Loss of Delta Waves on Exercise in an Asymptomatic Male with Wolff-Parkinson-White Syndrome

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Abstract
During a routine examination, healthy, asymptomatic patients may present with conditions such as tachycardia. Appropriate diagnosis of Wolff- Parkinson-White (WPW) syndrome, is important in determining risk for future arrhythmias or other complications. A 25-year-old, physically active, asymptomatic, African American male was detected with WPW during a pre-employment medical examination. His history was noncontributory. His electrocardiograph showed delta waves and short PR interval with occasional ventricular ectopies. Echocardiography showed normal valves, ventricular size and function. His exercise testing (Bruce Protocol) showed normal blood pressure response. Delta waves disappeared during exercise and reappeared during recovery. No arrhythmias were noted. However, the inability to induce an arrhythmia during electrophysiological testing does not guarantee lack of arrhythmia inducibility. Exercise test has high negative predictive value possibly eliminating the need for invasive electrophysiological testing. Delta waves disappearing during the exercise testing placed our patient at low risk; no further testing was advised. He remained asymptomatic on follow-up.

Keywords — arrhythmia, electrocardiogram, ventricular pre-excitation, Wolff-Parkinson-White syndrome.


I. INTRODUCTION
Wolff-Parkinson-White (WPW) syndrome is the most common form of ventricular pre-excitation with arrhythmia. Its symptoms often present as tachycardia or vasovagal syncope and it is often associated with other heart structure abnormalities including valve flow from the right atrium. Treatment may include medicines such as adenosine, antiarrhythmic drugs or amiodarone to control tachycardia, cardioversion, surgery or catheter ablations. In our case, the patient was symptomatic.

II. CASE PRESENTATION
A 25-year-old, physically active, African American male was detected to have WPW syndrome during the course of a routine pre-employment medical examination. He had no symptoms and his history was unremarkable. His family history was noncontributory. Finding of WPW on the electrocardiograph (ECG) prompted a referral to a cardiologist. His physical examination was unremarkable. ECG showed delta waves and short PR interval consistent with WPW with occasional ventricular ectopies (Figure 1). Echocardiography showed normal valves and normal ventricular size and function.

He underwent exercise testing using the Bruce Protocol. He exercised for 17.01 minutes achieving an estimated workload of 18 mets. He had a normal blood pressure response and was asymptomatic throughout the exercise and recovery phases. Delta waves disappeared during exercise (Figure 2). The delta waves reappeared during the recovery period. No arrhythmias were noted.

Figure 1. Baseline ECG showing WPW pattern and PVC.
III. DISCUSSION

WPW syndrome is the most common form of ventricular pre-excitation. In 1930, Wolff, Parkinson, and White reported paroxysms of tachycardia or atrial fibrillation in young patients who had a functional bundle branch block and an abnormally short PR interval on electrocardiograms recorded during sinus rhythm. In 1933, Holzmann and Scherf, and Wolferth and Wood reported that the mechanism in WPW consisted of an acceleration of passage of the impulse from the atria to the ventricles and not a block, as had been proposed by Wolff, Parkinson, and White. In 1944, Ohnell introduced the term "pre-excitation" to the medical literature, and along with Wood and his colleagues, confirmed the presence of accessory pathways by histologic studies. Subsequently, several different types of accessory pathways were described.

The prevalence of WPW has been estimated as 0.1 to 3.1/1000. The incidence of the familial WPW syndrome among patients with electrophysiologically proven accessory pathways is 34 per 1000 persons and the prevalence of pre-excitation in first-degree relatives of these patients is 5.5 per 1000 persons. The proposed mechanism of inheritance in a minority of cases is autosomal dominant; however, WPW can be inherited as part of a syndrome with extensive cardiac involvement, in which case it can be polygenic in inheritance. It is estimated that 7% to 20% of patients with WPW syndrome have other accompanying congenital abnormalities; the most common is the Ebstein anomaly. Other conditions that have been associated with this syndrome include hypertrophic obstructive cardiomyopathy, mitral valve prolapse, atrial septal defect, ventricular septal defect, transposition of the great arteries, coarctation of the aorta, dextrocardia, coronary sinus diverticula, and left atrial aneurysms, cardiac rhabdomyomas (as seen in patients with tuberous sclerosis), Marfan's syndrome.

