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Case Report

Unresponsive to Cardioversion Pre-excited Irregular Rhythm

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ABSTRACT

Case Summary: A 46-year-old man without known past medical history was referred to the tertiary hospital emergency department after being found collapsed at home. There are no any complaints of any headache, nausea, chest pain, or paralysis of extremities before losing consciousness. His chest examination to bilateral auscultation was clear. Chest X-ray, routine blood work, and transthoracic echocardiography did not reveal any abnormalities. The initial heart rate before referral was 250-300/min and the ECG shows irregular wide QRS complex tachycardia. The ECG after cardioversions shows sinus rhythm 86 bpm with WPW type A pattern. The patient got intravenous amiodarone and intravenous lidocaine during the transfer. And the ECG on arrival at the emergency department, the heart rate was 50-150 bpm irregularly and the ECG shows atrial fibrillation with a narrow QRS complex. **Discussion:** Rapid anterograde accessory pathway conduction during atrial fibrillation (AF) can result in sudden cardiac death. During pre-excited AF, delta waves as the key feature of Wolff-Parkinson-White (WPW) syndrome might be obscured. We should keep in mind the diagnosis of pre-excited AF in patients presenting with irregular and wide complex tachycardia.

Highlights:

1. Atrial fibrillation in the presence of an accessory pathway may present with confounding electrocardiographic signs.
2. The clinical recognition of WPW may be hindered by the presence of pre-excited AF.

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Introduction

The classical manifestation of WPW syndrome is the presence of bundle of Kent, also known as the auxiliary channel through which fast anterograde conduction can outperform slower atrioventricular (AV) node conduction. By causing the ventricles to depolarize relatively quickly, this route causes distinctive ECG alterations, including a short PR interval, a large QRS complex, and the nearly pathognomonic delta wave. In "concealed" WPW syndrome, the accessory pathway may not conduct in an anterograde fashion, making it challenging to detect any electrocardiographic anomalies at baseline. [1]

The atria can discharge at a rate of more than 300 impulses per minute during pre-excited AF, masking delta waves—the primary electrocardiographic hallmark of WPW syndrome. Due to decremental conduction, an intrinsic repolarization trait that permits the node to conduct more slowly when it receives quicker signals, the AV node often blocks the majority of these impulses. An auxiliary channel without such a built-in delay, on the other hand, allows for 1:1 conduction with ventricular rates reaching 300 bpm. Pre-excited AF is thus classified as a malignant arrhythmia since it can lead to abrupt cardiac death if it degenerates into ventricular fibrillation. [2,3]

The irregular wide complex tachycardia (WCT) with QRS of varied morphology and amplitude with sustained rates over 200 bpm is the key to recognizing WPW syndrome with pre-excited AF. Procainamide or ibutilide may be beneficial in reducing conduction velocity of the auxiliary route if the patient's blood pressure is steady. This rhythm can be difficult to distinguish from polymorphic ventricular tachycardia, although electrical cardioversion is the primary treatment for both in the context of hemodynamic instability. Radiofrequency ablation is the definitive treatment for pre-excited AF in WPW syndrome for the prevention of recurrent arrhythmias. [4]

Case Presentation

A 46-year-old man without known past medical history was referred to the tertiary hospital emergency department after being found collapsed at home. He did not complain of any headache, nausea, chest pain, or paralysis of extremities before losing consciousness. He was hospitalized for 3 days before the referral. On arrival, Glasgow Coma Scale was 456, his heart rate was 50-150 bpm irregularly, blood pressure of 150/90 mmHg, respiratory rate of 20/min, and peripheral oxygen saturation of 98% on 2 lpm nasal canula. His chest examination was clear to bilateral auscultation. Chest X-ray, routine blood work, and transthoracic echocardiography did not reveal any abnormalities.

His initial heart rate before referral was 250-300/min and initial electrocardiogram (ECG) on the referral hospital is shown in Figure 1. The patient underwent multiple cardioversions before being referred to our hospital. Figure 2 shows his ECG after cardioversions. He was on intravenous amiodarone and intravenous lidocaine during the transfer. His ECG on arrival at our emergency department is shown in Figure 3.

