CASE REPORTS

A case of wide complex tachycardia in wolff-parkinson-white syndrome

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ABSTRACT

The manifestation of atrial flutter, particularly with 1:1 conduction, is rare in patients with ventricular preexcitation secondary to Wolff-Parkinson-White Syndrome (WPW). Very few cases have been reported in the literature. We present a 40-year old male with a history of untreated WPW who presented with severe chest pain and shortness of breath. He was found to have a rapid, regular, wide complex tachycardia. He underwent successful synchronized cardioversion, in which the patient converted to normal sinus rhythm with classic WPW waveform characteristics, including a shortened PR interval and prolonged QRS complex with a slurred upstroke. Surprisingly, a subsequent electrophysiology study revealed atrial flutter, with bystander conduction of 1:1 atrial flutter being the most likely cause of the patient's presenting symptoms, and a posteroseptal accessory pathway consistent with the diagnosis of WPW. While considerably rarer than ventricular tachycardia or AVRT, it is nevertheless important for clinicians to consider atrial flutter with 1:1 conduction as a potential diagnosis in patients with WPW presenting with wide complex tachycardia.

Key Words: Wolff-Parkinson-White, Atrial flutter, Bystander conduction, Wide complex tachycardia

1. INTRODUCTION

In patients diagnosed with Wolff-Parkinson-White Syndrome (WPW), supraventricular tachyarrhythmias are frequently observed, most often atrioventricular reciprocating tachycardia (AVRT), although other mechanisms are seen. We present a case of a patient with untreated WPW who presented with wide-complex tachycardia (WCT).

2. CASE PRESENTATION

A 40 year old man presented to the hospital with worsening palpitations accompanied by non-radiating, retrosternal chest pain for six hours. The patient reported a long history of recurrent palpitations, usually lasting several minutes each and resolving with rest, and was diagnosed with WPW on electrocardiogram three years prior. At the time of his diagnosis he declined any further studies or therapy, and did not receive any medical treatment for his palpitations over the three year interim. Six hours prior to presentation he experienced another episode of palpitations, however rather than improving with rest his palpitations intensified over time and were soon accompanied by worsening chest pain. The patient had no documented medical history other than the aforementioned diagnosis of WPW, was not taking any medications, endorsed occasional alcohol use and denied any tobacco or illicit drug abuse. On arrival, the patient was alert, oriented, and suffering from rapid palpitations and severe left-sided,

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non-radiating chest pain with diaphoresis. His heart rate was 251 beats/min, with a blood pressure of 138/83 mmHg. Temperature was normal, respiratory rate was 18 breaths/min, lungs were clear to auscultation bilaterally and no obvious murmurs were detected. A twelve-lead ECG revealed WCT at 251 beats/min (see Figure 1).

