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Case Report

Myopericarditis Manifestation in Post-Dengue Infection Pediatric Patient in Endemic Country: Case Report

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Abstract

Background: Myopericarditis is one of the serious complications of severe dengue manifestations. The increased risk of myopericarditis in severe dengue cases may be associated with the immune system's overactivity. Dengue myocarditis has a higher prevalence in the pediatric population than in adults.

Case Illustration: An 11-year-old boy presented with heart failure syndrome after a recent dengue fever infection. Laboratory test results showed increased Erythrocyte Sedimentation Rate (ESR) and serum Lactate dehydrogenase (LDH), with a slight increase of serum troponin. Echocardiography and Cardiac Magnetic Resonance (CMR) studies confirm myocardial and pericardial involvement. Post-dengue myopericarditis diagnosis was made, and the patient was administered anti-heart failure medication and oral corticosteroid therapy. The patient improved after one month of therapy, as shown by the evaluation of laboratory and imaging criteria.

Conclusions: Myocarditis should be considered if one of the classic triad of myocarditis, such as chest pain, congestive heart failure, and/or arrhythmia, develops in a recent viral infection history. These phenomena suggest that the clinician consider and have a high index of suspicion toward latent complications of post-dengue myocardial and pericardial inflammation syndrome. Hence, early diagnosis and long-term longitudinal surveillance studies are of the utmost importance in dengue-induced myopericarditis, especially in the dengue-endemic area, in order to prevent myocardial remodeling progression to dilated cardiomyopathy.

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Introduction

Dengue is an endemic viral infection transmitted by the bite of the Aedes aegypti mosquito.[1] The most severe clinical manifestation of dengue is dengue shock syndrome, which has high mortality. On the other hand, another severe clinical manifestation of dengue is widely recognized as Expanded Dengue Syndrome (EDS). This spectrum of dengue manifestations can cause life-threatening cardiac complications, for instance, myocarditis and pulmonary artery thrombosis/embolism.[1-2] EDS is often overlooked, making it challenging for clinicians to recognize cardiac complications in dengue infection.[3]

Myocarditis is one of the serious complications of EDS. The prevalence of dengue-induced myocarditis is approximately 21 %, and one in eight mortalities in dengue cases was caused by cardiac events.[1] A meta-analysis reporting the prevalence of myocarditis in dengue is higher in the Asian population (15.2 %) compared to the Latin American population (3.6 %).[4] Dengue myocarditis has a higher prevalence in the pediatric population than in adults. The increased risk of myocarditis in severe dengue cases may be associated with the immune system's overactivity.[1]

Case Illustration

An 11-year-old boy presented to the outpatient cardiology service with chief complaints of chest discomfort and difficulty of breathing for 2 days prior to recent admission. The patient denied any recent chest trauma, cough, or fever at the time of recent admission. The patient's past medical history included Dengue Hemorrhagic Fever (DHF) ten days prior to recent admission. The clinical diagnosis of the prior admission was DHF, with positive anti-dengue IgM, which was documented. The patient was admitted for DHF for about 5 days and discharged home afterward. In the days following discharge from the first admission, facial and leg edema developed. The symptoms were aggravated by abdominal distension due to ascites and generalized edema one day before admission.

The patient's body weight and height were 30 kg and 130 cm, respectively, with a Body Surface Area (BSA) of 1.12 m^2. On admission, vital signs were heart rate 103 beats per minute, blood pressure 126/97 mmHg, respiratory rate 24 breaths per minute, and peripheral oxygen saturation 95%. Physical examination showed minimal pretibial edema and non-remarkable physical signs.

