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HYPERTENSION ETIOLOGY

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Annotation

Information about hypertension, its classification, mechanisms of development, elements of non-drug treatment and methods of preventing the disease are provided.

Key words: hypertension, nature of the course, treatment options, laboratory diagnostics, prevention.

Hypertension (HTN) is a chronic, stable increase in blood pressure, in which in people not receiving antihypertensive drugs, the level of systolic blood pressure is ≥140 mmHg. Art. and/or diastolic blood pressure level – ≥90 mm Hg. Art. (WHO and International Society of Hypertension recommendations 1999).

Arterial hypertension (AH) is a cause of heart attack and stroke. According to WHO estimates, >17.5 million people worldwide died from heart attacks and strokes in 2012.

Epidemiology. According to the epidemiological study "EPOCHA" (European part of Russia, 2002), the prevalence of hypertension is 39.2%, and effective treatment of hypertension is achieved in only 7.2% of patients. It is noted that it increases with age. Hypertension occurs in 50% of people aged 60–69 years and in 75% of people aged ≥70 years. Among patients under 40 years of age there are more men, in older age groups there are more women. However, not all people with high blood pressure (BP) are aware of this. Awareness of the presence of high blood pressure is 59% in women and 37% in men. It is also noted that not all people suffering from hypertension receive drug treatment (45% of women and 21% of men); however, target blood pressure values were achieved only in 17% of women and 5% of men.

The etiology of essential hypertension is unknown, and it is unlikely that a single cause could explain the variety of hemodynamic and pathophysiological disorders characteristic of this disease. In modern cardiology, hypertension is considered as a polygenic disease (i.e., caused by both hereditary structural disorders in various gene regions and environmental factors).

The basis for assuming its hereditary nature was the frequent identification of patients with a family history of cardiovascular diseases (CVD). Subsequent studies showed an increase in the expression level and the presence of “unfavorable” variants

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of polymorphism in more than 20 genes encoding pressor systems for blood pressure regulation, such as angiotensin-converting enzyme, angiotensinogen, angiotensin II receptors, etc. The role of these genetic changes in the pathogenesis of hypertension requires further study .,

Risk factors for developing hypertension include:

• hereditary burden of hypertension, CVD, DLP, DM;

• the patient has a history of CVD, DLP, DM;

• intoxication (smoking, alcohol);

• poor nutrition (salt overload, magnesium deficiency);

• obesity;

• low physical activity;

• snoring and indications of respiratory arrest during sleep (information from the patient's relatives);

• personal characteristics of the patient;

• emotional stress;

• occupational hazards (noise, constant strain on vision, attention);

• traumatic brain injury;

• age-related restructuring of the diencephalic-hypothalamic structures of the brain (during menopause);

• periodontal pathology*.

Epidemiological data indicate a potential association of periodontitis with increased blood pressure and the prevalence of hypertension. An increase in blood pressure in patients with periodontal pathology has been noted in a number of studies. Evidence from cross-sectional studies suggests that the presence of periodontitis in patients with hypertension may increase the risk and extent of end-organ damage.

The blood pressure level is determined by the ratio of cardiac output (CO) of blood and total peripheral vascular resistance. Whatever the pathogenetic mechanisms of hypertension, they should lead to an increase in total peripheral resistance (TPR) as a result of vasoconstriction or an increase in cardiac output (CO), or change both of these indicators.

Accordingly, the development of hypertension may be a consequence of:

1) increased OPS caused by spasm of peripheral vessels (neurogenically caused);

2) an increase in CO due to the intensification of myocardial work (neurogenically caused) or an increase in the intravascular volume of fluid (with sodium retention in the body);

3) a combination of increased cardiac output and increased total peripheral resistance.

