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CASE REPORT

# Electrocardiographic abnormalities in a patient with pre-excitation and acute myocardial infarction undergoing percutaneous coronary intervention and ablation

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**Abstract:** Pre-excitation syndrome can either mimic or mask myocardial infarction, making the diagnosis of acute myocardial infarction difficult. Herein, we report the case of a male patient with Wolff-Parkinson-White (WPW) syndrome who presented to our emergency department with severe chest pain. Non-ST-elevation myocardial infarction was suspected because of cardiac enzyme elevation and abnormal ST-T changes identified through electrocardiography. The patient underwent percutaneous coronary intervention; a left anterior descending artery stenotic lesion was dilated, and drug-eluting stents were implanted. One month later, he underwent successful radiofrequency catheter ablation for his accessory pathway and tachycardia. We present the series of electrocardiographic ST-T abnormalities to raise awareness of the value of diagnosing myocardial injury early in patients with WPW syndrome.

**Keywords:** Electrocardiography; Myocardial infarction; Pre-excitation

## 1. INTRODUCTION

The electrocardiographic diagnosis of acute myocardial infarction (AMI) in patients with Wolff-Parkinson-White (WPW) syndrome is often difficult[1-3] because the abnormal activation sequence revealed through electrocardiography (ECG) in WPW syndrome may mask the characteristic ECG findings of MI. The presence of Q waves is often confusing for patients with pre-excitation and possible infarction. Moreover, the apparent T-wave abnormalities of patients with pre-excitation may mimic the clinical ECG changes of acute coronary syndrome (ACS).[4,5] Herein, we report the case of a male patient with WPW syndrome who underwent percutaneous coronary intervention for non-ST-elevation MI (NSTEMI) and radiofrequency catheter ablation (RFCA) for his accessory pathway during 2 hospitalizations. The series of ECG abnormalities are presented to raise awareness of the value of diagnosing myocardial injury early in patients with WPW syndrome.

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## 2. CASE REPORT

A 60-year-old man presented to our emergency department with chest pain, which had developed during exercise. His past medical history was unremarkable except for a 20-year history of smoking. His initial ECG revealed normal sinus rhythm, positive delta waves in leads I, II, and aVL and precordial leads V2-V6, and negative delta waves in leads III and aVF. The ST segment was depressed in leads I and aVL (Fig. 1A). A physical examination was unremarkable, and the laboratory data revealed levels of troponin I, creatine kinase (CK), and CK-myocardial band (MB) within the normal limits. Most of his chest discomfort resolved after the sublingual administration of a nitroglycerin tablet; however, mild chest pressure persisted. A follow-up at 4 hours revealed normal cardiac enzymes, but a 12.7-U/L elevation in CK-MB and 1.19-ng/mL elevation in troponin I were discovered at 8 hours. Subsequent ECG revealed ST-segment normalization in leads I and aVL (initial finding: ST-segment depression) and T-wave inversion in leads V4 and V5 (initial finding: positive T waves; Fig. 1B). NSTEMI was suspected; therefore, cardiac catheterization was recommended and performed the next day, revealing a significant stenotic lesion in the proximal left anterior descending artery. Percutaneous coronary intervention was performed with drug-eluting stents deployed in the proximal-to-middle left anterior descending artery. ECG 2 days after the coronary intervention revealed progressive symmetrical T-wave inversion in leads V3-V6 (Fig. 1C). One month later, ECG was performed, revealing persistent pre-excitation and the resolution of the repolarization abnormalities in leads I, aVL, and V3-V6 (all positive T waves; Fig. 2A). The patient then underwent an invasive electrophysiological study, and atrioventricular orthodromic reciprocating tachycardia was induced in a laboratory. Successful RFCA of the accessory pathway was

