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Case Report

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Subepicardial ischemia vs cardiac memory after tachycardia by intranodal reentry. case report and literature review

Abstract

Cardiac memory is an electrophysiological property that is expressed as persistent electrophysiological changes in response to variations in the rate and sequence of cardiac electric activation and is defined as the persistent changes of the T wave on the electrocardiogram after a period of rhythms of wide QRS widened, and that becomes evident once the normal ventricular activation pattern is restored.[1] The first evidence of its existence arises from case reports where changes in the polarity of the T wave were observed after transient episodes of tachyarrhythmias. Subsequently, it was demonstrated how ventricular activation by external sources in a prolonged way induces persistent changes in the electrocardiogram, and those changes persist after eliminating this source.[6,8] Electrical memory can occur in a short and long term, when it is short-term, the changes are due to the modulation of the electrical activity of individual cardiac cells or cell to cell interaction, while long-term memory involves the synthesis of proteins and / or structural changes.[8]

Keywords: electrical memory, long term. short term, tachyarrhythmias, T wave, ventricular activation, persistent changes, electrical activity

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Abbreviations: VAS, visual analogue acale; LVEF, left ventricular ejection fraction; cAMP, cyclic adenosine monophosphate; WPW, wolff parkinson white; TWI ,T wave inversion; LBBB, left bundle branch block

Introduction

T-wave inversion secondary to electrical memory changes after a period of ventricular activation should be recognized as a differential diagnosis of ischemic heart disease or other structural abnormalities.

Material and methods

The literature review was used on articles and medical journals about electrocardiographic alterations and diagnostic algorithms were carried out to identify it.

Case report

This is a 27-year-old male patient who was admitted to the emergency room due to the presence of palpitations, dizziness and stabbing in the precordium of intensity 8/10 on the VAS scale, without apparent radiation. The patient reports multiple episodes of ventricular tachycardia in the las 4 years, without data of hemodynamic instability in all the episodes, which remitted with pharmacological cardioversion amiodarone 150-300mg Table 1.

Table 1 History and medical interventions days

Diagnosis Date	20-Aug-17
Last Episode	28-May-22
Medical Intervention	4-Jun-22

Since his diagnostic in 2017, treatment was started with amiodarone 200mg every 12 hours for 6 months, with subsequent change to propafenone 150mg every 12 hours until the present. After admission to the room, an electrocardiogram was taken, showing QRS complex tachycardia (grater than 120 ms), with no data of hemodynamic instability, so it was decided to perdorm pharmacological cardioversion with amiodarone 300mg, achieving reversion to sinus rhythm. After cardioversion, a new twelve-lead electrocardiogram is taken for control (image 1) in which it is observed; sinus rhythm, heart rate 75 bpm, QRS: 80ms, aQRS: +60°, regular R-R, isoelectric ST segment, negative T waves in DII, DIII, aVF and precordial leads V3-V6, QTc (Bazzet): 447ms. Biochemical analyzes without alternations. Transthoracic echocardiogram with preserved biventricular systolic function, LVEF >60%, without focal or segmental mobility disorders Image 1.

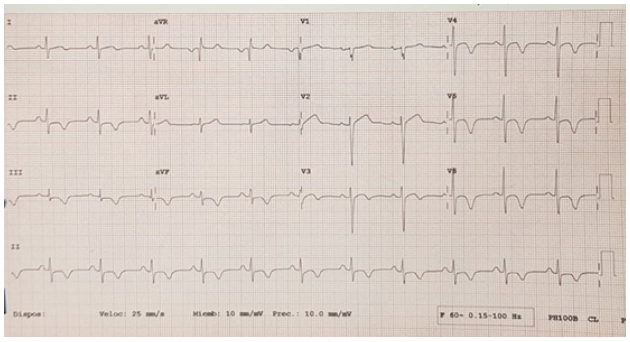

Image 1 12-lead electrocardiogram, sinus rhythm, FC 71bpm, aQRS -30°, PR 130ms, QRS 100ms, QT 400ms, QTc (Bazzet) 435ms, ST; inverted T wave V4 toV6, DII, DIII and aVF.

