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Intermittent Preexcitation and Electrotonic Modulation of Repolarization as a Cause of Macroscopic T Wave Alternans

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Abstract - Alterations in ventricular depolarization are recognized to lead to inverted T-waves which manifest once the ventricular activation returns to normal. This phenomenon referred to as 'T-wave memory' or 'electrotonic modulation' may be caused by several clinical conditions producing a shift in ventricular depolarization. Currently, T wave alternans (TWA), either macroscopic or microscopic refers to the beat to beat alteration in the repolarization heterogeneity that repeats with every other beat without changes in the QRS complex. We present the case of a 43-year old man with symptomatic intermittent pre-excitation who underwent 24-hour Holter monitoring revealing TWA secondary to electrotonic modulation. It is assumed that this phenomenon does not carry any prognostic significance.

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Intermittent Preexcitation and Electrotonic Modulation of Repolarization as a Cause of Macroscopic T Wave Alternans

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Abstract - Alterations in ventricular depolarization are recognized to lead to inverted T-waves which manifest once the ventricular activation returns to normal. This phenomenon referred to as 'T-wave memory' or 'electrotonic modulation' may be caused by several clinical conditions producing a shift in ventricular depolarization. Currently, T wave alternans (TWA), either macroscopic or microscopic refers to the beat to beat alteration in the repolarization heterogeneity that repeats with every other beat without changes in the QRS complex. We present the case of a 43-year old man with symptomatic intermittent pre-excitation who underwent 24-hour Holter monitoring revealing TWA secondary to electrotonic modulation. It is assumed that this phenomenon does not carry any prognostic significance.

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I. Introduction

Transient changes in the sequence of ventricular depolarization are recognized to give rise to gradual changes in ventricular repolarization which manifests as T-wave inversion. Alterations in ventricular depolarization may be caused by several mechanisms including intermittent ventricular pacing, rate-dependent bundle branch block, tachyarrhythmias with aberrant QRS complexes and ventricular preexcitation. The resultant change in ventricular repolarization remains masked by the secondary T-wave changes induced by the conduction disturbance, and are only unveiled once normal ventricular activation is restored. This phenol-menon is referred to as 'T-wave memory' or 'electrotonic modulation'[1].

Reports on T wave alternans (TWA) refer to the beat to beat alternation in the repolarization heterogeneity that repeats with every other beat with the same QRS complex and has recently been linked with an increased vulnerability to ventricular arrhythmias in certain conditions such as myocardial ischemia, Prinzmetal's angina, states of altered autonomic tone, electrolyte abnormalities and in the long QT syndrome[2]. We present the case of intermittent pre-excitation as the cause of TWA secondary to electrotonic modulation.

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II. Case Report

A 43-year old man with symptomatic intermittent preexcitation underwent investigations with 24-hour Holter monitoring. The upper rhythm strip (Figure 1) demonstrates the presence of both preexcited (black arrow) and non-preexcited beats (dotted arrow). In the lower rhythm strip (Figure 1), the corresponding T-wave morphology is seen to alternate with a positive T-wave in the presence of a preexcited beat (grey arrow) and a negative T-wave in the presence of a non-preexcited beat (white arrow). The electrotonic modulation associated with the intermittent preexcitation produced TWA. The patient subsequently underwent an uncomplicated electrophy-siology study with successful radiofrequency ablation of a right mid-septal accessory pathway. Figures 2A (pre-ablation) and 2B (post-ablation) show the development of overt T-wave inversion in leads II, III and aVF (post-ablation of the accessory pathway) in association with normalization of the QRS complex as a manifestation of cardiac memory[3].

III. Discussion

The cellular and electrophysiological basis that gives rise to the T wave observed on a 12-lead electrocardiogram (ECG) remains controversial[4]. Under normal conditions, the concordant polarity of the T wave and the R wave in the surface ECG indicates that the repolarization sequence proceeds in the opposite direction to that followed by the depolarization process[5]. As the direction of ventricular repolarization depends on the course of ventricular depolarization, any shift of the latter results in an instantaneous modification of the T waves, whose spatial orientation tends to be opposite to that of the abnormal QRS complexes. Alterations in the direction of depolarization, through pre-excitation as highlighted in this case, may give rise to changes in ventricular repolarization that are unmasked once normal ventricular activation is restored.

In the present case, the conditioning stimulus giving rise to the altered depolarization, and subsequent repolarization, was only present in alternating beats which consequently resulted in the generation of TWA. TWA has been reported to be associated with an increased vulnerability to ventricular arrhythmias in a

variety of pathophysiological conditions. In the present case, we demonstrate that TWA may be caused by electrotonic modulation associated with intermittent pre-excitation, although there is no suggestion that in this context the TWA seen is of any prognostic relevance.

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Figure Legends

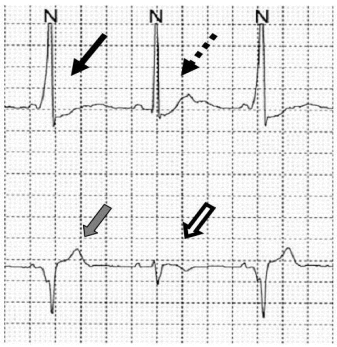

Figure 1

The upper rhythm strip demonstrates the presence of both preexcited (black arrow) and non-preexcited beats (dotted arrow). In the lower rhythm strip the corresponding T-wave morphology is seen to alternate with a positive T-wave in the presence of a preexcited beat (grey arrow) and a negative T-wave in the presence of a non-preexcited beat (white arrow). The electrotonic modulation associated with the intermittent preexcitation produced TWA.

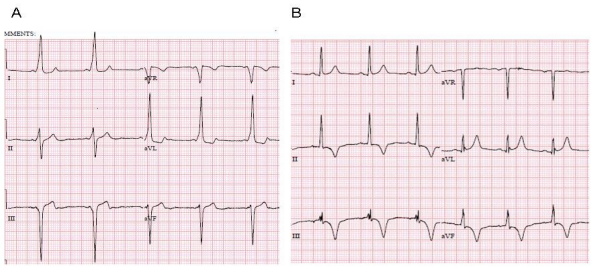

Figure 2

Limb leads pre (A) and post (B) ablation showing the development of overt T-wave inversion in leads II, III and aVF (post-ablation of the accessory pathway) in association with normalization of the QRS complex as a manifestation of cardiac memory.