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Features of Atrial Fibrillation and Obesity

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Abstract: Metabolic syndrome is one of the most common chronic diseases in the world today. Its spread is so great that it has become a non-contagious epidemic. Obesity creates the basis for the development of a number of cardiovascular diseases. Metabolic syndrome remains one of the main etiological factors, especially in the development of atrial fibrillation. Lipotoxic damage to the heart is one of the important mechanisms for the development of various forms of ventricular fibrillation.

Keywords: Metabolic syndrome, Obesity, atrial fibrillation, lipotoxic cardiomyopathy, waist circumference, hip circumference.

Relevance

Weight gain is observed all over the world today and has become an important health and social problem in countries. Excess weight It is no secret that it is an important factor in the development of a number of diseases.

By solving the problem of malignant tumors, the average life expectancy of people increases by 1 year, and by solving the problem of obesity, people's lives increase by 4 years.

Obesity is a global epidemic and its prevalence has doubled over the past 30 years. In Russia >24% of the population are overweight.

It has been proven that an increase in body mass index (BMI) by 5 kg/m2 increases the risk of heart rhythm disorders by 30%.

Goal of the work:

To study the features of obesity and ventricular fibrillation.

Obesity is a chronic, progressive metabolic disease characterized by excessive accumulation and recurrence of adipose tissue. Obesity is one of the most common chronic diseases in the world today. Its spread is so great that it has become a non-contagious epidemic.Obesity poses a serious health risk andthe disease is accompanied by the development of the following serious diseases: diabetes mellitus (DM) type 2, arterial hypertension (AH), coronary heart disease (CHD), myocardial infarction (MI), malignant tumors. These diseases, in turn, lead to decreased ability to work, early disability and shortened life expectancy. The risk of death from any disease, including cardiovascular disease and cancer, is higher in women and men of all ages who are obese.Obesity was once thought to be a problem in countries with a high standard of living, but the number of overweight and obese children is also increasing in low-middle-income countries.Currently, more than 30 million overweight children live in developing countries and 10 million in developed countries.[1].

Obesity is a global epidemic and its prevalence has doubled over the past 30 years. In Russia >24% of the population are overweight[2].

It has been proven that an increase in body mass index (BMI) by 5 kg/m2 increases the risk of heart rhythm disorders by 30%.[3].

It is noteworthy that in obese humans, conditions exist for the emergence of a special form of myocardial damage, "lipotoxic cardiomyopathy," which was first described in 2000 in obese diabetic (QD) rats. At the same time, an experiment on rats showed that an increase in the delivery of long-chain fatty acids to cardiomyocytes leads to excessive accumulation of triglycerides (TG) in cardiomyocytes (in the form of fat droplets). .[4].

Infiltration of the conducting structures of the heart by fat cells (sinus node, atrioventricular node, His bundle and its legs) may be accompanied by conduction disturbances along these pathways..[5].

It is now known that obesity directly affects the structure and function of the heart. As a result, this can lead to the development of heart failure. Obesity is associated with remodeling of the heart, which affects all its chambers..[6].

Long-term or severe obesity can cause enlargement of the left ventricular cavity, i.e. thickening of the wall and narrowing of the cavity. today this is interpreted as "eccentric remodeling" and causes hemodynamic changes, particularly increases in circulating blood and cardiac volume..[7,8].

The pathophysiological basis for the development of obesity is the discrepancy between the body's energy needs and incoming energy. The main way to obtain energy is by eating food. Energy expenditure goes towards metabolic processes, heat production and physical activity. Fighting excess body weight in adolescents is no easier than in adults, so it is very important to prevent obesity. It is necessary to increase the content of plant fiber in the diet, which will help you feel full faster. In addition, vegetables and fruits contain antioxidants - substances that normalize metabolism.[10]

The incidence of hypertension in middle-aged obese people is 50% higher than in people with normal body weight, and according to the Framingham study, blood pressure increases in parallel with an increase in body mass index (BMI). For every additional 4.5 kg of weight, systolic blood pressure increases by 4 mmHg. Art. in men and by 4.2 mm Hg. Art. among women.[eleven]

