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Hypertension and Obesity

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Abstract: *Hypertension and obesity are most often mixed factors of vascular risk. The relationship between obesity and hypertension demonstrated that the increase in body mass index (BMI) relative to the height significantly increases the prevalence of hypertension in different age groups for both sexes.*
Keywords: *hypertension, obesity, overweight, insulin, leptin.*

I. Introduction

It is known that the presence of risk factors (RF) plays a big role in the development of noninfectious diseases. So far, more than 200 risk factors are known, and their number is increasing annually. The results of the INTERHEART study identified 9 risk factors, playing important role in the development of acute myocardial infarction: lipid storage disease, diabetes, stress, hypertension (HT), obesity, insufficient fruit and vegetable consumption, sedentary lifestyle and alcohol abuse. The results of the study indicate a high prevalence of overweight or obesity among men of active working age [1]. It is not only the problem of economically developed countries, where risk factors of overweight and obesity occur twice as often, but of all other countries. Hypertension and obesity are most often mixed factors of vascular risk. The relationship between obesity and hypertension was documented in the Framingham Heart Study, which demonstrated that the increase in body mass index (BMI) relative to the height significantly increases the prevalence of hypertension in different age groups for both sexes. It is remarkable that one of the important factors of hypertension development is a recent weight gain; According to the Framingham Heart Study, approximately 70% of newly diagnosed hypertension cases was associated with recent weight gain or obesity [4,7,9].

Increase in body mass by 4.5 kg is accompanied by 4.4 mm Hg of increase in systolic pressure in men, which is 4.2 mm Hg in women; increase in body mass by 5% within 4 years makes the hypertension risk to rise up to 30%. As reported by WHO, in Western countries, hypertension increase is caused by overweight in one third of patients, and this level reaches 60% in men up to 45 years old.

Obesity raises a number of hemodynamic changes, in particular, increase in the volume of circulating blood, stroke volume, and cardiac output at relatively normal vascular resistance [7, 8]. It is assumed that hypertension in obese patients is mainly associated with increased cardiac output during "inappropriately normal" peripheral resistance [7, 9]. Such hemodynamic status has a stimulating effect on the two antagonistic regulatory systems that control blood volume and peripheral resistance - renin-angiotensin-aldosterone system (RAAS) and natriuretic peptide system of the heart. Their impaired regulation could significantly explain the high cardiac output in obese patients with hypertension. Moreover, these cardiovascular regulatory systems are involved in the metabolic changes associated with overweight in cardiovascular diseases [10, 11].

However, in patients with visceral obesity, the level regulation is disturbed in system circulating components of RAAS [9]. Despite increased hypertension, sodium and fluid retention, as well as the increase in the volume of circulating blood, plasma and aldosterone renin activity remains normal or even slightly elevated [13]. Such dysregulation of RAAS in obesity can be the result of increasing formation of RAAS components, and/or secondary growth of their concentration is caused by defects in the system of natriuretic peptides (NP). It is assumed that fatty tissue redundancy can lead to an increase in plasma clearance of NP, thus reducing their activity in the kidneys and facilitating the development of hypertension. In kidneys, the activity of NP is manifested in several ways: this is modulation of resistance of renal vessels, reducing of renal pressure, increasing of glomerular filtration and inhibition of the sodium reabsorption. The NP also reduce the activity of RAAS, peripheral vascular resistance and thereby reduce the hypertension. The role of NP in obesity was studied in several clinical studies described by A. Mark [11]. According to one of those studies, weight loss during caloric restriction leads to abundant natriuresis, diuresis, and decrease in hypertension. In obesity, especially, of visceral type, Renin activity in plasma is retained in normal or slightly elevated level, where the levels of antionsinogen and autoantibody of II. type are increased [15]. Regardless of the fact that the basic characteristics of cardiovascular system in young people with overweight and obesity remain within age standards, there is a tendency towards the development of cardiac and vascular remodeling in the form of the increased intima-media thickness of common carotid artery (IMT of CCA), carotid-femoral pulse wave velocity (PWV) and the development of concentric left ventricular hypertrophy (LVH). Interaction was not found between BMI, level of metabolic disorders and increasing blood pressure, endothelial function indicators [12].

