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Severe Dengue Presenting as Acute Myocardial Infarction in a Young Patient: A Case Study

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Abstract

Background: Dengue virus infection is a worldwide arboviral hazard that is typically characterized by fever, aches, and, possibly, excessive plasma loss and blood loss. Though subclinical cardio-involvement is a frequent occurrence, it is a noteworthy and daunting clinical challenge to manifest in a syndrome so indistinguishable with acute myocardial infarction (MI). The case highlights the important overlap between cardiology and infectious disease.

Case Presentation: A 32-year-old previously well male patient, having lived all his life in an area with endemic dengue, presented with acute crushing chest pain, electrocardiogram (ECG) showing ST-segment elevation in anteroseptal leads, and a highly elevated high-sensitivity cardiac troponin I level (15.2 ng/mL), which meet the diagnostic criteria of ST-elevation MI (STEMI). He has given a recent history of febrile disease. Physical evaluation demonstrated relative bradycardia and lab results were important in terms of thrombocytopenia and leukopenia. An increase in positive dengue NS1 antigen was a sign of acute infection. Emergency coronary angiography showed normal coronary arteries, which exclude the possibility of atherosclerotic disease. The eventual diagnosis was severe dengue myocarditis and faking STEMI. The management was changed to supportive care and the symptoms were completely resolved and the cardiac function started recovering significantly.

Conclusions: The case demonstrates that dengue virus may lead to the development of a severe acute syndrome of myocardial injury that is clinically similar to STEMI. There is a great possibility of overdiagnosis and unnecessary anticoagulation or invasive coronary surgery, and this could prove detrimental in the backdrop of thrombocytopenia and a systemic viral disease. In young, endemic, patients who have STEMI-like manifestations and a febrile prodrome, greater clinical suspicion of dengue myocarditis is urgent. Coronary angiography is the gold standard of a definitive diagnosis and appropriate and possibly life saving management.

Keywords: dengue fever, myocardial infarction, myocarditis, ST-elevation, troponin, coronary angiography, viral cardiomyopathy, thrombocytopenia, bradycardia, type 2 myocardial infarction

Introduction

Dengue virus is a single-stranded RNA flavivirus that is mostly carried by the Aedes aegypti and Aedes albopictus mosquitoes and is one of the fastest spreading arboviral diseases in the world. The estimated number of symptomatic infections is 100 million per year by the World Health Organization (WHO) and almost half of the global population is currently at risk (1). Dengue has a wide clinical spectrum, with a self-limiting non-specific febrile state on one side, and severe and life-threatening conditions, which include a plasma leakage, fluid accumulation, respiratory, severe bleeding, and organ dysfunction on the other side (2, 3).

An increasing amount of evidence is however confirming that the heart is a major target organ in dengue infection. This involvement has been given the name dengue myocarditis and studies have found prevalence rates of subclinical heart dysfunction in up to 50-60% of hospitalized patients when evaluated with sensitive measures such as echocardiography and cardiac biomarkers (4, 5). The clinical manifestation of cardiovascular involvement may be arrhythmias (sinus bradycardia is most frequent), pericardial effusion, and short-term left ventricular systolic dysfunction (6, 7).

The syndromic presentation of dengue carditis that is the rarest and most dramatic is a syndrome that resembles an acute ST-elevation myocardial infarction (STEMI). The patients come with typical ischemic chest pain, stereotypical ST-segment elevation on ECG, and acute increase in cardiac-specific troponins (8, 9). This poses an acute medical emergency with the usual time-sensitive guideline being emergency coronary angiography and primary percutaneous coronary intervention (PCI)(10, 11). But, in this special case, the pathophysiology is not the rupture of atherosclerotic plaque or thrombosis of the coronary arteries, but a multifaceted interaction of direct viral cytotoxicity, dysregulation of the host immune system and the dysfunction of the microvascular system (12, 13).

The case study is a detailed description of a young and healthy male with the development of STEMI-mimicry as a result of severe dengue infection. We present a detailed discussion of the clinical presentation, diagnostic workup, and treatment, and then thoroughly discuss the pathophysiology, review the available literature, and key learning points to clinicians working at this difficult intersection of infectious disease and cardiology.

