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DEPENDENCIES IN THE CLINIC AND DIAGNOSIS OF CORONARY HEART DISEASE AND ARTERIAL HYPERTENSION

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Annotation

Study of the relationship between the symptoms of both diseases in the clinic of coronary heart disease and hypertension. Identification of the relationships observed in the diagnosis of coronary heart disease and hypertension.

Key words: coronary heart disease, angina pectoris, arterial hypertension, diagnosis, prevention. Symptoms, difficulty breathing, radiate, Blood Pressure,

Angina Angina (angina pectoris) is a clinical syndrome manifested by a feeling of discomfort or pain in the chest, the development of which is associated with transient myocardial ischemia due to a discrepancy between the myocardial oxygen demand and its delivery through the coronary arteries. This situation occurs when the lumen of the coronary arteries narrows by 50–70%.

Clinic and diagnosis The main clinical manifestations of the disease are pain in the chest, which patients characterize as pressing, squeezing, bursting, burning. They most often point to the place of pain not with one finger, but with the entire palm or fist, pressing them to the sternum or to the left of it. The pain can spread (radiate) to the neck, left shoulder, left arm, back and, less commonly, to the lower jaw, epigastric region, right half of the chest and right arm. The attack occurs at the height of physical activity (angina pectoris). The pain may be accompanied by a feeling of fear of death, anxiety, general weakness, sweating, and nausea. The duration of painful attacks does not exceed usually 15 min. They disappear completely after stopping physical activity or using nitroglycerin for a few minutes. Angina attacks are provoked by physical and psycho-emotional stress, cooling, smoking, increased blood pressure, rich food, and

unfavorable weather conditions. Typically, angina attacks occur when the patient leaves the room to go outside in cold, windy weather. Atypical manifestations of angina pectoris are possible: patients note a feeling of heaviness in the chest, difficulty breathing, lack of air, weakness, palpitations, pain in the epigastric region, heartburn. These equivalents of angina occur and resolve under the same conditions as chest pain. Timely diagnosis of angina pectoris is important for determining further tactics of medical care; algorithms for its diagnosis have been developed based on clinical symptoms.

In general, angina pectoris is not characterized by pain: • acute, piercing in nature;

• changing with breathing, changing body position; • lasting several hours;

• localized above the lower jaw, below the epigastric region;

• localized in a small area in the left half of the chest Angina pectoris can be stable or unstable. Stable angina is characterized by a fairly long (at least 2 months) persistence at the same level of strength, frequency and duration of pain attacks that occur in certain situations (fast walking, after eating, during psycho-emotional stress, etc.). Increased activation of the plasma, and in particular the tissue renin-angiotensin-aldosterone system (RAAS), can be detected already in the early stages of both hypertension and the atherosclerotic process. The RAAS, in essence, plays an extremely important role in the following chain of events: “damaging” factors - oxidative stress - endothelial dysfunction - activation of the RAAS - imbalance of nitric oxide and AT-II - increased oxidative reactions - further aggravation of endothelial dysfunction - further pathological processes, affecting “target organs”. Naturally, angiotensin-converting enzyme inhibitors (ACE inhibitors) can play a significant role in influencing this pathological chain. Indeed, these drugs have a wide spectrum of action, potentially useful in disorders of neurohormonal regulation not only in hypertension and coronary artery disease, but also in chronic heart failure (CHF). According to modern concepts, ACEIs cause not only a decrease in tissue and plasma AT-II, but also reduce the activity of the sympathetic-adrenal system (SAS) and increase the production of bradykinin [7]. Reduced degradation of bradykinin causes a vasodilator effect, increased production of nitric oxide, prostacyclin, and tissue plasminogen activator. Nitric oxide, in addition to powerful vasodilation, prevents platelet aggregation and activation of a number of cells (especially monocytes that can transform into lipid-containing macrophages), and also inhibits the proliferation of smooth muscle cells - integral components of atherosclerotic arterial lesions. It is assumed that endothelial damage primarily affects the production of endothelial synthetase, responsible for the synthesis of nitric oxide.

An appropriately sized blood pressure cuff is placed on the upper arm. An appropriately sized cuff covers two-thirds of the biceps; the cuff chamber is long enough to cover >80% of the arm, and the cuff chamber width is at least 40% of the

arm circumference. Thus, a larger cuff size is required in obese patients. The physician inflates the cuff above the expected systolic pressure and gradually releases the air while listening to the brachial pulse. The pressure at which the first heart sound is heard during a decrease in pressure is the systolic blood pressure. The complete disappearance of sounds indicates diastolic blood pressure. The same principles should be followed for measuring blood pressure in the forearm (radial artery) and thigh (popliteal artery). artery). Mechanical instruments must be periodically calibrated; automatic machines often show inaccurate data (1).

Blood pressure is measured in both arms, because if the difference in blood pressure is > 15 mm Hg. Art. on one arm versus the other, a study of the vascular system of the upper body is necessary.

Blood pressure is measured at the thigh for coarctation of the aorta, especially in patients with a weakened pulse or delayed pulse wave at the femoral artery (using a much larger cuff); With coarctation, blood pressure in the lower extremities is significantly lower.

If blood pressure is in the range of stage 1 hypertension or is markedly labile, then it is advisable to measure blood pressure more often. Rarely, blood pressure readings may be high before hypertension becomes persistent; this phenomenon likely explains "white coat hypertension", in which blood pressure is elevated when measured in a doctor's office but normal when measured at home or with ambulatory blood pressure monitoring.

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