Determining the type of LVH is important to assess the risk of CVD. Especially in patients with hypertension and metabolic syndrome (Hua Q. et al., 2006, Chinali M., 2007). A direct correlation of obesity and dyslipidemia was identified with growth of IMT of CCA (Mavri A. et al., 2001, Stevens J. et al., 2002, Wunsch R. et al, 2006) and a change in elastic properties of arteries (Wildman R.P. et al., 2003, Im J.A. et al., 2007, Davy K.R. et al., 2004, Cruickshank K. et al., 2002).

With obesity, especially during its abdominal variant, the activation of sympathetic nervous system (SNS) is observed very often [9, 26, 27]. NAS **(Normotensive Aging Study)** studies found increase of noradrenaline in urine, which is proportional to body mass index [48]. When the weight reduces, SNS activity decreases [9, 27]. Increased activity of SNS in obesity contributes to the presence of hyperinsulinemia (HI) and insulin resistance (IR). IR increases plasma insulin level, which in turn is directly related to the increase in the level of catecholamines and plays an important role in the pathogenesis of hypertension [11,18] by sympathetic stimulation of the heart, blood vessels and kidneys [7]. IL promotes the development of hypertension mainly through the activation of sympathoadrenal system (SAS), and an increase in the glucose filtration of renal glomeruli leads to increased reabsorption of glucose together with sodium in the proximal tubules of the nephron [4,7]. This leads to hypovolemia and increases the content of sodium and calcium in the walls of blood vessels, causing the spasm of the latter and the increase in peripheral resistance of vessels. Insulin increases the activity of SNS, thus increasing cardiac output, and at the level of vessels, it causes their spasm and the increase in peripheral resistance of vessels. Insulin, as a mitogenic factor, increases the proliferation of fibroblasts and vascular smooth muscle cells by stimulation of tissue growth factor and collagen synthesis in atherosclerotic plaques, narrowing their gap and further increasing peripheral resistance of vessels [3,11**]. Insulin is a direct vasodilating agent, which is completely NO-dependent [18], therefore IR itself contributes to the increase in peripheral resistance of vessels [14].**

Some contribution to the genesis and formation of hypertension contribute to the dysfunction of vascular endothelium. One of the main biochemical markers of dysfunction of endothelium is the deficiency of nitric oxide-NO (or the lack of its products or its inactivation). In hypertension, NO deficiency can cause formation of excess amounts of free radicals and the degradation of bradykinin [19]. Being a characteristic feature of insulin-induced hypertension, renal hypersympathicotonia occurs as a consequence of GI stimulation of the central mechanisms of the SNS and as a result of increased allocation of NA in sympathetic synapses of kidneys as a result of the activation of renal tissues of RAAS under IR conditions. By blocking the transmembrane ion exchange mechanisms (by reducing the activity of transmembrane enzyme of Na^+, K^+ and Ca^{2+}– dependent ATPase), GI increases the content of Na^+ и Ca^{2+} and decreases the content of K^+, Mg^{2+}, pH within the cells, including the smooth myocytes. This leads to an increased sensitivity of the vascular wall to pressor effects of catecholamines, AT-II and increase in hypertension [2,3,4].

Insulin can increase the activity of SAS by itself, but this may be due to the action of leptin. It is known that with increasing level of obesity, the fasting level of leptin, which is secreted by adipocytes, increases. Leptin increases the activity of the SNA, particularly in the kidneys. This leads, on the one hand, to the high output and increasing of the heart rate, and on the other hand, to the the increasing of the reabsorption of sodium and the increasing of intravascular blood volume.

A relationship was found between RAAS and SNS. Activation of SNS is associated with the increased renin secretion in the kidneys, and it happens irrespective of intrarenal sensory system regulating renin secretion by the kidneys. Moreover, the increase in cyclic adenosine monophosphate under the influence of catecholamines stimulates the expression of angiotensinogen in the adipocytes of human [29]. Increased levels of AT II enhances the activity of the SNS. It has been established that AT II activates local SNS participating in the increase in body temperature (thermogenesis). Cold processing leads to an increase in the content of AT II in adipocytes without a concomitant variation in the level of AT II in plasma [30]. Thus, the dysregulation of RAAS in obesity can also stimulate the activity of SNS. It is significant that in obesity there occurs a dysfunction of the mechanisms of regulation of RAAS [12]. Under physiological conditions, the increase in the activity of the RAAS leads to increased peripheral vascular resistance and hence an increase in blood pressure. Under the principle of feedback, increased hypertension should cause decrease in the secretion of renin, drop in the AT II level and reduce the content of aldosterone. This, in turn, reduces the retention of fluid and sodium and supports hypertension at normal level.

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