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Risk Factors for Atrial Fibrillation (Literature Review)

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Abstract: Atrial fibrillation is one of the most common cardiac arrhythmias, causing dangerous complications. This article reviews the main risk factors associated with the development of atrial fibrillation, including hypertension, diabetes, smoking, obesity, and metabolic syndrome. Analysis of modifiable risk factors can contribute to the development of preventive measures aimed at reducing the incidence of this arrhythmia.

Keywords: atrial fibrillation, hypertension, diabetes, smoking, obesity and metabolic syndrome.

Introduction

Atrial fibrillation (AF) is the most common cardiac arrhythmia disorder among adults in the world. It is interesting to note that the incidence of this condition increases by 15–20% in individuals over the age of 80 years. By 2050, the number of patients with AF is expected to increase dramatically, which in turn will increase healthcare system costs due to the need for continuous monitoring and pharmacological treatment. Identifying the mechanisms underlying the initiation and maintenance of AF can be the first step towards the development of more effective medical, surgical and interventional approaches to treatment [1].

Purpose of the study

Study of some risk factors for the development of atrial fibrillation based on the results of clinical studies.

Results and analysis

Arterial hypertension. Due to chronic arterial hypertension, documented changes have been observed, such as increased atrial size and the development of fibrosis, as well as decreased connexin expression [7–9]. These physiological changes entail modifications in the electrophysiological characteristics of the myocardium and disruption of the normal functioning of calcium ions [10].

Together, these mechanisms cause conduction abnormalities that contribute to the reentry mechanism and are associated with more frequent and prolonged episodes of atrial fibrillation [8,12,13].

Structural remodeling in chronic hypertension, in particular, is partly explained by activation of the renin-angiotensin-aldosterone system (RAAS) due to increased atrial pressure [7, 11,18].

Hypertension can lead to coronary heart disease (CHD) and myocardial infarction (MI), with a subsequent increased risk for AF. For primary prevention of AF in hypertensive patients, the optimal target systolic blood pressure during treatment should be less than 130 mm Hg. Art. [1].

Hypertension is a significant risk factor for stroke in AF. Uncontrolled high blood pressure increases the likelihood of stroke and hemorrhagic complications, and can lead to relapses of arrhythmia. In summary, hypertension-induced atrial remodeling occurs rapidly, and over time, a variety of mechanisms are involved in this process, promoting the development of the reentry mechanism, and careful control of blood pressure should be an integral part of the treatment of patients with AF.

Diabetes mellitus (DM). In recent years, diabetes and elevated blood glucose levels have been potential risk factors for the development of atrial fibrillation (AF). The results of numerous studies [1,5] indicate an increase in the incidence of AF in patients with diabetes mellitus (DM). An analysis of 7 cohort studies and 4 case-control studies involving more than 1,600,000 people by Huxley et al. [1], found that patients with diabetes had a 39% greater risk of developing AF compared with those without diabetes. In addition, a relationship has been identified between the duration of diabetes and an increased risk of developing AF [5]. For each year of diabetes, the risk of AF increased by 3%, and this risk was also correlated with worse glycemic control. Close glycemic control, including measurement of glycated hemoglobin, was associated with a lower risk of developing AF. Elevated levels of glycated hemoglobin, body mass index (BMI), and older age increased the risk of recurrent AF after catheter ablation in patients with diabetes [1,3,4].

Data also indicate an effect of metformin on the development of AF [4,7,14]. A study of 645,710 diabetic patients taking metformin and excluding other diabetic medications found a reduction in the incidence of AF in patients using metformin over a 13-year period. These data support the suggestion that metformin may have a protective effect on the development of AF in patients with diabetes. Data also indicate an effect of metformin on the development of AF [4,7,14]. A study of 645,710 diabetic patients taking metformin and excluding other diabetic medications found a reduction in the incidence of AF in patients using metformin over a 13-year period. These data support the suggestion that metformin may have a protective effect on the development of AF in patients with diabetes.

Smoking. Literature suggests that smoking is a risk factor for the development of atrial fibrillation (AF). The risk of developing AF is the same in both male and female smokers [1]. It is emphasized that both ever smokers and active smokers are at higher risk of developing AF compared to non-smokers. Active smokers have an approximately 10% risk of developing AF. Some studies demonstrate a dose-dependent effect, with the highest risk of AF in people with a long history of smoking and in smokers with a higher number of cigarettes smoked per day [15,24]. There is also evidence indicating an increased risk of developing AF in adults exposed to secondhand smoke in utero or childhood [1].

Experimental and clinical studies have discussed several mechanisms responsible for the increased risk of developing AF with smoking. Nicotine and cigarettes predispose to inflammation, changes in atrial electrical activity, atrial fibrosis, decreased pulmonary function, as well as coronary heart disease and ischemic myocardial failure [15]. These factors, in turn, may contribute to the development of AF. Smoking also increases the risk of stroke, intracranial hemorrhage, and death in patients with AF [1,18,19]. Research indicates stronger associations between smoking and AF in current smokers compared to former smokers [1,25]. There was also a decrease in the incidence of AF in people with coronary heart disease and heart failure who stopped smoking. A reduction in the risk of AF has also been demonstrated after catheter ablation with smoking cessation [10,16,23].

Obesity and metabolic syndrome. Existing evidence suggests that obesity may contribute to the development of atrial fibrillation (AF) through several mechanisms, including inflammatory

changes, epicardial adipose tissue (EAT) biology, cardiac structural remodeling, and atrial fibrosis [12,13,22]. Obesity and metabolic syndrome. Existing evidence suggests that obesity may contribute to the development of atrial fibrillation (AF) through several mechanisms, including inflammatory changes, epicardial adipose tissue (EAT) biology, cardiac structural remodeling, and atrial fibrosis [12,13,21]. Genetic and environmental factors can lead to changes in EAT characterized by a proinflammatory and profibrotic phenotype. Due to its close location to the coronary arteries, thickened EAT actively promotes regional processes of atrial remodeling. In addition to classical paracrine transmission, EAT can directly release transmitters into the walls of the coronary arteries using a mechanism known as vasocrine. In addition, infiltration of adipocytes into the atrial myocardium can also cause disorganization of the depolarization wave front, which contributes to the microreentry mechanism and local block of electrical impulse conduction. Epicardial mapping in patients undergoing cardiac surgery has also shown a higher incidence of conduction abnormalities in obese patients compared with non-obese patients [2,17,20].

Conclusion. Thus, the data presented above indicate the presence of many risk factors for the development of AF. Prevention of this arrhythmia requires an individual approach. Many of the risk factors for AF can be prevented and/or modified through lifestyle changes and adequate treatment of concomitant diseases. Modification of unhealthy diet, cessation of smoking, alcohol consumption, daily physical activity should be under strict control of both the patient and the doctor. General practitioners and local therapists play a major role in improving the educational status of patients in this aspect and adherence to prevention. Careful monitoring of patients is necessary. Particular attention should be paid to adolescents and young adults, who may be at risk of heart disease due to the prevalence of obesity, poor diet, smoking and alcohol abuse, and a sedentary lifestyle. Therefore, a lifelong approach to modifying the risk of CVD, including AF, is required, subject to close doctor-patient cooperation.

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