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Case Report

Dengue-Associated Acute ST-Elevation Myocardial Infarction: Navigating Anti-Thrombotic Therapy in Severe Thrombocytopenia

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Abstract:

Dengue fever is a mosquito-borne viral disease transmitted by Aedes mosquitoes particularly Aedes aegypti. There are 4 distinct serotypes (DEN1, DEN2, DEN3, and DEN4) causing dengue fever. The clinical manifestations of dengue fever are variable- fever, headache, body ache, retro-orbital pain, rash, & joint pain. It has a wide range of clinical manifestations, ranging from asymptomatic disease to a more complicated form -dengue hemorrhagic fever or dengue shock syndrome (DHF/DSS), which is characterized by increased vascular permeability, thrombocytopenia (platelets <100,000), bleeding tendency, and sometimes circulatory shock. Atypical manifestations, such as central nervous system, renal, and cardiac involvement, are increasingly being reported. Bleeding manifestations in dengue are well known, but thrombotic events are uncommon. Only a few case studies have previously documented the effects of dengue fever on the cardiovascular system, including conduction abnormalities, hypotension, arrhythmias, myocarditis, cardiomyopathy, and, infrequently, myocardial infarction. Some cardiac manifestations in dengue hemorrhagic fever, such as atrioventricular blocks, sinus node dysfunction, and ectopic ventricular beats, are noticed. The separation between myocarditis and myocardial infarction is necessary, for which echocardiography and coronary angiography can be helpful. ST-elevation myocardial infarction during dengue is rare but poses an important management dilemma. We present three patients with dengue fever with ST elevation myocardial infarction, and our experience in the management of those patients. We suggest individualized case-based management of acute coronary syndrome in dengue, which should be guided by the degree of thrombocytopenia, bleeding risk & comorbidities.

Key words: *STEMI, Dengue, Thrombocytopenia*

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Introduction:

Most people suffering from dengue have mild symptoms, but some can suffer from life-threatening complications like dengue hemorrhagic fever or dengue shock syndrome. A small percentage of patients suffer from cardiac, renal, or CNS involvement. Dengue-associated cardiac involvement has various presentations, such as myocarditis, myocardial infarction, arrhythmia, and A-V conduction block. Acute coronary syndrome in a dengue patient is a rare presentation, and it's challenging to manage due to various factors like thrombocytopenia, plasma leakage, coagulopathy, and dengue-induced hepatitis. Here, we present three cases of dengue fever complicated by ST elevation myocardial infarction, each with a distinct presentation and management approach.

CASE 1:

A 50-year-old man, nondiabetic, normotensive, was admitted during a dengue outbreak because of 4 days of intermittent high-grade fever, headache, arthralgias, and myalgias. He had taken paracetamol orally for relief of chills that accompanied a temperature of 105°F. Additionally, he had 5 to 6 loose stools during the past 2 days. He had a 15-pack-year smoking habit.

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Components	On Admission day	Day 2 after admission	DURING DISCHARGE
Vital Signs	BP- 100/70 mmHg, Temp -102°F,	BP- 90/70 mmHg	BP- 130/80 mmHg
Investigation	NS1-positive, Haemoglobin-16 g/dl, hematocrit - 50% , TC 11630, Neutrophil 53%, Platelet 96,000/mm^3.	Platelet -44,000/mm^3, HCT-54%	Platelet count - 190000/mm3
	ECG -normal	*ECG - ST-segment elevation in leads V1-V3, depression in leads I, aVL, V5, and V6, and Q-wave abnormalities in leads V2-V3. *Troponin-I -5.12 ng/mL, (cut-off >0.4 ng/ml) *Transthoracic echocardiography - ejection fraction of 40% due to hypokinesia with thinning of the anterior and anteroseptal walls. *NT-proBNP - 1072.6 pg/mL.	
	AST-120 U/L , ALT - 75 U/L RBS- 6.6 mmol/L CRP- negative		

