CASE REPORT

Cardiac Memory, an Underdiagnosed Condition

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Introduction

Cardiac memory, also termed Chatterjee phenomenon, is a known yet uncommonly recognized entity in which myocardial repolarization is altered after abnormal ventricular activation, such as with artificial pacemakers, intermittent left bundle branch block, ventricular premature beats, ventricular pre-excitation or episodes of tachycardia.[1] This phenomenon is characterized by persistent but reversible T-wave changes on surface electrocardiogram (ECG) induced by an abnormal electrical activation pattern. The extent and direction of T-wave deviation depend on the duration and direction of abnormal electrical activation and can persist for several weeks. Rosenbaum et al.[2] found that the T-wave "resembled" the abnormal QRS vector. The pathophysiology underlying cardiac memory is still complex and evolving, but alterations in the transient outward potassium current — Ito1 — and changes in the phosphorylation status of the cAMP responsive element binding protein (CREB) have been reported.[3]

Although it seems that cardiac memory is a relatively benign pathophysiologic finding, it may lead to unnecessary and invasive diagnostic investigation if it is not recognized.

Here we report a patient presenting deep T-wave inversion after implantation of cardiac pacemaker.

Keywords

Electrocardiography; Pacemaker Artificial, Pre-Excitation, Mahaim; Myocardial Contraction/physiopathology.

Case report

We report the case of a 71-year-old woman with history of hypertension, hyperlipidemia and obesity admitted in the cardiology ward with syncope. ECG revealed advanced atrioventricular block and a narrow ventricular escape rhythm with a heart rate of 35 beats / minute, without evidence of ischemia or altered repolarization (Figure 1).

Transthoracic echocardiogram revealed normal left ventricular mass and function and no significant valve abnormalities. Metabolic causes for atrioventricular block were ruled out. Dual-chamber pacemaker was implanted without complications and she was discharged asymptomatic four days after admission.

She was followed up in cardiology consultation as well as pacemaker consultation for routine pacemaker interrogation. Four months after device implantation, pacemaker analysis revealed paroxysmal atrial fibrillation that was not previously known. Her 12-lead EKG revealed sinus rhythm and T wave inversion in leads DII, DIII and aVF and V3 to V6 (Figure 2).

The patient was completely asymptomatic and did not change any of her regular medication: lisinopril, atorvastatin, and omeprazole. Hypertension was medically controlled and cardiopulmonary and neurological tests were unremarkable. Transthoracic echocardiogram was repeated, revealing similar findings to the echocardiogram performed before pacemaker implantation, namely normal left ventricular mass and function, no valve abnormalities, no pericardial effusion and correct placement of pacemaker electrodes. She also underwent pharmacological stress echocardiogram, which proved negative to myocardial ischemia. Based on this clinical scenario, the final diagnosis of cardiac memory was assumed.

