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Dengue-Associated Fulminant Myocarditis Successfully Treated with VA-ECMO and IABP: A Case Report

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1. Abstract

Dengue infection is a major global health problem, with severe forms occasionally leading to multi-organ failure. Cardiac involvement in dengue is increasingly recognized and may manifest as fulminant myocarditis with life-threatening cardiogenic shock. Management is mainly supportive, and the role of mechanical circulatory support remains insufficiently described. We report the case of a 53-year-old woman who presented with fatigue, abdominal distension, and chest tightness without fever. Shortly after admission, she developed severe cardiogenic shock with metabolic acidosis and markedly elevated cardiac biomarkers. Echocardiography revealed severe left ventricular dysfunction with a left ventricular ejection fraction of 27%. Despite high-dose vasoactive agents, the patient's hemodynamic status rapidly deteriorated, requiring Veno arterial extracorporeal membrane oxygenation (VA-ECMO) and intra-aortic balloon pump (IABP) support. Coronary imaging excluded obstructive coronary artery disease. Subsequent serologic testing and next-generation sequencing confirmed infection with dengue virus type 1. After five days of VA-ECMO support, cardiac function improved significantly, allowing successful device weaning. The patient recovered fully and was discharged with normal cardiac function. This case highlights that dengue-associated fulminant myocarditis can rapidly progress to cardiogenic shock and suggests that early initiation of temporary mechanical circulatory support may be life-saving in selected patients.

2. Introduction

Dengue fever is a common mosquito-borne arboviral disease caused by the dengue virus (DENV) in tropical and subtropical regions such as Southeast Asia, Latin America, and sub-Saharan Africa, with more than 100 million new dengue infections every year [1]. Approximately 5% symptomatic dengue cases progress to severe dengue (SD) requiring hospitalization [2]. Hepatic and renal failures were respectively observed in 50% of severe patients. The mortality of severe dengue was reported reaching up-to 30%, with 62% Circulatory failure and 9% of myocarditis required VA-ECMO [3]. The VA-ECMO served as a life-saving bridge, stabilizing the patient and allowing for recovery of cardiac function.

3. Case Report

We present a case of a 53-year-old female sanitation worker who was admitted to our medical center with a seven-day history of abdominal distension, a mild chest rash, fatigue, and chest tightness. Notably, she had no fever during this period. Upon admission to the intensive care unit (ICU), her vital signs were as follows: heart rate 104 beats/min, blood pressure 99/71 mmHg while receiving methoxamine at 4 μg/kg/min and norepinephrine at 0.275 μg/kg/min, arterial pH 7.254, arterial oxygen tension 55.1 mmHg on a fraction of inspired oxygen (FiO_2) of 100%, and serum lactate level of 12 mmol/L.

