UDC 616-056.527-008.6+616.12-008.331.1+616.1-06
DOI: 10.56871/RBR.2025.20.95.012

THE RELATIONSHIP BETWEEN OBESITY AND ARTERIAL HYPERTENSION (LECTURE)

© Rodion V. Korablev[1], Andrei G. Vasiliev[1], Sarng S. Pyurveev[1, 2], Tatyana V. Brus[1], Anna V. Vasilieva[1], Aleftina A. Kravtsova[1], Vladimir A. Evgrafov[1]

[1] Saint Petersburg State Pediatric Medical University. 2 Lithuania, Saint Petersburg 194100 Russian Federation
[2] Institute of Experimental Medicine. 12 Academician Pavlov str., Saint Petersburg 197022 Russian Federation

Contact information: Rodion V. Korablev — Candidate of Medical Sciences, Associate Professor, Department of Pathological Physiology with a Course of Immunopathology. E-mail: rodion.korablev@gmail.com ORCID: https://orcid.org/0009-0004-5754-8437 SPIN: 4969-6038

For citation: Korablev RV, Vasiliev AG, Pyurveev SS, Brus TV, Vasilieva AV, Kravtsova AA, Evgrafov VA. The relationship between obesity and arterial hypertension (lecture). Russian Biomedical Research. 2025;10(2):114–128. DOI: https://doi.org/10.56871/RBR.2025.20.95.012

Received: 21.03.2025 **Revised: 14.05.2025** **Accepted: 24.06.2025**

Abstract. The prevalence of obesity and arterial hypertension in recent decades has reached the scale of global epidemics with an alarming trend of increasing new cases, representing a huge burden for modern healthcare. According to WHO, arterial hypertension is one of the most significant risk factors for both disability and mortality, while obesity increases the risk of developing arterial hypertension by 70–80%. Weight loss, on the contrary, reduces the risk of arterial hypertension or improves the prognosis in patients with this diagnosis. This lecture, intended for students of medical universities and doctors of various specialties, briefly covers the issues of the etiology and pathogenesis of arterial hypertension and obesity, as well as the mechanisms of their relationship, the principles of drug and non-drug therapy.

Keywords: obesity, overweight, arterial hypertension, cardiovascular disease, insulin resistance, cardiovascular accidents

DOI: 10.56871/RBR.2025.20.95.012

ВЗАИМОСВЯЗЬ ОЖИРЕНИЯ И АРТЕРИАЛЬНОЙ ГИПЕРТЕНЗИИ (ЛЕКЦИЯ)

© Родион Владимирович Кораблев[1], Андрей Глебович Васильев[1], Сарнг Саналович Пюрвеев[1, 2], Татьяна Викторовна Брус[1], Анна Валентиновна Васильева[1], Алефтина Алексеевна Кравцова[1], Владимир Аркадьевич Евграфов[1]

[1] Санкт-Петербургский государственный педиатрический медицинский университет. 194100, г. Санкт-Петербург, ул. Литовская, д. 2, Российская Федерация

[2] Институт экспериментальной медицины. 197022, г. Санкт-Петербург, ул. Академика Павлова, д. 12, Российская Федерация

Контактная информация: Родион Владимирович Кораблев — к.м.н., доцент кафедры патологической физиологии с курсом иммунопатологии. E-mail: rodion.korablev@gmail.com ORCID: https://orcid.org/0009-0004-5754-8437 SPIN: 4969-6038

Для цитирования: Кораблев Р.В., Васильев А.Г., Пюрвеев С.С., Брус Т.В., Васильева А.В., Кравцова А.А., Евграфов В.А. Взаимосвязь ожирения и артериальной гипертензии (лекция). Российские биомедицинские исследования. 2025;10(2):114–128. DOI: https://doi.org/10.56871/RBR.2025.20.95.012

Поступила: 21.03.2025 **Одобрена: 14.05.2025** **Принята к печати: 24.06.2025**

Резюме. Распространенность ожирения и артериальной гипертензии за последние десятилетия достигла масштабов глобальных эпидемий с тревожным трендом роста новых случаев и является огромным бременем современного здравоохранения. Артериальная гипертензия, согласно данным Всемирной организации здравоохранения, является одним из наиболее значимых факторов риска как инвалидизации, так и смертности, при этом ожирение увеличивает риск развития артериальной гипертензии на 70–80%. Нормализация массы тела, напротив, снижает риск развития артериальной гипертензии или улучшает прогноз у пациентов с данным диагнозом. В настоящей лекции, предназначенной для студентов медицинских вузов и врачей различных специальностей, кратко освещены вопросы этиологии и патогенеза артериальной гипертензии и ожирения, а также механизмы их взаимосвязи, принципы медикаментозной и немедикаментозной терапии.

Ключевые слова: ожирение, избыточный вес, артериальная гипертензия, заболевания сердечно-сосудистой системы, инсулинорезистентность, сердечно-сосудистые осложнения

ARTERIAL HYPERTENSION AND OBESITY (DEFINITIONS, TERMINOLOGY, CLASSIFICATION)

According to the clinical guidelines of the Russian Federation (RF), the terms “arterial hypertension” and “hypertensive disease” are defined as follows:

“Arterial hypertension (AH) is a syndrome of elevated blood pressure (BP) in essential hypertension and secondary hypertension, defined by threshold values established through epidemiological and randomized controlled trials. These studies have demonstrated an association between these BP levels and increased cardiovascular risk (CVR), as well as the feasibility and benefit of treatment aimed at reducing BP below these thresholds (synonym: Arterial hypertonia) [1].