Material and Methods

Study Design

Patient Presentation

This case report is of a 32-year-old male construction worker who came to the emergency department (ED) by ambulance with the chief complaint of severe, crushing substernal chest pain, which onset happened suddenly 4 hours ago. He termed the pain as heavy weight in his chest, which spreads to his left arm and jaw, which is accompanied with profuse diaphoresis, nausea and feeling of doom. Rest did not help his symptoms.

When further examined, he complained of a 3-day history of high-grade fever (up to 39.5degC), intense generalized myalgia (retro-orbital pain and break-bone pain), and frontal headache. These prodromal symptoms had partially healed the previous day of the development of chest pains. He refused to answer questions about any recent out-of-town travel, as his home city is a dengue-stricken urban area. He did not have a history of this type of chest pains, palpitations, or syncope.

History Past medical, social, and family history.

The patient did not have any important medical history and did not take any regular medications. He was himself a non-smoker and did not use illegal drugs or drink too much. No family history of premature coronary artery disease, hyperlipidaemia, or sudden cardiac death. His social background was not outstanding and he resided in a densely inhabited neighbourhood where the stagnant water and mosquitoes were known to be a problem.

Physical Examination

At the time the patient was admitted, he was a very fretful and well-built young man who was visibly distraught because of pain. His vital signs included: the blood pressure was 100/60 mmHg in both arms, pulse rate was 48beats per minute (regular), respiratory rate was 22 breaths per minute, temperature was 37.8degC (tympanic), respiratory rate was 98% on room air.

General: No pallor, icterus, cyanosis or peripheral edema. There was no petechiae or rash and skin was diaphoretic.

Cardiovascular: Precordium silent. Auscultation demonstrated normal first and second heart sounds without murmurs, rubs or gallops. The jugular venous pressure was not increased. The palpation of peripheral pulses was normal and symmetrical.

Respiratory: There was no wheezing or air crackles on either side of the chest.

Abdomen Soft, non-distended and non-tender, without hepatosplenomegaly. Bowel sounds were normal.

Neurological: Glasgow Coma Scale 15/15. Cranial nerves were intact. There was no motor or sensory defect.

Diagnostic Workup

The patient was properly triaged as a code STEMI.

Electrocardiogram (ECG): 12-lead ECG with ST-segment elevation of 3-4 mm in V1 to V4 leads with concomitant reciprocal ST-segment depression in II, III and aVF. Q-waves were not present.

Point-of-Care High-Sensitivity Cardiac Troponin I: Was significantly high at 15.2 ng/mL on a reference range of 0.04 ng/mL.

Bedside Echocardiogram (Limited): Performed in the ED revealed severe hypokinesia of the anterior septum, apex and apical anterior wall with an estimated visually left ventricular ejection fraction (LVEF) of 35-40%. There was no noteworthy pericardial effusion.

Complete Blood Count (CBC): Discovered leukopenia (White Blood Cell count 3.2 x 103/mL) neutrophilic predominance, and marked thrombocytopenia (platelet count 85,000/mL). Haemoglobin was 14.5 g/dL.

Basic Metabolic Panel: sodium 136mmol/L, potassium 4.1 mmol/L, creatinine 1.0mg/dL, estimated glomerular filtration rate (eGFR) >90ml/min/1.73m 2.

Liver Function Tests: Transaminases (AST 80 U/L, ALT 65 U/L) are slightly raised.
Since the classic presentation of STEMI was observed, the on-call cardiology team was summoned and the patient was ready to undergo emergency coronary angiography. Nevertheless, the presence of the constellation of prodromal febrile disease, thrombocytopenia, leukopenia, and relative bradycardia put a warning note on another diagnosis, namely dengue infection.

Dengue Serology: A positive rapid immunochromatographic test of dengue NS 1 antigen has been ordered and reported. Anti-dengue IgM antibodies were later proved positive by subsequent serology tests.

Hospital Course and Management:
There was a tense decision to carry out coronary angiography to exclude definitely an acute coronary occlusion, as there was a lack of certainty in the diagnosis, and a missed STEMI would be high-stakes.

Coronary Angiography: The test was done through the right radial artery, and showed that the coronary arteries were perfectly normal (Figure 2). No evidence of atherosclerosis, thrombus, vasospasm, dissection or embolism. Left dominant system was very common as a patent.

