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Sudden death risk associated with asymptomatic Wolff-Parkinson-White syndrome

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Abstract. First described in 1930, the Wolff Parkinson White (WPW) syndrome has the accessory pathway as anatomical and electric substrate for different types of arrhythmia. In 1979 sudden cardiac death associated with WPW was mentioned for the first time.
The mechanism by which sudden death occurs is atrial fibrillation conducted rapidly over the accessory pathway or pathways with a short effective antegrade refractory period (EARP) that deteriorates into ventricular fibrillation. When WPW syndrome is associated with tachycardia, it is a good scenario, because the patient can figure out the presence of the accessory pathway and can at least evaluate it by electrophysiological study or treat it by radiofrequency catheter ablation.
We present the case of a medical school student, totally asymptomatic, who was randomly diagnosed with WPW syndrome during the clinical practice in the cardiology service, being volunteer for an electrocardiogram. The patient requested electrophysiological study after the steps of the procedure were properly explained. The study revealed the great risk of sudden death as during the procedure atrial fibrillation conducted over a short AERP accessory pathway was easily induced and degenerated into ventricular fibrillation. Five external electrical conversions and the rapid infusion of Amiodarone were needed to return and maintain the sinus rhythm. The catheter ablation of the accessory pathway was performed. During time, a number of risk factors for sudden death associated with WPW were found as follows: shortest preexcited RR interval during and its surrogate, the antegrade ERP of the accessory pathway; multiple accessory pathways; male gender and syncope.
Key words: *Wolff-Parkinson-White syndrome, sudden death, syncope, electrophysiological study, catheter ablation, atrial fibrillation*.

Introduction

The management of symptomatic Wolff-Parkinson-White (WPW) is relatively defined with catheter ablation of the accessory pathway as main curative approach recommendation, as for the asymptomatic WPW the management strategy is still controversial.
The syntagm of low risk asymptomatic WPW was challenged many times by sudden deaths that would have been prevented if the electrophysiological study had been performed as soon as possible (1).

Case report

A 22 years old student of medicine, was diagnosed with WPW syndrome during an internship in our Cardiology Department as he volunteered for an ECG demonstration during the cardiology.
The patient was asymptomatic, with no history of cardiovascular events or arrhythmia.
Electrophysiological study was recommended even more as the patient's desire was to perform extreme sports (bungee jumping and skydiving).
The 12 lead standard ECG revealed sinus rhythm, HR=72/min, preexcitation syndrome (Fig. 1) with typical changes- short PR, enlarged QRS complexes and obvious delta wave (Fig. 2).
The blood tests and the echocardiography performed at admission showed no pathological changes.
The invasive electrophysiological testing was performed.

During the incremental atrial pacing the patient developed atrial fibrillation (AF) with very rapid ventricular response, up to 300/min, via accessory pathway (Fig.3 and Fig. 4) and spontaneous degeneration into ventricular fibrillation (VF) (Fig 5).

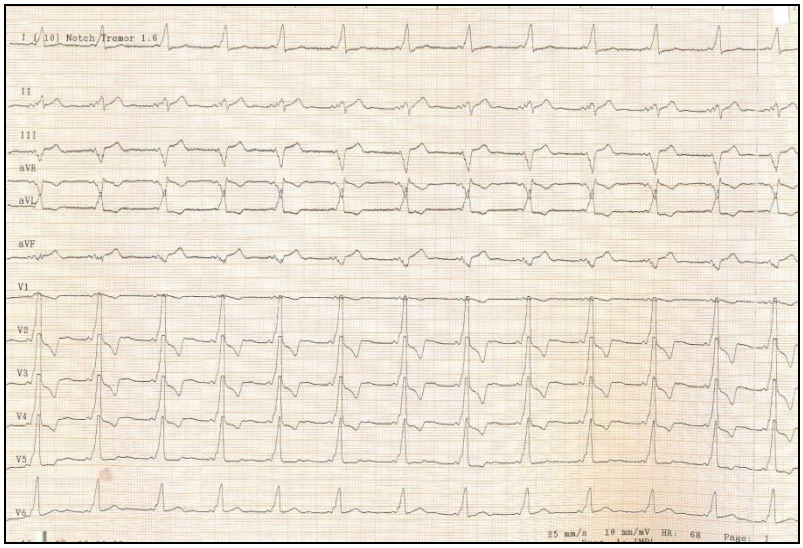

Figure 1. Twelve lead standard ECG.
Sinus rythm, HR=72/min, PR=80ms, QRS=140ms, QT=430ms, positive delta wave in leads I, II, aVL, V2-V6

