CASE REPORT

Regression of severe heart failure after combined left ventricular assist device placement and sleeve gastrectomy

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Abstract

Patients who suffer morbid obesity and heart failure (HF) present unique challenges. Two cases are described where concomitant use of laparoscopic sleeve gastrectomy (LSG) and left ventricular assist device (LVAD) placement enabled myocardial recovery and weight loss resulting in explantation. A 29-year-old male patient with a body mass index (BMI) of 59 kg/m^2 and severe HF with a left ventricular ejection fraction (LVEF) of 20–25% underwent concomitant LSG and LVAD placement. Sixteen months after surgery, his BMI was reduced to 34 kg/m^2 and his LVEF improved to 50–55%. A second 41-year-old male patient with a BMI of 44.8 kg/m^2 with severe HF underwent the same procedures. Twenty-four months later, his BMI was 31.1 kg/m^2 and his LVEF was 50–55%. In both cases, the LVAD was successfully explanted and patients remain asymptomatic. HF teams should consult and collaborate with bariatric experts to determine if LSG may improve the outcomes of their HF patients.

Introduction

In the USA, 39.8% of the adult population has a body mass index (BMI) > 30 kg/m^2 and 15.4% are considered to have morbid obesity (BMI > 40 kg/m^2).[1] Excess weight and excess body fat result in numerous obesity-related comorbidities that affect the cardiovascular, musculoskeletal and respiratory systems.[2,3] Numerous studies have shown the efficacy of bariatric surgery for the treatment of morbid obesity and its related comorbidities.[4–9]

Heart failure (HF) is typically progressive, and in advanced cases, patients may require a left ventricular (LV) assist device (LVAD) or cardiac transplantation. Bariatric surgery has been shown to improve cardiac function in patients with HF,[10–12] but its potential to significantly improve or normalize LV ejection fraction (LVEF) is debated. We present two patients with morbid obesity and advanced HF who significantly improved

after concomitant laparoscopic sleeve gastrectomy (LSG) and LVAD placement and resulted in explantation of the LVADs.

Case report

Patient 1

A 29-year-old man with morbid obesity (BMI 59.05 kg/m^2), asthma, and hypertension was admitted to an outside facility with worsening dyspnoea (*Table 1*). The hypertension was managed by angiotensin-converting enzyme inhibitors and beta-blocker as a part of guideline-directed medical therapy for his chronic systolic HF. Our institution uses clinical symptoms, laboratory results, and echocardiograms to diagnose HF per standard guideline. The modified Simpson method

Table 1 Baseline characteristics of patients

	Patient 1	Patient 2
Age (years)	29	41
Gender	Male	Male
Initial body mass index (kg/m^2)	59.0	44.8
Excess body weight (kg)	113.9	61.0
Comorbidities		
Hypertension	Yes	Yes
Obstructive sleep apnoea	No	Yes
Chronic kidney disease	No	Yes
Diabetes mellitus type II	No	No
Hyperlipidaemia	No	No

was utilized for assessment of LV systolic function, which is a standard method for evaluation of ejection fraction in patients.

An echocardiogram revealed dilated cardiac chambers and severe reduction in LV systolic function with an LVEF of 20–25% (*Table 2*). Extensive evaluation including diagnostic coronary angiography revealed no precipitating or reversible cause of HF. In view of worsening symptoms despite optimal

medical therapy, he was transferred to our facility for advanced therapy options.

Invasive haemodynamics revealed advanced HF. The patient was reviewed by the medical review board and approved for an LVAD. Given the morbid obesity, a concomitant LSG was performed in the hope that with weight loss, the patient will eventually be a heart transplant candidate. The patient received a Heartmate II™ (Thoratec Corporation, Pleasanton, CA) with concomitant LSG. Total operative time was 6 h and 58 min. Patient was extubated on post-operative Day 2 and had an uneventful recovery. He was discharged home on post-operative Day 11. Standard guideline therapy was continued for HF and the up-titrations of medication were based on patient's haemodynamics and symptoms.