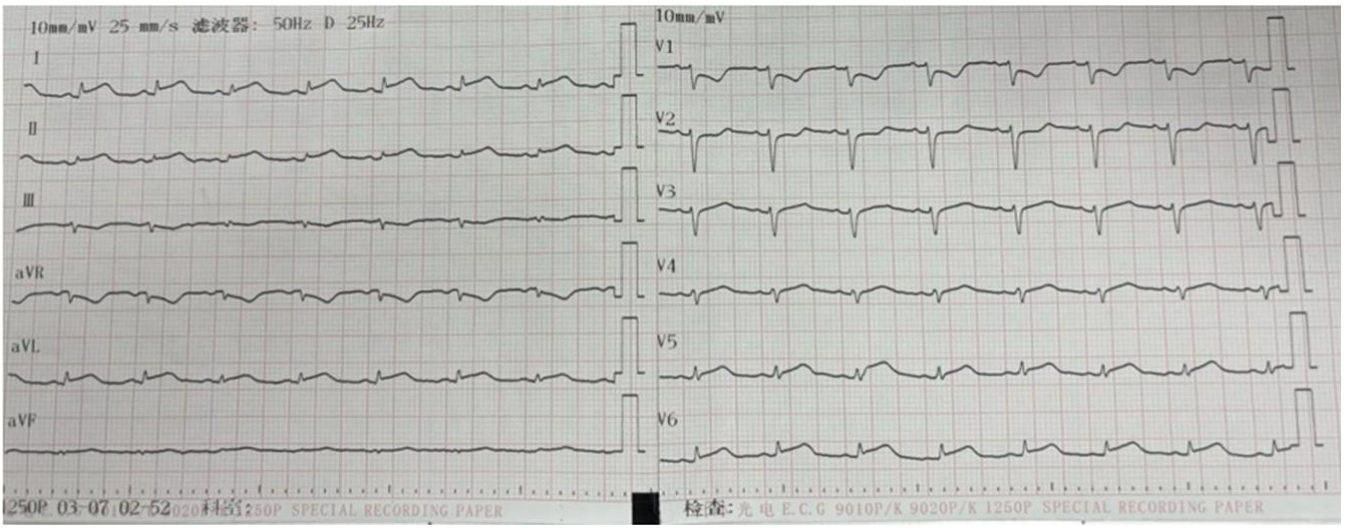

Figure 1: ECG findings suggestive of acute coronary syndrome.

Initial laboratory investigations revealed a serum creatinine level of 66 μmol/L and blood urea nitrogen of 9.2 mmol/L. The white blood cell count was 18.28 × 10⁹/L, hemoglobin 117 g/L, platelet count 414 × 10⁹/L, and hematocrit 0.368. Cardiac biomarkers were markedly elevated, including NT-proBNP (17,269 pg/mL), NT-TnI (1,188 ng/L), high-sensitivity cardiac troponin I (hs-cTnI, 10.13 μg/L), and CK-MB (30.7 ng/L). Liver function tests showed alanine aminotransferase (ALT) of 140 U/L, aspartate aminotransferase (AST) of 211 U/L, alkaline phosphatase (ALP) of 38 U/L, and total bilirubin of 12.1 μmol/L. Electrocardiography demonstrated changes suggestive of acute coronary syndrome (Figure 1). Transthoracic echocardiography revealed severe left ventricular systolic dysfunction, with a left ventricular outflow tract velocity–time integral (LVOT-VTI) of 6 cm (normal lower limit approximately 16 cm) and a left ventricular ejection fraction (LVEF) of 27%.

The patient subsequently developed rapid cardiac deterioration with hemodynamic instability and peripheral circulatory failure, necessitating initiation of VA-ECMO combined with IABP support. The patient had undergone coronary angiography in 2023, which showed no evidence of coronary artery stenosis. During the present admission in 2025, coronary computed tomography angiography also revealed no significant coronary artery disease, effectively excluding acute coronary syndrome. Other potential causes of cardiomyopathy, including immune-mediated disorders and myeloma-related cardiac involvement, were also ruled out. Two days after ICU admission, serological testing for dengue virus was positive. Next-generation sequencing (NGS) of peripheral blood further confirmed infection with dengue virus type 1. Cardiac magnetic resonance imaging was not performed because of hemodynamic instability. As the etiology of cardiogenic shock had been established, endomyocardial biopsy was not pursued.

Before cannulation, bedside vascular ultrasonography demonstrated bilateral femoral vein diameters of approximately 6.1 mm and identified a high bifurcation of the femoral artery near the inguinal ligament. To ensure safe cannulation, a surgical cut-down approach was selected. A 17-Fr arterial cannula and a 21-Fr venous cannula were inserted. The venous cannula was positioned at the junction of the inferior vena cava and right atrium, and the arterial cannula was advanced to a depth of 23 cm.

On the fifth day of VA-ECMO support (ICU Day 5), significant bleeding occurred at the arterial cannulation site (Figure 2). The patient was therefore evaluated for ECMO weaning. Transesophageal echocardiography demonstrated improvement in cardiac function with an ejection fraction of 45%. A reduced-flow trial was successfully performed, during which the patient maintained stable heart rate and blood pressure. The VA-ECMO cannulas were subsequently removed, and definitive hemostasis was achieved under direct visualization.

