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Editorial

Sudden Cardiac Death Associated to Auriculoventricular Accessory Pathways: Sleeping with the Enemy

Osmar Antonio Centurión[1,2]*, Judith María Torales[1,2] and Laura Beatriz García[1,2]

[1]*Department of Health Sciences Investigation, Sanatorio Metropolitano, Fernando de la Mora, Paraguay*

[2]*Cardiology Department, Clinic Hospital, Asunción National University (UNA), San Lorenzo, Paraguay*

***Corresponding Author:** Osmar Antonio Centurión, Professor of Medicine, Department of Health Sciences Investigation, Asuncion National University (UNA), Sanatorio Metropolitano, Fernando de la Mora, Paraguay.

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It may be a life-threatening situation when paroxysmal atrial fibrillation (AF) develops in patients with manifested Wolff-Parkinson-White (WPW) syndrome, that is, those who have anterograde conduction via the accessory pathway (AP) [1-4]. In this case, the ventricular rate may fastly accelerate if the AP has a short refractory period leading to ventricular fibrillation. Indeed, the presence of a high risk AP denotes the presence of an undeniable killer, and it is a red alert situation to a probable episode of sudden cardiac death (SCD). Since we do not want to sleep with the enemy, there are several diagnostic and therapeutic maneuvers that should be performed in order to localize and destroy the enemy to overcome a fatal situation caused by the AP [5-9].

A conventional electrocardiogram (ECG) is probable one of the cheapest diagnostic auxiliary method that is easily and widely available in the clinical setting. We can make the diagnosis of manifested WPW syndrome with a simple ECG when we found a short PR interval, a delta wave, and a wide QRS complex. Although, the diagnosis is easily made with the ECG, we still do not know if we are dealing with a high risk AP. At that time, we still do not know the predisposition to develop paroxysmal AF that will lead to fast ventricular response developing ventricular fibrillation and SCD. There are various mechanisms dealing with the development of paroxysmal AF in WPW patients. For example, the convertion of a macroreentrant tachycardia into AF is one of them, specially the antidromic tachycardia. The existence of a functional AP and inducible atrioventricular tachycardia in the electrophysiological laboratory has been found to play an important role in triggering AF in the WPW syndrome. It was demonstrated that these tachycardias can increase the vulnerability of the atrial myocardium due to the fast atrial rate, the augmented sympathetic tone and the stretching of atrial walls. In addition, the electrical properties of the AP and the presence of several AP are distinct mechanisms. It was mentioned that intermittent retrograde conduction over a second AP with faster conduction caused early atrial depolarization as a mechanism of premature atrial contraction that initiates atrial repetitive firing or intratrial reentry in the vulnerable period of the atrium during reciprocating tachycardia or during incremental right ventricular pacing [9]. Moreover, the effects of AP on atrial architecture, and the intrinsic abnormalities of the atrial muscle generating atrial vulnerability are other mechanisms [10-18].

Several studies observed that the anterograde AP refractoriness was shorter in patients with AF than in control patients without it. The anterograde effective refractory period of the AP was observed to be significantly shorter in WPW patients with AF that in those without clinical AF [11,19,20]. The AP itself develops as a consequence of an embryologic flaw in the formation of fibrous tissue at the level of the auriculoventricular annulus [21]. Electrophysiological alterations may be present in the atrial tissue adjacent to the AP leading to dispersion of atrial refractoriness and conduction abnormalities in the proximity of zones with different tissues such as the junction of the atrial myocardium and the AP. Histological or electrophysiological properties of the atrial muscle in the closeness of the AP may be very important in the development of atrial vulnerability to AF in the WPW syndrome.

