e-ISSN: 2582-5208
International Research Journal of Modernization in Engineering Technology and Science
(Peer-Reviewed, Open Access, Fully Refereed International Journal)
Volume:05/Issue:02/February-2023 Impact Factor- 6.752 www.irjmets.com

WOLFF-PARKINSON-WHITE (WPW) SYNDROME IN MAN, WOMAN AND OLD PATIENTS AND THE SIGNIFICANCE OF THE ABLATION IN TREATMENT

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ABSTRACT

Wolff-Parkinson-White syndrome is a type of rare congenital heart disease. Although the role of genes in the development of WPW is not fully understood, chromosome 7 has been linked to a rare autosomal dominant disorder known as familial Wolff-Parkinson-White syndrome. The individual who is suffering from this disease can't participate in athletic activities. Because of the short PR intervals, the ventricle pumps before it is completely filled, resulting in ventricle contraction and decreased cardiac output. If we observe the ECG of the patient, the "q" wave is absent and a new wave appears in the patient called the "delta wave." The patient associated with WPW will be complaining of palpitations, dizziness, vertigo, and syncopal attacks. There is a family history of sudden cardiac death in siblings. The drug of choice for this disease is oral flecainide to prevent future episodes. In emergency conditions, intravenously administered procainamide should be given. Radio frequency ablation is the treatment of choice for WPW syndrome, in which we use lasers to destroy the accessory pathway (Bundle of Kent).

I. INTRODUCTION

It was discovered in 1930 by Dr. Harold Wolff, Dr. Paul Dudley, & Sir John Parkinson. The name Wolff Parkinson white syndrome (WPW) was given for this reason only. WPW is when there is a activation of the ventricular muscles, whose impulse originates form the atrium 3 Otherwise known as per-excitation syndrome, WPW is characterized with episodes of tachycardia (supra ventricular tachycardia in 80% of cases).Someone with WPW may start having these episodes at anytime, infancy or adulthood. In females they can start in pregnancy.

The Wolff-Parkinson-White (WPW) syndrome was first described in 1930 in a landmark article in the American Heart Journal. In this article, the authors describe a case study of 11 otherwise healthy patients with electrocardiogram (ECG) findings of a short PR interval and "bundle branch block" morphology who also suffered from paroxysmal supra ventricular tachycardia (SVT). Wolff-Parkinson-White (WPW) syndrome is a type of heart problem present at birth in which the ventricles get contracted earlier than normal because of an extra conducting pathway (the bundle of Kent). The syndrome that arises from abnormal cardiac electrical conduction can result in symptomatic and life-threatening arrhythmia.

II. METHODOLOGY

The heart is normally composed of two electrically insulated units, the atria and the ventricles. These units are connected by a conduction system that allows for normal cardiac synchrony and function. The cardiac electrical potential originates from the sino-atrial node of the right atrium and propagates through the atria to the atrioventricular (AV) node. The action potential is delayed in the AV node and is then quickly transmitted through the bundle of His and Purkinje fibers to the ventricular myocytes, allowing for rapid ventricular depolarization and synchronized contraction. The person who is suffering from WPW syndrome has a

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supplement pathway (the "bundle of Kent") that violates the normal electrical conduction between the atria and ventricles, which can allow electrical impulses to bypass the AV node and cause pre-excitation of the ventricles. The ECG of the individual shows that WPW patterns are caused by the fusion of ventricular preexcitation through the extra conducting pathway and normal electrical conduction. Most people with WPW do not develop arrhythmia and remain asymptomatic. Some secondary pathways do not exhibit the typical ECG functions described, and as a result, some patients may develop tachycardia without prior ECG evidence of the pathway's presence. This is called a hidden bypass.

