Case Report

Intermittent Left Bundle Branch Block and Cardiac Memory in a Patient with Dengue Fever: A Case Report and Literature Review

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Abstract

Background: Arrhythmias and Electrocardiography changes in Dengue Fever (DF) cases can mimic electrocardiogram (ECG) appearance of threatening clinical conditions such as myocardial ischemia, albeit no significant ischemic injury at the heart muscle.

Case Presentation: We report a case of subsequent cardiac memory (CM) in a 50-year-old female with DF. A 12-lead ECG on admission revealed sinus rhythm with Left Bundle Branch Block (LBBB), and it resolved on day 4 of hospitalization. However, deep symmetric T wave inversions (TWI), which were later recognized as CM, occurred. The patient received treatment with Ringer's lactate and paracetamol infusion. A routine 12-lead ECG performed on day 7 of hospitalization revealed the resolution of TWI and normal sinus rhythm.

Conclusion: This case report highlights one intriguing phenomenon associated with LBBB in DF, which is CM TWI, where the T wave 'remembers' the direction of the QRS complex during prior abnormal ventricular activation. Due to its similarity with TWI caused by myocardial ischemia, understanding of this phenomenon may prevent unnecessary interventions.

Keywords: Bundle-Branch Block; Dengue; Case Reports.

Introduction

Cardiac manifestations in dengue infection are uncommon and typically arise only in severe cases. Both vascular damage, such as endothelial dysfunction and alterations in vascular permeability, and localized cardiac injury contribute to the development of these manifestations. Common cardiac complications associated with Dengue Fever (DF) include myocarditis, pericarditis, arrhythmias, left ventricular dysfunction, and hypotension.[1]

Among these, arrhythmias and asymptomatic electrocardiogram (ECG) changes are the most frequently observed. Various types of arrhythmias, including bundle branch block, have been reported in DF cases. Arrhythmias and ECG changes in DF may resemble the ECG patterns seen in more critical conditions, such as myocardial ischemia, despite the absence of significant ischemic injury to the myocardium.[1] In this case report, we present a patient with intermittent Left Bundle Branch Block (LBBB) followed by transient cardiac memory (CM) T-wave inversions, which mimicked myocardial ischemia in the context of DF, and discuss how to differentiate this from true ischemic injury.

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Case Report

A 50-year-old female presented to the emergency department with a primary complaint of fever for three days. She also reported severe joint and muscle pain, headache, and fatigue. The patient noted difficulty walking due to joint pain but denied any significant cardiac-related symptoms, such as chest pain or palpitations. Her medical, family, and travel histories were unremarkable.

Upon admission, the patient was alert and oriented. Physical examination revealed a blood pressure of 140/90 mmHg, a pulse rate of 75 beats per minute, a respiratory rate of 20 breaths per minute, and an axillary temperature of 38.9 °C. Other aspects of the physical examination were within normal limits. Initial laboratory findings showed a leukocyte count of 3.21 x 10^3/L, a platelet count of 117 x $10^3/\mu$L, a sodium level of 130 mmol/L, potassium level of 3.5 mmol/L, and a positive anti-Dengue IgM. Serial blood counts showed a further decrease in platelet count. A 12-lead ECG performed on admission revealed sinus rhythm with complete LBBB (Figure 1). The patient was treated with Ringer's lactate and paracetamol infusion.

On day 4 of hospitalization, a routine 12-lead ECG showed resolution of the LBBB and the subsequent development of T-wave inversions in leads V1-V4 (Figure 2). However, the patient did not exhibit any clinical deterioration. A transthoracic echocardiogram did not reveal any significant structural or functional abnormalities, and the troponin I level was undetectable. A repeat ECG performed on day 7 showed resolution of the T-wave inversions and return to normal sinus rhythm (Figure 3).

