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Features Antihypertensive Therapy Obesity

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ABSTRACT

Obesity negatively affects structure of renal tissue and increases the risk of developing renal failure and progression arterial hypertension. Adipose tissue is also a source of synthesis of all components renin-angiotensin system and can actively produce angiotensin.

KEYWORDS

Obesity, antihypertensive therapy, renin-angiotensin system.

INTRODUCTION

The main factors in the development of arterial hypertension against the background of obesity are hyperinsulinemia, hyperleptinemia, hypercortisolemia, renal dysfunction, altered structure and function of blood vessels, increased activity of the sympathetic and renin-angiotensin systems, decreased activity of natriuretic hormone

The main mechanisms of arterial formation hypertension in obesity:

- Changes in renal hemodynamics,
- Structural changes in the kidneys,

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- Activation of the sympatho-adrenal nervous system,
- Insulin resistance,
- Free fatty acids (FFA),
- Leptin.

Let's consider the listed mechanisms in more detail.Renal hemodynamic changes. A direct relationship was found between weight gain and sodium retention.The main mechanism of sodium and fluid retention during arterial hypertension against the background of obesity is the activation of the renin-angiotensin system (RAS), the sympathoadrenal system (SAS) and hyperinsulinemia . At the initial stage, a compensatory decrease in renal vascular resistance develops, an increase plasma flow through the kidneys, renal filtration rate,which partially prevents enhanced sodium reabsorption.

However, subsequently, against this background, the production of angiotensin II and cytokines in combination activations. Obese patients have an inadequately low natriuretic response. These changes increase the "stress"the walls of the glomeruli and against the background of other risk factors (hyperlipidemia and hyperglycemia) and quickly lead to the development of glomerulosclerosis, proteinuria, microalbuminuria and functional nephron failure. Early renal hyperfiltration in obesity is similar to that of type 1 diabetes mellitus.

Activation of the sympathy-adrenal nervous system.The consequence of SAS hyper activation is a number of disorders that are of a diverse nature - insulin resistance, increased afferent innervations of the kidneys with an increase in interregnal pressure, which leads to the activation of renal mechanoreceptors, an increase in the level of free fatty acids, angiotensin II, leptin, potentiation of the sensitivity of central chemoreceptor's and impaired bar reflex regulation. The activation of muscles and kidneys in obesity was confirmed by the icroneurographic method. I wonder what drugs suppressing central sympathetic activity,cause a greater decrease in obese patients, than in non-obese patients. Insulin resistance.

Currently, hyperinsulinemia is considered a key factor in the development of hypertension in obesity. It is known that obesity significantly increases the level of insulin, which is due to the need to maintain the metabolism of carbohydrates and fatty acids at a higher level, and this occurs against the background of insulin resistance of peripheral tissues. Currently available data suggest that insulin resistance is impaired insulin-mediated vasodilatation, which contributes to an increase in.

Free fatty acids. It's believed that high level of free fatty acids due to an increase in sympathetic activity, or a vasospatic effect, realized through adrenergic receptors. With visceral obesity, too much free fatty acids enter the liver, which activates the hepatic afferent pathways, increases activity and promotes the development of insulin resistance. With obesity, the level of free fatty acids is about 2 times higher than in non-obese people. Leptin is a peptide consisting of 167 amino acids. It has various mechanisms of press or action and therefore plays an important role in the pathogenesis of both obesity and arterial hypertension. Leptin is secreted by white adiposities and its level directly correlates with the amount of adipose tissue and is always elevated in obese people. Leptin levels are higher in women than in men. Leptin crosses the blood-brain barrier into the central nervous system through endocytosis, where it binds to

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receptors (Ob-R) in the lateral and medial regions of the hypothalamus.

The binding of leptin to receptors causes their activation, which ensures the regulation of energy balance through decreased appetite and increased energy expenditure due to Evidence that leptin reduces appetite and weight regulation, supported by both experimental and clinical data. With impaired ability to synthesize leptin or existing leptin receptor mutations always develop severe obesity. The hypertensive effect of leptin is enhanced by endothelial dysfunction, which is almost always takes place in obesity. Press or effect of leptin almost completely disappears against the background of and adrenergic blockades, adipose tissue and activity of the rennin-angiotensin system In obesity, RAS is activated against the background of an increase in fluid volume and sodium retention.

