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When a Pseudo-Infarct Electrocardiogram (ECG) Pattern in a Posterior Accessory (Wolff-Parkinson-White) Pathway Masks a True Inferior Infarct

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Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Conflict of interest: None declared

Patient: Male, 61
Final Diagnosis: Wolff-Parkinson-White (WPW)
Symptoms: Palpitation
Medication: —
Clinical Procedure: Ablation of accessory pathway of supra-ventricular tachycardia
Specialty: Cardiology

Objective: Rare co-existance of disease or pathology

Background: Wolff-Parkinson-White (WPW) pattern is due to a pre-excitation leading to characteristic ECG changes in sinus rhythm as short PR interval, the presence of delta waves, wide QRS complexes, and potentially Q wave-T wave vector discordance (pseudo-infarct pattern). These later changes can mask the underlying ECG depolarizing solely through the His-Purkinje system. Our case highlights how the ECG of a WPW pattern with a pseudo-infarct pattern can in fact mask a true infarct on the underlying ECG without pre-excitation.

Case Report: A 61-year-old diabetic man with a recent history of supra-ventricular tachycardia (SVT) presented with the ECG characteristic of a Wolff-Parkinson-White pattern i-e short PR interval of 0.10 s (<0.12 s) and the presence of delta waves in sinus rhythm. In addition, there was a wide significant Q wave in the inferior leads meeting the criteria for significant and pathologic Q waves, related to the pre-excitation and known as a pseudo-infarct pattern. The patient underwent successful ablation of his left inferoseptal accessory pathway. The pre-excitation pattern (short PR and delta wave) disappeared after successful ablation revealed a narrower Q wave in inferior leads, likely from unexpected true old inferior infarction, which was later confirmed by 2D echocardiogram and nuclear stress test (fixed inferior defect).

Conclusions: The presence of pseudo-infarct pattern due to a WPW does not always preclude the presence of underlying true infarct pattern, especially in the presence of coronary artery disease risk factors.

MeSH Keywords: Catheter Ablation • Myocardial Infarction • Wolff-Parkinson-White Syndrome

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/909189

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Background

The diagnosis of Wolf-Parkinson-White syndrome can present as a pseudo-myocardial infarct pattern on the surface ECG [1]. In addition to the typical finding of short PR interval (<0.12 s) and broad QRS complex (>0.10 s), the ECG can indeed present with negative Q waves in inferior leads, leading to the pseudo-infarct pattern for inferior preexcited accessory pathway [2,3]. In the presentation of an obvious acute or chronic coronary syndrome, the diagnosis of underlying coronary artery disease or prior myocardial infarction might be difficult, especially in diabetic patients. However, when the pre-excitation on the ECG disappears either spontaneously or after an ablation, the underlying ECG might reveal a true infarct pattern.

Case Report

A 61-year-old diabetic man with a history of recurrent palpitations was found to have supra-ventricular tachycardias (SVT) on the ECG, with the typical characteristic of a Wolff-Parkinson-White pattern (short PR interval of 0.10 s [<0.12 s] and the presence of a delta wave) (Figure 1). In addition, there was a wide significant Q wave in the inferior leads, meeting the criteria for a significant and pathologic Q wave (≥0.04 s wide and ≥0.1 mV deep), often seen with prior inferior myocardial infarction (MI) [4]. Since the patient did not have any history of coronary artery disease or any active chest pain, the presence of a significant Q wave associated with a WPW was considered to be part of the "pseudo-infarct pattern" related to activation of part or all the ventricles through the posterior insertion of the accessory pathway. Based on the surface ECG, the pathway appeared to be inserted posteriorly (Q wave in the inferior lead) and left-sided with an R/S ratio >0.5 to 1 [5,6]. Since the patient was experiencing a significant amount of SVT refractory to medications (recorded on a Holter monitor), the patient underwent an electrophysiology study and ablation of his accessory pathway. After successful radiofrequency ablation of a left inferoseptal accessory pathway, the pre-excitation pattern (short PR and delta wave) disappeared. The native conduction through the physiologic His-Purkinje system showed a PR interval longer than 0.2 s (first-degree atrioventricular block). However, instead of the positive and narrower QRS complexes in inferior leads typically expected after elimination of such accessory pathways, the ECG showed a narrower Q wave in inferior leads (Figure 2). As persistent Q waves were highly suggestive of an old infarction, a 2D echocardiogram was done, which confirmed wall motion abnormality and an old inferior wall myocardial infarction (wall thin and akinetic) that was masked on the surface ECG by the ventricular pre-excitation. The patient underwent a nuclear stress test that showed a limited fixed defect in the inferior left ventricle without signs of ischemia in the rest of the myocardium and an ejection fraction of 50%. The patient was then started on the guideline-directed medical therapy (aspirin, metoprolol, statin, and angiotensin-converting enzyme inhibitor).