12-lead electrocardiography revealed sinus rhythm, PR interval 120 ms, QRS duration 80 ms with non-specific intraventricular conduction delay, and no ST-T changes observed (Figure 1). Chest X-ray showed cardiac silhouette enlargement with cephalization of the cardiac silhouette (Figure 2). Transthoracic echocardiography showed edematous ventricular wall with increased Interventricular Septum (IVS) thickness and Left Ventricular (LV) Posterior Wall at Diastole (PWd) (IVSd 12 mm, PWd 12 mm) with both Z score for the corresponding patient BSA was +3 which indicated above normal value from patient BSA.5 LV systolic function is mildly reduced with Left Ventricular Ejection Fraction (LVEF) of 48 % and reduced LV Global Longitudinal Strain (GLS) [-12 %] in heart rate 120 beats/min. The reduction in peak systolic strain is especially observed in the lateral LV segment. Heart valves were normal. The pericardial effusion was observed around the LV and at the superior segment of the right atrium (Figure 3)(Video 1).

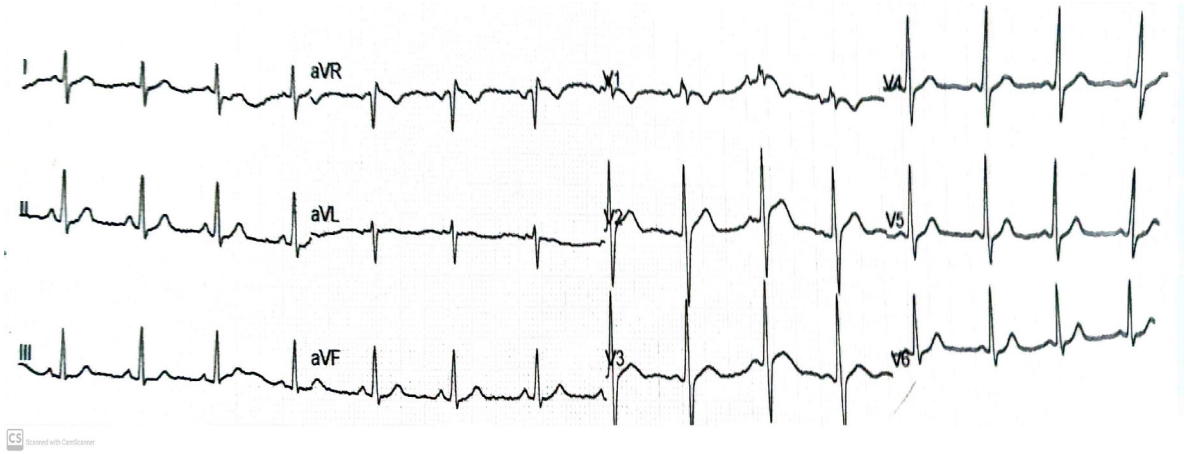


Figure 1. 12-lead electrocardiography.



Figure 2. Chest X-ray.

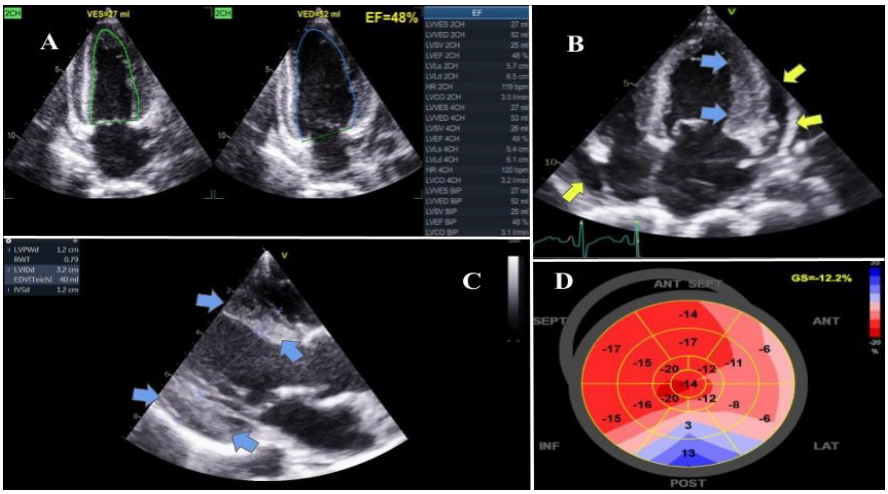


Figure 3. Baseline Echocardiography. A. LV ejection fraction mildly reduced to 48 % (Simpson); 4-chamber view showed minimal pericardial effusion on lateral LV and superior RA (yellow arrow); C. Increased LV wall thickness from interventricular septum and posterior wall which represent myocardial edema (blue arrow); D: LV global longitudinal strain measurement showed reduced segmental peak systolic strain in the posterolateral segment of LV.