Increases in obesity, stress and unhealthy lifestyles have resulted in more than 50 million people living with diabetes (DM) among Europeans aged 20-79 years. By 2030, their number is expected to reach 64 million, and 90 billion euros will be spent on treatment [36]. The 10-year risk of developing diabetes was calculated using a scale that asked different questions for a) the general population, b) obesity, hypertension, or/and severe genetic diseases causing diabetes, and c) patients with cardiovascular disease. .[thirty]

According to the literature, the nature of heart rhythm disturbances in obesity is very diverse. Fractional fibrillation (FF) is the most common type of heart rhythm disorder in obesity. There is also information about the development of supraventricular and ventricular extrasystole, supraventricular tachycardia...[12]

Modern literature describes several mechanisms of excess body weight that influence the development of arrhythmia: activation of the sympatho-adrenal glands, increased activity of the renin-angiotensin-aldosterone system (RAAS), the occurrence of arterial hypertension, increased insulin resistance, and impaired lipid metabolism. disorders, inflammation, the role of laning is debated..[10]

In obesity, the SAS is activated and the level of norepinephrine in the blood plasma increases, which is accompanied by an increase in vascular tone and an increase in total peripheral vascular resistance. Weight loss is known to reduce sympathetic activity.

In obesity, there is a clear activation of RAAT, the level of aldosterone significantly increases, and the mechanisms of blood circulation and tissue regulation of RAAS are disrupted. Activation of the RAAS in obesity plays a leading role in the electrophysiological remodeling of compartments and the development of FD..[13,14]

Obesity, vascular damage, aging, and infection can disrupt the complex balance of multiple factors sequestered by adipose tissue and promoting the growth of vascular smooth muscle cells (SMH), contributing to the development of atherosclerosis, arterial stenosis, and hypertension [15].

Fractional fibrillation (FF) is one of the most common arrhythmias. Its appearance leads to a decrease in the quality of life and a decrease in its duration due to thromboembolic complications. Obesity promotes structural and electrical remodeling of the myocardium, which leads to the appearance of ectopic foci in the ostia of the pulmonary vessels and disruption of normal electrical conductivity in this compartment. The main mechanisms for the formation of an arrhythmogenic substrate include systemic inflammation, myocardial fibrosis, overload of cardiomyocytes with Na+ and Ca2+ ions, accumulation of unoxidized metabolic products in cells, and the balance of autonomic regulation. Arterial hypertension, insulin resistance and obesity-related syndrome increase the risk of occurrence and development of arrhythmia. Studying the pathogenetic mechanisms of FD in obesity is necessary to develop new strategies for its prevention and create more effective methods of treating this group of patients. .[16]

Today, obesity is considered a leading factor leading to FD. 20% of patients with FD are obese.[17].

Less studied factors leading to the development of FD in obesity include lipid metabolism disorders. In obese patients, an increase in plasma triglyceride levels is accompanied by an increase in free fatty acids in the blood and causes the accumulation of acids and less oxidized products in cardiomyocytes.[18].

It has now been established that there are two pathogenetic mechanisms in the development of FD: 1) electrical heterogeneity of the myocardial compartment, creating conditions for the circulation of many independent waves of excitation simultaneously, and 2) high activity of the pulmonary vessels. localized focal triggers. Based on the first mechanism, constant and persistent FD arises, and on the basis of the second, a paroxysmal form of arrhythmia develops. According to the literature, epicardial obesity may play a role in the formation of electrical heterogeneity of the myocardium and an increase in local trigger activity..[19].

Even after normalization of systolic blood pressure, the interventricular septum remains the area most sensitive to changes in adipose tissue. The mechanisms leading to changes in the localization of wall thickening are not completely clear, but the barrier zone is sensitive to overhanging myocardial mass, which can cause asymmetric thickening..[20].

Impaired diastolic function leads to ventricular overdilation, and with obesity there is an increase in inflammation and inflammatory markers that promote the development of fibrosis and scar tissue, which in turn leads to increased permeability.[21].

Conclusions:

1. Obesity is a major factor leading to ventricular fibrillation. The fatty tissue enlarges the left ventricle, which causes electrical remodeling of the heart.
2. Losing body weight reduces the risk of developing FD. It also reduces the number of relapses after ablation.
3. In people with normal waist circumference, repeated paroxysms of atrial fibrillation were observed in 17.3%. 82.7% of repeated paroxysms were observed in patients with a waist circumference greater than normal.

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