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Editorial

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Ischemic atrial fibrillation: how to interpret its pathophysiology and the strategic decision of treatment concerning the arrhythmia

Fibrilación auricular isquémica: cómo interpretar su fisiopatología y la decisión estratégica de tratamiento en torno a la arritmia

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Atrial fibrillation (AF) is the arrhythmia which generates more scientific literature at present. This interest is due to several reasons, among which are the persistent ignorance about its mechanism and the difficult actions to take. However, the biggest factor in the interest in this arrhythmia is its high prevalence and the large economic and clinical impact it causes[1].

In this issue of CorSalud, Vicente Linares *et al.*[2], report a case with an episode of AF during an acute myocardial infarction (AMI) and its reversion to sinus rhythm after percutaneous coronary intervention (PCI). The authors discuss the therapeutic strategy based on the reversibility of arrhythmia after PCI.

The pathophysiology of AF is complex, and it is probably more complex during an acute coronary ischemic event, where the presence of risk factors associated with this arrhythmia has been demonstrated. These risk factors include: left ventricular failure; a high ventricular response, probably due to ventricular failure, the corresponding hemodynamic disorder, or both; and the low use of beta blockers and thrombolytics in some patients who present most episodes of AF during AMI[3].

Seventy percent of patients with AF are between 65 and 85 years of age[1], demonstrating that older age increases the risk of AF by the existence of inter- and intra-atrial conduction disorders. This is shown by higher P wave dispersion (Pd) values in the electrocardiogram. Patients with hypertension also have higher values of Pd, with proven paroxysms of AF[4-6]. In fact, hypertension confers a relative risk of 1.5 times after being adjusted for other risk factors[1]. The changes in the atrial wall that are shown in the electrocardiogram with increases in Pd are called structural and electromechanical remodeling of the atria. Patients with

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this disorder have a greater risk of AF[7].

Acute ischemic heart disease is also a risk factor associated with AF. Increases in Pd have been shown in these patients before AF episodes.

Clearly, the addition of risks increases the possibility of AF[4-6].

When assessing a patient, the ignorance of all the pathophysiological factors involved in the arrhythmia compels us to collect all clinical and epidemiological history, without forgetting any of the elements described in the onset and maintenance of arrhythmia[1,8].

Márquez *et al*.[8], when summarizing recent advances in the pathophysiology of AF, point out the following factors:

a) Genetic factor: involved in cases of familial AF.
b) Structural predisposing factor: atrial dilatation is the best known structural factor that allows the development of AF. Bachmann's bundle and interatrial conduction pathways are other predisposing structural factors that establish frequency gradients between left and right atrium, giving them a role in fibrillatory conduction.
c) Electrophysiological predisposing factors: heterogeneous refractory periods favor the generation of AF.
d) Trigger factors: the role of abnormal electrical activity (ectopic foci).
e) Modulating factors: the role of the autonomic nervous system.

From the electrophysiological standpoint (trigger factors), premature atrial complexes (PACs) are triggers in the onset of AF. These PACs may meet areas in refractory period, not conducting longitudinally in the fiber (longitudinal block), but transversely, to reenter then in the blocked area. Indeed, anisotropic reentry has been shown in normal ventricular and atrial muscles. Schmitt *et al*.[9], proposed a reentry model based on the longitudinal dissociation of a group of fibers that run parallel. Allessie *et al*.[10,11] were able to demonstrate, forty years later, reentry in rabbit anatomical parts where there were no anatomical barriers, and introduced the concept of leading circle. And Spach *et al*.[12,13] showed that in the normally arranged fibers the conduction speed in the longitudinal direction is much higher than in the transverse direction, since resistivity is higher in the transverse direction, because there are fewer intercalated disks, and this will make the premature stimuli to be blocked longitudinally and conducted transversely. Then, they will reenter distally and depolarize initially blocked areas. The atria, moreover, have a number of anatomical structures that facilitate conduction blocks and conduction in other directions, such as: *fossa ovalis*, *crista terminalis* and *musculae pectinati*[14].

All these electrophysiological alterations can initiate and perpetuate an AF that results from a PAC, when starting a fibrillatory conduction[9], in addition to the clinical and epidemiological conditions that can be found in patients with AF episodes (age, hypertension, acute myocardial infarction, diabetes mellitus, lung diseases), and the presence of a well known electrophysiological setting (PAC) as the trigger for the arrhythmia.

Regarding the treatment modality in a patient with AF, first it is important to organize all the risk factors affecting the patient. The AFFIRM study indicates that patients with AF at high risk of cerebrovascular accident (CVA) generally benefit from anticoagulation treatment even after sinus rhythm was restored. Therefore, the diagnosis of AF in patients with risk factors for thromboembolism should be accompanied by the introduction of an undefined oral anticoagulant (OAC) treatment, except when a reversible precipitating factor for AF has been identified, such as corrected hyperthyroidism[15]. To assess the risk of CVA, there is the CHA_2DS_2-VASc score [heart failure/left ventricular systolic dysfunction, hypertension, age > 75 years (2 points); diabetes, stroke (2 points); vascular disease, age between 65-74 years and female][16]. A patient who just meets these two conditions, hypertension and age between 65-74 years, would add 3 points according to the risk scale for CVA, therefore, should be anticoagulated. When the use of anticoagulation with vitamin K inhibitors could pose a high risk of bleeding, a reasonable alternative, with less bleeding risk, would be the new OAC (dabigatran, rivaroxaban, apixaban)[16].

The recent Consensus Paper of the Working Group on Thrombosis of the European Society of Cardiology, also supported by the European Heart Rhythm Association and the European Association of Percutaneous Cardiovascular Intervention[17], proposes the use of triple therapy in patients with AF who suffer an AMI or have a chronic ischemic heart disease, and are treated with stent implantation. Such therapy includes the short-term use of OAC with acetylsalicylic acid at a dose of 100 mg/day and clopidogrel at a dose 75

mg/day, followed by a longer treatment with OAC and a single antiplatelet agent. After 1 year, the use of an OAC, such as warfarin alone, seems to be enough.

The conventional stenting of a coronary artery occluded segment in a patient with AF risk factors (advanced age, hypertension, and an electrophysiological environment conducive to triggering and maintaining the arrhythmia) requires, as a therapeutic strategy, the use of triple therapy for four weeks[17].

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