After 10 months, a repeat echocardiogram revealed a normal LV systolic function (LVEF = 50–55%). He had lost 57.8 kg and had a BMI of 35.6 kg/m^2. Repeat echocardiogram at approximately 13 months post-op revealed continued normalization of systolic function. Given the sustained myocardial recovery, tolerability of guideline-directed medical therapy

Table 2 Echocardiogram data for patients

Patient 1			
	Just prior to LVAD/LSG procedure	Just prior to explantation procedure	Within 1 month of explantation
LVEF (%)	25	50	54
LV dilation	Severe	Normal	Normal
LVIDd/LVIDs (cm)	6.8/6.0	5.4/3.9	5.9/4.3
LA dilation	Severe	Normal	Normal
LA diameter (cm)	4.1	4.3	4.3
LA volume (mL)	74.0	93.1	76.9
LV hypertrophy	Severe, concentric	None	None
IVSd (cm)	1.4	1.2	1.2
LVPWd (cm)	1.5	1.1	1.2
RVSP	35 mmHg	Cannot be calculated; No TR noted	
RVSP (TR)	30 mmHg		
RA pressure	5 mmHg		

Patient 2				
	Just prior to LVAD/LSG procedure	Just prior to explantation procedure	Within 1 month of explantation	1 year follow-up appointment
LVEF (%)	16	54	59	45
LV dilation	Moderate	Normal	Normal	Mild
LVIDd/LVIDs (cm)	7.3/6.9	4.7/3.4	47/3.2	5.3/4.1
LA dilation	Severe	Mild	Mild	Moderate
LA diameter (cm)	5.4	3.5	4.2	4.2
LA volume (mL)	107	67.8	64.2	88.3
LV hypertrophy	None	None	None	None
IVSd	1.04	1.1 cm	0.8 cm	0.9 cm
LVPWd	1.04	1.0 cm	0.8 cm	0.9 cm
RVSP (mmHg)	36	28	33	IVC could not be visualized
RVSP (TR) (mmHg)	33	18	30	
RA pressure (mmHg)	3	10	3	

IVSd, interventricular septal end diastole; LA, left atrial; LSG, laparoscopic sleeve gastrectomy; LV, left ventricular; LVAD, left ventricular assist device; LVIDd, left ventricular internal diameter end diastole; LVIDs, left ventricular internal diameter end systole; LVPWd, left ventricular posterior wall thickness at end diastole; RA, right atrial; RVSP, right ventricular systolic pressure; TR, tricuspid regurgitation. The left ventricular ejection fraction was measured by the modified Simpson's method. All other measurements were recorded using standard 2D methods.

for HF, he underwent echocardiographic evaluation at lower pump speed, which revealed continued normalized LV function. The decision was made to proceed with the LVAD explantation at approximately 16 months post-surgery. At that time, his BMI was 34.44 kg/m^2. At the last follow-up (approximately 5 months after explantation), the patient was doing well. His BMI was 35.24 kg/m^2 (total weight loss 74.8 kg).

Patient 2

A 41-year-old male patient with chronic systolic HF, hypertension, obstructive sleep apnoea, morbid obesity (BMI 44 kg/m^2), and stage 2 chronic kidney disease was admitted to an outside hospital with worsening dyspnoea and New York Heart Association class 4 symptoms (*Table 1*). The hypertension was managed by angiotensin-converting enzyme inhibitors and beta-blocker as a part of guideline-directed medical therapy for his chronic systolic HF. An echocardiogram revealed severe LV systolic dysfunction (LVEF = 20–25%) and severe mitral regurgitation (*Table 2*). Haemodynamics indicated cardiogenic shock. Extensive evaluation including diagnostic coronary angiography revealed no precipitating or reversible cause of HF. The patient was reviewed by the medical review board and approved for a Heartmate II™. Given the morbid obesity, a concurrent LSG alongside the LVAD implant was carried out. Total operative time was 7 h and 20 min. The patient was extubated on post-operative Day 1. Patient was discharged home on post-operative Day 16. Standard guideline therapy was continued for HF and the up-titrations of medication were based on patient's haemodynamics and symptoms.