Distinctive feature is a significant increase in the level aldosterone which may be related to the release specific hepatic factor (possibly oily acid), which has a stimulating effect on its synthesis. It is known that adipose tissue has its own ASD, which plays an important role in the functioning of adipose tissue. Fabrics. Adiposities are capable of synthesizing all components of the RAS. They also stimulate Ang II receptors, increasing their affinity to peregrine Ang II. It is possible that local Ang can be a growth factor for fat cells. It has been shown that at an increased level 1-hydroxy steroid dehydrogenate involved in the formation of cortical, arterial hypertension with all signs of metabolic syndrome. Naturally, all of the humeral disorders described above in arterial hypertension and obesity lead to to changes in the cardiovascular system:

- Vascular changes;
- Changes in the heart;
- Changes in microcirculation;
- Markers of inflammation

Obesity changes occur at the cellular and molecular levels, leading to increase vascular tone. Insulin is normal possesses the properties of a vasodilator due to the ability suppress the voltage-dependent flow of Ca2 + ions. This leads to the stimulation of glucose transport and its phosphorylation with the formation of glucose-6-phosphate, which then activates the transcription of Ca-ATP-ase and ultimately reduces the level of intracellular calcium and vascular resistance. In obesity against the background of insulin resistance, these mechanisms are violated, and this leads to an increase in vascular resistance. A decrease in the elasticity of large vessels was revealed. data of nuclear magnetic resonance, which directly correlated with an increase in the mass of abdominal visceral fat. It is important to note that weight loss with obesity, it is accompanied by a pronounced decrease vascular resistance.

Thus, despite the fact that all antihypertensive drugs achieve the goal and lower blood pressure, in the treatment of hypertension in combination with obesity, the use of those drugs that have an adverse effect on lipids and carbohydrate metabolism, that is, diuretics and beta-blockers, should be limited. It is preferable to prescribe drugs, the point of application of which is the RAAS, primarily ACE inhibitors and antagonists of antiotensin II receptors. This is due to the fact that these drugs: 1) effectively reduce blood pressure in hypertensive patients with obesity; 2) do not cause lipid side effects; 3) favorably modify glucose metabolism, reducing insulin resistance; 4) in connection with the existence of the relationship that exists between the RAAS and the SAS, they also affect other obesity disorders caused by hyperadrenergy.

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REFERENCES

1. Nedogoda S.V. Obesity and arterial hypertension: theory and practice of choosing the optimal antihypertensive drug. M .: Medikom, 2012.
2. Bergman RN, Van Citters GW, Mittelman SD et al. Central role of adipocyte in the metabolic syndrome. J Invest Med 2001
3. Chu NF, Spiegelman D, Hotamisligil GS et al. Plasma insulin, leptin, and soluble TNF receptors levels in relation to obesity-related atherogenic and thrombogenic cardiovascular disease factors among men. Atherosclerosis 2001,
4. Drenick EJ, Bale GS, Seltzer F, Johnson DG. Excessive mortality and cause of death in morbidly obese men. JAMA 1980,
5. Esler M The sympathetic system and hypertension. Am J Hypertens, 2000,
6. Gorzelniak K, Engeli S, Janke J et al. Hormonal regulation of the human adipose-tissue renin-angiotensin system: relationship to obesity and hypertension. J Hypertens, 2002,
7. Gupta AK, Clark RV, Kirchner KA. Effects of insulin on renal sodium excretion. Hypertension 1992,
8. Hall JE. Mechanisms of abnormal renal sodium handling in obesity hypertension. Am J Hypertens, 1997,
9. Kuo J, Jones OB, Hall JE. Inhibition of NO synthesis enhances chronic cardiovascular and renal actions of leptin. Hypertension 2001,
10. Licata G, Scaglione, Capuana G, Parrinello G, Divincenzo D, Mazzola G. Hypertension in obese subjects: distinct hypertensive subgroup. J Hum Hypertens, 1990,