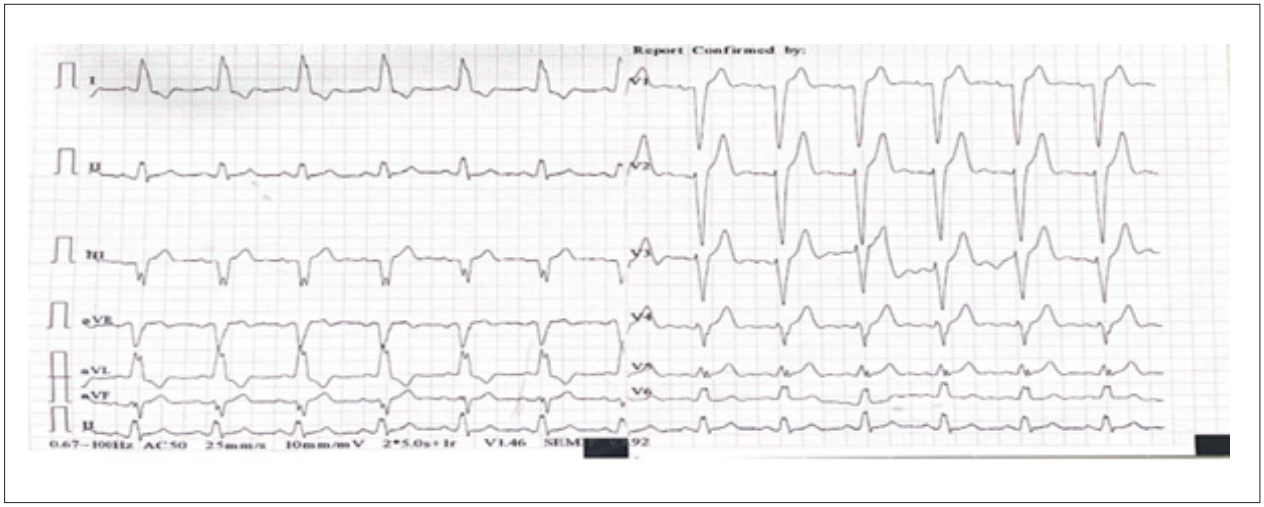

Figure 1 – *Electrocardiography on admission showed sinus rhythm with complete LBBB.*

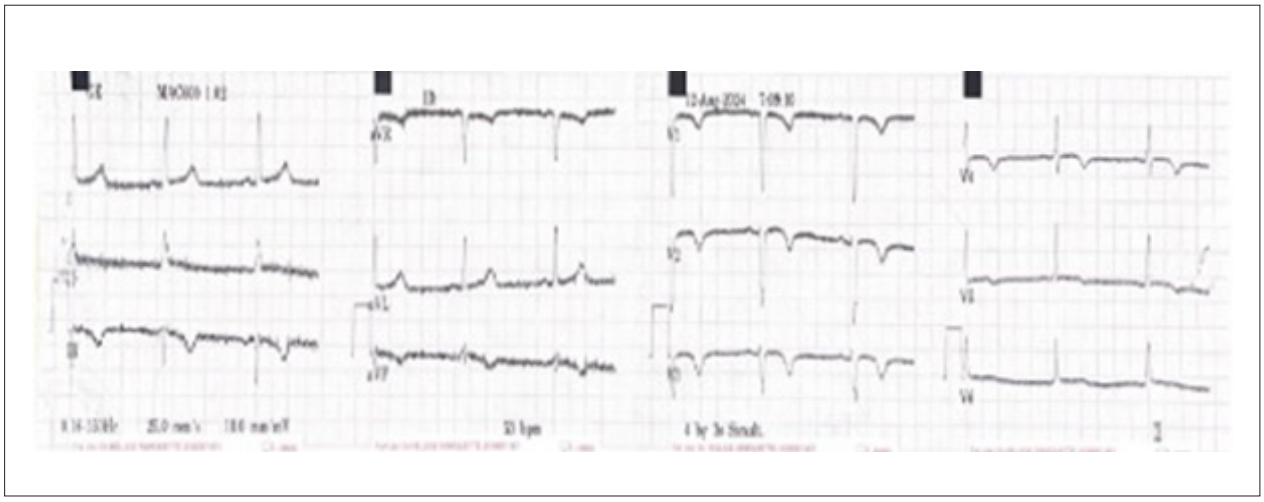

Figure 2 – *Monitoring electrocardiography revealed the resolution of LBBB and subsequent TWI in V1-V4.*