About 48 hours later, the patient complained of the sudden onset of severe central chest pain, profuse vomiting, and sweating. 12 lead ECG was done & it showed anteroseptal STEMI. The patient was conservatively managed with loading doses of aspirin, atorvastatin, nitroglycerin, and ramipril. The patient remained under strict observation in a high dependency care area. The packed cell volume was measured every 3 hours, and a point-of-care ultrasound was performed every 8 hours to assess plasma loss. Complete blood counts were performed to assess platelet levels. Aphaeretic platelets were made readily available for emergency transfusions if needed. The hospital stay was uneventful, with no bleeding or hemodynamic instability. The platelet counts showed progressive improvement, with a count of 190,000/mm^3 at hospital discharge. The patient was discharged on antiplatelet therapy, a statin, a beta-blocker, an ACE inhibitor, and spironolactone. The ECG showed improvement from ischemic changes. At subsequent follow-ups, the patient remained stable and experienced no complications. Echocardiography showed improvement in the LVEF to 50%.

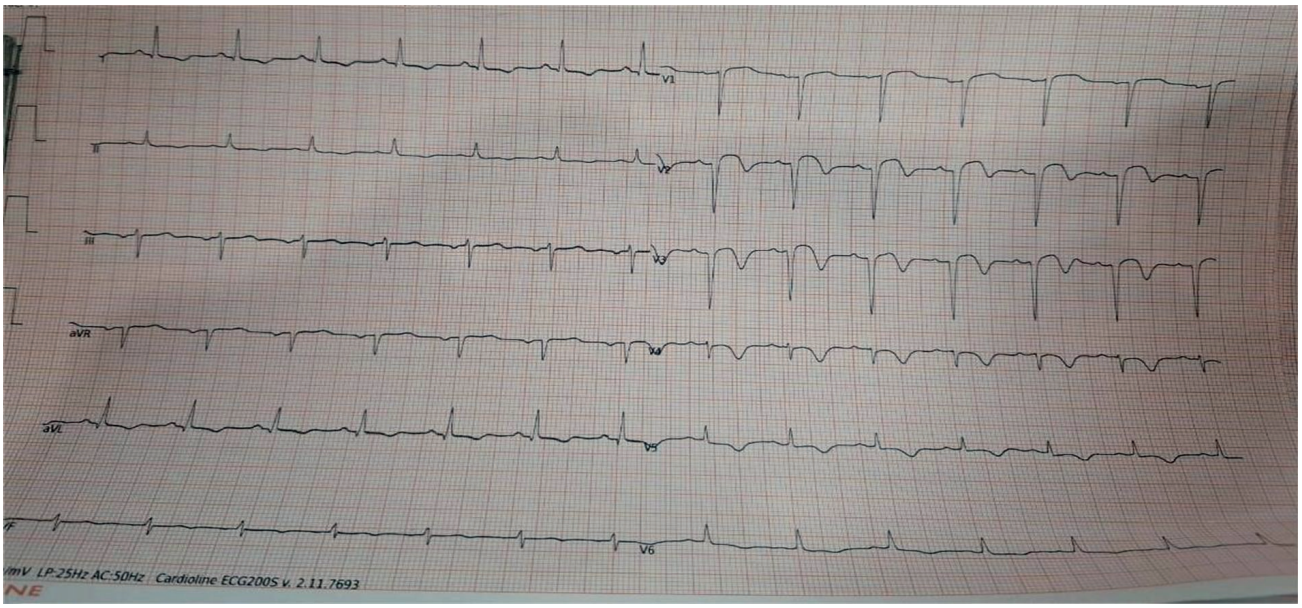


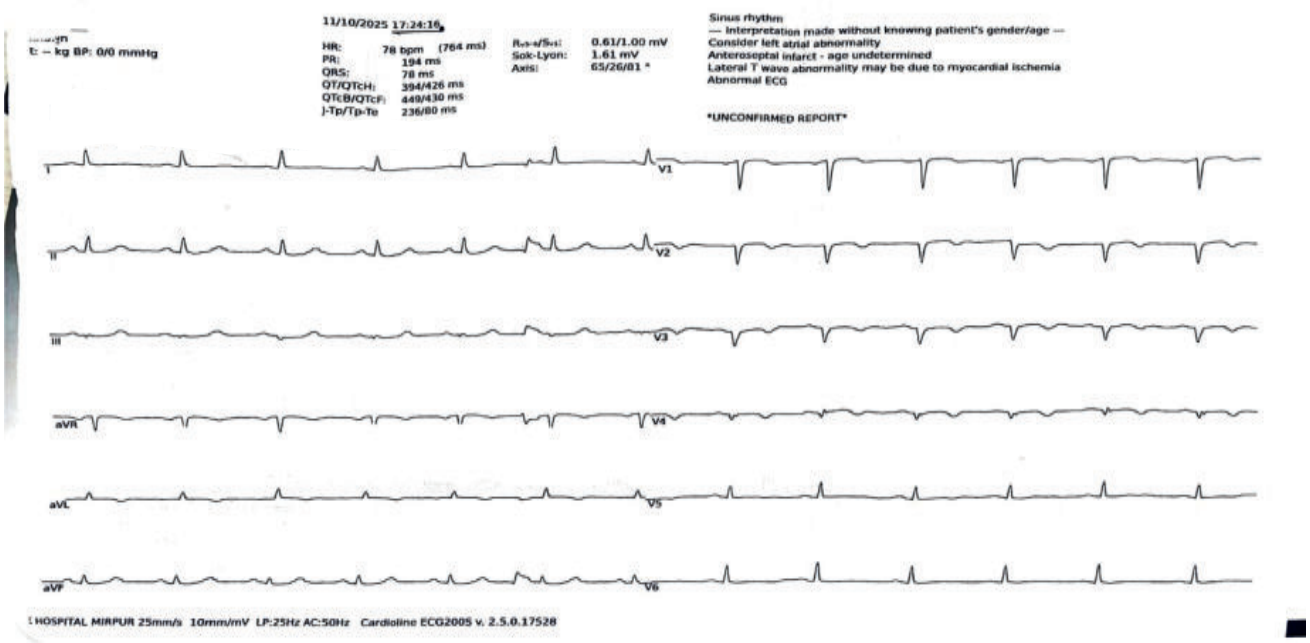
Figure 1: 12 lead ECG was done & it showed anteroseptal STEMI

Case 2:

A 60 -year-old man was admitted with high-grade fever with chills for 3 days. He had associated arthralgia, myalgia, and loose stools in several episodes. He had hypertension for 10 years and a past ischemic stroke.

Components	Admission day	Day 1 after admission	During discharge
Vitals	BP -140/85 mmHg, Temp - 104°F, pulse 116/min	BP-100/60 mmHg Pulse rate 96/min	
