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Methods for Treating Arterial Hypertension in Metabolic Syndrome

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Annotation: This review summarizes the literature evidence on humoral disturbances in arterial hypertension (AH), as well as on AH interrelationship with individual components of metabolic syndrome (MS). Based on the results of multi-centre randomised trials, the rationale for the use of antihypertensive agents with favourable metabolic profile is demonstrated, in particular, for antagonists of slow calcium channels, angiotensin-converting enzyme inhibitors, and selective imidazoline receptor agonists.

Key words: Arterial hypertension, metabolic syndrome, antihypertensive therapy.

It is known that arterial hypertension (AH) is accompanied by various endocrine and metabolic disorders, such as increased activity of the renin-angiotensin-aldosterone system (RAAS), hypercholesterolemia (HCS), insulin resistance (IR), with compensatory hyperinsulinemia vascular diseases (CVD). There is no doubt that the origin and course of hypertension depends on a complex ratio of activation of a number of pressor and depressor mechanisms, as well as overloads of the mechanisms of volume regulation and the state of renal hemodynamics. Pressor factors or factors that directly or indirectly increase blood pressure (BP) include: vasopressin, angiotensin II (AT II), catecholamines, endothelin (ET), prostaglandins F2, histamine, thromboxane A2 (TxA2), leukotrienes C4 and D4 and others. Vascular tone is also influenced by osmoregulatory factors and the state of the vascular wall (ionic permeability of its membrane, intimal proliferation). The action of the listed vasoactivators is opposed by vasodepressor systems - prostaglandins E, endothelial relaxing factor, nitric oxide (NO) system [2]. There is evidence that IR and increased sensitivity to Na + are related. Almost 2/3 of patients with primary hypertension have IR from the very onset of the disease and regardless of their body weight (BM). Other indicators of IR are hyperuricemia, hypertriglyceridemia (HTG) and fatty liver, hereditary burden of CVD and the thickness of the subcutaneous fat layer [3]. According to some reports, the risk of developing diseases associated with obesity (Ob), to a greater extent (st.) Depends on the nature of the distribution of fat and to a lesser extent on st. Ozh. It turned out that vascular complications are more typical for patients with predominant fat accumulation on the trunk and in the abdominal cavity (visceral or upper obesity) and less typical for patients with predominant fat deposition on the buttocks and thighs (gluteofemoral or lower obesity). Increases in blood pressure and IR are more often observed in patients with abdominal obesity. The mechanism of this phenomenon is associated with the fact that adipocytes of intra-abdominal tissue are initially more sensitive to the lipolytic action of catecholamines and less sensitive to the anti-lipolytic action of insulin [17]. It is believed that the mechanisms underlying the differences in fat distribution are associated with impaired metabolism of glucocorticoids and androgens. A prolonged increase in glucose concentration (in addition to disturbing the balance of redox processes, enhancement of free radical reactions, glycolization) leads to an increase in the biosynthesis of such components of the basement membrane of vessels as fibronectin, type IV collagen and laminin, which probably causes its thickening (an important pathomorphological a sign of changes in the vascular wall). As a result, in people with obesity, the probability of developing hypertension is 50% higher than in people with normal body weight (BM), and the target organs of hypertension in them are affected much earlier, and their changes are much more pronounced than in hypertension without obesity. It was also found that in middle-aged patients with excessive MT (BMI), the risk of developing hypertension is increased 3 times, and in young people - 6 times [4]. Aspects of the relationship between AH syndrome and factors such as BMI, disorders of lipid and purine metabolism, IR, glucose intolerance (IGT) are presented in numerous literary sources. The conjugation of hypertension was detected both with individual of the

