

The heart failure and obesity paradox: would you suggest your patients gain weight?

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Hot Topics

Obesity is a well-established risk factor for cardiovascular disease, including hypertension (HTN), heart failure (HF), and coronary heart disease (CHD) [1]. Interestingly, for each increment of 1 unit in body-mass index (BMI), an increase in the risk of heart failure of 5% for men and 7% for women has been estimated [2]. However, once the disease is established, data from clinical cohorts suggest that overweight and obese patients with HTN, HF, and CHD tend to have a more favorable short- and long-term prognosis, a phenomenon termed as “obesity paradox” [3,4]. Although the underlying mechanisms remain elusive, the obesity paradox has led physicians to question whether obesity should be treated when associated with HF.

Obesity paradox clearly represents a controversial area, and this is reflected both in the lack of finite directions from the HF international guidelines, as well in some of the issues regarding data in the literature. First of all, the available data are mostly retrospective in nature and, as such, cannot replace prospective studies. Moreover, although weight loss in chronic HF has consistently been linked to impaired survival [5], there are no data to support that purposeful weight gain improves clinical outcomes of HF patients. In addition, none of the major studies accounted for non-purposeful weight loss (an important phenotype of advance HF patients) or duration of disease. Indeed, over the past 50 years, the prevalence of HF has continuously risen due to improved survival [6]. As a consequence, HF patients present with a longer and a more advanced disease history, which is associated with an increased catabolic state. In this regard, obese patients with HF may represent a patient population with a greater metabolic reserve [5,7] or, in other words, a less advanced disease stage. In fact, obese patients with HF are, on average, younger, have lower natriuretic peptide levels, have more skeletal muscle, better appetite and are less catabolic.

On the other hand, when assessed in other cardiovascular disease setting such as CHD, purposeful weight loss as a result of cardiac rehabilitation program appears to have a favorable impact on long-term outcome, regardless of initial BMI [8]. Moreover, despite the obesity paradox in HF, trials have suggested that weight loss can induce improvements in left-ventricular (LV) mass as well as in systolic and diastolic ventricular function [9–11]. In addition, it has been suggested that, when separately assessed, weight loss and fat loss may have a different impact on mortality rates [12,13].

As outlined, the “weight” of evidence does not support weight gain as a treatment choice in HF patients. Systematic studies on purposeful changes in body composition in HF are warranted to establish the pathophysiology and evidence-driven management of nutritional status in this patient population. •

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