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Case Report

Chest pain associated with rate-related left bundle branch block and cardiac memory mimicking ischemia

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ABSTRACT

Background: Intermittent left bundle branch block (LBBB) has been linked to chest pain, and causes cardiac memory electrocardiographic (ECG) changes mimicking ischemia.
Purpose: To present a case of chest pain with ECG abnormalities suggestive of ischemia, both likely caused by LBBB.
Case: A 33-year-old hypertensive female evaluated for chest pain and LBBB by ECG was treated with lisinopril and metoprolol, and scheduled for stress testing. A 12-lead ECG performed prior to the stress test, due to recurrence of the chest pain the preceding night, showed resolution of the LBBB with a lower heart rate, and T-wave inversions in the precordial leads suggestive of ischemia. She developed chest pains with reappearance of LBBB during stress testing, which prompted cardiac catheterization. This revealed normal coronaries and left ventricular systolic function. The ECG abnormalities were in retrospect likely due to cardiac memory. Her chest pains may have been caused by the intermittent, rate-related LBBB, as control of her heart rate and blood pressure with metoprolol and lisinopril improved her symptoms on follow-up.
Conclusion: Intermittent LBBB causes chest pain and electrocardiographic abnormalities suggestive of ischemia in the absence of obstructive coronary disease. Certain clinical and electrocardiographic features may provide clues to a non-ischemic etiology.
<**Learning objective:** In the absence of obstructive coronary disease, rate-related left bundle branch block is associated with chest pain described as local, non-radiating, with palpitations and walk-through phenomenon. It can also cause electrocardiographic (ECG) changes of cardiac memory, which mimic myocardial ischemia, but with T waves that are positive in lead aVL, positive or isoelectric in lead I, and more inverted in the precordial leads compared with lead III. These clinical and ECG features may provide clues to non-coronary etiology of chest pain.>

Introduction

Exercise-induced left bundle branch block (LBBB) has been described in 0.38% of patients referred for stress testing [1], and has been shown to be an independent predictor of a higher risk of death and major cardiac events [2]. Rate-related LBBB can happen spontaneously without exercise at any heart rate [3]. It has been linked to the development of angina-quality chest pain [4] and the appearance of abnormal, ischemic-type T wave inversions of cardiac memory [5], in the absence of obstructive coronary disease. A case that combines these features is presented, together with review of pertinent literature.

Case report

A 33-year-old female was referred to the cardiology clinic for evaluation of chest pain and palpitations. She reported intermittent left-sided chest discomfort radiating to her left arm. The pain was not always associated with exertional activities. Her symptoms would only last a few seconds and abate spontaneously. She also complained of dyspnea on moderate exertion and rare palpitations. She is a non-smoker and does not have family history of premature coronary artery disease. She was only taking oral contraceptive pills. Blood pressure was recorded as 170/110 mmHg with a regular heart rate of 100 bpm; the remainder of her physical examination was unremarkable. Electrocardiography (ECG) revealed sinus tachycardia with LBBB (Fig. 1), which was a new finding compared

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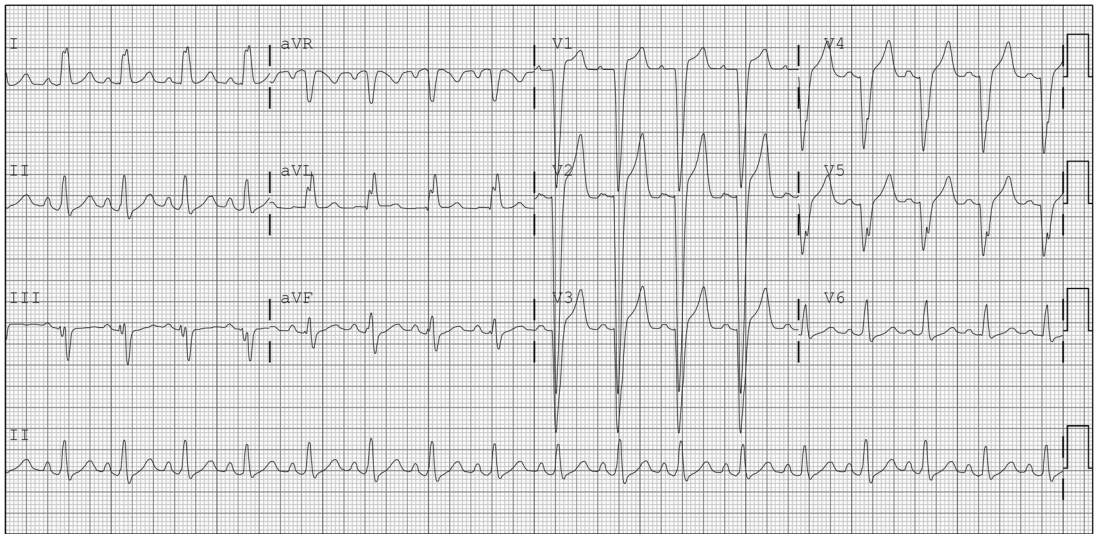

Fig. 1. Electrocardiogram upon presentation to cardiology clinic reveals sinus tachycardia at 103 bpm with left bundle branch block.

to a prior ECG seven years earlier (Fig. 2). Basic blood work revealed normal creatinine, electrolytes, glucose, thyroid stimulating hormone, and complete blood count.

