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OVERWEIGHTING AND OBESITY AS PROMOTERS OF CARDIOVASCULAR DISEASES

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Relevance. Almost one third of the world population suffers overweighting or obesity requiring about half of the total treatment cost among all chronic diseases. Cell damage, high metabolic demand, neurohormonal disorders, and inflammation were described as risk factors of cardiovascular diseases.

Aim: to find clinically relevant proves of pathologic impact of overweighting or obesity on to cardiovascular diseases (CVD) development in published literature.

Materials and methods. Detailed analysis via search of published literature in 2015-2025 was done. Keywords “overweighting”, “obesity”, “myocardial dysfunction”, “biomarkers”, “heart failure”, “echocardiography”, “left ventricular remodeling”, “epicardial fat”, “lipotoxic cardiomyopathy”, “leptin’ were used for article search. Article was selected for analysis in case of more than 5 keywords were presented in its text. Among found articles twelve were corresponded to criteria.

Results and their discussion. Obesity is associated with a chronic inflammation that persists in the visceral adipose tissue due to reduced production of adiponectin and contrary increased secretion of resistin, leptin, and pro-inflammatory adipokines and cytokines. High leptin level promotes sodium retention, cardiac remodeling, impaired vascular relaxation, and finally cardiac diastolic dysfunction and heart failure (HF), thus contributing to cardiovascular morbidity. Obesity, insulin resistance and diabetes promote hypertriglyceridemia and increased plasma levels of fatty acids, that is stored in lipid droplets in heart. Intramyocardial lipid exceeding storage capacity and its incomplete b-oxidation induce non-ischemic and non-hypertensive cardiomyopathy known as lipotoxic cardiomyopathy that leads to heart failure with preserved ejection fraction (HFpEF). The "cardiomyopathy of obesity" refers to obesity-associated changes in cardiac function. Research from the Multi-Ethnic Study of Atherosclerosis (MESA) study found that obesity is linked to concentric left ventricle (LV) hypertrophy (LVH) and HFpEF. Obese cohort demonstrates an increased hemodynamic load with LV remodeling,atrial remodeling, atrial severe mitral and tricuspid valve insufficiency and elevated natriuretic peptides plasma level. Notably, obesity is the most significant risk factor for LV remodeling, even in patients with hypertension. The buildup of fat in the epicardium can trigger a cascade of events leading to impaired myocardial circulation, increased myocardial stiffness, diastolic dysfunction, and left atrial enlargement. These changes are characteristic of HFpEF, a condition disproportionately affecting obese individuals. The epicardial fat depot suggested to be a key target for therapeutic intervention in HFpEF. Significant weight loss can reduce epicardial fat volume and associated systemic inflammation, thereby lowering the risk of heart failure. Additionally, certain medications have been shown to decrease epicardial fat accumulation and inflammation, potentially mitigating the development of HFpEF.

Conclusion. Obesity is recognized as a heterogeneous condition in which individuals with similar body mass index may have distinct metabolic and cardiovascular risk profiles. Susceptibility to obesity-related cardiovascular complications is not mediated solely by overall body fat mass but depends largely on individual differences in regional body fat distribution, which negatively affect cardiac structure and function. With increasing prevalence of obesity in populations with a longer life span, there is a need to evaluate mechanisms underlying obesity-related CVD and to improve the management of patients with obesity through future research. In addition, the dramatic increase in the proportion of young patients with severe obesity invokes the need for more upstream interventions for the primary prevention of CVD and better treatment of obesity as a chronic disease.