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CASE REPORT

A Rare Case of Fulminant Myocarditis Caused by Epstein-Barr Virus Infection in a Middle-Aged Man

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ABSTRACT

Background: **Fulminant myocarditis (FM) is a highly lethal syndrome of abrupt, diffuse cardiac inflammation that precipitates rapid-onset hemodynamic collapse. FM caused by Epstein-Barr virus (EBV) is rare and infrequently reported.**
Methods: **We reported a case of EBV-related FM in a man with respiratory and circulatory failure.**
Results: **The clinical diagnosis was made based on the patient's signs, ECG, ECHO, and significantly elevated levels of inflammatory factors and myocardial injury markers. The etiology was confirmed by mNGS detection of EBV DNA. The patient was successfully treated with VA-ECMO combination with medication therapy.**
Conclusions: **The risk of FM precipitated by EBV infection should not be underestimated. Prompt recognition, targeted therapy, and early circulatory support may avert fatal outcomes.**
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KEYWORDS

fulminant myocarditis, Epstein-Barr virus, VA-ECMO, circulatory failure

INTRODUCTION

Myocarditis is an inflammatory injury of the heart muscle caused by viral infections, allergic reactions, and autoimmune etiologies [1]. Fulminant myocarditis (FM) is a rare, sudden onset, and most severe form of diffuse myocarditis, characterized by rapidly progressive cardiac compromise and a high mortality rate [2]. FM cases with established microbial etiologies are less infrequent [3]. We hereby present a case of a 54-year-old male with FM caused by Epstein-Barr virus (EBV). The patient was successfully treated with veno-arterial extra corporeal membrane oxygenation (VA-ECMO) support and optimal medical therapy.

CASE PRESENTATION

A 54-year-old Chinese man was admitted to the hospital because of fever for 3 days. Laboratory findings reveal-

Table 1. Results of laboratory tests during the patient's treatment.

A

Laboratory test	Reference range	Time						
		Day 1	Day 4	Day 7	Day 9	Day 10	Day 11	Day 14
PCT (ng/mL)	0 - 0.25	1.24	1.21	2.81	1.58	0.77	0.34	0.17
IL-6 (pg/mL)	0 - 7	55.28	626.5	432.4	4.85	3.29	4.44	3.45
WBC (10^9/L)	3.5 - 9.5	14.24	20.94	20.55	10.21	7.00	6.00	13.82
hsCRP (mg/L)	0 - 8	98.82	128.14	157.93	57.34	32.11	19.13	7.93

B

Laboratory test	Reference rang	Day 2	Day 4	Day 7	Day 8	Day 9	Day 10	Day 12	Day 17
BNP (pg/mL)	0 - 100	/	178.89	2,775.17	> 4,911.00	2,243.18	2,661.04	1,105.3	117.93
cTnT (μg/L)	0 - 0.1	0.007	/	/	0.174	0.118	0.071	0.065	0.017

C

Negative	Positive
Blood culture (anaerobic + aerobic)	EBV nucleic acid 2.91 x 10^6 IU/mL (< 500 IU/mL)
Influenza virus A nucleic acid	mNGS EBV reads: 45, relative abundance: 97.7%
Influenza virus B nucleic acid	Rubella virus IgM 29.5 AU/mL (< 20 AU/m)
Human rhinovirus nucleic acid	Rubella virus IgG 61.3 IU/mL (< 9 IU/m)
Adenovirus nucleic acid	T-cell Spot Test
Respiratory syncytial virus nucleic acid	
Mycoplasma pneumoniae nucleic acid	
COVID-19 nucleic acid	
Cytomegalovirus nucleic acid	

A: Inflammatory factors, B: Markers of myocardial injury, C: Laboratory investigation for infectious pathogens.

ed elevated white blood cell count (14.24 x 10^9/L), percentage of neutrophils (85.3%), high-sensitivity C-reactive protein (98.82 mg/L), PCT (1.24 ng/mL), IL-6 (55.28 pg/mL), B-type natriuretic peptide (178.89 pg/mL), fibrinogen (8.12 g/L), D-Dimer (529.28 ug/L). Na (134.7 mmol/L) and thrombin time determination (10.2 second) were decreased. No significant abnormalities were found in the liver and kidney function, myocardial injury markers, or electrocardiogram findings. Chest CT revealed no infectious lesions (Figure S1, panels A and B). After admission, the patient had recurrent high fever, and various inflammatory factors were elevated (Table 1A). The patient was treated with moxifloxacin and piperacillin tazobactam, but the fever continued. On the 4th day after admission, chest CT revealed bilateral patchy pulmonary shadows (Figure S1, panels C and D). Therefore, meropenem was administered as anti-infective therapy.

