



Acute myocardial infarction in a patient with Wolff-Parkinson-White syndrome

Xu-Gang TANG^{1,*}, Jing WEN^{2,*}, Xue-Sen ZHANG¹, Xiang-Jun LI³, Da-Chun JIANG^{1,#}

¹Department of Cardiology, No. 37 Hospital of PLA, Ya'an, Sichuan Province, China

²Department of Pediatrics, People's Hospital of Hechuan District, Chongqing, China

³Department of Cardiology, No. 22 Hospital of PLA, Ge'ermu, Qinghai Province, China

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It is known that the Wolff-Parkinson-White syndrome (WPW) may either mimic myocardial infarction (MI) or mask the ECG changes of MI. Thus, the diagnosis of MI coexisting with WPW is frequently difficult. Furthermore, patients with WPW occurring acute MI may be life threatening. Therefore, early recognition and correct treatment allows rapid restoration of normal sinus rhythm and may decrease morbidity and mortality.

A 45-year-old male farmer was admitted to our hospital for five days of intermittent chest pain (pressure-like pain, less than ten minutes of time) with palpitations, associated closely with body work. He had no lung or gastrointestinal or neurological symptoms. He had undergone an intermittent and self-resolving palpitation from the past three years, with at least one or two episodes a week, diagnosed as WPW at other hospital. He took no medications, and had no family history. He was a nonsmoker and nondrinker, with no history documenting the presence of hypercholesterolemia, hypertension, and diabetes.

Physical examination was done. Blood pressure was 102/68 mm Hg, pulse was 211 beats/min, and arterial oxygen saturation was 98%. A 12-lead ECG showed a supraventricular tachycardia (Figure 1). Tachycardia was terminated by transesophageal atrial pacing (Figure 1S). However, tachycardia occurred 10 min later (Figure 2S). Intravenously amiodarone (0.15 g) was administered over a 10-min period. The heart rate decreased to between 150 and 180 beats/min. Seven minutes later, the farmer suddenly lost consciousness, involuntary twitching occurred in the hands and feet. Ventricular fibrillation was diagnosed on ECG monitor. The farmer was defibrillated successfully by a

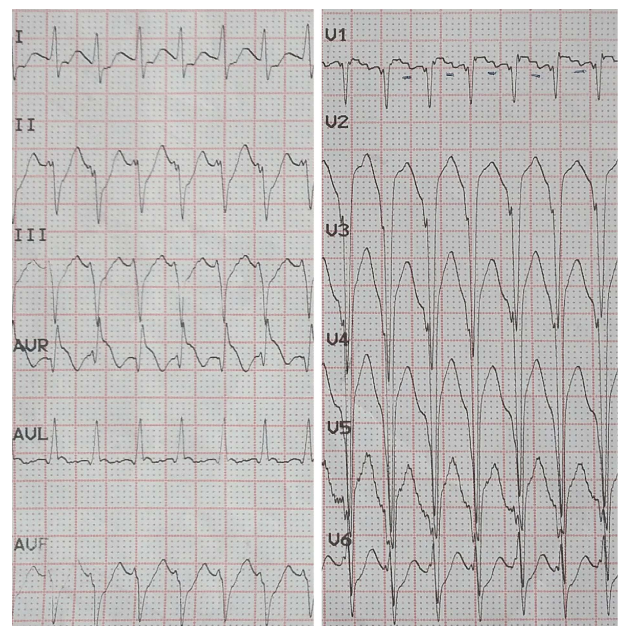


Figure 1. Onset of supraventricular tachycardia. ECG was recorded at a paper speed of 25 mm/s and a gain of 10 mm/mV.

direct current precordial shock of 200 J (Figure 3S) and reverted into sinus rhythm.

Troponin I taken after admission was significantly elevated at 5.78 ng/mL (reference threshold < 0.15 ng/mL). Cardiac enzymatic determinations showed increased serum lactic dehydrogenase (750 U/L, reference threshold < 245 U/L), creatine phosphokinase MB (37 U/L, reference threshold < 25 U/L), and glutamic oxalacetic transaminase (75 U/L, reference threshold < 37 U/L). The ECG showed a WPW pattern and the S-T segment elevation in leads V₂ through V₄ and the S-T segment depression in leads II, III, aVF, and V₆ (Figure 1S). An echocardiogram revealed an enlargement of the left atrium (41 mm) and left ventricle

*The two authors contributed equally to this work.