Essential hypertension (EH) is a chronically progressing condition whose primary clinical manifestation is elevated BP unrelated to identifiable causes of secondary (symptomatic) hypertension” [1].

The classification of office-measured blood pressure and the definition of hypertension grades are presented in Table 1.

According to the clinical guidelines of the RF, obesity is a chronic disease characterized by excessive accumulation of adipose tissue in the body, which poses health risks and serves as a major risk factor for several other chronic conditions, including type 2 diabetes mellitus (T2DM) and cardiovascular diseases (CVD) [2].

Overweight and obesity are classified based on body mass index (BMI) measurements, a concept proposed by Quetelet in the mid-19th century. BMI is calculated using the following formula:

$$\text{BMI (kg/m}^2\text{)} = \text{body weight (kg)} / (\text{height in m})^2.$$

The diagnostic criterion for obesity is BMI ≥30.0 (WHO 1997 classification (Table 2)) [3].

It should be considered that BMI does not always objectively reflect the amount and distribution of adipose tissue in the body. Examples include individuals with hypertrophied skeletal musculature, low adipose tissue content, and BMI values above normal ranges.

According to the etiological classification principle, obesity is divided into *primary* (alimentary, exogenous-constitutional) and *secondary* (symptomatic), which is further subdivided into cerebral obesity, obesity with an identified genetic defect, obesity due to endocrinopathies, and iatrogenic obesity.

Table 1

Classification of blood pressure measured in a medical institution and determination of the degrees of hypertension

Таблица 1

Классификация артериального давления, измеренного в медицинском учреждении, и определение степеней гипертензии

Категории артериального давления / Blood pressure categories	Систолическое артериальное давление, мм рт.ст. / Systolic blood pressure, mm Hg		Диастолическое артериальное давление, мм рт.ст. / Diastolic blood pressure, mmHg
Оптимальное / Optimal	<120	и/and	<80
Нормальное / Normal	120–129	и/или / and/or	80–84
Высокое нормальное / High normal	130–139	и/или / and/or	85–89
Артериальная гипертензия I степени / Arterial hypertension stage I	140–159	и/или / and/or	90–99
Артериальная гипертензия II степени / Arterial hypertension stage II	160–179	и/или / and/or	100–109
Артериальная гипертензия III степени / Arterial hypertension stage III	≥180	и/или / and/or	≥110
Изолированная систолическая артериальная гипертензия / Isolated systolic hypertension	≥140	и / and	<90
Изолированная диастолическая артериальная гипертензия / Isolated diastolic hypertension	<140	и / and	≥90

Table 2

Classification of overweight in adults according to BMI [3]

Таблица 2

Классификация избыточной массы тела в соответствии с ИМТ [3]

Классификация / Classification	ИМТ, кг/м^2 / BMI kg/m^2	Риск сопутствующих заболеваний / Risk of co-morbidities
Дефицит массы тела / Underweight	<18,5	Низкий / Low
Нормальная масса тела / Normal rage	18,5–24,9	Обычный / Average
Избыточная масса тела / Pre-obese	25–29,9	Повышенный / Increased
Ожирение I степени / Obese class I	30,0–34,9	Высокий / Moderate
Ожирение II степени / Obese class II	35,0–39,9	Очень высокий /Severe
Ожирение III степени / Obese class III	>40	Чрезвычайно высокий / Very severe

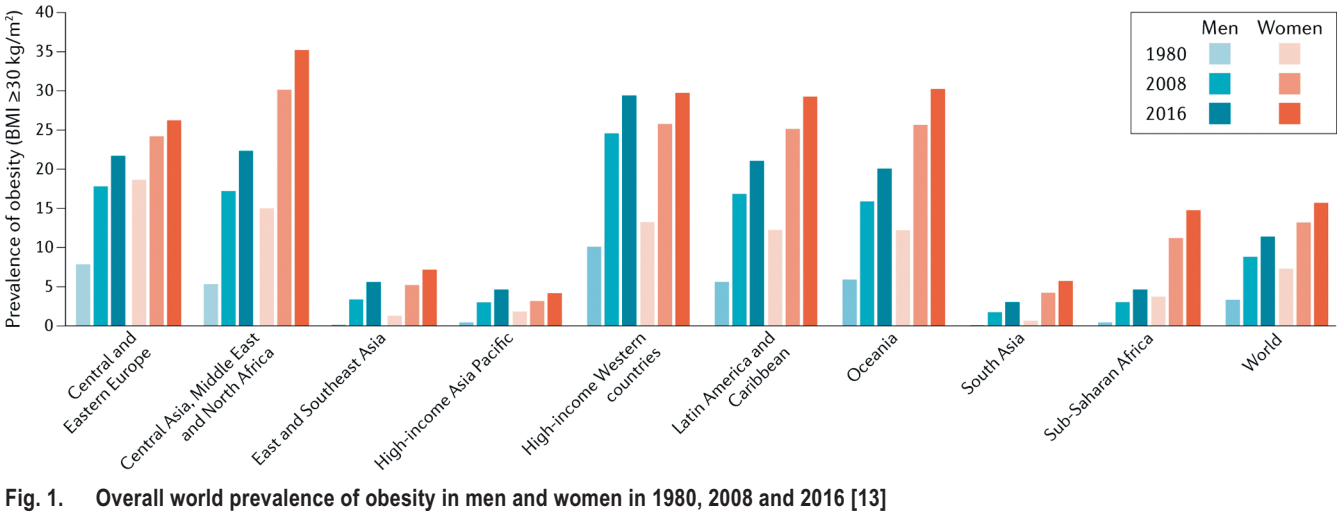

Fig. 1. Overall world prevalence of obesity in men and women in 1980, 2008 and 2016 [13]