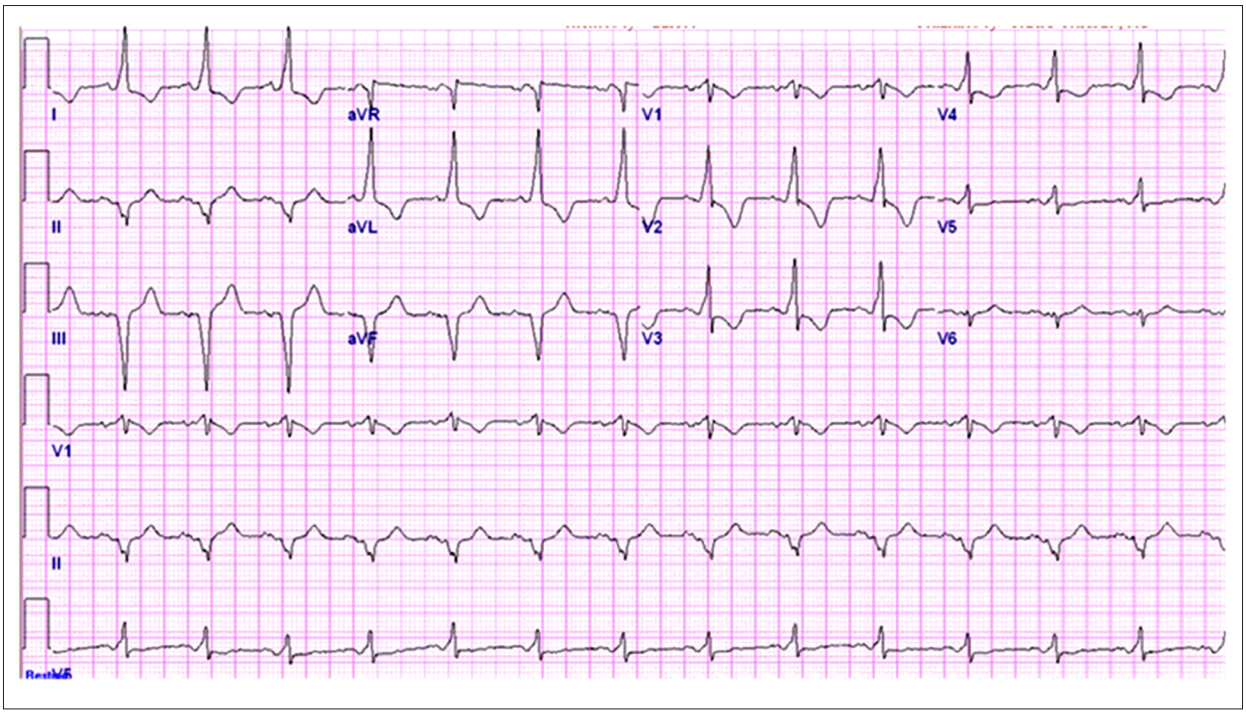

Figure 1. Presence of delta waves, wide QRS complexes, prominent T waves and pathologic Q waves with Q wave-T wave vector discordance in inferior leads II and II aVF (pseudo-infarct pattern).

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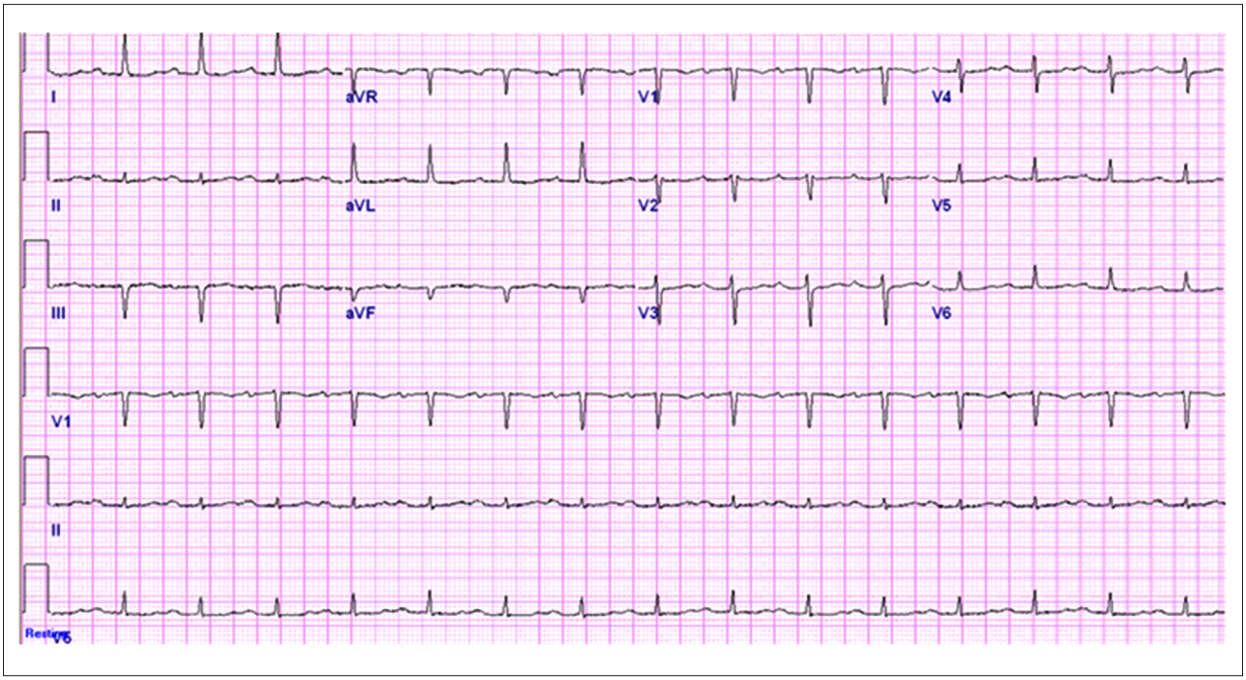

