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EC PULMONOLOGY AND RESPIRATORY MEDICINE

Case Report

Cardiac Memory T Cells: A Benign Phenomenon

Rajan Kumar[1] and Archana Sinha[2]*

[1]*Department of Internal Medicine, Howard University Hospital, Washington DC, District of Columbia, USA*

[2]*Department of Cardiology, Ozarks Medical Center, West Plains, Missouri, USA*

***Corresponding Author:** Archana Sinha, Department of Cardiology, Ozarks Medical Center, West Plains, Missouri, USA.

Received: February 05, 2018; **Published:** March 22, 2018

Abstract

Cardiac ischemia is possible when a patient seeks medical attention for chest pain with deep T-wave inversions on electrocardiogram (EKG). Besides cardiac ischemia, other potential causes of T wave inversions on EKG are acute cerebral events, apical hypertrophic cardiomyopathy as well as cardiac memory T cells. Cardiac memory represents a benign phenomenon. In this article, we illustrate a case of cardiac memory related T wave inversion and review the pathophysiology and proposed EKG diagnostic criteria.

Keywords: *T Wave Inversion; Cardiac Ischemia' Cardiac Memory T Cells*

Introduction

Cardiac memory is a process whereby precordial T waves remain persistently inverted with respect to the QRS complex following abnormal myocardial depolarization patterns usually a bundle-branch block, ventricular pacing (right ventricular or biventricular pacing), ventricular arrhythmias, or Wolf-Parkinson-White syndrome/atrioventricular bypass tract. It is still a poorly understood process, in spite of being first described in 1980's [1].

T wave changes of cardiac memory mimic those of ischemic heart disease and can also impact the effects of anti-arrhythmic drugs and of other drugs that affect K+ channel function on EKG [1-3]. Cardiac memory may often be unrecognized, but it has potentially important clinical implications, such as alteration in the action of anti-arrhythmic drugs, with reduced efficacy or potential pro-arrhythmic effects [3].

Case Presentation

84-year-old Caucasian gentleman presented to the emergency room with complaints of dull diffuse abdominal pain, distension and uncharacterized chest pain. He also complained of associated shortness of breath. He has past medical history of paroxysmal atrial fibrillation, coronary artery disease with multiple remote coronary interventions, hypertension, dyslipidemia and history of implantable cardioverter defibrillator for ischemic cardiomyopathy.

EKG showed atrial fibrillation with demand ventricular paced pacing. T wave inversion in anterolateral and inferior leads (Figure 1). Previous EKG showed biventricular paced rhythm with upright T waves (Figure 2). His labs were within normal limits including troponins were negative. CT scan of head did not reveal any significant intracranial abnormalities. Recent pharmacological stress test did not show any evidence of ischemia. He refused to stay in the hospital and left against medical advice. He came again the following day with similar complaints and troponin was still negative. His follow up EKG showed ventricular paced rhythm with upright T waves (Figure 3). He was treated for gastritis.