Most WPW patients whom undergo AP catheter ablation do not develop reciprocating tachycardias neither AF anymore. Although, AF catheter ablation may be a definite curative procedure, sustained clinical episodes of AF may still happen in certain patients even though there is no more conduction through the AP. However, this recurrent episode of AF in the absence of AP conduction post-ablation is no longer fatal in the arrhythmogenic setting since there is no more anterograde conduction through the AP. Although, AF may still occur in nearly one third of the patients with WPW syndrome after successful catheter ablation of the AP, the dreadful killer (the AP) has been silenced. Indeed, the AP has disappeared from the clinical scenario, so the patient will no longer be sleeping with the enemy.

Now, why does the patient still develops AF despite the successful catheter ablation of the AP? A plausible explanation may be the presence of an underlying atrial myocardial disease [11,13,22]. Some researchers observed that the induction of sustained episodes of AF in the laboratory was more frequent in patients with documented episodes of clinical AF. Others have analyzed the atrial electrophysiological substrate that may predispose to AF, evaluating atrial refractoriness, intra-atrial and interatrial conduction times, atrial endocardial electrograms morphology in sinus rhythm, and several electrophysiological parameters for augmented atrial vulnerability, namely, fragmented atrial activity, repetitive atrial firing, and intra-atrial conduction delay.

Regarding the latter parameters of augmented atrial vulnerability, which can be elicited by programmed atrial stimulation, it was demonstrated that patients with WPW syndrome associated with paroxysmal AF have a wider fragmented atrial activity zone, and a wider repetitive atrial firing zone than those WPW patients without AF [13]. The widening of the zones of fragmented atrial activity and, the repetitive atrial firing is associated to the development of AF in WPW patients [13,23]. It was also observed that the zone width of interatrial conduction delay in WPW patients with clinical episodes of paroxysmal AF was broader than in those without AF [13]. Therefore, the WPW patients with clinical episodes of paroxysmal AF tend to show a greater incidence of slow interatrial conduction.

When atrial endocardial mapping during sinus rhythm is performed with catheter electrodes, abnormal atrial electrograms may be recorded. These abnormally prolonged and fractionated atrial endocardial electrograms may reflect slow and anisotropic conduction through a diseased atrial muscle [24-26]. Abnormal atrial electrograms were recorded in 83% of WPW patients who had paroxysmal AF, but only in 10% of those WPW patients without AF. These abnormal electrograms are due to asynchronous activation of myocardial fibers where the myocardial cells are separated by connective tissue [27]. These abnormal electrograms may indicate the presence of certain zones of the atria prone to develop reentrant circuits.

A very interesting finding was observed in a large multicenter, international study performed by Etheridge SP., *et al* [28]. They demonstrated that SCD occurred most often in adolescent males who were not engaged in competition. Although competitive athletics are considered to increase SCD risk [29], sports restriction would not have prevented SCD in the 73% of their patients whose events did not occur with competition. This finding was consistent with a previous series exploring SCD in the young, where most events occurred during rest or sleep [30,31]. Due to this interesting finding, they conclude that they do not support unrestricted sports participation in patients with WPW syndrome but demonstrated that sports restriction does not keep adolescents safe. Therefore, they advocate a low threshold for catheter ablation to cure WPW syndrome.

In conclusion, there are several mechanisms related to the AP itself and the intrinsic atrial vulnerability to develop AF. This clinical scenario in the presence of a fast anterograde conducting AP may lead to ventricular fibrillation and SCD. Most of these life threatening clinical events occur at rest in young adolescents. Even the asymptomatic patient is not immune from SCD. Since catheter ablation of the AP can cure WPW syndrome and eliminate SCD risk, there should be a serious, detailed and criterious decision making balancing the small long-term risk of an episode of SCD with the immediate very low risk of a catheter ablation complication. Therefore, we should lower our threshold even further for catheter ablation to cure WPW syndrome in certain asymptomatic patients. As a physician, it is easier to think otherwise when we are not the one that has the sword of Damocles over our heads. As long as the dreadful killer, the AP, is stalking around we should not let our patients be peacefully sleeping with the enemy.

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