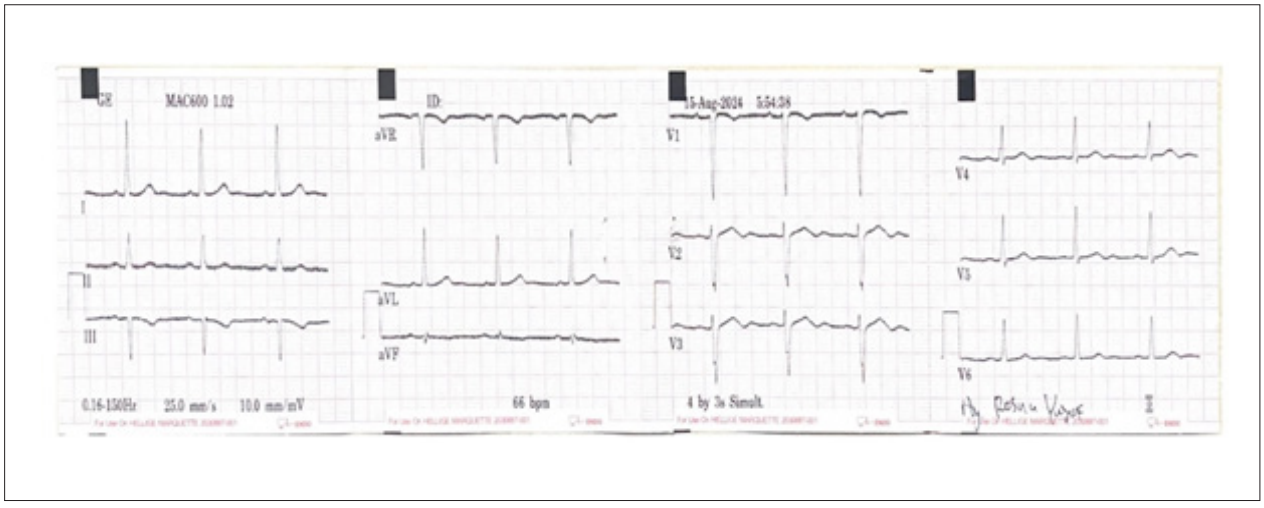

Figure 3 – *A repeat ECG performed on day 7 of hospitalization showed the resolution of TWI and normal sinus rhythm.*

Discussion

LBBB is a conduction block where the cells of the ventricle must rely on a relatively slow myocyte-to-myocyte spread of electrical activity traveling from the unaffected ventricle rather than rapid uniform stimulation by Purkinje fibers.[2] LBBB itself is asymptomatic, and there are no signs or symptoms other than a marked pattern on ECG.[3] However, it may have a significant role in certain clinical contexts. This article reports on the occurrence of intermittent LBBB in DF. At first, it was unclear whether the LBBB was associated with the infection because there was no prior ECG documented. However, the rhythm turned into normal sinus rhythm on day 4 of hospitalization, and it was aligned with the patient's clinical improvement.

Cardiac complications in DF are rare. However, the cases are increasingly being reported in various studies. The most frequent forms of abnormalities include sinus tachycardia, sinus bradycardia, and non-specific ST-T changes.[4] Additionally, one of the cardiovascular disturbances that may appear is bundle branch block. The incidence of bundle branch block in dengue patients has been reported to range between 1.6% to 5.5%.[4,5]