Laboratory results showed significant increase in Erythrocyte Sedimentation Rate (ESR) of 120 mm/hour with minimal increase in cardiac troponin I of 0.1 ng/mL, Lactate dehydrogenase level (LDH) also increased to 476 U/L (normal value < 332), reduced serum albumin to 2.8 mg/dL, neutrophil and lymphocyte count were 59.6 and 29.4, respectively with Neutrophil-Lymphocyte Ratio (NLR) of 2.02. Other laboratory parameters were unremarkable, including normal renal function tests, a nonreactive qualitative C-reactive protein test, and a negative qualitative Anti-streptolysin O antibody (ASTO) test.

The differential diagnosis based on these modalities is myopericarditis, which is associated with the recent dengue convalescent phase. The negative qualitative ASTO excludes the possibility of a rheumatic process as the origin. The other possibility is related to idiopathic viral myopericarditis.

Cardiac Magnetic Resonance (CMR) imaging was performed in this patient and revealed a midwall pattern of Late-gadolinium Enhancement (LGE) in the basal-to-mid inferolateral LV wall (Figure 4). The pattern of the LGE from CMR confirmed fibrosis of the midwall LV, which is caused by a non-ischemic etiology; in this case, myocarditis. Therefore, the diagnosis of myopericarditis that is associated with dengue was made. This conclusion was made because of pericardial involvement was more dominant than myocardial involvement, whereas we observed only a minimal reduction in LVEF but an obvious pericardial effusion.

The patient was administered Intravenous (IV) furosemide 30 mg b.d., IV methylprednisolone 4 mg t.i.d., ramipril 2.5 mg o.d., bisoprolol 1.25 mg o.d., and spironolactone 25 mg o.d. The patient was also administered paracetamol for symptomatic therapy. Intravenous administration of methylprednisolone 4 mg three times daily was considered due to an increase in serum LDH that reflected myocardial injury despite a mild troponin rise. The patient was discharged on the sixth day of admission with resolution of dyspnea and congestion symptoms. The patient was then discharged from the hospital uneventfully. Prednisone 2 mg/kg body weight, divided into 4 doses per day, was prescribed as part of the anti-inflammatory regimen at discharge.

Three weeks after discharge, an outpatient visit was scheduled, and the patient was asymptomatic. Laboratory test results showed an improvement in ESR to 39 mm/hour, although it remained above the normal range. Serum albumin normalized to 4.4 mg/dL. A week later, we observed normalization of the ESR to 14 mm/hour. We performed an echocardiographic evaluation, which showed resolution of pericardial fluid and reduced LV myocardial wall thickness (IVSd 8 mm, PWd 8 mm), with Z-scores of +1 and 0, respectively.[5] The LVEF improved to 50 % along with an improvement in LV GLS of [-14 %] at a heart rate of 77 beats/min (Figure 5) (Video 2). The prednisone dose was tapered down gradually every week. The ramipril and bisoprolol were continued for several more months. The periodic follow-ups were documented on the 3rd, 6th, and 12th months post-discharge. The patient's condition was well, with no symptoms remaining.

