

CASE REPORT

Medicine Science 2017;6(4):780-2

Atrial fibrillation with Wolff–Parkinson–White syndrome: A case report

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Received 01 July 2017; Accepted 17 August 2017

Available online 16.11.2017 with doi: 10.5455/medscience.2017.06.8676

Abstract

Wolff-Parkinson-White (WPW) syndrome is a conduction disturbance characterized by the presence of an accessory pathway between the atria and the ventricles. The accessory pathway predisposes patients to tachyarrhythmias and sudden cardiac death. Among patients with WPW syndrome, atrioventricular reentrant tachycardia (AVRT) is the most common arrhythmia, accounting for 95% of re-entrant tachycardias. It has been estimated that one-third of patients with WPW syndrome have atrial fibrillation (AF). AF is a potentially life-threatening arrhythmia and can degenerate to ventricular fibrillation. In this paper, we aimed to present an AF with WPW case.

Keywords: Atrial fibrillation, tachycardia, Wolff-Parkinson-White syndrome

Introduction

The atrias are almost completely isolated from the ventricles electrically, thanks to the separation by fibrous atrioventricular rings. Normally the only electrical connection between the atria and the ventricles is by AV node [1]. Patients with a pre-excitation syndrome have an additional or alternative pathway, known as an accessory pathway, which directly connects the atria and ventricle and bypasses the AV node [2]. Wolff-Parkinson-White syndrome (WPW) is the most common form of ventricular pre-excitation. The prevalence of WPW syndrome is between 0.1% and 0.3% in the general population. It appears on electrocardiogram (ECG) as a short PR interval, a wide QRS complex and a delta wave [3]. The most common arrhythmia associated with this syndrome is the atrioventricular reentrant tachycardia (AVRT). Atrial fibrillation (AF) is a potentially life-threatening arrhythmia in patients with WPW syndrome as it can degenerate to ventricular fibrillation (VF) [4]. We reported a case of wide QRS tachycardia which was subsequently diagnosed as WPW syndrome with AF.

Case report

A 40-years-old female patient admitted to emergency service due to palpitation and chest pain that started 3 hours ago. She had no medical history of note. On physical examination, we determined blood pressure 126/78 mmHg, pulse rate 180-200beats/minute, temperature 37°C and SPO₂ 98% by pulse-oximeter in room air. But the patient was stable hemodynamically. Laboratory tests

(complete blood count, biochemistry and troponin I) were normal. The initial 12-lead ECG revealed irregularly irregular rhythm, narrow and broad complexes at a rate of 186. (Figure-1). The patient were underwent synchronized cardioversion and converted to sinus rhythm successfully. Repeat ECG following cardioversion showed a shortened PR interval with a distinct slurring of QRS complex (Figure 2), which is diagnostic for the WPW syndrome. Transthoracic echocardiography revealed normal findings and left ventricular systolic function. Then, she was referred for electrophysiological studies. The left-sided accessory pathway was identified and successfully treated with radiofrequency ablation (Figure-3).

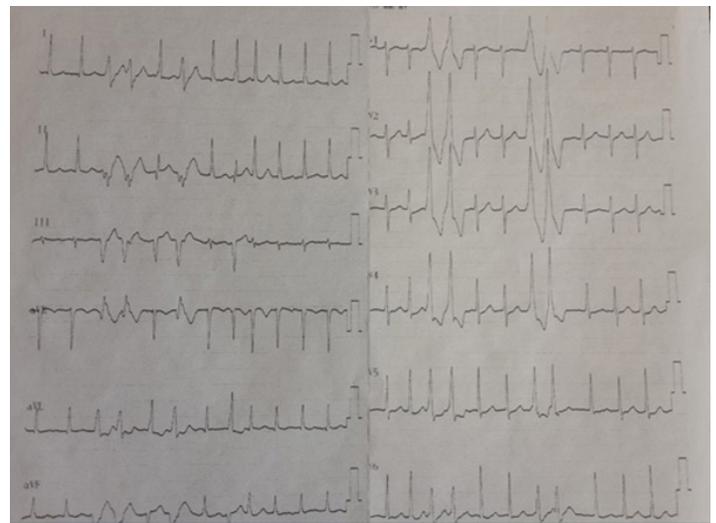


Figure 1. A 12-lead electrocardiogram showing a heart rate of 186 bpm, narrow and wide QRS complexes by the irregularity of the R-R interval

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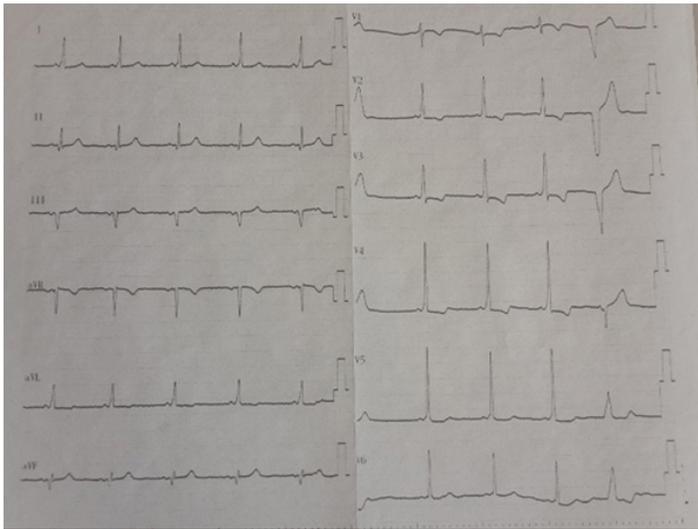