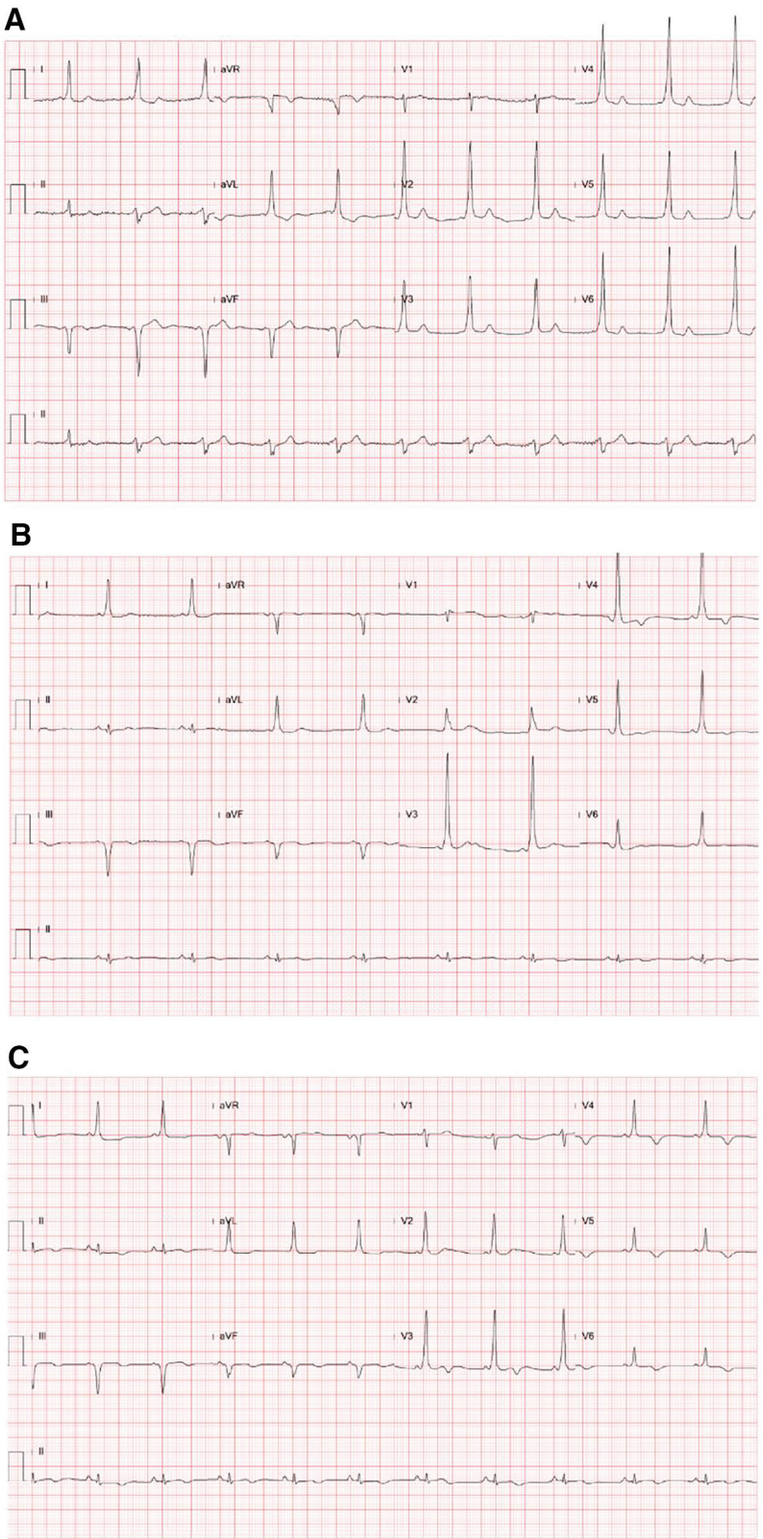


**Fig. 1** A, ECG in the emergency department demonstrating sinus rhythm and pre-excitation; ST segment is depressed in leads I and aVL. B, Twelve hours later, ECG reveals symmetrical T-wave inversion in leads V4 and V5. C, ECG 2 days after coronary intervention revealing progressive symmetrical T-wave inversion in leads V3–V6. ECG = electrocardiography.