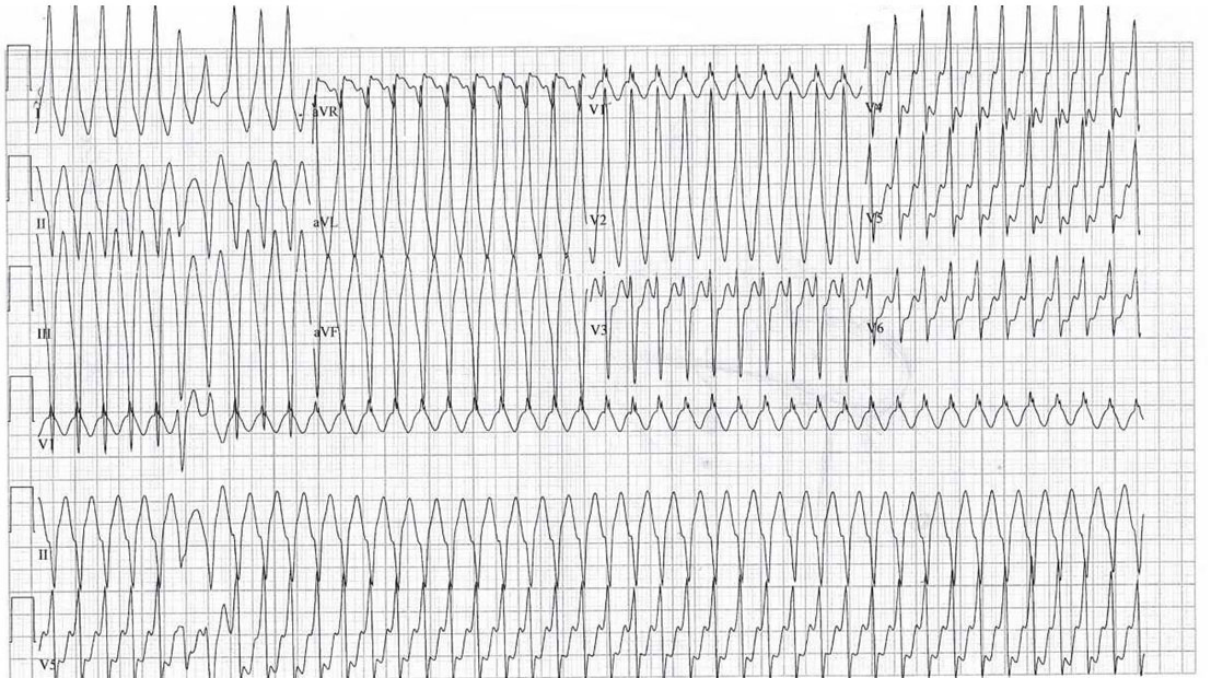

Figure 1. Twelve-lead electrocardiogram obtained following presentation to the ED, showing a regular wide complex tachycardia (QRS duration 136 ms) at 251 beats/min.

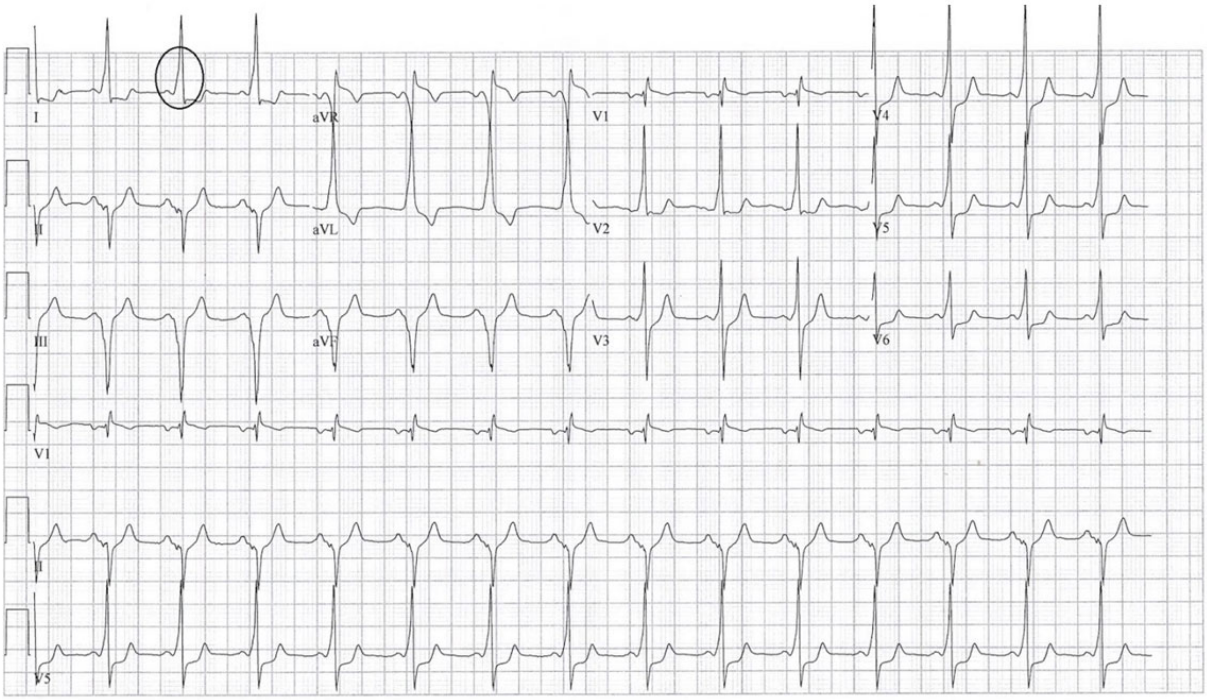

Figure 2. Twelve-lead electrocardiogram obtained after cardioversion showing normal sinus rhythm at 88 beats/min with shortened PR interval (130 ms) and prolonged QRS complex duration (120 ms) with slurred upstroke (delta wave).