III. CAUSES

Most cases of WPW syndrome occur randomly in the general population for no apparent reason (sporadically) and are not inherited. Some cases of WPW syndrome are inherited and can be transmitted in an autosomal dominant manner. Genetic diseases are determined by two genes inherited from the father and the mother. Dominant genetic disorders occur when only one copy of the abnormal gene is needed to cause the disease. The abnormal gene may be inherited from one of the parents or be the result of a new mutation (gene change) in an affected individual. The risk of passing the abnormal gene from an affected parent to the offspring is 50% for each pregnancy, regardless of the sex of the fetus. There is no proper mutation in the gene that has been identified in individuals associated with WPW syndrome, and the role of the gene in the development of the syndrome is not yet fully understood. About 7–20% of people with WPW syndrome have a congenital heart defect, such as Epstein's anomaly, a condition in which the tricuspid valve is deformed. Symptoms of WPW syndrome are due to the presence of alternative electrical pathways. A normal heart has a single pathway (His bundle) that carries electrical impulses from the small chambers of the heart (the atria) to the larger chambers (the ventricles). These electrical impulses contract and relax the muscles in the atria and the ventricles, pumping blood throughout the body. The person who is suffering from WPW syndrome has an accessory conducting pathway (Bundle of Kent) that sends extra electrical impulses from the SA node to those of the ventricles. This accessory pathway bypasses the normal route and causes the arrhythmia of normal heartbeats and generates irregularities, usually exceptionally rapid heartbeats known as "atrial flutter, atrial fibrillation, or paroxysmal SVT."

IV. DIAGNOSIS

The diagnosis of WPW syndrome is based upon a clinical evaluation, such as a detailed patient family history (are there any of the patient's siblings with a history of sudden death?) and a variety of specialized tests like an electrocardiogram (ECG), a Holter monitor, and an electrophysiology study. During electrophysiological studies, a thin tube (catheter) is inserted into a blood vessel and guided into the heart to measure electrical activity. Each of these tests can detect the abnormal heartbeat associated with WPW syndrome. Some patients associated with WPW syndrome may be clinically asymptomatic, meaning they have no symptoms associated with the disorder, including no abnormal findings on various heart tests.

V. TREATMENT

For the treatment of the WPW syndrome, we observe the patient and use various medications, as well as a surgical procedure known as catheter (radio frequency) ablation. The therapeutic procedures and interventions may vary, depending upon many factors, such as the kind of arrhythmia present, the type and severity of associated symptoms, the risk of cardiac arrest, an individual's age and general health, the individual's sex, and/or other factors. For the use of particular interventions, the decision should be made by the doctor and the members of the health care team in consultation with the patient, based upon the patient's proper disease conditions, a thorough discussion of the potential benefits and risks, patient preference, and other appropriate factors.

Regular follow-up visits are required to monitor heart function. A variety of drugs may be used to control episodes of arrhythmia in some people with WPW syndrome. The drugs for controlling the arrhythmia include adenosine, procainamide, sotalol, flecainide, ibutylide, and amiodarone. Some medicines, such as verapamil, may increase the risk of ventricular fibrillation and should be used with caution. The cardiac drug digoxin is contraindicated in adults with WPW syndrome. However, it is sometimes used as a prophylactic treatment in children with WPW syndrome who do not have a ventricular pregnancy.