Рис. 1. Распространенность ожирения у мужчин и женщин в 1980, 2008 и 2016 годах в мире [13]

The pattern of fat distribution represents an important marker for the development of various pathological processes [4].

The principal obesity types were first classified by J. Vague in 1956 [5]:

- the gynoid (gluteofemoral) type, characterized by predominant fat deposition in the hip and gluteal regions, more prevalent in women;
- the android (central) type, characterized by predominant fat accumulation in the trunk region, particularly in the abdominal area, more common in men.

Abdominal obesity involves the accumulation of adipose tissue within the abdominal cavity, which possesses distinct anatomical, vascular, metabolic, and hormonal characteristics [6]. Visceral fat in particular is associated with multiple obesity-related pathological conditions. Consequently, waist circumference serves as a key supplementary anthropometric measure, with normal values not exceeding 94 cm for men and 80 cm for women [2].

PREVALENCE OF ARTERIAL HYPERTENSION

The prevalence of AH shows a persistent upward trend globally (Fig. 1). From 1990 to 2019, the number of affected individuals aged 30–79 years doubled (increasing from 331 million women and 317 million men in 1990 to 626 million women and 652 million men in 2019), despite stable age-standardized global prevalence. The lowest AH prevalence is observed in: Peru and Canada for both sexes; South Korea, Taiwan, Japan, and certain Western European countries (Switzerland, Spain, and the United Kingdom) among women; and several low- and middle-income countries (Ethiopia, Bangladesh, Eritrea, and Solomon Islands) among men. AH prevalence exceeds 50% among women in two countries and among men in nine countries across Central and Eastern Europe, Central Asia, Oceania, and Latin America [7].

In the RF, the AH prevalence stands at 53.9% (men: 56.0%, women: 52.1%) [8]. The AH and its associated

complications are responsible for 8.5 million annual deaths worldwide [9].

PREVALENCE OF OBESITY

According to WHO data, the global number of adults with obesity in 2022 had doubled since 1990 and tripled since 1975. By 2022, one in eight people worldwide (totaling 890 million individuals) had obesity, while 2.5 billion adults were classified as overweight [10].

Among children and adolescents, obesity prevalence has increased sharply since 1990 — showing a four-fold rise (from 8% to 20%) with no gender differences. A total of 390 million children and adolescents aged 5–19 years had excess body weight, including 160 million already living with obesity. This alarming trend has prompted the WHO to emphasize "the urgent need to tackle the obesity crisis" [10].

According to projections, by 2030 approximately 1.12 billion people worldwide will have obesity, while 2.16 billion will be overweight [11].

In the Russian population in 2016, obesity was present in 26.2% of adults, with an additional 62.0% classified as overweight [12].

ETIOLOGY AND PATHOGENESIS OF ARTERIAL HYPERTENSION: CORE CONCEPTS

Blood pressure is determined by three principal hemodynamic factors: cardiac output, circulating blood volume (CBV), and systemic vascular resistance (SVR). An increase in any of these parameters elevates BP. CBV is regulated through the balance of fluid intake and excretion, plasma concentrations of key cations (Na^+, Ca^{2+}, K^+), their membrane transport activity, circulating natriuretic peptides, antidiuretic hormone (ADH) secretion, and renin-angiotensin-aldosterone system (RAAS) activity. SVR depends on vascular length, vessel diameter, and blood rheological properties. Vessel diameter is modulated by neurogenic and humoral vasoactive substances and pathological changes like atherosclerosis and intimal hyperplasia. Cardiac output is determined by stroke volume and heart rate (HR). It is influenced by venous return (which depends on CBV), SVR, and neurohumoral factors.

The pathogenesis of AH involves a progressive imbalance between depressor and pressor blood pressure regulatory mechanisms, with a shift toward pressor dominance. Key pathogenic factors include: activation of RAAS, sympathetic nervous system hyperactivity, deficient vasodilator systems (nitric oxide, kinins, prostaglandins), and impaired baroreceptor sensitivity in the aortic and carotid arteries.

The age-related progression of AH appears to be mediated by gradually developing blood pressure regulation disorders (approximately 60% of individuals aged over 60 years have AH) [14]. However, progression patterns vary across populations [15].

Both pulse pressure and systolic blood pressure (SBP) show progressive increases throughout life. Diastolic blood pressure (DBP) exhibits a similar pattern to SBP, reaching peak values between 50–60 years of age, followed by a characteristic decline [16].

These blood pressure progression trends are consistent across both sexes.

The etiology of AH remains unknown.

