Obesity and the Heart: Cardiovascular Magnetic Resonance Imaging Evidence of the Beneficial Effects of Bariatric Surgery

Oliver J. Rider, James P. Byrne, Stefan Neubauer

ABSTRACT

Obesity is associated with cardiac hypertrophy and increased aortic stiffness, which are independent predictors of cardiovascular risk. In the setting of obesity, bariatric surgical weight loss has been shown to reduce mortality. We present a case showing that, in a severely obese individual without comorbidities, these cardiovascular responses to excess weight are reversible during weight loss over a period of 1 year.

Key Words: Obesity, Bariatric surgery, Left ventricle, Aortic stiffness, Cardiac magnetic resonance, Myocardial energetics, Diastole.

INTRODUCTION

Obesity, defined by a body-mass index (BMI) >30kg/m², is associated with increased mortality rate, and even greater risk is associated with a BMI of ≥35kg/m². Both ventricular hypertrophy and aortic stiffness are present in obesity,2–4 and a growing body of literature has demonstrated a strong relationship between left ventricular hypertrophy and all cause mortality on one hand,5,6 and between impairment of aortic elastic function and cardiovascular events in healthy and diseased populations on the other hand.

Excess adiposity is associated with a hyperdynamic circulation and increased blood volume which, in combination with the hormonal changes in obesity, including leptin,7 produce a spectrum of cardiac changes ranging from asymptomatic left ventricular hypertrophy through diastolic dysfunction to overt systolic heart failure.2,6,8,9 Because obesity is linked to left ventricular hypertrophy, diastolic dysfunction, and aortic stiffness (all independent predictors of cardiovascular mortality5,10) and increased mortality, this hypertrophic response provides a potential mechanism by which obesity modulates cardiovascular risk.

Bariatric surgical weight loss is now known to confer mortality benefit,11 and left ventricular hypertrophy aortic stiffness and diastolic function are seen to improve with laparoscopic bariatric surgical weight loss.9

CASE STUDY AND DISCUSSION

Here we present anthropometric data, serum markers of obesity, and cardiovascular magnetic resonance imaging data of the left ventricular structure and function as well as aortic distensibility of a 38-y-old Caucasian female before and after laparoscopic adjustable gastric banding for the treatment of obesity to highlight the effects of weight loss on left ventricular structure.

Prior to the surgery, the patient weighed 108kg (BMI of 37.3kg/m²), had no history of cardiovascular disease, no history of smoking, and was normotensive. Her fasting plasma glucose, total cholesterol, and insulin were within the normal adult range (Table 1). Calculated insulin resistance using the Homeostasis Model of Insulin Resis-
distance (HOMA) was 2.69. Serum leptin, and C-reactive protein (CRP) levels were however elevated above the normal range (Table 1).

Twelve months after bariatric surgery she had lost 28kg in weight, representing a 79% loss of her excess body weight. After weight loss, blood pressure, glucose, total cholesterol, insulin, and HOMA levels were similar to those at before weight loss (Table 1). In comparison, serum leptin and CRP levels were markedly lower.

Figure 1A shows a midventricular short axis cardiac magnetic resonance image prior to surgery demonstrating left ventricular mass and cavity size at the upper end of the normal range (LV mass 130g, [upper normal limit 138g] LV end-diastolic volume 164mL [upper normal limit 187mL]). Figure 1B was taken 12 mo after weight loss surgery, at the same midventricular level and image magnification. The postoperative image shows both a 25% reduction in left ventricular mass and the decrease in cavity size that occurred as a consequence of the weight loss surgery (LV mass 102g, LV end-diastolic volume 154mL). The gastric band is clearly visible in the image below the left ventricle (labelled GB). In addition to this, left ventricular diastolic function and aortic distensibility were seen to increase (at all 3 levels measured) indicating improved aortic elastic function (Table 1). Given the reduction in serum leptin and CRP, both of which have been linked to left ventricular hypertrophy and aortic stiffness, it is plausible that these hormones and their reduction with weight loss play at least some role in the cardiovascular changes that occur with obesity and subsequent weight loss.