Investigation	RT PCR for dengue and chikungunya - positive, Hb -14.5 g/dl, hematocrit - 45 %, TC-3940, Neutrophil 35%, Platelet 80,000/mm^3.	Platelet -18,000/mm^3, HCT-50%	Platelet-221000 /mm^3.
	ECG -normal	*ECG - ST elevation in V1-V2, and T inversion in anterolateral leads (V1-V4), Q wave in V2-V3. *High sensitive Troponin I - 72 ng/L (<57) *NT pro BNP 986 pg/ml. *Echocardiogram -EF 35% with anterior and antero-septal hypokinesia.	
	AST-145 U/L , ALT - 85 U/L RBS- 12.3 mmol/L CRP- negative Albumin 3.2g/dl	AST-345 U/L , ALT -120 U/L HbA1c- 7.5%	

On day 3, he complained of sudden chest pain and chest tightness, shortness of breath & sweating. Anterior STEMI was treated cautiously with only aspirin, atorvastatin, ramipril, & beta blocker. During his hospital stay, fluid was given cautiously to prevent fluid overload, and his platelet count began to increase. With proper fluid management, his vital signs improved. Serial ECGs revealed Q-wave development. Dual antiplatelet medication was started when the platelet count was above 100000/mmb. During discharge, his platelet count was 22/000/mmb. An outpatient coronary angiography was scheduled with a cardiologist review. Repeat echocardiography showed an improved ejection fraction of 45% 4 weeks later.



Case 3:

A 42-year-old male patient came to the emergency department with symptoms of high grade fever, lethargy, and bleeding gums for the last 3 days. The patient had no previous history of any cardiovascular disease, diabetes mellitus, hypertension, or other chronic illness. Also, there is no family history of early coronary artery disease. The patient weighed 74 kg.