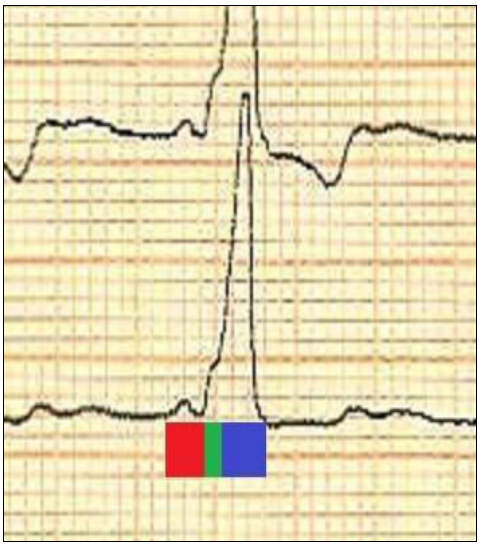

Figure 2. Detailed image of a QRS complex recorded in V5.
It showing typical WPW changes: red- short PR, green - delta wave, blue- the rest of the QRS complex

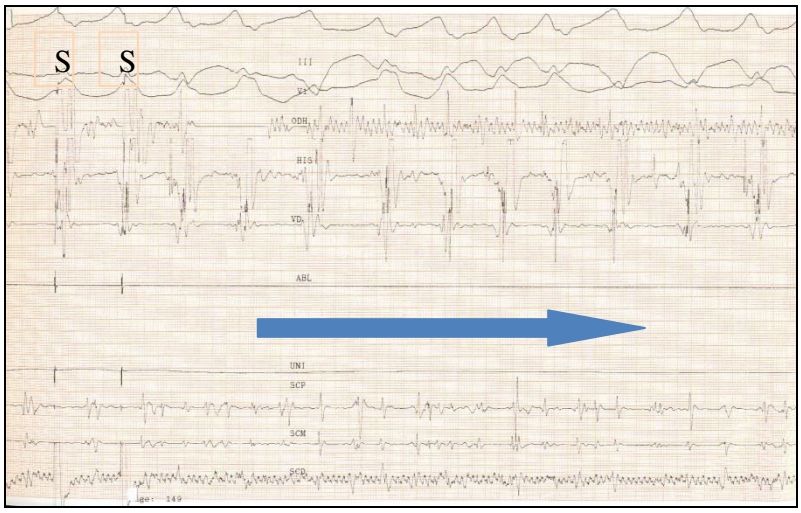

Figure 3. Atrial fibrillation with fast ventricular response via accessory pathway revealed during incremental atrial pacing
DI, DIII, V1- ECG derivations, ODH- high right atrium lateral wall, HIS- His electrogram, VD-right ventricule, ABL- ablation channel, UNI- unipolar derivation, SCP- proximal coronary sinus, SCM- medium coronary sinus, SCD- distal coronary sinus
S- last two atrial stimuli

