Editorial

Left Ventricular Morphology and Diastolic Function in Severe Obesity: Current Views

Morfología ventricular izquierda y función diastólica en la obesidad grave: perspectivas actuales

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Early observations of cardiac morphology in obesity focused primarily on the prominence of epicardial fat.1,2 These include anecdotal descriptions by such luminaries as René Laennec, William Harvey, and Jean Nicholas Corvisart.1 In 1933, Smith and Willius reported autopsy findings on 135 obese patients including 4 who were severely obese.1–3 In this study heart weight increased in proportion to the degree of obesity up to 105 kg and to a lesser extent thereafter.1,3 Heart weight was greater than predicted for normal weight and height in both men and women and was far greater than expected in severely obese persons. Nearly all patients had excess epicardial fat and the authors ascribed increased heart weight to this phenomenon. In 1957, Lillington reported biventricular hypertrophy in a severely obese man who died of pulmonary embolism, thus raising the question of left heart involvement in obesity.1,2 In 1962, Alexander reported the results of a retrospective survey of autopsy studies of extremely obese patients previously cited as having cor pulmonale. Left ventricular hypertrophy (LVH) and pulmonary congestion had been present in each case.1,2 In 1965, Amad et al. reported the results of postmortem studies of 12 severely obese patients.1,2 They confirmed that heart weight was increased in all individuals, but demonstrated that it was largely due to LVH. The left ventricle (LV) wall thickness was increased in 11 of the 12 patients and myocyte hypertrophy on microscopic examination was present in all 12. Right ventricular wall thickness was increased in 1 patient and epicardial fat was increased in 6. A later study of 12 severely obese patients by Warnes and Roberts reported increased heart weight, LV wall thickness, microscpic LVH, and excess epicardial fat in all cases and increased right ventricular wall thickness in 4 patients.1,2 Complicating these observations was the fact that systemic hypertension and/or coronary artery disease were present premortem in many of the subjects in these studies.

The advent of echocardiography permitted premortem assessment of cardiac structure in morbidly obese patients. In a study of 62 normotensive severely obese subjects, Alpert et al. reported LV enlargement in 40%, increased LV wall thickness in 56%, increased LV mass in 64%, left atrial enlargement in 50%, and right ventricular enlargement in 32%.1,2 Multiple studies have compared LV morphology in obese patients to that of lean subjects.3–5 LV mass, mass index, mass/height index, or mass/height2,3 were significantly greater in obese than in lean patients in all of these studies regardless of the severity of obesity.3–4 The LV internal dimension in diastole was significantly larger in mildly, moderately, and severely obese patients compared to lean subjects.2–4 Interventricular septal thickness and LV posterior wall thickness were significantly greater in obese than in lean patients regardless of severity of obesity.2–4 Calculated LV radius to thickness ratios were elevated in most but not all of these studies, suggesting that LVH in obese patients, when present, is usually eccentric.2 It is notable that not all of these studies excluded hypertensive patients. A study by Kasper and coworkers of 409 lean and 43 severely obese patients with heart failure showed a higher prevalence of dilated cardiomyopathy in obese than in lean patients.4 A specific cause was identified in 64% of lean and only 23% of obese patients, thus supporting the existence of a cardiomyopathy of obesity. Myocyte hypertrophy was present in 67% of biopsies of obese patients. Based on postmortem and echocardiographic studies reported during the 1980s and 1990s the following cardiac structural abnormalities were identified in obese subjects: increased heart weight, LV enlargement, increased LV wall thickness, an increased LV radius to thickness ratio, increased LV mass, microscopic LVH, left atrial enlargement, right ventricular enlargement and hypertrophy, and excessive epicardial fat.

With the exception of excessive epicardial fat, the aforementioned abnormalities associated with obesity evolve from altered hemodynamics.5–7 Obesity, particularly severe obesity, produces hemodynamic alterations that predispose to changes in cardiac structure and function.5–7 Excessive adipose accumulation together with increased fat-free mass combine to increase central blood volume. In the absence of systemic hypertension a reduction in systemic vascular resistance facilitates augmentation of cardiac output.5–7 Since heart rate changes little if at all with increasing fat mass, the rise in cardiac output is due entirely to increased LV stroke volume. Augmentation of cardiac output leads to dilation of the LV, left atrium, and right ventricle. The increase in LV chamber size (and therefore radius) causes LV wall stress to rise in accordance with the law of LaPlace.5–7 This in turn is thought to
Concepts related to the progression of LVH include eccentric LVH and concentric LVH. Eccentric LVH is characterized by increased left ventricular wall thickness with normal LV mass, whereas concentric LVH is characterized by increased LV mass with normal wall thickness.}

Eccentric LVH and concentric LVH are often associated with different clinical manifestations and outcomes. For example, eccentric LVH is typically associated with hypertension, diabetes, and obesity, while concentric LVH is often associated with systemic hypertension, left ventricular hypertrophy, and pulmonary hypertension.

The progression of LVH is often linked to factors such as age, gender, family history, and lifestyle factors. For example, a sedentary lifestyle and poor diet have been associated with a higher risk of LVH.

To prevent the progression of LVH, it is important to identify and manage risk factors such as hypertension, diabetes, and obesity. Treatment options for LVH may include lifestyle modifications, medication, and medical interventions such as endarterectomy or pacemaker implantation.
LV diastolic function that presumably are load independent. The authors suggest that failure of load-independent LV diastolic function to improve despite regression of LV following substantial weight loss in severely obese patients may be due to myocardial fibrosis. That may well be the case and may explain disparate LV filling pressure responses to weight reduction in severely obese patients. It would have been of interest to compare and contrast LV diastolic function in patients with and without LVH in this study.

The preceding discussion has focused on obesity-related changes in cardiac morphology and performance in adults. An increasing body of evidence suggests that similar LV remodeling occurs in overweight and obese children and adolescents. In conclusion, the study by Luaces et al., published in Revista Española de Cardiología demonstrates that LV remodeling occurs commonly in severely obese patients. Although eccentric LVH predominated, concentric LVH or remodeling was noted in a substantial percentage of patients. Substantial weight loss from bariatric surgery had a beneficial effect on LV geometry. At 1 year nearly 60% of patients demonstrated normal LV geometry. However, LV diastolic function assessed by tissue Doppler did not significantly change following substantial weight loss despite a decrease in LV mass. This suggests that nonmodifiable factors such as fibrosis may contribute to LV diastolic dysfunction in severe obesity and may prevent improvement in LV diastolic function following weight loss despite regression of LVH.

CONFLICTS OF INTEREST

None declared.

REFERENCES