ETIOLOGY AND PATHOGENESIS OF OBESITY: CORE CONCEPTS

The acknowledgment that obesity is a chronic disease — rather than a result of inadequate body weight self-regulation or lack of willpower in eating behaviors — has been made possible through 70 years of research. These studies have deepened our understanding of the physiological mechanisms underlying body weight regulation, including adipose tissue expansion, its metabolic adaptation, secretory functions, and associated comorbid conditions. These comorbidities include dyslipidemia, cardiovascular diseases, non-alcoholic fatty liver disease, obstructive sleep apnea, and other chronic disorders [17].

Obesity is a complex disease whose development involves both modifiable and non-modifiable factors.

The primary etiological factors include:

I. Chronic energy imbalance, defined as sustained caloric intake exceeding daily energy expenditure and/or insufficient physical activity (key drivers of the global obesity epidemic) [18].

II. Social determinants of health.

Three decades of research confirm that biopsychosocial factors influence weight gain more significantly than individual psychological characteristics [18].

The Foresight obesity report emphasizes that the social environment fundamentally alters eating behaviors, thereby promoting obesity [19].

Recent decades have witnessed increased accessibility of high-calorie foods across all socioeconomic strata, marked by: the proliferation of fast-food chains, expanded vending machine availability, and growth of prepared meal delivery services. Aggressive marketing of products high in rapidly digestible carbohydrates and/or fats exacerbates the obesity epidemic by activating the same brain reward pathways as addictive substances, creating dependency [20].

Occupational patterns have shifted toward sedentary employment. Private vehicle ownership has become commonplace across broad population segments. The proliferation of technological conveniences has fostered sedentary behaviors across all age groups.

III. Genetic predisposition and epigenetic influences.

The genetic causes of obesity can be classified into the following categories:

1) Monogenic obesity — results from single-gene mutations primarily affecting the leptin-melanocortin signaling pathway. Examples include genes encoding agouti-related peptide (AgRP), orexigenic peptide YY (PYY), and melanocortin-4 receptor (MC4R) that have been identified in monogenic obesity disrupting appetite and body weight regulation [21].
2) Syndromic obesity — a rare form of obesity resulting from neurodevelopmental abnormalities and congenital malformations of multiple organ systems, caused by alterations in single or multiple genes [22]. Examples of obesity-associated syndromes include Prader–Willi syndrome (mutations in MKRN3, MAGEL2, NDN, and NPAP1 genes) and Bardet-Biedl syndrome (mutations in BBS1-BBS26 genes).
3) Polygenic obesity — associated with mutations in multiple genes (FTO, INSIG2, MC4R, BDNF, etc.), with proven cumulative effects. Key distinguishing features from monogenic obesity include: high population prevalence, relatively small contribution of each individual gene to obesity development, and strong influence of environmental factors on obesity manifestation. A child with one obese parent has a threefold higher risk of overweight in adulthood, while having two obese parents increases the obesity risk tenfold [20]. An observational study of 260 children (139 girls and 121 boys aged 2.4 to 17.2 years) demonstrated that family history of cardiometabolic disorders constitutes a critical risk factor for childhood obesity severity [23].

IV. Dysregulation of neuroendocrine control of eating behavior. Eating behavior is regulated by the interplay between orexigenic factors (stimulating food intake: somatoliberin, somatostatin, ghrelin, β-endorphin, galanin) and anorexigenic factors (inhibiting food intake: leptin, serotonin, glucagon-like peptide-1, corticoliberin, cholecystokinin). Leptin, a principal appetite regulator, is secreted by adipocytes in concentrations directly proportional to adipose tissue mass [24]. Obesity is characterized by excessive leptin secretion, which ultimately leads to leptin resistance. This results in the development of false hunger signaling in the hypothalamus, consequently increasing appetite. The hypothalamus additionally responds to circulating levels of gut-derived peptides including cholecystokinin, ghrelin, and glucagon-like peptide-1, which are released in response to food intake. Hypocretins (orexins) released from the lateral hypothalamic area project to the medial hypothalamus and modulate reward-related neural circuits involved in motivation and addictive behaviors. Dysregulation of this intricate neuroendocrine system promotes hedonic feeding despite adequate energy stores, thereby creating a significant barrier to effective appetite regulation and weight management in affected individuals.

V. Development of insulin resistance and chronic inflammation. The association between impaired carbohydrate metabolism and obesity has been well established in numerous studies. The risk of developing insulin resistance and type 2 diabetes mellitus increases with expanding adipose tissue mass. As an endocrine organ, adipose tissue secretes leptin, adiponectin, and free fatty acids. Adipose tissue expansion leads to adipocyte hypertrophy, accompanied by increased secretion of these compounds along with reactive oxygen species and proinflammatory cytokines (tumor necrosis factor-α, interleukin-6, etc.). These processes result in insulin resistance, endothelial dysfunction, and chronic low-grade inflammation, likely explaining the well-established causal links between obesity and three major disorders: atherosclerosis, arterial hypertension, and type 2 diabetes mellitus.

MECHANISMS UNDERLYING THE OBESITY-HYPERTENSION RELATIONSHIP

An imbalance between nutrient intake (predominantly carbohydrates) and their reduced utilization — specifically, excess energy intake over expenditure — induces compensatory hyperinsulinemia that maintains blood glucose within normal physiological ranges for a limited time.