listed factors, and with their combination [2-4]. These connections have been studied so much that the results allow us to speak about the dependence of these processes. These parallels are most clearly generalized and integrated in the so-called metabolic syndrome (MS). The incidence of diseases associated with IR has increased especially over the past 20 years. The explanation for this is seen in the strengthening of the action of such factors characteristic of the modern lifestyle as BMI, physical inactivity, unsuccessful heredity, excessive consumption of saturated fats with food. The complete picture of the MS is explained by the joint presence of: IR; BMI with predominant fat deposition on the trunk; essential hypertension; a moderate increase in the level of total cholesterol (TC), with a reduced content of high-density lipoprotein cholesterol (HDL-C); Tretyakov Gallery; NTG, increasing to a clear diabetes mellitus (DM); hyperuricemia (gout). At the same time, it is believed that these characters have a common origin, in which the primary (probably genetically determined) tissue IR plays a key role. Due to the combination of all these features, patients with MS have an extremely high risk of developing atherosclerosis and CVD. In the aspect of the problem of the relationship between the above signs, it should be said that even with isolated essential AH, GI can occur [8]. It is known that antihypertensive therapy (AHT) affects metabolic disorders in hypertensive patients. Under the influence of therapy with some β-adrenergic receptor blockers (β-AB), the progression of IR, an increase in insulin levels and a decrease in the rate of metabolic clearance of glucose in the blood serum in comparison with the indicators before the start of treatment were revealed. Unfortunately, the peripheral antiadrenergic effect of β-AB, which is responsible for the favorable cardiovascular consequences of their use, does not provide positive effects on carbohydrate and lipid metabolism and may even contribute to the progression of metabolic disorders and an increase in MT. At the same time, in patients receiving therapy with slow calcium channel blockers (AAs) or angiotensin converting enzyme (ACE inhibitors) inhibitors, a decrease in insulin levels and the degree (st.) Of IR was found [7]. A favorable metabolic profile of ACE inhibitors and AAs was shown in the large Antihypertensive and Lipid-Lowering treatment to prevent Heart Attack Trial (ALLHAT) study [12], where lisinopril and amlodipine provided fewer new cases of diabetes in comparison with chlorthalidone. In another study, the number of obese patients who managed to achieve normalization of blood pressure in the lisinopril group was significantly higher than in the group. hydrochlorothiazide (HCT), although the latter was used in doses up to 50 mg / day. [15]. In this case, the level of glucose in the blood serum in gr. lisinopril decreased, and in gr. Ght, on the contrary, increased. Potassium level in gr. lisinopril practically did not change, while in gr. Гхт it has significantly decreased. This gives grounds to recommend lisinopril for the treatment of hypertension in obese patients. The metabolic neutrality of dihydropyridine AAs makes their combination with an ACE inhibitor one of the most promising for the treatment of patients with hypertension and MS [5,12]. Thus, there is a renewed interest in drugs that, in addition to their own antihypertensive effect, have a beneficial effect on carbohydrate and lipid metabolism within MS. This becomes even more relevant, taking into account the fact that none of the main classes of antihypertensive drugs (AGP) provides a pathogenetic effect on the formation of IR, namely: suppression of hyperactivity of the sympathetic nervous system (SNS) [9]. Meanwhile, the relationship between SNS and essential hypertension in the entire spectrum of its clinical manifestations is obvious, the mechanisms of its pathogenesis suggest either an increase in sympathetic activity or a decrease in parasympathetic, but with an absolute or relative increase in hypersympathicotonia. It has been shown that SNS activation is important not only in the early stages of AH formation, but also contributes to the development of cardiovascular risk (CVR) in the future [11]. One of the evidence in favor of the role of SNS activation in hypertension may be the lack of it in secondary forms of hypertension, which, in turn, may be one of the explanations for the absence of secondary metabolic disorders in symptomatic hypertension. A high-calorie diet is known to increase SNS and BP function. An additional and very powerful factor contributing to an increase in SNS activity is a sedentary lifestyle [13]. In all likelihood, the common link between the negative aspects of the modern lifestyle and the development of these RFs is the hyperactivity of the SNS [9]. Consequently, a therapeutic approach that provides a decrease in SNS activity seems to be very attractive for reducing the negative effect of RF and reducing the rate of occurrence and progression of hypertension and its cardiovascular complications [15]. Centrally

acting drugs that primarily affect the sympathetic link in the regulation of vascular tone, reducing peripheral vascular resistance, were originally created based on the role of SNS hyperactivity in the pathogenesis of hypertension [6]. But the large number of side effects (AEs) characteristic of the stimulation of central α2-adrenergic receptors limited the widespread use of first-generation central drugs such as clonidine and guanfacine. The new generation of drugs, represented by rilmenidine and moxonidine, is favorably distinguished by selectivity for imidazoline receptors and, accordingly, a weak expression of PE, which provides a "renaissance" of this group. medicines [18]. Selective agonists of imidazoline receptors (AIRs) are now increasingly called the drugs of choice in the treatment of patients with MS, since they have shown a decrease in IR and a beneficial effect on lipid metabolism [10]. Recent studies have shown that rilmenidine not only does not have an adverse effect on lipid and carbohydrate metabolism, but reduces the manifestations of IR and IGT and, with prolonged use, leads to changes in the blood lipid spectrum that are favorable in relation to the risk of cardiovascular complications [1,14]. In this regard, lipid metabolism disorders, along with IR, can serve as indications for the use of moxonidine and rilmenidine as mototherapy for hypertension or in combination with other metabolically neutral drugs.

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