

Picture Quiz Answer: A young man with palpitations

A Shaukat & MC Grey

Answers

1. ECG No 1 (Figure 1) shows a broad complex irregularly irregular rhythm which is consistent with atrial fibrillation. The ventricular rate is very rapid and there is slow upstroke of R wave in the chest leads indicative of pre-excitation.
2. DC Cardioversion is a safe option. In this patient DC Cardioversion was chosen due to the worrying rapid ventricular rate and risk of degeneration into ventricular fibrillation (VF). Amiodrone in this situation can act as an atrio-ventricular (AV) node blocking agent and lead to unopposed AV conduction and risk of VF.
3. ECG No 2 (Figure 2) shows classical features of pre-excitation with short PR interval and delta wave in leads V2-V6; this is suggestive of the Wolff Parkinson White (WPW) syndrome.
4. Radiofrequency ablation of the accessory pathway is the treatment of choice

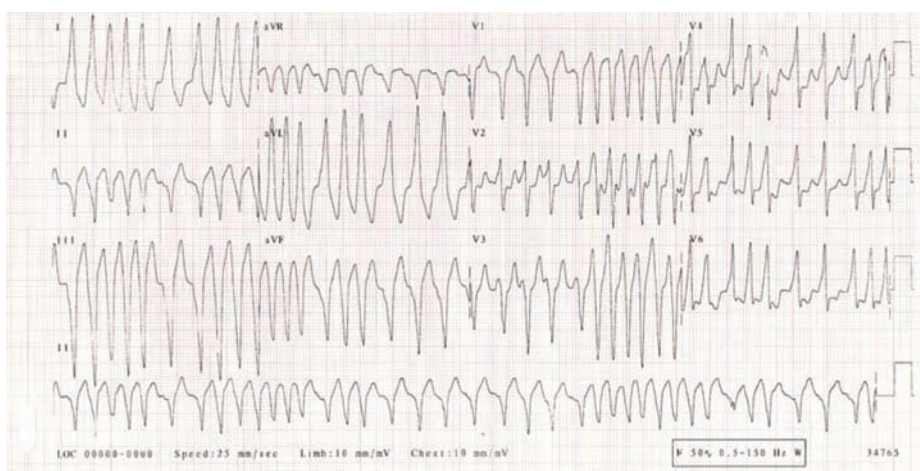


Figure 1.

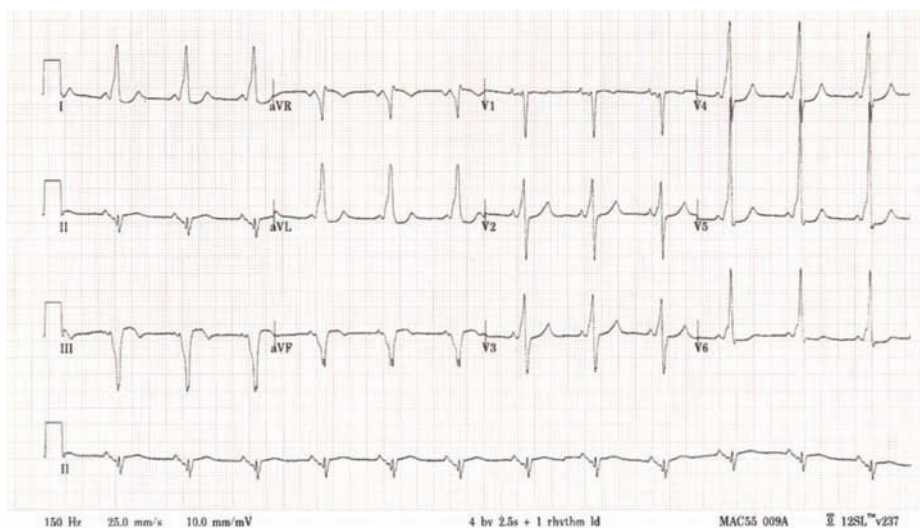


Figure 2.

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Discussion

In 1930, Wolff, Parkinson and White presented a group of eleven young patients who demonstrated an ECG with a bundle branch block pattern, short PR interval (<120ms) and paroxysms of tachycardia.¹ In 1940 the term Wolff-Parkinson-White (WPW) syndrome was coined to describe the combination of pre excitation and paroxysms of tachycardia. This syndrome which is estimated to occur in 0.1-0.3% of the population² arises from an embryological cardiac defect caused by an abnormal connection in the atrio-ventricular electrical insulation. Thus, strands of myocardium are found within the normally insulating AV annulus³ thereby forming an accessory pathway between the atria and ventricle which is termed the Bundle of Kent.

The AV node is described as decremental as it only allows a limited number of atrial impulses to pass through to the ventricle per period of time. However, the majority of accessory pathways are non decremental and have the ability to conduct both anterogradely (from atria to ventricle) and retrogradely (ventricle to atria). Consequently, the most frequent dysrhythmia in patients with WPW is an AV reciprocating tachycardia whereby a re-entry circuit is established between the atria, AV node, ventricles and accessory pathway thereby demonstrating a narrow complex tachycardia on ECG. However, in a minority of individuals, the above circuit will run in an antidromic/retrograde direction from the atria, through the accessory pathway to the ventricle and back up through the AV node to the ventricle and thereby establishing a wide complex tachycardia.² If such a retrograde circuit is established in the presence of atrial fibrillation (AF) the result will be a wide complex irregularly irregular tachycardia.

The incidence of AF in patients with WPW has been reported to be between 11.5 to 39%.⁴ However, AF is particularly dangerous in WPW, due to the non decremental properties of the accessory pathway. Lacking the refractory period of the atrioventricular node (AVN), the accessory pathway can conduct impulses to the ventricle at a rate of up to 300 / minute. Hence, the rhythm can easily degenerate into ventricular fibrillation⁵

This problem is thought to be the most common cause of death in patients with WPW, occurring at a rate of up to 0.6% per year.⁶

Due to its potential dangers, rapid termination of this arrhythmia is essential. The first step in this process involves recognition of the AF related to WPW. Clues to the diagnosis include:

- The irregularity of the rhythm
- Very rapid ventricular response (often >200 /minute)
- The wide, bizarre QRS complex morphology, which varies from beat-to-beat and may intermittently present a delta wave.
- The occasional presence of a narrow QRS complex, when conduction occurs via the AV node

When patients present with haemodynamic compromise due to their arrhythmia, DC cardioversion should be considered first line treatment, particularly when the ventricular rate is rapid, as in this case. Chemical cardioversion may be considered when patients are haemodynamically stable with a slower ventricular rate. Procainamide and amiodarone have both been proposed as therapies for cardioversion of AF associated with WPW.^{7,8} However, the slow onset of action of procainamide may be a concern for a rhythm which can potentially degenerate into VF. Furthermore, some authors have suggested that amiodarone can cause acceleration of ventricular rate, leading to degeneration into VF due to its acute AV node blocking properties.^{9,10}

Agents which prolong conduction through the AVN should be avoided as these will encourage conduction through the accessory pathway and thereby increase ventricular response to the AF. Hence, Digitalis, adenosine, calcium channel blockers and beta-blockers should not be used if WPW AF is considered a likely diagnosis.

The long term treatment of accessory pathway is with anti-arrhythmics or catheter ablation. Anti-arrhythmics are associated with cost, inconvenience and adverse effects. Radiofrequency ablation techniques provide a success rate of approximately 95% and a recurrence rate after the initial procedure in the order of only 5%.^{11,12}

References

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