



UDC 616.127-005.8

## TO STUDY CHANGES IN VASCULAR WALL STIFFNESS IN OBESE PATIENTS WITH PAROXYSMAL AF DURING THE PERIOD OF SINUS RHYTHM PRESERVATION

Ruziyev Shavkat Rakhmatovich

Philadelphia, USA

### ARTICLE INFO

Received: 28<sup>th</sup> October 2023Accepted: 29<sup>th</sup> October 2023Online: 30<sup>th</sup> October 2023

### KEYWORDS:

chronic heart failure,  
inflammatory heart  
diseases, body weight.

### ANNOTATION:

Obesity is a growing epidemic, with its prevalence doubling in the past 30 years. Overweight is considered as an independent risk factor for the development of arterial hypertension (AH), diabetes mellitus, coronary heart disease, and chronic heart failure.

**Relevance.** Currently, the role of obesity as one of the predisposing factors for the occurrence of atrial fibrillation (AF) is being discussed. An increase in body mass index (BMI) of 5 kg/m<sup>2</sup> has been shown to increase the risk of developing cardiac arrhythmias by 30% [1,3]. Several mechanisms of overweight influence on the development of arrhythmias are described in the current literature: activation of the sympathoadrenal nervous system, increased activity of the renin-angiotensin aldosterone system, the appearance of hypertension, insulin resistance, impaired lipid metabolism, and the development of systemic inflammation [2,7]. One of the least studied mechanisms associated with the development of AF in obesity is considered to be remodeling of the cardiovascular system. It has been proven that an increase in arterial wall stiffness is accompanied by the development of left ventricular diastolic dysfunction and left atrial volume overload, which underlies the pathogenesis of AF [5,8,9]. For the first time, the effect of obesity on the elastic properties of the vascular wall was demonstrated in the work of J. J. Toto-Moukouo et al. When comparing groups of patients with long-term hypertension who were normal and overweight, it turned out that regardless of age, gender, and blood pressure, the pulse wave velocity in the vessels of the upper extremities was significantly higher in obese patients.

The independent role of obesity in the development of vascular changes was demonstrated by J. Orr et al. [4,6]. The study included young healthy men who underwent measurement of vascular wall stiffness on a high-calorie diet, which leads to an increase in body weight by an average of 5 kg in 6-8 weeks.

With weight gain, the authors noted a statistically significant increase in carotid artery stiffness and a decrease in their compliance. More pronounced vascular changes were associated with the presence of visceral obesity, determined by computed tomography and an increase in waist circumference. Researchers have shown that moderate weight gain induced by a high-calorie diet, even in healthy individuals, can lead to increased arterial stiffness. In the modern medical literature, there are practically no clinical works devoted to the study of vascular wall stiffness in patients with obesity and paroxysmal AF.

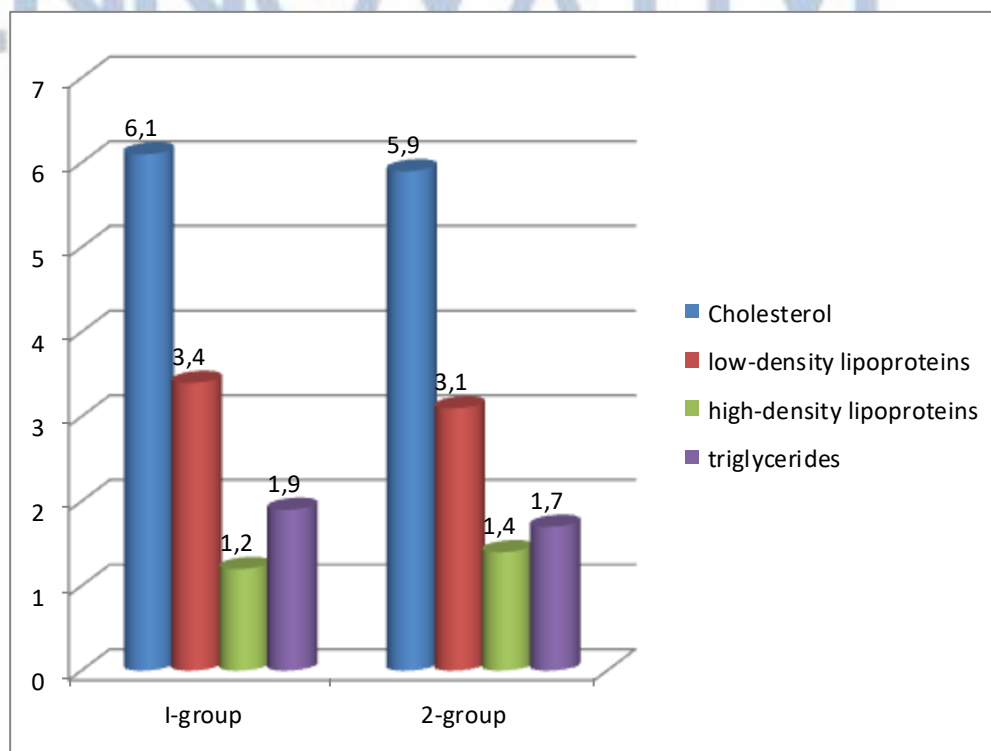
**Objective of the study:** to study changes in vascular wall stiffness in obese patients with paroxysmal AF during the period of sinus rhythm preservation.