Symptoms occur in 50% of patients and most often as tachyarrhythmias. The most common arrhythmia is atrioventricular reentrant tachycardias (70 to 80%), followed by atrial fibrillation (12% - 39%), and atrial flutter (<5%). It rarely causes sudden cardiac death (0.018%) with possible warning factors being younger age (<30 years), male gender, associated congenital or other heart disease, and familial WPW. Sudden cardiac death is rarely the first manifestation of WPW syndrome. Recently Pappone et al reported a higher incidence of serious arrhythmias (than in previous studies) in asymptomatic patients with WPW with incidences of ventricular fibrillation, sudden death and life threatening syncopal arrhythmias at 0.3%, 0.1% and 1.3% per year respectively.

Physicians are often faced with otherwise healthy and asymptomatic patients who are detected to have WPW during the course of routine examination. The question is “How should the patient be managed?”

The mechanism for sudden cardiac death in WPW is the onset of atrioventricular reentry tachycardia that degenerates into atrial fibrillation followed by ventricular fibrillation. The likelihood of an asymptomatic WPW patient having sustained atrioventricular reentry tachycardia in the future is much greater if the accessory pathway can also conduct retrogradely. In asymptomatic WPW patients, no atrioventricular reentry tachycardia or noninducibility identifies subjects at very low risk for development of subsequent spontaneous arrhythmias. Assessment of the future ventricular fibrillation risk in an asymptomatic patient with WPW is not easy.

There are two modalities available for the risk stratification: Electrophysiological studies (EPS) (transesophageal and transvenous) and exercise stress testing. During EPS, the shortest pre-excited R–R interval <250ms during inducible AFib strongly correlates to risk of sudden death. Because of the very low incidence of sudden cardiac death in asymptomatic WPW patients, current guidelines do not support routine electrophysiological testing in these patients. Exceptions to these recommendations are asymptomatic patients with a family history of sudden cardiac death, asymptomatic patients in special professions (such as airline pilots) and competitive athletes. The argument in favor of electrophysiological testing of asymptomatic WPW patients is to identify patients at risk of subsequent ventricular fibrillation, which can be fatal. However, the inability to induce an arrhythmia during electrophysiological testing does not guarantee that an arrhythmia will not be inducible later in life.

Exercise stress testing is simple and noninvasive. Markers such as sudden and complete loss of pre-excitation, loss of accessory pathway conduction on exercise stress testing, and loss of accessory pathway conduction after treatment with antiarrhythmic drugs are generally associated with a benign prognosis. Sudden disappearance of the delta wave and normalization of the QRS complex on exercise suggest long effective refractory period and thus a low risk for sudden cardiac death. Exercise test has high negative predictive value that could potentially eliminate the need for invasive risk stratification in a significant proportion of patients.

The disappearance of the delta wave is an interesting observation that has been reported by several investigators. The sudden versus gradual disappearance appears to help with identification of risk for sudden cardiac death, secondary to rapid tachyarrhythmias. The sudden disappearance of the delta wave signifies a complete conduction block in the accessory pathway and a long anterograde effective refractory period. This has been contrasted with gradual or incomplete disappearance of the delta wave.
wave, which is secondary to a slow but gradual increase in conduction through the AV node as the sympathetic tone increases.

Short accessory pathway-refractory periods lead to the fastest heart rates during atrial fibrillation, which can in turn lead to ventricular fibrillation and sudden death. This concept studied in small populations showed that the longest accessory pathway-refractory periods were noted in the patients who demonstrated the sudden disappearance of the delta waves. This was associated with low risk of ventricular fibrillation as longer refractory periods lead to slower heart rates. The negative predictive value of disappearance of delta waves may be used in risk stratification especially if done prior to invasive testing in order to minimize the latter.

IV. CONCLUSIONS

Diagnostic electrophysiology studies usually are associated with minor and non-life-threatening complications; while radiofrequency ablation is associated with serious complications such as stroke, cardiac tamponade, urgent cardiac surgery, and death. Considering the risk of radiofrequency ablation, this procedure is not warranted in low-risk asymptomatic patients. The patient had a normal heart rate and blood pressure response with no arrhythmias during exercise or recovery. The disappearance of delta waves during the exercise placed him at low risk for sudden cardiac death. Therefore, no further testing was advised. The patient has remained asymptomatic on follow-up visits since the initial diagnosis of WPW syndrome was made. This case also points to the utility of exercise testing in the WPW population for risk stratification.

V. REFERENCES