Discussion

In 1915, Paul Dudley White described transient T-wave inversion after premature ventricular complexes. In 1940, a series of cases of electrical memory after paroxysmal tachycardia was described for the first time. It was believed that this was secondary to anoxia. and "fatigue" of the cardiac cell due to longer diastoles. In 1982 Rosenbaun et al.[1] introduced the term "cardiac memory" describing the case of T wave inversion.[1]

Cardiac electrical memory may be due to electrical stimulation by pacemakers, Wolf Parkinson White syndrome, left bundle branch block, widening of the QRS following calcium channel toxicity, secondary to propafenone administration, tachyarrhythmias, or Subepicardial ischemia secondary to obstruction of the left anterior descending artery (Wellens syndrome), this results from a stimulus that leads to a pattern of ventricular activation different from that imposed by sinus rhythm.[2,1]

Although this, in a variable period of time, may represent an explanation for the initiation of the impulse and myocardial activation, from the point of view of the previous history of the heart beats, this represents a rare event. If recurrent, this event can lead to changes, over minutes to hours, in gap junction density, myocyte size, repolarization pattern, ion currents, action potential, and T wave on the electrocardiogram. Activation sequence and recovery properties are strongly associated; Apparently, the areas of the ventricle that are activated early have a longer refractory period and those activated late have a shorter refractory period, which may be a protective mechanism for the formation of reentries. It has been proposed that the alteration in the electrocardiogram is secondary to the alteration of potassium channels and cAMP.

In different models, when the site of stimulation has been changed along with a new activation sequence, the repolarization sequence also changes, but it does not necessarily occur in the opposite direction of activation, as it normally would. Rosenbaum et al.[3] demonstrated that during the period of cardiac electrical memory, the T wave resembles the direction of the abnormal QRS vector.

Because cardiac memory can mimic T-wave inversions typical of myocardial ischemia, differentiation between these two events is important. Shvilkin et al, proposed in 2005 electrocardiographic criteria for the diagnosis of cardiac memory; presence of positive T wave in aVL, positive or isoelectric in DI, and greater T wave amplitude of any precordial lead compared to greater T wave polarity in DIII, with a sensitivity of 92% and specificity of 100 %.[1,4] Later, in 2016, T. Nakagawa et al, reproduced these criteria in a study that included 9 patients with T-wave inversion secondary to idiopathic left ventricular tachycardia and 48 patients with acute coronary syndromes, extra to the criteria proposed by Shvilkin et al, added the corrected QT measurement <430 ms, reaching a sensitivity of 100% and a specificity of 96% for the differential diagnosis of subendocardial ischemia as a cause of T wave inversion, likewise they demonstrate in this study the deviation of the QRS electrical axis between -120° and -30° compared to those patients with coronary artery disease.[5,1] However, these criteria may vary depending on the location of the source that promoted the abnormal ventricular activation Figure 1.

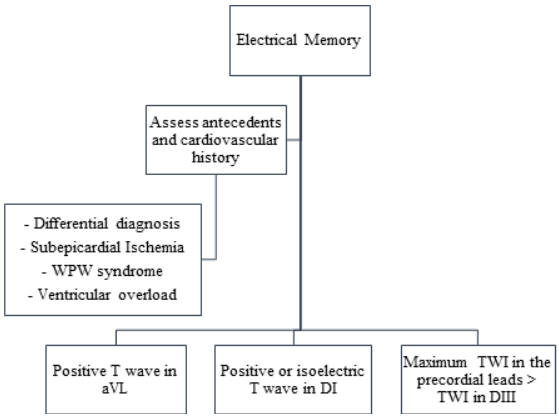

Figure 1 Diagnostic algorithm of electrical memory in case of inversion of T waves.

Conclusion

The T-wave memory phenomenon is perhaps the only widely validated electrical memory event, so it should be considered in the interpretation of such electrocardiographic deflection disorders. The interpretation of an inverted T wave should not be based towards a cause-effect event due to ischemia or hypertrophy, but the possibility that it is the consequence of a preceding tachyarrhythmia or bundle branch block should also be considered. The interrogation and the correct interpretation of the electrocardiogram using these diagnostic algorithms is very useful to make the differential diagnosis and thus avoid the misuse of personal or material resources.

Acknowledgments

This work was a pleasure to develop thanks to the contributions of the authors and collaborators.

Ethical statement

For the clinical case report, no animals were involved, nor was the physical integrity of the participants damaged during the entire investigation, complying with the regulations established by the "Committee of Ethics and Prevention of Conflict of Interest of Cardiovascular Institute of Puebla, Mexico".

Conflicts of interest

We don´t have any interest conflict, to publish this information about case report.

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