

THE ROLE OF VASCULAR WALL RIGIDITY IN THE DEVELOPMENT OF CORONARY HEART DISEASE WITH COMORBID KIDNEY DYSFUNCTION

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Abstract. *The article about the role of vascular wall stiffness in the development of coronary heart disease and renal dysfunction. At the beginning of the review, the definition of the stiffness of the vascular wall is given. Rigidity of the aorta and large vessels of the elastic type is being studied as an independent marker of the risk of developing coronary artery disease and how its measurement allows a more accurate assessment of the prognosis of the disease Methods for noninvasive determination of vascular stiffness by pulse wave propagation velocity, central pulse pressure level, and index of pulse pressure increase in the aorta allows to define formation of high-risk groups for the purpose of its further correction.*

Keywords: *vascular wall stiffness, coronary artery disease, pulse wave propagation velocity, central pulse pressure, index of pulse pressure.*

РОЛЬ ЖЕСТКОСТИ СОСУДИСТОЙ СТЕНКИ В РАЗВИТИИ ИШЕМИЧЕСКОЙ БОЛЕЗНИ СЕРДЦА С СОПУТСТВУЮЩЕЙ ДИСФУНКЦИЕЙ ПОЧЕК

Аннотация. *Статья о роли жесткости сосудистой стенки в развитии ишемической болезни сердца и нарушениях функции почек. В начале обзора дается определение жесткости сосудистой стенки. Ригидность аорты и крупных сосудов эластического типа изучается как независимый маркер риска развития ИБС и как ее измерение позволяет более точно оценить прогноз заболевания Методы неинвазивного определения жесткости сосудов по пульсу скорость распространения волны, уровень центрального пульсового давления и индекс повышения пульсового давления в аорте позволяют определить формирование групп высокого риска с целью его дальнейшей коррекции.*

Ключевые слова: *жесткость сосудистой стенки, ишемическая болезнь сердца, скорость распространения пульсовой волны, центральное пульсовое давление, индекс пульсового давления.*

Relevance: The development of the most socially significant cardiovascular diseases (CVD), which are based on the progression of atherosclerosis with the further occurrence of its

complications, has been considered in recent decades from the standpoint of the "cardiovascular continuum".

Currently, the search for new prognostic factors continues, the correction of which would allow to influence mortality from coronary artery disease [4]. Diagnosis and treatment of chronic coronary heart disease (CHD) is well developed, but in the management of patients with comorbid diseases, difficulties often arise. A very common chronic kidney disease (CKD) occupies a special place among comorbidities, which is considered a major risk factor for cardiovascular disease. The cause of death in patients with kidney dysfunction is significantly more often CHD than end-stage renal disease (ESRD) [14]. The urgency of this problem is increasing every year due to an increase in the number of elderly patients with coronary artery disease and severe kidney dysfunction. In this regard, one of the most important characteristics of the vascular system, arterial stiffness, is of great interest. Arterial stiffness is defined as the ability of the arterial wall to resist stretching under the influence of blood flow.

Vascular stiffness depends on the ratio of the structural proteins of elastin and collagen, as well as the tone of the smooth muscle cells that make up the middle shell. The wall of the aorta and its large branches contains mainly elastin [6]. Such vessels are classified as elastic type. In peripheral arteries (muscle type), the predominant components are smooth muscle cells and collagen, which, when stretched, offers more resistance than elastin. The higher the blood pressure on the vessel wall, the greater the wall stress. This served as the basis for studying the role of vascular stiffness in the formation and progression of coronary artery disease. The main function of the aorta and large arteries is to buffer fluctuations in blood pressure (BP) resulting from intermittent ejection of blood from the cavity of the left ventricle. Highly elastic arterial system provides stable blood flow in most tissues of the body without the impact of peak systolic pressure; this mechanism is so effective that it practically prevents the decrease in diastolic pressure from the ascending aorta to the peripheral arteries. With the loss of distensibility, the aorta becomes more rigid and loses the ability to accommodate the volume of blood ejected from the left ventricle, which leads to an increase in systole pressure and an increase in pulse pressure.

Purpose of the study: to study the magnitude of the stiffness of the aortic wall in patients with coronary artery disease and kidney dysfunction as an independent marker.

Material and research methods. We examined 40 patients with coronary artery disease aged 40 to 70 years (mean age 61.5 ± 6.5 years).

The first group consisted of 20 people, including 14 men and 6 women (mean age was 58.4 ± 7.5 years) without hemodynamically significant lesions of the coronary arteries (CA) and renal vessels. The second group included 20 people (12 men and 8 women), mean age 63.0 ± 5.6 years, with hemodynamically significant damage to one or two coronary arteries. The control group consisted of 20 patients aged 40 to 70 years old (56.5 ± 6.5 years), including 13 men and 7 women with excluded cardiovascular and renal diseases. Work with each patient included: questioning (complaints anamnesis), physical examination (measurement of the height and weight of the patient's body with the calculation of BMI, waist circumference (WC), as well as a study of the functional state of the cardiovascular system and kidneys. The examination included: complete blood count and urine, biochemical blood test (determination of the level of creatinine, urea, cholesterol, lipid spectrum), coagulogram, ECG, dopplerography of the renal vessels. Arterial stiffness was determined by applanation tonometry using the SphygmoCor apparatus (AtCor Medical, Australia). Studied: central systolic blood pressure (cSBP), central diastolic blood pressure (cDAP), central pulse pressure (CPP), aortic augmentation (AA), augmentation index (AIx), pulse wave velocity (PWV), which were used to determine the rigidity of the vascular wall. In the position of the patient lying down, after a 10-minute rest, he entered into the computer program and passport, anthropometric data of the patient, SBP and DBP, measured by manual tonometry. To assess PWV, the distance (in mm) from the site of the pulsation of the femoral artery under the pupartite ligament to the clavicle (distal distance) and the distance (in mm) from the site of the pulsation of the carotid artery in the triangle of the same name to the clavicle (proximal distance) were indicated. Next, three electrodes were applied to the upper limbs and the left leg, and the pulse wave was sequentially recorded on the carotid and femoral arteries with synchronous ECG recording, and automatic PWV calculations were made. Statistical processing of the study results was carried out using the Microsoft Excel 2016 statistical software package [12].

