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Cardiology

An Episode of Paroxysmal Atrial Fibrillation Revealing Wolff-Parkinson-White Syndrome, a Case Report

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Abstract **Case Report**

Wolff-Parkinson-White syndrome involves abnormal electrical conduction in the heart through accessory pathways. Pre-excited atrial fibrillation arises from the non-decremental conduction of atrial fibrillation to the ventricles via these pathways, resulting in supraventricular tachycardias and, albeit rarely, sudden cardiac death due to ventricular fibrillation. Our case report aims to elucidate the specific characteristics of this condition and explore different methods of managing it.

Keywords: Wolff-Parkinson-White syndrome, atrial fibrillation, accessory pathways, Sudden cardiac death, Case report.

INTRODUCTION

Wolff-Parkinson-White (WPW) syndrome is a relatively uncommon condition characterized by the presence of an atrioventricular accessory pathway. Its clinical significance stems from episodes of paroxysmal supraventricular tachycardia (SVT) facilitated by this accessory pathway. The most alarming complication associated with WPW sudden cardiac arrest, particularly when atrial fibrillation sets in, potentially leading to ventricular fibrillation.

When it comes to managing pre-excited atrial fibrillation in WPW syndrome, treatment options depend on the patient's hemodynamic stability. Specific antiarrhythmic medications or electrical cardioversion may be employed to restore a normal heart rhythm. However, the ultimate curative approach involves the use of radiofrequency ablation to eliminate the abnormal accessory pathway.

Here we present the case of a young patient which was admitted for a hemodynamically intolerated pre-excited atrial fibrillation, through which we expose the particularity of this pathology and the specificities of its treatment.

CASE REPORT

A 22 years old male without specific pathological history, consulted in the emergency department for palpitations of rapid onset occurring at rest following a night of excessive alcohol drinking. The admission examination found a conscious normotensive patient with an arterial pressure of 132/67 mmHg, tachycardic with a heart rate of around 160 beats per minute, eupneic at rest, without signs of shock or heart failure.

A few minutes later, the patient developed hemodynamic instability with systolic blood pressure around 73 mmHg. An urgent electrocardiogram was performed, which revealed an irregular tachycardia with wide QRS at 224 beats per minute for which the patient was sedated and received a synchronized DC cardioversion.

Post-critical EKG showed a sinus rhythm, with a heart rate of 74 beats per minute, a short PR interval, prolonged QRS with a delta wave, and secondary repolarization disorders. In this context we concluded that it was pre-excited atrial fibrillation.

The initial biology workup was without significant abnormalities. Transthoracic echocardiography showed a normal bi ventricular

systolic function, no significant valvular disease, and normal atrial dimensions.

The patient was referred to a specialized center where he underwent electrophysiological exploration which revealed a left posterior WPW with a refractory period at baseline less than 260 ms which was ablated by radiofrequency with definitive disappearance of the accessory pathway without anterograde or retrograde conduction.

