EDITORIAL COMMENT

The Growing Problem of Obesity and the Heart

The Plot “Thickens”*

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There is widespread recognition of the developing epidemic of obesity worldwide (1). Obesity imparts a major risk for the secondary complications of diabetes and is associated with increased cardiovascular morbidity and mortality (2). The Framingham investigators found that after adjustment for known coronary risk factors, the relative risk of developing heart failure over 14 years of follow-up increased by 5% to 7% for each increment of one in body mass index (BMI) (3).

In the Framingham cohort, a reduced left ventricular (LV) ejection fraction was present in the majority (66%) of the obese patients who had an echocardiogram performed within 30 days of the diagnosis of heart failure (3). However, this study did not provide data on how many of the patients who developed heart failure had evidence of ischemic heart disease. This is an important consideration because obesity may predispose to heart failure because it is a risk factor for atherosclerosis rather than a direct cause of heart failure. Moreover, the mean age of the patients in that study was 55 years—a population that has a significant risk for coronary disease, even after adjustment for baseline risk factors. In contrast, at least two recent studies have found that younger (mean ages, ∼44 and 31 years, respectively) and otherwise healthy patients with various degrees of obesity have normal LV ejection fractions (8,9). Nonetheless, both groups reported reductions in LV tissue Doppler velocities and/or strain indices that correlated with the severity of obesity. Wong et al. (8) also found that the LV tissue Doppler velocities in both systole and diastole correlated moderately with exercise capacity. Thus, it seems that uncomplicated obesity is associated with subclinical LV dysfunction that can be shown with sensitive measurement techniques.

The Framingham Heart Study criteria for heart failure may define overlapping populations with left, right, or biventricular heart failure (10). Because obesity is the main risk factor for obstructive sleep apnea, it is important to also consider right ventricular (RV) dysfunction as a possible mechanism of heart failure in the obese population. Prior studies have not directly assessed the right ventricle in severely obese patients. In this issue of the Journal, Wong et al. (11) assessed RV function using conventional echocardiographic measures (RV volumes and RV ejection fraction) and newer approaches based on tissue Doppler measurements of long-axis tricuspid annulus velocities and RV strain indices. These studies were performed in 112 overweight or obese patients without organic heart disease and in 36 normal-weight control patients. A subset of patients had overnight polysomnography performed to quantify the severity of sleep apnea. An important control group consisting of normal-weight patients with documented sleep apnea was also studied. Although RV volumes and wall thickness were increased in the obese group, the RV ejection fractions were not different between obese and normal-weight patients. However, the tissue Doppler measurements of tricuspid annular velocities in systole and diastole and the RV strain indices were reduced in the obese patients, particularly those with the highest BMIs. The tissue Doppler velocities had an inverse and independent relationship with BMI but were unrelated to the severity of sleep apnea. The normal-weight patients with sleep apnea did not have tissue Doppler evidence of RV dysfunction.

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A surprise from this study is that RV ejection fraction in severely obese patients was normal and only relatively mild changes in RV systolic function were detected using tissue Doppler imaging. These findings are very similar to those reported previously with respect to LV function in obese patients (8,9). Likewise, RV diastolic function as assessed by tricuspid annular diastolic velocities was also only mildly impaired in severe obesity. Taken together, the accumulating data call into question the widely held perception that obesity is independently associated with a cardiomyopathy per se. It seems somewhat unlikely that this subtle degree of dysfunction is a cause of overt heart failure. Perhaps over longer periods of time more severe abnormalities of systolic and diastolic function are likely to develop. However, the current data did not show a relationship between duration of obesity and the severity of RV dysfunction. In addition, the magnitude of LV dysfunction detected by tissue Doppler in a younger group and an older group of obese patients were comparable (6,8). Thus, the available evidence does not support the notion that LV dysfunction progresses over time in severe obesity.

Despite the fact that the myocardial abnormalities were relatively mild, there were significant correlations between the measures of RV function and maximal exercise tolerance. This finding makes the case that the abnormalities seen on tissue Doppler imaging are clinically relevant. Moreover, the present results reinforce the notion that the ejection fraction (of either ventricle) is an insensitive measure of systolic dysfunction, whereas tissue Doppler measurements are more sensitive for detecting such abnormalities (12,13). In addition, the high signal-to-noise ratio of tissue Doppler measurements makes them easier to record, relatively simple to quantify, and very reproducible as compared with approaches that require clear endocardial visualization. Lastly, tissue Doppler measurements are advantageous because they take into account the longitudinal component of contraction (which may be particularly important for the right ventricle) and do not require any geometric assumptions. They will likely become the standard method of assessing RV function.

Additional unexpected findings are seen in this study. Severely obese patients, even those with significant sleep apnea, have very little tricuspid valve regurgitation. Those that do have tricuspid regurgitation seem to have fairly normal pulmonary artery systolic pressures. Not only do these findings show a surprising degree of adaptation to an extreme physiological condition, but they also suggest that LV filling pressures are not elevated enough to cause passive pulmonary hypertension. The notion that LV filling pressures may be fairly normal in extreme obesity is also supported by the findings of normal E/E' values in obese patients (8,9) and brain natriuretic peptide levels that are reduced compared with those in lean patients (14). Of note, previous findings from the Framingham Heart Study suggest that the prevalence of valvular insufficiency is lower in obese patients than in normal-weight people (15).

With the rapid increase in the prevalence and severity of obesity around the globe, it is extraordinarily important to better understand this condition and its effects on the heart. Given the interesting new data by Wong et al. (11), are we closer to this goal? Their findings, taken in context with other recent publications, convincingly show that obesity is consistently associated with mild but significant reductions in systolic and diastolic function of both left and right ventricles. The severity of the dysfunction is related to the magnitude of obesity. Comorbidities are not prerequisites for the presence of myocardial contractile abnormalities. Nonetheless, the current data do not prove that obesity directly leads to heart failure, nor do they help us to determine the future risk of heart failure in individual obese patients. Moreover, the incidence of heart failure has not increased at pace with the profound increase in obesity in our society. If obesity serves in large part as a risk factor for the development of coronary disease, then it makes sense that there would be a substantial lag between an increase in the incidence of obesity and a subsequent escalation of heart failure. Alternatively, there may be a distinct, but very slow (>20 years) progression of myocardial dysfunction in obesity. In either case, it seems that there may be a window of opportunity for treating related pathologies (i.e., hypertension) and preventing the final culprits in the pathogenesis of obesity-related heart failure (i.e., coronary artery disease). As yet, we do not know to what extent the myocardial dysfunction is reversible with weight loss. Lastly, before condemning the ill effects of obesity, we must ponder the considerable body of data showing improved survival in obese patients with heart failure or acute coronary syndromes (the obesity paradox) (16). Perhaps the cardiac hypertrophy and mild dysfunction are the price that is paid in exchange for the cardioprotective effects that have consistently been reported in obese patients. It would behoove us to consider the possibility that some aspect of obesity may precondition the heart against subsequent insults.

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REFERENCES