Table 1. Anthropometric, Serum Markers of Obesity, Left Ventricular Characteristics, and Aortic Distensibility Before and After Weight Loss

|                      | Pregastric Band | Postgastric Band |
|----------------------|-----------------|------------------|
| Body Mass Index (kg/m²) | 37.3            | 27.6             |
| Weight (kg)          | 107.9           | 79.7             |
| Systolic Blood Pressure (mm Hg) | 138            | 132              |
| Waist (Inches)       | 56              | 44               |
| Hip (Inches)         | 57              | 49               |
| Diastolic Blood Pressure (mm Hg) | 82            | 75               |
| Excess Weight (kg)   | 65              | 41               |
| Visceral Fat Mass (cm³) | 1287           | 420              |
| Fasting Serum Glucose (mmol/L) | 6.5           | 4.9              |
| Fasting Total Cholesterol (mmol/L) | 5.4          | 6.1              |
| HDL Cholesterol (mmol/L) | 0.92           | 1.0              |
| Triglycerides (mmol/L) | 0.8            | 0.1              |
| Fasting Insulin (mmol/L) | 5.3           | 9.2              |
| Serum Leptin (mmol/L) | 239             | 23               |
| C-reactive Protein (mg/L) | 10.1          | 0.39             |
| Fasting Free Fatty Acids (mol/L) | 0.61          | 0.60             |
| HOMA - IR            | 2.69            | 2.87             |
| Left Ventricular Mass (g) | 138            | 102              |
| Left Ventricular Stroke Volume (mL) | 106           | 106              |
| Left Ventricular End-Diastolic Volume (mL) | 164          | 155              |
| Left Ventricular End-Systolic Volume (mL) | 58            | 49               |
| Left Ventricular Ejection Fraction (%) | 65           | 68               |
| Left Ventricular Peak Diastolic Filling Rate (EDV/s) | 2.31         | 4.44             |
| Ascending Aortic Distensibility (mm Hg/mm² × 10⁻³) | 7.66         | 8.22             |
| Proximal Descending Aortic Distensibility (mm Hg/mm² × 10⁻³) | 2.40         | 5.84             |
| Abdominal Aortic Distensibility (mm Hg/mm² × 10⁻³) | 7.35          | 7.76             |

Figure 1. T1 weighted TSE images (A) pre and (B) post-laparoscopic adjustable gastric band showing a large reduction in visceral and subcutaneous fat. Short axis MRI view showing the reduction of left ventricular mass and size following weight loss due to laparoscopic adjustable gastric banding. Before gastric banding (C) and clearly visible gastric band (GB) in the right lower quadrant of the image (D).
CONCLUSION

These images illustrate succinctly the left ventricular adaptations to obesity next to a gastric band, an effective and safe surgical method of weight reduction associated with reduced long-term mortality in obese subjects.\textsuperscript{11} The regression in visceral fat, left ventricular mass, diastolic dysfunction, and aortic stiffness represents a potential mechanism to explain the reduction in mortality rates seen with significant effective weight loss highlighting the benefits of laparoscopic surgery in the fight against obesity related heart disease.

References:

1. Adams KF, Schatzkin A, Harris TB, et al. Overweight, obesity, and mortality in a large prospective cohort of persons 50 to 71 years old. \textit{N Engl J Med}. 2006;355:763–778.
2. Alpert MA. Obesity cardiomyopathy: pathophysiology and evolution of the clinical syndrome. \textit{Am J Med Sci}. 2001;321:225–236.
3. Sutton-Tyrrell K, Newman A, Simonsick EM, et al. Aortic stiffness is associated with visceral adiposity in older adults enrolled in the study of health, aging, and body composition. \textit{Hypertension}. 2001;38:429–433.
4. Mattace-Raso FU, van der Cammen TJ, Hofman A, et al. Arterial stiffness and risk of coronary heart disease and stroke: the Rotterdam Study. \textit{Circulation}. 2006;113:657–663.
5. Levy D, Anderson KM, Savage DD, et al. Echocardiographically detected left ventricular hypertrophy: prevalence and risk factors. The Framingham Heart Study. \textit{Ann Intern Med}. 1988;108:7–13.
6. Vyakili BA, Okin PM, Devereux RB. Prognostic implications of left ventricular hypertrophy. \textit{Am Heart J}. 2001;141:334–341.
7. Rajapurohitam V, Gan XT, Kirshenbaum LA, Karmazyn M. The obesity-associated peptide leptin induces hypertrophy in neonatal rat ventricular myocytes. \textit{Circ Res}. 2003;93:277–279.
8. Rider OJ, Francis JM, Ali MK, et al. Determinants of left ventricular mass in obesity: a cardiovascular magnetic resonance study. \textit{J Cardiovasc Magn Reson}. 2009;11:9.
9. Rider OJ, Francis JM, Ali MK, et al. Beneficial cardiovascular effects of bariatric surgical and dietary weight loss in obesity. \textit{J Am Coll Cardiol}. 2009;54:718–726.
10. Bhatia RS, Tu JV, Lee DS, et al. Outcome of heart failure with preserved ejection fraction in a population-based study. \textit{N Engl J Med}. 2006;355:260–269.
11. Adams TD, Gress RE, Smith SC et al. Long-term mortality after gastric bypass surgery. \textit{N Engl J Med}. 2007;357:753–761.