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ATRIAL FLUTTER WITH 1:1 CONDUCTION IN UNDIAGNOSED WOLFF-PARKINSON-WHITE SYNDROME

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□ **Abstract—Background: Atrial flutter with 1:1 atrioventricular conduction via an accessory pathway is an uncommon presentation of Wolff-Parkinson-White syndrome not previously reported in the emergency medicine literature. Wolff-Parkinson-White syndrome, a form of ventricular preexcitation sometimes initially seen and diagnosed in the emergency department (ED), can present with varied tachydysrhythmias for which certain treatments are contraindicated. For instance, atrial fibrillation with preexcited conduction needs specific consideration of medication choice to avoid potential degeneration into ventricular fibrillation. Case Report: We describe an adult female presenting with a very rapid, regular wide complex tachycardia successfully cardioverted in the ED followed by a normal electrocardiogram (ECG). Electrophysiology study confirmed atrial flutter with 1:1 conduction and revealed an accessory pathway consistent with Wolff-Parkinson-White syndrome, despite lack of ECG findings of preexcitation during sinus rhythm.**

Why should an emergency physician be aware of this? Ventricular tachycardia must be the first consideration in patients with regular wide complex tachycardia. However, clinicians should consider atrial flutter with 1:1 conduction related to an accessory pathway when treating patients with the triad of very rapid rate (>250 beats/min), wide QRS complex, and *regular* rhythm, especially when considering pharmacologic treatment. Emergency physicians also should be aware of electrocardiographically concealed accessory pathways, and that lack of delta waves does not rule out preexcitation syndromes such as Wolff-Parkinson-White syndrome. © 2013 Elsevier Inc.

□ **Keywords—preexcitation; Wolff-Parkinson-White syndrome; atrial flutter; wide complex tachycardia; accessory pathway**

INTRODUCTION

Preexcitation syndromes, such as Wolff-Parkinson-White syndrome, frequently cause supraventricular tachydysrhythmias, most commonly, atrioventricular reciprocating tachycardia and atrial fibrillation. We present a case of atrial flutter with 1:1 conduction in a patient ultimately diagnosed with Wolff-Parkinson-White syndrome.

CASE REPORT

A 48-year-old woman presented by private car after several 5–10-min episodes of retrosternal, nonradiating chest pain and palpitations over the prior 6 h. She briefly lost consciousness once, but initially refused to come to the hospital. She had similar, less severe episodes of palpitations monthly for two decades, but never sought medical attention. Her medical history included hypertension and hyperthyroidism treated with radiofrequency ablation therapy years earlier with subsequent hypothyroidism.

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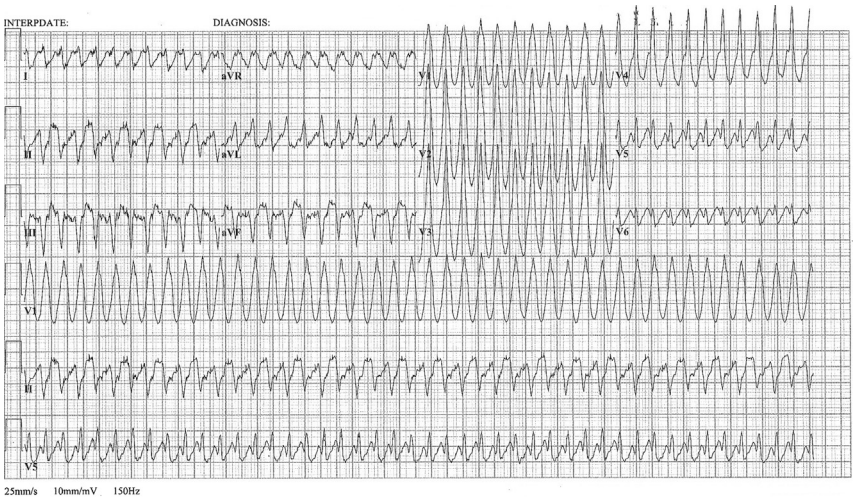

Figure 1. Twelve-lead electrocardiogram obtained shortly after presentation to the emergency department revealing a regular wide complex tachycardia (QRS duration 132 ms) at 270 beats/min.

She smoked cigarettes but denied illicit drug or alcohol use. Her medications included levothyroxine and lisinopril.

