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CME Article

ECG delta waves in patients with palpitation

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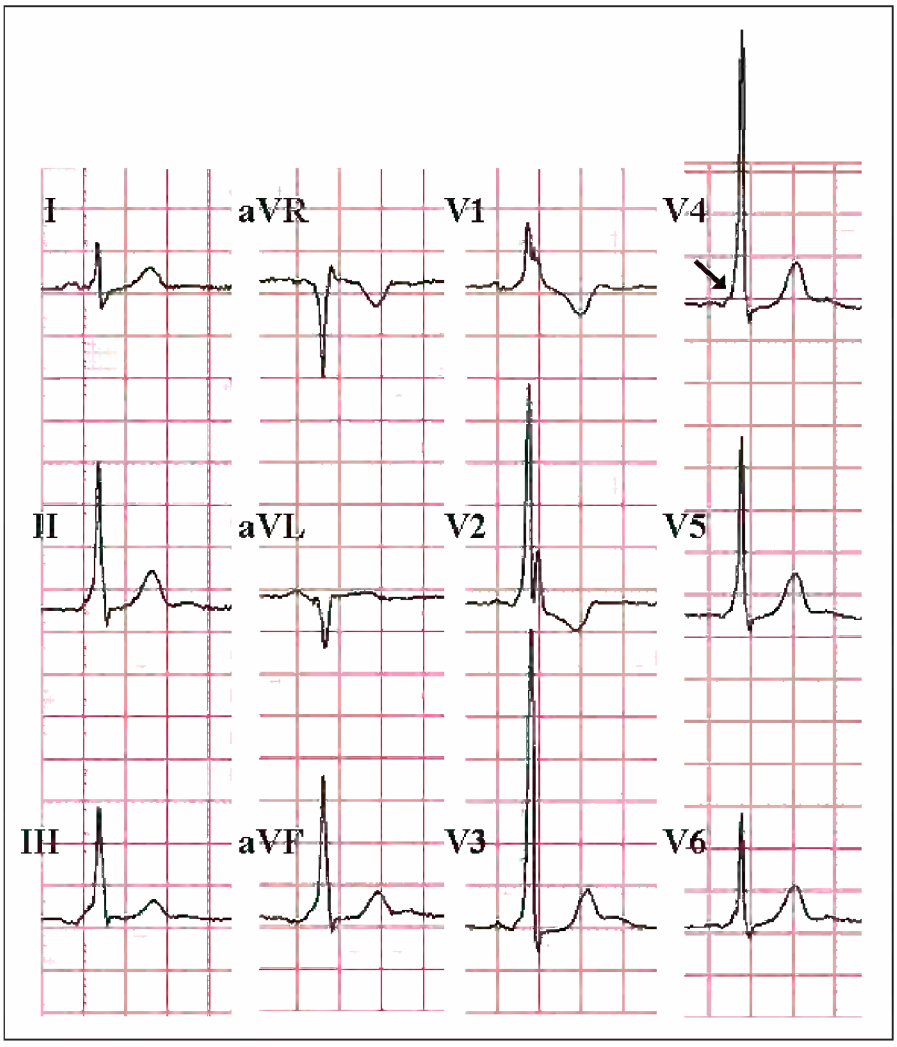

Fig. 1 ECG shows type A Wolff-Parkinson-White syndrome.

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CASE 1

CLINICAL PRESENTATION

A 37-year-old man was found to have an abnormal electrocardiogram (ECG) during health screening. He had a history of palpitation at a frequency of about once a month. Comment on the ECG shown in Fig. 1, which was recorded when the subject was not having palpitations. What are the ECG abnormalities? What is the diagnosis?

ECG INTERPRETATION

The 12-lead ECG shows widened QRS complex. Delta waves are seen in most of the leads (arrow in lead V4). The PR interval is short (80 milliseconds [ms]). The R wave in lead V1 is upright, suggesting the presence of a left-sided accessory pathway (i.e. between the left atrium and left ventricle).

CLINICAL COURSE

The patient had type A Wolff-Parkinson-White (WPW) syndrome. His palpitations were most likely due to atrioventricular reentrant tachycardia (AVRT). He underwent electrophysiology study, and a left lateral accessory pathway was confirmed. This was successfully ablated. He has not had a recurrence of palpitations since then.

CASE 2

CLINICAL PRESENTATION

A 61-year-old woman with a history of hypertension had been having intermittent palpitations for two years (Fig. 2). What are the abnormal ECG findings?