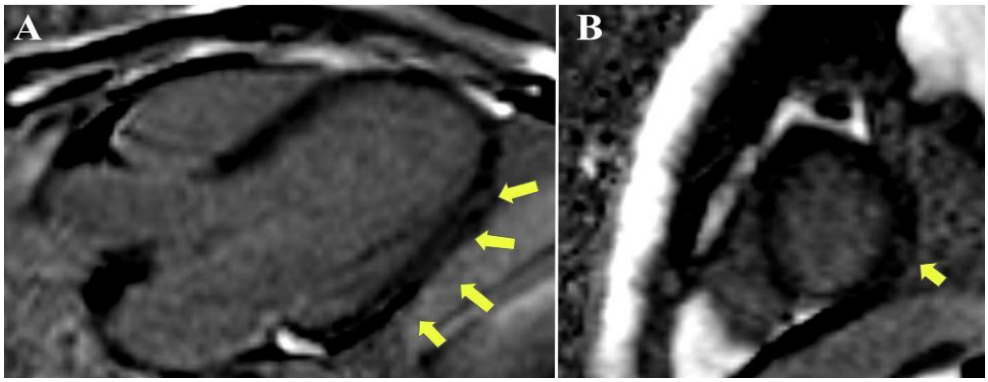


Figure 4. Cardiac magnetic resonance imaging showed LGE (yellow arrow) at: A. 4-chamber view basal-mid lateral LV; B. Short axis views inferolateral LV. [LGE: Late-gadolinium enhancement; LV: left ventricle].

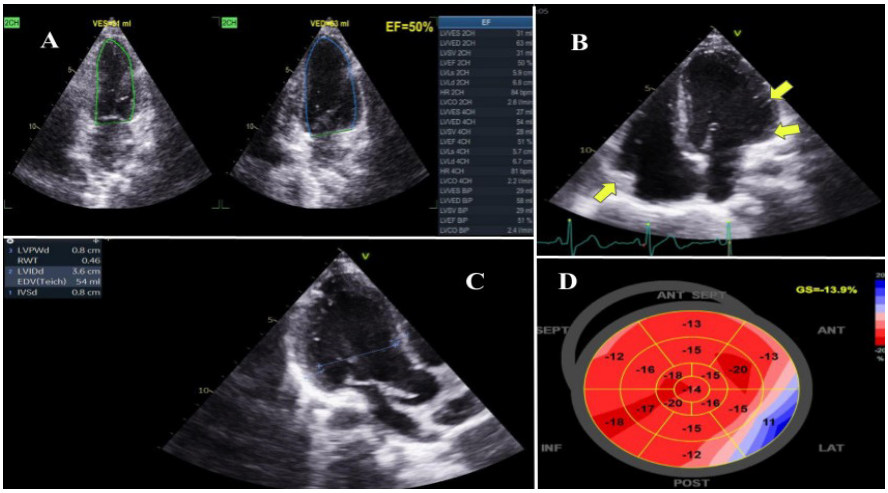


Figure 5. One month follow-up Echocardiography. A. LVEF improved to 50 % (Simpson); B. 4-chamber view showed resolution of pericardial effusion on lateral LV and superior RA; C. Reduced LV wall thickness; D. GLS value assessment showed improvement of peak systolic strain in LV. [LV: Left ventricular; RA: Right atrium; LVEF: Left ventricular ejection fraction; GLS: Global longitudinal strain].

