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Editorial

Cardiocentro ERNESTO CHE GUEVARA SANTA CLARA CUBA

Understanding atrial fibrillation by accessory pathway starts from knowing the electrophysiology of accessory bundles and the atrial wall

Entender la fibrilación auricular por vía accesoria parte del conocimiento de la electrofisiología de los haces accesorios y de la pared auricular

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Atrial fibrillation (AF) with anterograde accessory pathway (AP) conduction in patients with Wolff-Parkinson-White (WPW) syndrome is sometimes difficult to recognize at the emergency department, even by specialists of well-known experience. A study by Koźluk *et al*[1] showed that only 18% of survey respondent specialists could recognize an AF by AP; only 10% recognized an AF, and the rest (72%) made a mistaken diagnosis. Unfortunately, the lack of knowledge concerning this disease threatens the life of the patient, since the AF by AP can degenerate into ventricular fibrillation and cause sudden death to the patient.

It is worth commenting on some considerations of interest about the articles of Mata Cuevas *et al*[2] and Moreno-Martínez *et al*[3], published in this issue of CorSalud.

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Aspects of the pathophysiology of AF by AP

What is known as electrical and structural remodeling is well described in the pathophysiology of AF, that is gradually generated in paroxysmal episodes recurrently, which we will not explain here, as the reader can find it in the references of this editorial. From this electrical and mechanical remodeling comes the expression known in the bibliography as "atrial fibrillation begets atrial fibrillation" [4]. The latter is what occurs in part of the pathophysiology of this arrhythmia that Mata Cuevas *et al*[2] explained.

One of the issues that would suggest us that the pathophysiology of AF in the general population differs, somewhat, from the pathophysiology of AF by AP is that, once the ablation of the accessory bundle is accomplished, some of the patients never have more episodes of AF and others do. The pathophysiological differences of AF by AP are: a) electrophysiological accessory pathway properties b) the effect of AP's electrophysiology in atrial architecture at the site of its insertion c) intrinsic vulnerability of the atrial muscle[5].

These pathophysiological issues make the incidence of and AF by AP, described in 1977, higher in patients with WPW (10-30%) than in the general population[6].

Regarding the electrophysiology of the accessory bundle, there are the refractory short anterograde periods of AP, which are very common in young patients, as presented by Mata Cuevas *et al*[2]; in these patients, if the correct medical intervention does not take place, the final result can be fatal for the patient. In such case of AF by AP, it is very important to mention the shortest RR distance in the electrocardiogram, because one of the risk assessments that cannot be missed is the likely refractory period of the AP, determined by the shortest RR. If it is less than 250 milliseconds, it confers a high risk to the patient[7].

The description of atrioventricular reentry tachycardia that degenerate into AF by AP is related to the presence of multiple retrograde accessory pathways or multiple retrograde fibers, which cause several focal depolarizations in the atrial wall, and electrophysiological disorganization in this structure. The presence of retrograde atrial double potential in coronaries sinuses is well associated to the beginning of AF in patients with WPW syndrome, and it is not described in patients without this syndrome. These double potentials, that are involved in the pathophysiology of AF by AP, are triggered in relation to the presence of premature ventricular complexes, which conduct, retrogradely, into the atria by multiple accessory pathways or multiple retrograde fibers. The collision of multiple wave fronts in the atrial wall can be then the beginning or the perpetuation, or both, of the AF by AP[5].

The intrinsic vulnerability of the atrial muscle is given by the dispersion of refractoriness, by the homogeneity of the depolarization and repolarization, and by areas with shorter conduction speeds. These features, together with intraatrial conduction delays and longer durations of endocardial atrial electrocardiograms, are more common characteristics in patients with WPW and AF, than in those with the syndrome but without AF[5]. The latter, on the atrial muscle, are pathophysiological aspects that have been described in the AF of patients without WPW syndrome; it is clear that there are mixed pathophysiological issues and others that are purely of the WPW syndrome.

Knowledge of the pathophysiology combined with the complications that a disease can present is vital for the implementation of a treatment. That an AF by AP can degenerate into ventricular fibrillation and cause sudden death is the knowledge that motivates us to apply an urgent therapeutic approach in favor of the patient's life.

Treatment of AF by AP

The hemodynamic stability or instability of the patient with AF by AP is what can define the therapeutic behavior. For cases with hemodynamic instability, synchronized electrical cardioversion is always recommended. In either cases, because of the risk of ventricular fibrillation and sudden death mentioned before, in my opinion, there is well justified the synchronized electrical cardioversion[8,9].

In the case of patients with hemodynamic stability, it is useful using drugs intravenously as ibutilide, procainamide or drugs from the IC group, as flecainide. In its defect, amiodarone has been used, the latter with the risk of reducing conduction through the atrioventricular node and facilitating conduction through the AP, which would worsen the clinical-hemodynamic picture of the patient. Finally, once the AF by AP is resolved, each case will be solvable by performing the ablation of the AP, for which the patient has to be referred to invasive electrophysiology departments[8,9].

Mata Cuevas *et al*[2] mentioned, when referring to the AF's management guidelines, that currently, there are two approaches for the treatment of this arrhythmia, one of them is aimed at reducing the deleterious electromechanical effects of AF on the myocardium, and another to reduce the appearance of thromboembolic complications. In my opinion, this comment is important for the AF in the general population, not for patients with AF by AP.

The AF's classification of the clinical practice guidelines of the European Society of Cardiology in the general population, commented by Mata Cuevas *et al*[2], can help to orient toward a therapeutic approach, but in the case of AF by AP must not adjust to this classification, because doing it separates us from the true knowledge of the pathophysiology and treatment of AF by AP.

In the references of this editorial[1,4-12], it is possible to delve into the pathophysiological and therapeutic issues of AF by AP.

CONFLICT OF INTERESTS

None declared.

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