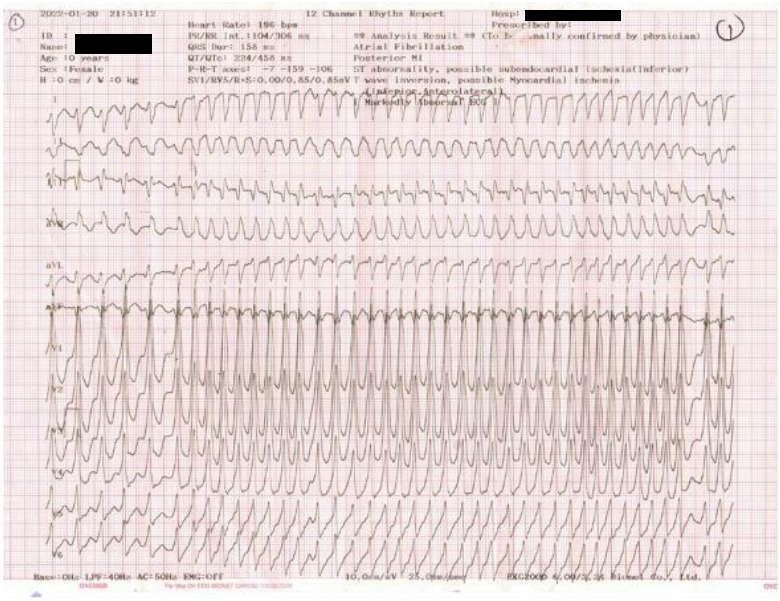

Figure 1. Initial electrocardiogram (ECG) at the referral hospital. This ECG shows irregular wide QRS complex tachycardia at a rate of around 150-200 bpm on the first 5 beats, followed by wide QRS complex tachycardia at a rate of 300 bpm suggesting ventricular tachycardia

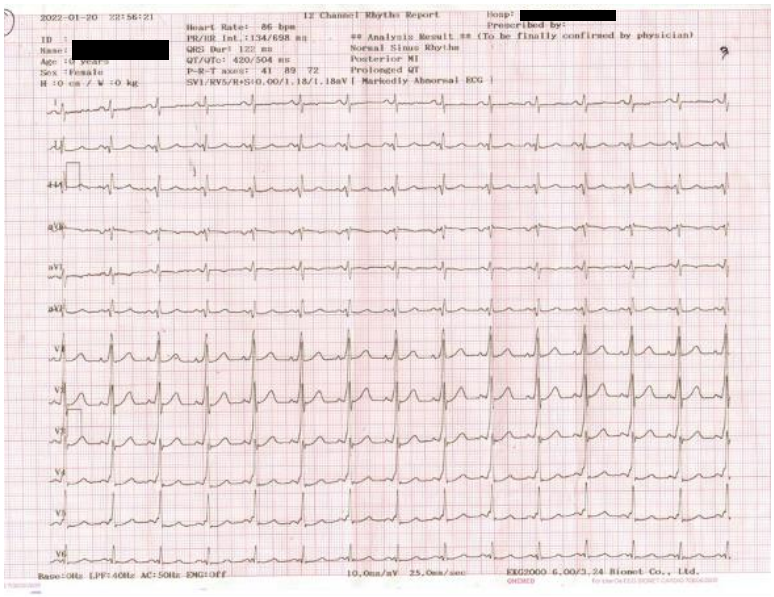

Figure 2. Patient's ECG after cardioversion. It shows sinus rhythm 86 bpm with WPW type A pattern

