

Supraventricular tachycardia from a left bundle branch block-An unusual presentation of Wolff-Parkinson-White syndrome

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Vol. 13, No. 1 (2009-01 - 2009-12)

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Key Words: Supraventricular tachycardia, Wolff-Parkinson-White syndrome, left bundle branch block

Accepted September 9 2008

Curr Pediatr Res 2009; 13(1 & 2): 1-3

Abstract

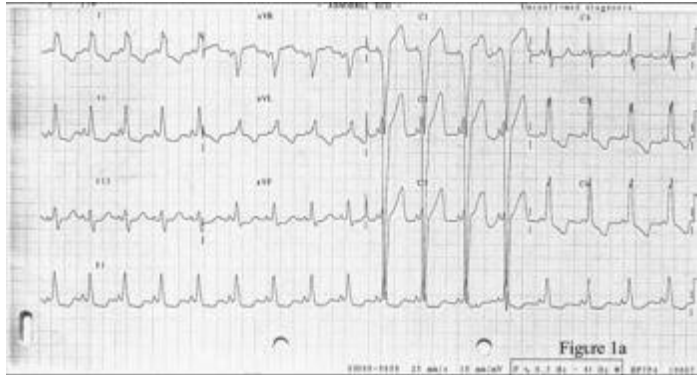
Wolff-Parkinson-White syndrome (WPW) is an electrocardiographic diagnosis based on a short PR interval with the presence of a delta wave, but it can mimic a variety of electrocardiographic conditions. We present a 6 year old Saudi female whose WPW was easily mistaken as a left bundle branch block.

Wolff-Parkinson-White syndrome, or as frequently abbreviated as WPW, accounts for about 20% of all supraventricular tachycardias (SVTs).[1] With an estimated incidence of 0.3%, it is the commonest of the atrioventricular re-entry tachycardias (AVRTs).[2] In the majority of cases, WPW presents as orthodromic AVRTs (i.e. The conduction of impulses travels through the AV node to the ventricles before being redirected back to the atria through the accessory pathway thus completing a loop, inverse of antidromic AVRT).

In about 20% of all cases of WPW, delta waves and PR shortening are not evident on the usual electrocardiogram (EKG), and are referred to as concealed pathways, as they allow SVTs to occur but are not evident outside of those times. Accessory pathways may also present intermittently or in cyclic fashion (concertina) on the EKG i.e. delta waves and short PR intervals may be seen after every few beats and then this cycle is repeated. Classically, WPW are easily recognized because the accessory pathway conducts first and through slowly conducting myocardium before the conducted pulse through the atrioventricular (AV) node and bundle of His overtakes the impulse and depolarizes the rest of the myocardium producing a delta wave prior to the narrow QRS complex. Depending on the size of the accessory pathway and the number of these tracts and their sites, the EKG has been described as representing right ventricular hypertrophy, posterior myocardial infarction, right bundle branch block (type A), or left ventricular hypertrophy, anterior myocardial infarction, or Left bundle branch block (type B).[3] If the size of the accessory tract depolarizes a large portion of the ventricle at its location, then there will be a short PR interval (<120 ms) followed by a wide QRS complex, and can mimic a bundle branch block. Preexcitation syndromes in children are less common compared to adults.[4] as are EKG patterns that fulfil the criteria of a left bundle branch block. We present a case report of a 5-year-old Saudi girl who suffered from intermittent episodic asthma, recurrent chest tightness, shortness of breath, and palpitations.

