Orthodromic Atrioventricular Reentrant Tachycardia Presenting To The Komfo Anokye Teaching Hospital, Kumasi, Ghana

I Owusu, A Bockarie, Y Amoako

Citation

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Abstract

In Wolf-Parkinson-White (WPW) pattern pre-excitation, an accessory pathway known as the bundle of Kent conducts electric impulses directly from the atrial to the ventricles. Patients with WPW pre-excitation are therefore predisposed to paroxysmal supraventricular tachycardia (PSVT). The PSVT can be a narrow QRS complex tachycardia or broad QRS complex tachycardia. This presentation is on a patient with WPW pattern pre-excitation presenting as a narrow QRS complex tachycardia.

INTRODUCTION

The first publication on WPW syndrome was in 1930. Wolff, Parkinson and White reported 11 young healthy people with ECG syndrome of short PR interval, wide QRS and paroxysmal tachycardias [1]. Since that time, many studies have been done on WPW syndrome, and today we understand the pathogenesis and the mechanisms of the arrhythmias associated with it [2,3,4].

Patients with WPW syndrome have accessory pathway that conducts electrical impulses directly from the atrial to the ventricles. Ventricular activation results from both early activation (pre-excitation) of the ventricle and from normal activation. The degree of unopposed pre-excitation depends upon the time required to conduct through the right and left atria, the accessory pathway and the ventricular myocardium as compared to conduction through the normal pathways. This results in a QRS complex that is a fusion of ventricular pre-excitation and normal excitation, with a shorter PR interval, a small delta wave, and some prolongation of the QRS duration. Patients with WPW pattern pre-excitation are predisposed to recurrent atrioventricular reentrant tachycardia (AVRT).

HISTORY

A 53 year old woman, a widow with 3 children and a known hypertensive for 5 years, presented to the medical emergency unit, Komfo Anokye Teaching Hospital, Kumasi on 26th April 2008 with a history of recurrent palpitations and dizziness for two years. On the day of admission, she developed palpitations which progressively increased in severity.

PHYSICAL EXAMINATION

On physical examination, she looked ill, sweating, not dyspnoeic at rest and she had no pedal oedema. Her respiratory rate was 24 breaths/minute; the radial pulse was very rapid and thready. The systolic blood pressure was 60mmHg by palpation and the diastolic blood pressure was not recordable. The jugular venous pressure was not raised and the apex beat was at the 5 th left intercostals line in the mid-clavicular line. The heart sounds were normal and no murmurs were heard. The chest, abdomen and the central nervous system were also normal.

INVESTIGATIONS

An urgent 12-lead ECG was done (figure 1). It showed a heart rate of 222/minute, regular sinus rhythm, QRS complex duration of 61ms and repolarization abnormalities. Blood was taken for full blood count, blood urea nitrogen, serum creatinine, fasting blood glucose and serum lipid profile. Holter and echocardiography were also done; the results of these investigations are shown on table 1.

Figure 1

Figure 1: 12-Lead ECG Of The Patient On Admission

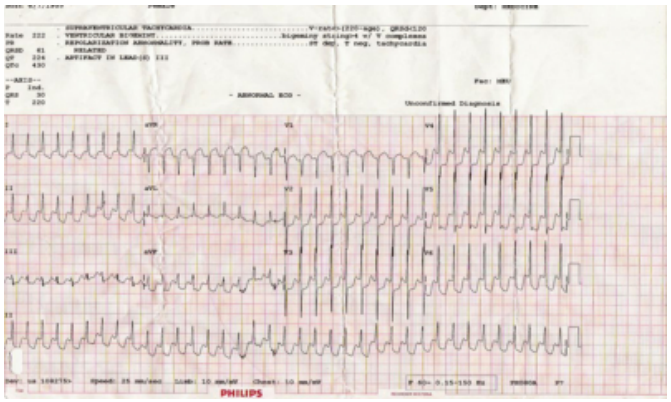


Figure 2

Table 1: Laboratory Investigation Of The Patient

HOLTER REPORT

- Minimum heart rate occurred at 10.00 PM while patient is asleep = 44/min
- Maximum heart rate = 90/min
- Average heart rate = 58/min
- Maximum sinus pause=1776ms
- VES=2/24hrs
- SVES=4/24hrs
- QRS complex showed WPW pre-excitation throughout recordings

