

Effect of obesity on atrial fibrillation

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Abstract

Obesity is a chronic civilisation disease with multifactorial aetiology that contributes to dysfunction of the entire organism, especially circulatory system diseases. The impact of obesity on the circulatory system is related to haemodynamic, inflammatory, and toxic effects of adipose tissue on the electromechanical activity of the heart. The association between obesity and atrial fibrillation (AF) has gained increasing scientific attention. Obesity has been proven to be an independent risk factor for AF. Haemodynamic, inflammatory, and toxic mechanisms contribute to decreased conduction velocity, and electrical remodeling, which results in alteration of the expression of ion channels, resulting in decreased atrial effective refractory period. Obesity affects the incidence and treatment in AF patients. Changes in eating habits and sufficient physical activity, as well as bariatric surgery, should be part of therapy because it effectively reduces body weight, resulting in the reversal of structural changes in the heart and reducing the risk of AF.

Introduction

Obesity is a chronic disease of civilisation with a multifactorial aetiology. The prevalence is increasing year by year, with the problem now affecting more than one billion people [1]. Obesity contributes to whole-body dysfunction, hepatic steatosis, diabetes, chronic kidney disease, metabolic syndrome, and particularly cardiovascular disease [1]. As the degree of obesity increases, the mortality rate also increases [2]. The degree of obesity can be assessed by body mass index (BMI), waist circumference measurement, waist-to-hip circumference ratio, skinfold thickness, and percentage of body fat [2, 3].

The incidence of atrial fibrillation (AF) is on an upward trend. More than 33 million people worldwide suffer from AF [4]. About 5 million new cases are diagnosed annually [4]. The risk of AF in people > 55 years of age is 37% [4]. AF increases the risk of stroke, dementia, heart failure, and death. The annual risk of stroke in these patients ranges from 0.5% to 9.3%. The burden of AF is high regardless of race and gender [2, 4].

Risk factors for AF include age, genetic factors, obesity, physical inactivity, sleep apnoea, hypertension, coronary artery disease, heart failure, and alcohol and cigarette addiction [4]. Moreover, obesity is the second most common risk factor after hypertension [3]. Recently, there has been increasing interest in the relationship between obesity and the incidence of AF.

Aim of the research

The purpose of this article is to review the literature and present the correlation between the prevalence

of obesity and the incidence of atrial fibrillation, and clarify the pathomechanisms involved. The article also aims to discuss the impact of obesity on the treatment of AF and the benefits of weight loss in patients with already diagnosed AF.

Methods

A review of recent scientific studies available in the PubMed and EBSCO databases was conducted. The key words that were used for the search were 'obesity and atrial fibrillation'. The search results were exported to Rayyan. First, the articles were checked by reviewing the titles and abstracts of the papers according to the inclusion and exclusion criteria and relevance in this work. Concerns about the inclusion of an article in a paper were resolved during the discussion of the paper's authors. Then the qualified scientific articles were again thoroughly reviewed. Inclusion criteria were randomised control trials, original research, systematic reviews, reviews, subject age over 18 years, transparent methodology, open access, scientific articles not older than 7 years. Excluded were articles that did not contain an English language version, studies without open access, and studies older than 7 years. After searching on PubMed and EBSCO, a total of 6446 scientific articles were identified. After excluding duplicate papers, 1492 articles remained. Subsequently, after a review based on inclusion and exclusion criteria for titles and abstracts, 1221 were excluded. After a thorough review of all articles, 30 studies were finally included in the paper. This review includes information also from European So-

ciety of Cardiology and American Heart Association guidelines.

Pathomechanism of AF development in obesity

Obesity causes increased arterial filling pressures and an increase in left atrial and left ventricular pressures. The haemodynamic effects of obesity are associated with an increase in the load on the left atrium and left ventricle, due to increased blood volume. To maintain greater cardiac output, the left ventricle dilates, and eccentric or concentric hypertrophy of myocytes occurs [3, 5]. Obese individuals have been shown to have an enlarged left atrium despite the absence of cardiovascular disease [6]. A study by Lim *et al.* included 120 patients with a mean age of 54.9 ± 11 years with persistent AF from the onset of the disease and 231 patients with a mean age of 57.2 ± 10.1 years who had progression from paroxysmal to persistent AF. By comparing the 2 groups, it was noted that the obese patients had larger dimensions of both the right and left atrium [6]. It has also been shown that obstructive sleep apnoea often affects obese individuals, which, combined with autonomic nervous system dysfunction, also predisposes to the development of AF. Magnetic resonance imaging is used to determine the volume of epicardial adipose tissue. Studies have shown that patients with persistent AF have more of it compared to patients with paroxysmal AF [6].