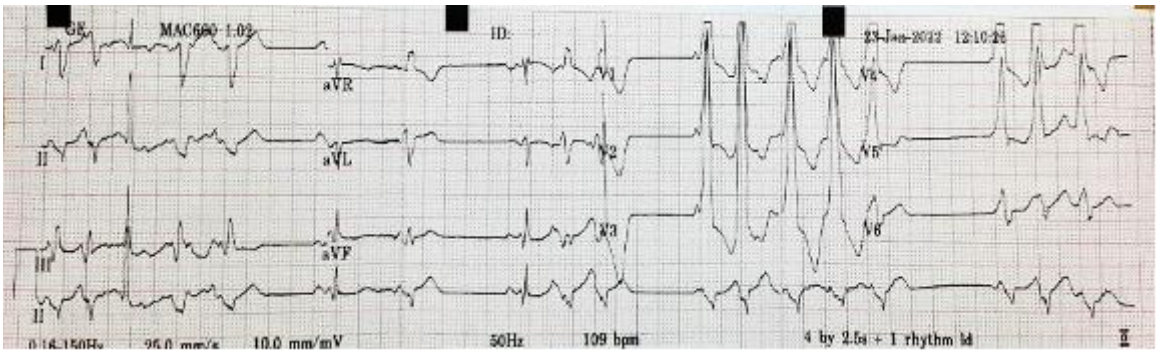

Figure 3. ECG on arrival at our emergency department. It shows atrial fibrillation with a narrow QRS complex alternating with a wide QRS complex

What Drug Would You Choose?

A. Verapamil

B. Amiodarone

C. Ibutilide

D. Propafenone

Diagnosis

Rapid Atrial Fibrillation with WPW Syndrome

What Drugs to Choose

C. Ibutilide

Electrical cardioversion should be readily available for hemodynamically compromised patients with pre-excited Atrial Fibrillation (AF). Atrioventricular (AV) node-modulating drugs (e.g. verapamil, beta blockers, digoxin) should be avoided as these drugs may suppress conduction via the AV node and aggravate conduction via the accessory pathway. Amiodarone may not be safe in pre-excited AF as it may enhance accessory pathway conduction. Pharmacological cardioversion can be attempted using ibutilide, whereas class I anti-arrhythmic drugs (procainamide, propafenone, flecainide) should be used with caution owing to their effect on the AVN.

Discussion

This case serves as a crucial reminder that atrial fibrillation (AF) in the presence of an accessory pathway may present with confounding electrocardiographic signs, perhaps resulting in inaccurate diagnosis and potentially fatal therapies. Even though there is a 10% to 30% prevalence of pre-excited AF in the presence of an accessory pathway and general medical practitioners are generally aware of the Wolff-Parkinson-White (WPW) syndrome, the clinical recognition of WPW may be hindered by the presence of pre-excited AF[4]. Wolff-Parkinson-White (WPW) syndrome is the commonest pre-excitation disorder with an incidence of 0.1–0.3% in the general population and an associated sudden cardiac death risk of less than 0.6%. [5]

The most important clinical significance of WPW syndrome is the frequent occurrence of supraventricular tachycardias, such as atrioventricular re-entrant tachycardia (AVRT), AF, and atrial flutter. Rapid anterograde accessory pathway conduction during AF can result in sudden cardiac death in patients with a manifest accessory pathway.[6] During pre-excited AF, the atria can discharge at a rate higher than 300 impulses per minute, obscuring delta waves—the key electrocardiographic feature of WPW syndrome. The AV node normally blocks most of these impulses due to decremental conduction, an intrinsic repolarization property that allows the node to conduct more slowly when it receives faster signals. However, an accessory pathway without such a built-in delay makes 1:1 conduction possible, with ventricular rates reaching 300 bpm. Pre-excited AF is thus characterized as a malignant arrhythmia, as sudden cardiac death may result from this rhythm degenerating into ventricular fibrillation. [2,7,8]

The presenting ECG of this patient shows wide QRS complex tachycardia at a rate of 300 bpm. Although there is no clear AV dissociation, the presence of a positive initial R wave in lead aVR, positive concordance in all precordial leads, and onset to nadir of R wave in lead V1 longer than 100 milliseconds, suggesting VT. The other less common cause is SVT with bundle-branch aberrancy or ventricular activation via an accessory pathway. Ventricular tachycardia and SVT are commonly regular rhythms. However, the tachycardia in this case was irregular and the ECG findings had no discernible P waves. Moreover, there was no evidence of QRS fusion or capture beats, which are characteristic of VT. In addition, the QRS morphology did not show a typical right bundle branch block (RSR' in lead V1). All those findings make VT diagnosis less likely. The patient experienced syncope, so synchronized direct-current cardioversion was performed.

