

Post-traumatic Wolff-Parkinson-White syndrome description and case presentation

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Abstract: The ventricular pre-excitation syndrome (Wolff-Parkinson-White syndrome) is defined as a premature activation of the entire ventricular myocardium by a super-ventricular impulse. The premature activation is made by accessory conduction pathways that produce a short circuit in the normal nodo-hisian pathway. The accessory pathways are made of atrial myocardial fibers that have a higher conduction velocity and a refractory period shorter than the nodo-hisian structures. The accessory bundle can be single or multiple; it refers to congenital structures that can be dormant and they are often activated under the action of different agents. We present the case of a male aged 34 who was the victim of an aggression being hit with fists and feet. In this case, the Wolff-Parkinson-White syndrome has appeared as a sudden atrial fibrillation and left ventricular failure that appeared immediately after a traumatism of the face and legs. In this case the patient suffered from severe symptoms due to the appearance of the Wolff-Parkinson-White and required immediate hospitalization, medical treatment and ablation of the Paladino-Kent bundle.

Key Words: WPW syndrome, accessory pathways, traumatism

Ventricular pre-excitation is defined as the premature activation of the entire ventricular myocardium by a super-ventricular impulse. The premature activation is made by accessory conduction pathways that produce a short circuit of the normal nodo-hisian pathway [1]. In a normal heart, the atria are electrically isolated from the ventricles through a series of fibrous atrioventricular rings. The accessory pathways are made of atrial myocardial fibers which have a higher conduction velocity and a refractory period shorter than the nodo-hisian structures. The accessory bundle can be single or multiple; it refers to congenital structures that can be dormant and they are often activated under the action of different agents [2].

This method of activation directly connects the atria and the ventricles passing over the atrioventricular node. As a consequence, the sinus impulse can move on the normal pathway through the atrioventricular node and also faster on the accessory pathway; this fact allows the impulse conducted on the accessory pathway to reach the ventricle faster initiating its contraction [3].

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The presence of two conduction pathways (the normal one and the accessory one) between the atria and the ventricles creates the risk of producing a “short-circuit” of the normal electrical pathway leading to tachycardia. There are two mechanisms of producing the tachycardia in Wolff-Parkinson-White (WPW) syndrome: atrial fibrillation and atrioventricular reentrant tachycardia.

In atrial fibrillation, the atria have a low frequency of contractions of about 350-600/minute. Usually, the atrioventricular node blocks those impulses so that the ventricular frequency (perceived as pulse) is usually up to a maximum of 170/minute. In the case of WPW syndrome, the impulse conduction is faster and it also passes also through the accessory pathway and the ventricles reaching 200-300 contractions/minute. Unlike the conduction through the atrioventricular node, the conduction on an accessory pathway during an episode of atrial fibrillation can cause a ventricular response that can be fatal. This hidden accessory conduction pathway seems to play a less important role than the atrioventricular node in the limitation of the ventricular response. Due to the fact that a significant augmentation of the sympathetic tonus can also increase the ventricular pre-excitation syndrome, the modification of the vagal tonus seems to have a minor effect in the accessory pathway conduction [4]. The specific electrocardiographic figure for the WPW atrial fibrillation includes the irregular rhythm, a quick ventricular response, the presence of delta waves and a wide strange conformation of the QRS complex.

In atrioventricular reentrant tachycardia the electrical impulse has a descendent trajectory starting from the atrium level to the atrial node and the ventricles and then the impulse returns to the atria on the accessory pathway. One of the most rare types of atrioventricular reentrant tachycardia involves an electrical circuit that descend from the atria towards the ventricle on accessory pathway and then backwards towards the atria on the pathway of the atrioventricular node. These reentrant circuits cause a ventricular contraction with a frequency of 140-220/minute [5].

WPW Syndrome- description

The WPW syndrome is the most frequent and specific form of ventricular pre-excitation. The exact preponderance of the WPW syndrome is difficult to be evaluated because the electrocardiographic abnormality is discontinuous and there are disguised or hidden forms which can be revealed by electrophysiological explorations. It is estimated that the WPW syndrome affects about 0,1-0,3% of the general population; most of the time, the syndrome appears in an undamaged heart and in 60-70% of the cases the patients show no antecedents of heart disease.

From the clinical point of view during tachycardia the patients can suffer from palpitations, dizziness, and syncope.

The most severe complication of the WPW syndrome is the sudden death that fortunately occurs relatively rare, due to transmission of paroxysmal atrial fibrillation on the accessory conduction pathway to the ventricles. In these conditions, the accessory pathway which has a short refractory period transmits to the ventricles a large number of stimuli along with the perturbation of ventricular activity [6].