The patient's condition gradually worsened over the hospitalization course, and on the 7th day, he was admitted to the intensive care unit (ICU) owing to respiratory failure and circulatory failure. Electrocardiography (ECG) revealed a sinus rhythm (91 bpm) and abnormal T waves (Figure 1, panel A). Chest radiography indicated cardiogenic pulmonary edema (Figure 1, panels B and C). An echocardiography (ECHO) demonstrated a reduced ejection fraction of 32% and an enlarged left atrium, accompanied by severe tricuspid regurgitation and moderate mitral regurgitation (Figure 1, panel D). BNP and cTnT levels were significantly increased (Table 1B). The following day, the patient received VA-ECMO along with an intravenous infusion of methylprednisolone (80 mg/d) and immunoglobulin (20 g/day).

Laboratory investigation for infectious pathogens was completed (Table 1C) to assess the etiology of FM. Metagenomic next-generation sequencing (mNGS) reported that EBV was detected in the blood, with 45 EBV nucleotide sequences. In addition, quantitative polymerase chain reaction (qPCR) testing revealed a high nucleic acid load for EBV (2.91 x 10^6 IU/mL). Therefore, antiviral therapy with ganciclovir (250 mg every 12 hours) was administered to reduce the viral load.

On the 13th day, the patient's cardiac function signifi-

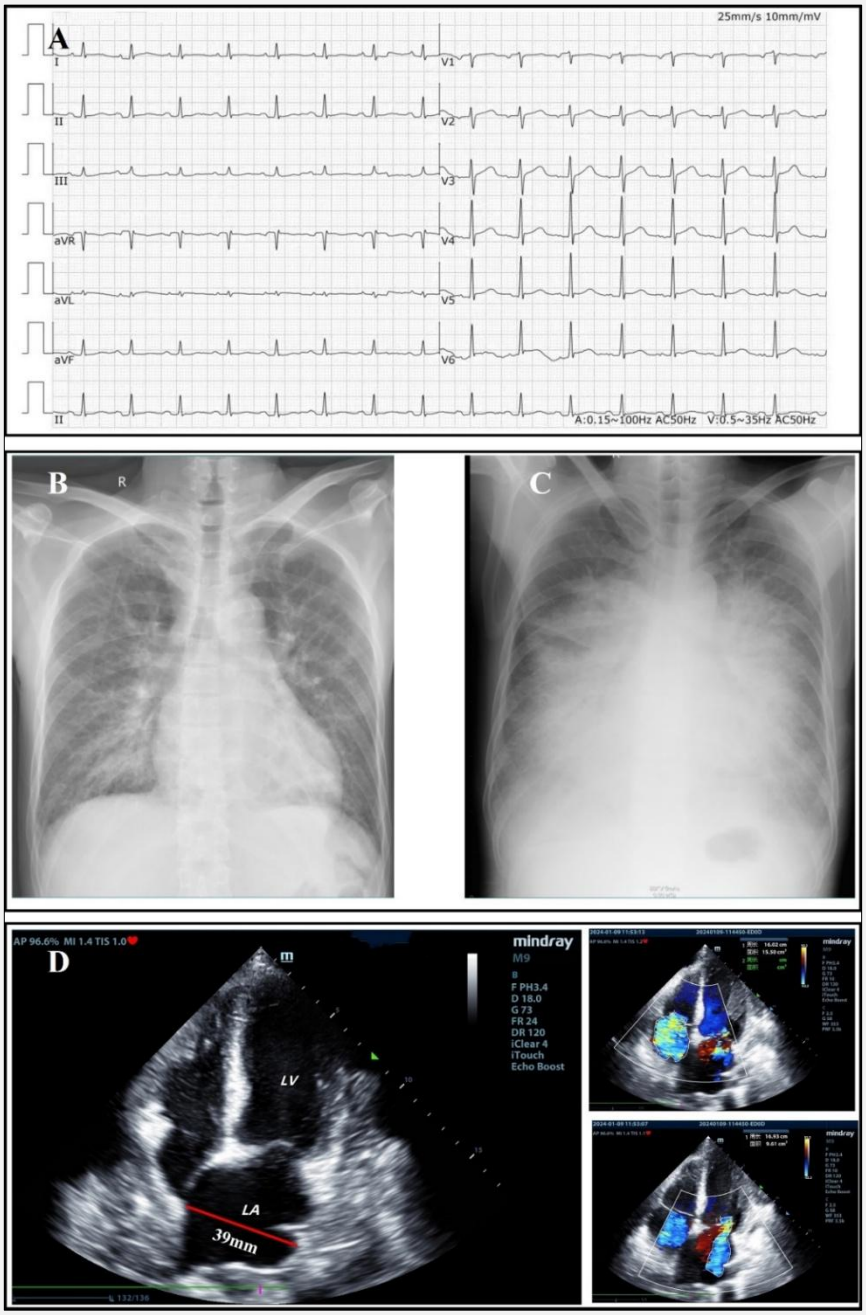

Figure 1. Instrumental findings.

Panel A: Bedside electrocardiogram at the admission to the ICU showed abnormal T waves.
Panel B: On the 7th day of admission, the patient's bedside chest X-ray revealed increased lung texture, Kerley B-line, and cardiac enlargement.
Panel C: On the 8th day of admission, the patient's bedside chest X-ray revealed butterfly sign and cardiac enlargement.
Panels D: Bedside echocardiogram at the time of ICU admission.

cantly improved, with a notable reduction in myocardial injury markers and stable respiratory and circulatory functions. Consequently, the VA-ECOM treatment was suspended, and the patient was transferred to the coro-

nary care unit (CCU) of the cardiology department for further treatment. After 27 days of hospitalization, the patient's general condition gradually improved and he was discharged. The patient was followed up at regular intervals, and at 9 months of follow-up, an echocardiogram showed a standard left heart size (Figure S2), and markers of cardiac injury returned to normal levels (Table S1).

DISCUSSION

