

The Link Between Atrial Fibrillation and Obesity

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Introduction

The obese population is currently increasing. Atrial fibrillation (AF) is the most common supraventricular arrhythmia. There is a close link between these two pathologies that we will detail in this article.

Epidemiology

The World Health Organization estimates that cases of obesity have tripled since 1975 with 39% of overweight adults and 13% of obese adults in 2016. Obesity is an independent risk factor of AF and those in spite of other associated comorbidity (sleep apnea, arterial hypertension, physical inactivity, diabetes mellitus ...) [1-4] The increase in the body mass index (BMI) is associated with an increase in the incidence of AF. Conversely, the standardization of BMI is associated with a decrease in the incidence of AF. [2] It is estimated that one in five of AF may be due to being overweight or obesity [5].

Physiopathology

Structural remodeling (atrial enlargement, increased fat depots, interstitial fibrosis, increased of left ventricular geometric abnormalities, altered hemodynamics) is a major factor by which obesity promotes AF. [6] Associated with oxidative stress, these two mechanisms are the main cause of AF in the obese patient. We will not discuss the particular pathophysiology of diabetes that is often associated with obesity in this population. Atrial electrical remodeling is a cause and consequence of AF. At the tissue level, electrical remodeling comprises effective refractory period shortening, conduction velocity slowing, wavelength reduction, and frequent atrial ectopic caused by calcium (Ca^{2+})-dependent triggered activity [7,8]. While Ca^{2+} mediated triggers serve as the critical initiators of AF, an appropriate substrate formed through progressive electrical and structural remodeling of the atria is required for the long-term perpetuation of the arrhythmia and its conversion from paroxysmal to chronic forms.

Pericardial fat volume which correlates with left atrial enlargement has been associated with increased risk of AF [9]. It expresses anti-inflammatory adipokines and pro-inflammatory cytokines (including interleukin (IL)-1 β , IL-6, IL-8, TNF- α). Reversal of atrial fat by weight loss is associated with reduced AF burden [9-11]. Other complications of obesity also favor the occurrence of AF. The main ones are developed heart disease, arterial hypertension, coronary disease, sleep apnea, diabetes and metabolic syndromes [12].

Management Issues Specific

The management of atrial fibrillation in obese patients begins with the global management of obesity. The weight loss is directly related to the dimming of AF. The management of its complications, such as sleep apnea syndromes, the treatment of arterial hypertension, the management of diabetes, and the development of cardiopathy can reduce prevail and recurrence of the AF. The management of AF in obese patients has a peculiarity that we will highlight in the following paragraph.

Special Feature of Anticoagulation

Of all the molecules used in the AF of the obese, a lot of study data are missing and requires more documentation. The historical treatment with anti-vitamin K is particular in the obese patient. It has been proven that there is a need for a larger dose of warfarin (for example) and that delay between initiation of treatment and steady state takes longer. [13] With regard to the new direct oral anticoagulant, the data is limited.

For example, the FDA conducted an analysis that compared the rates of stroke and systemic embolism (SSE) among rivaroxaban and warfarin patients from ROCKET AF across 5 weight categories (<50 kg, 50- \leq 70 kg, 70- \leq 90 kg, 90- \leq 110 kg, and >100 kg). [14] There was no statistical relationship between weight category and the rate of SSE. There are no published pharmacokinetic or clinical

analyses of dabigatran in patients with extreme weights extremes. A recent study on apixaban seems to show that, compared to warfarin in obese or overweight patients, there is less SSE without risk of bleeding [15].

Heart Rhythm Control or Rate Control

The difficulty of maintaining the heart rate under medical treatment is truer in this population. For example, it has been shown that clearance of amiodarone was significantly reduced in those with BMI >25 kg/m². [16] Higher shock energy was needed for increased first-shock success when transthoracic direct current cardioversion was studied in overweight and obese individuals. [17] However, the ablation is less effective in the obese. An index BMI ≥30 kg/m² led to a 1.2-fold increased likelihood of experiencing recurrent AF at 12-months follow-up as compared with overweight patients (HR 1.223; 95% CI 1.047 to 1.429; p=0.011), while no significant correlation was found between overweight and normal BMI groups (HR 0.954; 95% CI 0.798 to 1.140; p=0.605) and obese versus normal BMI (HR 1.16; 95% CI 0.965 to 1.412; p=0.112) [18].

The surgical approach seems interesting but remains to be confirmed. A meta-analysis of 6 studies of patients undergoing coronary artery bypass surgery suggested that posterior pericardiectomy significantly reduced the incidence of post-operative AF. [19] More recently, a study assessing injections of botulinum toxin into the epicardial fat pad reduced the recurrence rate of AF after cardiac surgery. [20] Ablation of atrial fibrillation alone by thoracotomy seems limited by the risk of surgery, especially by ventilatory problems. Obese patients undergoing rate control strategy were more likely to have higher and uncontrolled resting heart rate as compared with their leaner counterparts [21].

Conclusion

The management of AF in the obese patient is complex and justifies multidisciplinary and comprehensive management. The central point remains the prevention and early management of obesity.

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