ECG INTERPRETATION

The 12-lead ECG shows widened QRS complexes, as well as deep and wide Q waves in leads V1, II, III, and aVF. The PR interval is short (80 ms) and delta waves are seen in most of the leads (arrow in lead aVL). The ECG findings are classical for type B WPW syndrome.

CLINICAL COURSE

The patient had a right-sided accessory pathway. Her symptoms of palpitations were most likely due to AVRT. She underwent a transthoracic echocardiogram, which revealed Ebstein anomaly (Fig. 3). There is apical or downward insertion of the septal leaflet of the tricuspid valve. The distance between the insertion points of the mitral and tricuspid valves to the septum was abnormally long. It was measured to be 25 mm (19 mm/m^2, indexed to body surface area). There was no left ventricular regional wall motion abnormality in the inferior wall to suggest an old myocardial infarction. She was offered electrophysiology study with a view for radiofrequency ablation of the accessory pathway.

DISCUSSION

Patients with WPW pattern in the ECG show a short PR interval (< 120 ms), a wide QRS complex (> 120 ms) and a delta wave, which is the initial slow upstroke and slurring of the R wave. Those with WPW syndrome have both

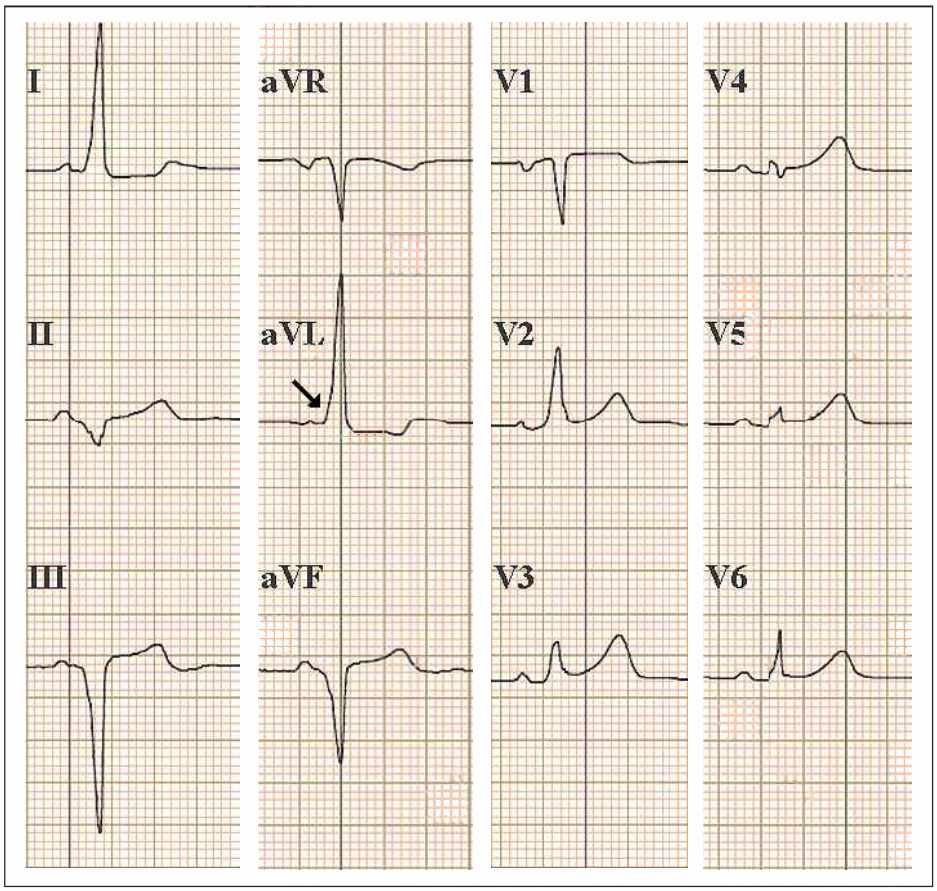

Fig. 2 ECG shows type B Wolff-Parkinson-White syndrome.