#Correspondence to: jdc_37@163.com

(LVDd, 59 mm). Together, these data suggested that we were dealing with an MI of the anterior wall of the left ventricle.

Coronary angiography confirmed single vessel disease with 90% stenosis of the left anterior descending coronary artery, successfully treated with percutaneous coronary intervention (PCI) using a drug-eluting stent (Figure 2). The electrophysiology study also was performed after PCI. A left lateral anterior wall accessory pathway was documented, which was successfully ablated (15 Watt, 120 s) (Figure 3). Notably, atrial fibrillation was induced during electrophysi-

ologic testing after the accessory pathway ablation (Figure 4S), and self resolved after the farmer sent back to the coronary care unit (Figure 5S).

He was discharged after nine days hospitalization, with free chest pain or palpitations. ECG taken after discharge three months showed a normal P-R interval and Q waves in leads V₁ through V₄. The left atrium was 38 mm, and LVDd was 54 mm measured using ultrasonic cardiography.

Acute MI and WPW may present simultaneously, which is an emergent and potentially lethal event. Correct and

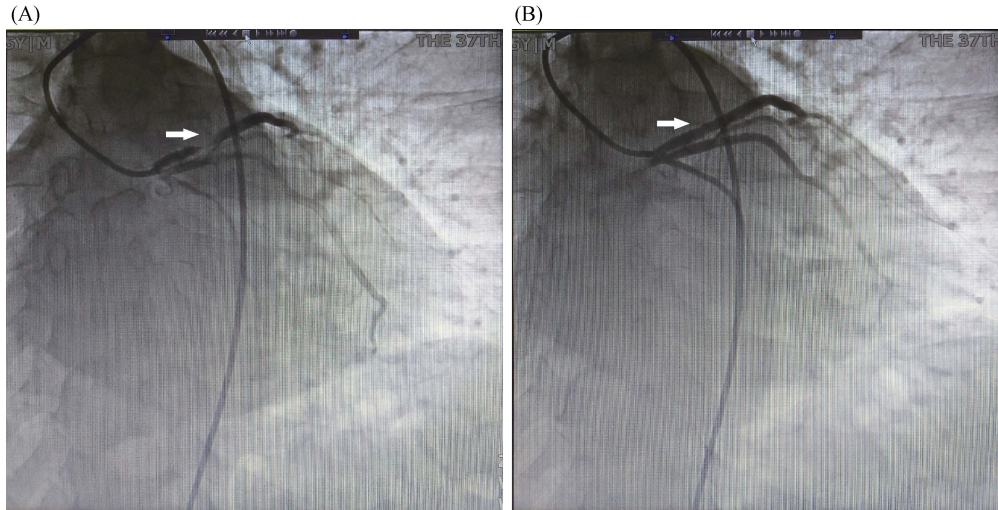


Figure 2. Coronary angiography before (A) and after (B) percutaneous coronary intervention. Arrows indicating stenosis site.