Figure 2. A postconversion electrocardiogram showing slurring of the wide QRS complex and shortened PR interval consistent with the diagnosis of Wolf-Parkinson-White syndrome

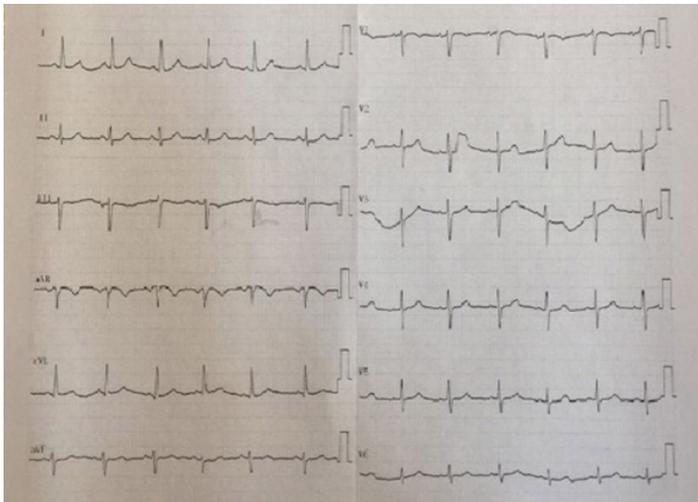


Figure 3. Twelve lead ECG of the patient after radiofrequency ablation

Discussion

In a normal heart, conduction from atrium to ventricles occurs through the AV-His-Purkinje system. But, patients with WPW syndrome have an accessory pathway that bypasses the AV node. The existence of an accessory pathway without the physiological delay of the cardiac impulse in the AV node causes arrhythmias [5]. The ECG changes are due to an accessory pathway, usually a bundle of Kent. The classic ECG pattern of WPW syndrome consists of a short PR interval, a slurred, thickened initial upstroke of the QRS complex, which is termed as delta wave, and a slight widening of the QRS deflexion with increased ventricular activation time [3].

The most important clinical significance of WPW syndrome is the frequent occurrence of supraventricular tachycardias, such as AVRT, AF and atrial flutter. Tachyarrhythmia can be facilitated by the formation of a reentry circuit involving the accessory pathway, termed AVRT. There are 2 forms of AVRT: orthodromic and antidromic. In orthodromic AVRT, the atrial impulse is conducted to the ventricles via the normal AV node route and is conducted retrogradely via an accessory pathway; this retrograde conduction

allows the impulse to reenter the conduction system and perpetuate the tachycardia. In AVRT with antidromic conduction, the accessory pathway conducts the impulse antegradely, resulting in preexcitation of the ventricles, which is followed by retrograde conduction through the AV node [6,7]. Tachyarrhythmia may also be facilitated by direct conduction from the atria to the ventricles via the accessory pathway, bypassing the AV node, seen with AF or atrial flutter in conjunction with WPW. Rapid anterograd accessory pathway conduction during AF can result in sudden cardiac death in patients with a manifest accessory pathway [8].

The incidence of paroxysmal AF has been reported to be between 10% and 38% patients with WPW syndrome [9] and can result in an extremely fast, very irregular wide and narrow complex tachycardia [10]. In particular, frequent tachycardias may also promote electrical remodeling and an increased atrial vulnerability to AF, which has been shown to more frequently induce sustained episodes of AF. In the present case, the ECG of patient was diagnosed as pre-excited AF because of the irregularity of rhythm, presence of delta wave, rapid ventricular response, narrow and wide QRS complexes.

Acute treatment of patients with WPW syndrome presenting with a tachydysrhythmia differs depending upon the patient's clinical presentation. Hemodynamically unstable patients, regardless of the QRS duration or regularity, should receive immediate synchronized cardioversion [11]. According to current ACC/AHA/HRS Guideline for the Management of Adult Patients With Supraventricular Tachycardia, hemodynamically stable patients who present with tachycardia caused by pre-excited AF must be monitored very carefully due to the increased risk of sudden death in intensive care unit. Acute treatment of pre-excited AF requires a rapid acting drug that can be given intravenously and can slow conduction in the accessory pathway. Small observational studies support the use of ibutilide or intravenous procainamide for the treatment of pre-excited AF in stable patients. Both medications can decrease ventricular rate by slowing conduction over the accessory pathway. Additionally, class III anti arrhythmics (amiodarone) and AV node blockers (β -blockers, calcium channel blockers and digoxin) should not be used for pre-excited AF. Because, inhibition of AV node conduction can increase pre-excitation and lead to ventricular fibrillation. Catheter ablation is the recommended treatment for the the long term therapy of pre-excited AF [8].

In conclusion, we should keep in mind pre-excited AF in irregular and wide complex tachycardias. Pre-excited AF is a life threatening arrhythmia. The main treatment of pre-excited AF in our country is electrical cardioversion whichever patients are stable or not, because of the absence of drugs. But the only long term therapy is catheter ablation.

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