Components	Admission day	22 hours after admission	During discharge
Vitals	BP -90/70 mmHg, Temp -104°F, pulse 120/min, Respiratory rate 22 breaths/min	BP-110/90 mmHg Hb - 15.59 m/dL	BP-130/80 mmHg Pulse - 88/min
Investigation	Dengue NS1 - positive , Hb -13.9 g/dl, hematocrit - 43% , WBC- 4470/mm^3, Neutrophil 75%, Platelet 35,000/mm^3.	Platelet -18,000/mm^3, HCT-50% Hematocrit WBC - 3500/mm^3	Hematocrit- 37.1%, WBC - 13200/mm^3 Platelets - 175000/mm^3
	ECG -normal	*ECG- ST segment elevation in leads I, aVL, and V2 to V5, and reciprocal ST segment depression in leads II, III, and aVF. *The high sensitivity troponin I (>25,000ng/L). *NT-proBNP - 10,800 pg/mL, *Transthoracic echocardiography -regional wall motion abnormalities in the anteroseptal wall and hypokinesia in the inferior wall with ejection fraction of 38%	
	AST-180 U/L , ALT - 82 U/L	AST-540 U/L , ALT- 380 U/L	
	RBS- 18.31 mmol/L	HbA1c- 9.2%	
	CRP- negative		
	Albumin 3.2 g/dl	Albumin 2.5 gm/dl	

With regard to findings of fever, thrombocytopenia, mucosal bleeding, and positivity for NS1 antigen, it was determined that this patient had dengue hemorrhagic fever (DHF). Care for this patient consisted of conservative treatment using IV fluids prudently, antipyretics for fever, and insulin for hyperglycemia.

About 22 hours post-admission, he began to experience sudden chest pain and shortness of breath 12 lead ECG was done & it showed antero-lateral MI. The patient was started on aspirin, atorvastatin, ramipril, and metoprolol. Percutaneous primary coronary intervention was withheld due to the patient's severe thrombocytopenia with a high risk of active bleeding. Supportive care consisted of oxygen therapy and the intravenous administration of 25% human albumin together with furosemide (40mg) for pulmonary edema. Intravenous albumin and diuretic therapy were continued for the next two days. In the days that followed, the patient became afebrile and clinically improved. The patient remained hemodynamically stable and clinically improved, and thus allowed discharge and instruction to go for elective coronary angiography upon recovery.