Figure 2. After ablation of the left inferoseptal accessory pathway with prolongation of PR interval, the disappearance of the delta waves but unmasking of the narrow Q waves (from old infarction) in inferior leads II, II aVF. There is also noticeable relative flattening of T waves.

Discussion

Wolff-Parkinson-White is a pre-excitation syndrome with a symptomatic accessory pathway leading to characteristic ECG changes in sinus rhythm of short PR interval, the presence of delta waves, wide QRS complexes, and potentially Q wave-T wave vector discordance. This constellation of ECG findings is known as the pseudo-infarct pattern [7]. The WPW pattern is the pre-excitation findings in sinus rhythm on the ECG in the absence of symptomatic arrhythmias, whereas the WPW syndrome is the pre-excitation findings in the setting of symptomatic arrhythmias. Although the WPW pattern is more prevalent than the syndrome, both are relatively rare in the general population (the prevalence of WPW syndrome in the general population is 0.1 to 3.1 per 1000 persons) [8]. The AV bypass tract conducts faster than the normally-located AV nodal pathways, resulting in early excitation of the ventricle connected to the accessory pathways. Myocardial infarction and myocardial ischemia can affect the electrophysiologic characteristics of normal AV conduction system, and can lead to changes in QRS morphology, AV node prolongation, new Q waves, and changes in the ST segments or T wave.

The picture becomes further convoluted as WPW itself can also alter both depolarization and repolarization and can mimic acute infarctions. It can also mask true infarction, as in our case. In our patient, because of the left inferoseptal location of the accessory pathway, the ventricles were activated posteriorly and inferiorly first, then down to up, in a reverse sequence compared to physiologic activation and therefore generated negative forces on the ECG in the form of a Q wave in inferior leads. The pseudo-infarct Q waves in the inferior leads were associated with a short PR and positive T waves (known as Q wave-T wave discordance). This characteristic Q wave-T wave vector discordance results from secondary repolarization changes due to altered ventricular activation [7]. However, the presence of T wave inversion with inferior Q waves and a normal PR interval (referred to as Q wave-T wave concordance) was strongly suggestive of inferior ischemia, and further evaluation (echocardiogram) for infarction was warranted. A case report by Smolders et al. mentioned the intravenous use of procainamide to differentiate between the myocardial infarction and WPW in a similar situation [9]. Procainamide leads to initial 2: 1 blockage and later complete block of conduction over accessory pathway, and pseudo-infarction pattern of posterior inferior MI type disappeared. In the case presented here, there was an important shift from a very positive T wave with pre-excitation to a T wave slightly positive to almost isoelectric T wave without pre-excitation and a narrower Q wave. The T wave might stay slightly positive instead of negative, as seen with ischemia, because of the T wave memory phenomenon in which the T wave follows ("remembers") a previously altered QRS vector due to WPW [10]. Nonetheless, the presence of significant Q waves in inferior leads was highly suspicious in the absence of pre-excitation.

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Conclusions

The presence of pseudo-infarct pattern does not always preclude the presence of an underlying true infarct pattern. The presence of an underlying myocardial infarction should not be overlooked in the presence of a pseudo-infarct pattern due to an accessory pathway, especially in the presence of cardiovascular risk factors for coronary artery disease, as the presence of an underlying true prior infarct can strongly modify the medical management of such patients.

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