SVOA Medical Research
e-ISSN 2977-3644

ScienceVolks Open Access

Case Report

A Case of Dengue-Induced Myocarditis Leading to Pulmonary Edema

Faiza[1*], Imtiaz Hussain[1]

[1]Doctor Hospital and Medical Center, Lahore, Pakistan.

***Corresponding Author:** Faiza, Doctor Hospital and Medical Center, Lahore, Pakistan.

https://doi.org/10.58624/SVOAMR.2026.04.004

Received: January 12, 2026

Published: February 09, 2026

Citation: Faiza, Hussain I. A Case of Dengue-Induced Myocarditis Leading to Pulmonary Edema. *SVOA Medical Research* 2026, 4:1, 21-24. doi: 10.58624/SVOAMR.2026.04.004

Abstract

Dengue myocarditis is an uncommon but clinically important complication of dengue fever and may present with acute respiratory distress and dynamic changes in cardiac function. We report the case of a 33-year-old male who presented with high-grade fever and shortness of breath and was found to have pulmonary edema with an initially preserved left ventricular ejection fraction (LVEF). During the critical phase of dengue illness, repeat echocardiography demonstrated a marked but transient reduction in LVEF, consistent with dengue-induced myocarditis. With early recognition and supportive management, the patient achieved complete clinical and echocardiographic recovery. This case underscores the importance of serial cardiac evaluation in dengue patients presenting with unexplained dyspnea or pulmonary congestion.

Keywords: *Dengue fever, Myocarditis, Pulmonary edema, Left ventricular dysfunction*

Background

Dengue fever is a mosquito-borne viral illness that typically presents with fever, myalgia, headache, and thrombocytopenia. Although most cases are self-limiting, severe complications such as plasma leakage, hemorrhage, hepatic dysfunction, and shock may occur. Cardiac involvement, particularly dengue myocarditis, is rare but potentially life-threatening and may result from direct viral myocardial injury or immune-mediated inflammation.

The clinical spectrum of dengue myocarditis ranges from asymptomatic electrocardiographic abnormalities to acute heart failure and cardiogenic shock. Pulmonary edema in dengue patients may result from capillary leak syndrome, excessive intravenous fluid administration, or underlying cardiac dysfunction. Distinguishing between these mechanisms is critical, especially during the critical phase of dengue illness. This case highlights an atypical presentation in which dengue myocarditis manifested with pulmonary edema despite a normal initial echocardiogram. [1-12]

Case Presentation

A 33-year-old male with a history of intermittent smoking and recent statin use (two months) presented with a 3-day history of high-grade fever and progressive shortness of breath. He denied chest pain, orthopnea, paroxysmal nocturnal dyspnea, bleeding manifestations, gastrointestinal symptoms, or abdominal pain.

On presentation, his oxygen saturation was 88% on 2 L/min of supplemental oxygen, blood pressure was 110/70 mmHg, and pulse rate was 104 beats per minute. Respiratory examination revealed bilateral rhonchi. The abdomen was soft and non-tender, and there was no peripheral edema.

Prior to admission, the patient received intravenous ceftriaxone (1 g twice daily for 2 days), oral azithromycin (500 mg daily for 2 days), and approximately 3 liters of intravenous fluids. Dengue NS1 antigen testing performed on day 1 of illness was positive.

Initial investigations revealed a declining platelet count (175 → 154 ×10⁹/L). Chest radiography demonstrated pulmonary fluid overload, which was confirmed on high-resolution computed tomography (HRCT) of the chest. Electrocardiography showed normal sinus rhythm without ischemic changes (Figure 1). Transthoracic echocardiography performed showed normal biventricular systolic function with an LVEF of 67%.

The patient was treated with intravenous furosemide (40 mg), resulting in significant symptomatic improvement. COVID-19 PCR testing was negative.