The trigger point of pathogenesis is considered to be hyperactivity of the nerve centers for blood pressure regulation, expressed in increased pressor effects, which is

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carried out by increasing: the activity of the sympathetic nervous system (sympathetic-adrenal system); production of renal pressor substances (switching on of the renin-angiotensin mechanism, the appearance of secondary hyperaldosteronism, increased production of prostaglandin F2α and cyclic nucleotides); release of vasopressin.

An increase in the activity of the sympathetic-adrenal system is manifested by hypersecretion of catecholamines, which affect total peripheral resistance and cardiac output by:

a) direct alpha-adrenergic stimulation of arterioles and veins, which causes spasm of peripheral vessels and leads to an increase in peripheral vascular resistance;

b) beta-adrenergic stimulation, which increases CO;

c) an indirect increase in CO due to peripheral venoconstriction with a decrease in the intravascular blood volume in them and an increase in the central, cardiopulmonary volume, which causes an increase in the venous return of blood to the ventricles of the heart and an increase in the stroke volume of the heart.

Increased activity of the sympathetic-adrenal system is the main factor in increasing blood pressure in the initial period of hypertension (formation period). During this period, a hyperkinetic type of blood circulation is formed, which is characterized by an increase in CO with a slightly changed OPS. Neurogenic stimuli, due to the high sensitivity of the renal arterioles to constrictor effects, cause, through renal ischemia, hyperproduction of pressor-active humoral substances of varying duration and severity: the proteolytic enzyme renin, angiotensin II, aldosterone and others. Increased release of the latter into the blood leads to increased blood pressure. They acquire primary importance during the period of persistent hypertension (stabilization period).

The severity and stability of hypertension is determined not only by an increase in the production of pressor agents, but also by a decrease in active depressor effects:

1) decreased release of prostaglandins E2, D, A and prostacyclin J2;

2) inhibition of the kinin system;

3) decreased production of the renin inhibitor – phospholipid peptide;

4) reconfiguration of receptors in the sinocarotid zone of the aortic arch.

Stimulated by renal ischemia in the early stages, the hyperfunction of the juxtaglomerular apparatus is replaced during this period by its hyperplasia and hypertrophy, which leads to a constant change in the production of renin and stimulation of the production of angiotensin II and aldosterone. Hypersecretion of aldosterone causes sodium retention in the walls of arterioles, increasing their sensitivity to the effects of pressor factors. Together with sodium, a significant amount of calcium diffuses into the cell during the depolarization phase, which increases the tone of vascular smooth muscles.

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Depending on the predominance of violations of one or another link in the humoral regulation of blood pressure, pathogenetic variants of hypertension are distinguished, differing in the level of renin in the palasma: hyperrenin, normorenin, hyporenin.

Regardless of the clinical and pathogenetic variants of the course of hypertension, elevated blood pressure leads to damage to target organs, the functional state of which determines the course and outcome of hypertension.

In elderly and senile people, in the pathogenesis of arterial hypertension, in addition to the general mechanisms causing a persistent increase in blood pressure, age-related changes in the cardiovascular system, diencephalic-hypothalamic structures of the brain, kidneys, as well as an increase in exposure to bad habits, an increase in body weight and a decrease in physical activity play a role. .

The consequence of physiological restructuring of the structure and function of the heart and blood vessels is the increasing frequency of isolated systolic arterial hypertension (ISAH) with aging and an increase in the proportion of people with low-renin form of hypertension among the population of “age-related hypertensives”.

A feature of the pathogenesis of hypertension in elderly patients is its development or progression against the background of a significant number of accumulated metabolic disorders and concomitant diseases that have developed by this age period, which increase the overall risk of developing cardiovascular complications and death. Separately, it is worth noting the contribution to the increase in blood pressure of drug therapy received by elderly and elderly patients for multiple comorbid pathologies, as well as their uncontrolled self-administration of a number of medications due to the formation of geriatric syndromes (metamizole (analgin), non-steroidal anti-inflammatory drugs (NSAIDs), sympathomimetics in the eyes and nasal drops, etc.).

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