Editorial

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Atrial fibrillation and bronchial asthma: two conditions linked by systemic inflammation

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ARTICLE INFORMATION

Received on November 18, 2025.
Accepted after review on November 29, 2025.
www.revistafac.org.ar

There are no conflicts of interest to disclose.

Keywords:
Atrial fibrillation,
asthma,
chronic inflammation.

ABSTRACT

Atrial fibrillation (AF) is known to be an arrhythmia characterized by uncoordinated atrial electrical activation. It is the most common sustained arrhythmia worldwide and is associated with significant morbidity and mortality. AF can range from asymptomatic (subclinical) to manifest episodes of arrhythmias (paroxysms) to permanent (formerly called chronic). Sometimes its first clinical manifestation can be a stroke (cerebrovascular event or cerebrovascular accident). Therefore, it is important to identify patients with a higher propensity to develop AF.

The association between AF and asthma is bidirectional. On the one hand, evidence suggests that AF shares a background of systemic inflammation with asthma. Asthma, being a systemic inflammatory disease, could be affecting the atria and thus favoring the development of atrial cardiomyopathy, which would predispose to the onset of AF.

In this issue of the Revista de FAC (Journal of FAC), Carlos Paterno et al. present the data from a retrospective study on 435 patients, of whom 93 were chronic and asymptomatic, a group that was compared with the rest, who had diseases categorized as "systemic autoinflammatory diseases" (SAIDs), such as psoriasis or rheumatoid arthritis, among others.[1] Although values of inflammatory activity were not compared directly, the authors compare both groups, considering them as subrogate to endothelial damage by inflammation: blood pressure values and the presence of atrial fibrillation (AF). Interestingly, neither blood pressure, nor AF frequency were different between both groups, so they assume there is similar systemic inflammation between asthma and the rest of SAIDs.[1]

This editorial will focus on AF, asthma and inflammation. It is known that AF is an arrhythmia characterized by disorganized atrial electrical activation, being the most usual sustained arrhythmia at world level, and associated to a significant morbidity-mortality.[2] AF could be asymptomatic (subclinical), manifest episodes of arrhythmias (paroxysms) and finally be permanent (previously called chronic). At times, the first clinical manifestation could be stroke.[3] Therefore, it is important to identify patients with a greater tendency to develop AF, and for this reason, there are different screening strategies. AF could be viewed from two angles: as a disease per se, or as a symptom of another disease.[2] For example for the second view, AF is frequently found in acute pulmonary thromboembolism (PTE), where it is considered secondary to right atrium dilatation caused by hemodynamic alterations of pulmonary artery occlusion. Recently, the presence of atrial cardiomyopathy has been studied as a background for AF, mainly in cases where no direct cause for AF can be found (PTE, hyperthyroidism, acute ischemic heart disease, etc.).[4]

In terms of the pathophysiology of AF and its association with respiratory disorders, not considering the hemodynamic ones that have been described above for PTE, the proposed mechanisms include an elevated inflammatory activity, endothelial dysfunction and sympathetic activation; mechanisms mainly studied in sleep apnea.[5] In the studies made in several laboratories, including ours, patients with sleep apnea were detected to possibly generate AF by common pathophysiological mechanisms, like increased sympathetic tone, intermittent hypoxemia, systemic inflammation and endothelial dysfunction.[5] However, more recently, other respiratory diseases, such as asthma, are starting to also show an association with AF.

The association between AF and asthma is bidirectional. On one hand, evidence suggests that AF shares a background of systemic inflammation with asthma, and on the other, treatments for asthma, such as β2-adrenergic agonists, have been associated with a greater risk of arrhythmias.[6] Although the main mechanism of action of β2-adrenergic agonists, such as salbutamol, is their bronchodilator effect,

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they also have cardiovascular, uterine, metabolic and neurological effects.[7] In regards to the heart, studies in asymptomatic patients show that salbutamol, administered by inhalation or orally, is associated with an increase in heart rate of 23 and 28%, respectively.[7]

Thus, asthma (as it is a systemic autoinflammatory disease, as the investigators of this publication point out) could be affecting the atria, and thus, favor the emergence of atrial cardiomyopathy, which would predispose to the appearance of AF. On the other hand, plenty of AF episodes in asthmatic patients could be the consequence of using bronchodilators with sympathomimetic properties (β2-adrenergic agonists), as with patients who already have supraventricular tachycardia.[8] In this case, using this drugs in patients with the abovementioned substrate would make them act as AF "triggers", through an increase in sympathetic tone.

In asthma, inflammation is mediated by numerous pathways. In the eicosanoid pathway, arachidonic acid is catalyzed by 5-lipoxygenase in leukotrienes, which are strong paracrine inflammatory substances, mainly formed in leukocytes. Leukotrienes mediate bronchoconstriction, mucus secretion and local edema in bronchioles. Leukotrienes has systemic effects; leukotriene receptors largely express in the heart and are intrinsically associated with the conduction system.[9]

Different inflammatory cytokines mediate ion channel function and atrial remodeling. Inflammation mediators have been described, such as C-reactive protein and interleukine-6, elevated in patients with AF, and their influence has even been demonstrated in AF ablation success.[6]

Tattersall et al. in 2020 raised the hypothesis that persistent asthmatic patients present a greater risk of developing AF, and that this association would be attenuated by adjusting the baseline markers of systemic inflammation. In the MESA *(Multi-Ethnic Study of Atherosclerosis)*, a prospective longitudinal study in adults with no cardiovascular disease at the onset of the study, with a multi-ethnic cohort of 6615 participants with almost 13 years of follow-up, the aim was detecting the appearance of AF in these patients. They concluded that persistent asthma was associated with a 1.5 times increase in incident AF risk, that this association was not attenuated when adjusting baseline inflammatory biomarkers, and that persistent asthma could be associated to a higher relative risk of AF in women.[9]

Given the high prevalence of undiagnosed AF, which in turn may lead to severe consequences, such as thromboembolic events, heart failure and progression of AF, it is important to identify populations with a greater risk of developing AF, to make a diagnosis or start an early treatment of AF, and act timely on risk factors such as asthma, obesity and obstructive sleep apnea. Equally, in this association, it has been proven that treating obesity by weight-loss programs and treating sleep apnea in asthmatic patients improves the symptoms of asthma.

Current guidelines do not contemplate this association (AF and asthma), but it is evident that both pathologies share pathophysiological mechanisms, mainly systemic inflammation. When reviewing the 2020 and 2024 ESC guidelines on AF, between the risk factors and co-morbidities, COPD (chronic obstructive pulmonary disease) and sleep apnea are included, but asthma is not considered.[10,11] The 2025 GINA guideline *(Global Initiative for Asthma)* suggests managing multi-morbidity, including co-morbidities like rhinosinusitis, obesity, anxiety/depression and gastroesophageal reflux, which may contribute to respiratory symptoms, and even contribute with a poor control of asthma, but do not take into account cardiovascular pathologies, among them AF.[12]

Our reflection, due to the prevalence of asthma and AF still increasing, is to consider this association, to seek it intentionally, and simultaneously, to carry out more investigations on the possible mechanisms of this association.

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