During hospitalization, he experienced intermittent fever spikes, with a maximum recorded temperature of 101°F. By day 4 he still had oxygen requirement and was still dyspneic, so repeat echocardiography was done which demonstrated a marked reduction in LVEF to 35%, raising suspicion for dengue myocarditis. So, diuretics were continued and heart failure medications were optimized. Within next 3 days his dyspnea got better and oxygen requirement tapered off.

The patient remained hospitalized for three days, encompassing the critical phase of dengue illness. He remained hemodynamically stable with complete resolution of respiratory symptoms. At the request of the patient and family, he was discharged with appropriate medications, counseling, and strict return precautions.

At follow-up one month later repeat echocardiography demonstrated recovery of LVEF to 60% (27 oct), confirming reversible myocardial dysfunction. The patient was asymptomatic, afebrile, and clinically stable.

Laboratory Investigations

Serial complete blood counts demonstrated progressive thrombocytopenia (155 → 144 ×10⁹/L) and leukopenia (4.83 → 3.54 ×10⁹/L). Hemoglobin remained stable at 16.4 g/dL.

Inflammatory markers showed mildly elevated C-reactive protein (17.7 mg/L). NT-proBNP was 242 pg/mL. Renal function was preserved (serum creatinine 1.11 mg/dL; eGFR 87 mL/min/1.73 m²). Liver function tests revealed mild transaminitis (AST 112 U/L, ALT 74 U/L) with normal serum albumin (4.2 g/dL).

Influenza A and B testing and COVID-19 PCR were negative.

Discussion

Dengue myocarditis is an uncommon but increasingly recognized complication of dengue virus infection. Its true incidence is likely underestimated due to subclinical presentations and lack of routine cardiac evaluation in dengue patients. Studies using systematic echocardiographic and electrocardiographic screening have reported cardiac involvement in 9–27% of hospitalized dengue patients, ranging from mild functional abnormalities to overt myocarditis and heart failure [1,2].

The pathophysiology of dengue myocarditis is complex and multifactorial. Proposed mechanisms include direct viral invasion of cardiomyocytes, immune-mediated myocardial injury, and cytokine-induced myocardial depression.

Elevated levels of inflammatory mediators such as tumor necrosis factor-α, interleukin-6, and interleukin-10 have been implicated in myocardial edema, transient systolic dysfunction, and conduction abnormalities [3,4]. Histopathological studies have demonstrated myocardial inflammation with interstitial edema and viral antigens within cardiomyocytes, supporting a role for direct viral myocardial involvement [5].

Clinical manifestations of dengue myocarditis are heterogeneous and nonspecific, making diagnosis challenging. Patients may present with dyspnea, chest discomfort, hypotension, disproportionate tachycardia, pulmonary edema, or arrhythmias, while some remain asymptomatic [6]. Electrocardiographic changes, including sinus tachycardia, ST–T wave abnormalities, atrioventricular block, and atrial or ventricular arrhythmias, have been reported; however, a normal ECG does not exclude myocardial involvement [7]. Similarly, cardiac biomarkers such as troponin and natriuretic peptides may be normal or only mildly elevated, limiting their diagnostic sensitivity [8].

A key feature of this case is the presence of pulmonary edema with an initially preserved left ventricular ejection fraction. In dengue infection, pulmonary edema is most commonly attributed to plasma leakage resulting from increased capillary permeability during the critical phase. However, excessive intravenous fluid administration—often necessary early in the disease—may unmask or worsen underlying myocardial dysfunction [9]. The subsequent decline in LVEF during the critical phase, followed by complete recovery, strongly supports the diagnosis of transient dengue myocarditis rather than isolated fluid overload.

The dynamic and reversible nature of cardiac dysfunction observed in this patient has been described in previous studies, where serial echocardiography revealed transient systolic and diastolic dysfunction that resolved with clinical recovery [2,10]. This highlights an important clinical lesson: a normal early echocardiogram does not rule out evolving myocarditis, and repeat cardiac imaging should be considered in dengue patients with persistent dyspnea, pulmonary congestion, or hemodynamic instability.

