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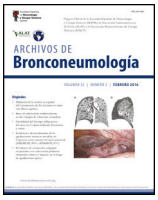

Editorial

Relationship between SAHS and cardiac arrhythmias☆

Relación entre el SAHS y las arritmias cardiacas

Cardiac arrhythmias are a major health problem in our setting, particularly atrial fibrillation (AF), which is the most common form. In individuals over 40 years of age, the prevalence is higher than 4% in both sexes[1] and the aging of the population is expected to cause increases in the next few years that may reach epidemic levels. Arrythmias reduce quality of life and generate high social health costs.[2]

Current studies show that the onset of AF is related more to cardiovascular risk factors that are closely linked to lifestyle than to the presence of established structural heart disease. Thus, factors such as obesity, sedentary lifestyle, hypertension, diabetes, and sleep apnea and hypopnea syndrome (SAHS) have been described as risk factors for developing AF.

This editorial aims to set out the basic principles that prioritize the diagnosis and treatment of SAHS in patients with arrhythmias.

SAHS and atrial fibrillation

SAHS and AF share risk factors, such as increased age, obesity, heart failure, and hypertension,[3] so it is not unusual to find a close relationship between the two diseases.

SAHS is more common in patients with AF (18%–74%) than in patients without AF (3%–49%).[4] Furthermore, in patients under 65 years of age, sleep-disordered breathing confers the highest risk for AF (HR 3.3).[5]

The presence of SAHS is also a risk factor for recurrence of AF after electrical ablation[6] or cardioversion.[7] Treating SAHS with CPAP can normalize this risk.[8]

The main mechanisms by which SAHS may favor the development of AF are intermittent nocturnal hypoxemia, sympathetic activation, transmural pressure changes, and systemic inflammation. These factors contribute to atrial remodeling, both structural and electrical, that makes the atrial tissue more susceptible to the genesis and perpetuation of AF.[9]

In short, SAHS increases the risk of AF and is a poor prognostic factor in the outcome of AF treatment. In these patients, it is important to detect and treat SAHS because CPAP treatment normalizes this risk.

SAHS and atrial flutter

AF-free atrial flutter is characterized by less unstructured atrial tissue and less fibrosis, so successful preventive treatment is more likely. In this context, it makes sense to explore a possible etiopathogenic relationship between SAHS and atrial flutter since treatment of SAHS with CPAP may also be effective in preventing progression from flutter to AF.

Increased pulmonary artery pressure increases the risk of AF and flutter and may be the mechanism by which SAHS causes the appearance of these arrhythmias. It is known that during obstructive episodes of the upper airway, the right atrial and ventricular afterload is raised by a paroxysmal increase in pressure in the pulmonary artery.

There is little evidence of the relationship between the two entities, and for this reason our group studied a prospective series of 56 patients with flutter who underwent radiofrequency ablation. In this series, 82% had SAHS and 45% had severe SAHS. In follow-up after ablation, 38% of patients presented AF. In the multivariate analysis, the absence of paroxysmal AF episodes prior to ablation and CPAP therapy in patients with SAHS were the only protective factors. Thus, only 6% of the group of SAHS patients treated with CPAP developed AF. The protective effect of CPAP was only observed in patients with no previous episodes of AF, suggesting that we can only delay the progress of less fibrotic atria.[10]

These findings are relevant, as many patients with flutter develop AF a few years after successful AF ablation. Diagnosing and treating SAHS in patients with no previous history of AF may delay or prevent the onset of AF and its impact on morbidity and mortality.

SAHS and ventricular tachycardia and sudden cardiac death

SAHS has also been associated with ventricular arrhythmias. Up to 60% of these patients have SAHS and 22% have severe SAHS.[11] Factors involved in this relationship are ventricular hypertrophy, QT interval prolongation, and autonomic dysfunction in patients with SAHS.[12]

Gami et al. demonstrated that sudden cardiac death in patients with SAHS peaks during sleep, while in the population without SAHS it occurs during the day.[13] This finding was subsequently confirmed in a 15-year longitudinal study that defined age >60 years, low nocturnal oxygen saturation, and apnea-hypopnea index (AHI) >20 as risk factors for sudden death.[14] Treatment with CPAP has been shown to protect against these serious cardiovascular events.[15]

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Future challenges

1 A randomized, interventional trial is still needed to demonstrate a causal relationship between SAHS and cardiac arrhythmias and to demonstrate reduced arrhythmic load by treating SAHS.
2 Do we have to act sooner? Specifically in moderate SAHS (AHI 15–30), to avoid structural remodeling of the atrium.
3 The medical community has conventionally seen SAHS as a problem for pulmonologists, but given an aging population, the obesity pandemic, and the lifestyle of Western countries, it is important that other specialists work together to reduce its consequences. Cardiologists and internists should be aware of the close relationship between cardiac arrhythmias and SAHS and the benefits of early diagnosis and treatment of SAHS in reducing cardiovascular morbidity and mortality. This is especially important in patients who need to undergo cardiac ablation or cardioversion.

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