ECHOCARDIOGRAPHIC REPORT

Aortic Root: 2.8 cm	Right Atrium: Normal size
Left Atrium: 3.0 cm	Right Ventricle: Normal size
IVS: 1.0 cm	FS: 45 %
LVPW: 1.1 cm	EF: 74 %
LVIDd: 4.0 cm	LV mass index:
LVIDs: 2.2 cm	TAPSE: 2.4 cm MVAD: 1.6 cm

Normal 2 dimensional and Doppler echocardiographic findings

HAEMATOLOGY AND BIOCHEMISTRY REPORT

- Heamogloin= 13.4 g/dL, white blood cells= 5.37x 10^9/L, platelets= 269x10^9/L
- Urea= 3.5mmol/L, creatinine=86.4 umol/L, sodium=140mmol/L, potassium=3.8mmol/L, fasting blood glucose=4.5mmol/L, total cholesterol= 3.8 mmol/L, LDL cholesterol=1.6 mmol/L, HDL cholesterol=1.5mmol/L, Triglycerides=1.7mmol/L

DIAGNOSIS AND MANAGEMENT

A diagnosis of narrow QRS complex tachycardia with haemodynamic instability was made. Carotid sinus massage was done but the tachycardia did not stop. Adenosine and a defibrillator were not readily available on the ward and therefore the patient was put on oral verapamil 120 mg immediately. Figure 2 shows the ECG of the patient some hours after the first dose of the verapamil. She had cardioverted to sinus rhythm, and the heart rate was now 69/minute. QRS complex duration was 71ms.

Figure 3 shows another ECG which was done two days after admission. This ECG showed a heart rate of 79/minute, sinus rhythm, and WPW pattern pre-excitation. The patient was discharged after 72 hours of admission on verapamil 120 mg twice daily orally, to be followed-up at the cardiac clinic.

Figure 3

Figure 2: 12-Lead ECG Of The Patient Some Hours After The First Dose Of Verapamil

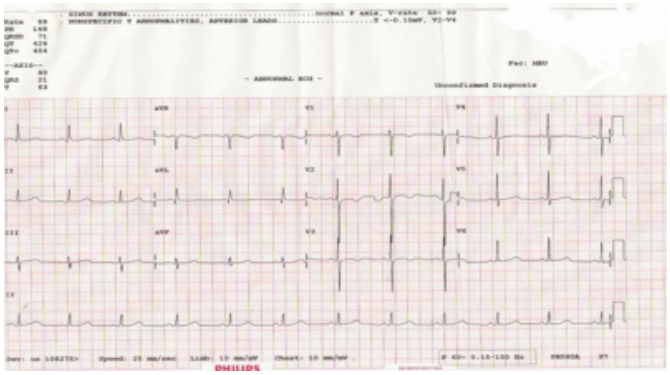
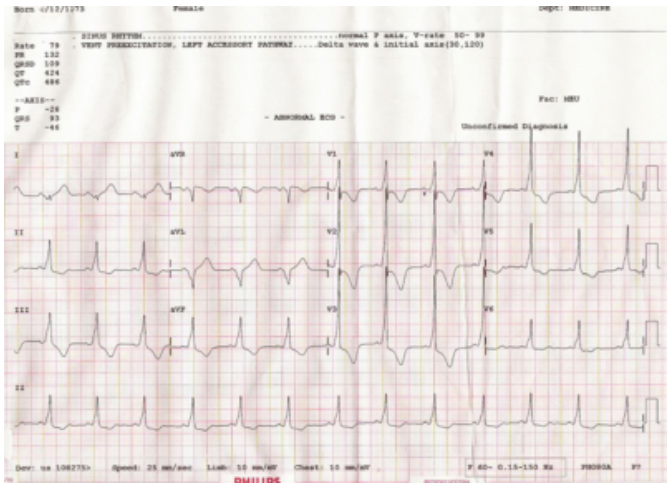


Figure 4

Figure 3: 12-Lead ECG Done Three Days After Admission



DISCUSSION

Tachycardias associated with the WPW syndrome and the accessory pathways can be classified into those in which the accessory pathway is necessary for initiation and maintenance of the tachycardia eg, atrioventricular reentrant tachycardia (AVRT)) and those in which the bypass tract acts as a “bystander”, providing a route of conduction from the anatomic site of tachycardia origin to other regions of the heart eg, atrial fibrillation and atrial flutter [5] .

