Case Report

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Internal Medicine Section

Reversible Wenckebach Phenomenon in a Young Patient with Dengue Fever: A Case Report

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ABSTRACT

Dengue fever, caused by the Dengue Virus (DENV) of the Flaviviridae family, is widespread in tropical and subtropical regions, with India experiencing a high burden of disease. Although many cases remain asymptomatic or mild, severe complications, including cardiac manifestations, can occur. This article reports uncommon presentation of dengue-associated transient conduction disturbances without overt myocardial injury. A previously healthy 22-year-old male presented with intermittent fever, chills, generalised myalgia, gastrointestinal symptoms and dehydration for five days. Initial evaluation at a local clinic revealed thrombocytopenia and a normal Electrocardiogram (ECG). On hospital admission, the patient exhibited dehydration, bleeding manifestations (oral mucosal and gingival haemorrhages), and an irregular pulse. Laboratory tests indicated thrombocytopenia, transaminitis, and pre-renal acute kidney injury, while dengue NS1 antigen and IgM antibodies were positive. A 12-lead ECG performed on admission revealed a second-degree Mobitz Type I (Wenckebach) atrioventricular block, which was confirmed by 24-hour Holter monitoring. Cardiac enzymes were within normal limits, and echocardiography showed preserved left ventricular function without evidence of myocarditis. Management included intravenous hydration and platelet transfusions. Serial ECG monitoring demonstrated resolution of the Atrio-ventricular (AV) block by day 4 of hospitalisation, and the patient's renal and hepatic functions normalised, leading to discharge on day 7. This case underscores the need for diligent cardiac monitoring in dengue fever, as transient conduction disturbances may occur even without significant myocardial involvement. Transient low-degree AV block in dengue fever, although seemingly benign, may herald severe cardiac complications. Prompt recognition and supportive management are pivotal in mitigating potential cardiac complications.

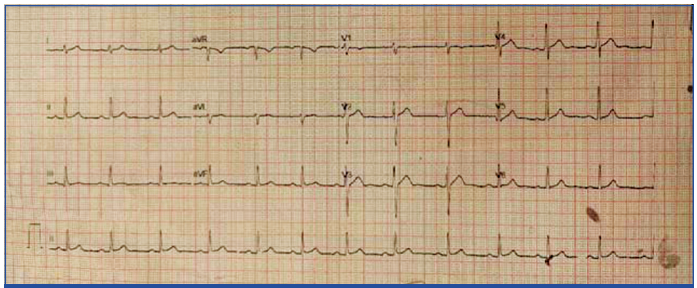
Keywords: Conduction disturbances, Dehydration, Dengue fever, Mobitz type I atrioventricular block, Myocarditis, Thrombocytopenia

CASE REPORT

A 22-year-old male, with no known co-morbidities, reported with 5-day history of intermittent fever associated with chills and rigours along with generalised myalgia. He had complaints of 3-4 episodes of vomiting over three days, with food as vomitus; the vomitus was non-bilious and non-blood-stained. Patient gave no history of cardiac complaints and denied history of cardiac disease in family.

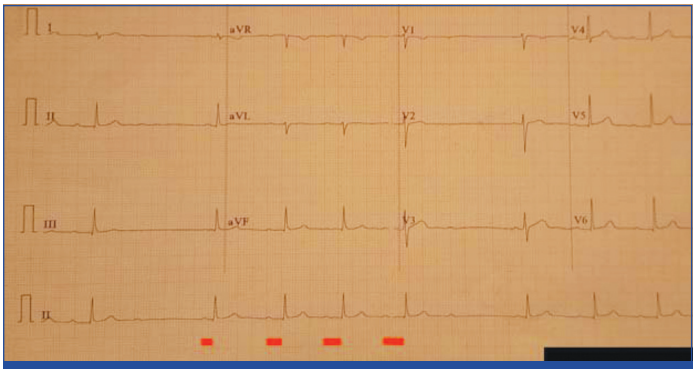
Patient was initially evaluated in a local primary healthcare clinic, where haemogram showed thrombocytopenia and ECG showed no significant abnormalities [Table/Fig-1]. Patient came to the hospital two days later. On examination, his blood pressure was 100/70 mmHg, his pulse rate was 62 beats per minute-low in volume and regularly irregular in rhythm-and his temperature was 102°F. No hypoxaemia or tachypnoea was observed. Patient exhibited signs of dehydration with parched lips, oral mucosal and gingival bleeding, and multiple petechiae observed over his limbs, trunk, and palate [Table/Fig-2]. Mild tenderness was noted on palpating epigastric and right hypochondriac quadrants of abdomen. Other system examination were non-contributory. ECG showed Mobitz type I second-degree atrioventricular heart block [Table/Fig-3], which was confirmed by 24-hour Holter monitoring.

