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Choice of Management in a Rare Case of Symptomatic Wolff-Parkinson-White (WPW) Syndrome Type B

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Abstract

Introduction: Wolff-Parkinson-White (WPW) syndrome type B is a congenital condition involving abnormal electrical conduction between the atria and the ventricles that provide an accessory pathway (AP) for a re-entrant tachycardia circuit. Background: This case report illustrates a 28-year-old male who presented with multiple episodes of rapid, regular palpitations associated with dizziness and nausea. These episodes of palpitations often resolve spontaneously. Physical examination revealed normal first and second heart sounds with no audible murmurs. Other systemic examinations were unremarkable. A 12 lead electrocardiogram showed an atrioventricular re-entrant tachycardia (AVRT) of 210 beats per minute. Intravenous amiodarone was given to which he responded. Post pharmacological cardioversion, the repeated ECG showed shortened PR interval and broad QRS complexes associated with negative delta waves in lead V1, T-wave inversion in inferior leads and precordial leads of V5-V6 which represented a Type B pattern. He was then referred to the electrophysiology unit at the National Heart Institute for radiofrequency ablation (RFA) and Holter monitoring. Conclusion: Wolff-Parkinson-White (WPW) Type B should be considered as a differential diagnosis for a young individual who presents with frequent palpitations. RFA is well known as a permanent solution to the prevention of tachyarrhythmia.

Keywords

Wolff-Parkinson-White Type B, Accessory Pathway, Amiodarone, Palpitation, Radiofrequency Ablation

Subject Areas: **Cardiology**

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1. Introduction

Wolff-Parkinson-White (WPW) syndrome represents one of many conditions with paroxysms of tachyarrhythmias that belong to a unifying pathophysiology of pre-excitation [1]. Specifically, there is a congenital embryological formation of accessory conductive pathway that connects the atrium and ventricle that bypasses the atrioventricular node. Hence, the presence of two atrioventricular connections and differing refractory periods between them can eventually lead to unique supraventricular tachycardia called an atrioventricular re-entrant tachycardia (AVRT) [1]-[3]. Estimates of WPW syndrome prevalence range from 0.068% to 0.174%, with WPW syndrome being twice as common in those with coronary heart disease [4] [5]. WPW syndrome is divided into type A and Type B with unknown measures of prevalence in the population. However it is thought that Type A is more common as left sided accessory pathways (APs) are much more common than right APs as visualized by electrophysiological studies. All WPW syndromes have a small risk of sudden cardiac death with an incidence of 1 to 1000 patient-years in those with presence of ventricular pre-excitation [3]. There are also two other pre-excitation syndromes, namely Lown-Ganong-Levine syndrome and Mahaim types [1] [2].

2. Case Presentation

A 28-year-old Malay gentleman presented with multiple episodes of palpitations for the past one year in which he described the palpitations as rapid and regular. The palpitation was associated with dizziness, nausea and dyspnoea. He denied any syncope or chest pain. There were no symptoms of heart failure. He had no family history of sudden cardiac death (SCD) or palpitations. He denied any use of over the counter, herbal, traditional, stimulant and recreational drug use. He almost never consumed any caffeinated products. He is a non-smoker and a teetotaler. He works at a textile factory. Physical examination revealed an alert male who was clinically pink with no jaundice. He had no obvious goiter. He was afebrile with a blood pressure of 139/76 mmhg. The heart rate was 210 beats per minute regularly regular. Oxygen saturation was 100% on room air. Bedside capillary glucose was 5.2 mmol/L. The cardiovascular examination revealed first and second heart sounds with no audible murmurs. Jugular venous pulsations were not elevated and the apex beat was not displaced. Lungs were clear on auscultation. Other systemic examinations were unremarkable. There was no lower limb edema. The 12 lead electrocardiogram showed atrioventricular re-entrant tachycardia (AVRT) of 210 beats per minute. The complete blood count, renal profile, liver function test, electrolytes and thyroid function test are as shown in **Table 1**. The patient was given intravenous amiodarone 150 mg which was diluted in 100 ml of Dextrose 5% solution over 15 minutes followed by 360 mg of amiodarone over 8 hours and then 540 mg over 16 hours. The

Table 1. Blood parameters of the patient.

