Worth Remembering: Cardiac Memory Presenting as Deep Anterior T-Wave Inversions Explained by Intermittent Left Bundle Branch Block

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Cardiac memory is a common cause of deep T-wave inversions (TWI) in the anterior precordial leads and can be difficult to distinguish from alternative causes of TWI such as myocardial ischemia. Cardiac memory is generally a benign condition except in the setting of prolonged QT when it can contribute to the precipitation of torsades de pointes. Herein, we describe the presentation and clinical course of a case of cardiac memory due to intermittent left bundle branch block (LBBB) that presented asymptomatically to our outpatient cardiology clinic with deep anterior TWI. We discuss common causes of and mechanisms underlying cardiac memory and how to distinguish it from alternative causes of TWI based on 12-lead electrocardiogram. In conclusion, intermittent LBBB is an under-recognized cause of cardiac memory that can present as deep anterior TWI mimicking cardiac ischemia, and awareness of this clinical entity may help prevent unnecessary invasive and expensive testing on otherwise healthy patients. **(Am J Cardiol 2020;135:174−176)**

Cardiac memory refers to the characteristic repolarization abnormalities on electrocardiogram (ECG) in patients whose QRS waveform returns to normal ventricular conduction following a period of abnormal ventricular depolarization.[1] In these patients, the vector of the precordial T-wave mirrors (or "remembers") the direction of the major QRS complex deflection during the period of abnormal depolarization. Cardiac memory is an under-recognized cause of T-wave inversions (TWI), particularly in patients with pacemakers.[1,2] Here, we present a rare case of cardiac memory due to intermittent LBBB and discuss the diagnostic and management considerations for patients who present with deep anterior TWI on ECG.

Case Description

A 74-year-old woman with known rate-dependent left bundle branch block (LBBB; **Figure 1**) presented to the outpatient cardiology clinic for routine follow-up. A 12-lead electrocardiogram (ECG) demonstrated new deep anterior T-wave inversions (TWI) in leads V_1 through V_4 (**Figure 2**). The patient was asymptomatic. Her blood pressure was 130/80 mm Hg, pulse 78 beats/minute and O_2 saturation 95%. The physical exam was unremarkable. Previous studies had disclosed calcific coronary atherosclerosis on computed tomography and a small previous myocardial infarct on myocardial perfusion imaging. Serum troponin levels were normal and there were no wall motion abnormalities on echocardiogram. Coronary angiography showed normal coronary arteries. The patient was prescribed a 7-day Zio-XT (iRhythm Technologies) continuous ambulatory ECG monitor (**Figure 3**).

Her ECG on presentation (**Figure 2**) demonstrates sinus bradycardia; left ventricular enlargement by Cornell, Peguerro LoPresti and total 12-lead QRS voltage criteria;[3] ST-segment depression in inferior leads II, III, and aVF; and marked deep TWI in anterior precordial leads V_1 through V_4. The T-wave axis mimics that of LBBB. Continuous ambulatory ECG monitoring captured 2 instances of interventricular conduction delay (**Figure 3**). Zio-XT records only a single channel, precluding accurate characterization of the conduction delay. However, based on the patient's previous 12-lead ECG, it is likely LBBB. **Figure 3** demonstrates the waveform transitioning from LBBB pattern to a normal conduction pattern with TWI following a premature ventricular contraction (PVC; circled). This transition is likely to have occurred due to the longer cycle created by the fully compensatory pause following the PVC. **Figure 3** shows spontaneous conversion from LBBB to normal conduction, this time due to a decrease in the sinus rate. Together, **Figure 3** shows that the LBBB is dependent upon higher heart rates and shorter cycle lengths. Based on the presence of anterior TWI following episodes of LBBB, the TWI was attributed to cardiac memory due to intermittent LBBB. The patient continues to do well 6 months later without further intervention.

