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Obesity and Heart Failure With Preserved Ejection Fraction: What is There to EAT?

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Heart failure with preserved ejection fraction (HFpEF) is a heterogenous syndrome in which different comorbidities contribute to the progression of the disease, i.e. "outside-in" paradigm. However, many of these comorbidities co-exist in heart failure with reduced ejection fraction (HFrEF), but this syndrome is not defined by "outside-in" paradigm. Another way of looking at HFpEF is mandatory, and it is obvious where to look because the epidemic of HFpEF has tracked closely with the epidemic of obesity over the last 50 years[1]. However, obesity defined by body mass index (BMI) has limitations, since BMI depends on bone and skeletal muscle structure and is not very sensitive to visceral fat distribution. A much better way for assessment of cardiometabolic risk is visceral adiposity that encompasses adipose tissue around major organs, and unlike subcutaneous adipose tissue, is metabolically active, and proinflammatory if excessive. Visceral adiposity is defined by waist to height ratio of 0.5 or higher, and it predicts HFpEF years before its onset. A recent analysis involving patients with HFpEF, found that 49% have obesity as defined by BMI, but 96% have visceral adiposity[2]. Enormous overlap between obesity and HFpEF is mediated by visceral adipose tissue (VAT), which is the largest endocrine organ. Epicardial adipose tissue (EAT) is a distinct form of VAT that can affect heart through inflammation, fibrosis, and neural dysregulation. EAT communicates its state to the myocardium through paracrine secretion of messenger molecules called adipokines. Majority of EAT (42%) is located nearby right ventricle[3]. During nutrient overload, which is a hallmark of obesity, EAT becomes hypertrophic and inflamed. Since EAT is supplied by the branches of epicardial coronary arteries without muscular fascia between EAT and the heart, intensive crosstalk is unobstructed. Abundant EAT may start infiltrating and compressing the underlying myocardium, promoting diastolic dysfunction. Furthermore, thickened EAT contributes to obesity-related HFpEF by mechanical effect analogous to that of a constrictive pericarditis[4]. Namely, if a pericardial dilatation is non-congruent with EAT and intravascular volume expansion, increased EAT volume constrains the myocardium, resulting in a diastolic dysfunction. Invasive hemodynamic testing in individuals with HFpEF and obesity confirmed elevated left ventricular filling pressures and an increased left ventricular eccentricity index, implying that increased left and right ventricular coupling is directly related to excessive EAT thickness due to pericardial constraint[5]. In conclusion, EAT is an active contributor to obesity-related HFpEF phenotype with metabolic and hemodynamic implications.

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