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Preexcitation Syndrome and Myocardial Infarction a Case Report and Literature Review

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Abstract: The electrocardiogram allows easy recognition of myocardial infarction and preexcitation syndromes but may cause confusion when both conditions are present simultaneously. The preexcitation syndrome may either simulate myocardial infarction or mask it. A case report where myocardial infarction is simultaneously masked and mimicked by a preexcitation syndrome is presented.
Keywords: Preexcitation; Acccessory pathway Myocardial infarction; Electrocardiographic manifestations.

INTRODUCTION

Ventricular preexcitation results from premature activation of part or all of the ventricular myocardium by supraventricular impulses due to the existence of accessory conduction pathways that completely or partial bypass the normal conduction pathways [1].

In 1930, Wolff, Parkinson, and White described the syndrome as the most significant aspect of ventricular preexcitation, associating delta wave, short PR interval, and paroxysmal tachycardia, but the existence of accessory pathways of conduction had been reported since 1914 by Kent [2].

Myocardial infarction and preexcitation are two distinct electrocardiographic (ECG) entities, with marked effects on ventricular intramural conduction. When these effects occur simultaneously, they may result in confusing ECG patterns. This is demonstrated by the following observation [3, 4].

CASE REPORT

A 56-year-old male, chronic smoking, presenting with chest pain less than 12 hours from onset of symptoms. The 12 lead ECG (Figure 1) showed sinus rhythm with a rate of 63 beats per minute, positive delta waves in leads I, aVL, and V2 through V6, and negative delta waves in aVF, consistent with an posteroseptal located of accessory pathway, it's showed also Q-wave formation in leads III, and aVF. Enzymatic markers of myocardial necrosis were high. Ultrasound examination showed aspect of anterior myocardial infarction and no abnormalities in inferior wall with LVEF at 40%. An emergency coronary angiogram showed a proximal left anterior descending artery occlusion, which was successfully stented. Following this procedure, the patient became asymptomatic. In a new ECG performed 12 hours later (figure 2), there was no left ventricular preexcitation, inferior leads were normal, and Q waves remain visible in leads V1 through V4, indicating myocardial injury in the anterior wall.

DISCUSSION

Initial phase of QRS is modified by both ventricular preexcitation and myocardial necrosis. Depending on the location of the accessory pathway, and therefore the orientation of the delta wave vector, ventricular preexcitation may masking or mimicking myocardial infarction [5]. An inferior myocardial necrosis can be simulated by a posterior preexcitation translated by negative delta wave in leads DII, DIII and aVF, likewise a left lateral preexcitation with negative delta wave in DI , aVL and V6, can simulate lateral infarction. Conversely, ventricular preexcitation type A characterized by an exclusive R wave in leads V1-V2, masks anteroseptal myocardial necrosis. In Our case, preexcitation masked anterior infarction and simultaneously simulated inferior infarction. All reported studies emphasize the importance of the availability of ECG from the same patient without preexcitation for comparison when one is considering an MI in possible preexcited ECG.