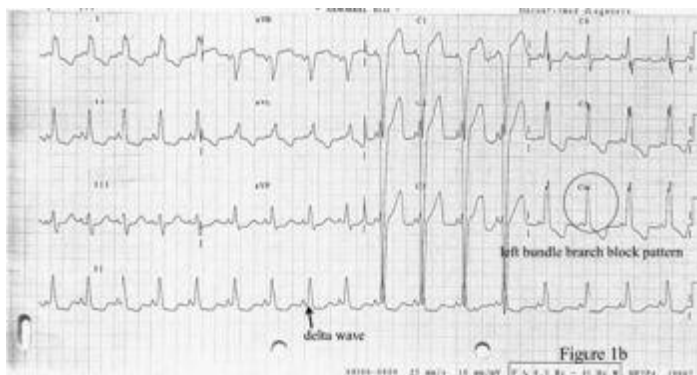
Case report

A 5-year-old Saudi girl who suffers from intermittent episodic asthma was first admitted 6 months previously with an acute exacerbation of her asthma and received 2.5mg of nebulised salbutamol. She developed SVT that was observed on the cardiac monitor in the emergency room. This was aborted via vagal manoeuvres, and an EKG (Fig. 1) was obtained the next day. She was discharged "well", and provided with salbutamol and budesonide metered dose inhalers.



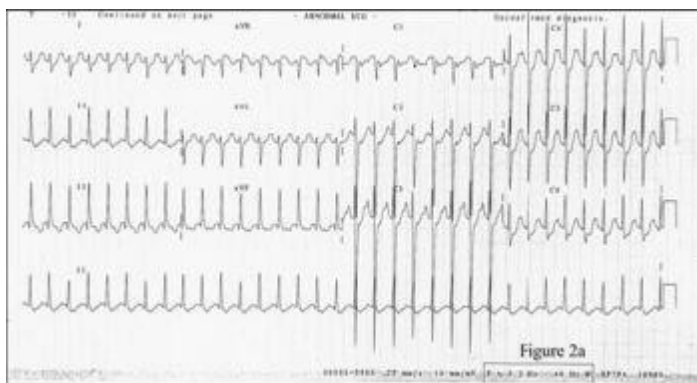
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Figure 1a. Electrocardiogram demonstrating a left bundle branch block in a 5 year old girl.



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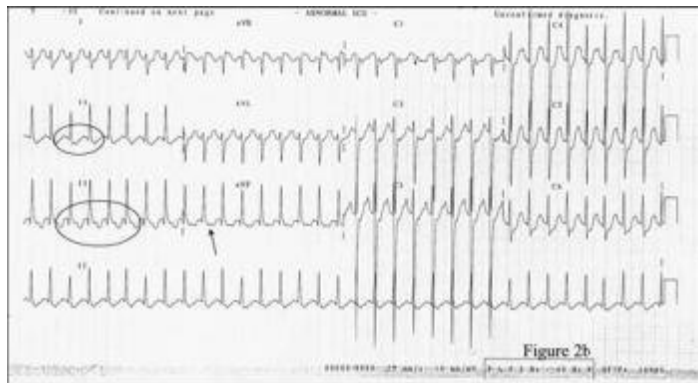
Figure 1b. Delta wave and LBBB are highlighted from figure 1a by circles, fulfilling part of the criteria for Wolff-Parkinson-White syndrome



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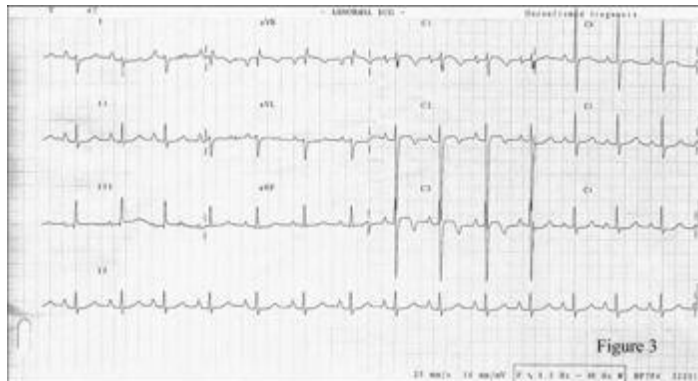
Figure 2a. Supraventricular tachycardia in the same patient.

Six months later, she suffered another episode of chest tightness associated with wheezing. She was again given nebulized salbutamol and developed SVT with a heart rate of about 250 bpm. This time vagal maneuvers were unsuccessful and adenosine was employed in increasing doses starting at 100mcg/kg till 300mcg/kg/dose was reached in 5 attempts that were unsuccessful, this was not captured during the emergency. IV amiodarone infusion was then started and an EKG (Fig. 2) was obtained after half an hour of admission to our pediatric intensive care unit.