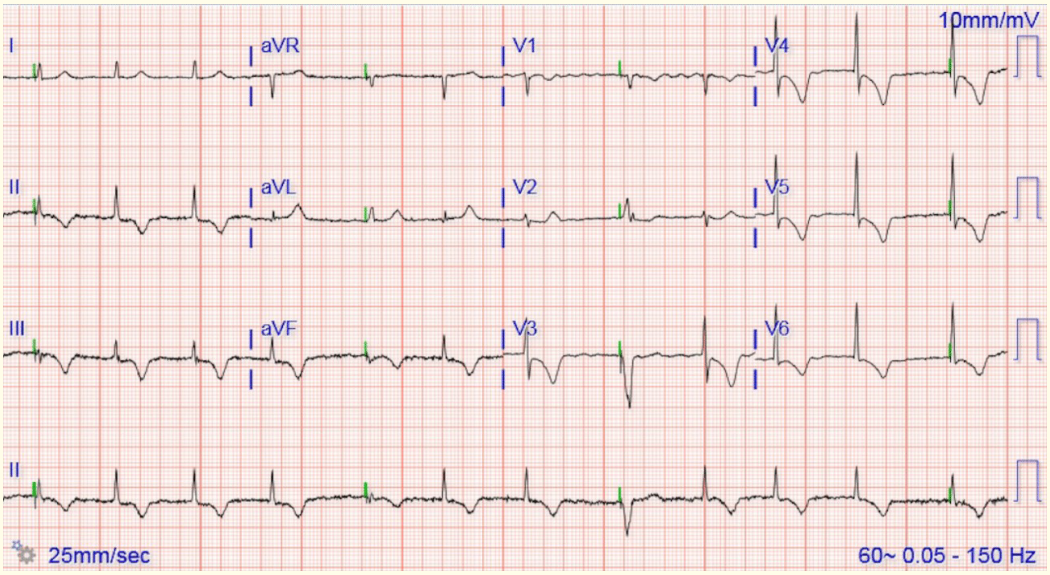

Figure 1: *Atrial fibrillation with demand biventricular pacing. T wave inversion in anterolateral and inferior leads.*

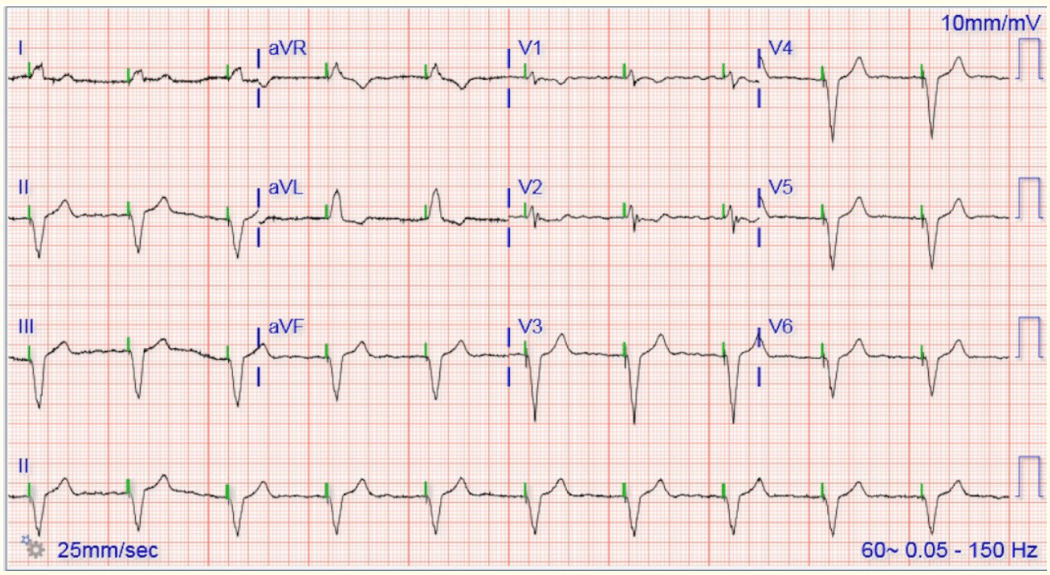

Figure 2: *Biventricular paced rhythm with upright T waves.*

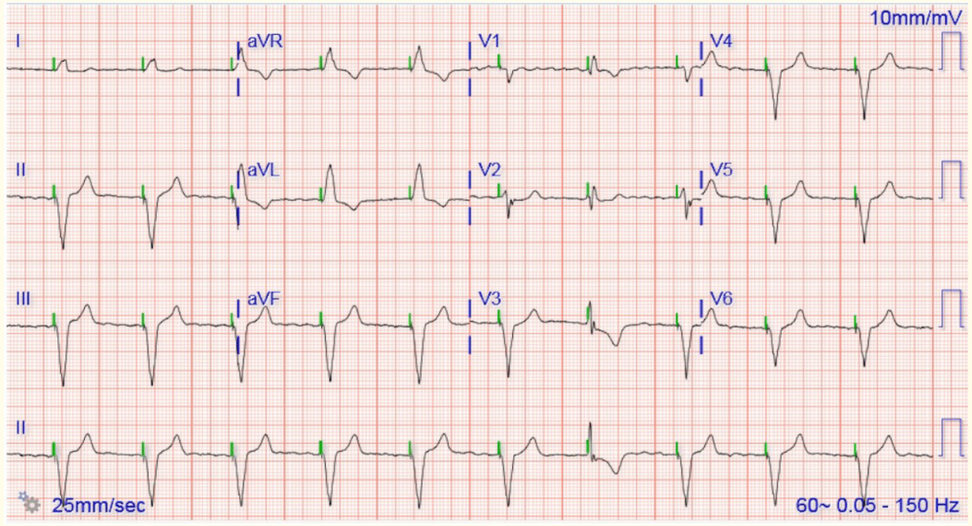

Figure 3: *Right ventricular paced rhythm with upright T waves and occasional biventricular paced rhythm with inverted T wave.*