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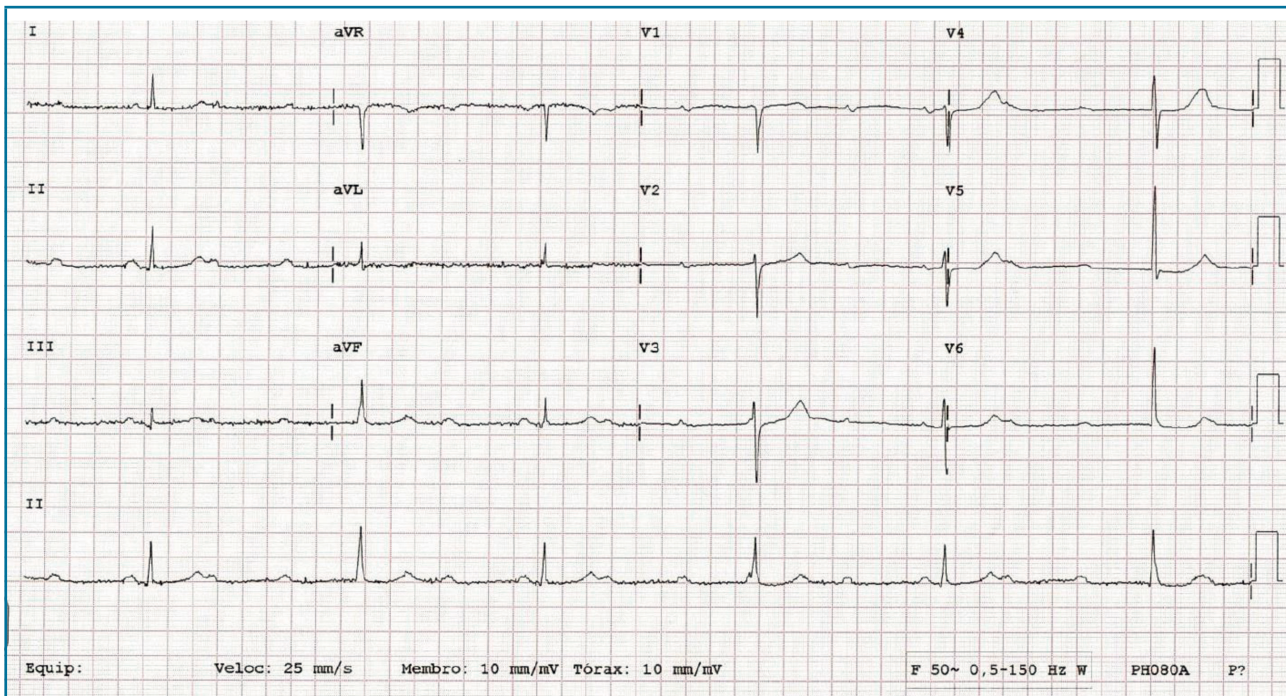**Figure 1** – *Electrocardiogram showing advanced atrioventricular block at hospital admission*
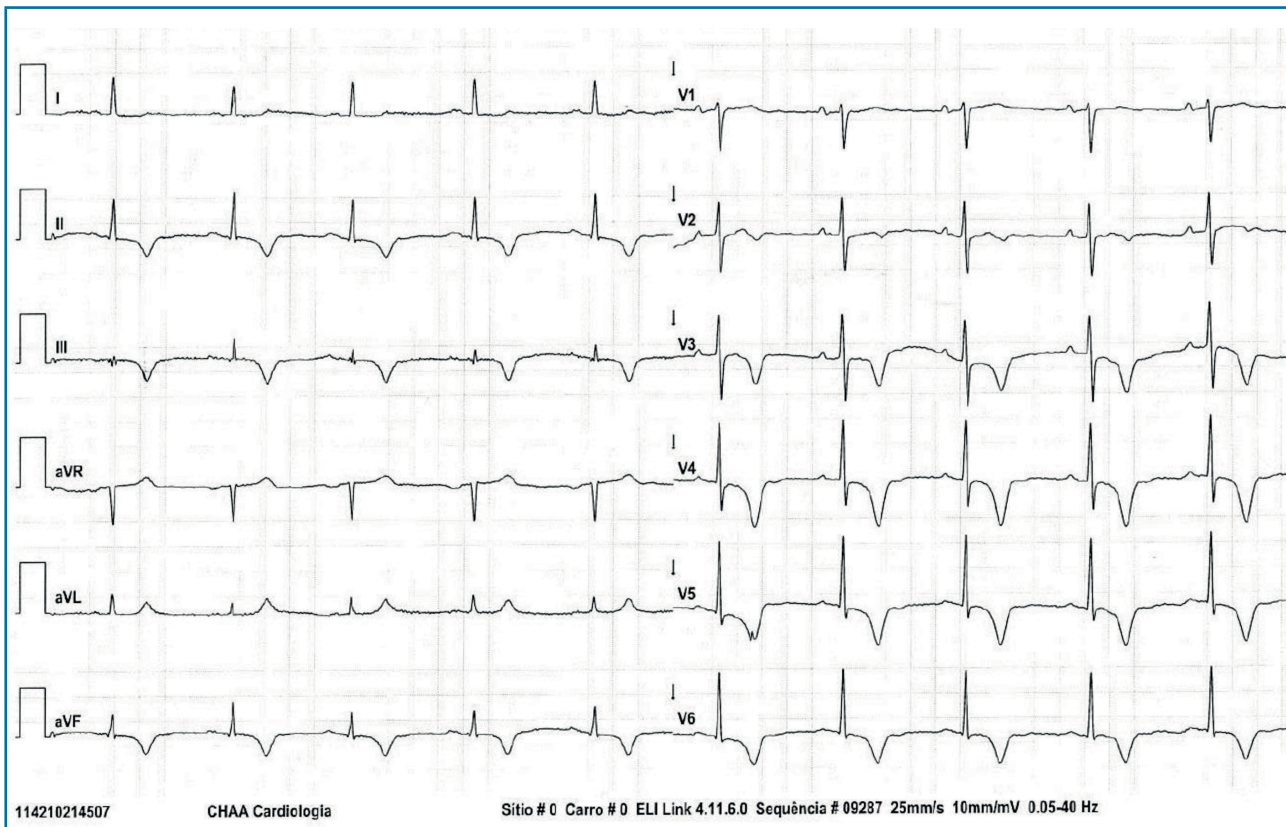

Figure 2 – *Electrocardiogram showing T-wave inversion in leads DII, DIII and aVF and V3 to V6 in the follow-up after pacemaker implantation*

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Discussion

Cardiac memory is an underdiagnosed condition and physicians should be aware of this diagnosis to avoid unnecessary tests, hospital admissions and health costs. Our clinical case raises the awareness for this diagnosis among physicians.

Diagnosis of cardiac memory is based on defining the phenomenon itself. It is an exclusion diagnosis when a patient presents with new altered T-waves after a period of abnormal electrical activation and once we have ruled out other causes for repolarization changes. It is also reported that the combination of (a) positive T-wave in lead aVL, (b) positive or isoelectric T-wave in lead I, and (c) maximal voltage of T-wave inversion in precordial leads higher than in lead III has been shown to be associated to a sensitivity of 92% and a specificity of 100% for the diagnosis of cardiac memory.[4] It is also helpful, when possible, to document the normalization of repolarization changes.