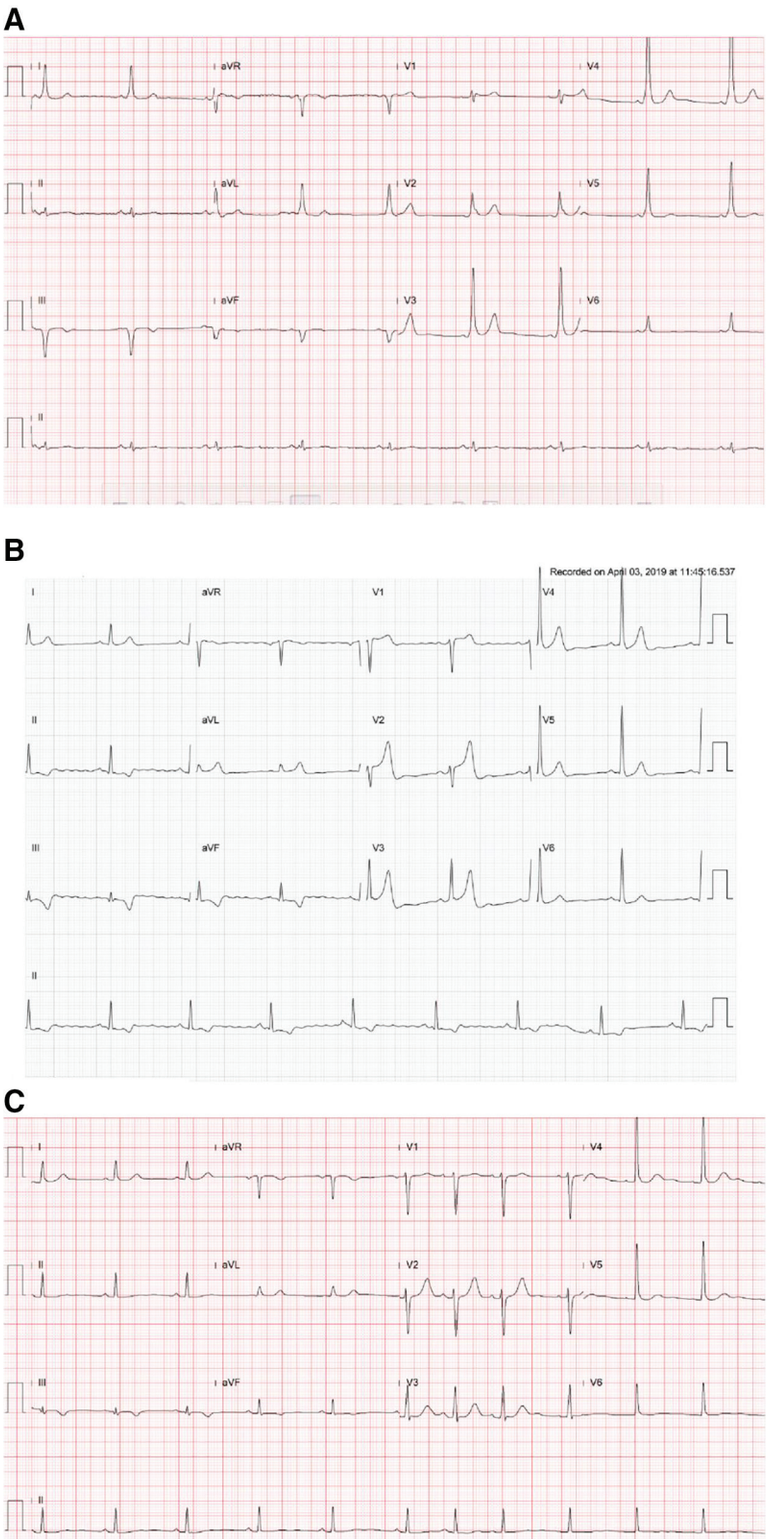


**Fig. 2** A, ECG 1 month after myocardial infarction revealing persistent pre-excitation without significant ST-T abnormalities. B, ECG following successful ablation in the laboratory, revealing sinus rhythm without pre-excitation. ST elevation and tall T waves in the precordial leads and ST depression in the inferior leads suggestive of cardiac memory. C, ECG 1 year later revealing normal sinus rhythm without pre-excitation or notable ST-T abnormalities. ECG = electrocardiography.

performed on the right posterior septal aspect of the tricuspid ring. ECG after successful ablation revealed positive memory T waves in the precordial leads and negative T waves in the inferior leads (Fig. 2B). The patient was followed up regularly for 1 year in our clinic without any discomfort. ECG after 1 year demonstrated that the memory T waves had disappeared (Fig. 2C).