Discussion

The differential diagnosis for anterior TWI includes several potentially life-threatening conditions including proximal left anterior descending artery lesion (Wellens' syndrome), posterior myocardial infarction, pulmonary embolism, pericarditis, hypokalemia, or central nervous system events.[2] Other potential causes include myocarditis and cardiac memory. Given the patient's history of known

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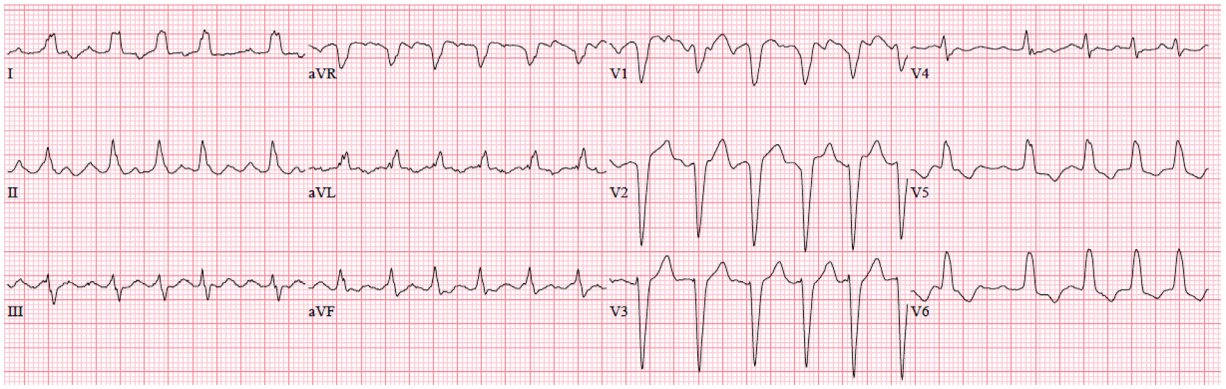

Figure 1. ECG obtained during previous admission demonstrating rate-related LBBB during a transient episode of atrial flutter.

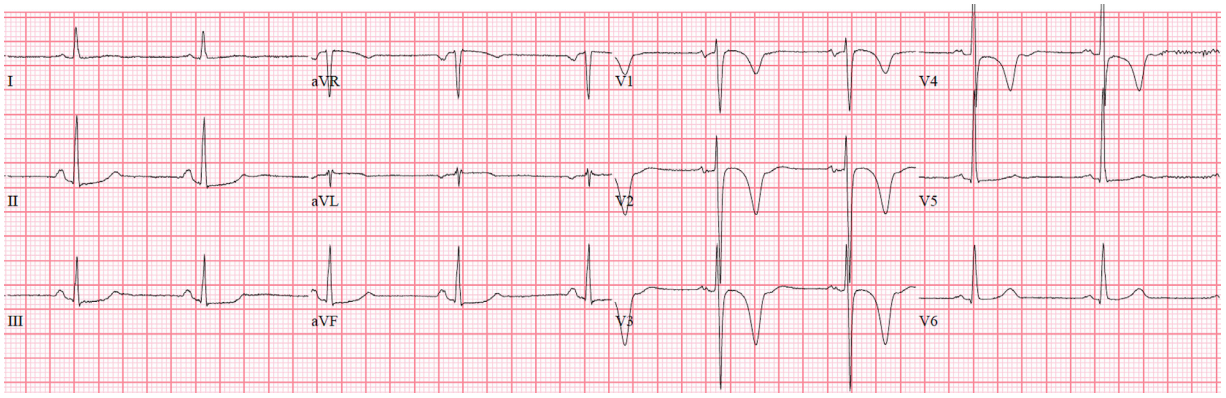

Figure 2. Twelve-lead ECG obtained previous to admission.