**Material and methods of research.** The study included 86 patients with a BMI of 30 to 44 kg /m<sup>2</sup> aged 38 to 72 years (mean age 61.2±4.7 years). Depending on the presence of AF, patients were divided into two groups. The criterion for inclusion of patients in Group I (n=42) was the presence of documented AF paroxysm in obese patients, confirmed by electrocardiography (ECG) or Holter ECG monitoring the comparison group (group II; n=44) consisted of obese patients without cardiac arrhythmias. Exclusion criteria: functional class I-IV stress angina, a history of myocardial infarction or cerebral stroke, acute coronary syndrome, functional class III-IV chronic heart failure, inflammatory heart diseases, heart defects, severe kidney, liver, lung diseases, anemia, oncological diseases, pregnancy, and mental illnesses. All patients signed the patient's written informed consent. When patients were included in the study, all patients underwent a general clinical examination with an assessment of anthropometric parameters: BMI, waist circumference (OT), hip circumference (OB), ratio of OT/OB and OT to height (OT/height), sagittal abdominal diameter. Visceral obesity was diagnosed in patients with values of the OT/OB ratio >1.0 and the OT/height ratio >0.6. To assess the stiffness of the wall of the main arteries VaSera, the VaSera (VS-1000) device (Fucuda Denshi, Japan) was used, which made it possible to automatically determine and calculate the main indicators of vascular wall stiffness – the cardio-ankle vascular index (CAVI), the biological age of the arteries and the ankle-brachial index (ABI) in the main arteries on the right and left. The CAVI index was calculated automatically based on the registration of plethysmograms of 4 limbs, electrocardiograms, and phonocardiograms using a special algorithm for calculations (the Bramwell-

Hil formula). The structural and functional state of the myocardium was evaluated by echocardiography using a Siemens device (Germany), a sensor with a frequency of 3.74 MHz. Standard positions were used. Statistical processing of the obtained results was carried out on a personal computer using the SPSS23.0 program using standard statistical methods of information processing. Numerical data were described using the arithmetic mean (M) and its root-mean-square deviation ( $\sigma$ ). Statistical analysis was performed using the Student's parametric criterion. Correlation analysis was performed using Pearson's r correlation criterion. The result was considered statistically significant for the probability of error in patients of group I and II.

**Research results.** Most of the patients included in the study had more than two risk factors, which determined a high risk of cardiovascular events. When comparing the total number of risk factors, there were no statistically significant differences between the groups. The level of total plasma cholesterol in group I was  $6.1 \pm 1.9$  mmol/l, in group II- $5.9 \pm 2.6$  mmol/L, low-density lipoproteins- $3.4 \pm 0.3$  and  $3.1 \pm 0.2$  mmol/L, high-density lipoproteins- $1.2 \pm 0.1$  and  $1.4 \pm 0.1$  mmol/L, triglycerides- $1.9$  and  $1.7 \pm 0.1$  mmol/l, glucose levels of  $5.9 \pm 1.1$  and  $6.2 \pm 1.2$  mmol/L, respectively, and had no statistically significant differences. See 1-picture.

**Picture 1. Comparison of lipoproteins in 2 groups.**



When performing anthropometric measurements, it was found that BMI did not have statistically significant differences in patients of the studied groups. Anthropometric indicators of visceral obesity were statistically significantly higher in group I than in group II. The prevalence of visceral obesity in patients of group I was statistically significantly higher than in patients of group II: 42 (100%) patients versus 32 (72%) patients; ( $p=0.0002$ ). The study of vascular wall rigidity revealed a statistically significant increase in the CAVI index in patients with paroxysmal AF than in patients without cardiac arrhythmia.