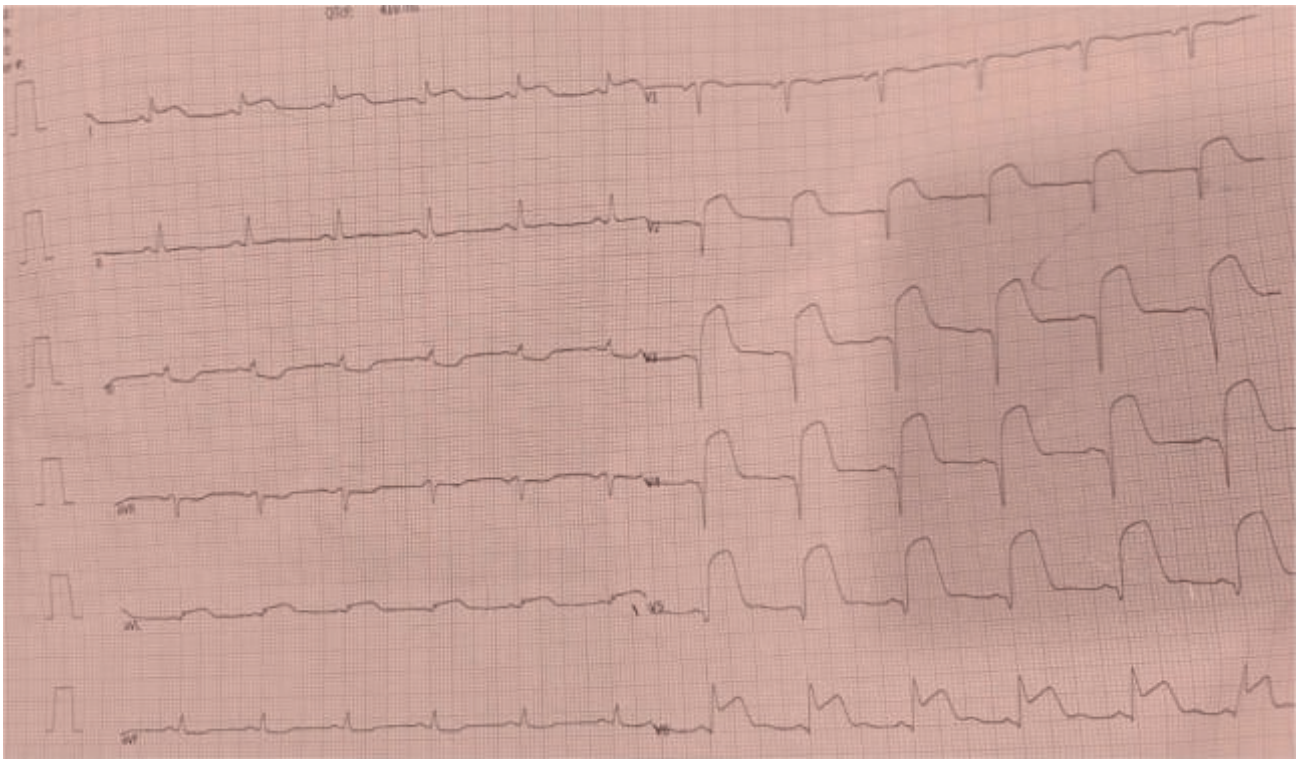


Fig.-3 : 13 lead ECG showing Anterolateral STEMI

Cardiac manifestations of dengue infection have long been acknowledged and it ranges from conduction disturbances, shock, arrhythmias, myocarditis, pericarditis, heart failure, and cardiomyopathies. Myocardial infarction as a complication of dengue fever is even more uncommon and has been documented by the currently available literature only through sporadic case reports. Two cases of dengue fever complicated by acute ST-elevation myocardial infarction (STEMI), which were managed conservatively, were reported by Wijayabandara et al.[1] A case of concurrent STEMI involving multiple vessels was documented by the Taiwanese study by Lin et al.[2] and involved subtotal occlusions of the left anterior descending and left circumflex arteries on coronary angiography due to dengue infection-induced inflammation that led to rupture of pre-existing unstable atherosclerotic plaque lesions in the prothrombotic environment favored by the infection.

The pathophysiological mechanisms underlying the cardiac manifestations of dengue infection include extensive endothelial dysfunction, increased vascular permeability, and micro-myocardial injury, including myocardial necrosis and inflammation. [3] Inflammatory mediators such as cytokines, tumor necrosis factor, and free oxygen radicals have been implicated in the etiopathogenesis of myocarditis and may progress to idiopathic dilated cardiomyopathy in some instances. [3]

Electrocardiogram, cardiac biomarkers, echocardiogram, coronary angiogram, and cardiac magnetic resonance imaging are helpful diagnostic procedures in determining the presence of cardiac complications in dengue infection. The echocardiogram and coronary angiogram are important procedures for distinguishing acute myocarditis from acute myocardial infarction.[4] But the execution of the coronary angiogram in patients with dengue infection can be difficult because of thrombocytopenia and the hazards associated with bleeding complications from invasive procedures.[1,5] Endomyocardial biopsy is the definitive method for diagnosing acute myocarditis; however, it is frequently limited in clinical application due to its invasiveness. Cardiac magnetic resonance imaging is a valuable tool for diagnosis in selected cases in a noninvasive manner.[5]

There are certain clinical and radiological features that suggest myocardial infarction rather than myocarditis, including ST-segment elevation in anterior precordial leads associated with reciprocal ST-segment depression in inferior leads, evolution of pathological Q waves, and wall motion abnormalities detected by echocardiography.

Conclusion

Patients with dengue fever can present with a variety of cardiac problems, and early detection and optimal therapy are critical since the disease can quickly progress to a fulminant stage. Because there is no data to support any

therapy method in this subgroup of patients, it is critical to distinguish acute myocardial infarction from dengue myocarditis and develop a personalized management strategy. Further investigations and enhanced clinical awareness are important for refining diagnostic techniques and therapeutic strategies to manage dengue with myocardial infarction effectively.

Ethical approval

Our institution does not require ethical approval for reporting individual cases or case series.

Informed consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Declaration of conflicting interests

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