The second ECG revealed the patient's baseline ECG as sinus rhythm and type A WPW pattern with shortened PR and positive delta waves in V1. Hence, the presenting rhythm was not consistent with classic bundle-branch block patterns. This raises the possibility of a wide complex rhythm (Figure 1) being atrial fibrillation with accessory pathway conduction. Normal sinus rhythm with pre-excitation suggestive of left lateral or left anterolateral accessory pathway was noted.

The third ECG shows atrial fibrillation with variation of QRS morphology and width. Varying QRS morphology is often present in patients with pre-excited AF owing to beat-to-beat variation in the conduction to the ventricle between the accessory pathway and AV node. These findings make pre-excited AF more likely.

AF with WPW syndrome should be suspected in tachycardia with wide and irregular QRS complexes. Several important features lead to the diagnosis of AF with WPW syndromes, such as an irregular rhythm, rapid ventricular response (too fast for conduction through the AV node), and the wide-bizarre QRS complex. Occasionally a narrow QRS may be seen, indicating conduction through the AV node. Careful interpretation of the ECG must be confirmed with the clinical context. The probability of AF with WPW syndrome is increased in younger patients <50 age with a previous history of palpitations, rapid heart rate, syncope, or a documented history of WPW syndrome. However, the rapid ventricular rate and wide QRS complex are poor differentiators of AF with WPW syndrome from other wide-complex tachyarrhythmias. Meanwhile, irregular rates and variations of bizarre QRS complex morphologies suggest AF with WPW syndrome.

The ECG features of polymorphic VT are similar to those of AF with WPW syndrome. Polymorphic VT has wide QRS complexes with a fast-ventricular rate (150-300 beats/min), variable RR intervals, and frequently changing QRS complexes. Torsade de pointes is a subtype of polymorphic VT with undulating baselines that distinguishes it from AF with the WPW syndrome, which usually has a stable baseline with no alteration in the polarity of the QRS complex.

Atrial fibrillation with aberrant ventricular conduction is observed when the impulse from AF is conducted to the ventricle with a pre-existing bundle branch block or rate-dependent bundle branch block. The ECG shows irregular broad complex tachycardia with monotonous QRS configuration, unlike AF with WPW syndrome with variable QRS configuration.

AF with an antegrade conduction accessory pathway increases the risk of sudden cardiac death by rapid conduction over the accessory pathway. A shortest pre-excited RR interval of <250 ms during AF predicts an increased risk of degeneration to ventricular fibrillation. AV nodal blocking agents should be avoided because they increase the risk of preferential conduction down the accessory pathway with degeneration to ventricular fibrillation.[9,10]

Acute treatment of pre-excited AF requires a rapid-acting drug that can be given intravenously and can slow conduction in the accessory pathway. Patients with pre-excited AF who are hemodynamically stable may be treated with intravenous ibutilide (prolongs refractoriness of both AV node and accessory pathways) or procainamide (because of its effects on the atrial myocardium). Intravenous amiodarone can be used if ibutilide or procainamide is unavailable, but these patients should be monitored closely because of the effects of amiodarone on the AV node. The most recent European Society of Cardiology SVT guidelines

report a class III recommendation for amiodaron in pre-excited AF. Hemodynamically unstable patients should undergo immediate cardioversion. [10]

Patient Outcome

The patient underwent an electrophysiological study during his stay and it was found that there are several mechanisms underlying his ECG patterns: Antidromic Atrioventricular Re-entry Tachycardia, WPW Syndrome with Left Lateral/ Left Anterolateral accessory pathway, and intermittent Atrial Flutter. He underwent ablation but the results were not satisfactory. Due to logistic limitations, he was discharged on Propafenone 300 mg three times daily and was scheduled for re-ablation with a transeptal approach. During outpatient follow-up, the patient did not experience any syncopal episodes and his symptoms were minimal.

Conclusion

In conclusion, we should keep in mind pre-excited Atrial Fibrillation or Atrial Flutter in irregular and wide complex tachycardias. Wolff-Parkinson-White (WPW) syndrome should always be suspected in a patient presenting with symptomatic irregularly irregular broad complex tachycardia. Pre-excited AF is a life-threatening arrhythmia. The main treatment of pre-excited AF in our country is electrical cardioversion whether patients are stable or not, because of the absence of available drugs (intravenous ibutilide and procainamide). But the only long-term therapy is catheter ablation.

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