Ruling out other causes for T-wave inversion is essential for the diagnosis of cardiac memory. There are several causes for T-wave inversion: myocardial ischemia, intracranial events, left ventricular hypertrophy, pericarditis, pulmonary embolus, myocardial contusion, mitral valve prolapse, myocarditis, apical septal hypertrophy, medications etc. Myocardial ischemia is one of the most serious and worrying diagnoses to be ruled out. It demands a careful and focused clinical history and an ischemia diagnostic test, in some cases, and even an invasive diagnostic method. It is also important to rule out intracranial bleeding by performing neurological tests and, when there are still doubts, computed tomography (CT) of the head. Echocardiogram can also help ruling out regional wall motion abnormalities, hypertrophy, mitral valve prolapse, pericardial fluid commonly present in pericarditis and cardiac repercussion of a significant pulmonary embolism. It is also important to review all medications taken by the patient, mainly antiarrhythmic drugs that can alter cardiac repolarization.

In our clinical case, clinical history and full physical examination, including neurological tests, allowed us to rule out several causes of new T-wave inversion, namely acute coronary syndrome, acute myocarditis, pulmonary embolism, stroke, status epilepticus or medications. Our patient also presented ECG clues, pointing towards the diagnosis of cardiac memory (Figure 2). Echocardiogram also allowed us to rule out several causes of T-wave inversion, such as left ventricular dysfunction, regional wall motion abnormalities due to myocardial infarct or myocarditis, left ventricular hypertrophy or pericardial effusion. Considering the patient age and the coexistence of cardiovascular risk factors, we decided to perform a pharmacological stress echocardiogram to rule out coronary heart disease. After a negative result in stress echocardiogram we assumed the diagnosis of cardiac memory.[4]

Cardiac memory was first recognized in 1940, but was named and studied in 1982 by Rosenbaum et al.[2] Electrophysiological remodeling following altered activation is characterized by distinct changes in the proximal (early-activated) versus distal (late-activated) areas from the site of altered activation. During ventricular activation, by artificial pacemaker, for example, there is a change in the action potential duration in the early (mild prolongation) and late (significant prolongation) activated myocardial areas. Consequently, regionally heterogeneous action potential remodeling occurs, which enhances regional repolarization gradients. This changes the repolarization vector direction that underlies the electrophysiological basis for T-wave memory(5). Several mechanisms have been proposed to underlie the development of cardiac memory: altered expression of transient outward potassium current, angiotensin II receptor-mediated signaling and, more recently, mechanical-strain-induced changes in action potential.

Despite early beliefs that cardiac memory was benign, some detrimental consequences of altered ventricular activation have been discussed, which include worsened mechanical function[5] and increased susceptibility to arrhythmias.[6] In addition, cardiac memory can also be responsible for patients being submitted to invasive diagnostic tests that put patients under unnecessary risks.

Pacing is an evolving area in cardiology and we are just starting to understand the pathophysiological consequences of the implantation of these devices. Cardiologists should definitely be aware of cardiac memory and be able to raise this suspicion whenever indicated, avoiding further unnecessary diagnostic workup.

Author contributions

Conception and design of the research: Oliveira M, Azevedo O, Ribeiro S. Acquisition of data: Oliveira M, Calvo L, Ribeiro S. Writing of the manuscript: Oliveira M, Calvo L, Faria B. Critical revision of the manuscript

for intellectual content: Azevedo O, Faria B, Ribeiro S, Lourenço A.

Potential Conflict of Interest

Sources of Funding

Study Association

References

1. Katz AM. T wave 'memory': possible causal relationship to stress-induced changes in cardiac ion channels? J Cardiovasc Eletrophysiol. 1992;3(2):150-9.

2. Rosenbaum MB, Blanco HH, Elizari MV, Lázzari JO, Davidenko JM. Eletrotonic modulation of the T wave and cardiac memory. Am J Cardiol. 1982;50(2):213-22.

3. Ozgen N, Rosen MR. Cardiac memory: a work in progress. Heart Rhythm. 2009;6(4):564-70.

4. Shvilkin A, Ho KK, Rosen MR, Josephson ME. T-vector direction differentiates postpacing from ischemic T-wave inversion in precordial leads. Circulation. 2005;111(8):969-74.

5. Jeyaraj D, Ashwath M, Rosenbaum DS. Pathophysiology and clinical implications of cardiac memory. Pacing Clin Electrophysiol. 2010;33(3):346-52.

6. Haverkamp W, Hordt M, Breithardt G, Borggrefe M. Torsade de pointes secondary to d,l-sotalol after catheter ablation of incessant atrioventricular reentrant tachycardia--evidence for a significant contribution of the "cardiac memory". Clin Cardiol. 1998;21:55-8.