation and impaired contractility, it can be triggered by viral infections, genetic predispositions, or toxic exposures but does not typically involve eosinophilic inflammation. The manifestations of DCM include progressive heart failure manifestations like dyspnea, fatigue, and fluid retention, however, unlike EM, DCM lacks eosinophilia or systemic allergic symptoms. For the therapeutic approaches, both EM and DCM are required to manage

heart failure, including diuretics, beta-blockers and ACE inhibitors, whereas EM requires some specific immunosuppressive therapies such as corticosteroids and immunosuppressants. In this case, myocardial biopsy failed to provide definite evidence of EM, and subsequent specialist echocardiography revealed LV enlargement, with severely reduced coherence in LV wall motion and a lack of overall wall movement coordination. All the above results increased the difficulties of the EM diagnostic. It is reported that EM can feature LV enlargement due to the inflammatory response and subsequent myocardial damage, although the presentation may vary among patients [25, 26]. Differential diagnosis of EM is essential for effective management and treatment, as the clinical presentation can overlap with various other forms of myocarditis, and systemic diseases such as eosinophilic granulomatosis with polyangiitis (EGPA) - a vasculitis linked to ANCA (anti-neutrophil cytoplasmic antibodies) -were considered in the differential diagnosis. For instance, viral myocarditis [27] often caused by viral infections presents with similar symptoms such as chest pain, dyspnea, and elevated cardiac biomarkers, but it typically shows inflammatory infiltrates on biopsy but lacks the eosinophilic component characteristic of EM. Another hypersensitivity myocarditis [28] occurs due to an allergic reaction to drugs or environmental agents, leading to eosinophilic infiltration. While it shares some similarities with EM, hypersensitivity myocarditis often has a clearer association with specific allergens. As for some systemic diseases like EGPA, it is a rare systemic vasculitis characterized by asthma, eosinophilia, and vasculitis affecting multiple organs, including the heart. Cardiac involvement in EGPA can manifest as acute eosinophilic myocarditis or chronic inflammatory cardiomyopathy, while the presence of ANCA antibodies and systemic symptoms can help differentiate it from isolated EM [29]. In the current case, the results of ANCA tests were negative.

Collectively, the patient in this case rapidly developed malignant arrhythmia after admission. Despite the administration of medications, electric defibrillation, endotracheal intubation, ventilator-assisted respiration, and other resuscitative measures, there was no significant improvement. Following the initiation of ECMO and IABP life support, the patient's condition did not reach the expected state of recovery. Ultimately, the patient underwent heart transplantation, and cardiac tissue samples were sent for pathology examination. The diagnostic report revealed a significant presence of eosinophils, supporting the diagnosis of EM. This case underlined the importance of early recognition and treatment of EM, which is crucial to prevent progression to severe heart failure or FM. Clinicians should maintain a high suspicion for EM in patients presenting with unexplained chest pain, especially when accompanied by hypereosinophilia or a history of allergic conditions, and the application of comprehensive laboratory tests and examinations, effective imaging tools such as CMR and PET scans are necessary, and myocardial biopsy remains the gold standard for EM diagnosis, which can confirm eosinophilic infiltration and guide targeted therapy. Besides, a multidisciplinary team involving cardiologists, respirologists, hematologists, and radiologists can enhance diagnostic accuracy and treatment planning.

Abbreviations

DCM	Dilated cardiomyopathy
EM	Eosinophilic myocarditis
FM	Fulminant myocarditis
IABP	Intra-aortic balloon pump
ECMO	Extracorporeal membrane oxygenation
LV	Left ventricular

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None.

Author contributions

M.L. designed the case report, collected the data, drafted the initial manuscript, and revised the manuscript; H.C. reviewed and revised the manuscript; and all the authors approved the final manuscript as submitted.

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Data availability

Data is provided within the manuscript or supplementary information files.

Declarations

Ethics approval and consent to participate

The Biomedical Ethics Committee of West China Hospital of Sichuan University waived ethical approval of the study due to the nature of the case report. Clinical trial number: not applicable. The informed consent of the patient was obtained.

Consent for publication

Written informed consent was obtained from the patient for all the manuscripts that included images and details.

Competing interests

The authors declare no competing interests.

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