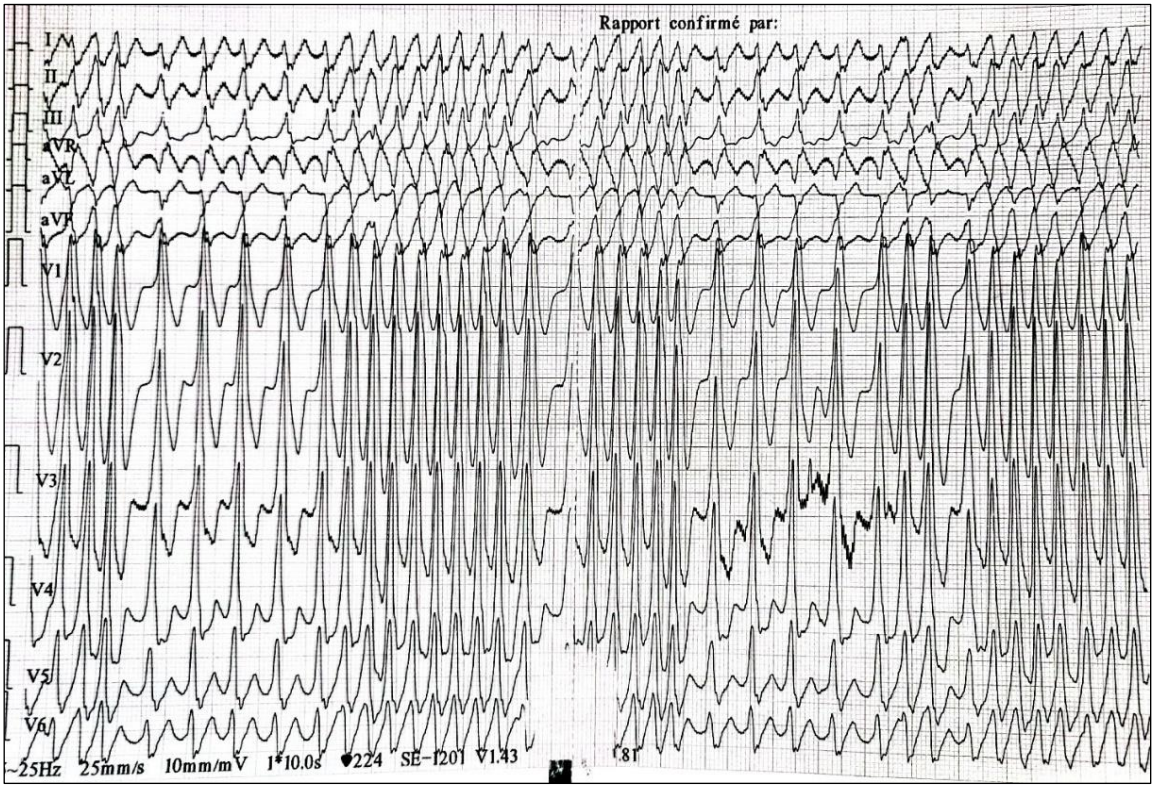

Figure 1: EKG at admission showing a supraventricular tachycardia

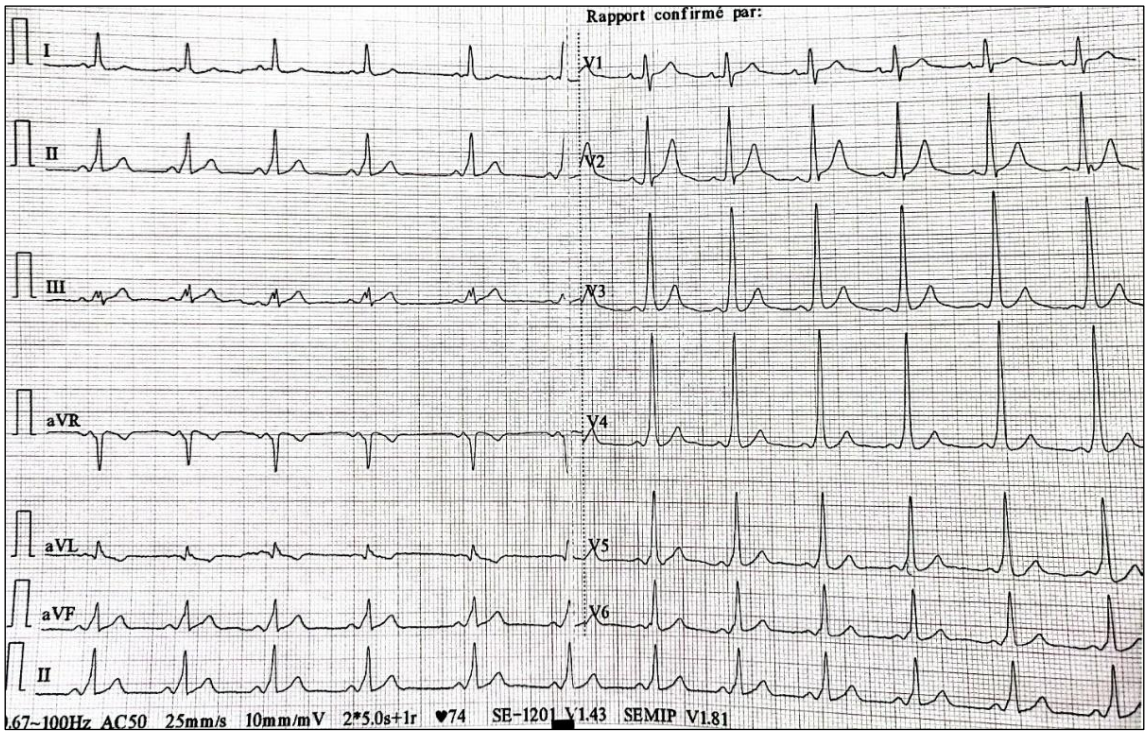

Figure 2: EKG after Synchronized DC cardioversion showing signs of WPW syndrome

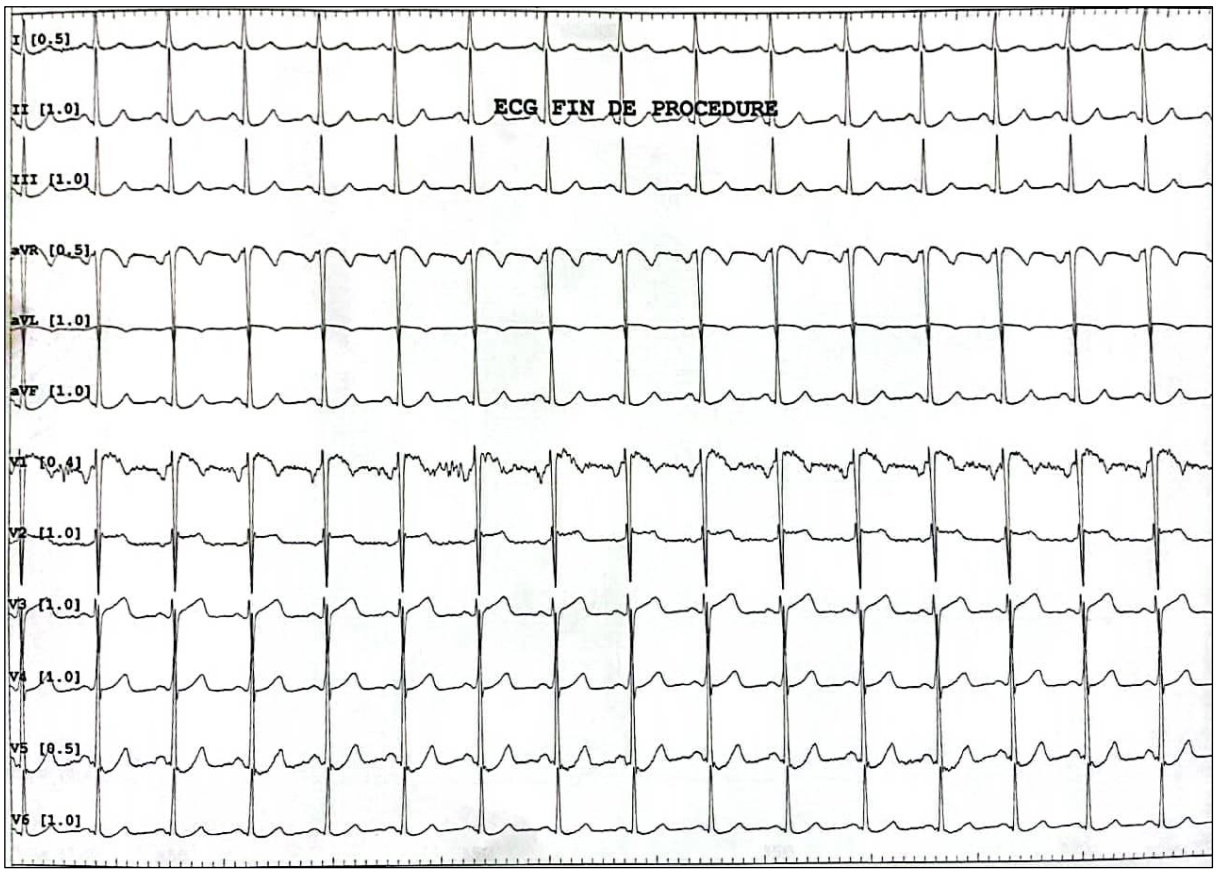

Figure 3: EKG after ablation of the accessory pathway

DISCUSSION

Wolff-Parkinson-White (WPW) syndrome is a congenital cardiac preexcitation syndrome that is secondary to an abnormal cardiac electrical conduction through an accessory pathway that can result in symptomatic and life-threatening arrhythmias [1].

The diagnosis of WPW syndrome rely on clinical symptoms namely palpitations, episodic lightheadedness, presyncope, syncope, or even cardiac arrest along with electrocardiographic findings consistent with WPW pattern or preexcitation such as short PR interval and prolonged QRS with an initial slurring upstroke ("delta" wave) in the presence of sinus rhythm [1].

On the anatomical level, a normal heart is formed by two electrically insulated units, the atria and the ventricles, these units are connected by the atrioventricular (AV) node, that transmits the action potential originating in the sinoatrial node, to the ventricles with a delay allowing a homogeneous ventricular depolarization and synchronized contraction [2].