Insulin exerts its primary effects during the postprandial period (elevated glucose levels stimulate insulin secretion). It mediates multiple anabolic processes: glucose uptake by skeletal muscle myocytes for glycogen synthesis and by adipocytes for triglyceride production (lipogenesis), while simultaneously inhibiting proteolysis, glycogenolysis, ketogenesis, and gluconeogenesis. These insulin actions are mediated through activation of two signaling pathways: the mitogen-activated protein kinase (MAPK/RAS) and phosphatidylinositol-3-kinase (PI3K)/Akt pathways. Consequently, dysfunction at any point in these complex biological cascades — from the insulin receptor to expression of target genes — can lead to insulin resistance, characterized by progressively diminished cellular responsiveness to insulin. Most commonly, these defects occur at the post-receptor level.

The mechanism of insulin resistance in obesity involves impaired glucose transport into insulin-sensitive tissues (adipose tissue, skeletal muscle, and liver). This dysfunction stems from inhibited GLUT4 translocation and/or activity dysregulation, reduced insulin receptor autophosphorylation, and decreased receptor density in adipocytes and myocytes, ultimately impairing intracellular insulin signaling.

Thus, progressive hyperinsulinemia secondary to insulin resistance drives expansion of adipose tissue mass. As this mass increases, daily energy requirements rise correspondingly, which modifies feeding behavior and thereby closes the vicious cycle.

Hyperinsulinemia associated with obesity promotes cellular proliferation, including that of arterial smooth muscle cells, as demonstrated in human and primate smooth muscle cell cultures [25]. The consequent smooth muscle cell hypertrophy, along with enhanced proliferation of collagen-secreting fibroblasts, progressively leads to vascular wall rigidity. These pathological changes ultimately increase SVR, representing a fundamental mechanism in arterial hypertension pathogenesis.

In addition to its effects on vascular smooth muscle cells, obesity induces endothelial dysfunction. The pathophysiological mechanisms involve reduced nitric oxide (NO) bioavailability due to impaired endothelial nitric oxide synthase (eNOS) synthesis or activity, compromising endothelium-dependent vasodilation [26]. Furthermore, obesity promotes endothelial oxidative stress [27], endoplasmic reticulum stress [28], autophagy dysregulation [29], and chronic low-grade inflammation [30].

Additionally, obesity reduces the expression of the NAD^+-dependent deacetylase sirtuin-1 (SIRT1). Sirtuins catalyze NAD^+-dependent lysine deacetylation, and their diminished activity is linked to decreased lifespan, cellular stress, and particularly to endothelial dysfunction [31].

Despite its direct vasodilatory action, insulin paradoxically stimulates the sympathoadrenal system, resulting in a predominant net vasoconstrictor effect.

Hyperinsulinemia and obesity are characterized by increased sympathetic nervous system activity accompanied by reduced parasympathetic tone. This occurs via insulin's ability to cross the blood-brain barrier and access hypothalamic periventricular regions, potentiating neuronal signaling to spinal sympathetic nuclei [32]. One physiological explanation for this sympathetic activation involves compensatory energy expenditure to restore metabolic homeostasis.

Obese patients exhibit elevated urinary levels of norepinephrine and its metabolites compared to normal-BMI individuals [33], with concurrently increased resting sympathetic activity in skeletal muscles [34].

This heightened sympathetic tone may lead to multisystem effects. Within the cardiovascular system, this manifests as increased HR, augmented stroke volume, and induced vasoconstriction, collectively elevating SVR and BP.

The relationship between BP, RAAS activity, and tissue insulin sensitivity has been well-established in multiple studies [35, 36]. During hyperinsulinemia, sympathetic nervous system activation causes renal artery constriction, thereby stimulating the RAAS. Moreover, insulin induces intracellular accumulation of Na^+ and Ca^{++} through inhibition of Na/K-ATPase and Ca/Mg-ATPase activity in cell membranes, potentially enhancing vascular receptor sensitivity to vasoconstrictors.

Renin cleaves angiotensinogen to yield angiotensin I, which is converted to angiotensin II by angiotensin-converting enzyme (ACE) — the principal RAAS effector that directly mediates cardiometabolic dysfunction. Through receptor activation, angiotensin II induces vasoconstriction, promotes sodium retention in renal proximal tubules, and triggers aldosterone secretion from the adrenal zona glomerulosa, collectively increasing CBV and cardiac output [37].

Components of RAAS (angiotensinogen, ACE, angiotensin II) have been detected in pancreatic tissue, skeletal muscle, and adipose tissue [38]. Angiotensin II can promote adipocyte hypertrophy and modulate their differentiation [39].

Thus, local RAAS activation promotes adipose tissue expansion and insulin resistance. Importantly, antihypertensive therapy using angiotensin-converting enzyme inhibitors (ACEIs) or angiotensin II receptor blockers (ARBs) demonstrates significantly greater improvement in tissue insulin sensitivity compared to other antihypertensive drug classes [40–43].

In skeletal muscle, locally produced angiotensin II adversely affects both hemodynamics and metabolism by reducing microcirculatory perfusion, impairing GLUT-4 translocation, decreasing insulin-stimulated glucose uptake, and disrupting intracellular insulin signaling pathways [43].

Beyond metabolic dysfunction, perirenal adipose tissue causes mechanical kidney compression and promotes structural renal changes. These alterations decrease urinary Na^+ excretion while increasing tubular Na^+ reabsorption, ultimately raising BP [44].