She arrived alert, oriented, and mildly anxious. The heart rate was 270 beats/min, with a blood pressure of 130/106 mm Hg. Respiratory rate was 20 breaths/min, and temperature was normal. Lungs were clear to auscultation. She had no obvious murmurs. Radial pulses were thready but symmetric and she had acrocyanosis. Twelve-lead electrocardiogram (ECG) revealed regular wide complex tachycardia at 270 beats/min (Figure 1).

Due to signs of decreased perfusion, the patient underwent procedural sedation with propofol and fentanyl followed by synchronized cardioversion with a single biphasic shock of 200 Joules. Postcardioversion ECG demonstrated normal sinus rhythm (Figure 2). Chest pressure and acrocyanosis resolved after cardioversion.

Laboratory workup was unrevealing as to potential causes of tachydysrhythmia, with normal complete blood count, chemistry panel, chest radiograph, and thyroid-stimulating hormone. Cardiology and Electrophysiology consultants saw her in the hospital and she had cardiac catheterization the following day, revealing mild nonocclusive coronary artery disease. During the procedure, she developed sustained narrow complex tachycardia at 190–200 beats/min, but remained hemodynamically stable with only mild symptoms. She then went to electrophysiology study and was found to have a left posteroseptal atrioventricular (AV) accessory pathway that conducted in both antegrade and retrograde directions very rapidly. AV orthodromic reciprocating tachycardia using this AV accessory pathway was induced easily and repeatedly. She had no preexcitation in baseline sinus rhythm, but developed maximal preexcitation during

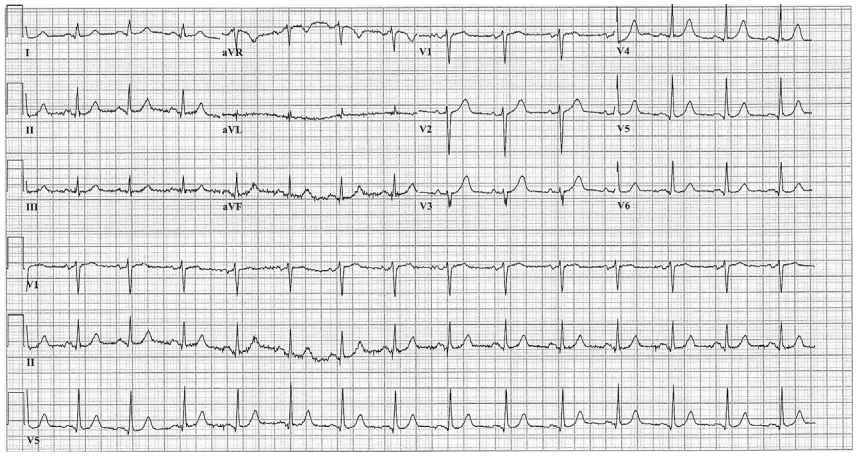

Figure 2. Twelve-lead electrocardiogram obtained after direct current cardioversion showing normal sinus rhythm with PR interval 136 ms and QRS duration 74 ms, and no appreciable delta waves.

rapid atrial pacing at 270 beats/min with ECG similar to her clinical dysrhythmia (Figure 3A). The patient subsequently went into spontaneous atrial flutter with 1:1 AV conduction via the accessory pathway at 270 beats/min, confirmed by intracardiac electrogram, with a 12-lead ECG identical to her ECG with wide complex tachycardia in the emergency department (ED) (Figure 3B). No ventricular tachycardia was inducible. She underwent successful transseptal puncture with three-dimensional mapping and catheter ablation of the AV accessory pathway. Transthoracic echocardiography later that day revealed normal right and left ventricular size and function with estimated ejection fraction of 60%. Troponin I peaked at 0.050 ng/mL (normal 0–0.03 ng/mL).

Holter monitoring 6 weeks later showed two asymptomatic runs of nonsustained atrial tachycardia without preexcitation. She later developed isolated episodes of palpitations without associated ischemic symptoms or presyncope. She refused to wear an event monitor and has not sought further medical attention.