On the first day after ECMO removal (ICU Day 6), follow-up echocardiography showed further improvement in cardiac function with an LVEF of 60%, and vasoactive medications were gradually tapered. The IABP was removed on ICUday 8. On day 9, the patient was successfully extubated, vasoactive agents were discontinued, and she was transferred to the

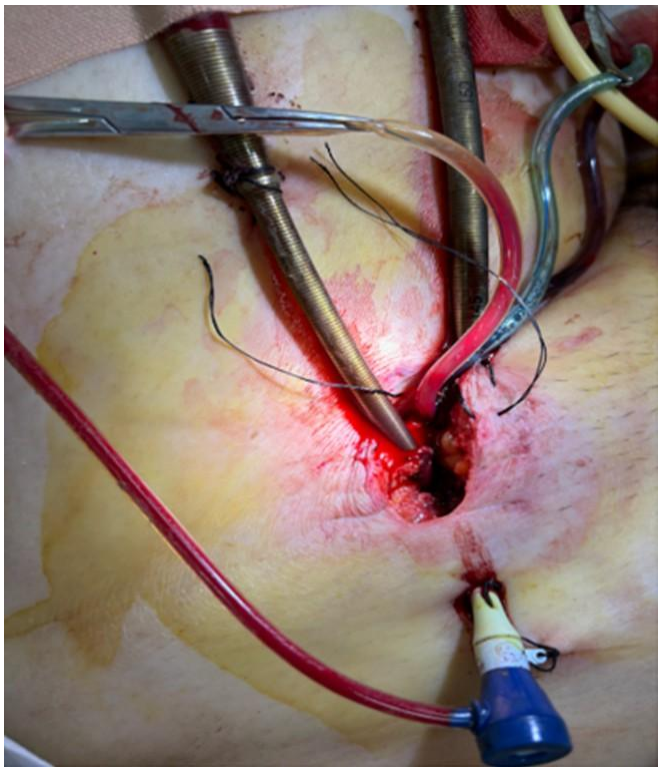

Figure 2: Bleeding at the arterial cannulation site during VA-ECMO support.

cardiology ward for further management. The patient was later discharged in stable condition. At one-month follow-up, she had recovered well and had resumed normal daily activities.

4. Discussion

Dengue fever is an acute infectious disease caused by the dengue virus and transmitted primarily through the bite of infected Aedes mosquitoes. The disease is endemic in many tropical and subtropical regions worldwide. Typical clinical manifestations include fever, myalgia, retro-orbital pain, arthralgia, rash, abdominal pain, diarrhea, and bleeding tendencies, although some individuals may remain asymptomatic.

Severe dengue is defined by one or more of the following conditions: (i) plasma leakage leading to shock (dengue shock syndrome) and/or fluid accumulation with or without respiratory distress; (ii) severe bleeding; or (iii) severe organ impairment. Cardiac involvement is an increasingly recognized complication of severe dengue infection. Among these complications, myocarditis is particularly important because it may progress to life-threatening cardiogenic shock. However, the true incidence of dengue-associated myocarditis remains uncertain. A recent meta-analysis reported that the overall incidence of cardiac events in dengue infection was 27.6%, with myocarditis accounting for approximately 10.9% of these cases 4.

Data on fatal dengue-associated viral myocarditis remain limited. A retrospective analysis of the 2014 dengue outbreak in Guangzhou reported a myocarditis prevalence of 11.28% among hospitalized dengue patients, with only two deaths among 1,728 cases5. In contrast, another retrospective study found that 12.5% of patients who died from severe dengue experienced fatal cardiac complications, including malignant arrhythmias and refractory heart failure6. Although dengue infection is self-limiting in most patients, the development of myocarditis may lead to severe cardiac dysfunction and cardiogenic shock.

Dengue myocarditis often lacks specific clinical manifestations and reliable diagnostic markers, which makes early diagnosis challenging. Most evidence regarding dengue-associated myocarditis is derived from limited case reports. In the present case, the patient did not initially exhibit the typical febrile manifestations of dengue infection. Instead, she presented with nonspecific symptoms such as fatigue and chest discomfort, followed by rapid deterioration of cardiac function leading to cardiogenic shock. Early recognition of hemodynamic instability prompted the timely initiation of mechanical circulatory support, including IABP and VA-ECMO.

4.1. Pathogenesis

The mechanisms by which dengue virus infection leads to myocarditis remain incompletely understood. It has been proposed that dengue virus may induce myocardial injury through mechanisms similar to those observed in coxsackievirus infection, involving immune-mediated damage and cytokine storm–induced myocardial inflammation. Pathologically, this process is typically characterized by lymphocytic myocarditis [7,8]. Most studies suggest that dengue virus primarily infects myeloid lineage cells, including monocytes, macrophages, and dendritic cells, particularly Langerhans cells in the skin. Additional target cells include hepatocytes and Kupffer cells in the liver, as well as vascular endothelial cells. However, direct cardiac tropism of the dengue virus remains poorly characterized [9].

