MODERN THERAPY OF ARTERIAL HYPERTENSION

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ABSTRACT

Arterial hypertension is a persistent increase in resting systolic blood pressure (≥ 130 mm Hg) and/or diastolic blood pressure (≥ 80 mm Hg). An increase in blood pressure without a known cause (primary, essential hypertension) is the most common. Hypertension with an identified cause (secondary hypertension) is usually due to primary aldosteronism. Sleep apnea, chronic kidney disease, obesity, or renal artery stenosis are other causes of secondary hypertension. Usually, symptoms appear only with severe or prolonged course. Diagnosis is based on sphygmomanometry. Diagnosis can determine the cause, evaluate organ damage, and identify other cardiovascular risk factors. Treatment includes lifestyle changes and medications, including diuretics, angiotensin-converting enzyme (ACE) inhibitors, angiotensin II receptor blockers, and calcium channel blockers.

Hypertension is defined as systolic blood pressure ≥ 130 mmHg. or diastolic blood pressure ≥ 80 mmHg. or taking medications to treat high blood pressure. Nearly half of US adults have hypertension. Many of these people do not know that they have hypertension. Approximately 80% of adults with hypertension are

recommended drug therapy and lifestyle changes, but only about 50% of patients with hypertension receive treatment.

Only about 1 in 5 adults with hypertension have their blood pressure (BP) under control. Even with medical treatment and lifestyle changes, only 26% of patients achieve blood pressure control, and among adults on antihypertensive therapy whose blood pressure is not controlled, almost 60% of blood pressure is ≥ 140/90 mmHg.

Keywords: *neurohumoral system, epidemiological monitoring, Essential hypertension, symptomatic arterial hypertension, ACE inhibitors, dihydropyridines.*

The primary factor is hereditary predisposition. According to the concept of Yu.V. Postnov, it consists in widespread violations of the ion-transport function and the structure of the cytoplasmic membrane of cells. Under these conditions, the preservation of the specific function of cells is ensured by the mechanism of cellular adaptation associated with the regulation of calcium metabolism, with a change in hormonal-cellular relationships, with an increase in the activity of neurohumoral systems (hypothalamic-pituitary-adrenal, renin-angiotensin-aldosterone, insular).

Over the past few years, according to the epidemiological monitoring of hypertension in Uzbekistan, many patients do not regularly take treatment. Only 23.2% of women and 15.4% of men take antihypertensive drugs daily. although the effectiveness of treatment in the population does not exceed 30%. It seems relevant to search for new and optimize existing pharmacological approaches to the treatment of hypertension in order to achieve its higher efficiency. The effect of antihypertensive drugs on the prognosis of hypertensive patients depends not only on the ability to reduce blood pressure and maintain it at the target level, but also on the ability to inhibit or reverse the development of remodeling in target organs.

When examining patients with arterial hypertension, attention should be paid to the etiology of the disease. Essential hypertension (hypertension) accounts for 90-

95% of cases of hypertension. In other cases, secondary, symptomatic arterial hypertension is diagnosed: renal (nephrogenic) - 3-4%, endocrine - 0.1-0.3%, hemodynamic, neurological, stress, caused by the intake of certain substances (iatrogenic) and hypertension in pregnant women, in which an increase in blood pressure is one of the symptoms of the underlying disease. Among iatrogenic hypertension, those caused by the intake of biologically active additives and drugs stand out. Women taking hormonal contraceptives are more likely to develop hypertension (this is especially noticeable in obese women, women who smoke, and older women).

The main classes of antihypertensive drugs were: diuretics, β-blockers, slow calcium channel blockers (CCBs), angiotensin converting enzyme inhibitors (ACE inhibitors), angiotensin (AT) receptor blockers (ARBs) II. But in modern therapy, to achieve the target blood pressure, combined antihypertensive therapy is necessary.

Current guidelines on hypertension emphasize the importance of using rational combined regimens to ensure reliable BP control. A rational combination involves the use of drugs of different classes (with different mechanisms of action) to obtain an additional hypotensive effect and reduce adverse events. Patients receiving combination therapy are less likely to refuse treatment than patients who are prescribed any monotherapy. Another advantage is the presence of physiological and pharmacological synergy between drugs of different classes, which can not only contribute to a more pronounced decrease in blood pressure, but also cause fewer side effects and provide more pronounced benefits than one drug. It is irrational to combine drugs with the same mechanism of action and a similar spectrum of adverse events, for example, a combination of a thiazide diuretic and β-blockers. The combination of drugs of these 2 classes can lead to an increased risk of developing hyperglycemia. An example of a favorable combination is the combination of BMCC with ACE inhibitors, which leads to a decrease in the severity of edema associated

with the use of dihydropyridines. In addition, the combination of non-dihydropyridine CBCA with an ACE inhibitor can potentially enhance the positive effect of the latter on vascular elastic properties. In addition, in recent years, a new class of drug has appeared - a direct renin inhibitor (RIR) - aliskiren, with a different effect on the RAAS than ACE inhibitors and ARBs. ACE inhibitors block ACE, ARBs block AT II receptors, and PIRs act on renin to decrease plasma renin activity. All these groups of drugs reduce blood pressure and prevent damage to the kidneys, heart, and blood vessels. With the appointment of ACE inhibitors and ARBs, the release of renin from the kidneys increases, the plasma activity of renin increases, and the vicious cascade starts again. Associated with this is the elusive effect on ACE inhibitors. Aliskiren is the only selective PIR to date. Aliskiren, by binding to the active center of the renin molecule, prevents the conversion of angiotensinogen into AT I. Aliskiren's molecule is stable, has a non-peptide structure and a high affinity for human renin. Aliskiren acts at the starting point of RAAS activation, reducing plasma renin activity and preventing the formation of AT I from angiotensinogen, the vicious cascade is not triggered and the feedback mechanism is not activated. An increase in plasma renin activity is a proven independent risk factor for cardiovascular mortality and complications. Aliskerin should be administered orally at a dose of 150 mg 1 time / day. With persistent hypertension, it is possible to increase the dose to 300 mg per day. Highest daily dose: 300 mg. In addition, when examining pediatric patients, it is necessary to exclude congenital adrenal hyperplasia and renin-producing tumors. According to clinical guidelines in the treatment of resistant forms of arterial hypertension, combination therapy is the main one.

Conclusion. Modern ideas about arterial hypertension, formed on the basis of epidemiological studies, include such concepts as target blood pressure, which is the same for patients of all ages - less than 140 and 90 mm Hg. Art., and for patients with diabetes - less than 140 and 85 mm Hg. Art., risk reduction, risk assessment of CVD and their complications.

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