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THE IMPACT OF OBESITY ON THE CARDIOVASCULAR SYSTEM

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Abstract. In recent years, obesity has become one of the most pressing global public health problems. According to the World Health Organization (WHO), approximately 39% of the adult population worldwide is overweight, and 13% suffers from obesity. Obesity is considered one of the leading risk factors for cardiovascular diseases (CVD), significantly increasing the risk of coronary artery disease, arterial hypertension, stroke, and heart failure.

Keywords: arterial hypertension, hyperinsulinemia, dyslipidemia, lipoproteins, body weight.

Discussion and Results.

Pathophysiological relationship between obesity and the cardiovascular system.

Obesity is accompanied by metabolic and hemodynamic changes in the body. An increase in adipose tissue leads to the development of insulin resistance, hyperinsulinemia, and dyslipidemia. As a result, levels of low-density lipoproteins (LDL) increase, while high-density lipoproteins (HDL) decrease, accelerating the progression of atherosclerosis. Studies indicate that individuals with a body mass index (BMI) greater than 30 kg/m² have a 2–3 times higher risk of developing atherosclerotic cardiovascular disease compared to individuals with normal body weight[1].

Obesity and arterial hypertension.

Obesity is one of the major causes of arterial hypertension. Excess body weight enhances sympathetic nervous system activity, activates the renin–angiotensin–aldosterone system (RAAS), and increases sodium retention. Scientific data show that arterial hypertension is diagnosed in 60–70% of obese patients. An increase in BMI by every 5 kg/m² is associated with an average rise in systolic blood pressure of 5–7 mmHg[2].

Structural and functional changes of the heart.

Obesity causes excessive workload on the myocardium. Increased blood volume leads to left ventricular hypertrophy and the development of diastolic dysfunction. Echocardiographic studies demonstrate that in obese patients, left ventricular wall thickness is 20–30% greater compared to individuals with normal body weight[3]. These changes significantly increase the risk of heart failure.

Coronary heart disease and thrombotic complications.

Obesity is associated with a chronic low-grade inflammatory state. Elevated levels of adipokines and pro-inflammatory cytokines contribute to endothelial dysfunction and increased thrombotic tendency[4]. Research shows that obesity increases the risk of coronary heart disease by 50–100%, while the risk of stroke rises by up to 64%.

Additional mechanisms linking obesity and cardiovascular disease

Role of visceral adiposity.

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Visceral (abdominal) obesity plays a particularly important role in cardiovascular risk. Unlike subcutaneous fat, visceral adipose tissue is metabolically active and secretes large amounts of pro-inflammatory cytokines, including tumor necrosis factor-α (TNF-α), interleukin-6 (IL-6), and resistin[5,6]. These mediators promote systemic inflammation, endothelial dysfunction, and progression of atherosclerosis. Epidemiological studies demonstrate that waist circumference is a stronger predictor of cardiovascular events than BMI alone, increasing the risk of myocardial infarction by approximately 40–50%.

Endothelial dysfunction and oxidative stress.

Obesity is strongly associated with endothelial dysfunction due to reduced bioavailability of nitric oxide (NO) and increased oxidative stress[7]. Elevated free fatty acids and chronic inflammation impair vasodilation and promote vascular stiffness. As a result, obese individuals exhibit increased arterial stiffness and pulse wave velocity, which are independent predictors of cardiovascular mortality.

Obesity, type 2 diabetes, and cardiovascular risk.

Obesity is the primary risk factor for type 2 diabetes mellitus, which further amplifies cardiovascular risk. Approximately 80–90% of patients with type 2 diabetes are overweight or obese[8,9]. The coexistence of obesity and diabetes increases the risk of coronary artery disease by 2–4 times, while cardiovascular mortality rises by nearly 70% compared to non-diabetic individuals.

Impact on cardiac rhythm and sudden cardiac death.

Obesity is also associated with cardiac rhythm disturbances, particularly atrial fibrillation. Clinical data indicate that each 1 kg/m² increase in BMI raises the risk of atrial fibrillation by about 4–5%[10]. Structural remodeling of the atria, myocardial fat infiltration, and autonomic imbalance contribute to arrhythmogenesis and increase the risk of sudden cardiac death.

Benefits of weight reduction on cardiovascular outcomes.

Weight loss has been shown to significantly improve cardiovascular parameters. A reduction of body weight by 5–10% leads to a decrease in systolic blood pressure by 5–10 mmHg, LDL cholesterol by 10–15%, and insulin resistance by approximately 30%[11,12]. Long-term studies confirm that sustained weight reduction lowers the incidence of cardiovascular events and improves overall survival.

Public health and preventive implications.

Given the strong association between obesity and cardiovascular diseases, preventive strategies should focus on early lifestyle interventions. Population-based programs promoting physical activity, healthy nutrition, and weight control have demonstrated a reduction in cardiovascular morbidity by 20–25% over a 10-year period.

Conclusion.

Obesity exerts a multifaceted and adverse impact on the cardiovascular system. It plays a crucial etiological role in the development of arterial hypertension, atherosclerosis, coronary heart disease, and heart failure. Therefore, obesity prevention through the promotion of a healthy

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lifestyle, balanced nutrition, and increased physical activity remains one of the key strategies in reducing cardiovascular morbidity and mortality.

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