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REVIEW

Heart Failure

Targeting Obesity for Heart Failure

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Abstract

Obesity is a growing global health challenge and a significant contributor to the rising prevalence of heart failure (HF), affecting both HF with preserved and reduced ejection fraction. While obesity increases HF incidence through haemodynamic stress, neurohormonal activation, metabolic dysfunction and systemic inflammation, the so-called 'obesity paradox' complicates our understanding of prognosis in established HF. This review examines the complex bidirectional relationship between obesity and HF, summarising evidence for the effects of intentional weight loss on cardiac structure, function and clinical outcomes. Bariatric surgery provides consistent benefits across HF phenotypes, while dietary and pharmacological interventions, particularly glucagon-like peptide-1 (GLP-1) receptor agonists, show promise for cardiometabolic improvement. Emerging evidence also supports structured exercise and rehabilitation programmes. However, gaps remain regarding the long-term efficacy of interventions and the additive benefit of GLP-1 receptor agonists with sodium–glucose cotransporter-2 inhibitors. Understanding and targeting obesity in HF is essential for the improvement in morbidity and mortality in this high-risk population.

Keywords

Heart failure, obesity, weight loss

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Heart failure (HF) is a growing global health burden, affecting more than 56 million people worldwide, with significant morbidity, mortality and economic consequences.[1] While hypertension, diabetes and coronary artery disease have traditionally dominated the aetiological landscape of HF, the global rise in obesity, now affecting more than 880 million adults, has brought increasing attention to its role in the pathogenesis of HF.[2–4]

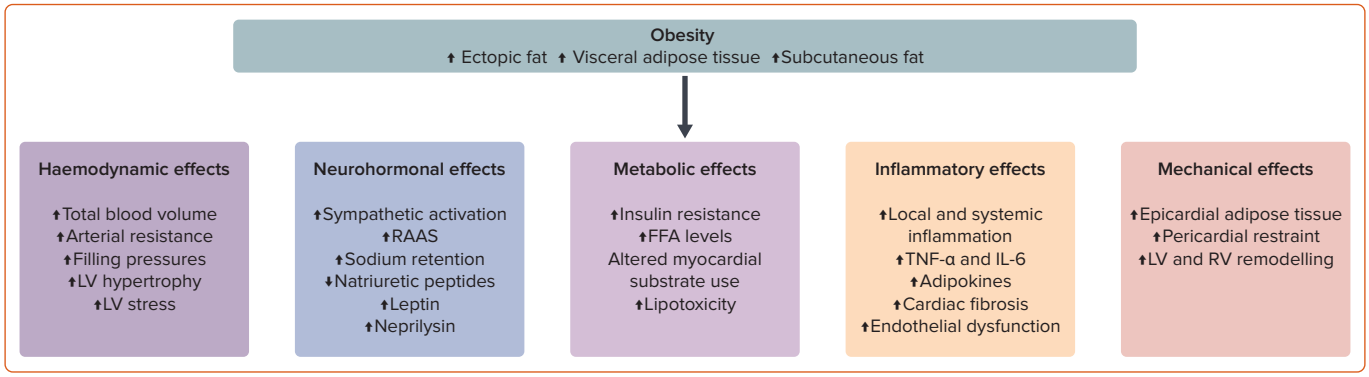
The link between obesity and HF has been recognised for over two decades, with epidemiological studies suggesting that for every unit increase in BMI above normal, the risk of developing HF increases by 5–7%.[5] This is particularly the case for HF with preserved ejection fraction (HFpEF), with 1 SD increase in BMI associated with a 1.34-fold increased hazard of future HFpEF and a 1.18-fold increased hazard of future HF with reduced ejection fraction (HFrEF).[6] Despite these associations, the relationship between obesity and HF remains complex, particularly given the obesity paradox: the counterintuitive observation that a higher BMI is associated with improved survival in patients with established HF.[7,8]

Due to concerns regarding the obesity paradox, the 2016 European Society of Cardiology (ESC) guidelines advised that weight loss could not be recommended for HF patients with moderate degrees of obesity (BMI <35 kg/m^2).[9] In more advanced obesity, the ESC advised that weight loss may be considered for symptomatic benefit and the 2019 Heart Failure Society of America guidelines recommend a weight reduction of 5–10% for patients with HF and a BMI ≥35 kg/m^2.[9,10]

Mechanistically, obesity contributes to both structural and functional cardiac changes. Increased adiposity is associated with left ventricular (LV) hypertrophy, increased stroke volume demand and altered myocardial metabolism, predisposing individuals to HF.[3] Additionally, obesity is a pro-inflammatory state, driving insulin resistance, endothelial dysfunction and neurohormonal activation – processes implicated in HF progression.[11] However, while obesity contributes to HF pathogenesis, its impact on disease trajectory and management remains a subject of ongoing debate.