Initial investigations [Table/Fig-4] revealed a haemoglobin level of 16.5 g/dL, packed cell volume of 49%, and a platelet count of 13,000/mm^3. The total leucocyte count was 4,560/mm^3. Liver function tests revealed transaminitis, with Serum Glutamate Oxaloacetate Transaminase (SGOT) at 244 IU/L and Serum Glutamate Pyruvate Transaminase (SGPT) at 151 IU/L. Renal function tests were impaired, showing a serum creatinine 1.6 mg/dL and serum urea of 58 mg/dL, suggestive of pre-renal Acute Kidney Injury (AKI) on clinical correlation. Inflammatory markers like Erythrocyte Sedimentation Rate (ESR) and C-Reactive Protein (CRP) were elevated. Ultrasonography of abdomen revealed features suggestive of polyserositis. There was very strong suspicion of tropical fever on correlating clinical symptoms with geographical location, thrombocytopenia, transaminitis, and serositis. Dengue NS1 antigen and IgM antibodies turned out positive. His serum electrolytes, calcium, and magnesium values turned out not contributory to the aetiology of the AV block. In suspicion of dengue myocarditis, echocardiogram was obtained but revealed normal left ventricular function and no other anomalies [Table/Fig-5]. Cardiac enzymes were not elevated. Hence, patient was diagnosed to have dengue-associated second-degree Mobitz type I (Wenckebach) atrioventricular block without evidence of overt myocarditis.



[Table/Fig-1]: The baseline ECG obtained at a local healthcare facility 2 days prior to hospital admission shows no significant abnormalities in rhythm, rate, or conduction. This highlights the initial absence of cardiac involvement in the early stages of dengue fever, prior to the onset of the Atrioventricular (AV) conduction disturbance noted later.

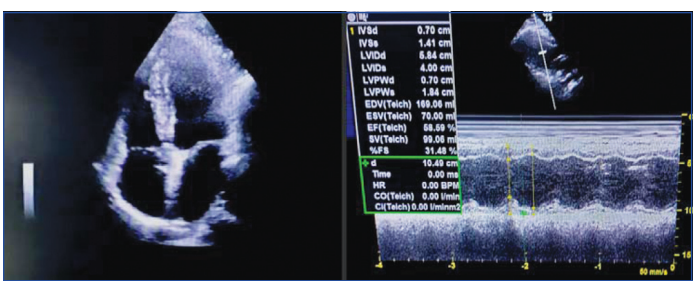
[Table/Fig-2]: Patient had parched lips with oral mucosal and gingival bleeding with multiple petechiae observed over his limbs, trunk and palate.



[Table/Fig-3]: ECG performed on hospital admission reveals a Mobitz Type I (Wenckebach) second-degree AV block. This is characterised by the progressive PR interval prolongation (Red bars) followed by a dropped QRS complex in the ECG, indicating transient conduction delay at the AV node.

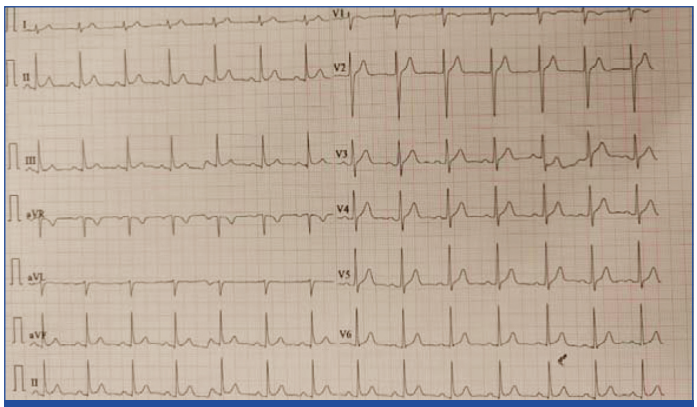
Test	Result	Normal range
Haemoglobin (g/dL)	16.5	Male 13-17, Female 12-15
Total leukocyte count (cells/mm^3)	4560	4000-10000
Platelet count (lakh/mm^3)	0.13	1.5-4.5
Aspartate aminotransferase (AST/SGOT) (IU/L)	244	10-40
Alanine aminotransferase (ALT/SGPT) (IU/L)	151	7-56
Serum creatinine (mg/dL)	1.6	0.6-1.2
Serum urea (mg/dL)	58	17-43
Erythrocyte Sedimentation Rate (ESR) (mm/hour)	20	0-15 (male), 0-20 (female)
C-Reactive Protein (CRP) (mg/L)	164	0-10