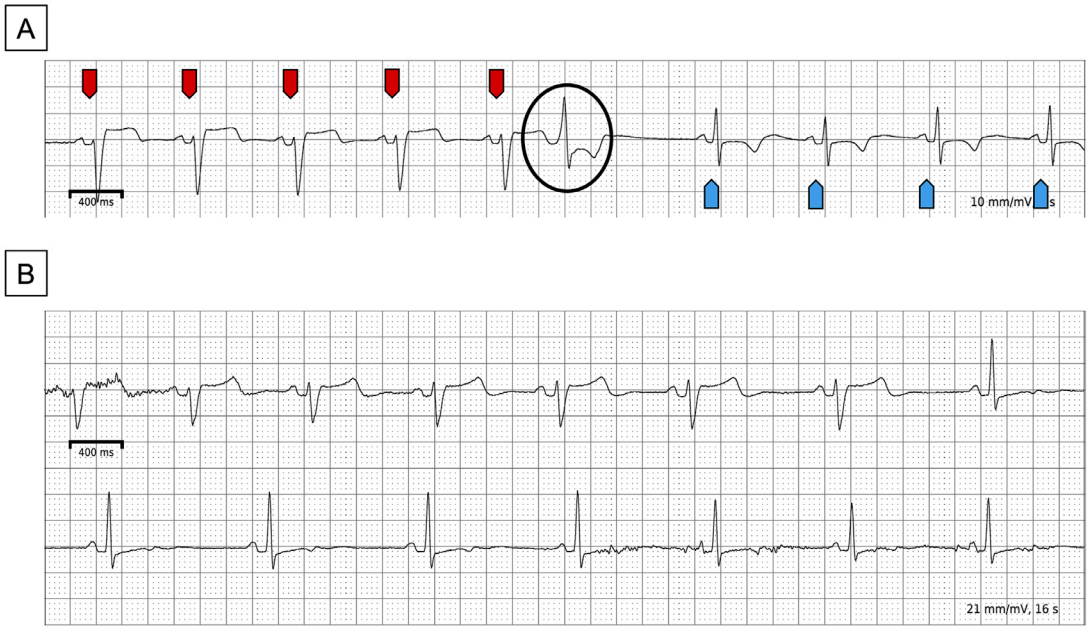

Figure 3. Continuous ambulatory ECG strips following hospital discharge. Top arrows indicate left bundle branch block (LBBB). Bottom arrows indicate normal conduction following the circled premature ventricular contraction.

atherosclerotic disease, her presentation was most concerning for Wellens' syndrome. However, this was ruled out by the presence of angiographically normal coronary arteries. Capturing these events on ambulatory ECG monitoring supported the diagnosis of cardiac memory, which is otherwise a diagnosis of exclusion.

Cardiac memory is most commonly seen in patients with pacemakers whose rhythm returns to sinus rhythm

Table 1
Proposed ECG criteria for differentiating cardiac memory from acute coronary syndromes as the source of T-wave inversion

Study Characteristics	Nakagawa et al. (2016)[4]	Shvilkin et al. (2005)[5]
Mechanism of cardiac memory for participants in study	Idiopathic left ventricular tachycardia	Cardiac pacemaker
Sensitivity (%)	100	92
Specificity (%)	96	100
Criteria suggesting cardiac memory		
1	Positive T-wave in aVL	Positive T-wave in aVL
2	Negative or isoelectric T-wave in II	Positive or isoelectric T-wave in I
3	Negative T-wave in V_{4-6} OR QTc < 430ms	Maximal precordial T-wave inversion greater than the T-wave inversion in III

following a period of pacing, but it has also been reported in patients with intermittent LBBB, intermittent preexcitation and ventricular tachycardia.[1,4,5] The magnitude of the T-wave inversion is a reflection of the duration of the period of abnormal depolarization wherein longer periods of abnormal ventricular depolarization typically result in more prominent and enduring TWI, which can last from minutes to several days. This may explain the difference between the magnitude of TWI seen on ambulatory ECG in our patient following periods of LBBB (**Figure 3**), although the duration of preceding LBBB previous to these 2 recorded events is unknown. Two similar sets of criteria have been found to effectively differentiate cardiac memory from acute coronary syndromes (ACS) in patients with cardiac memory (**Table**).[4,5] In both studies, a positive T-wave in aVL was 100% sensitive for cardiac memory as a stand-alone criterion suggesting that negative or isoelectric T-waves in aVL should raise suspicion for alternative causes of TWI besides cardiac memory.

Mechanistically, repolarization abnormalities in cardiac memory are due to altered ventricular depolarization that causes myocardial stretch-induced changes in potassium and calcium handling, primarily the transient outward current I_{to}.[5−7] Abnormal ion handling then triggers a cascade of downstream autocrine and paracrine signaling, possibly triggered by release of angiotensin II, that change both ion channel conductance in the short term and expression in the long term.[6−8]

While generally thought to be a benign, cardiac memory may be arrhythmogenic, most notably with QT-prolonging medications or conditions.[9] Two cases have been described showing that the combination of QT-prolongation and cardiac memory has the potential to precipitate torsade de pointes (TdP).[10,11] In both, alteration of pacemaker settings was sufficient to prevent future episodes of TdP. Further research is needed to determine what, if any, additional interventions may be needed in this population.

Declaration of Interests

The authors declare that they have no known competing financial interests or personal relations that could have appeared to influence the work reported in this study.

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