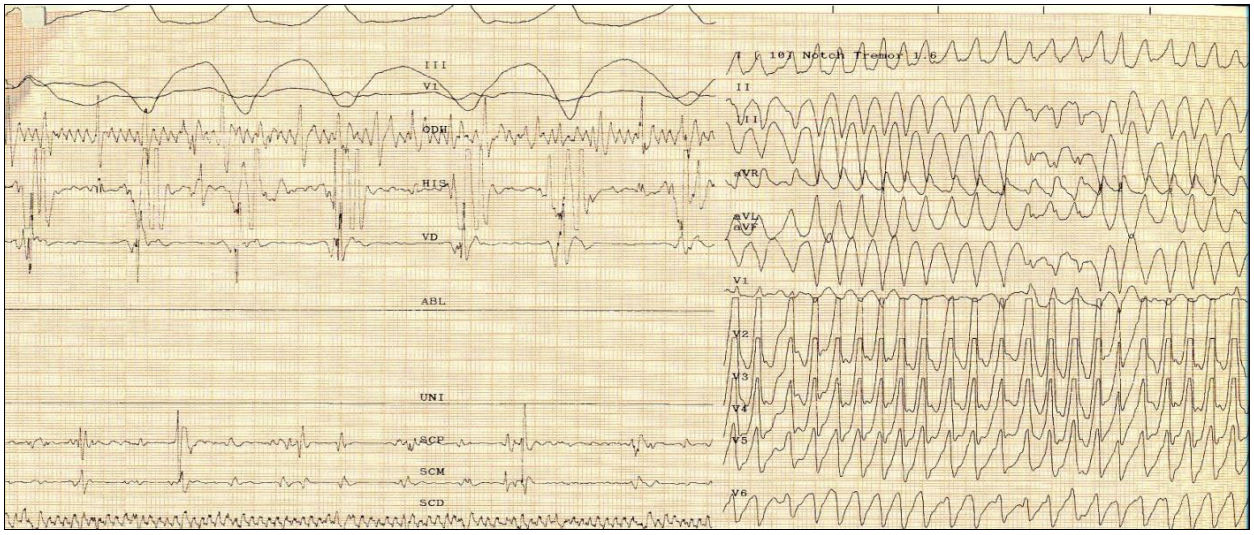

Figure 4. Endocavitary (left) and 12 lead standard ECG (right) showing atrial fibrillation with very fast ventricular response up to 300/min and short RR preexcited interval

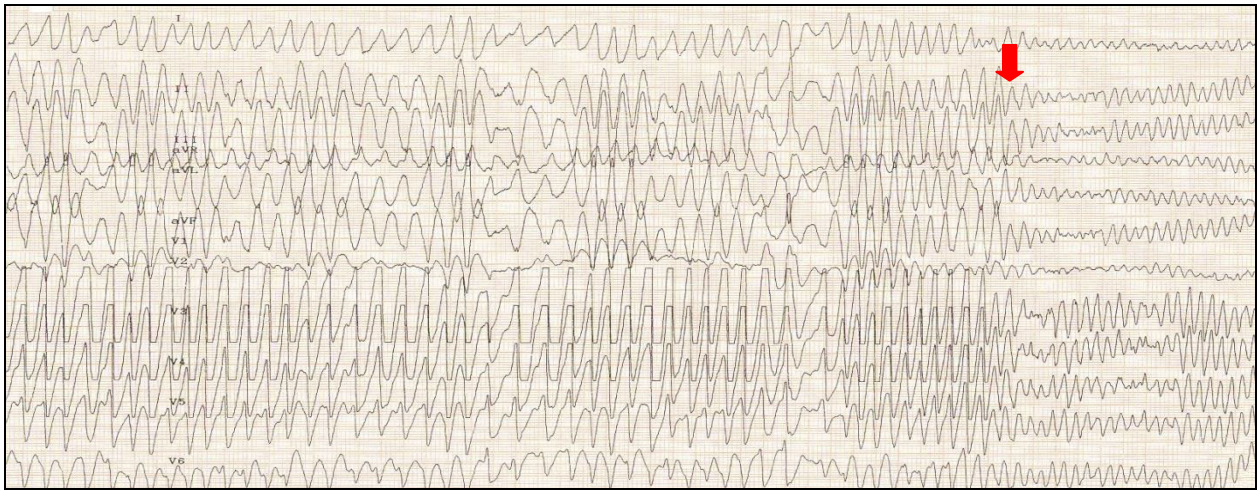

Figure 5. Atrial fibrillation spontaneously degenerate into ventricular fibrillation (red arrow)

Five 270J external electrical conversions (Fig.6) tempted to reestablish sinus rhythm. Rapid infusion of 600mg of amiodarone over 20 minutes was necessary to maintain sinus rhythm.