Obesity initially elevates glomerular filtration rate and effective renal plasma flow, but ultimately leads to glomerular damage and AH, creating a vicious cycle.

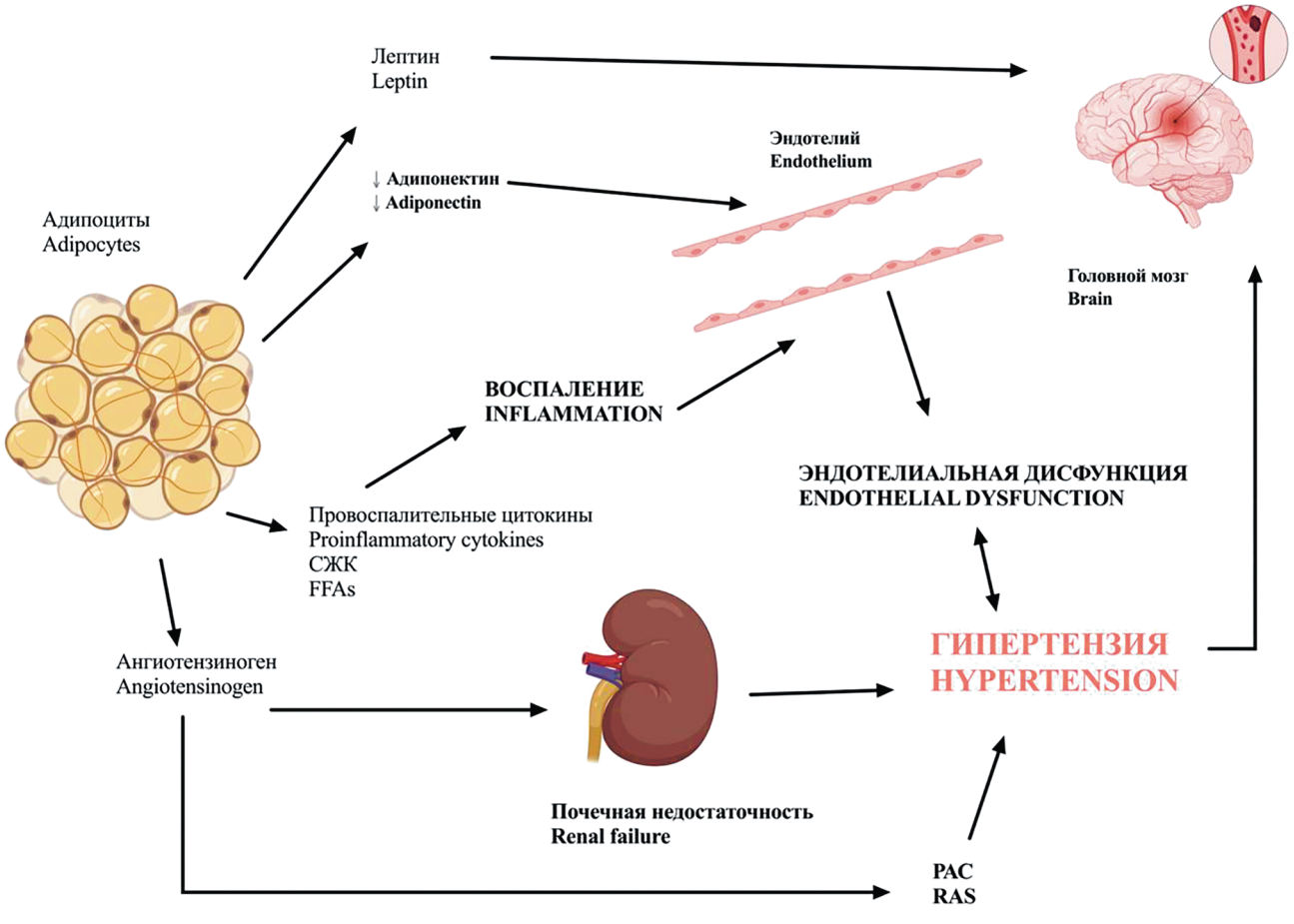

Fig. 2. The relationship between obesity and arterial hypertension

Рис. 2. Взаимосвязь ожирения и артериальной гипертензии

Thus, excessive body weight or obesity can result in AH and a cascade of associated cardiorenal and metabolic disorders (Fig. 2).

Global population studies demonstrate a near-linear relationship between BMI and SBP [45]. The Framingham Heart Study Multidetector Computed Tomography Study found SBP increases of 2.0 mm Hg per standard deviation in subcutaneous adipose tissue volume and 3.3 mm Hg per standard deviation in visceral adipose tissue volume [46]. These data indicate that 78% of AH cases in men and 65% in women are attributable to overweight or obesity [45]. Maintaining BMI <25 kg/m² effectively prevents AH onset, while BMI normalization in patients with AH reduces both BP and associated cardiovascular risks [47, 48].

PRINCIPLES OF ARTERIAL HYPERTENSION MANAGEMENT

The management of AH incorporates both pharmacological and non-pharmacological approaches.

Key approaches to pharmacological treatment

The management of AH should target not only BP control but also reduction of cardiovascular complications (CV complications). The target BP is 140/90 mmHg, or 130/80 mm Hg and below if well tolerated. Pharmacological therapy is recommended for all AH patients except those at low risk with BP below 150/90 mmHg. Initial treatment typically requires combination therapy from different antihypertensive drug classes, as adequate BP control with monotherapy is achieved in only a subset of patients.