Management of dengue myocarditis is primarily supportive, as no specific antiviral therapy exists. Careful fluid management is crucial, balancing the risks of hypovolemia and shock against fluid overload and pulmonary edema. Diuretics may be used cautiously in patients with clinical and radiological evidence of heart failure, as demonstrated in this case [11]. Close monitoring during the critical phase is essential, as rapid deterioration may occur.

Prognosis of dengue myocarditis is generally favorable, with most patients demonstrating complete recovery of ventricular function within days to weeks [1,10]. However, severe presentations, including fulminant myocarditis, cardiogenic shock, and fatal arrhythmias, have been reported, emphasizing the need for early recognition and vigilant monitoring [12].

This case reinforces the importance of maintaining a high index of suspicion for myocardial involvement in dengue patients presenting with unexplained dyspnea or pulmonary edema. Serial cardiac evaluation and judicious fluid therapy are essential to prevent iatrogenic complications and to ensure optimal outcomes.

Conclusion

Dengue myocarditis can present with unexplained dyspnea and pulmonary edema, even when initial echocardiography is normal. A high index of suspicion, serial cardiac evaluation, and meticulous fluid management are crucial for timely diagnosis and favorable outcomes. Early recognition and supportive care can result in complete recovery of cardiac function.

Funding

None

Conflict of Interest

The authors declare no conflict of interest.

References

1. Yacoub S, Griffiths A, Chau TT, Simmons CP, Wills B, Hien TT, et al. Cardiac function in Vietnamese patients with different dengue severity grades. *Crit Care Med*. 2012;40(2):477-83.
2. Kabra SK, Juneja R, Madhulika, Jain Y, Singhal T, Dar L, et al. Myocardial dysfunction in children with dengue haemorrhagic fever. *Natl Med J India*. 1998;11(2):59-61.
3. Martina BE, Koraka P, Osterhaus AD. Dengue virus pathogenesis: an integrated view. *Clin Microbiol Rev*. 2009;22(4):564-81.
4. Wills BA, Oragui EE, Dung NM, Loan HT, Chau NV, Farrar JJ, et al. Size and charge characteristics of the protein leak in dengue shock syndrome. *J Infect Dis*. 2004;190(4):810-8.
5. Salgado DM, Panqueba CA, Castro D, Vega MR, Rodriguez JA. Myocarditis in dengue hemorrhagic fever: a case report and review of the literature. *J Med Case Rep*. 2014;8:379.
6. Miranda CH, Borges MC, Schmidt A, Pazin-Filho A, Rossi MA, Ramos SG, et al. A case presentation of dengue myocarditis showing evidence for dengue virus-induced lesion. *BMC Infect Dis*. 2013;13:268.
7. Gupta VK, Gadpayle AK. Subclinical cardiac involvement in dengue haemorrhagic fever. *J Indian Acad Clin Med*. 2010;11(2):107-11.
8. Lee IK, Lee WH, Liu JW, Yang KD. Acute myocarditis in dengue hemorrhagic fever: a case report and review of literature. *Int J Infect Dis*. 2010;14(10):e919-22.
9. World Health Organization. *Dengue: Guidelines for Diagnosis, Treatment, Prevention and Control*. Geneva: WHO; 2009.
10. Kumar R, Tripathi P, Tripathi S, Kanodia A, Venkatesh V. Prevalence of dengue infection in north Indian children with acute hepatic failure. *Ann Hepatol*. 2008;7(1):59-62.
11. Guzman MG, Harris E. Dengue. *Lancet*. 2015;385(9966):453-65.
12. Horta Veloso H, Ferreira Júnior A, Braga de Paiva J, Faria Filho L. Fulminant myocarditis associated with dengue fever. *Braz J Infect Dis*. 2017;21(3):338-40.