Despite a logical expectation that intentional weight loss would induce reverse LV remodelling, evidence supporting improvements in cardiac geometry and function among obese patients with HFrEF remains limited. Currently, data are restricted to a small prospective dietary intervention study (n=14), which reported modest improvements in diastolic function and systolic fractional shortening, and two small retrospective echocardiographic studies that demonstrated LV ejection fraction (LVEF) improvements of 5–12% following bariatric surgery in patients with established systolic dysfunction.[12,13] While these findings are promising, no prospective randomised trials have evaluated the impact of intentional weight loss in obese individuals with HFrEF, leaving a significant gap in the evidence base for clinical guidance. Although systematic reviews of unintentional weight loss in HFrEF have been published, none address intentional weight loss.[14,15] Nevertheless, available evidence suggests that dietary weight loss may improve myocardial contractility and reduce demand for adenosine triphosphate (ATP). This suggests that enhanced metabolic efficiency could underlie functional cardiac recovery in this population.[16]

Figure 1: Simplified Pathophysiology Linking Obesity and Heart Failure



FFA = free fatty acid; IL = interleukin; LV = left ventricular; RAAS = renin–angiotensin–aldosterone system; RV = right ventricular; TNF = tumour necrosis factor.

These uncertainties underscore the need for a clearer understanding of how obesity influences HF pathophysiology, prognosis and management. This review, therefore, aims to:

- Examine the pathophysiological links between obesity and HF.
- Evaluate the obesity paradox and its implications for HF prognosis.
- Assess the effects of intentional weight loss on cardiac morphology and function.
- Explore emerging weight loss interventions, including lifestyle, pharmacologic and surgical approaches.

The article builds on recent reviews and the ESC Heart Failure Association position statement by emphasising emerging trial data and therapeutic approaches in both HFrEF and HFpEF.[3,17,18]

Pathophysiological Links Between Obesity and Heart Failure

Beyond its established association with HF incidence, obesity exerts diverse and complex effects on cardiovascular physiology (*Figure 1*).[19] Haemodynamically, obesity leads to a chronic volume-expanded state. Excess adiposity increases metabolic demand on the body, resulting in increased cardiac output and total blood volume. Through the traditional theory, when there is a hyperdynamic circulation, LV cavity dilatation and subsequent hypertrophy (LVH) occur secondary to increased wall stress.[20] While this mechanism explains the eccentric hypertrophic remodelling observed in obesity, concentric modelling has also been reported.[21] The latter pattern appears more commonly in the context of insulin resistance, diabetes, myocardial steatosis and greater levels of visceral adipose tissue.[11] It has been suggested that increased systemic vascular resistance in obesity may contribute to concentric remodelling.[22]

From a neuro-hormonal perspective, obese individuals exhibit heightened sympathetic nervous system activity and activation of the renin–angiotensin–aldosterone system (RAAS). This promotes myocardial fibrosis, vascular remodelling and fluid retention.[3,23,24] Elevated levels of the adipokine leptin, characteristic of diet-induced obesity, further stimulates RAAS.[25] Increased RAAS activity contributes to glomerular hyperfiltration and progressive nephron injury, underpinning the pathophysiological connection between obesity, HF and chronic kidney disease (CKD). Obesity can independently contribute to kidney function decline and has been associated with accelerated estimated glomerular filtration rate (eGFR) loss in HFpEF patients.[26] The coexistence of CKD in HF is also linked to increased mortality and HF hospitalisations. It is also hypothesised that obesity may predispose to greater haemodynamic-driven fluctuations in renal function, compounding risk.[23]

Importantly, obesity is a well-established risk factor for AF, with increasing adiposity linked to left atrial enlargement, AF and electrical remodelling.[24] Inflammatory signalling and epicardial fat infiltration are thought to contribute to the arrhythmogenic substrate that underlies obesity-related AF. AF may, in turn, exacerbate HF symptoms by impairing diastolic filling and reducing cardiac output, with AF burden linked to worse HF outcomes.[18,24]

Metabolically, obesity is characterised by systemic insulin resistance and altered myocardial substrate usage. In healthy individuals, the myocardium can flexibly switch between pyruvate and fatty acid oxidation to meet its energetic demands.[27] However, in obesity, there is a shift towards chronic fatty acid reliance. This impairs myocardial energy efficiency and leads to increased oxygen consumption per unit of ATP produced.[28] The resultant inflexibility contributes to a decline in cardiac energetics, evidenced by observed reductions in the phosphocreatine-to-ATP (PCr/ATP) ratio in obese individuals.[29] Lipotoxicity – the accumulation of ectopic lipid intermediates such as ceramides and diacylglycerols within cardiomyocytes – has been implicated in promoting cardiomyocyte apoptosis and cardiac dysfunction.[30]

Taken together, the bidirectional relationship between obesity and HF extends beyond epidemiological correlation. Through a combination of haemodynamic burden, metabolic dysregulation, neurohormonal activation and inflammation, obesity creates a permissive environment for the development and progression of HF.