Five principal drug classes are utilized in the management of AH. These include:

1. Angiotensin-converting enzyme inhibitors (ACEIs) represent the most commonly prescribed antihypertensive agents. ACEIs act by inhibiting angiotensin II formation, thereby reducing SVR. Furthermore, they prevent degradation of bradykinin, substance P, and kallikrein, which enhances endothelial function, exerts antiproliferative effects, decreases plasminogen activator inhibitor-1 synthesis, and inhibits platelet aggregation. The nephroprotective effects

of ACEIs are mediated through both hemodynamic and non-hemodynamic mechanisms. Hemodynamic effects include: reduced intraglomerular pressure, increased natriuresis, and antiproteinuric action. Non-hemodynamic mechanisms involve: decreased glomerular membrane permeability and improved renal endothelial function. Therapeutic administration of ACEIs decreases cardiac preload and afterload, reduces pulmonary artery pressure, attenuates left ventricular remodeling, and improves ejection fraction.

2. Angiotensin II receptor blockers (ARBs) inhibit the binding of angiotensin II to type 1 receptors, resulting in reduced vascular tone and attenuation of pathological remodeling in both the vascular wall and myocardium. Compared to ACE inhibitors, ARBs offer several clinical advantages, including an excellent safety profile, complete blockade of the RAAS, and a low incidence of adverse effects comparable to placebo.

3. Beta-blockers. The pharmacological action of beta-blockers is mediated through competitive inhibition of both β_1- and β_2-adrenergic receptors, leading to decreased HR, reduced myocardial contractility, lowered SVR (mediated by β_2-adrenergic receptors), and suppressed renin secretion (via β_1-adrenergic receptor blockade). Beta-blockers are specifically indicated for hypertensive patients presenting with the following comorbid cardiovascular conditions: heart failure (chronic forms), myocardial infarction (post-infarction period), and exertional angina (stable forms).

4. Diuretics are classified into four main categories: loop diuretics, thiazides, thiazide-like agents, and potassium-sparing diuretics. In the management of arterial hypertension, thiazide and thiazide-like diuretics are most frequently used. These drugs act by inhibiting the Na^+-Cl^- symporter in distal convoluted tubule epithelial cells, resulting in natriuresis, reduced CBV, and decreased SVR. It should be noted that thiazide diuretics promote potassium (K^+) excretion, which may lead to ventricular arrhythmias. The mechanism of loop diuretics involves blockade of ion transporters in the thick ascending limb of Henle's loop, enhancing urinary excretion of Na^+, Ca^{++}, K^+, Mg^{++}, and Cl^-. Their antihypertensive effect demonstrates a linear dose-response relationship. Compared with thiazide diuretics, loop diuretics exhibit more potent diuretic action, even in cases of renal impairment.

5. Calcium antagonists (calcium channel blockers, CCBs). The unifying mechanism of this pharmacologically heterogeneous drug class involves inhibition of calcium ion (Ca^{++}) influx from the extracellular space into vascular smooth muscle cells, leading to vasodilation. Select CCBs additionally reduce myocardial contractility, enhance coronary blood flow, and decrease atrioventricular node conduction velocity, resulting in reduced HR.

Key approaches to non-pharmacological treatment

Non-pharmacological therapy is indicated for all patients with AH and includes a range of lifestyle modification measures aimed at reducing the risk of CV complications. These include:

1. Dietary interventions.

Research into effective dietary approaches originated from observational studies demonstrating that vegetarian diets, as well as diets with reduced saturated fat content and increased vegetable and fruit intake, are effective for both lowering BP and maintaining BP control in AH patients.

Since 1996, the DASH diet has been implemented in clinical practice. The DASH is the acronym for Dietary Approaches to Stop Hypertension. The DASH diet is based on: vegetables, fruits, whole grains, low-fat dairy products, poultry, fish, and nuts, while requiring reduced consumption of table salt, red meat, processed meat products, and sweets. Two versions exist: the standard DASH diet (table salt intake up to 2,300 mg per day) and the low-sodium DASH diet (up to 1,500 mg per day). Patient adherence to the standard DASH diet can reduce systolic BP by 5–6 mm Hg, with the low-sodium version providing an additional 6–8 mm Hg reduction [49].

Notably, salt intake in most populations globally is nearly universally excessive. The majority of dietary salt intake comes from so-called "hidden salt" (e.g., processed foods and restaurant meals), making an accurate assessment of daily consumption particularly challenging. Salt sensitivity varies substantially among individuals, affecting both normotensive subjects and AH patients. Approximately one-quarter of adults are salt-sensitive, meaning they exhibit BP elevation in response to increased salt intake without being aware of this response [50].

Dietary planning must account for the patient's sex, age, occupation, and physical activity. Care should be taken to consider not only total caloric intake (kcal) but also the protein-to-fat-to-carbohydrate ratio per kg of body weight (proteins: 1.0–1.5 g; fats [plant and animal]: 0.9–1.0 g; carbohydrates: 1.5–4.0 g). Daily carbohydrate intake requires continuous adjustment, as carbohydrate restriction specifically reduces adipose tissue mass through its utilization under energy deficit conditions.