DISCUSSION

We present a case of very rapid, regular wide complex tachycardia in the setting of previously undiagnosed Wolff-Parkinson-White syndrome in a patient lacking evidence of preexcitation during sinus rhythm on her ECG. In the ED, we did not definitively diagnose this tachydysrhythmia due to lack of intracardiac monitoring and because electrical cardioversion was indicated regardless of the underlying rhythm due to impaired peripheral perfusion. However, she had an identical tachydysrhythmia during her electrophysiology study. That study revealed the presence of an AV accessory pathway with bidirectional conduction. In addition to easily inducible orthodromic reciprocating tachycardia, she developed atrial flutter at 270 beats/min with 1:1 AV conduction via the accessory pathway and reproduced her clinical wide complex tachycardia. The patient underwent successful catheter ablation of the accessory pathway and had no recurrent problem at follow-up.

Differential diagnosis of regular wide complex tachycardia is fairly limited. Most commonly, the dysrhythmia is generated in the ventricles. However, several mechanisms can lead to supraventricular wide complex tachycardia—aberrant conduction through the AV node and His-Purkinje system, conduction through an accessory pathway, or conduction through both the AV node/His-Purkinje system and an accessory pathway (Table 1).

Atrial flutter with 1:1 conduction is rarely reported in the cardiology literature, especially in conjunction with preexcitation. To have atrial flutter with 1:1 conduction, a patient must have both an accessory pathway with an extremely short refractory period and a slow atrial flutter. Our patient's atrial flutter cycle length was 222 ms and her accessory pathway refractory period was <200 ms. Most often, 1:1 conduction is reported in patients taking sodium channel blockers, which, by slowing the rate of atrial conduction, slow the atrial flutter rate itself; but it has been reported to happen spontaneously (1–3). When treating atrial flutter with 2:1 conduction, one should avoid medications that slow AV conduction

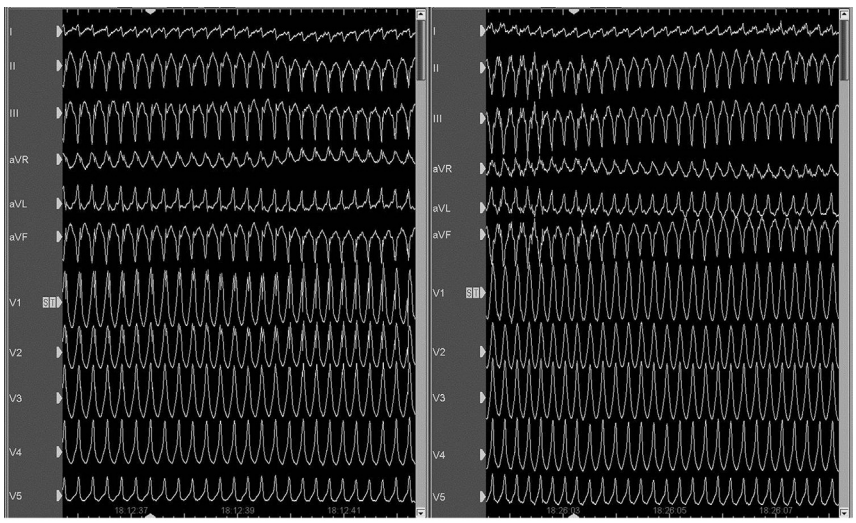

Figure 3. (A) Twelve-lead electrocardiogram (ECG) obtained during the electrophysiology study when the high right atrium was paced at 270 beats/min. Note that the maximal preexcitation pattern was identical to the patient's regular wide complex tachycardia in the Emergency Department (Figure 1). (B) Twelve-lead ECG obtained during the electrophysiology study when atrial flutter was induced with 1:1 atrioventricular conduction at 270 beats/min and was identical to her clinical tachycardia. Lead V6 was not included here due to the poor quality of the tracing.

Table 1. Differential Diagnosis of Regular Wide Complex Tachycardia

Ventricular tachycardia
Supraventricular tachycardias
Atrioventricular (AV) aberrant conduction via AV node and His-Purkinje system
AV nodal reentrant tachycardia (AVNRT) with aberrancy
Orthodromic AV reciprocating tachycardia (ORT) with aberrancy
Atrial tachycardia with 1:1 AV conduction with aberrancy
Atrial flutter with 1:1 AV conduction with aberrancy
Atrioventricular (AV) conduction via an accessory pathway
Antidromic reciprocating tachycardia (ART)
Atrioventricular (AV) conduction via an accessory pathway and AV node
Atrial tachycardia with 1:1 AV conduction via an accessory pathway
Atrial flutter with 1:1 AV conduction via an accessory pathway

prior to giving AV nodal blocking agents. By slowing the atrial rate with procainamide, for instance, one can paradoxically change conduction from 2:1 to 1:1 and then have an increased ventricular rate, which can degenerate into ventricular fibrillation.