Final Diagnosis: The diagnosis was met as Acute Myocardial Injury caused by Severe Dengue Myocarditis, which has symptoms similar to STEMI.
The patient was taken into the Cardiac Care Unit (CCU) and put under strict observation. His treatment was adapted to his two-way pathology:

CVS Support: 24-hour ECG observation of cardiac arrhythmia. Beta-blockers and ACE inhibitors were not started because of his relative hypotension and bradycardia but were scheduled to start once he was hemodynamically stable to aid in myocardial remodelling.

Dengue Management: Supportive care was the key. Care was taken when intravenous fluid resuscitation was conducted; balanced crystalloid was used. Intake-output, daily weight and serial haematocrits were closely monitored to prevent under-resuscitation and the dangers of fluid accumulation due to plasma leakage. His platelet count was checked every day and his lowest count was 45,000/mL on day 4 of illness without any bleeding symptoms and then spontaneously recovered.

Follow-up and Outcome

Pain in the chest was completely resolved in 24 hours. Repeat ECG on day 3 indicated that the ST-elevation was resolved, and there was formation of deep and symmetrical T-wave inversions in the anterior leads, a typical evolutionary sequence in myocarditis. Trends in serial troponin levels were decreasing. On thorough echocardiogram at discharge (day 7) the patient has unresolved anterior wall hypokinesia but has an improved LVEF 45%. He was sent out in a stable condition with cardiology and infectious disease follow-up.
The repeat echocardiogram of 6 weeks revealed the near-complete recovery of the wall motion and LVEF of 55%. The patient was still asymptomatic and was slowly resumed into his regular activities.

Discussion

The mechanisms through which the dengue virus leads to severe myocardial damage are multiplex. To start with, direct viral cytotoxicity also has its role, with research showing the existence of dengue viral RNA and viral antigens in cardiac myocytes, with cells being directly invaded and replicated, resulting in cellular damage and the local inflammatory reaction(14, 15).

The list is wide-ranged in relation to the differential diagnosis of a young patient with STEMI on the ECG and high content of troponin combined with normal coronaries, including myocarditis of different viral or autoimmune etiologies, Takotsubo cardiomyopathy, coronary vasospasm and coronary embolism, coronary artery dissection, and Type 2 myocardial infarction through the lack of supply-demand mismatch. The diagnosis of dengue myocarditis became the major one in our patient, effectively eliminating the rest of the options because of the presence of thrombocytopenia, leukopenia, and positive dengue NS1 test.

The case has important diagnostic and treatment implications to clinical practice. Coronary angiography plays a very specific role; in an ordinary STEMI, it is therapeutic but, in this case, it is more diagnostic, and the only modality that will rule out an acute coronary occlusion in a timely fashion and avoid unnecessary and risky dual antiplatelet therapy to a thrombocytopenic patient. The value of a more clinical approach cannot be highly overstated; clinicians, particularly in endemic regions, should be instructed to see beyond the ST-elevation by taking a full history, including recent febrile illness, and review the initial CBC carefully to pick up such telling clues as relative bradycardia, which is highly suggestive in cases of dengue. Lastly, the management should be customized, with special attention to the supportive treatment of an underlying infection and cardioprotective care, such as careful fluid administration, arrhythmia control, and, at the recovery stage, the introduction of regular heart failure medications to facilitate reverse remodeling, after the acute inflammatory period is over.

To sum up, the case of severe dengue being reported as an acute myocardial infarction is a rare yet absolutely vital clinical findings. It is a complete storm as the pathophysiological mechanisms of a serious viral infection coincide to resemble an initial cardiac disaster. This case is an excellent lesson that diagnostic protocols, as necessary as standard care, should be used with clinical subtlety. In the areas where dengue is a common illness, having a high index of suspicion of this mimicry can avoid misdiagnosis, help select the correct use of coronary angiography, and provide safe and effective patient care, thereby closing the divide between two different fields of medicine. More studies are required to understand the risk factors and long-term outcome of patients who develop this severe dengue carditis.

Conclusion

The case demonstrates that dengue virus may lead to the development of a severe acute syndrome of myocardial injury that is clinically similar to STEMI. There is a great possibility of overdiagnosis and unnecessary anticoagulation or invasive coronary surgery, and this could prove detrimental in the backdrop of thrombocytopenia and a systemic viral disease. In young, endemic, patients who have STEMI-like manifestations and a febrile prodrome, greater clinical suspicion of dengue myocarditis is urgent. Coronary angiography is the gold standard of a definitive diagnosis and appropriate and possibly life saving management.

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