

Myopericarditis - A catastrophic complication of dengue fever

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ABSTRACT

Dengue is a mosquito-borne viral fever seen commonly in the urban areas of tropical and sub-tropical countries. The spectrum of this disease varies from a simple fever which can be managed with anti-pyretics to dengue shock syndrome, which requires urgent intensive care admission. Dengue shock syndrome can be a life-threatening condition. Dengue fever can progress to dengue shock and later to dengue shock syndrome if not managed with promptness. Apart from the spectrum of progression known to us, dengue fever can have a varied set of atypical presentations. These atypical presentations can be neurological, cardiac, or even gastro-intestinal. Cardiac complications, although rare, have been reported, such as various atrioventricular arrhythmias, myocarditis, pericarditis, and even myopericarditis. Here, we report a case of a 65-year-old male who presented to us with severe dyspnea and fever. Examination and investigations revealed dengue fever with myopericarditis.

Keywords: Arrythmias, cardiac, neurological, shock

Introduction

Dengue fever is a fast-spreading global infectious disease that is characterised by an increasing number of unusual symptoms.^[1] Dengue fever is an arboviral disease spread by the mosquito Aedes aegypti. It is caused by a flavivirus. Dengue fever is endemic in Africa, Central and Latin America, and South and Southeast Asia and has four antigenically different serotypes (DEN 1, DEN 2, DEN 3, and DEN 4). According to the World Health Organization (WHO), 40%

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Received: 10-02-2022 **Accepted:** 15-06-2022 **Revised:** 03-06-2022 **Published:** 31-10-2022

Video available on: www.jfmpc.com

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Website:
www.jfmpc.com
DOI:
10.4103/jfmpc.jfmpc_345_22

of the world's population lives in tropical and sub-tropical cities, where they are at high risk of contracting dengue fever.

Fever, headache, retro-orbital pain, rash, severe myalgia, and arthralgia are some of the clinical symptoms. Dengue haemorrhagic fever or dengue shock syndrome (DHF or DSS) is marked by an increase in vascular permeability, thrombocytopenia (platelets less than 1,00,000), bleeding tendencies, and circulatory shock in a limited percentage of patients.^[2]

Patients suffering from moderate to severe dengue go through three phases: an initial phase highlighted by fever and de-hydration, a critical period marked by shock from plasma leakage, haemorrhage or organ damage, and a recovery phase marked by extra-vascular water re-absorption. End-organ failure, coagulopathy, and capillary leak are predicted to occur in 1% to 5% of dengue patients who arrive to hospitals.^[3]

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How to cite this article: Giri A, Acharya S, Kamat S, Shukla S, Kumar S. Myopericarditis - A catastrophic complication of dengue fever. J Family Med Prim Care 2022;11:6553-5.

Dengue epidemics are known to cause a non-specific afebrile sickness, a milder form of DHF and severe DSS. Atrioventricular conduction abnormalities, supra-ventricular arrhythmias, myocarditis, pericarditis, and even myopericarditis are among the cardiac problems that have been observed in dengue patients. DSS is a severe form of the disease characterised by circulatory shock and haemorrhagic consequences, which can cause transient myocardial depression. Dengue pericarditis is uncommon; however, it has been recorded. Although pericardial effusions are relatively rare, they have been reported in severe dengue cases. Very rarely, the combination of myocarditis along with pericarditis presenting as myopericarditis has also been reported.

Case Report

A 65-year-old male patient presented to the Emergency department of the hospital with chief complaints of fever, body ache, palpitations, and breathlessness for 4 days.

The patient was apparently alright 4 days back when he started experiencing fever, which was sudden in onset and gradually progressing. It was of high grade, was intermittent, and was associated with chills and rigours. Fever was relieved on taking anti-pyretics. Fever was associated with generalised body ache and joint pain. The patient also complained of palpitations which were present even at rest. It was associated breathlessness at rest (NYHA grade 4). There was no history of any co-morbidities.

The patient's general examination revealed a pulse rate of 120/min, regular. Peripheral pulses were feeble. The blood pressure was 90/60 mm of Hg. Oxygen Saturation was 97% while breathing ambient air. The Jugular venous pressure was increased at 12 cm of water. On cardiovascular system examination, S1 and S2 were soft and S3 was audible with gallop rhythm. There was an audible pericardial rub present just inside the apex. There were no murmurs. The respiratory system revealed bibasal inspiratory crepitations. P/A and the central nervous system did not reveal any abnormalities.

Laboratory investigations revealed CBC, Hb-11 gm%, leukopenia with a WBC count of 2200/cumm with 58% lymphocytes, and thrombocytopenia with a platelet count of 70,000/cumm. The fever profiles for malaria, leptospirosis, and scrub typhus were negative. The dengue NS-1 antigen was positive. Peripheral smear did not reveal the malarial parasite. Cardiac markers revealed CKMB- 95 IU/L and Trop-I- 12000 ng/ml. NT- Pro BNP was 1200 pg/ml. Kidney function tests revealed urea of 50 mg%, creatinine of 1.9 mg%, sodium of 138 mEq/L, and potassium of 5.0 Meq/L. Liver function tests revealed mild transaminitis.

