Clinical Spotlight

Take Heart: Bariatric Surgery in Obese Patients with Severe Heart Failure. Two Case Reports

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Bariatric surgery may be an effective treatment for obese heart failure patients, enabling access to cardiac transplantation and/or improvement of symptoms. We report the outcomes of two morbidly obese patients with end-stage heart failure, where obesity precluded cardiac transplantation and underwent laparoscopic gastric banding. A 42 year-old male with idiopathic dilated cardiomyopathy weighing 124.4 kg (BMI 42 kg/m^2) lost 34 kg and was successfully transplanted 11 months later. A 40 year-old woman with familial dilated cardiomyopathy weighing 105 kg (BMI 40 kg/m^2) lost 14 kg with sufficient symptomatic resolution to no longer require cardiac transplantation. In selected patients with severe heart failure and concomitant morbid obesity, bariatric surgery may be a reasonable treatment option.

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Introduction

Obesity has reached epidemic proportions in much of the industrialised world, with 60% of adults classified as overweight or obese [1]. Obesity impacts significantly on the development of numerous diseases, including cardiovascular disease, diabetes mellitus and hypertension. Bariatric surgery is currently the only effective, long-term treatment for obesity [2,3]. Obesity impacts on all aspects of cardiac function, including exacerbation of cardiac risk factors, increased cardiac output and potentially cardiac remodelling. Current literature demonstrates that patients with heart failure and BMI >35 kg/m^2 have significantly diminished long-term survival and greater morbidity when compared to patients with BMI 18.5–34.99 kg/m^2 [4].

Case 1

A 42 year-old man with a 16-year history of idiopathic cardiomyopathy and NYHA Class III heart failure was referred for heart transplant assessment. He was managed with carvedilol 25 mg bd, spironolactone 25 mg mane, ramipril 2.5 mg nocte, frusemide 250 mg mane, atorvastatin 80 mg nocte, fenofibrate 145 mg nocte and warfarin and had a pacemaker in situ. He had insulin-treated type 2 diabetes mellitus (10 years) treated with preprandial insulin aspart (90 units daily), glargine 30 units nocte and metformin 500 mg t.d.s. The HbA1c of 9.4% indicated poor glycaemic control. Past history included three episodes of pancreatitis with severe hypertriglyceridemia (up to 48.3 mmol/L). Fasting lipids showed total cholesterol 5.7 mmol/L, HDL 0.6 mmol/L and triglycerides 10.3 mmol/L. In the preceding two years, he had gained 20 kg to reach a peak weight of 124.4 kg, BMI 42 kg/m^2, associated with symptomatic deterioration of heart failure. His echocardiogram showed severe left ventricular dilatation and dysfunction, with a left ventricular ejection fraction of approximately 20%. In line with national guidelines [5], he was considered unsuitable for cardiac transplantation due to morbid obesity.

The patient was placed on a supervised calorie-restricted diet with the option of using meal replacement shakes providing 140 kCal per serve. Insulin doses were reduced commensurate with the caloric restriction and electrolytes and renal function monitored. Dose reductions in insulin and diuretic therapy were required; however, there was minimal weight loss (2 kg) after three months. Bariatric surgery was undertaken with laparoscopic gastric banding placement. Intra-operative management included monitoring of right heart pressures

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Table 1. *The Effect of Gastric Banding on Anthropometric, Metabolic and Cardiac Measures in Two Morbidly Obese Patients with Severe Heart Failure.*

Variable	Case 1			Case 2		
	Baseline	6 months	12 months	Baseline	6 months	12 months
Weight	124.4 kg	98.7 kg	89 kg	105 kg	91 kg	91 kg
BMI	42 kg/m^2	34.6 kg/m^2	31.2 kg/m^2	40 kg/m^2	34.7 kg/m^2	34.7 kg/m^2
HbA1c	9.9%	6.6%	6.7%	–	–	–
Total cholesterol	5.7 mmol/L	3.2 mmol/L	3.8 mmol/L	3.3 mmol/L	3.9 mmol/L	
Triglycerides	10.3 mmol/L	3.0 mmol/L	2.2 mmol/L	1.2 mmol/L	1.2 mmol/L	
LV ejection fraction	20%	–	15%	15%	–	10–15%

by Swan–Ganz catheterisation, continuing for 24 h post-op for close management of cardiac function. His course was uncomplicated and he had an unremarkable recovery.

Three months post-op, the patient had reduced to 107.5 kg. His diabetes and hypertriglyceridemia had improved (HbA1c 6.6%).