Systolic blood pressure (BP) in patients of group I was  $147\pm 5.2$  mm Hg, in group II- $152\pm 6.8$  mm Hg, diastolic blood pressure- $88.7\pm 2.8$  and  $86\pm 2.1$  mm Hg, heart rate (HR) –  $77.0\pm 3.8$  and  $75.5\pm 2.9$  beats / min, respectively. There were no statistically significant differences in blood pressure and heart rate between the study groups. We studied the relationship between anthropometric indicators of visceral obesity and vascular wall stiffness indices in the study groups. In patients with obesity and paroxysmal AF, CAVI correlations with OT, OT/OB and OT/height ratios were found. Echocardiography revealed normal values of the left ventricular ejection fraction (LVEF), end-diastolic and end-systolic left ventricular volume in all patients included in the study. Patients of group I had statistically significantly larger left atrial volume (LV), LV myocardial mass index, and epicardial adipose tissue thickness when compared with similar indicators in group II. At the same time, the values of the E/A ratio in obese patients with paroxysmal AF were less than 1.0, which characterizes the appearance of LV diastolic myocardial dysfunction. To assess the relationship between the parameters of vascular wall stiffness and the frequency of arrhythmia paroxysms in patients of group I, a correlation analysis was performed. A positive direct relationship was found between the CAVI index and the frequency of AF attacks during the year ( $r=0.782$ ,  $p=0.001$ ;). The obtained relationship proves a direct link between vascular lesions and the development of AF paroxysms in patients with visceral obesity. There were no statistically significant associations between the CAVI index and age. In our study, it was found that the occurrence of frequent AF paroxysms correlated with an increase in the CAVI index, and the obtained relationship proves a direct relationship between vascular wall damage and the development of arrhythmic complications in visceral obesity: from increased vascular wall stiffness, development of LV diastolic dysfunction, increased LP volume, and the appearance of AF.

**Conclusion.** Statistically significant increase in the CAVI index, indicating a change in the stiffness of the vascular wall, was found in patients with obesity and paroxysmal AF when compared with overweight patients without cardiac arrhythmias. An increase in the CAVI index was associated with an increase in OT, the ratio of OT/OB, OT/growth. A statistically significant direct relationship was found between the CAVI index and the frequency of arrhythmia attacks in obese patients with paroxysmal AF.

### List of literature

1. Wong C.X., Sullivan T., Sun M.T., et al. Obesity and the risk of incident, post-operative, and post-ablation atrial fibrillation: a meta-analysis of 626,603 individuals in 51 studies. *JACC Clinical Electrophysiology*. 2015;1:139-52. DOI:10.1016/j.jacep.2015.04.004.

2. Podzolkov V.I., Tarzimanova A.I., Gataulin R.G., et al. the Role of obesity in the development of atrial fibrillation: the current state of the problem. *Cardiovascular Therapy and Prevention*. 2019;18(4):109- 14 (In Russ.) [Podzolkov V.I., Tarzimanova A.I., Gataulin R.G., et al.. The role of obesity in the development of atrial fibrillation: current state of the problem. *Cardiovascular Therapy and Prevention*. 2019;18(4):109-14]. DOI:10.15829/1728-8800-2019-4-109-114.

3. Goudis C.A., Korantzopoulos P., Ntalas I.V., et al. Obesity and atrial fibrillation: A comprehensive review of the pathophysiological mechanisms and links. *J Cardiol*. 2015;66(5):361-9. DOI:10.1016/ j.jjcc.2015.04.002.

4. Kotsis V., Stabouli S., Papakatsika S., et al. Mechanisms of obesity-induced hypertension. *Hypertens Res*. 2010;33(5):386-93. DOI:10.1038/hr.2010.9.

5. Ogunsua A.A., Shaikh A.Y., Ahmed M., McManus D.D. Atrial Fibrillation and Hypertension: Mechanistic, Epidemiologic, and Treatment Parallels. *Methodist DebaqueyCardiovasc J*. 2015;11(4):228-34. DOI:10.14797/mdcj-11-4-228.

6. Yang T., Yang P., Roden D.M., Darbar D. Novel KCNA5 mutation implicates tyrosine kinase signaling in human atrial fibrillation. *Heart Rhythm*. 2010;7(9):1246-52. DOI:10.1016/j.hrthm.2010.05.032.

7. Lumeng C.N., DelProposto J.B., Westcott D.J., et al. Phenotypic switching of adipose tissue macrophages with obesity is generated by spatiotemporal differences in macrophage subtypes. *Diabetes*. 2008;57(12):3239-46. DOI:10.2337/db08-0872.

8. Zaripova D. Ya., Negmatullayeva M. N., Tuksanova D. I. The role of Aleandronic acid (Ostalon) in the treatment of perimenopausal osteoporosis. Dr. akhborotnomasi 2019; 4(3) pages23-27.

9. Zaripova D. Ya., Negmatullayeva M. N., Tuksanova D. I., Ashurova N. G. Influence of magnesium deficiency state and imbalance of steroid hormones in the vital activity of a woman's body. Tibbietda yangi kun. №4.2019 p. 45-49.