Epicardial adipose tissue produces pro-inflammatory adipocytokines and secretes profibrotic factors, i.e., adipo-fibrokinases, which induce the deposition of collagenous tissue in the myocardium with the help of myofibroblasts. The concentration of interleukin-6 (IL-6) and tumour necrosis factor- α (TNF- α) increases, contributing to local inflammation and fibrosis. Antibody production by B lymphocytes is stimulated by protein IL-6. IL-6 initiates the transformation of type 17 effector lymphocytes, which leads to myocardial fibrosis due to an increase in IL-17 [5, 7]. In addition, IL-6, during the acute phase of inflammation, affects the gene responsible for the transcription of C-reactive protein (CRP), a protein synthesised by the liver in response to systemic inflammation, which contributes to the increase of this protein in inflammation [8]. TNF- α is a protein secreted by macrophages and monocytes, among others, that initiates the inflammatory response and contributes to the dilatation of blood vessels [9, 10]. Inflammation and the associated increase in CRP, IL-6, and TNF- α cause activation of the renin-angiotensin-aldosterone system (RAAS), which in turn leads to the development of hypertension, which is also a risk factor for atrial fibrillation [6]. Fibrosis, autonomic nervous system dysfunction, and structural remodeling of the left atrium are important factors in the development of an arrhythmogenic substrate that results in the development of AF [3]. In contrast, AF contributes

to inflammation, oxidative stress, and mitochondrial dysfunction, a self-perpetuating vicious circle [1].

Local and systemic effects of adipose tissue

The main role in the contraction and diastole of the cardiomyocyte is played by calcium ions. They are accumulated in the endoplasmic reticulum, which contains Ca^{2+} ATP-ase 2a (SER- Ca^{2+}) responsible for releasing calcium from the sarcoplasmic reticulum needed for cardiomyocyte contraction and re-storing it in the sarcoplasmic reticulum during the diastolic phase of cardiac muscle. In myocardial diseases, there is often reduced expression of SER- Ca^{2+} , which impairs the regulation of calcium concentration in the cardiomyocyte and impairs its contraction and diastole. As a result of excess adipose tissue, an increase in TNF- α reduces SER- Ca^{2+} expression, resulting in delayed afterdepolarisation in cardiomyocytes [1, 11]. Other local effects of the presence of excess adipose tissue include structural remodeling, i.e. inflammation, fibrosis, oxidative stress, and apoptosis of cardiomyocytes – resulting in decreased conduction velocity, and electrical remodeling, which results in decreased expression sodium channels and L-type calcium channels and increased expression potassium channels, in turn resulting in decreased atrial effective refractory period (AERP) [1]. Electromechanical delay measured by tissue Doppler imaging (TDI) in the left atrium as well as between the atria is prolonged in the obese population compared to normal-weight subjects. TDI allows assessment of electrical events in different regions with high temporal resolution and measurement of atrial electromechanical delay from the beginning of the P wave on the ECG to the onset of atrial contraction [6]. There was also a correlation between interatrial electromechanical delay and highly sensitive CRP (hsCRP) levels, demonstrating how the inflammation has a significant impact on atrial fibrillation. Nullifying inflammation may help reverse or prevent AF [6]. Systemic effects of obesity include insulin resistance, autonomic nervous system dysfunction, and cardiac infiltration. All these factors contribute to the development of AF [1].

Inflammatory markers associated with atrial fibrillation and obesity

As mentioned above, epicardial adipose tissue is a source of increased production of inflammatory cytokines (IL-6, TNF- α), which adversely affect myocardial tissue remodeling. IL-6, which is increased in patients with atrial fibrillation and metabolic syndrome, is correlated with right and left atrial volumes and with epicardial fat thickness [1]. Similarly, TNF- α and CRP, which are correlated with epicardial adipose tissue thickness, increase in patients with atrial fibrillation and metabolic syndrome [1]. Obese patients with paroxysmal atrial fibrillation have significantly higher

levels of MMP-9. MMP-9 is a matrix metalloproteinase whose expression increases under the influence of pro-inflammatory factors, resulting in an increase under the influence of pro-inflammatory factors, resulting in an increase in collagen deposition in the cardiac matrix, and is positively correlated with left atrial volume. MMP-9 levels are significantly higher in patients with obesity and paroxysmal atrial fibrillation [1].

Obesity and atrial fibrillation

Obesity is an important factor for atrial fibrillation that certainly needs to be modified. The risk of atrial fibrillation in obese individuals has most often been attributed to cardiovascular risk factors, i.e. type 2 diabetes, hypertension, and dyslipidaemia [3, 12]. However, a study was conducted that divided obese people into those with serious comorbidities and obese people without accompanying metabolic disease. It showed that both groups had an increased risk of AF, proving that obesity is an independent risk factor for the development of AF [3]. Previous studies confirm an association between the presence of obesity and an increase in risk [3, 4, 13]. Obesity has been shown to increase AF risk 1.5-fold, with a 4% increase in AF risk per 1 unit increase in BMI > 25 kg/m² [4]. In addition, atrial fibrillation incidents increase by 29% for every 5-unit increase in BMI. For every 5-unit increase in BMI, the risk of post-operative AF increases by 10%, and post-ablation AF increases by 13% [4]. Obesity has also been shown to predispose to progression of paroxysmal AF to persistent [4].