After 13 months, a repeat echocardiogram demonstrated normal LV size and systolic function (LVEF = 50–55%). The up-titration of the patient's HF therapy was at maximum tolerated doses (carvedilol 25 mg/twice per day; sacubitril/valsartan 24/26 mg/once per day; and aldactone 12.5 mg/once per day). Given the myocardial recovery, he underwent LVAD explantation at approximately 24 months post-surgery. At that time, his BMI was 31.1 kg/m^2 (total weight loss 42.3 kg). At approximately 22 months post-explantation, his LVEF remained normal (50–55%). At last follow-up approximately 29 months after explantation, the patient was doing well, and his medical therapy has continued (carvedilol 9.375 mg/twice per day). His BMI was 35.88 kg/m^2 (total weight loss 30.7 kg).

Discussion

This study describes two cases of advanced HF with morbid obesity and severe LV systolic dysfunction who underwent concomitant LSG and LVAD placement. Sixteen and 24 months post-op, both patients had a normalized LVEF (50–55%) and complete resolution of their HF-related symptoms. Their BMIs were below 35, and both successfully underwent LVAD explantation. The improvement in LVEF for these two patients (25–30%) was significantly higher than patients reported in previously published studies[10–18] and obviated the need for cardiac transplantation.

Patients with obesity have an overall twofold risk of developing HF as compared with the normal population, with an increased risk of 5% in men and 7% in women for each increment increase of 1 kg/m^2 in BMI.[19] Research indicates that obesity leads to heart disease through several direct and indirect pathways. Excess body fat results in myocardial accumulation of triglycerides,[20] and this accumulation causes a chronic, systemic inflammatory response with an increased production of cytokines and acute-phase proteins.[21] These two mechanisms can directly lead to myocardial fibrosis.[20,21] On the other hand, obesity-related comorbidities indirectly affect cardiac function. Hypertension increases LV afterload, which may cause left cardiac hypertrophy.[22] Dyslipidaemia and diabetes mellitus promote atherosclerosis, which increases the risk of ischaemic cardiomyopathy.[23] Insulin resistance decreases myocardium contractility and potentiates the negative impact of hypertension on cardiac function by activating the renin–angiotensin–aldosterone system.[24,25] Obstructive sleep apnoea syndrome causes hypoxaemia, hypercapnia, and oxidative stress with subsequent sympathetic activation and vascular inflammation.[26] These various mechanisms may result in arrythmias, systolic/diastolic dysfunctions, and ultimately HF. Due to the multifactorial cause, innovative methods are needed to treat obesity in the HF population.

Because the prevalence and severity of obesity have been constantly growing in the USA in the past years,[1] a concomitant rise in the number of patients with obesity and HF is expected. The positive impact of bariatric surgery on HF was described in the 1970s by Backman et al.[27] and has been supported by other recent articles.[10–13,15,16,18] A number of physiological mechanisms that positively influence cardiovascular function among patients who undergo bariatric surgery have been identified.[11,28,29] They include, among others, favourable changes in left atrial and ventricular mass and shape, abolition of high output state, and improvement of cardiovascular risk factors such as hypertension, diabetes, and hyperlipidaemia. Several recent studies advocated for concomitant bariatric surgery and LVAD placement as a bridge to cardiac transplantation for selected patients with obesity-related HF.[13–15,17,18] LSG, as a relatively straightforward procedure without intestinal anastomosis, has a safer long-term risk profile compared with other bariatric procedures such as Roux-en-Y gastric bypass or biliopancreatic diversion.[30–33] Therefore, it has emerged as the procedure of choice for patients who are at higher risk of developing severe complications as well as those who may need a heart transplant.[34,35] There have been reports of modest

improvement in LVEF after bariatric surgery in patients with HF, which in some cases obviated the need for cardiac transplantation.[10–12,15,16] There is, however, to the best of our knowledge, no previous report of LVEF normalization after bariatric surgery in patients with obesity and severe HF requiring LVAD therapy.

In conclusion, these two patients represent a subgroup of patients with severe HF and obesity, whose heart function can be significantly improved after bariatric surgery and LVAD support without the need for cardiac transplantation. These findings could potentially favour more aggressive use of bariatric surgery for these critically ill patients. Factors predicting potential HF reversibility and normalization of LVEF in patients with obesity-related severe HF are currently unknown, and further studies are needed to identify them.

Acknowledgements

Conflict of interest

None declared.

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