Obesity, Weight Reduction and Survival in Heart Failure

We read with great interest the study by Horwich et al. (1), and while we generally agree with their intriguing findings alluding to better survival with higher body mass index (BMI) among patients with severe systolic heart failure (HF), we strongly object to their conclusions suggesting that promotion of weight loss “may even be potentially harmful.”

In a much smaller cohort of patients with moderate systolic HF who were followed for an average of 19 months (2), we noted that 83 obese patients (BMI >30 kg/m²) had fewer major events (cardiovascular death or urgent transplantation) than 142 nonobese patients (10% events vs. 17% events), although this did not quite meet statistical significance (p = 0.10). Event-free survivors had significantly greater percent body fat (26.4 ± 7.6% vs. 22.6 ± 6.7%; p = 0.02) than those with a major event. When we divided patients into quartiles of percent body fat, the lowest quartile had an annual event rate of 11% compared with 5% in those in the highest quartile (p < 0.05), again implying that body composition may predict prognosis in chronic HF (3), as is also supported by data from others (4). However, whether this relationship is merely an association or is causal is subject to debate, and we strongly suspect the former relationship. It is likely that lower body weight (e.g., lower BMI or lower % body fat) is associated with a heightened metabolic state, associated with higher levels of tumor necrosis factor and other cytokines in the “cardiac cachexia” of severe HF. Clearly, cachexia and wasting appears to be an independent risk factor for mortality in HF (5). In this regard, involuntary weight loss would likely be associated with a bad prognosis, whereas we doubt that this would also apply to purposeful weight reduction.

Clearly, obesity has been associated with numerous adverse effects on hemodynamics and cardiac structure and function, including eccentric left ventricular hypertrophy (LVH) and systolic and diastolic abnormalities, as well as a propensity for more ventricular arrhythmias and sudden cardiac death (6–10). In a study of 74 morbidly obese patients, Alpert et al. (11) demonstrated that nearly one-third had clinical evidence of HF, and the probability of HF increased with increasing duration of morbid obesity (at 20 and 25 years of obesity duration, the probability of congestive heart failure was 66% and 93%, respectively). They demonstrated significant improvements in New York Heart Association (NYHA) functional class in nearly 90% of patients who achieved marked weight reduction; these patients also had significant improvements in systolic and diastolic ventricular function. Likewise, weight reduction has been shown to significantly reduce LVH in obese hypertensives (6,7,10) and to be associated with marked improvements in exercise capacity and coronary risk factors in patients with coronary artery disease (12).

Finally, we urge caution that a “risk marker” should not be confused with a “risk factor” and sweeping conclusions not be entertained. Thus, whereas low serum cholesterol may mark a metabolically severe state of HF (13), it should not be immediately assumed that therapeutic interventions to increase cholesterol shall yield benefits in HF. Although we agree that further research is needed on this topic, it has been well documented that obesity takes a “heavy” toll on the body, particularly on the heart. Until proven otherwise, we believe that purposeful weight reduction remains a viable therapy in the prevention and treatment of most cardiovascular disorders, including HF.

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REFERENCES


REPLY

We appreciate the interest of Lavie et al. with regard to our recent analysis of obesity’s role in the prognosis of advanced heart failure (HF) (1) and the supporting data they have provided from their own institution (2). Our study has found that obese HF patients