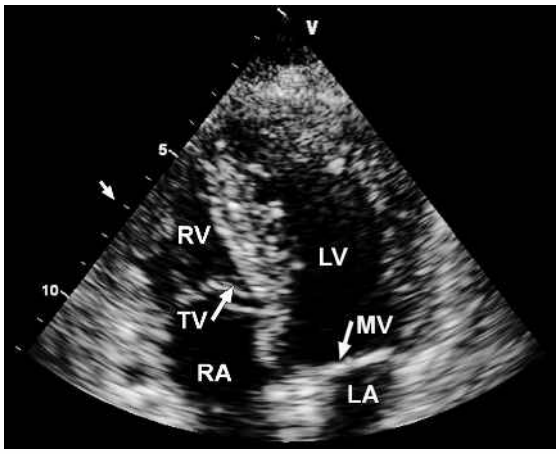

Fig. 3 Transthoracic ECG at the apical 4-chamber view shows the typical features of Ebstein anomaly. There is abnormal apical insertion of the tricuspid valve (TV) and ventricularisation of the right atrium (RA). LA: left atrium; LV: left ventricle; MV: mitral valve; RV: right ventricle. Arrows indicate the position of the atrioventricular valves (MV and TV).

the WPW ECG pattern and paroxysmal supraventricular tachyarrhythmias.[1]

The WPW ECG is a great mimicker of cardiac abnormalities. Deep and wide Q waves simulate old transmural myocardial infarction. Tall ventricular complexes simulate left and right ventricular hypertrophy. ST segment depression simulates ischaemic heart disease, and wide QRS complexes simulate bundle branch block. In WPW syndrome, clinicopathologic studies have revealed the presence of accessory pathways (APs) between the atria and ventricles, which enables the conduction of atrial impulses outside the normal conduction pathway. These microscopic strands are located along the cardiac annulus or septum. More than 50% of APs are located at the left free wall, 5%–10% at the anteroseptum, 20%–30% at the posteroseptum and 10%–20% at the right free wall.[2] In patients with an antegradely conducting AP, ventricular activation during sinus rhythm occurs simultaneously via both the AP and the atrioventricular (AV) node, resulting in a fusion complex.

APs may be classified into several types.[3] Manifest APs are those that conduct more rapidly in the antegrade direction than the AV node, resulting in a discernible delta wave on the surface ECG. Concealed APs conduct only in the retrograde direction, and no delta wave is documented in the ECG. Several algorithms have been developed to localise the site of APs from the surface ECG.[4,5] These are usually based on delta wave amplitude, sum of delta wave polarities, R/S wave ratio and QRS axis. For example, we can localise the AP insertion site in Case 1 to be most likely in the left lateral position. Although this is interesting and important, detailed intracardiac mapping is often still needed for successful pathway ablation.

AVRT is a reentrant arrhythmia and can be divided into the orthodromic and antidromic variants. During orthodromic tachycardia, the antegrade limb is the AV node-His-Purkinje system, and the retrograde conduction is the AP. As AVRT uses the normal conduction system as its antegrade limb, it results in a narrow QRS complex tachycardia. In contrast, antidromic AVRT, which is much less common, results in fully preexcited wide QRS complexes. This gives rise to a regular wide-complex tachycardia that may be mistaken for ventricular tachycardia.

Most patients with WPW syndrome have no structural heart disease. However, it may be associated with several cardiac conditions, most notably Ebstein anomaly. This is a congenital abnormality of the tricuspid valve. There is marked apical displacement of the septal leaflet insertion, resulting in downward displacement of the functional annulus. The right ventricle becomes atrialised and the true tricuspid annulus dilates. The effective right atrium (including the atrialised right ventricle) is invariably large. This becomes more so in the presence of the commonly associated significant tricuspid regurgitation. When patients with Ebstein anomaly undergo radiofrequency ablation, the success rate is lower and the recurrence rate of WPW is higher because they tend to have multiple APs.

Besides AVRT, patients can also develop atrial arrhythmias, including atrial fibrillation and atrial flutter. Ventricular fibrillation has been reported to occur in 2%–3% of symptomatic WPW patients over a long period of follow-up.[6] In some patients, sudden death may be the first manifestation of this syndrome. However, individuals who lose antegrade AP conduction over time have a lower mortality risk. Treadmill ECG exercise test has been used to assess the robustness or malignant potential of an AP's antegrade conduction.[7] The hypothesis is that the delta wave should disappear with exercise when APs have longer effective refractory periods than normal AV conduction. Therefore, during exercise, impulses will be conducted down the AV node-His-Purkinje system, resulting in narrow complex ECGs. Some studies have shown that this situation may be more benign. However, whether it is predictive of a lower long-term risk of sudden death is unclear, and studies have not been systemically conducted in a large WPW-syndrome population.