[Table/Fig-4]: Laboratory parameters on admission showing haematological, hepatic, renal, and inflammatory markers.



[Table/Fig-5]: The transthoracic echocardiogram shows normal left ventricular function with no regional wall motion abnormalities, normal chamber dimensions, and no other structural abnormalities - ruling out myocarditis or overt myocardial dysfunction.

The patient was adequately hydrated, and six units of random donor platelets were transfused in view of bleeding manifestations and severe thrombocytopenia. Other supportive medications were administered. Patient showed significant improvement of clinical symptoms, with no signs of hypotension or further bleeding manifestations. His thrombocytopenia gradually recovered over the course of a few days. Serial ECG monitoring was done and patient's ECG had returned to normal sinus rhythm on day 4 of hospital stay [Table/Fig-6]. This was accompanied with rapid resolution of renal and liver function parameters: SGOT 68 IU/L, SGPT 56 IU/L; renal function tests revealed serum creatinine of 0.8 mg/dL and serum urea of 9 mg/dL. Patient was discharged on day 7 of his hospital stay. ECG and cardiac evaluation done on follow-up visit after one week showed no significant abnormalities.



[Table/Fig-6]: ECG on day 4 of hospital stay demonstrates restoration of normal sinus rhythm, with no evidence of conduction anomalies.

DISCUSSION

Dengue, a virus from the Flaviviridae family, is the world's most prevalent arthropod-borne viral disease, with four serologically distinct types {Dengue Virus (DENV)-1 to DENV-4} transmitted predominantly by Aedes aegypti and Aedes albopictus mosquitoes [1]. Although most infections are asymptomatic or mild-characterised by fever, headache, myalgia, vomiting, and malaise-severe illness and mortality can occur. India, with its conducive urban and semi-urban breeding environments, frequently endures outbreaks despite nationwide vector control programmes, enhanced diagnostics, and ongoing vaccine research. Rapid urbanisation, insufficient control measures, and climate change have driven an increasing dengue burden, leading to more hospitalisations for complications, including cardiac involvement, even as the case fatality rate remains below 1% [1]. In 2009, the World Health Organisation (WHO) reclassified dengue into three categories-without warning signs, with warning signs, and severe dengue-to better stratify risk [2]. Warning signs such as mucosal bleeding (epistaxis, gum bleeding), gastrointestinal haemorrhage, dyspnoea, altered sensorium, ascites, haemoconcentration with thrombocytopenia, shock, elevated transaminases, gallbladder wall thickening, and secondary infections are all linked to progression toward severe disease [3]. Thrombocytopenia and a higher haematocrit value show significant correlation with dengue-associated cardiac involvement [4].

Cardiac conduction disturbances are an uncommon but reported complication of dengue infection. This patient's Mobitz type I second-degree AV block (Wenckebach) is similar to prior cases of dengue-associated AV block, which were transient and resolved spontaneously with supportive care. Khongphatthallayothin A et al., reported two children with dengue haemorrhagic fever who developing transient Mobitz I AV block during recovery phase, without any structural heart issues. In this article, it was proposed that transient AV block may be a functional impairment of the AV node influenced by altered autonomic tone [5]. These observations suggest that a dengue-related Wenckebach block is generally

benign and self-limited. However, dengue can rarely precipitate more severe conduction disturbances, including Mobitz type II second-degree block or complete heart block, particularly in patients with severe dengue [6]. In an article reported by Nigam AK et al., a middle-aged female diagnosed to have dengue fever with normal ECG on admission but developed Mobitz type II second-degree AV block on 5th day of admission. Patient reverted to normal rhythm on the 6th day of admission and showed no abnormalities in her ECG on follow-up [7]. In rare cases, dengue-associated myocarditis can lead to long-term complications like dilated cardiomyopathy, as reported by Tahir H et al., [8].