## 3. DISCUSSION

The diagnosis of AMI in patients with WPW syndrome in outpatient clinics or emergency departments is challenging because the typical ECG findings of AMI can be masked by repolarization changes caused by pre-excitation.[1-6] Moreover, the abnormal Q wave (delta wave) in the ECG results of patients with WPW may easily lead to misdiagnosis of MI in clinical practice.[2] The key to the diagnosis of AMI in WPW syndrome is the serial evolution of ECG changes.[1,6] However, serial ECG is time-consuming and delays the diagnosis of STEMI and thus primary coronary intervention for myocardial rescue. In addition, ST elevation may be difficult to identify in patients with WPW syndrome because of existing secondary ST-T changes. In our case, AMI was diagnosed on the basis of typical chest pain, serial ECG ST-T changes, and cardiac enzyme elevation. Angiographic findings confirmed the presence of significant stenotic lesion related to AMI. Notably, this case provides a unique opportunity to study the continual ECG changes in AMI after RFCA. Postablation ECG revealed ST elevation and tall T waves in the precordial leads and considerable ST depression in the inferior leads in the form of memory T waves.[7]

For the early diagnosis of ACS or AMI in patients with WPW syndrome, the specific electrocardiographic ST-T abnormalities are more valuable than the elevation of cardiac enzymes. Secondary ST-T changes are common features in patients with WPW without ischemic episodes. Such secondary ST-T changes include ST-T abnormalities deflected in the opposite direction of the delta wave vector, ST-T changes that are positively correlated with the degree of delta waves, nonhorizontal ST-T changes, and nonsymmetrical T-wave inversion. When secondary ST-T changes in patients with WPW occur along with acute chest pain, the early diagnosis of ACS or AMI is difficult. In our case, we observed ST depression in leads I and aVL in the initial ECG (Fig. 1A) on presentation to our emergency department. We were unable to ascertain whether the ST-T abnormality was caused by acute ischemic change or secondary ST-T change because of the pre-excitation. Notably, less severe ST-T abnormality in leads I and aVL was observed in subsequent ECG screenings (Fig. 1B,C). This might be because the ST depression was caused by acute ischemic injury.

Because secondary ST-T changes in patients with WPW syndrome might mask ischemic or injury-induced ST-T changes, understanding the primary specific ST-T abnormalities of WPW syndrome is essential. Such ECG findings are as follows: ST-T changes concordant or discordant with the delta wave vector, horizontal ST depression, symmetrical T-wave inversion, and ST-T changes in 2 or more contiguous leads in the presence of clinical ischemic symptoms. We identified typical symmetrical T-wave inversion in our patient's precordial ECG leads (Fig. 1B,C), which strongly suggested acute ischemic injury. Coronary intervention was recommended, especially because of the cardiac enzyme elevation. The ST-T abnormalities were resolved withing 1 month through management of the disease (Fig. 2A).

The patient underwent an electrophysiological study, which confirmed the presence of an accessory pathway, and the right posterior septal aspect of the tricuspid valve was successfully ablated. Postablation ECG revealed substantial ST elevation in the precordial leads and ST depression in the inferior leads, indicating cardiac memory delta waves (Fig. 2B). Abnormal T-wave memory typically resolves within several months (Fig. 2C). If the patient has recurrent angina, defining the specific ischemic ST-T changes as the absence of delta waves in ECG will be straightforward.

In conclusion, AMI and pre-excitation can occur simultaneously, and dynamic repolarization abnormalities that differ from secondary ST-T changes may suggest acute ischemia in patients with WPW syndrome. Early and prompt coronary intervention may rescue the myocardium and save the patient's life. Clinicians should be familiar with the ECG abnormalities specific to this condition to avoid the overdiagnosis of AMI in patients with WPW syndrome.

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