Atrial fibrillation in obese patients and medical treatments and procedures

If pharmacological treatment of atrial fibrillation is ineffective or intolerable, the European Society of Cardiology recommends percutaneous ablation. During ablation, patients are not only subjected to X-rays during the procedure but also a pre-procedure computed tomography (CT) scan. However, this procedure in obese patients is associated with several difficulties, i.e. in obese patients it is often impossible to insert catheters into the femoral vein, the radiation dose required for this procedure is significantly higher than in normal-weight patients, and there is a higher rate of postoperative complications. Previous studies have shown that obese patients receive a 75% higher radiation dose compared to normal-weight patients [14, 15].

A study in the Korean population evaluated the effect of abdominal obesity (defined as waist circumference ≥ 90 cm in men and ≥ 85 cm in women) on the long-term efficacy and safety of catheter ablation of atrial fibrillation. The study included 5397 patients, 1759 of whom presented with abdominal obesity. Abdominal obesity was shown to be associated with

higher AF recurrence rates at 3 and 6 years, with no significant statistical difference between the two groups 1 year after ablation [16]. In addition, in obese patients, Kaplan-Meier analysis based on body mass index and waist circumference showed a significantly higher rate of AF recurrence. Patients with abdominal obesity are also at risk for an increased incidence of ischemic stroke, as observed at 3- and 6-year follow-up, and intracranial haemorrhage. In contrast, total perioperative complications were not associated with abdominal obesity [16].

A study conducted by Serban *et al.* in a population of patients undergoing cardiac surgery for coronary artery disease or valvular heart disease proved that the median duration and number of prolonged episodes of atrial fibrillation were higher in patients with obesity compared to those without obesity. It was also found that obesity predisposed to a higher burden of atrial fibrillation in the early postoperative period, especially on day 3 [17].

Effects of weight reduction on risk of atrial fibrillation

Abed *et al.* conducted a study of overweight and obese patients with atrial fibrillation. During this study, which lasted 15 months, patients changed their lifestyles and had their weight controlled. The effects of the changes were evaluated every month by observing the severity of AF symptoms. The group that received the weight control intervention had a reduction in AF severity, as well as positive changes in heart structure [18, 19]. Weight reduction has been shown to be associated with a reduction in AF symptoms, and there was a reduction in ventricular septal thickness and left atrial area [20].

A total of 355 patients were enrolled in the LEGACY study. They were then divided into 3 groups according to percentage weight loss. Group 1 lost < 3% of body weight, group 2 from 3% to 9%, and group 3 $\geq 10\%$. In group 1, 26% of patients had a change from persistent to paroxysmal atrial fibrillation or the atrial fibrillation stopped. In group 2, 49% of patients had the changes described above, and in group 3, 88% had remission of the disease. The above data prove the dynamic relationship between weight loss and atrial fibrillation remission. This study clearly shows that the greater the weight reduction, the more patients experience a change from persistent to paroxysmal atrial fibrillation or remission of atrial fibrillation. Those with weight loss > 10% had a sixfold greater chance of arrhythmia-free survival compared to the other 2 groups [18, 21].

Mohanty *et al.* examined the effect of weight loss on long-term persistent AF treated with catheter ablation. The 58 participants in the study received the intervention, which consisted of moderate physical activity of 150 minutes per week and an introduced

diet. In contrast, 32 participants were in the control group. Weight loss was shown to lead on an improved quality of life, while it had no effect on the severity of symptoms or the long-term outcome of the ablation procedure [18, 22].

The SORT-AF study focused on evaluating the impact of weight reduction on AF ablation outcomes. Weight reduction was made possible by dietary counseling and physical training. The 133 patients with symptomatic atrial fibrillation, post-AF ablation, and a BMI between 30 and 40 kg/m² were randomly divided into a primary care or weight reduction group. Weight reduction in patients with persistent AF has been shown to reduce the rate of AF recurrence after ablation [18, 23].

Physical activity and atrial fibrillation

A study of 120 patients with paroxysmal or persistent symptomatic atrial fibrillation showed that after 6 months of aerobic exercise, the severity of atrial fibrillation symptoms was lower in the exercise group compared with the control group. Moreover, the differences between the 2 groups persisted beyond 12 months. Resolution of atrial fibrillation was observed in 40% of patients in the exercise group and in 20% in the control group. In contrast, changes in cardiac structure or function, BMI, or blood pressure were not observed in either group [24]. However, attention should be paid to exercise intensity, as chronic high-intensity endurance exercise increases the risk of atrial fibrillation [20, 25]. Physical activity applied before surgery also contributes to reducing the risk of atrial fibrillation in the postoperative period. [26].

