FACTORS CAUSING ESSENTIAL HYPERTENSION AND COURSE OF THE DISEASE

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Summary. Modern ideas about the participation of the inflammatory process in pathogenesis are considered hypertension. The role of chronic subclinical vascular inflammation as a binder has been noted links of essential hypertension, endothelial dysfunction, atherosclerosis. Data presented reflecting the importance of the characteristics of concomitant pathology in the formation of the inflammatory phenotype in patients with hypertension. The significance of metabolic syndrome from the point of view of its participation in maintaining the activity of the systemic inflammatory process. Thus, the proportion of people with arterial hypertension that meets criteria for metabolic syndrome in selected populations reaches 80%. A key role in the pathogenesis of this syndrome belongs to insulin resistance, which induces the development of compensatory hyperinsulinemia, hypertriglyceridemia, arterial hypertension and visceral (abdominal) obesity. A frequent companion to metabolic syndrome is a disorder purine metabolism. It has been established that elevated levels of uric acid in the blood serum are an important independent risk factor for systemic hypertension and cardiovascular events. High concentration in serum uric acid may induce the development of hypertension through stimulation of oxidative stress, endothelial dysfunction, activation of the renin-angiotensin-aldosterone system. Degree uricemia correlates with blood levels of C-reactive protein, fibrinogen, interleukin 6 and factor tumor necrosis. The proinflammatory effects of renin-angiotensin-aldosterone hormones are considered systems for essential hypertension and metabolic syndrome. Data on pleiotropic anti-inflammatory effectiveness of antihypertensive therapy based on the use of antagonists angiotensin II and aldosterone receptors.

Key words: essential hypertension, inflammation, risk factor, atherosclerosis, endothelial dysfunction, oxidative stress, metabolic syndrome

Essential arterial hypertension (AG) is not only a very common independent disease, but also the most important risk factor for the development of associated with atherosclerosis, damage to the cardiovascular systems. Over the past two

decades in the problem is actively discussed in scientific publications inflammation in cardiovascular diseases, its pathogenetic and prognostic significance. It has been firmly established that the nonspecific subclinical inflammation observed in hypertension is associated with damage target organs – left ventricular hypertrophy , atherosclerosis of the aorta and large arteries, renal dysfunction. The level of high-sensitivity C-reactive protein, which is one of markers of chronic subclinical inflammation, correlates with the risk of developing non-fatal and fatal cardiovascular complications. It is argued that increased blood pressure (BP) level is an element of the inflammatory process.

The problem of inflammatory changes in patients Hypertension cannot be considered in isolation. The inflammatory phenotype is characteristic of concomitant hypertension conditions (atherosclerosis, metabolic syndrome, heart failure). By in the figurative expression of Academician A.L. Myasnikova– author of the fundamental monograph “Hypertension and Atherosclerosis”, published in 1965, “atherosclerosis follows hypertension like a shadow behind the person" . In accordance with modern views, along with dyslipidemia, oxidative stress, endothelial dysfunction and inflammation represent the basis for the development and progression of atherosclerosis . Endothelial cells play an important role in the regulation of vascular tone, proinflammatory and prothrombotic processes through the production of numerous factors, including nitric oxide, cytokines, prothrombotic agents and anticoagulant factors. In normal conditions, there is a balance in the secretion of these substances, which ensures the integrity of the vascular walls. Endothelial dysfunction is characterized by a predominance of vasoconstriction, adhesion leukocytes, readiness for thrombus formation. Presence of cardiovascular risk factors, such as hypertension, diabetes, hypercholesterolemia, smoking disrupts the structural and functional state of endothelial cells. Long-lasting against the background of low-grade inflammation, endothelial dysfunction is accompanied by a decrease in the elasticity of the aorta and large arteries and an increase in pulse wave velocity in patients with hypertension.

Clinical and experimental studies have made it possible to establish a relationship between high blood pressure values and various markers and mediators of inflammation, thereby showing that

hypertension is a subclinical inflammatory process. L.E. Bautista et al. [12] showed that essential hypertension, the incidence of which averages 30% among adults population, is much more widespread among subjects with finding the level

of C-reactive protein in the top quartile (60% of the population) relative to subjects with C-reactive protein levels in the lowest quartile (14% of the population).

This study also established correlation of C-reactive protein content in the blood and values of systolic and pulse blood pressure, which turned out to be independent of the presence of other factors cardiovascular risk (age, gender, aggravated heredity, body mass index, fasting glycemia, sedentary lifestyle) [12]. Association of blood pressure values within the normotensive and hypertensive ranges with the level of C-reactive protein has been confirmed by a number of other researchers even after adjustment by known factors cardiovascular risk. Own observation of a group of young men with prehypertension and no history of cardiovascular disease diseases classified according to the Framingham scale to low or moderate cardiovascular risk, showed differences not only in average daily values blood pressure, heart rate, atherogenic fractions of lipoproteins, but also the values some laboratory markers of inflammation.

In general, in the pathogenesis of hypertension the problem of chronic subclinical inflammation. The presence and severity of the inflammatory phenotype in essential hypertension depends significantly on the spectrum cardiovascular risk factors, comorbid diseases, conditions of neurohumoral regulation of blood circulation (inadequate consumption table salt, dyslipidemia, hyperuricemia, insulin resistance, RAAS hyperactivity, MS, diabetes mellitus, atrial fibrillation). The inflammatory component serves as a unifying link of hypertension, endothelial dysfunction and atherosclerosis. An integrated approach to the treatment of hypertension based on modification lifestyle, correction of concomitant pathology, the use of RAAS blockers promotes not only to achieve stable normotension, but also reducing the activity of the inflammatory process.

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