Parameters (units)	Values (normal range)
Haemoglobin (g/dL)	13.8 (13 - 18)
White cell count (10^9 L)	9.5 (4 - 11)
Platelet (10^9 L)	253 (150 - 450)
Albumin (g/L)	40 (35 - 55)
AST (U/L)	30 (10 - 40)
ALT (U/L)	25 (10 - 40)
Creatinine kinase (U/L)	153 (52 - 336)
Sodium (mmol/l)	140 (136 - 145)
Potassium (mmol/l)	4 (3.5 - 5)
Serum Creatinine (μmol/l)	78 (60 - 110)
Calcium (mmol/l)	2.5 (2.2 - 2.6)
Magnesium (mmol/l)	0.9 (0.7 - 1.1)
Phosphate (mmol/l)	0.9 (0.8 - 1.5)

12 lead electrocardiogram post pharmacological cardioversion showed shortened PR interval, slurred downstroke of the QRS complex in V1, broad QRS complex with T-wave inversion in V5, V6 and inferior leads as shown in **Figure 1**. A 2-dimensional transthoracic echocardiogram showed no structural abnormality with good left ventricular ejection fraction of 65% and no left ventricular hypertrophy. Chest radiograph showed clear lung fields with a normal cardiac size. A diagnosis of WPW syndrome type B was made. Patient was then referred for RFA and Holter monitoring at the National Heart Institute.

3. Discussion

Wolff-Parkinson-White (WPW) is a syndrome that presents with paroxysms of tachycardia that have a distinctive pathological feature, namely pre-excitation [1]-[3]. It requires 1) an alternate pathway of conduction that connects the atrium and ventricle (Bundle of Kent) and 2) differing electrical characteristics of these two pathways that predispose it to a re-entrant tachyarrhythmia [1]. Current electrophysiological evidence supports the theory advanced first by de Boer in 1926 that describes a circus-like movement in electrical conductance of the heart consisting of an impulse from the atrium to atrioventricular junction followed by the His-bundle towards the ventricle and finally back up to the atrium via AV anomalous conduction (later known as an Orthodromic AVRT) [6]. Often, when this occurs, patients may present to the hospital with bouts of palpitations with the characteristic resemblance of a supraventricular tachycardia (SVT) on a standard 12-lead electrocardiogram. Symptoms are variable with differing severity related to the meaningful cardiac output such as palpitations, syncope, chest pain and dyspnea [1] [3]. When the AVRT ceases, the patient would return to the premorbid state of health before the next attack occurs. These pathways usually exhibit separate conductive properties specifically in their refractory periods and/or speeds [1] [3]. In this particular scenario, the properties of the AP in WPW syndrome are as follows [1]:

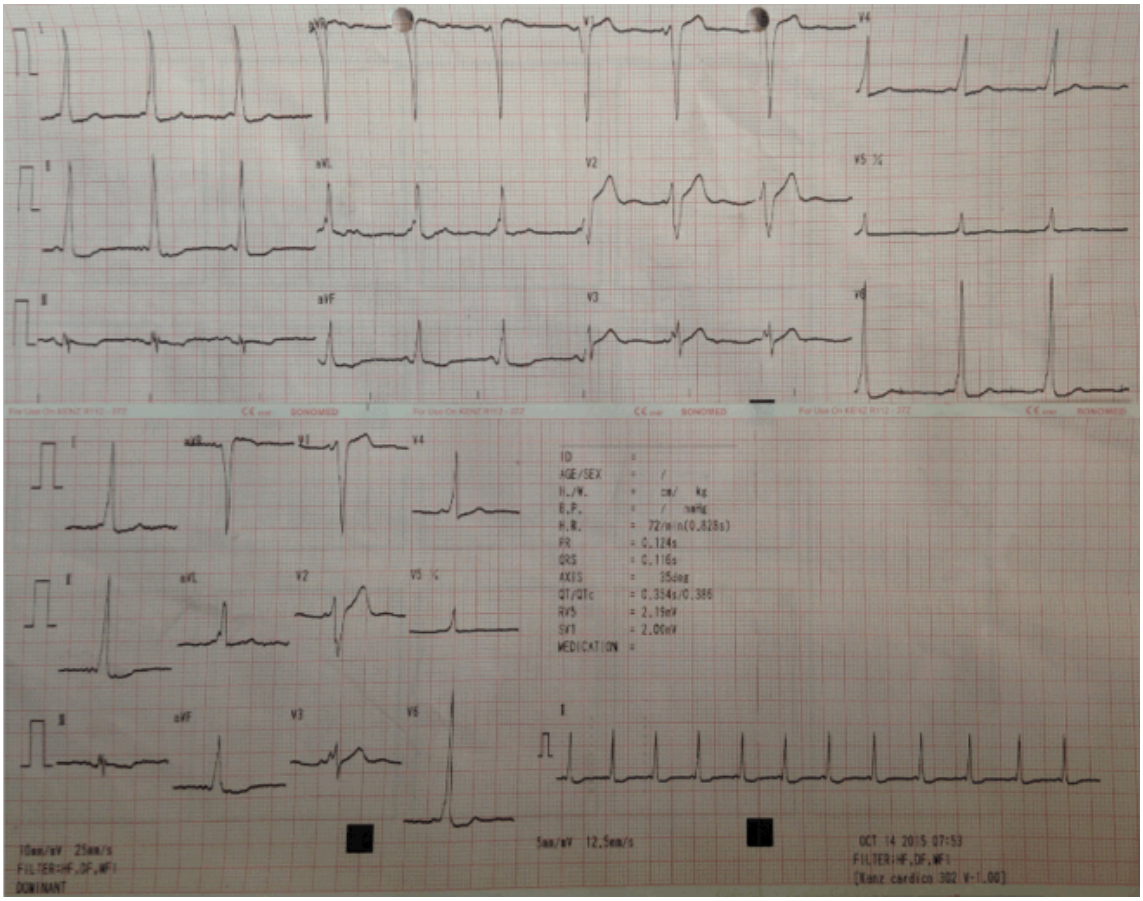

Figure 1. 12 lead electrocardiogram of patient.

1) Faster conduction speeds than the AV-His bundle (as the AV node has a normal delay to allow ventricular filling.
2) Longer refractory period than the AV-His bundle