2. Moderate-intensity aerobic exercise. The recommended regimen consists of at least 30 minutes of moderate-intensity activity performed 5–7 days per week. This intervention improves organ perfusion and delivery of both oxygen and nutrients while activating key metabolic processes (including skeletal muscle glucose uptake and energy deficit generation). It concurrently reduces cortisol secretion and enhances functional capacity of specific immunocompetent cell populations.

3. Alcohol intake restriction. Ethanol metabolites initially induce transient vasodilation and BP reduction, followed by

a rebound pressor response. Chronic alcohol consumption, particularly in excessive amounts, contributes to AH development. The primary mechanism involves RAAS activation, resulting in increased CBV and SVR. Additionally, ethanol stimulates cortisol and vasopressin secretion while impairing baroreceptor sensitivity.

Patients with AH should not exceed the following weekly limits: 14 units for men or 8 units for women. One alcohol unit (10 mL or 8 g pure ethanol) equals about 125 mL wine or 250 mL beer.

4. Smoking cessation. The combination of AH and smoking is highly prevalent in most populations. The risk of developing AH exhibits a linear relationship with both daily cigarette consumption and smoking duration. A single cigarette can induce transient BP elevation lasting up to 15 minutes [51]. Smokers with AH have twice the risk of all-cause premature mortality and a fivefold higher risk of fatal CV complications compared to non-smoking AH patients [51, 52].

Endothelial cells serve as a common target for both tobacco smoke metabolites and elevated BP. Smoking induces chronic endothelial inflammation, which reduces endogenous NO bioavailability, accelerates atherosclerosis progression, increases thrombotic risk, and promotes various other pathological manifestations. Additionally, tobacco use activates the sympathetic nervous system, resulting in sustained vasoconstriction. Over time, the vascular wall undergoes structural remodeling characterized by an altered elastin-to-collagen ratio, ultimately leading to increased rigidity. Thus, smoking and AH demonstrate synergistic effects that increase the risk of multiple complications.

PRINCIPLES OF OBESITY MANAGEMENT

The primary goal of obesity therapy is to reduce the risk of obesity-related complications (including AH, type 2 diabetes mellitus, non-alcoholic fatty liver disease, and obstructive sleep apnea) while simultaneously improving quality of life.

Maintaining target BMI following treatment remains the primary challenge for patients with obesity. Like all chronic diseases, obesity requires a long-term, multidisciplinary approach that considers each patient's therapeutic goals along with the benefit-risk ratio of various treatment modalities.

The main management strategies for obesity include:

1) non-pharmacological therapy;
2) pharmacological therapy;
3) surgical treatment.

Non-pharmacological therapy constitutes the foundational and mandatory first phase of obesity treatment, comprising dietary modifications and physical activity regulation. The recommended daily caloric deficit is 500–700 kcal below individual maintenance needs, while maintaining a nutritionally balanced diet adjusted for age and sex. The expected weight loss ranges from 500–1000 g per week. Importantly, fasting is not recommended for obesity treatment due to insufficient evidence of efficacy and safety. Patients should engage in at least 150 minutes of moderate-intensity aerobic exercise weekly.

Pharmacological therapy is indicated for patients with a BMI ≥30 kg/m², as well as for those who fail to achieve therapeutic targets through non-pharmacological methods or cannot maintain their weight loss. Currently, three pharmacological agents are approved in the Russian Federation: sibutramine, orlistat, and liraglutide. Of particular concern, sibutramine therapy has been associated with significantly increased risks of cardiac arrhythmias and cardiac arrest. Consequently, in January 2010, the European Medicines Agency (EMA) suspended its marketing authorization across the European Union due to elevated risks of non-fatal myocardial infarctions and strokes. Subsequently, in October 2010, the U.S. Food and Drug Administration (FDA) enforced a complete market withdrawal of sibutramine because of its demonstrated association with major adverse cardiovascular events (MACE) [53].

Surgical treatment (bariatric surgery) is indicated for patients with BMI >40 kg/m² demonstrating an inadequate response to conservative management. The primary surgical interventions include gastric bypass, biliopancreatic diversion, sleeve gastrectomy, and gastric banding. Long-term studies have demonstrated that bariatric surgical procedures typically result in sustained weight loss of 25% and prompt, durable reduction of obesity-associated complication risks [54].

CONCLUSION

Obesity and AH share common pathogenic mechanisms and necessitate a multidisciplinary treatment approach. Given the high prevalence of obesity, AH, and their associated complications, a thorough understanding of these conditions' pathophysiology is essential for clinicians across all medical specialties.

ADDITIONAL INFORMATION

Author contribution. Thereby, all authors made a substantial contribution to the conception of the study, acquisition, analysis, interpretation of data for the work, drafting and revising the article, final approval of the version to be published and agree to be accountable for all aspects of the study.

Competing interests. The authors declare that they have no competing interests.

Funding source. This study was not supported by any external sources of funding.

ДОПОЛНИТЕЛЬНАЯ ИНФОРМАЦИЯ

Вклад авторов. Все авторы внесли существенный вклад в разработку концепции, проведение исследования и подготовку статьи, прочли и одобрили финальную версию перед публикацией.

Конфликт интересов. Авторы декларируют отсутствие явных и потенциальных конфликтов интересов, связанных с публикацией настоящей статьи.

Источник финансирования. Авторы заявляют об отсутствии внешнего финансирования при проведении исследования.

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