Research results. It was found that with an increase in the degree of atherosclerotic lesions, the level of SBP does not change, and the level of DBP tends to decrease. With an increase in the degree of atherosclerotic lesions, the values of peripheral and central pulse BP, AIx, AIx@HR75 and PWV increase statistically significantly. With an increase in the degree of damage to the coronary arteries, the indicators characterizing the augmentation of the central pulse pressure and the stiffness of the vascular wall increase.

In the first group, with an increase in the number of affected vessels, the following increase significantly: peripheral augmentation index, central augmentation index, peripheral and central pulse blood pressure and PWV (cSAP– 156.4 ± 14.0 mm Hg ($p=0.015$), CPD - 59.8 ± 8.50 1 mm Hg. Art. ($p=0.0001$), AIx - $54.4 \pm 9.0\%$ ($p=0.025$) AIx@HR75 - $28.9 \pm 4.1\%$ ($p=0.031$) and PWV - 18.6 ± 4.2 m/s ($p<0.05$). In the second group, there was a significant increase in central pressure, augmentation index, central augmentation index, peripheral and central pulse BP and PWV. When comparing the parameters of the three groups, depending on the degree of coronary injury, it was found that the maximum values of cSAP were 140.4 ± 13.0 mm Hg. Art. ($p=0.015$), CPD - 55.8 ± 9.50 1 mm Hg. Art. ($p=0.0001$), AIx - $44.4 \pm 9.0\%$ ($p=0.025$) AIx@HR75 - $22.9 \pm 3.8\%$ ($p=0.031$) and PWV - 15.7 ± 3.8 m/s ($p=0.0015$) were found in the second group with 3-vessel atherosclerotic lesions of the coronary artery and renal vessels. Also in the second group, the value of PWV was 4.8 m/s higher than in patients in the first group with a similar lesion of the coronary artery ($p=0.0005$). In the third control group, the value of cSBP was 135.4 ± 5.0 mm Hg. Art. ($p=0.015$), CPD – 55.3 ± 7.50 1 mm Hg. Art. ($p=0.0001$), AIx – $32.1 \pm 8.0\%$ ($p=0.025$) AIx@HR75 – $26.3 \pm 3.8\%$ ($p=0.032$) and PWV – 7.7 ± 4.4 m/s ($p=0.0015$).

Urea levels and creatinine values (98.80 ± 11.67 $\mu\text{mol/l}$) in the first and third groups of the examined did not exceed normal values. In the second group, there was a significant increase in these indicators (138.40 ± 14.56 $\mu\text{mol/l}$). From this it follows that the majority of patients with CHF of ischemic origin had chronic renal dysfunction in the absence of primary renal pathology.

Doppler ultrasonography (USDG) of the renal vessels showed no hemodynamic disturbances in patients in the third group; in the majority of patients in the first and second groups, hemodynamic disorders were detected (42.5% and 61.8%): an increase in resistive characteristics - 17.6%, renal artery stenosis with lumen obliteration - 44.2%. Kidney function was reduced in 100% of patients in the second group ($p < 0.05$).

Discussion: Thus, a therapeutic strategy that aims to reduce arterial stiffness may lead to a greater improvement in patient prognosis than the correction of individual risk factors. The PWV level reflects the severity of coronary atherosclerosis in patients with established coronary artery disease and renal dysfunction [14]. In studies using Dopplerography of the renal vessels, it was shown that the stiffness of the renal vessels increases in proportion to the number of coronary arteries affected by atherosclerosis, as well as the length and degree of their stenosis, an increase in the amount of creatinine and urea in the blood indicates a significant lesion of the renal

parenchyma. In addition, PWV is an independent parameter, positively associated with vascular stiffness and the degree of coronary artery stenosis [6].

In patients with coronary heart disease, with an increase in the degree of damage to the coronary arteries, an increase in pulse blood pressure, augmentation of central systolic blood pressure, augmentation index and PWV was noted. In patients with coronary heart disease, as the number of affected coronary arteries increases, the pulse wave velocity in the elastic type arteries increases significantly, while the parameters of the central aortic pressure remain unchanged. On the one hand, this indicates the severity of the atherosclerotic process, on the other hand, an increase in the central pulse blood pressure, the degree of augmentation (AIx) and the speed of propagation of the pulse wave indicates the integral nature of the damaging effect of the pulse wave on the vascular wall and reflects an increase in the risk of cardiovascular complications, as well as damage to the vessels of the kidneys increases in proportion to the number of coronary arteries affected by atherosclerosis [10].

Thus, the relationship of coronary heart disease and kidney dysfunction with an increase in the incidence of diseases and a worse prognosis in patients with coronary artery disease with kidney dysfunction and the impact of a decrease in kidney function on the clinical picture of coronary disease are determined.

Conclusions. The stiffness of the aorta and large vessels of the elastic type is an independent marker of the risk of cardiovascular complications in coronary artery disease. Its measurement allows a more accurate assessment of the prognosis and the formation of high-risk groups for the purpose of its further correction. Methods for noninvasive determination of vascular stiffness by pulse wave propagation velocity, central pulse pressure level, and index of pulse pressure increase in the aorta have been developed and tested. This allows the methods to be widely used in clinical practice.

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