This leads to the faster AP being blocked due to its own extended refractory time allowing the impulse in the AV-His bundle pathway to continue retrograde up the AP, predisposing a re-entrant tachycardia [1]. Re-entrant type tachycardia can occur either due to APs or even presence of ischemic tissue [1]. A firm grasp of the basis of AVRT underscores the ultimate aim of medical cardioversion in which slowing conduction speeds and prolonging refractory periods in the AP, to allow more AV conductance is the desired outcome [3]. Following this revelation, AVRTs can be precipitated by any AV nodal blocking drugs such as Adenosine, Verapamil, or Digitalis and many well-revised guidelines have recommended avoiding these classes of drugs instead suggesting Class 1a, 1c and 3 anti-arrhythmic medications in its place [2]. The overwhelming majority of ECG patterns in sinus rhythm follow the type A pattern with a smaller incidence of type B patterns. The ECG findings between type A and type B are as tabulated in **Table 2** [2]. These different types merely signify the site of the AP with Type A patterns occurring due to left sided APs and Type B patterns occurring due to right-sided APs [7]. This finding has been reinforced with evidence from various sources such as when Durrer and Roos were the very first ones to locate a right free wall AP via intraoperative mapping and cooling [7]. Since the advent of radiofrequency ablation as a suitable method to successfully treat this condition, algorithms have been developed to accurately predict APs in ECGs for multiple reasons; to reduce morbidity, shorten procedure time, selection of the ablator with appropriate curves and to minimize mechanical trauma [8] [9]. One of the first known attempts was in an article written in 1987 by Milsten and Sharma *et al*. published an algorithm by attempting to identify four APs in 141 patients, namely right free wall, anteroseptal, posteroseptal and left free wall using the morphology of delta wave [8]. Following that, Chiang and Chen et al in 1995 began to embark on the same endpoint by including the R/S ratio of the QRS complex along with the Delta wave morphology in 369 patients with only anterograde aberrant pathways. They did this by first examining 182 patients' pre-ablation ECGs and electrophysiology findings, noting the polarity of delta waves of the earliest pre-excitations among the precordial leads. They then went on to develop a relatively simple 4-step process that required examining 4 leads: V1, V2, Lead III and aVF. They discovered that their algorithm was able to predict the location of APs with an overall accuracy of 93% when they used the algorithm to predict their remaining 187 patients as compared to 86%, 85% and 85% respectively [9]. In 2014, a paper by Taguchi and Yoshida et al recognized that using the delta wave morphology analysis method can often be difficult with many limitations such as the need for maximal pre-excitation. They proposed amendments to just use the R/S ratio to accurately predict the location of these APs. Using 142 patients (88 post ablative ECGs of patients to construct algorithm and 54 patients for prospective testing of developed algorithm) they were able to just use only the R/S ratios with assessments of V1, V2 and aVF to correctly identify the correct aberrant pathway 51 out of 54 patients. This translated to an overall sensitivity of 94% and specificity of 98% [10]. Since its introduction in 1987, the utilization of RFA has now been recognized as a treatment of choice for WPW with tachyarrhythmias due to its relative safety and efficacy, as well as for the primary rationale of preventing the occurrence of sudden cardiac deaths [4] [11]. Electrophysiology studies, which involve the placement of electrode catheters within the cardiac chambers, enable inter-cardiac signals to be recorded in real-time and are coupled with RFA procedures to map out the APs that need to be ablated [12]. The overall success rate of RFA is approximately 93%, of which ablation of left free wall APs have a success rate of 95%, followed by right free wall (90%) and posteroseptal APs (88%) [4]. This is in part due to the difficulty

Table 2. ECG findings of WPW type A and Type B.

Type A findings	Type B Findings
Short PR interval (<120 ms) waves in sinus rhythm	Short PR interval (<120 ms) waves in sinus rhythm
Presence of Broad QRS complex accompanied with slurred upstroke R component which could be a possible RBBB mimicker (Delta wave)	Presence of Broad QRS complex accompanied with slurred downstroke S component which could a possible LBBB mimicker (Delta wave)
Dominant R wave In V1 (Left sided AP)	Dominant S wave in V1 (right sided AP)
Tall R waves accompanied with inverted T waves in leads V1 until V3 (mimicker of RVH)	Tall R waves accompanied with inverted T waves in the inferior leads and V4 till V6 (mimicker of LVH)

in ablating the latter two APs, which is also reflected in the high proportion of second ablation procedures required. In addition, high volume ablation centers also concur with relatively higher rates of success [13]. A recent epidemiological study in Taiwan suggests that the implementation of RFA in its health system has contributed to a lower prevalence rate of WPW compared to prior population studies, with an overall prevalence of (0.36/1000) and a peak prevalence rate of 0.61/1000 [4]. Current debate revolves around the appropriate clinical approach to asymptomatic WPW patients and the need to strike a balancing act between taking necessary measures to prevent sudden cardiac death while avoiding the potential complications brought about by the invasive nature of RFA. In an observational study spanning 8 years by Pappone *et al*. on 2169 patients, it was found that prophylactic RFA was able to reduce the frequency of arrhythmias in patients diagnosed with WPW syndrome [14].

4. Conclusion

WPW type B should always be considered as an important differential diagnosis in a young male who presents with frequent palpitations. It causes for sudden cardiac death. Hence a high index of suspicion coupled with a good clinical workup and management may help prevent morbidity and mortality associated with this disorder. RFA remains the mainstay modality of treatment with regards to a permanent cessation of its associated tachyarrhythmias.

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Informed Consent

Informed consent was obtained from the patient for the publication of this study.

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