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PATHOGENETIC MECHANISMS OF METABOLIC DISORDERS IN PATIENTS WITH HYPERTENSION AND OBESITY.

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In many countries, the prevalence of metabolic syndrome, obesity and type 2 diabetes is increasing, forming a dangerous, clinically difficult situation that may slow or even slow down progress in the treatment of vascular diseases that has been observed in recent years. At present, insulin resistance is considered as the key etiological category in the pathogenesis of metabolic syndrome [3]. As a cornerstone in the cascade of metabolic disorders formation, metabolic syndrome plays a major role in the formation of arterial hypertension, dyslipidemia, kidney, and abnormalities in the function of adipose tissue [1]. It is known that insulin resistance (IR) is interrelated with the distribution of fatty tissue in an organism and causes the formation of visceral obesity. Evidence of the cause-and-effect relationship of obesity with severe metabolic disorders and cardiovascular diseases determine the importance of this problem for modern health care [5].

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As recent studies have shown, adipose tissue is also an endocrine gland [2], which secretes a significant amount of hormones and biologically active peptides, most of which have an effect on the increased severity of IR. The adipose tissue hormones, with the exception of adiponectin, decreases the sensitivity of peripheral tissues to insulin, which is accompanied by an increase in the severity of insulin resistance that is involved and is the main link in the pathogenesis of type 2 diabetes [2]. As you know, insulin possesses the qualities of the vasodilator due to the ability to suppress the potential-dependent flow of Ca^{2+} ions. This leads to stimulation of glucose transport and its phosphorylation to the formation of glucose-6-phosphate, which then activates the transcription of calcium adenosine triphosphatase (Ca-ATPase) and ultimately reduces the content of intracellular Ca^{2+} and vascular resistance. When obesity against the background of insulin resistance, these mechanisms are disturbed, and this leads to increased vascular resistance. It is important to note that the decrease in body weight during obesity is accompanied by a marked decrease in vascular resistance and mean blood pressure levels. There are also data suggesting that fatty tissue production of inflammatory mediators increases the risk of thrombosis. In patients with android type of obesity on the background of a lipid peroxidation oxidation disturbances platelet aggregation amplication is observed, and these disoders disappear with weight loss [2].

The increase in the prevalence of obesity among the population, the increase in the number of patients with complicated forms of the disease (carbohydrate metabolism disturbances, insulin resistance, dyslipidemia, arterial hypertension, ovarian hyperandrogenism) explains a significant interest in understanding the physiology of adipose tissue, and in the particular role of adipokines in the development and progression of metabolic disoders in obesity. Many elements of the metabolic syndrome (such as arterial hypertension, dyslipidemia, type 2 diabetes) are well-established risk factors for cardiovascular diseases. The role of insulin resistance and hyperinsulinemia as independent cardiovascular risk factors remains not fully proven. It is clear that the most insulin-resistant part of the population has

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an increased risk of cardiovascular disease, however, the extent to which it depends on generally accepted risk factors (or they are mediators) remains unclear.

A number of researchers believe that obesity can actually be considered a condition of "chronic inflammation". Of course, there is a relationship between obesity and the increased risk of developing cardiovascular complications to a large extent it is due to the high level of inflammatory mediators. At present, the role of resistin as a substance that potentially binds obesity to diabetes is actively discussed. It is believed that resistin can reduce the sensitivity of peripheral tissues to insulin action. With the action opposite adiponectin, the level of resistin positively correlates with the values of proinflammatory cytokines, the level of IR, the degree of calcification of coronary arteries [4, 7], which allows us to consider resistin as a possible factor that binds together metabolic disorders, inflammation and atherosclerosis . Taking into account the participation of resistin in stimulating the mechanisms of inflammation, activation of the endothelium and proliferation of smooth muscle cells in vessels, this cytokine is also interesting as a possible link between metabolic and vascular disorders. Some researchers suggest to consider it as a marker or even an etiological factor for the development of cardiovascular disease in metabolic syndrome [6].

An important place in the modern neuroendocrine theory of metabolic syndrome and cardiovascular system diseases development is given by the TNF-α, which normally plays a fundamental physiological role in immunoregulation, but in some cases, it can perform pathological action, taking part in the development and progression of inflammation, microvascular hypercoagulation, hemodynamic disturbances and metabolic exhaustion at various diseases of a person as an infectious, and not infectious cause. It has been shown that TNF-α disturbs insulin signals in muscle and adipose tissue and thus contributes to the development and progression of insulin resistance [3, 2, 6]. The values of TNF-α are positively correlated with insulin resistance, which makes it possible to use this cytokine as an early marker for the development of diabetes mellitus [6].

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Therefore, it is important to study changes in the concentration of these cytokines in patients with arterial hypertension with type 2 diabetes or IR, namely at different stages of the disease progression, from the stage of disturbance of glucose regulation up to type 2 diabetes, which will enable doctors and scientists to overcome these diseases together, yet at the stage of laboratory changes to the occurrence of such serious complications as myocardial infarction and stroke, using a change in therapeutic tactics.

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