CASE REPORT

Cardiac memory: an under-recognised cause of deep T wave inversion in a patient presenting with chest pain

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SUMMARY

T wave inversion (TWI) has many differential diagnoses with acute myocardial ischaemia being the highest on the list of potential causes. Cardiac T wave memory is a benign, under-recognised and clinically important phenomenon seen after periods of altered ventricular conduction. After normal ventricular conduction is restored, the T wave 'remembers' and mirrors the direction of the wide QRS complex. Therefore, negative T waves are seen in leads that had negative wide QRS complexes. We describe the case of a 60-year-old truck driver with chest pain, deep TWI and traditional cardiovascular risk factors. After ruling out significant myocardial ischaemia, it was crucial to determine the cause of his T wave changes to provide reassurance and provide commercial license medical clearance. While it is currently a diagnosis of exclusion, it remains an important clinical entity for clinicians to recognise to provide an explanation for certain T wave changes to avoid future unnecessary cardiac testing.

BACKGROUND

Cardiac memory is a term used to describe the process in which a T wave maintains, or 'remembers', the vector of a preceding abnormal QRS complex once normal ventricular conduction is restored. In other words, once a previously wide QRS complex on the ECG returns to a narrow QRS complex, the T wave tracks the direction of the preceding wide QRS complex. The longer the duration that abnormal ventricular conduction occurs, the greater the degree and persistence of the resulting T wave on return of normal ventricular conduction.¹

Cardiac memory is associated with a number of conditions, such as intermittent left bundle branch block, ventricular pre-excitation, ventricular extra systole, right ventricular pacing and ventricular tachycardia.²⁻⁶ As a result of this phenomenon, T wave inversion (TWI) can be intermittently present on the ECG in patients who experience changes to normal ventricular conduction, as seen in the above conditions.

While cardiac memory is generally considered benign,⁷ and can occur in hearts that have no evidence of haemodynamic instability, structural abnormalities or ischaemic compromise,¹ the subsequent TWI on restoration of normal ventricular conduction can mimic the ECG findings seen in pathological conditions such as myocardial ischaemia or infarction. While cardiac electrophysiologists (EPs) are familiar with this phenomenon in the electrophysiology laboratory, many non-EP cardiologists and general physicians are not widely aware of this important clinical entity.

CASE PRESENTATION

A 60-year-old truck driver presented to our emergency department with chest pain. His medical history included smoking and hypertension with perindopril being his only regular medical therapy.

He described a 3-day history of chest pain. The pain was pleuritic in nature, worse on lying flat and mostly occurred at rest. It was not associated with any other symptoms. Physical examination was unremarkable with dual heart sounds, no murmurs and clear chest on auscultation. His first 12-lead ECG (figure 1) was consistent with normal sinus rhythm and left bundle branch block. As part of his routine

Figure 1 Sinus rhythm with left bundle branch block.

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Figure 2 Sinus rhythm with deep T wave inversion in the precordial leads.

assessment, a second 12-lead ECG (figure 2) was performed 2 hours later, which showed resolution of left bundle branch block but development of new deep TWI in the precordial leads. Serial cardiac troponins were negative (<0.015 ng/mL) with a negative D dimer. He had a mildly elevated C-reactive protein (CRP) and white cell count (WCC).

The history of chest pain, although atypical, coupled with the above dynamic changes in the 12-lead ECG recordings, was suspicious for coronary artery ischaemia, which prompted a referral for inpatient invasive coronary angiography. This was performed via the right radial artery and showed non-obstructive coronary arteries with preserved left ventricular function. The pain responded to simple analgesia, and the patient was discharged home 2 days later.

OUTCOME AND FOLLOW-UP

Our patient's presentation was likely consistent with acute pericarditis and his pain resolved with anti-inflammatory medication. The dynamic T wave changes observed on his ECG were due to cardiac memory. To further examine the cause of his intermittent left bundle branch block and exclude other significant myocardial structural abnormalities, he underwent a transthoracic echocardiogram and cardiac MRI, both of which did not show any significant structural abnormalities.

Given the benign nature of cardiac memory, the patient was given medical clearance for his commercial licence and was handed copies of his ECGs and medical summary to avoid future unnecessary cardiac testing should he present to another institution.

DISCUSSION

Myocardial ischaemia must always be considered as a differential diagnosis in patients presenting with TWI, particularly when it is associated with chest pain as was the case in our patient's presentation. Deep TWI in the precordial leads can represent significant stenosis of the left anterior descending coronary artery; hence, a diagnosis of cardiac memory should not be made until coronary artery ischaemia is excluded. Other causes of TWI that need to be considered include hypertrophic cardiomyopathy, right ventricular strain from conditions such as pulmonary embolus and metabolic disturbances.⁸

Intermittent left bundle branch block, as seen in our patient, resulted in precordial TWI. This is a recognised cause of cardiac memory.⁹ However, given cardiac memory can persist for weeks, not every patient will present with ECGs demonstrating serial changes that occur with altered ventricular conduction. This makes recognition of cardiac memory difficult in many cases.¹³

Distinguishing TWI due to cardiac memory from that of ischaemia poses a challenge. It has been proposed that cardiac memory may result in ECG changes that are distinct from ischaemic precordial TWI, thus eliminating the need for invasive work-up. In a

study of 60 patients comparing TWI from cardiac memory induced by ventricular pacing, with those from ischaemia, the combination of (1) a positive T wave in aVL, (2) a positive or isoelectric T wave in lead I and (3) a maximal precordial TWI that is greater than the TWI in lead III was 92% sensitive and 100% specific for cardiac memory.¹⁰ Another study of 64 patients identified that TWI in cardiac memory following idiopathic left ventricular tachycardia was similar to TWI induced by acute coronary syndrome involving the right coronary artery. However, they found that (1) a positive T wave in aVL, (2) a negative or isoelectric T wave in lead II and (3) a negative T in V_{4-6} or (4) $QTc < 430$ ms were 100% sensitive and 96% specific for the cardiac memory group.¹¹

While both of the above criteria are met in the ECG of our patient, larger studies are needed to externally validate these criteria. The studies also did not look at intermittent left bundle branch block, which was the cause of cardiac memory in our patient. Without

Patient's perspective

I was happy to know that I did not have coronary artery disease but was more reassured to get down to the cause of my ECG changes and know that cardiac memory is a benign condition. I will be able to go back to driving my truck.

Learning points

- ▶ Cardiac memory is an under-recognised, but important clinical phenomenon.
- ▶ It occurs after a period of altered ventricular conduction (right ventricular pacing, left bundle branch block or ventricular arrhythmia) is restored to normal conduction.
- ▶ The T wave 'remembers' the direction of the preceding abnormal QRS complex.
- ▶ Hence, deep T wave inversion (TWI) can be observed after a period of left bundle branch block.
- ▶ While robust criteria are still being validated to differentiate cardiac memory from ischaemic T wave changes, myocardial ischaemia should also be the top differential for dynamic T wave changes.
- ▶ It is important for clinicians to recognise cardiac memory, especially in patients where ischaemia and other causes of pathological TWI have been excluded to provide further reassurance and potentially avoid future unnecessary testing.

fully validated tools to distinguish cardiac memory from ischaemia, features on ECG alone cannot be used.

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