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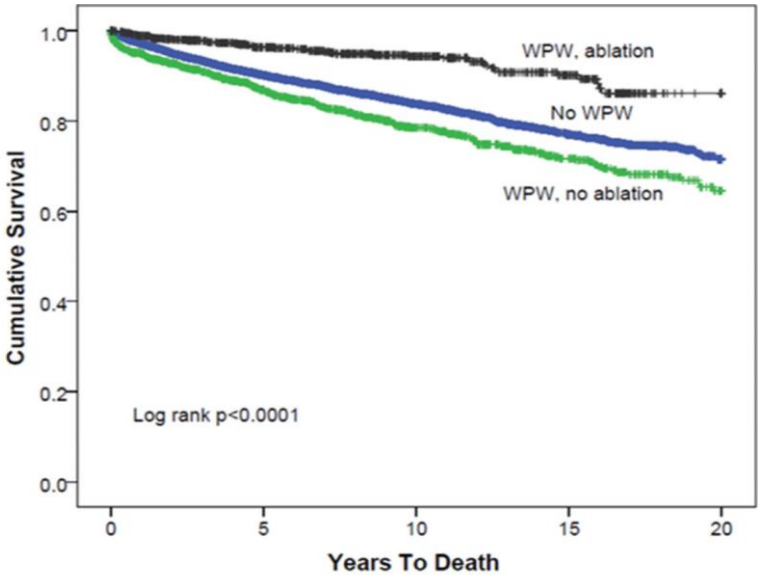
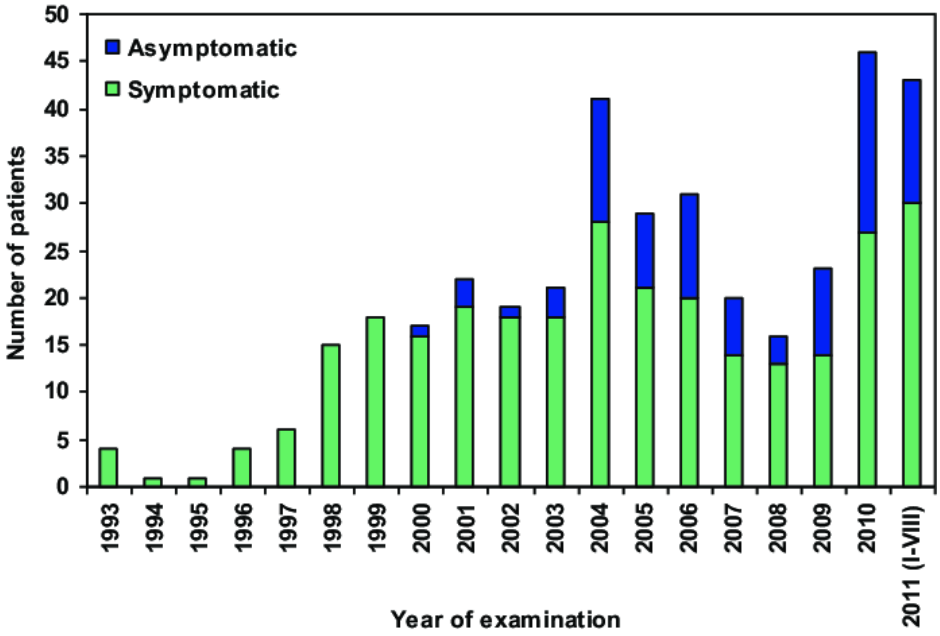


Proportion surviving at each timepoint

No WPW	100%	88.9%	82.6%	76.0%	71.6%
WPW, no ablation	100%	84.7%	77.4%	69.9%	63.5%
WPW, ablation	100%	95.5%	93.7%	87.3%	85.9%



VI. CONCLUSION

It is a type of heart problem in which pre-excitation of the ventricle occurs. The electrical signal is conducted via the accessory pathway (bundle of Kent) instead of the normal pathway (SAN to AVN). About 7–20% of people with WPW syndrome have a congenital heart defect, such as Epstein's anomaly, a condition in which the tricuspid valve is deformed. There is a shorter PR interval in the ECG of this patient. Most of the patients with this disease are asymptomatic; only the patient's ECG is relevant to the diagnosis of this disease. Generally, after physical activity in a normal, healthy person, the PR interval decreases, but in the person associated with WPW, there is no change in the PR interval after physical activity. Medication may not control an abnormal heartbeat in some cases or may cause the patient to become intolerant to the medication. In these cases, a surgical procedure known as catheter ablation may be used. This procedure may also be used for individuals at high risk of cardiac arrest and sudden death, including some asymptomatic individuals. During catheter ablation, a small, thin tube (catheter) is inserted into the heart and guided through an abnormal path that uses high-frequency electrical energy to destroy (ablate) the tissue that forms the abnormal path. This form of treatment is very successful and can reduce the need for many people to take medications. In the past, open heart surgery

was used to treat patients with WPW syndrome. Open heart surgery is rarely performed in people with WPW syndrome because of the success of catheter (radio frequency) ablation, a less invasive procedure.

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