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Case Report

Atrial Fibrillation with Ventricular Pre-Excitation: A Diagnosis That Must Not Be Missed

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Abstract

Wolff-Parkinson-White (WPW) syndrome is caused by an accessory pathway that communicates between the atria and ventricles known as the Bundle of Kent. The development of atrial fibrillation, can result in the atrial impulses all being conducted via the accessory pathway and result in a sinister, board complex, irregular tachycardia, with varying QRS morphology (known as pre-excited atrial fibrillation) Adenosine is a potent atrioventricular node blocker, which can be used in the treatment of supraventricular tachycardias, but also has diagnostic utility, particularly in differentiating between supraventricular tachycardia with aberrant conduction (which would often terminate) and a ventricular tachycardia (which would not respond to adenosine). However, the administration of adenosine in pre-excited atrial fibrillation can precipitate 1:1 atrial to ventricular conduction, which can degenerate into life-threatening ventricular arrythmias. This case describes a patient who presented with pre-excited atrial fibrillation and received intravenous adenosine that resulted in development of broad complex tachycardia with haemodynamic compromise. In patients with pre-exited atrial fibrillation, AV nodal blocking agents should be avoided and direct current cardioversion should be utilised.

Key words: wolff-parkinson-white; haemodynamic; atrial fibrillation

INTRODUCTION

Wolff-Parkinson-White (WPW) syndrome is caused by an accessory pathway that communicates between the atria and ventricles known as the Bundle of Kent. The development of atrial fibrillation, can result in the atrial impulses all being conducted via the accessory pathway and result in a sinister, board complex, irregular tachycardia, with varying QRS morphology (known as pre-excited atrial fibrillation).[1] This case describes a patient in whom this diagnosis was not initially recognised and the administration of intravenous adenosine precipitated a broad complex tachycardia with haemodynamic compromise.

Case report

A young male who was previously fit and well presented to hospital due to palpitations. These began gradually and worsened over the preceding hour. On arrival he still had palpitations, but did not report any chest pain, shortness of breath or dizziness. He denied having any other medical conditions, any family history of cardiac disease or sudden unexplained death, and denied recreational drug use. Clinical examination was unremarkable apart from a resting tachycardia (167 bpm) and his blood pressure was normal (132/75 mmHg). The rhythm strip of his electrocardiogram (ECG) demonstrated an irregular broad complex tachycardia, with variable QRS morphology (Figure 1).

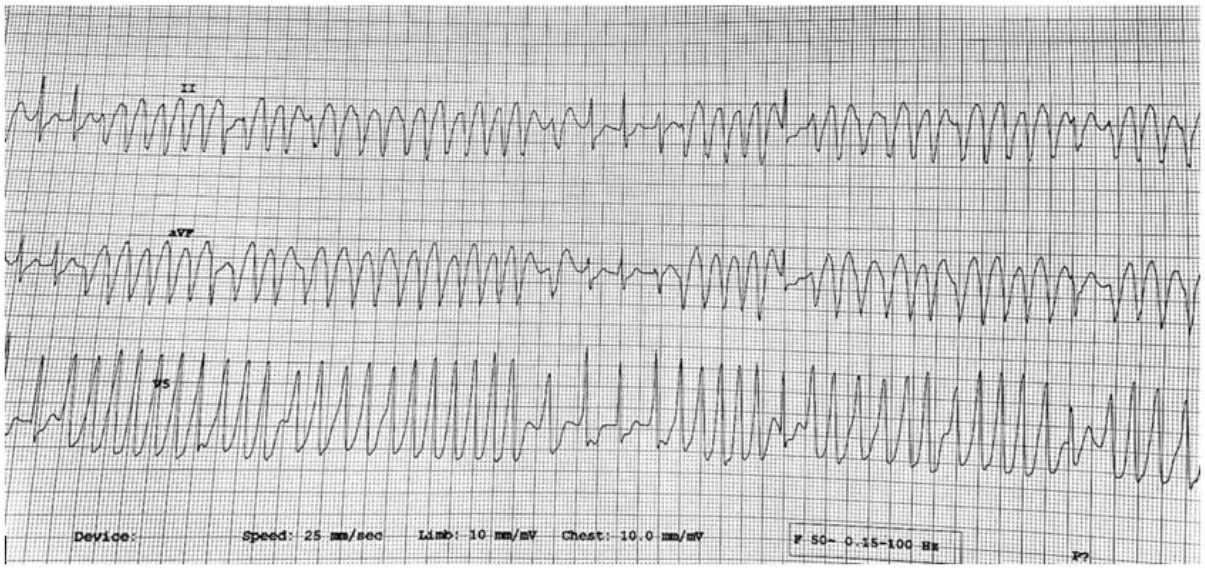

Figure 1: Broad complex irregular tachycardia consistent with pre-exited atrial fibrillation.

Following the initial assessment he was given 6mg of intravenous adenosine in order to differentiate whether this tachyarrhythmia was related to a supraventricular tachycardia with aberrant conduction or a ventricular tachycardia. On administration of the adenosine he became lightheaded, his heart rate rapidly increased to 220 bpm and his blood pressure decreased to 94/63 mmHg. The cardiac monitor showed a rapid regular broad complex tachycardia. He subsequently underwent immediate direct current (DC) cardioversion with a single biphasic synchronised shock of 200J. This successfully cardioverted the patient into sinus rhythm. His resting 12-lead ECG demonstrated a short PR interval of 100 msec, narrow QRS complexes (111 msec) and a delta wave, which was most prominent in the inferior leads (Figure 2). In view of the ECG findings he was diagnosed with Wolff-Parkinson-White (WPW) syndrome.