The pathophysiology of dengue-related cardiac effects is likely multifactorial. Proposed mechanisms include:

- **Immune-mediated injury:** The intense inflammatory response in dengue (cytokine surge and immune cell infiltration) can inflame the myocardium and conduction system. This immune-mediated injury may transiently impair AV nodal function [9].
- **Direct viral invasion:** DENV may directly infect cardiac myocytes. Viral components have been detected in myocardial tissue, suggesting direct cytopathic damage that disrupts electrical conduction [9].
- **Ischaemic/metabolic stress:** In severe dengue, hypotension and shock cause systemic hypoperfusion and metabolic acidosis, which can transiently disturb AV nodal conduction [9].
- **Autonomic dysfunction:** During defervescence, heightened vagal tone (relative bradycardia) can functionally slow AV nodal conduction. In dengue patients with Wenckebach block, exercise (reducing vagal tone) normalised AV conduction, while Valsalva manoeuvers worsened it, implicating autonomic effects in the block [9].

These mechanisms may act in concert. Overall, dengue-related myocardial dysfunction likely results from a combination of direct viral injury and immune-mediated damage, with systemic hypoperfusion or autonomic factors exacerbating the cardiac involvement. The normal troponin (Troponin I-0.03 ng/mL) and echocardiogram in our patient support a predominantly functional or mild inflammatory process rather than extensive myocarditis.

This case underscores the importance of serial ECG monitoring in dengue fever, even when patients are asymptomatic. Holter studies have demonstrated a high incidence of transient, often asymptomatic ECG changes in dengue, including bradyarrhythmias, ST/T-wave changes, and minor conduction blocks [10,11]. Given this, regular ECGs during the acute and recovery phases are prudent to detect occult arrhythmias early. Early identification of an AV block enables closer observation and supportive measures (e.g., hydration, electrolyte correction) to prevent haemodynamic compromise. In our patient, daily ECGs allowed timely recognition of the Wenckebach phenomenon and confirmed its resolution by day 4 of hospitalisation.

Identifying dengue-related AV block is crucial since it may signal broader cardiac involvement. While Mobitz I block in dengue is usually self-limited, progression to higher-grade AV block or more malignant arrhythmias is possible if myocardial injury worsens [6]. Rare case of complete heart block have been documented in dengue patients [12], sometimes accompanied by acute myocarditis and haemodynamic instability. If myocardial involvement becomes severe, patients can develop syncope, acute heart failure, or even sudden cardiac arrest. Fulminant dengue myocarditis-though uncommon-has been reported to cause cardiogenic shock and life-threatening arrhythmias [13].

A new-onset AV block in a dengue patient should prompt evaluation for myocarditis (including cardiac biomarkers and echocardiography) and careful fluid management to avoid cardiac overload. One report emphasises that arrhythmias in dengue "can be an early indicator for myocarditis," underscoring the need for vigilant monitoring. Prompt supportive therapy-judicious i.v. fluids, oxygen, and correction of metabolic disturbances-is key to preventing progression of cardiac complications. In rare instances of high-degree AV block with haemodynamic compromise, temporary pacing may be required. Fortunately, most dengue-related rhythm disturbances, as in our case, resolve with supportive care alone, with normal conduction returning in a few days as the infection subsides.

CONCLUSION(S)

Dengue fever can occasionally involve the heart, causing transient arrhythmias such as a Mobitz type I AV block. Our 22-year-old patient's Wenckebach block resolved by day 4 of hospitalisation with supportive treatment, illustrating the generally reversible nature of this complication. A review of similar cases indicates that dengue-associated AV blocks typically abate spontaneously once the acute phase passes, likely due to functional autonomic effects or mild, transient myocarditis. Clinicians should maintain a high index of suspicion for cardiac involvement in dengue-even in young patients without prior cardiac issues-and implement serial ECG monitoring during acute illness. Early recognition of conduction disturbances enables appropriate supportive management and prevents potential progression to more severe arrhythmias or fulminant myocarditis. This case reinforces that diligent cardiac monitoring and supportive care are essential in dengue fever, contributing to favourable outcomes.

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