Electrophysiologic study and pathway ablation are well established in the treatment of symptomatic patients with WPW syndrome. The approach to asymptomatic patients is less clear, but based on the recommendations of the ACC/AHA/ESC guidelines, it is a class IIa indication for catheter ablation.[8]

ABSTRACT

It is important to recognise Wolff-Parkinson-White (WPW) syndrome in electrocardiograms (ECG), as it may mimic ischaemic heart disease, ventricular hypertrophy and bundle branch block. In addition, ECG can aid in the localisation of the accessory pathway. Recognising WPW syndrome allows for risk stratification, the identification of associated conditions and the institution of appropriate management.

Keywords: accessory pathway, diagnosis, early repolarisation, Ebstein anomaly, management, risk stratification, Wolff-Parkinson-White

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REFERENCES

1. Gallagher JJ, Pritchett EL, Sealy WC, Kasell J, Wallace AG. The preexcitation syndromes. Prog Cardiovasc Dis 1978; 20:285-327.
2. Wellens HJ, Atie J, Penn OC, et al. Diagnosis and treatment of patients with accessory pathways. Cardiol Clin 1990; 8:503-21.
3. Lee KW, Badhwar N, Scheinman MM. Supraventricular tachycardia – part I. Curr Probl Cardiol 2008; 33:467-546.
4. Fitzpatrick AP, Gonzales RP, Lesh MD, et al. New algorithm for the localization of accessory atrioventricular connections using a baseline electrocardiogram. J Am Coll Cardiol 1994; 23:107-16.
5. Arruda MS, McClelland JH, Wang X, et al. Development and validation of an ECG algorithm for identifying accessory pathway ablation site in Wolff-Parkinson-White syndrome. J Cardiovasc Electrophysiol 1998; 9:2-12.
6. Berkman NL, Lamb LE. The Wolff-Parkinson-White electrocardiogram. A follow-up study of five to twenty-eight years. N Engl J Med 1968; 278:492-4.
7. Gaita F, Giustetto C, Riccardi R, Mangiardi L, Brusca A. Stress and pharmacologic tests as methods to identify patients with Wolff-Parkinson-White syndrome at risk of sudden death. Am J Cardiol 1989; 64:487-90.
8. Blomstrom-Lundqvist C, Scheinman MM, Aliot EM, et al. ACC/AHA/ESC guidelines for the management of patients with supraventricular arrhythmias – executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines (Writing Committee to Develop Guidelines for the Management of Patients With Supraventricular Arrhythmias). Circulation 2003; 108:1871-909.

SINGAPORE MEDICAL COUNCIL CATEGORY 3B CME PROGRAMME
Multiple Choice Questions (Code SMJ 201102A)

	True	False
Question 1. What is the underlying cause of palpitations in the patients who are presented?		
(a) Acute myocardial infarction.	☐	☐
(b) Thyrotoxicosis.	☐	☐
(c) Lown-Ganong-Levine syndrome.	☐	☐
(d) Wolff-Parkinson-White (WPW) syndrome.	☐	☐
Question 2. The following are ECG features of WPW syndrome:		
(a) Presence of delta wave.	☐	☐
(b) Shortened PR interval of < 0.12 sec.	☐	☐
(c) PR depression.	☐	☐
(d) Prolonged QRS interval of > 0.12 sec.	☐	☐
Question 3. The following are known conditions associated with WPW syndrome:		
(a) ST elevation myocardial infarction.	☐	☐
(b) Ebstein anomaly.	☐	☐
(c) Acute pericarditis.	☐	☐
(d) Marfan's syndrome.	☐	☐
Question 4. The WPW ECG may mimic the following:		
(a) Old myocardial infarction.	☐	☐
(b) Left ventricular hypertrophy.	☐	☐
(c) Right ventricular hypertrophy.	☐	☐
(d) Bundle branch block.	☐	☐
Question 5. The following statements regarding accessory pathways (APs) are correct:		
(a) APs connect the atria with the ventricles.	☐	☐
(b) There may be no delta wave in the ECG of concealed APs.	☐	☐
(c) Electrophysiologic study and pathway ablation are established treatments for symptomatic patients with WPW syndrome.	☐	☐
(d) More than 50% of APs are located at the left free wall.	☐	☐

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SUBMISSION INSTRUCTIONS:
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RESULTS:
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Deadline for submission: (February 2011 SMJ 3B CME programme): 12 noon, 28 March 2011.