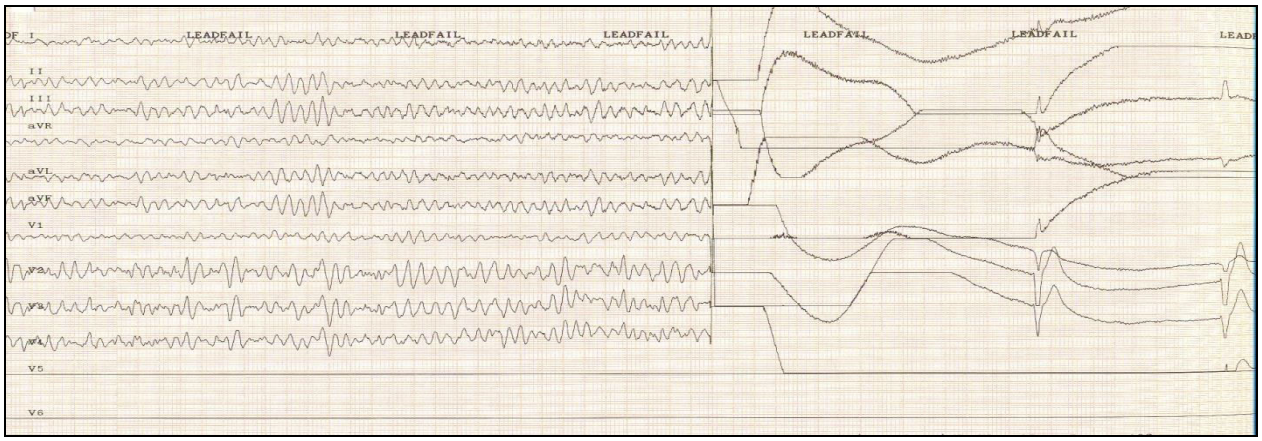

Figure 6. Electrical conversion of the ventricular fibrillation to slow junctional rhythm.

After stable sinus rhythm was obtained, successfully ablation of a short anterograde refractory period (AERP < 220 ms) left anterior accessory pathway was performed (Fig.7).

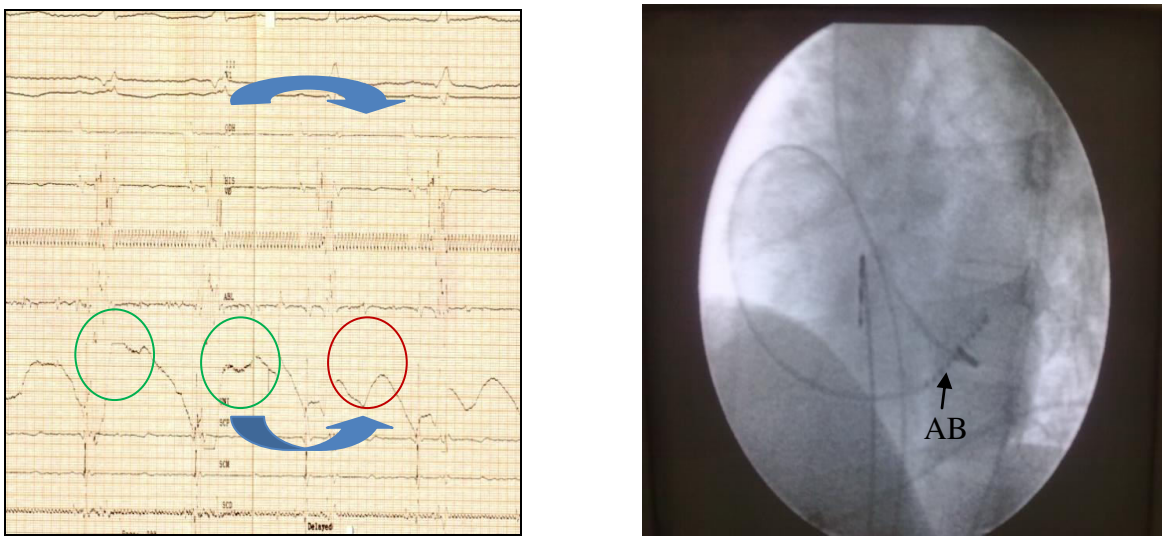

Figure *7. **Left:** the moment of successful ablation- separation of the A and V potentials recorded on the ablation catheter channel at the ablation target point and changing of QRS morphology registered by the surface ECG channels. **Right:** Left anterior oblique projection in fluoroscopy. ABL- ablation catheter positioned at the left anterior accessory pathway target site.*

Conclusion

Sudden death among the patients with WPW syndrome is not well documented and probably underestimated and could be the first clinical manifestation of this syndrome. Risk factors for potentially life-threatening arrhythmic events in WPW syndrome were established as it follows: short AP-AERP (<250ms) allowing a rapid ventricular response during AF; inductility of tachyarrhythmia during EPS (AVRT and/or AF); short pre-excited RR interval during AF (<250ms); multiple APs; male gender; age and syncope (2,3).
We strongly recommend electrophysiological study in every patient with WPW syndrome.

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