FM caused by BEV in the present case is rare. Endomyocardial biopsy (EMB), the gold-standard test for myocarditis, is seldom used because it is invasive [4]. Cardiac magnetic resonance imaging (MRI) is an alternative choice for diagnosing FM if EMB is not accessible [5]. In our case, due to the patient's unstable condition, he did not undergo EMB or MRI. The clinical diagnosis was made based on the patient's signs, ECG, ECHO, and significantly elevated levels of inflammatory factors and myocardial injury markers. Additionally, the etiological diagnosis was made based on the detection of infectious pathogens. Although blood culture did not detect any microorganisms, qPCR revealed a significant number of EBV DNA copies in the blood. Meanwhile, mNGS excluded other potential pathogens, confirming EBV as the sole detectable pathogen in the blood. All those evidences support the direct association between EBV infection and FM.

Currently, the treatment strategy for FM primarily involves symptomatic supportive therapy, including general supportive care, antiviral therapy, immunomodulatory therapy, vasoactive medications, and mechanical circulatory support (MCS) [6]. In rescuing clinically critical FM patients experiencing pump failure, MCS devices, such as ECMO, can provide effective respiratory and circulatory support [7]. Current research suggests that FM patients receiving ECMO exhibit improvements and discharge rates ranging from 55% to 81.8% [8].

In our case, after ECOM implantation combined with immunomodulation, the levels of inflammatory factors (Table 1A) and myocardial injury markers (Table 1B) rapidly decreased and returned to normal on the 5th and 9th day, respectively. It should be noted that antiviral therapy is debated even when viral infection is considered as the initiating factor of FM according to clinical prodromal symptoms [9]. However, we chose ganciclovir for etiological therapy based on the identification of the source of infection by mNGS testing. Our patient survived and had a good prognosis. This indicates that rapid diagnosis, correct medication, and early use of circulatory support therapy may be crucial in preventing fatal outcomes.

Availability of Data and Materials:
The original data and materials presented in the study are included in the article/supplementary material. Further inquiries can be directed to the corresponding author.

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Declaration of Interest:
The authors declare no competing interests.

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Additional material can be found online at:
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