By 11 months, the cumulative weight loss was 34, to a weight of 89 kg. He had on-target glycaemic control with HbA1c 6.7%, requiring only glargine 15 units nocte and metformin. As there was no improvement in objective measures of cardiac function by echocardiography (Table 1), clinical signs or the patient's NYHA Class III symptoms of heart failure, he was listed for cardiac transplantation. A donor heart became available two months later and the patient underwent successful heart transplantation. The explanted heart weighed 432 g. The histology showed severe myopathic features with a severe degree of anisocytosis, severe myocytolysis and a severe diffuse degree of interstitial fibrosis. Atheromatous plaque was present in some coronary arteries, with no occlusions beyond 50%. The findings were consistent with a severe dilated cardiomyopathy.

Case 2

A 40 year-old obese woman with an eight year history of familial dilated cardiomyopathy and NYHA Class III symptomatic heart failure was referred for heart transplant assessment. The patient had undergone mitral valve annuloplasty after cardiomyopathy diagnosis, with temporarily improved left ventricular ejection fraction (from 15% to 29%)) and implantation of an automated cardioverter-defibrillator. Medications were carvedilol 6.25 mg bd, rosuvastatin 20 mg, frusemide 80 mg, spironolactone 25 mg, perindopril 2.5 mg and warfarin.

Exercise tolerance declined, with walking reduced to less than 600 m flat grade or a maximum of 14 stairs. An echocardiogram revealed severe left ventricular dilatation, moderate mitral regurgitation and an ejection fraction of 15%. The patient was placed on the cardiac transplantation waiting list, weighing 92 kg (BMI 35 kg/m^2). She ceased smoking, however gained weight over 7 months, up to 105 kg, with BMI 40 kg/m^2. She was delisted for transplantation due to worsening obesity.

The patient made several unsuccessful attempts at weight loss (maximum 3 kg loss over 6 months). Laparoscopic gastric band placement was performed with no anaesthetic or operative complications.

Three months post-op, the patient had lost 1 kg, to a weight of 95 kg. She was able to exercise up to 30 min daily. At six months, the cumulative weight loss was 14 kg, with weight 91 kg, BMI 34.7 kg/m^2. The patient's heart failure symptoms improved to NYHA Class II and she was judged to be too well to be reconsidered for heart transplantation, despite little echocardiographic change: the left ventricle remained dilated, with an ejection fraction of 10–15% and pulmonary artery systolic pressure of 24 mmHg.

Discussion

These cases highlight the benefits of bariatric surgery in treating obese patients with severe heart failure, particularly after unsuccessful attempts at weight loss. Obesity is a contraindication for cardiac transplantation, due to poor outcomes [6]. In the cases above, achieving a lower BMI after bariatric surgery provided access to cardiac transplantation with positive outcomes for both. Case 1 underwent successful transplantation and Case 2 experienced symptomatic improvement such that transplantation was no longer necessary.

The link between obesity and heart failure is established. The Framingham Heart Study reported doubling of the risk of incident heart failure in the obese [7]. Obesity increases cardiac load by effects on blood volume, heart rate, left ventricular loading, as well as contributing to risk factors that impact on the heart, including blood pressure, hyperlipidaemia, diabetes risk, insulin resistance and sleep apnoea/hypoxia. Weight loss induces multiple cardiovascular benefits in obese patients, including improved cardiac remodelling, blood pressure, lipid profile and glucose tolerance. In both cases presented, the patients were euvolaemic at all times. Thus, the improvement in heart failure symptoms in Case 2 cannot be attributed to weight loss-induced improvements in fluid overload. However, there are only early reports on the impact of bariatric surgery on obese patients with severe heart failure. One report of 14 subjects reported improved left ventricular ejection fraction six months after bariatric surgery; 85% had downgrading of their NYHA classification from Class IV or III to II [8]. In two obese patients with NYHA Class IV heart failure, bariatric surgery improved symptoms sufficiently that cardiac transplantation was no longer necessary [9], as occurred in our Case 2. With only a small number of case reports and a single small series, the published literature includes diverse heart failure aetiologies. Furthermore, no randomised trials have yet reported

the effects of bariatric surgery on the obese with severe heart failure.

It is hypothetically feasible that obesity-related cardiomyopathy may improve after weight loss, however much is unclear about the response of different forms of cardiomyopathy to weight loss. In this report, one case with idiopathic dilated cardiomyopathy had no improvement after massive weight loss. In contrast, the second case with familial cardiomyopathy had symptomatic improvement sufficient to no longer require transplantation. Given limited organ availability, reducing demand is desirable. Our cases demonstrate that heart failure in some obese patients may respond to bariatric surgery, which may be a feasible intervention in centres where anaesthetic and intensive care expertise supports peri-operative management of these critically unwell patients.

The varying response for both cases highlights the need for further research into the effects of bariatric surgery on patients with non-obesity-related cardiomyopathy, particularly whether baseline criteria may be determined to predict response to bariatric surgery. Even without cardiac improvement following weight loss, bariatric surgery may be a means of permitting access to lifesaving cardiac transplantation in patients with severe heart failure where obesity has previously prevented this therapeutic option.

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