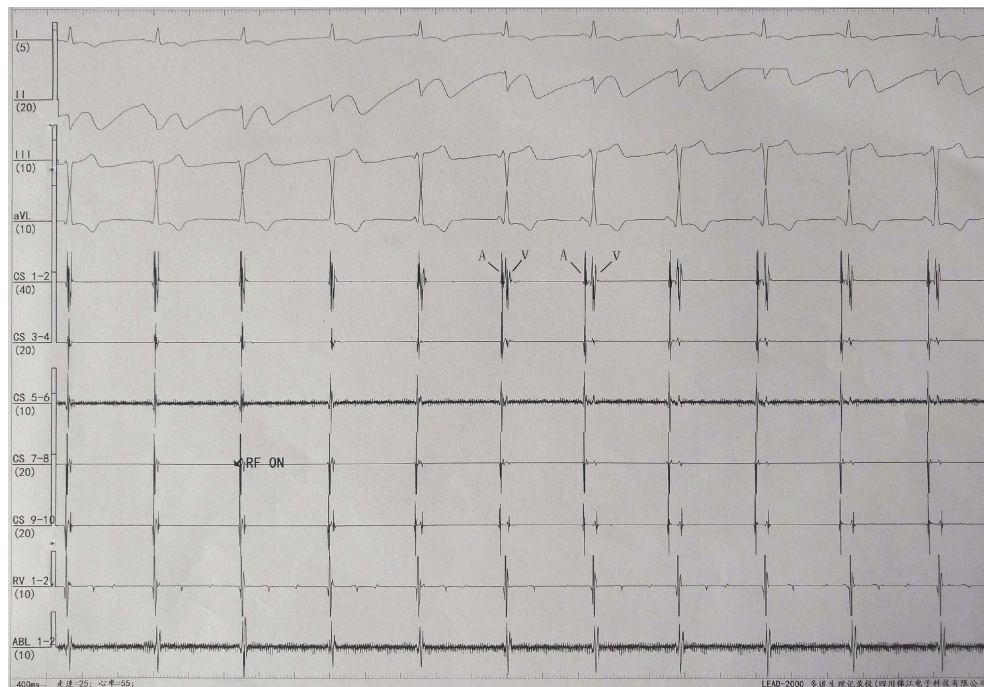


Figure 3. Loss of pre-excitation during radiofrequency energy application.

timely diagnosis is critical for the effective control of this event. Here, we report a case of WPW with acute MI with multiple tachycardias occurring. Our study may be of help to doctors for clinical works.

Interestingly, the farmer was free of traditional cardiac risk factors, such as smoking habits, fat, hypercholesterolemia, hypertension, and diabetes, except for exposure to long working hours. The farmer was frequently working 9-11 hours a day, which may be the most possible risk factor for his acute MI according to the literatures.^[1,2] Because of the existence of WPW, we initially couldn't make a affirmative diagnosis of acute MI from the surface ECG (Figure 1 & 1S), although the greater ST-segment elevation in lead V1-V4 seemed unusual in the common form of type-A WPW syndrome. It is known that ECG may result in confusion when WPW and MI present simultaneously.^[3,4] Thus, a misdiagnosis may be made on some conditions. Taking note of the newly occurring chest pain, we further tested the Troponin I and cardiac enzyme levels to exclude the potential cardiovascular diseases. With the significantly elevated Troponin I and cardiac enzyme levels, the farmer was highly suspected of suffering from an acute MI. The stenosis of the left anterior descending coronary artery was confirmed by coronary angiography (Figure 2). Given WPW and MI may have co-existed, we must take good care of WPW patients with new-onset chest pain, such as in our case, collapse,^[5] syncope,^[6] shock,^[7] do not focus only on the surface ECG.

Patient of WPW with acute MI may occur multiple tachycardias.^[8] In our case, the farmer suffered from supraventricular tachycardia (Figure 1), WPW with atrial fibrillation (Figure 2S), and ventricular fibrillation (Figure 3S) in a short time. Fortunately, he was defibrillated successfully by a direct current precordial shock. It is well known that both WPW and MI are closely associated with atrial fibrillation, and atrial fibrillation with a fast ventricular response may degenerate to catastrophic ventricular fibrillation, which is the most frequent cause (reason) of sudden cardiac death in WPW patients.^[9,10] For stable patient of WPW with atrial fibrillation, intravenous procainamide is recommended,^[11] however, this drug in our hospital is unavailable at that time. Amiodarone was initially used as an antianginal drug, which could increase coronary blood flow, and decrease myocardial oxygen consumption. Nowadays, amiodarone is widely used to control multiple arrhythmias in patients.^[12,13] Guidelines gave the superiority of amiodarone in the use of antiarrhythmia in the stable WPW with atrial fibrillation patients.^[14] However, some researchers argued against the superiority of administering amiodarone in WPW with atrial fibrillation patients because of the pro-

dysrhythmic events in some cases.^[15,16] Indeed, study didn't show the same prodysrhythmic events in some other cases.^[17] Therefore, the newly developed guideline indicates that intravenous amiodarone should be used with caution in WPW with atrial fibrillation patients.^[18] In our case, intravenous amiodarone was suitable for control of rapid ventricular response in atrial fibrillation in the farmer with true MI according to the literatures.^[12,18,19] It is well known that malignant dysrhythmia may happen to a patient with atrial fibrillation with fast ventricular response, acute MI, etc. Amiodarone should not be accounted for malignant arrhythmic events in the patient with WPW and atrial fibrillation due to the lack of well-designed studies now.