The patient's electrocardiogram (ECG) was suggestive of pericarditis [Figure 1]. 2D echocardiography (ECHO) showed that all four chambers were dilated, a dilated LV with global hypokinesia, a depressed left ventricular ejection fraction around 30%, and moderate pericardial effusion [Figure 2 and Video 1].



Figure 1: A 12 lead ECG showing sinus tachycardia HR-130 bpm along with widespread subtle concave ST elevation with PR depression in the pre-cordial (V2-6) and inferior leads (II, III, aVF). PR elevation in a VR suggestive of pericarditis

The patient was started on ionotropic support, monitored iv fluids, supplemental oxygen, iv diuretics, steroids, anti-pyretics, Tab Colchicine 0.5 mg Bd, and Inj Prednisolone 1 mg/kg per day. Within 72 hours, the patient's symptoms started resolving, breathlessness settled, and fever subsided, and within 7 days, the patient was discharged.

On discharge, investigations were repeated, which showed CBC, Hb-11.2 gm%, a WBC count of 5200/cumm, and a platelet count of 1,20,000/cumm. Kidney function tests showed urea of 50 mg%, creatinine of 1.1 mg%, sodium of 135 mEq/L, and potassium of 4.6 mEq/L. Liver function tests and all other routine tests were normal. Repeat 2D ECHO was performed, which showed that the effusion had gone down. The patient was then discharged on oral prednisolone with tapering doses and with the advice of follow-up after 2 weeks.

Discussion

Cardiac involvement in dengue fever and associated pathogenesis has been rarely described and studied. The production of inflammatory mediators and/or direct action of the virus on cardiomyocytes, as shown in acute myocarditis caused by other viruses, could be the mechanism of cardiac injury in dengue.

When comparing individuals with DSS to those with DF or non-shock DHF, myocardial dysfunction has been found to be more severe. In dengue fever, the pathogenesis of cardiac cell damage is still unknown. Direct DEN invasion of the cardiac muscles, a cytokine-mediated immune response, or both can cause myocardial involvement in dengue. Elevated vascular permeability and shock are associated with increased serum tumour necrosis factor; interleukins 6, 13, and 18; and cytotoxic factors in dengue patients; whether these cytokines play a role in the development of myocardial cell injury is uncertain. Only DEN-2 and DEN-3 have been identified as the causative viruses in dengue patients with cardiac problems when the DEN serotype has been indicated.^[4]

ECG abnormalities and elevations in cardiac bio-markers can indicate dengue myopericarditis, which can mimic acute myocardial infarction. The cardiotropic characteristic of the



Figure 2: 2 D ECHO PLAX view showing evidence of pericardial effusion

virus is likely to blame for myocyte destruction, with serotypes DEN 1, DEN 2, and DEN 3 usually implicated in fulmimnant myopericarditis patients. With the right fluid therapy and inotropes, ECHO may be able to help guide early management of refractory shock. Cardiac magnetic resonance imaging is a useful research technique for confirming myocardial involvement, which appears on T2-weighted images with early and late gadolinium enhancement as hyper-intense signals.

Severe dengue has no specific treatment, and care is purely supportive, including fluid resuscitation when necessary. During the recovering phase of dengue myopericarditis, fluid overload has been linked to higher morbidity. Despite fluid resuscitation, around 5% of patients with shock require inotropes.^[5,6] To treat life-threatening hypotension or hypotension progressing to cardiogenic shock, inotropes or vasopressors are utilised. In dengue myocarditis, there is no evidence to support the use of particular anti-viral treatment, steroids, or immunoglobulin. In dengue-affected patients with severe myocarditis, early detection of cardiac involvement, fluid resuscitation while avoiding overload, and inotropic support with constant monitoring remain the mainstays of therapy.^[7]

Conclusion

The primary care physician is the first to initiate the diagnosis and treatment of all infectious diseases presenting as acute febrile illness including dengue fever. Therefore, it is of utmost importance that the primary care physician is well aware of all the atypical and lethal complications of such commonly occurring infectious diseases. The aim of our case report is to create awareness to the primary care physicians about these unusual complications of seemingly common infections, dengue fever in this report, so that prompt initial management and referral to a tertiary care hospital can be performed as soon as possible, which will not only save the life of the patient but also appropriately utilize the health care infrastructure. It is imperative that more research and reporting of this fatal complication is performed so that it is easier for fellow physicians to diagnose and treat this deadly life-threatening complication at the earliest. Cardiac complications along with all other atypical complications of dengue fever must be diagnosed at the earliest and treated with prompt care to save the life of the patient.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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