WPW pattern is the result of ventricular preexcitation through abnormal accessory pathway formed during improper early atrial and ventricular folding in cardiac embryogenesis. This pathway usually has non-decremental conduction contrary to the AV node. the initiation and transmission of an arrhythmia leading to WPW syndrome, depends on location and number of accessory pathways, their speed of conduction, direction of conduction, and refractory period [2, 3].

The accessory pathways in WPW syndrome can initiate and maintain an atrioventricular reentrant tachycardia (AVRT) through a cycle between the atria, AV node, ventricles, and the accessory pathway. Orthodromic AVRT is usually characterized by narrow QRS complex and occurs when conduction progresses from the atria with antegrade conduction through the AV node and retrograde conduction through the accessory pathway while Antidromic AVRT follows the opposite path with antegrade conduction passing through the accessory pathway and retrograde conduction back up the AV node and is usually associated with a wide complex QRS [1].

Another way accessory pathways can lead to arrhythmia is by allowing non-decremental conduction of an arrhythmia generated in the atria responsible of immediate and non-delayed depolarization of the ventricles, with a rapid ventricular response in the case of atrial fibrillation (AF) or atrial flutter that can degenerate into ventricular fibrillation (VF) and cardiac arrest [1, 4].

The Most common arrythmia encountered in patients with WPW is AVRT with a frequency around 80%, succeeded by atrial fibrillation in 20 to 30%. Sudden cardiac death is a rare complication of WPW, it is the result of ventricular fibrillation secondary to AF, its frequency has been estimated at 0.9–2.4 per 1000 person-year [5].

In the event of pre-excited AF, usual treatment such as beta blockers, digoxin and amiodarone should be avoided. Their modulating effect on the AV node, favors the passage through accessory pathways, in this context Flecainide or propafenone could be considered in patients hemodynamically stable. In hemodynamically unstable patients, Synchronized DC cardioversion is recommended. Catheter ablation of accessory pathways is recommended in patients with symptomatic, recurrent AVRT [5, 6].

Our patient was admitted in pre-excited atrial fibrillation and benefited of a synchronized direct current defibrillation and was ulteriorly referred to a specialized center for ablation.

CONCLUSION

The onset of paroxysmal AF during WPW syndrome can be life-threatening in the short term and constitute a prognostic factor for the occurrence of sudden cardiac arrest. The electrocardiographic presentation can be misleading, leading to the prescription of potentially dangerous treatment in this context such as beta-blockers. The management of pre-excited AF must be rapid and is guided by the hemodynamics of the patient. However, the curative treatment is based on the ablation of the accessory pathways, which depends on the local expertise and the availability of the technical platform.

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