

HYPERTENSION DISEASE AND ITS TYPES, CAUSES

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Abstract: High blood pressure or hypertension is one of the most common diseases of the cardiovascular system today. The disease is manifested by an increase in arterial blood pressure, and often its indicator exceeds 140/90. According to many experts in the field of cardiovascular diseases, hypertension often occurs as a result of blood circulation disorders. Heart failure is also included in the list of its causes. This disease can trigger the development of secondary diseases in patients, for example, stroke, heart attack.

Key words: hypertension, stroke, ischemic heart disease, arteriosclerosis, hyalinosis, heart attack.

Hypertension disease (primary arterial hypertension) is a nosological form clinically characterized by a long-term and constant rise in arterial pressure. The characteristics of this disease are as follows: 1) its prevalence, 2) the fact that sometimes it passes without symptoms for a long time even before the last stage begins, 3) the development of life-threatening complications. High blood pressure is the most important risk factor for the onset of ischemic heart disease, cerebrovascular disease, and nephrosclerosis leading to kidney failure. As a person grows older, the tendency to hypertension increases, but this disease can also be observed in young people and can be very dangerous. Most primary hypertension in women is observed at a young age, while in men it is observed after the age of 50. Primary arterial hypertension (idiopathic, essential hypertension), which occurs in 90% of cases, should be distinguished from secondary or symptomatic hypertension can pass safely or dangerously.

Safe hypertension is more common, it is distinguished by the fact that arterial pressure remains the same for many years. In this case, if myocardial infarction or stroke is not added, patients live longer. This type of hypertension leads to the onset of nephrosclerosis, which is safe. Sometimes (in 5 percent of cases) dangerous hypertension is observed less often (in 5 percent of cases), in which arterial pressure rises from the very beginning of the disease to very high numbers (up to 220 | 140 mm Hg), and most tend to rise again. Organic changes in the vessels and organs are rapidly increasing. Dangerous nephrosclerosis, neuroretinopathy (bilateral cysts of the retina and optic nerve discs, hemorrhages) progressing with worsening kidney failure. Hypertensive encephalopathy, heart and kidney failure begin. Most dangerous hypertension begins at the age of 40.

Arterial pressure office bending mechanism. It is known that the level of arterial pressure (systolic and diastolic pressure) depends on a number of factors, among them the volume of blood arriving at the core of the vessels per unit of time (the volume of blood ejected from the heart) and the resistance of peripheral vessels (the degree of vasocomstriction) is in the main position. The nervous system, baroreceptor and chemoreceptor systems, humoral factor should be mentioned among the physiological mechanisms controlling the interaction of the two aspects just mentioned. However, the excretory mechanism of the kidneys occupies a special place in the control of arterial pressure, and it is necessary to examine this mechanism in order to understand the pathogenesis of hypertension. Renin-angiotensin system. Kidneys participate in blood pressure control with the help of renin and angiotensin II, renin is produced by the cells of the supraglomerular complex. Angiotensin II is formed when the decapeptide angiotensin 1 is released from plasma angiotensin under the influence of renin, and is considered the most important link of the renin-angiotensin system, because it causes vasoconstriction and stimulates the secretion of aldosterone, aldosterone in the renal tubules by increasing the reabsorption of sodium and water, it creates conditions for further narrowing of the vessels and further increase in arterial pressure. This is due to the fact that, in addition to increasing the volume of blood ejected from the heart, sodium retention in the body changes the sensitivity of smooth muscle cells to vasoactive effects and increases peripheral resistance.

Etiology and pathogenesis. The etiology and pathogenesis of primary and secondary hypertension are different. In this regard, it is appropriate to review the ulama separately. The causes and mechanism of exacerbation of primary, i.e., essential hypertension are not known, as evidenced by the fact that there are many hypotheses about them. It is considered that the increase in arterial pressure depends on the influence of nerves, hormones, various factors related to the kidney, reflex and genetic factors. According to G. F. Lang's neurogenic theory, the disruption of higher nerve activity due to external influences, that is, the long-term activation of autonomic centers that control blood circulation and the rise of blood pressure, is the leading link in the pathogenesis of hypertension. "im" ("remaining feelings"). The role of hormonal changes that predispose to high blood pressure is especially clear in women in the form of climacteric neurosis. When the estrogen and androgen functions of the gonads decrease, in response to this, as a compensatory reaction, the function of the adrenal glands increases, catecholamines and adrenaline are produced in excess. Micronutrients are less important in the etiology and pathogenesis of hypertension. Currently, only cadmium and magnesium have been reported to be important. chronic arterial hypertension was created when cadmium was injected into the body with food or drinking water. In this regard, it is worth noting the data that hypertension is somewhat more common among people who use soft water with very few minerals. It is assumed that the beginning of local vasoconstriction, retention of sodium in the body, and increase in renin activity in the plasma are the basis of the hypertensive effect of cadmium.

Damage to small arteries and arterioles is the most typical symptom of arterial hypertension. At the same time, three types of main structural changes are observed in them - arteriosclerosis, hyalinosis or fibrinoid necrosis. For arteries of elastic and muscular-elastic type,

elasto-fibrosis and atherosclerosis are pathognomonic, since hypertension is a serious risk factor for the onset of atherosclerosis. The nature of the morphological changes in the vessels depends on the stage of the development of arterial hypertension and how the disease progresses (safely or dangerously). For example, in dangerous hypertension, vessels and organs are damaged in the early stages, and hypertensive crises are more frequent. Morphologically, hypertensive crises are characterized by folding and changes in the structure of the basement membrane of the endothelium, which indicate spasm of arterioles. In addition, plasmatic infiltration or fibrinoid necrosis is observed in the wall of arterioles. In the safe passage of hypertension, there are three main stages, which differ in the nature of morphological changes. For example, in stage I of hypertension (transient hypertension stage), structural changes are in the form of muscle layer hypertrophy and elastosis, and are found only in arterioles and small arteries. Hypertrophy of the muscular layer of vessels depends on the hyperplasia and hypertrophy of smooth muscle cells that pass from the expanded areas of the internal elastic membrane to the intima. Sometimes, between the endothelium and the internal elastic membrane, a region of smooth muscle cells with two or more layers appears. In this case, the wall of arterioles becomes thicker. References

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