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Figure 2b. The figure is the same as figure 2a, but with the circles illustrating the inverted p waves seen after the QRS complexes in leads II and III. The arrow points to the appearance of P wave after the QRS complex in aVF.



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Figure 3. Electrocardiogram obtained 11 days after amiodarone conversion from the same patient showing disappearance of the LBBB

Figure 3 was obtained 7 days after being discharged on oral amiodarone, and she is currently awaiting electrophysiology testing and radiofrequency ablation at a neighboring centre as we do not have the facility nor expertise at our centre.

Discussion

Features of Wolff-Parkinson-White syndrome that can and had been mistaken for bundle branch blocks, and was overlooked as the possible cause for this child's supraventricular tachycardia is shown in Figure.1a and b. The cardinal features are short PR interval, presence of delta waves and a wideQRS complex that resembled a left BBB (see Figure 1b). Figure 2a and b also demonstrate a feature more typical of orthodromic atrioventricular

reentrant tachycardia (AVRT); regular inverted P waves are seen after the narrow QRS complexes (see Figure 2b). Figure 3 shows prolongation of the PR interval and abolition of the delta wave after 11 days of treatment on amiodarone.

Symptomatic presentations of WPW are uncommon in childhood, and more so in a young child who may not be able to complain about the symptoms appropriately [4]. In this case, the addition of salbutamol, a β adrenergic agonist may have facilitated the appearance of SVT.

Many algorithms have been designed to locate the accessory pathway (AP) in WPW, with Milstein, et al and Lindsay, et al among the better known.[1,5]. These 2 different algorithms are 90% accurate in accessing the site of the AP on the basis of polarity of the delta wave in the ordinary 12 lead ECG. This child demonstrates a left BBB, with isoelectric delta waves in leads III, aVF and a negative delta wave in aVR, with R>S waves from V4 – V6, and probably represents multiple tracts occurring in the right lateral free wall (negative aVR), and right anteroseptal planes, although electrophysiological testing would be required to determine this.

WPW is not a benign condition with instances of sudden death in individuals of WPW in both those symptomatic and asymptomatic. There is still often debate on the best treatment for cases of asymptomatic WPW syndrome as the risk of sudden death in such cases are low [5]. As alluded to in the case history, a trial of vagal maneuvers may be used to abort the SVT, before pharmacological measures are employed.

Adenosine is usually the drug of first choice. It can safely induce an atrioventricular block. However, adenosine has the ability to cause an atrial arrhythmia and reinduce the SVT or atrial fibrillation with a rapid ventricular response rate [2].

In this case, we elected to use loading doses of IV amiodarone to terminate the tachycardia. Many adult centers tend not to use IV amiodarone as it can result in an antidromic conduction through the accessory tract, because IV amiodarone has its fastest and greatest effect at the AV node, and causes QT prolongation, hence allowing for the same effect as digoxin and verapamil, inducing atrial arrhythmias and possible antidromic AVRT, with the risk of rapid ventricular rates and ventricular fibrillation. In many cases, a slow bolus infusion over a period of 30 mins often is sufficient to control the heart rate and terminate the tachycardia in a well-observed setting, such as the intensive care unit. Oral therapy can also be started simultaneously while waiting for the oral drug to slowly take its effect. The use of this drug would also require the need for frequent monitoring because it has effects on the thyroid gland (both hyper and hypothyroidism) and lens of the eye, which are frequently reversible with the stoppage of this drug. We did not opt for sotalol or the β -blocking agents in this girl because of her asthma, nor did we choose procainamide, again because of its proarrhythmic properties – with instances of ventricular fibrillation [6].

There have been important observations of childhood dilated cardiomyopathy in WPW, traditionally attributed to chronic and prolonged tachycardia, but more recent case reports suggest dilated cardiomyopathy may occur despite the control of tachycardia, perhaps via dyskinetic segments [7]. Hence our decision to send the child for electrophysiology testing and radiofrequency ablation despite a normally appearing echocardiogram for this child.

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