Wolff-Parkinson-White syndrome is a form of ventricular preexcitation estimated to occur in 0.1–3% of the general population first described in the 1930s (4,5). Electrical impulse from the atria reaches the ventricles by a pathway other than the AV node, termed the accessory pathway (AP), which may conduct electrical impulse more quickly than the AV node. The pathophysiology and varied ECG findings of preexcitation are well described by Mark et al. (6).

Wide complex tachycardia can develop in patients with Wolff-Parkinson-White syndrome through multiple mechanisms because the impulse from the atria can transmit to the ventricles via the AV node, an accessory pathway, or both. In supraventricular tachycardia transmitted via the AV node, aberrant conduction through the His-Purkinje system can lead to a wide QRS complex. Second, if the supraventricular tachycardia is transmitted down an accessory pathway and back up the AV node in a retrograde fashion, the QRS will also be wide due to impulse being conducted through the ventricular myocardium. In Wolff-Parkinson-White syndrome with atrial fibrillation or flutter, as in our patient, atrial impulses approach the AV node and accessory pathway very rapidly and can transmit to the ventricles via both. The widened QRS in this case represents fusion of two separate complexes—1) depolarization from the rapidly conducting accessory pathway directly to the ventricular myocardium, and 2) depolarization from the slowly conducting AV node to the rapidly conducting Purkinje system. The AV node's long refractory period protects the ventricles from conducting at extremely rapid rates, usually no faster than 200 beats/min, however, an accessory pathway in a patient with Wolff-Parkinson-White syndrome may have a much shorter refractory period that can allow more rapid impulses to reach the ventricles.

The true incidence of preexcitation is difficult to determine. Many patients do not present due to minimal symptoms, and ECG may not always be performed when preexcitation is present. Priorities for the emergency physician are to suspect preexcitation, manage dysrhythmias safely and effectively, and arrange appropriate follow-up.

Suspicion for Preexcitation

Preexcitation should be suspected in patients with a history of recurrent episodes of palpitations, especially those with symptoms of poor perfusion such as syncope, near-syncope, chest pain, shortness of breath, or lightheadedness.

Electrocardiography can offer clues to preexcitation, but may be limited such as in this case. The classic ECG findings of Wolff-Parkinson-White syndrome include: 1) short P-R interval, 2) delta waves, 3) prolonged QRS complex (>120 ms), and 4) secondary ST-T wave changes discordant to the QRS vector (6,7). The location and magnitude of the ST-T changes vary depending on the accessory pathway's location and electrophysiological characteristics.

However, a normal baseline ECG does not rule out Wolff-Parkinson-White syndrome. Our patient did not have a delta wave at baseline; therefore, her accessory pathway is concealed electrocardiographically. Concealed accessory pathways occur by several mechanisms. Some accessory pathways can only transmit in a retrograde fashion. Others that can transmit in an antegrade direction do so only intermittently (8). In some instances, the conduction velocity through the AV node could be relatively brisk as compared to accessory pathway. As a result, the ventricles are activated predominantly via the His-Purkinje system and make the preexcitation not obvious. Other pathways, such as the one in our patient, are located so far from the sinus node that the impulse transmits though the AV node before it reaches the AP. Electrophysiologic properties of accessory pathways and the surrounding myocardium can also change as a result of many factors, such as age, heart rate, electrolyte balance, hypoxia, cardioactive medications, and autonomic nervous system tone (9,10).