After determining that the patient was displaying signs of hemodynamic instability, he was placed on supplemental oxygen via non-rebreather mask and underwent procedural sedation, followed by synchronized cardioversion with a single shock of 200 joules. Post-cardioversion ECG demonstrated normal sinus rhythm with classic WPW waveform characteristics, including a shortened PR interval and delta waves (see Figure 2). The patient's chest pain and diaphoresis resolved following synchronized cardioversion.

Laboratory workup was unrevealing, with normal complete blood count, chemistry panel, chest radiograph, cardiac troponins, and thyroid stimulating hormone. An echocardiogram showed a normal ejection fraction with trace aortic and mitral regurgitation. Following improvement of his symptoms the patient had an electrophysiology (EP) study, which confirmed a posteroseptal atrioventricular (AV) accessory pathway. During the electrophysiology study, orthodromic reciprocating tachycardia (ORT) was not induced. Catheter manipulation resulted in two episodes of atrial fibrillation during catheter placement, one of which required cardioversion and the other of which organized into atrial flutter and spontaneously terminated. The atrial flutter observed demonstrated variable cycle length (CL) from 210 to 240 ms, and conducted in a 2:1 pattern with pre-excitation. Subsequent burst pacing from the coronary sinus initiated clockwise cavotricuspid isthmus (CTI)-dependent atrial flutter. The patient underwent successful catheter ablation of both the posteroseptal accessory pathway and clockwise isthmus dependent atrial flutter. Given the findings on the EKG and during the EP study, the presenting rhythm was most likely 1:1 atrial flutter with bystander conduction down the accessory pathway.

3. Discussion

We present a case of very rapid, monomorphic WCT in the setting of Wolff-Parkinson-White Syndrome. While the specific etiology of this patient's tachyarrhythmia was unclear at the time of presentation, electrical cardioversion was still indicated due to the patient's accompanying ischemic chest pain and diaphoresis. His follow-up electrophysiology study revealed a posteroseptal accessory pathway and clockwise CTI-dependent atrial flutter, which were successfully ablated.

In a normal heart, the AV node's long refractory period insulates the ventricles from rapid atrial impulses.[1] However, accessory pathways can have shorter refractory periods than the AV node, thereby circumventing the AV node's protective delaying properties and allowing for more rapid conduction to the ventricles.[1–3] While normal conduction through the AV node and His-Purkinje system results in synchronized ventricular depolarization and thus a narrow QRS complex, most accessory pathways insert directly into the ventricular myocardium and conduct in an aberrant fashion, causing the ventricles to contract out of sync and thereby producing a wide QRS complex. In addition to direct conduction from the atria to the ventricles, accessory pathways can also cause wide complex tachycardias by forming reentrant circuits. For instance, antidromic AVRT is a reentrant tachycardia utilizing an accessory pathway as the antegrade limb of the tachycardia, and the AV node as the retrograde limb.[4–6] Furthermore, accessory pathways may also be bystanders in other forms of SVT, where they are not a critical part of the arrhythmic mechanism. In the setting of SVT, atrial fibrillation, or more rarely atrial flutter (as in this case), when the accessory pathway is not a part of the arrhythmia mechanism, the wide QRS complex is a result of the fusion of atrial impulses conducting slowly across the AV node and more rapidly across the accessory pathway.[2, 3, 7, 8]

We believe the clinician should be aware of certain considerations when treating patients with pre-excitation syndromes and atrial arrhythmias such as atrial fibrillation and atrial flutter. For instance, physicians should avoid using AV-nodal blocking agents in these patients, as this can perpetuate the conduction of rapid atrial impulses down the accessory pathway and lead to rapid ventricular response.[9] Rather than AV nodal blocking antiarrhythmics, current guidelines recommend medications that can directly slow conduction over the accessory pathway, including procainamide, and flecainide, or that terminate atrial flutter without promoting 1:1 conduction (ibutilide).[10]

4. Conclusion

While ventricular tachycardia, supraventricular tachycardia coupled with aberrancy, or AVRT may be more common causes of wide complex tachycardias in patients with WPW, physicians must also consider atrial arrhythmias such as atrial flutter with bystander conduction as an underlying mechanism. Physicians should endeavor to avoid using AV-nodal blocking agents in this patient population, as doing so can lead to preferred conduction of atrial impulses down the accessory pathway and subsequent rapid ventricular rates. Once stabilized, management of WPW and counseling patients regarding next steps is important to prevent further episodes. Patients with WPW who are symptomatic should be referred for an electrophysiology evaluation with the possibility of radiofrequency ablation of the accessory pathway.

Conflicts of Interest Disclosure

The author declares no conflict of interest

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