Important notes about Wolf-Parkinson-White syndrome.

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Abstract

Wolff-Parkinson-White (WPW) syndrome is a potentially life-threatening cardiac conduction disorder. Arrhythmias caused by Wolff-Parkinson-White syndrome account for 20% of all supraventricular tachycardias occurring in the general population. Clinical manifestations range from asymptomatic to sudden cardiac arrest. The risk of sudden death is ever present in his WPW syndrome and is the driving force behind the evaluation and management of this syndrome. Current diagnostic methods can accurately identify patients with WPW syndrome but lack the sensitivity to predict sudden cardiac death. This article provides an overview of the history of WPW syndrome and its general features, diagnostic criteria, treatments, and implications for care.

Keywords: Wolff-Parkinson-White syndrome, Atrial fibrillation, Anxiety, Panic attacks, Sudden death.

Introduction

Wolff-Parkinson-White (WPW) syndrome is a relatively common arrhythmia, affecting about 1-3 per 1000 people. Mutations in PRKAG2 have been reported in rare patients associated with cardiomyopathy. However, the genetic basis of her WPW in individuals with structurally normal hearts is still poorly understood. Sudden death from Atrial Fibrillation (AF) can also occur in these individuals. Several studies have shown that despite ablation of the alternative pathway, patient's risk of atrial fibrillation remains elevated compared to the general population [1].

WPW ECG patterns are caused by abnormal electrical conduction through alternative pathways that bypass the heart's normal conduction system. This alternative pathway allows cardiac electrical activity to bypass conduction delays in the atrioventricular node and reach the ventricles early, causing premature ventricular depolarization. This preexcitation also bypasses the rapidly conducting His-Purkinje system and evokes a short PR-interval ECG pattern with a 'flickering' onset of the QRS complex known as delta, called a wave, early but slowly. Result in progressive ventricular depolarization. The rest of the normal QRS erases this delta wave as normal cardiac conduction catches up after rapid conduction by the atrioventricular node delay and His-Purkinje system [2].

Wolff-Parkinson-White syndrome is the most common form of ventricular preexcitation. This is a cardiac conduction disorder caused by accessory conduction causing tachyarrhythmia. The definition of Wolff-Parkinson-White syndrome is based on the following ECG features: a PR interval less than 0.12s, an ambiguous initial segment of the QRS complex known as the delta wave, a total QRS complex spread greater than 0.12s, and a secondary repolarization change appearing in the ST-reflecting segmental T-wave change, is generally opposed (discordant) with large changes in delta waves and QRS complexes [3].

The Wolff-Parkinson-White pattern refers to electrocardiographic manifestations in sinus rhythm in which the paraventricular pathway shortens the PR interval and causes masking of the QRS rise. It may be asymptomatic or associated with orthodromic reciprocal tachycardia. Rarely, however, even in children, it has been associated with sudden death from ventricular fibrillation due to the rapid response of the alternative pathway to atrial fibrillation, itself thought to be due to orthodromic reciprocal tachycardia. . In the past, patients were at risk of sudden death due to the presence of symptoms and minimal pre-excitation RR intervals during induced atrial fibrillation. It is marked as 250ms. Because of the relatively high prevalence of the asymptomatic Wolff-Parkinson-White pattern and the availability of catheter ablation, there was a need to identify risk in asymptomatic patients. Recent guidelines recommend invasive testing for patients whose preexcitation does not clearly resolve during exercise testing. This strategy only has a high negative predictive value. The accuracy of this approach is under further investigation, especially in the light of other considerations. Patients with intermittent preexcitation previously thought to be at minimal risk may no longer be at risk, suggesting the role of isoproterenol in risk assessment [4].

WPW syndrome is the most common form of ventricular preexcitation. The ventricular myocardium is activated earlier than expected by an auxiliary pathway that allows direct electrical communication between the atria and ventricles.

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Although many patients remain asymptomatic throughout their lives, about half of those with WPW syndrome have symptoms secondary to tachyarrhythmia, such as: B. Paroxysmal supraventricular tachycardia, atrial fibrillation, atrial flutter and rarely ventricular fibrillation and sudden cardiac death. Symptoms include palpitations, dizziness, fainting, and difficulty breathing. Diagnosis is usually made by ECG findings, but more tests may be needed to confirm the diagnosis [5].

Conclusion

Wolf-Parkinson-White syndrome is a rare but dangerous condition. A high degree of clinical suspicion and close attention to symptoms are critical for diagnosis. Once the diagnosis or sufficient concern has been established, further evaluation and an interprofessional approach to treatment are required. This approach coupled with education and shared decision-making with patients and their families will guide treatment planning. It is often difficult to design and conduct well-structured and rigorous studies on rare diseases. Wolff-Parkinson-White syndrome is no exception, with most of the evidence coming from case series and population studies. Shown to be low. Although ablation is the most definitive treatment in high-risk patients, future further studies will clarify medical management and ablation thresholds for some low-risk patients.

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