Our patient was seen in office in a week and was doing well. His chest pain had resolved. Our patient's memory T cells was secondary to ventricular pacing, so no further evaluation and therapy was warranted.

Differential diagnosis

T wave inversions can be seen in ischemia, intracranial bleed, apical hypertrophic cardiomyopathy, stress cardiomyopathy and pulmonary edema. Our patient's troponins were negative. CT scan did not show any acute intracranial bleed or ischemia. His previous echocardiogram showed no evidence of hypertrophic cardiomyopathy. His follow up EKG and negative troponins further confirmed the findings.

Discussion

Cardiac memory is used to describe a specific form of re-modelling seen as an altered electrocardiographic T wave [1]. After an interval of ventricular pacing or arrhythmia, when sinus rhythm returns the T wave persists in tracking the vector angle and amplitude of the QRS complex that characterized the paced or arrhythmic state. It can also be present when one wide complex rhythm is replaced by another e.g. ventricular paced rhythm with LBBB. Although normally concerning for ischemia, T-wave inversions in the case of cardiac memory have no correlation with myocardial ischemia. Such changes may persist for weeks after the inciting abnormal depolarization, although the exact temporal relationships are not well known. Extensive workups under these circumstances including cardiac catheterization may not be warranted. EKG criteria (Table 1) have been proposed that may be useful in discriminating an essentially benign condition from a potentially life-threatening one in the emergency department.

RV apical pacing [3]	Intermittent LBBB [4]	Wide complex tachycardia [4]	Ventricular pre-excitation [4]	Idiopathic LV tachycardia [5]
Positive T_{aVL} Positive or isoelectric T_I Maximal precordial TWI in precordial leads > TWI_{III} 92% sensitive 100% specific.	QRS complex axis in the frontal plane is directed more superiorly during right ventricular apical pacing than during LBBB.	T waves negative in those ECG leads in which the conditioning aberrant QRS complexes had been pre- dominantly negative.	A repetitive abnormal depolarization process giving rise to positive QRS complexes may induce subsequent tall peaked positive T waves whenever normal ventricular activation resumes.	Positive T in aVL Negative or isoelectric T in II Negative T in V_{4-6} QTc < 430 ms 100% sensitive and 96% specific

Table 1: *EKG criteria for Cardiac Memory T cells.*

Molecular Mechanisms in evolution of cardiac memory

Pacing alters activation and stretch, resulting in angiotensin II synthesis or release, trafficking and internalization of the AT1 receptor–Kv4.3–KChIP2 complex from its membrane site and a reduction in current. Increased Ca^{2+} from L type Ca^{2+}, Na^{+}–Ca^{2+} exchanger and stretch-activated channels may be a second messenger activating changes in nuclear transcriptional factors e.g. CREB, is reduced. However, long-term changes in Ito, IKr and ICa L have been demonstrated as well, all of which would be expected to contribute to the altered action potentials and ECG changes of cardiac memory [3].

Memory is a specialized form of stress response in both central nervous system and cardiovascular system. There is advancement of structure and function in the former and a return to an earlier structure/function entity in the latter. Cardiac memory creates action potential and cardiac repolarization phenotypes very similar to that in neonatal heart.

Conclusion

The awareness of cardiac memory T cells is important for health care providers, to facilitate appropriate evaluation and management. Differentiation between T wave inversion of memory and ischemia is important. Comparison to prior EKG's and use of diagnostic EKG criteria help in identifying cardiac memory T cells from ischemia. Cardiac memory, itself does not require any therapy.

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Volume 7 Issue 4 April 2018