In a fatal case of dengue infection, Salgado et al. [10], demonstrated extensive viral infection involving cardiac tissue, including myocardial endothelial cells and cardiomyocytes. Their findings further suggested that both cardiac and skeletal muscle cells may serve as targets for dengue virus infection10. Similarly, Miranda et al. reported immunohistochemical evidence of dengue virus in myocardial tissue from two fatal cases of refractory cardiogenic shock. Viral staining was observed prominently in monocytes and weakly in the cytoplasm of cardiomyocytes [11].

Recent advances in experimental models have further improved understanding of dengue-associated cardiac injury. Kangussu et al. [12], established an experimental model of dengue virus type 3–induced cardiac inflammation and oxidative stress. Their findings indicated that the virus primarily infected inflammatory monocytes, resulting in myocarditis characterized by infiltration of monocytes and neutrophils surrounding blood vessels12. More recent studies have shown that dengue virus can directly infect cardiomyocytes in interferon α/β receptor knockout mice, leading to viral myocarditis. These findings suggest that impaired immune responses may facilitate viral invasion of myocardial tissue, particularly in immunocompromised hosts [13].

4.2. Therapeutic Considerations

Currently, no specific antiviral therapy is available for dengue infection. Although large randomized clinical trials are lacking, several clinical guidelines and expert consensus statements recommend immunomodulatory therapy, including methylprednisolone and intravenous immunoglobulin (IVIG), in selected cases of severe viral myocarditis [14,15].

Recent studies have suggested that the combination of temporary mechanical circulatory support (t-MCS) and immunomodulatory therapy may improve survival in patients with fulminant viral myocarditis. However, the relative contribution of each therapeutic component remains unclear and requires further investigation16.Acute viral myocarditis represents an inflammatory process of the myocardium caused by viral infection and often presents with highly variable and nonspecific clinical manifestations. Diagnostic confirmation typically relies on cardiac magnetic resonance imaging (CMR) or endomyocardial biopsy. However, in critically ill patients with rapidly deteriorating hemodynamics, these diagnostic procedures are frequently not feasible.

In the present case, IABP therapy was initiated promptly according to the institutional clinical protocol. When the patient developed progressive metabolic acidosis and elevated lactate levels, VA-ECMO was initiated, and the patient was transferred to the ICU for advanced hemodynamic monitoring and supportive care [15,17].

During ECMO support, the patient developed bleeding at the arterial cannulation site. This complication was not related to dengue-associated thrombocytopenia. Previous retrospective studies have shown that cannulation sites represent the most common location of bleeding complications during ECMO support. Identified risk factors include D-dimer levels exceeding 1,000 ng/mL within two days, fibrinogen levels below 2 g/L, platelet counts below 50×10^9/L, arterial pH below 7.12, and body mass index (BMI) below 25 kg/m². In the present case, the patient had two of these risk factors: low fibrinogen levels and a BMI below 25 kg/m² [18-20]. Management included transfusion of red blood cells and fresh frozen plasma, as well as fibrinogen supplementation. Following guideline recommendations, ECMO weaning was performed once clinical and laboratory parameters improved, including sustained lactate levels below 2 mmol/L and stable hemodynamics [21].

5. Conclusion

In summary, dengue fever is generally a self-limiting illness; however, dengue-associated viral myocarditis is a rare but potentially life-threatening complication. A review of the PubMed database identified only a limited number of reported cases, with relatively few successful outcomes. In previously reported outbreaks, dengue virus type 2 has been the most frequently implicated serotype in cases of fulminant myocarditis.

The present case demonstrates that dengue virus type 1 infection can also lead to severe myocarditis and cardiogenic shock. Early recognition of cardiac involvement and timely initiation of mechanical circulatory support, such as VA-ECMO and IABP, may be crucial for patient survival. Further studies are needed to clarify the mechanisms of dengue-associated myocardial injury and to improve the differential diagnosis between dengue-related myocarditis and other causes of cardiac dysfunction, such as septic cardiomyopathy.

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