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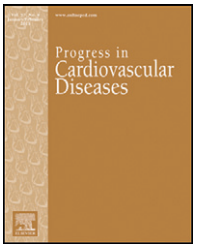

Update on Obesity and Obesity Paradox in Heart Failure

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ABSTRACT

Obesity has reached epidemic proportions in most of the Westernized world. Overweightness and obesity adversely impact cardiac structure and function, including on both the right and, especially, left sides of the heart, with adverse affects on systolic and, especially, diastolic ventricular function. Therefore, it is not surprising that obesity markedly increases the prevalence of heart failure (HF). Nevertheless, many studies have documented an obesity paradox in large cohorts with HF, where overweight and obese have a better prognosis, at least in the short-term, compared with lean HF patients. Although weight loss clearly improves cardiac structure and function and reduces symptoms in HF, there are no large studies on the impact of weight loss on clinical events in HF, preventing definitive guidelines on optimal body composition in patients with HF.

Overweight and obesity are generally defined by body mass index (BMI) criteria, which is useful in large population studies, but flawed in individual patients, since BMI reflects both fat mass and non-fat mass (mostly muscle and skeletal mass), leading to suggestions that alternative methods to define obesity, including waist circumference (WC), waist-to-hip-ratio, and percent body fat (% BF) may better reflect at risk body fatness. [1–3] Obesity is certainly a major risk factor for most cardiovascular (CV) diseases (CVDs), including for hypertension (HTN) and coronary heart disease (CHD), two of the major risk factors for the development of heart failure (HF).[1,4–6] Additionally, obesity has more direct effects on the pathogenesis of HF, due to its negative impact on cardiac morphology and performance, including negative impact on both systolic and, especially, diastolic left ventricular (LV) function.[5–8] Therefore, it is of no surprise that HF is dramatically increased in the setting of obesity.

However, despite the adverse effects that obesity has on CVD risk factors and its association with increased incidence of most CVD, including HTN and CHD, (both of which are strongly related with the increased risk of HF), and for HF itself, many large studies have demonstrated the powerful

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Abbreviations and Acronyms

% BF = percent body fat
BMI = body mass index
BP = blood pressure
CHD = coronary heart disease
CO = cardiac output
CRF = cardiorespiratory fitness
CV = cardiovascular
CVD = cardiovascular disease
HF = heart failure
HTN = hypertension
LA = left atrium or atrial
LV = left ventricular
LVH = left ventricular hypertrophy
LVM = left ventricular mass
RV = right ventricle
VO_2 = oxygen consumption
WC = waist circumference

"obesity paradox" in cohorts with established HF. In fact, the presence of overweight and obese, at least mild obesity in those with HF, appears to confer a better short- and intermediate-term prognosis than in their leaner counterparts who have similar degrees of HF.[5,6,9]

In this review, we describe the adverse effects of overweight/obesity on hemodynamics, as well as on cardiac structure and ventricular function. The impact of obesity on HF prevalence is reviewed, as well as studies showing an obesity paradox in HF. Finally, we discuss the role of fitness and weight loss in obese patients with HF, as well as the need for more studies on this topic.

Impact of obesity on cardiac morphology and performance

Alpert and colleagues[7,8] have recently reviewed this topic in detail. Considerable evidence underscores the adverse effects of excessive adipose accumulation and associated fat-free mass on central and peripheral hemodynamics, as well as on cardiac structure and systolic and diastolic LV function (Fig 1, Table 1).[5,7]

Hemodynamics

There are many hemodynamic alterations in obesity, leading to increased total and central blood volume and increased stroke volume and cardiac output (CO).[5,7,8] Typically, obesity leads to a reduction in systemic vascular resistance in normotensive obese persons, which facilitates the increase in CO despite the fact that heart rate is not substantially impacted in obesity. Arteriovenous oxygen differences are usually widened in obesity, due to lower oxygen extraction by adipose tissue. Thus, blood flow per unit of weight is reduced, especially in more severe degrees of obesity and in combined obesity/HTN, where systemic vascular resistance is also increased. [5,7,8]

In Class II and III obesity, increased venous return leads to increased preload with increased LV filling pressure. This further contributes to elevated pulmonary artery and right heart pressures.[5,7,8] With exercise in Class III obesity, central blood volume increased by 20%, LV end-diastolic pressure increased by 50%, and the LV dP/dt increased by 57%. [5,10]

Cardiac structure

Obesity also produces marked abnormalities in cardiac structure, including LV hypertrophy (LVH),[5,7,8,11] as well as enlargement of the atria and right side of the heart. Although LV mass (LVM) is commonly increased in obesity,[5,7,8,11,12] blood pressure (BP) seems to be an important component of such augmentation. Additionally, LVM markedly increases progressively with the severity of obesity. Based on the hemodynamic alterations discussed previously, it seems logical that the elevated blood volume and CO would lead to LV dilatation and more eccentric LVH. However, several studies suggest that in Class I and II obesity, the incidence of concentric remodeling and concentric LVH may exceed the incidence of eccentric LVH.[5,7,8] However, without elevated BP, eccentric LVH may be more prevalent. Obviously, studies that assess the impact of obesity on LV geometry should account for obesity severity and levels of BP, as well as duration of obesity.[5,7,8]

Ventricular function

Obesity also has adverse effects on ventricular function.[5,7,8,11,12] Most studies assessing LV systolic function in obese patients using LV ejection phase indices have reported normal or even hyperdynamic LV function. Even in severe obesity, LV systolic dysfunction is relatively uncommon in the absence of coexisting CVD. In severe obesity, however, the presence of severe LVH causes blunting of the exercise induced rise in LV systolic function. Additionally, studies using tissue Doppler imaging have reported subclinical LV systolic dysfunction in obesity that is thought to be load independent, suggesting the presence of intrinsic abnormalities of LV contraction.[5,7,8,11,12]

More importantly, however, studies demonstrate impaired LV diastolic function in obese patients, especially in combination with LVH.[5,7,8] Nevertheless, some studies have reported diastolic LV dysfunction independent of LVM. Although LV diastolic dysfunction has been reported for many years with several imaging techniques,[5,7,8,12,13] more recent studies with tissue Doppler have particularly identified LV diastolic dysfunction as a subclinical disorder in obesity. The prevalence of LV diastolic dysfunction increases with increases of severity of obesity, from 12% of Class I, 35% of Class II, and 45% of Class III obese patients in one study. [14] Most studies demonstrating impaired LV diastolic function in obesity have reported a high prevalence of LVH, with progressive impairment of LV diastolic function with increasing LVM (presumably due to muscle, fibrosis and intra-myocardial fat).[5,7,8]

Additionally, obesity has adverse affects on the left atrium (LA) and right ventricle (RV), and particularly abnormalities are noted with the common combination of obesity, HTN, and LVH.[5,7,8,12,13,15] Recent studies have reported abnormal LA strain in obesity, particularly in those with more LA enlargement and coexisting HTN. [7,16] RV function has not been as extensively studied in obesity, partly due to difficulties with older imaging techniques in obese patients, but studies employing tissue Doppler imaging show reduced systolic and diastolic velocities of the lateral tricuspid valve annulus and reduced RV strain.[7]