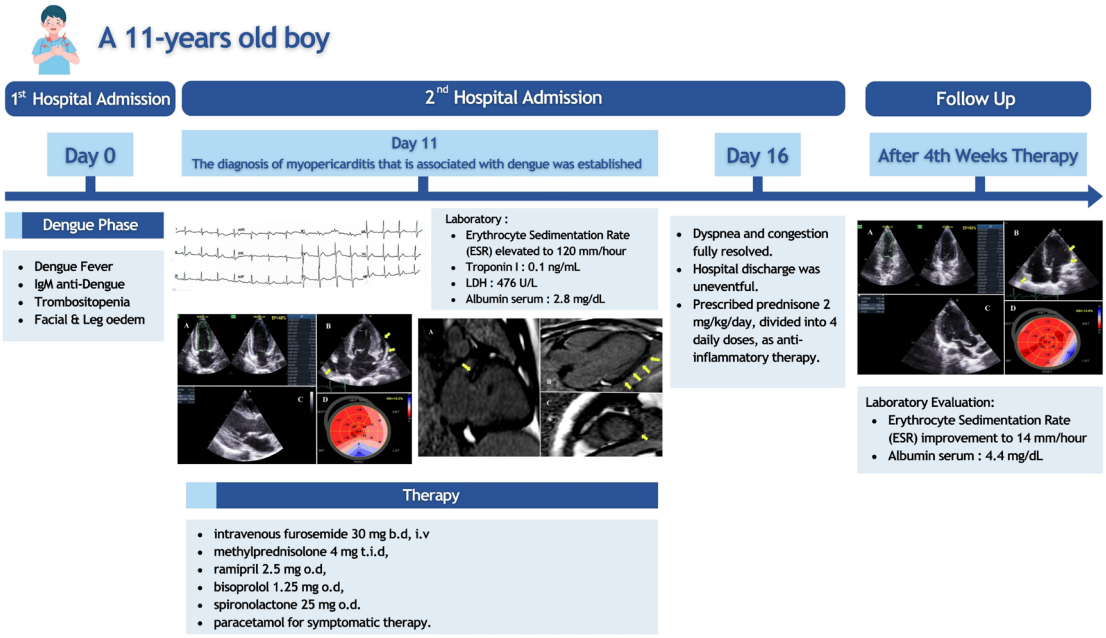


Figure 6. Summary figure.

Discussion

The congestive and dyspnea symptoms in recent viral infection could raise a clinician's suspicion of myocardial and/or pericardial inflammation as the origin.[6] Multimodality imaging, including echocardiography and CMR, could establish the diagnosis and deliver prompt management. Myocarditis should be considered if one of the classic triad of myocarditis, such as chest pain, congestive heart failure, and/or arrhythmia, develops in a recent viral infection history.[3,6] Although in this condition, the differential diagnosis should include other causes of myocarditis, such as toxins, viral infections, tuberculosis, or rheumatic processes. However, in this circumstance, due to a very recent dengue virus infection being documented, dengue-induced myocardial and/or pericardial inflammation becomes the top differential diagnosis.

Echocardiographic findings of increased LV wall thickness, accompanied by reduced systolic function and pericardial effusion in our case, support myocardial and pericardial involvement, respectively, as sources of the patient's symptoms. Our patient demonstrated post-dengue infection myopericarditis in the pediatric population, whereas the patient presented with overt heart failure symptoms. These phenomena suggest that the clinician consider and have a high index of suspicion toward latent complications of post-dengue myocardial and pericardial inflammation syndrome.[7]

However, electrical disturbance was also reported as a dominant manifestation of myocarditis. Khan et al. reported a post-dengue myocarditis case in an adolescent male with complete heart block that necessitated a permanent pacemaker.[2] Dengue myocarditis is also often reported in the adult population, but dengue myocarditis has a higher prevalence in the pediatric population than in adults.[6,9] Furthermore, dengue infection cases that are complicated by myocarditis have a higher in-hospital mortality rate compared to dengue without myocarditis.[6]

Myocarditis in the pediatric population can result in adverse long-term complications if left untreated, especially in the inflammatory phase that potentially progresses into dilated cardiomyopathy.[9] This case report highlighted the importance of clinical awareness and early detection of this pathology, especially in dengue-endemic areas and in resource-limited settings. Furthermore, long-term longitudinal surveillance and follow-up are of the utmost importance in dengue-induced myocarditis to prevent progression of myocardial remodeling to dilated cardiomyopathy.[2]

CMR is important to provide specific information regarding the presence and type of myocardial tissue injury. CMR also plays a fundamental role as the primary imaging modality for the diagnosis of acute myopericarditis.[2,8,10-11] Degiovanni et al. reported that the reduction of LV GLS by speckle-tracking echocardiography has a high correlation

with myocardial edema and LGE on CMR. In addition to the high correlation with LGE-positive myocarditis, the study found that segmental GLS reduction correlates strongly with the corresponding LGE location on CMR in 74% of patients.[10] Other studies reported that myocarditis LGE distribution is frequently found in the inferior and inferolateral territory.[11] Our case CMR finding also showed LGE distribution consistent with the segmental reduction pattern of GLS echocardiography, predominantly seen in the inferolateral LV (Figure 3).