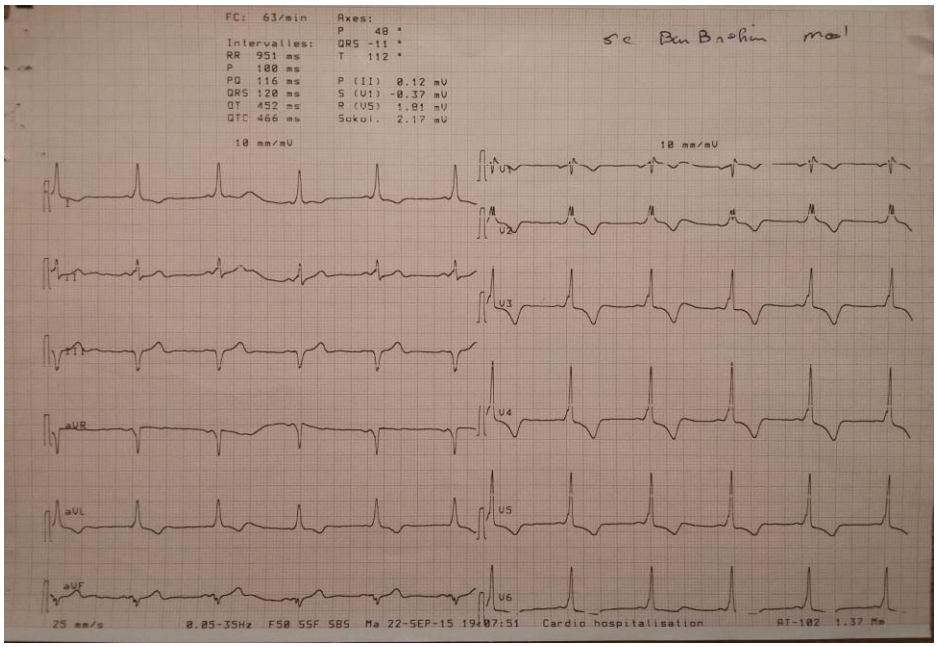

Fig-1: 12-Lead ECG 9 hours from onset of symptoms displaying ventricular preexcitation masking anterior myocardial infarction (MI) and mimicking inferior MI

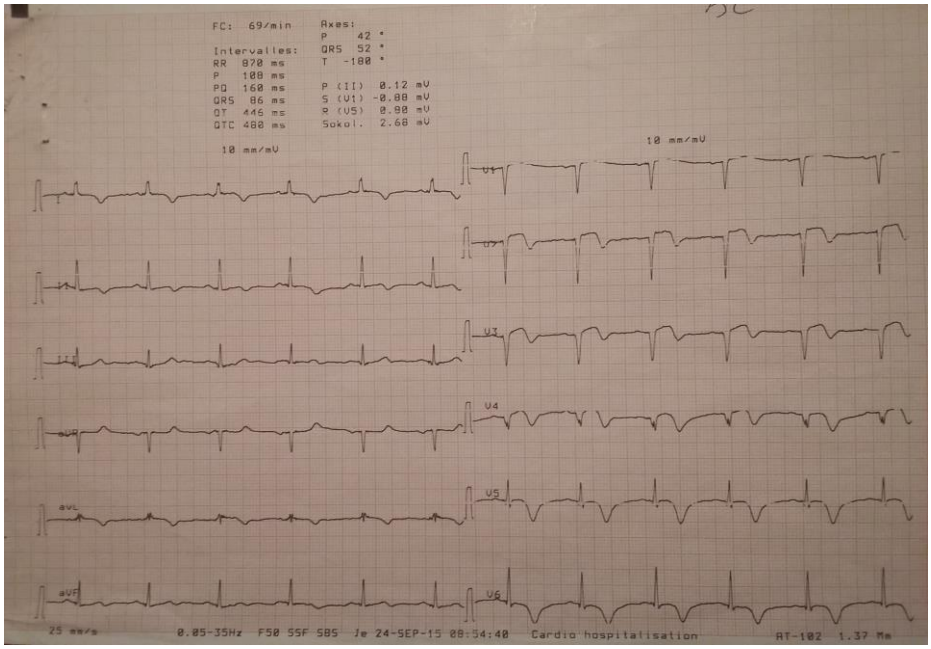

Fig-2: 12-Lead ECG 24 hours from onset of symptoms without ventricular preexcitataion displaying Q waves in anterior

Noninvasive techniques, to block preexcitation intermittently, may be useful for diagnostic accuracy of both acute and chronic conditions. One of those is Holter monitoring, where preexcitation may be intermittent because of changes in sinus rate. Exercise testing may be used to block the accessory pathway because at a given heart rate, the refractory period of the accessory pathway is attained, resulting in sudden disappearance of the preexcitation pattern [6]. In addition, increasing contribution through the normal AV-nodal–His-Purkinje axis due to increased sympathetic tone results in gradual diminishment of preexcitation, leading to gradual disappearance of the pseudo-infarction pattern. Similarly, sinus carotid massage may lead to different contribution over both conduction axes, leading to changes in pseudo-infarction patterns [7]. Also, the observation of a narrow QRS tachycardia (without an infarction pattern) will unmask preexcitation as the underlying mechanism. Class 1A drugs, such as procainamide and ajmaline, can block the accessory pathway without interrupting AV nodal conduction [8, 9].

CONCLUSION

Acute MI and preexcitation may occur simultaneously, which can lead to both masking and mimicking of either entity, whereas preexcitation alone is able to mimic a chronic MI. To avoid misdiagnosis, especially in emergency situations, clinicians should be aware of this ECG pitfall and understand how to distinguish between the two conditions.

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