The specialty literature describes cases of sudden death in persons who suffered from asymptomatic WPW and also cases in which members of the same family have been diagnosed with identical localization of the accessory pathway. These aspects emphasize the importance of the early diagnose of this syndrome and the need to warn the patient's relatives.

A certain part of the persons that have accessory pathway can present a normal EKG because the accessory pathway is active only during a tachycardia, thus being a hidden accessory pathway [7].

The treatment for the WPW syndrome must be customized according to each patient. In patients who have WPW syndrome electrocardiographically proved but without manifest tachycardia it is necessary to carry out a careful clinical monitoring. If the symptoms related to tachycardia occur, it is necessary to carry out an electrophysiological study [8]. There are rare situations when the syndrome is treated with antiarrhythmic medication but most of the time the patients opt for the radiofrequency ablation of the accessory pathways which consists of intracavitary electrophysiological identification of the accessory bundle, i.e. location of the accessory bundle, conduction velocity, refractory period, followed by the application of radiofrequency energy by placing the right electrode on the accessory bundle. This method show very good results in 85% of the cases [9].

Case presentation

We present the case of a 34 years old man who has been the victim of an aggression, being punched and kicked, respectively “throttled”. Immediately after the aggression he was hospitalized for: general feeling of weakness and consciousness impairment, atrial fibrillation with ventricular frequency of 250-290/minute.

The patients was diagnosed with “Atrial fibrillation. Wolff-White syndrome. Left ventricular failure.” The patient also had a few injuries, namely: on both suborbital and malar area reddish-purple bruises and moderate swelling, on the anterior side of the right leg a red-purple bruise and moderate swelling.

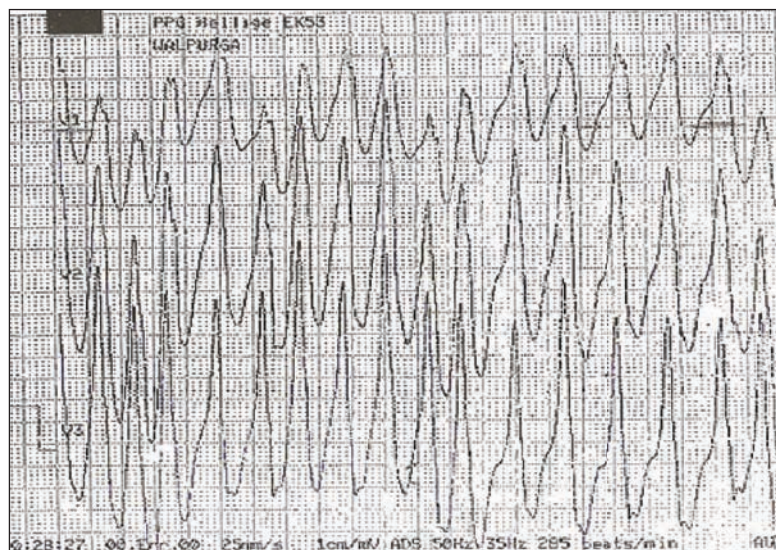


Figure 1. EKG at admission in the hospital – pericardial leads

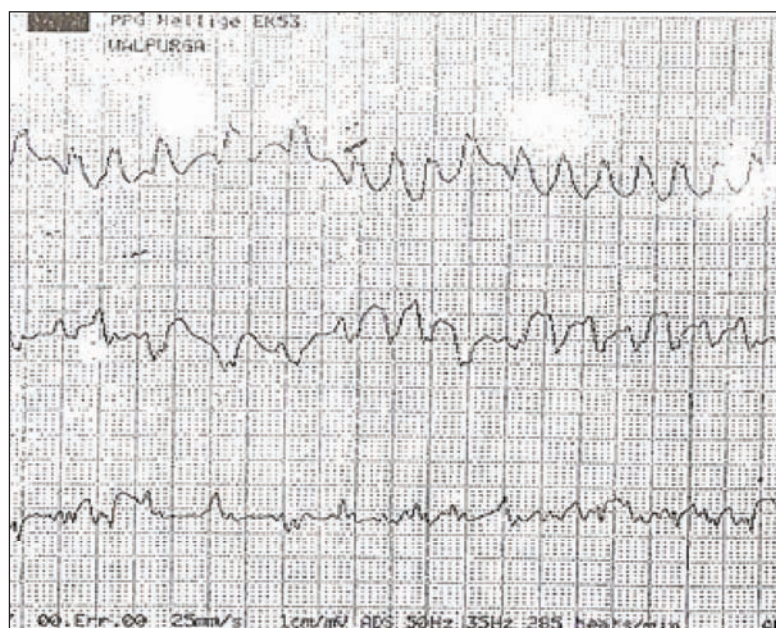


Figure 2. EKG at admission in the hospital– standard leads

stimulation with progressive decrement extra-stimulus reveals the presence of a retrograde conduction with an atrial premature depolarization in retrograde conduction in the area of the distal coronary sinus.