In conclusion, acute MI and WPW may present simultaneously. Urgent direct current cardioversion should be considered in unstable patients. Intravenous amiodarone should be used with caution in WPW with atrial fibrillation patients. Further study is needed for assessment of intravenous amiodarone for heart rate control in WPW with atrial fibrillation patients.

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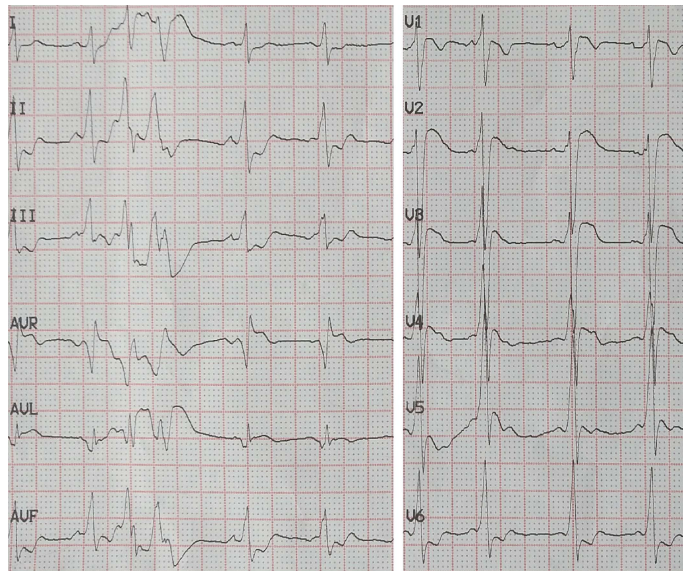


Figure 1S. ECG obtained after tachycardia terminated by transesophageal atrial pacing showing WPW (type A), ECG was recorded at a paper speed of 25 mm/s and a gain of 10 mm/mV.



Figure 2S. WPW and concomitant atrial fibrillation. ECG was recorded at a paper speed of 25 mm/s and a gain of 10 mm/mV.

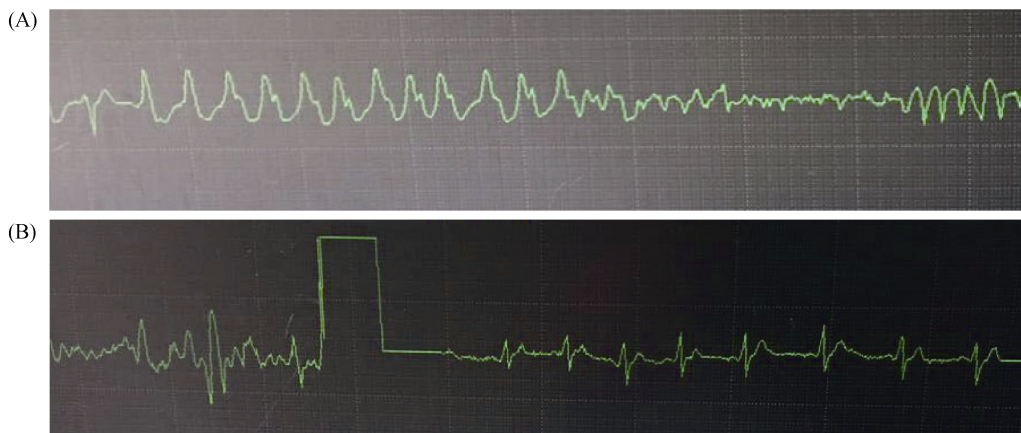


Figure 3S. ECG on the monitor before (A) and after (B) a direct current precordial shock.