Management of Wide Complex Tachydysrhythmias

Most wide complex tachycardias are ventricular tachycardia (VT), especially in the presence of structural heart disease. Next most frequent is supraventricular tachycardia with aberrant conduction. However, less common

Table 2. ECG Distinctions for Diagnosis of Wide QRS Complex Tachycardia

Favor Supraventricular Tachycardia (SVT)	Favor Ventricular Tachycardia (VT)
Initiation with premature atrial contraction (PAC)	Initiation with premature ventricular contraction (PVC)
QRS complexes identical to those in sinus rhythm	QRS complexes in tachycardia identical to PVCs during sinus rhythm
Changes in P-P interval precede changes in R-R interval	Changes in R-R interval precede changes in P-P interval
QRS morphology matches with aberrant conduction	QRS morphology does not match with aberrant conduction
QRS duration <0.14 s	QRS duration >0.14 s
Slowing or termination with vagal maneuvers	AV dissociation, fusions beats or capture beats
	Left axis deviation (180–270°)

AV = atrioventricular.

etiologies such as preexcitation must be considered, especially when contemplating antidysrhythmic therapy (Table 1). Many criteria have been proposed to aid in the differentiation between VT and supraventricular tachycardia (SVT) with aberrancy based on a 12-lead ECG, however, none are perfect (11,12). Table 2 lists factors that favor either VT or SVT. Exact diagnosis of the tachydysrhythmia is, in many cases, irrelevant to emergency management of the dysrhythmia, especially when the patient has hemodynamic instability. When the diagnosis cannot be made definitively, it is prudent for the emergency physician to assume the diagnosis is ventricular tachycardia.

The notable exception is atrial fibrillation with Wolff-Parkinson-White syndrome, which is suggested by the combination of wide QRS complex, extremely fast rate, and irregular rhythm on the ECG. Medications that slow conduction through the AV node, such as beta-adrenergic blockers, calcium channel blockers, digitalis, and adenosine, should be avoided due to concern about causing preferential conduction down the accessory pathway and precipitating an increasingly rapid ventricular rate and possible cardiovascular collapse. We propose having similar concerns when seeing the patient with wide complex, *extremely* fast heart rate, and *regular* rhythm, as this could be atrial flutter with preexcitation.

In the ED, clinical stability is a more important determinant of treatment than underlying rhythm. Hemodynamic instability thought to be related specifically to the presenting tachydysrhythmia should prompt immediate direct current cardioversion, regardless of the specific etiology of the rhythm.

Arrange Specialty Consultation

Counseling the patient with suspected or confirmed tachydysrhythmia or incidentally found delta waves can be challenging. Many patients with delta waves will never have a symptomatic dysrhythmia and some patients have concealed accessory pathways. However, there is a low, but measurable risk of sudden cardiac death in this patient population (13). Fitzsimmons et al. found a 0.2%/patient-year rate of sudden cardiac death in patients with Wolff-Parkinson-White ECG findings (14).

Electrophysiology evaluation with radiofrequency ablation is very effective at preventing recurrent dysrhythmias and sudden cardiac deaths. Patients with suspected or documented tachydysrhythmias should be referred for outpatient monitoring or cardiology consultation. Asymptomatic patients with incidentally discovered delta waves should also be referred for counseling and risk stratification as there have been reports of Wolff-Parkinson-White syndrome presenting as sudden cardiac death (15).

Some patients are at higher risk of life-threatening dysrhythmia, but only some have predictors that are apparent to the emergency physician. Obviously, patients presenting with hemodynamic instability, documented ventricular fibrillation, or resuscitated cardiac arrest are at high risk of recurrence. Patients with persistent delta waves on ECG, documented tachydysrhythmias, and more than one type of supraventricular tachycardia are also higher risk (16). Patients with documented shortest R-R interval of <220 ms give clear proof that their pathway can conduct very quickly and would benefit from ablation (17).

WHY SHOULD AN EMERGENCY PHYSICIAN BE AWARE OF THIS?

Patients with preexcitation syndromes such as Wolff-Parkinson-White syndrome can be difficult to diagnose and risk-stratify. Emergent management of the patient with a tachydysrhythmia should initially be dictated by the patient's clinical status. Although ventricular tachycardia must be the first consideration in evaluating patients with wide complex tachycardia and regular rhythm, clinicians should consider atrial flutter with 1:1 conduction related to an accessory pathway when treating patients with the triad of very rapid rate (>250 bpm), wide QRS complex, and *regular* rhythm. Pharmacologic management of this condition should be guided by the same principles as atrial fibrillation with suspected preexcitation conduction.

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