The patient was started on lisinopril 20 mg daily and metoprolol tartrate 25 mg twice daily to treat her hypertension and palpitations. Given her symptoms and the newly diagnosed LBBB, she was scheduled for pharmacologic stress nuclear testing to evaluate any structural or functional abnormalities which may explain her chest discomfort, dyspnea on exertion, and palpitations.

Two weeks later the patient presented for her scheduled tests. She reported one recurrent episode of chest discomfort the preceding evening. An ECG was obtained (Fig. 3) which revealed a slower heart rate of 64 bpm and resolution of the LBBB when compared with her clinic ECG (Fig. 1). T-wave inversions were noted in the precordial leads suggestive of ischemia, which were new compared to her previous baseline ECG (Fig. 2). Pharmacologic stress testing using regadenoson was performed, with re-emergence of the LBBB at 94 bpm, associated with her usual non-radiating, left-sided chest discomfort. Given the persistence of lingering chest discomfort while the patient was being observed after regadenoson was stopped, it was decided to proceed to coronary angiography. Left ventriculography was normal, with an estimated ejection fraction of 55%, and no mitral regurgitation. Coronary arteries were normal. Review of her pharmacologic stress test results revealed normal stress and rest myocardial perfusion with no evidence of ischemia; LVEF was 55%, in the normal range, and regional wall motion was normal. A follow-up visit three weeks later documented better blood pressure and heart rate control on metoprolol and lisinopril, with improved symptoms.

Discussion

Chatterjee et al. [6] described massive T wave inversion and ST depression in the unpaced ECG subsequent to ventricular pacing, which persisted for a varying length of time depending on the duration of pacing. Although similar findings of T wave inversions were subsequently reported with intermittent LBBB [7], it was not until 1982 when Rosenbaum et al. [8] coined the term "cardiac memory" to describe the phenomenon of reversible T-wave changes following abnormal ventricular rhythms. They illustrated alterations in the sinus T-waves that seemed to reflect the major vector of the

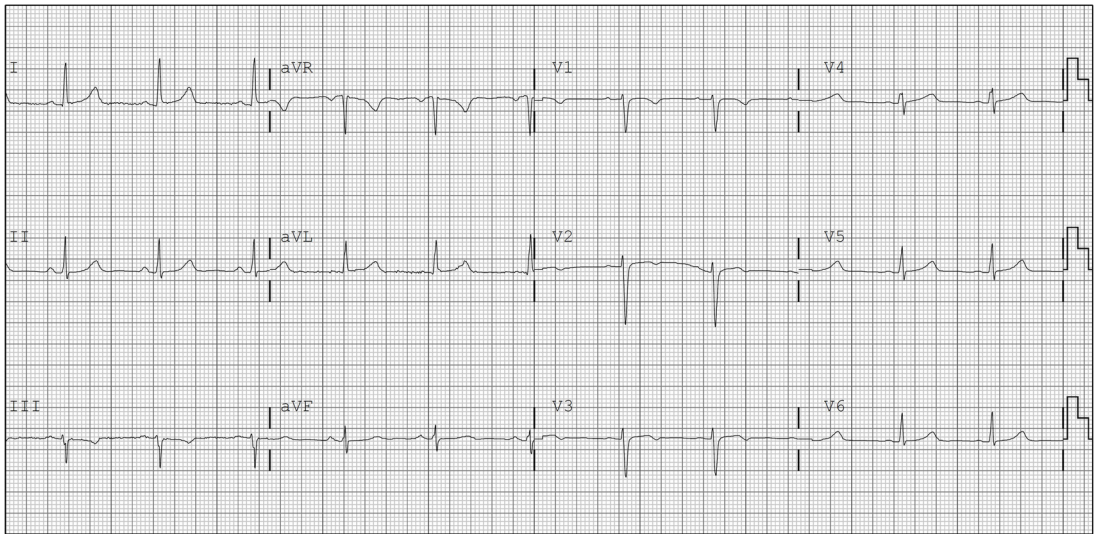

Fig. 2. Baseline electrocardiogram, seven years earlier, reveals sinus rhythm with non-specific anterior T wave abnormalities.