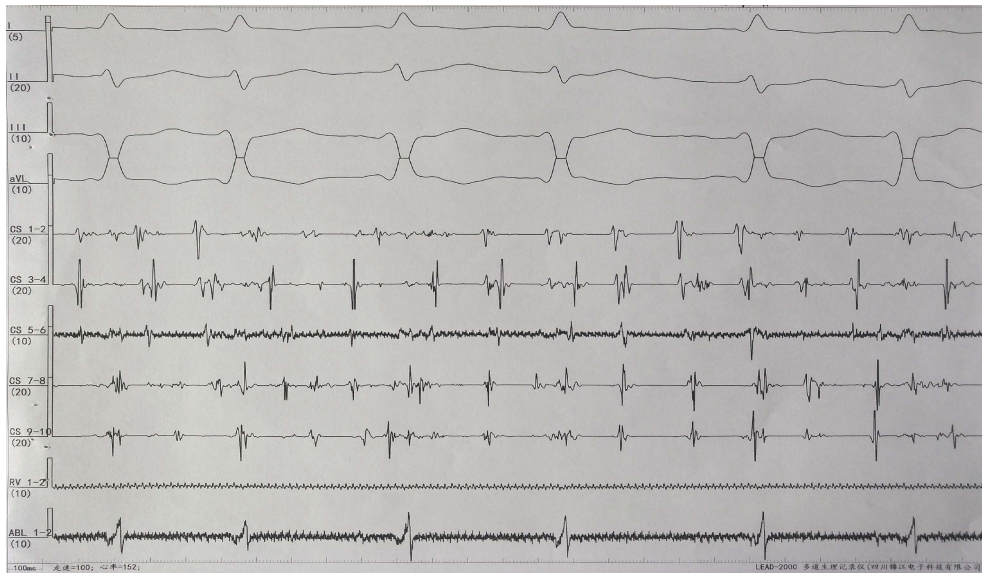


Figure 4S. Atrial fibrillation was induced during electrophysiologic testing.

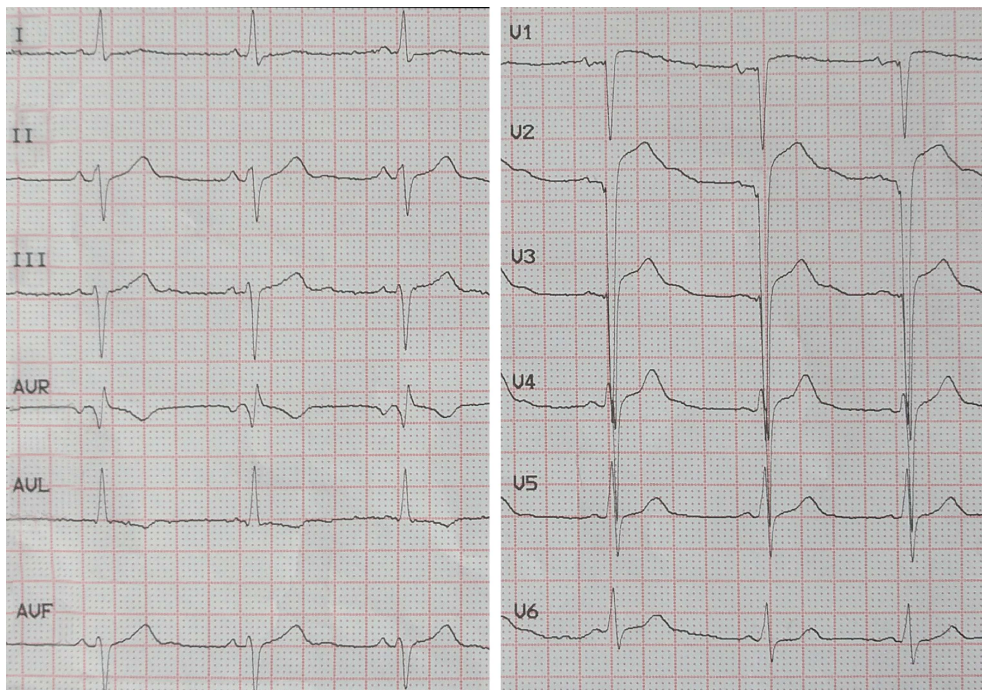


Figure 5S. ECG after PCI and radiofrequency catheter ablation. ECG was recorded at a paper speed of 25 mm/s and a gain of 10 mm/mV.