Moreover, Meindl et al. reported that among the preserved LVEF myocarditis population, a lower mean GLS was observed in myocarditis than in the healthy control group. This study also observed that the myocarditis group had thicker diastolic LV PWd than the control group.[11] This finding was also observed in our case. The more sensitive, non-invasive, and quick-to-use strain-derived echocardiography, such as GLS, can be an additional diagnostic modality for identifying early changes of myocardial systolic function and for rapid detection of myocarditis.[2] This approach can become a helpful tool for early diagnosis in clinically suspicious myocarditis cases in an endemic area with a resource-limited setting, especially when the CMR modality is not available. However, given that GLS is heart-rate-dependent, the interpretation of GLS measurements from different heart rates should be done with caution to prevent misinterpretation.

Immunosuppressive therapy, which has a role in reducing myopericardial inflammatory burden, in addition to standard cardioprotective anti-heart failure therapy, was the mainstay therapeutic approach in our case, although this approach is still controversial.[12] General consensus outlined that not all myocarditis cases need an immunosuppressive approach. Immunosuppressive therapy is restricted to a particular type of myocarditis, such as eosinophilic, giant cell, sarcoidosis, immune checkpoint-related, and autoimmune-related myocarditis. Unfortunately, a definitive myocarditis diagnosis requires an endomyocardial biopsy-proven approach and a viral antigen-negative test for immunosuppressive suitability, which were not feasible at our center.[6] This has become our case limitation.

However, expert consensus stated that IV methylprednisolone boluses for 3 days, followed by oral prednisone 1 mg/kg/day, then followed by tapering off with steroid-sparing immunosuppressive drugs, showed an association with clinical benefit.[6] As demonstrated in this case, the patient responded well to pharmacological management, as seen in the resolution of pericardial fluid, myocardial edema, and a significant reduction in ESR as an inflammatory marker within one month after pharmacological management.

Conclusion

We reported a pediatric patient with acute myopericarditis in the setting of a recent dengue infection. The recent timeline of dengue infection makes dengue more likely as the etiology and should raise clinicians' suspicion for myopericarditis. Early recognition is a very important measure, especially in dengue-endemic areas and in resource-limited settings. Long-term surveillance studies of the utmost importance in this situation to prevent progression to dilated cardiomyopathy.

List of Abbreviations

ASTO	Anti-streptolysin O
BSA	Body Surface Area
CMR	Cardiac Magnetic Resonance
DHF	Dengue Hemorrhagic Fever
EDS	Expanded Dengue Syndrome
ESR	Erythrocyte Sedimentation Rate
GLS	Global longitudinal strain
IV	Intervenous
IVS	Interventricular Septum
LDH	Lactate dehydrogenase
LGE	Late-gadolinium Enhancement
LV	Left Ventricular
LVEF	Left Ventricular Ejection Fraction
NLR	Neutrophil-Lymphocyte Ratio
PWd	Posterior Wall at Diastole

Ethical Clearance

Not applicable.

Publication Approval

All authors consent to the publication of this manuscript.

Authors' Contributions

All authors have made a significant intellectual contribution to the manuscript according to the criteria formulated by the International Committee of Medical Journal Editors.

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Conflict of Interest

None.

Availability of Data and Materials

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Generative AI and AI-Assisted Technologies in the Writing Process

The authors declare that no artificial intelligence (AI) was used in the writing, analysis, or preparation of this manuscript

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