There are two major forms of AVRT, orthodromic and antidromic, which can be differentiated in part by the findings on the ECG. In orthodromic AVRT, the QRS

complex is narrow. Antegrade coduction occurs through the atrioventricular (AV) node/His-Purkinje system; in this setting, the delta wave seen during sinus rhythm is lost since antegrade conduction is not via the accessory pathway. Orthodromic AVRT comprises 95 percent of the reentrant tachycardias associated with the WPW syndrome [6] .

In antidromic AVRT, the tachycardia has a wide QRS complex as the antegrade limb is usually the accessory pathway. Antidromic AVRT occurs in only 5 to 10 percent of patients WPW syndrome [7,8,9] . Antidromic AVRT can result in considerable diagnostic and therapeutic uncertainty because the differential diagnosis includes all other wide QRS tachycardias, such as ventricular tachycardia, and supraventricular tachycardia conducted aberrantly.

Our patient presented with orthodromic AVRT because during the tachycardia the ECG showed narrow QRS complex tachycardia but when she cardioverted to sinus rhythm after the oral verapamil was initiated, the ECG initiallly showed a narrow complex QRS (figure 2), and then WPW pattern pre-excitation (figure 3). The verapamil initially blocked both the antegrade and retrograde conduction leading to sinus rhythm with narrow complex QRS and the loss of the delta wave. Later, conduction through the accessory pathway was recovered and therefore delta wave appeared in the patient's ECG.

In conclusion, arrhythmias are very common in Africa and physicians should be encouraged to do ECG more often when necessary, especially in patients presenting with cardiogenic shock.

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CORRESPONDENCE TO

DR ISAAC KOFI OWUSU, FWACP DEPARTMENT OF MEDICINE KOMFO ANOKYE TEACHING HOSPITAL P. O. BOX 1934 KUMASI, GHANA E-MAIL: ikeowusu@yahoo.com

References

1. Wolff L, Parkinson J, White PD. Bundle branch block with short P-R interval in healthy young people prone to paroxysmal tachycardia. Am Heart J 1930;5:685.
2. Liebman, J, Zeno, JA, Olshansky, B, et al. Electrocardiographic body surface potential mapping in the Wolff-Parkinson-White syndrome. Noninvasive determination of the ventricular insertion sites of accessory atrioventricular connections. Circulation 1991; 83:886.
3. Benson, WD, Sterba, R, Gallagher, JJ, et al. Localization of the site of ventricular pre-excitation with body surface maps in patients with Wolff-Parkinson-White syndrome. Circulation 1982; 65:1259.
4. Gallagher, JJ, Pritchett, ELC, Sealy, WC, et al. The preexcitation syndromes. Prog Cardiovasc Dis 1978; 20:285.)
5. Yee, R, Klein, GJ, Sharma, AD, et al. Tachycardia associated with accessory atrioventricular pathways. In: Cardiac Electrophysiology, Zipes, DP, Jalife, J (Eds), WB Saunders, Philadelphia 1990. p.463.
6. Josephson, ME. Preexcitation Syndromes. Clinical Cardiac Electrophysiology. Lea & Febiger, Philadelphia 1993. p.311.
7. Yee, R, Klein, GJ, Sharma, AD, et al. Tachycardia associated with accessory atrioventricular pathways. In: Cardiac Electrophysiology, Zipes, DP, Jalife, J (Eds), WB Saunders, Philadelphia 1990. p.463
8. Bardy, GH, Packer, DL, German, LD, et al. Preexcited reciprocating tachycardia in patients with Wolff-Parkinson-White syndrome: Incidence and mechanisms. Circulation 1984; 70:377.
9. Kuck, KH, Brugada, P, Wellens, HJ. Observations on the antidromic type of circus movement tachycardia in the Wolff-Parkinson-White syndrome. J Am Coll Cardiol 1983; 2:1003.)

Author Information

Isaac Kofi Owusu, FWACP
Department of Medicine, Komfo Anokye Teaching Hospital

Ansumana Bockarie, PhD
Department of Medicine, Komfo Anokye Teaching Hospital

Yaw Ampem Amoako, MB, ChB
Department of Medicine, Komfo Anokye Teaching Hospital