Based on the pathophysiology of DF, which involves systemic inflammation, endothelial dysfunction, and possible subclinical myocarditis, we propose that LBBB in this case may be a result of the infection's direct impact on the cardiac conduction system. However, we also consider the possibility that LBBB could be an incidental finding unrelated to the infection. The mechanism of cardiac involvement, particularly arrhythmia including LBBB, consists of several factors that result in myocardial injury and following conduction impairments. Arrhythmia can occur from shifts in membrane potential that can arise from inflammatory processes and cytokine storms impacting myocytes and the surrounding tissue. Alterations in ventricular dynamics, such as increased wall strain and myocardial oxygen demand, can also provoke arrhythmias. Furthermore, low platelet counts increase the risk of bleeding around the sinoatrial or atrioventricular node, which eventually can result in conduction defects and arrhythmias. Additionally, electrolyte imbalance, specifically potassium level, might cause transient rhythm abnormalities.[5] In this patient, we found no significant cardiac associated complaints nor an increase of cardiac enzyme, which contradicts other similar case reports. Despite the fact that there are Sgarbossa criteria to diagnose acute myocardial infarction in a setting of LBBB, we suggest further research in determining the importance of cardiac enzyme for the diagnosis of patients with arrhythmias such as LBBB, particularly in DF cases.

One intriguing phenomenon associated with LBBB is CM T Wave Inversions (TWI), where the T wave 'remembers' the direction of the QRS complex during prior abnormal ventricular activation. CM is characterized by persistent TWI that occurs when normal ventricular activation is restored after a phase of abnormal ventricular conduction. White first documented the phenomenon of transient TWI in 1915, noting their occurrence after isolated ventricular premature beats. In the 1940s, TWI was also reported following the return to sinus rhythm after episodes of paroxysmal tachycardia. Since then, irregular T waves of different durations have been observed in a range of conditions, including intermittent ventricular pre-excitation, ventricular pacing, LBBB, and ventricular tachycardia.[6] CM itself is benign and does not have any clinical significance, but it is similar to pathological condition such as myocardial ischemia.[7] In this patient, we found CM following the resolution of prior LBBB, which is in line with other similar case reports.

In a study involving 60 patients conducted by Shvilkin *et al*. (2015), researchers compared TWI from CM caused by right ventricular pacing to that from myocardial ischemia. They found that positive T in lead a VL and positive/isoelectric T in lead I, and maximal precordial TWI greater than inferior TWI had a sensitivity of 92% and specificity of 100% for CM. These criteria can be applied in the case of intermittent LBBB due to the axis similarity of both cases with positive QRS complex in leads I and aVL.[8]

A review of the current case reports and literature reveals a paucity of data regarding ECG abnormalities, particularly LBBB, in patients with DF. Additionally, there is a significant lack of reports addressing the occurrence of CM in association with LBBB in this patient population. While various ECG abnormalities have been documented in DF, the relationship between LBBB and CM remains insufficiently explored. This gap in the literature underscores the contribution of our case report, which provides valuable insights into these uncommon ECG changes and highlights the clinical importance of recognizing such disturbances in the management of DF.[5]

Although all the criteria for diagnosis are met in our patient, further investigations are essential to establish a reliable diagnostic approach, especially in cases where concurrent myocardial ischemia is present. Additionally, close follow-up is warranted to determine whether this patient may have a predisposition to future conduction abnormalities, particularly in the presence of underlying cardiac conditions, ensuring comprehensive long-term care and management.

Conclusion

Arrhythmias in DF can be caused by inflammation of the heart muscle. Although self-limiting, early diagnosis and prompt monitoring are crucial for prognosis. This case report highlights one intriguing phenomenon associated with LBBB in DF, which is CM TWI, where the T wave 'remembers' the direction of the QRS complex during prior abnormal ventricular activation. Due to its similarity with TWI caused by myocardial ischemia, understanding this phenomenon may prevent unnecessary interventions.

Author Contributions

Conception and design of the research and acquisition of data: Alkatiri JI; analysis and interpretation of the data and critical revision of the manuscript for intellectual content: Siregar MNI; writing of the manuscript: Alkatiri JI, Siregar MNI.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Study Association

This study is not associated with any thesis or dissertation work.

Ethics Approval and Consent to Participate

All procedures performed in this study follow the ethical standards of the institutional and national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. Informed consent was obtained from participants included in this study.

Use of Artificial Intelligence

The authors did not use any artificial intelligence tools in the development of this work.

Availability of Research Data and Other Materials

The underlying content of the research text is contained within the manuscript.

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