Bariatric surgery as a prelude to treatment

In obese patients, the effectiveness of atrial fibrillation treatment with current therapies may not be reduced. Satisfactory results in these patients can be helped by bariatric surgery. Bariatric surgery is applicable to patients with a BMI ≥ 40 kg/m² or ≥ 35 kg/m² if they have high-risk comorbidities, i.e. cardiomyopathy, asthma, diabetes type 2, obstructive sleep apnoea, and musculoskeletal diseases. It is also acceptable to perform this surgery in patients with a BMI of 30–34.9 kg/m² if there is coexisting type 2 diabetes unresponsive to treatment with insulin and oral medications. Bariatric surgery has been proven to effectively reduce body weight, resulting in reversal of structural changes of the heart, i.e. left atrial dilatation. Patients who underwent such therapy had a 29% reduced risk of atrial fibrillation, reduced risk of hospitalisation due to AF or heart failure, reduced cardiovascular risk, and reduced inflammatory markers and blood pressure [14, 27]. For non-valvular atrial fibrillation, patients who have undergone bariatric surgery do not have an increased risk of bleeding or thrombosis from oral anticoagulants. The preferred drug in bariatric patients is warfarin, due to more studies confirming

its safety. Oral anticoagulants (DOACs) can be used as an alternative to warfarin if warfarin is ineffective or not tolerated [28, 29].

The study by Jamaly *et al.* included 4021 obese patients who had never previously suffered from atrial fibrillation or other cardiac arrhythmias. They found that patients who underwent bariatric surgery had a 29% lower risk of a first diagnosis of atrial fibrillation compared to controls [18, 30].

The study by Donnellan *et al.* included 220 patients with giant obesity (BMI > 40 kg/m²) who were followed up before and after bariatric surgery. The greatest effectiveness in reversing the type of AF from persistent to paroxysmal occurred in gastric bypass patients, in up to 71% of patients. Sleeve gastrectomy and gastric banding were less effective, as they led to the reversal of AF type from persistent to paroxysmal in 56% of patients and 50% of patients, respectively. Analysis showed a significant association between percent weight loss and AF type reversal [18, 27].

Screening tests

Screening for atrial fibrillation in obese patients, especially in the general population, is a controversial topic. The European Society of Cardiology recommends performing an electrocardiogram or palpation test for atrial fibrillation in patients > 65 years of age [29, 31]. U.S. guidelines do not recommend screening for early detection of atrial fibrillation [32, 33].

Effect of intermittent fasting on atrial fibrillation

Patients with obesity often suffer from insulin resistance, which is a factor in the development of atrial fibrillation. There have been studies that have proven the effectiveness of intermittent fasting in people with insulin resistance, but so far it has not been confirmed whether intermittent fasting reverses atrial fibrillation. A study in mice documented that intermittent fasting reversed atrial remodeling, including left atrial dilatation, cardiac hypertrophy, and fibrosis, independent of weight loss [34]. SIRT3 is a protein (sirtuin 3) that causes mitochondrial fusion in mice having a high-fat diet. Intermittent fasting improves mitochondrial function and fusion in mouse white adipose tissue [35]. However, the effect of intermittent fasting was strongly dependent on the amount of SIRT3 protein. Both pharmacological inhibition of SIRT3 and genetic suppression of this protein resulted in reduced benefits associated with intermittent fasting. Because of this, lifestyle modification in the form of intermittent fasting improves the treatment of atrial fibrillation [34].

Conclusions

Obesity is a chronic civilisation disease with a multifactorial aetiology that is the second most common

risk factor for the development of atrial fibrillation. Epicardial adipose tissue stimulates the production of pro-inflammatory cytokines, contributing to local myocarditis, myocardial fibrosis, and dilatation of both the left and right atria. Local myocardial inflammation impairs the expression of the calcium pump and sodium and potassium channels, resulting in delayed afterdepolarisation in cardiomyocytes as well as a reduction in the effective atrial refractory period. Obesity has been proven to be an independent risk factor for atrial fibrillation, prompting weight loss measures for prevention. Studies confirm the positive effects of lifestyle changes and weight control on the course and treatment of atrial fibrillation. In such patients, physical activity results in a reduction in AF symptoms, and a change from persistent to paroxysmal AF or remission of the disease. Current methods used to treat atrial fibrillation are less effective in obese patients. Given this, bariatric surgery should be part of the therapy because it effectively reduces body weight, resulting in the reversal of structural changes in the heart and reduced risk of AF and risk of hospitalisation due to AF or heart failure.

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Ethical approval

Not applicable.

Conflict of interest

The authors declare no conflict of interest.

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