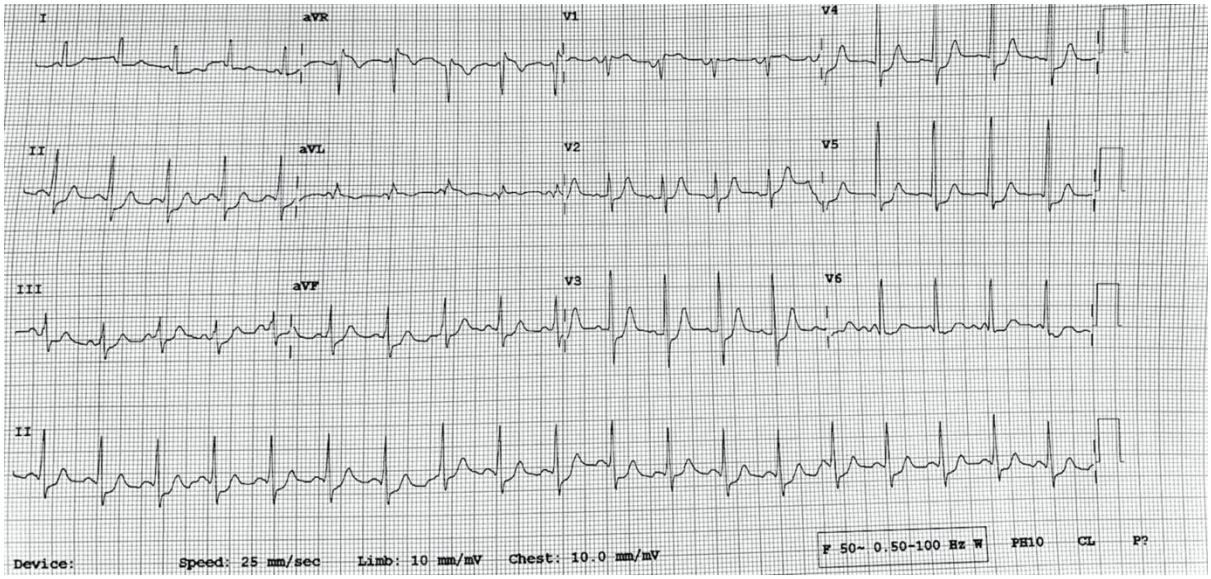

Figure 2: Sinus rhythm with a narrow PR interval, and a delta wave most prominent in the inferior leads.

His initial blood tests including a serum troponin revealed normal biochemistry and haematology. Transthoracic echocardiography demonstrated normal biventricular size and function and no valvular abnormalities.

He was admitted to the coronary care unit and the next day underwent an electrophysiology study followed by definitive treatment with an ablation of the accessory pathway. He made an uneventful recovery and was discharged 24-hours later.

Discussion

WPW is caused by an accessory pathway that communicates between the atria and ventricles known as the Bundle of Kent.[1] The accessory pathway does not share the rate limiting properties of the atrioventricular (AV) node and can conduct electrical activity at a much faster rate. If patients with WPW develop atrial fibrillation, the atrial impulses can all be conducted via the accessory pathway and result in a rapid heart rate with haemodynamic instability. Pharmacological agents that block the AV node (such as adenosine, beta-blockers and non-dihydropyridine calcium channel blockers) are contraindicated as they may exacerbate the syndrome by blocking the normal conduction pathway, and favouring 1:1 atrial to

ventricular conduction via the accessory pathway.[2] This leads to a sinister, broad complex, irregular tachycardia which can degenerate into ventricular arrhythmias. Such patients should be treated in the acute setting with DC cardioversion and then have definitive treatment with an ablation of the accessory pathway.[3]

Intravenous adenosine is a potent AV node blocker with a short half-life, and has become the first line pharmacological agent in the treatment of supraventricular tachycardias. It often causes rapid termination of arrythmias that utilise the AV node. Adenosine can also be used as a diagnostic tool when the underlying arrhythmic mechanism is unclear. Atrial arrythmias, such as atrial flutter, do not terminate on the administration of adenosine but unmasking the atrial rhythm by temporarily blocking the AV node can be diagnostically beneficial.[4]

In patients presenting with a broad complex tachycardia, adenosine administration can be used to differentiate between a supraventricular tachycardia with aberrant conduction (which would often terminate) and a ventricular tachycardia (which would not respond to adenosine). This differentiation is extremely important as it has both short and long-term implications on subsequent patient investigations, treatment and prognosis. [5-8]

However, an important caveat to consider is that if the broad complex tachycardia is indeed a pre-excited atrial fibrillation, the administration of adenosine carries the risk of precipitating 1:1 atrial to ventricular conduction, which can degenerate into life-threatening ventricular arrythmias. Pre-excited atrial fibrillation often occurs in young otherwise healthy patients, and causes a rapid, board complex, irregular tachycardia, with variable QRS morphology. In the acute setting, AV nodal blocking agents should be avoided and DC cardioversion should be utilised.

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