Transaortic retrograde approach with positioning of the Cordis Webster ablation catheter with a tip of 4 mm at the level of the mitral ring. The lateral area of the mitral ring is mapped with the ablation catheter in atrial stimulation, with applications of RF (setup 30-35W/55grC) in a fit AV fusion area; during the application the anterograde conduction disappeared. On the basis of the electrocardiographic appearance and the result of the electrophysiological study it has been asserted the diagnosis of ventricular pre-excitation syndrome with lateral left accessory pathway with life-threatening risk. Based on this diagnosis, radiofrequency ablation of the accessory pathway was performed. The patient was discharged with sinus rhythm, mild QRS complex without pre-excitation conformation.

During hospitalization electrical conversion by a 200J impulse of the WPW syndrome was performed followed by improvement of the patient's clinical condition. Before being released from the hospital, the patient was programmed for ablation.

The medical certificate issued by the general practitioner proves that the patient was not registered with any chronic diseases before hospitalization.

A month after discharge, the patient was hospitalized in a specialized medical facility where radiofrequency ablation of the accessory pathways was performed.

The clinical and EKG examination showed sinus rhythm of 70/min frequency, QRS complex with configuration of ventricular pre-excitation with localization of the accessory pathway on the basis of the 12 EKG lateral left conformation (positive delta wave in V1 with R>S and positive in aVF). The electrophysiological study was also performed, with the right femoral vein being approached by using three catheters: 7F dodecapolar placed with the distal tip in the coronary sinus, 6F quadripolar placed in hisian and one mapping catheter. The atrial stimulation in continuum drive at the cycle of 500ms reveals anterograde conduction on the accessory pathway, in anterograde conduction the maximal fusion being revealed at the level of the distal pair of electrodes of the hisian catheter. The programmed ventricular

The cardiac examination of the patient performed after a few months from discharge showed: blood pressure of 120/70 mmHg, normal and rhythmic heart noises. The electrocardiography examination showed: sinus rhythm of 78/min, A+750PRO 16", normal morphology.

Discussion

In this case, a relatively young man suffered from a trauma, followed by sudden appearance of atrial fibrillation and left ventricular failure in the context of a Wolff-Parkinson-White syndrome. Medical evidences showed that the patient had no previous cardiac problems and he has not been the subject of any medical investigation before this date in order to establish the existence of this syndrome.

In this context, it can be concluded that the clinical manifestation of the Wolff-Parkinson-White syndrome could have appeared as a clinical expression of an intense acute stress due to trauma conditioned by the existence of the accessory pathway Paladino-Kent bundle, i.e. the trauma could have triggered the clinical manifestation of WPW syndrome. The trauma and the intense acute stress unlocked a ventricular pre-excitation syndrome on left lateral accessory pathway (Wolff-Parkinson-White syndrome) with super-ventricular paroxysmal tachycardia and atrial fibrillation.

The consciousness impairment showed by the patient immediately after the trauma was most likely the consequence of the left ventricular failure and atrial fibrillation (that required electrical conversion), with acute decrease in brain oxygenation. In this way the WPW syndrome with atrial fibrillation and severe left ventricular failure threatened the patient's life and required emergency electrical cardioversion.

Conclusions

The WPW syndrome appears due to the existence of a particular anatomical configuration characterized by the existence of a preferential conduction pathway that connects atria and ventricles (which are otherwise completely electrically isolated by the fibrous atrioventricular plane, the only electrical connection being the Aschoff-Tawara node). This additional bundle, i.e. Paladino-Kent bundle, is present in about 0,1-0,3% of the general population.

Most of the persons that have this bundle have a normal electrocardiographic aspect and no clinical symptoms. Furthermore, the electrocardiographic diagnosis is possible only if the examination is performed when the depolarization is produced on accessory pathway. In this way the disease can remain "silent" for a long time, both clinically and electrically.

In certain situations, such as trauma, the presence of the Paladino-Kent bundle (hence, the presence of two conduction pathways between atria and ventricles) can induce super-ventricular paroxysmal tachycardia by a reentrant mechanism, and WPW becomes clinically visible.

WPW syndrome requires early diagnose and adequate medical treatment as it predispose to severe heart failure and sudden death. In addition, the members of the patient's family need to be warned as they could show identical localization of the accessory pathway.

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