Obesity is, however, disproportionately linked with HFpEF compared to HFrEF.[6] Up to 80% of HFpEF patients are overweight or obese and excess body weight confers a higher risk of developing HFpEF than HFrEF.[3,6] Several pathophysiological mechanisms explain why obesity favours HFpEF. Excess adipose tissue, particularly visceral and epicardial fat, releases pro-inflammatory cytokines and adipokines (including tumour necrosis factor-α and interleukin-6), producing a state of chronic low-grade systemic inflammation.[31] The resulting inflammatory environment impairs coronary microvascular endothelial function. This occurs through disrupted nitric oxide signalling and capillary rarefaction, leading to myocardial fibrosis and stiffness.[32] Epicardial adipose tissue may contribute further through local paracrine effects on the myocardium, exacerbating oxidative stress, fibrosis and diastolic dysfunction.[33] The overall phenotype is a hypertrophic, non-dilated left ventricle with preserved ejection fraction but elevated diastolic filling pressures and chamber stiffness, characteristic of HFpEF.[34] By contrast, obesity alone rarely induces the cardiomyocyte loss or eccentric remodelling typical of HFrEF, which more commonly arises from ischaemic damage or primary cardiomyopathies.[35]

The Obesity Paradox

Despite obesity being a significant risk factor for developing HF, a higher BMI has been associated with improved survival in patients with established HF. This paradox has been consistently demonstrated in meta-analyses of large HF cohorts. A meta-analysis of 28,209 HF patients found that both overweight and obese individuals had lower all-cause and cardiovascular mortality rates compared to those with normal or low BMI.[36] This association has been observed across different populations, in both acute and chronic HF settings and in patients with HFpEF and HFrEF.[8,37–39] A retrospective analysis of more than 200,000 acute HF hospitalisations in the US found that higher BMI groups experienced significantly reduced in-hospital mortality and length of hospitalisation.[40]

Several explanations have been proposed to account for the obesity paradox. One widely accepted hypothesis is that it arises due to survival bias and reverse causality. HF is a catabolic state that can lead to unintentional weight loss. Patients with more advanced disease often experience muscle wasting and cachexia, both of which are strongly linked to a poorer prognosis.[41] As a result, studies such as CHARM that do not differentiate between intentional and unintentional weight loss risk attributing a protective effect to obesity, when lower BMI may simply indicate a greater disease severity.[8] Observational studies may be influenced by selection bias. Individuals with obesity who survive to develop HF may represent a relatively healthier subgroup, while leaner HF patients are more likely to include those with frailty or other comorbid conditions that contribute to worse outcomes. It should also be recognised that while most studies adjust for age, HF patients with obesity are often younger than other patients.[42]

Another proposed explanation for the paradox is the metabolic reserve hypothesis, which suggests that excess adipose tissue provides an energy reserve that protects against the catabolic stress of HF. In this model, patients with greater fat stores may have a survival advantage in times of metabolic stress, reducing their risk of cardiac cachexia and associated complications.[43]

A major limitation of the obesity paradox concept is its reliance on BMI as the primary measure of adiposity. BMI does not differentiate between fat mass and lean muscle mass, nor does it account for fat distribution, which may be more relevant in determining HF prognosis. Several studies have explored alternative anthropometric measures, including waist circumference (WC) and waist-to-hip ratio (WHR), to assess the association between adiposity, mortality and HF outcomes.

A retrospective analysis of 209 ambulatory patients with chronic HF found that higher percentage body fat and total fat, measured using the skinfold technique, were associated with improved event-free survival from cardiovascular death and urgent transplantation.[44] Notably, this study focused predominantly on an HFrEF cohort. In contrast, a study of 3,310 HFpEF patients from the TOPCAT trial found that increased WC (≥102 cm in men and ≥88 cm in women) was associated with higher all-cause, cardiovascular and non-cardiovascular mortality.[45]

However, findings on the obesity paradox remain inconsistent. A smaller study of 344 HFrEF patients found that high WC or a combination of high WC and BMI was associated with improved survival, supporting the presence of an obesity paradox.[46] Conflicting with these results, a larger analysis of 8,399 HFrEF patients from the PARADIGM-HF trial found no obesity paradox when WHR was used instead of BMI. When adjustments were made in the analysis for N-terminal pro-B-type natriuretic peptide (NT-proBNP) and a broader range of prognostic variables, any survival advantage associated with higher BMI disappeared. Additionally, three alternative anthropometric indices (WHR, relative fat mass and body roundness index) demonstrated a significantly higher risk of HF hospitalisation and the composite outcome of HF hospitalisation or cardiovascular death in patients with greater adiposity. When BMI was used, this was less pronounced.[47]

A potential explanation for these conflicting findings is the differing impact of obesity in HFpEF and HFrEF. As mentioned above, obesity is more strongly associated with HFpEF, where metabolic dysfunction, inflammation and increased epicardial adipose tissue contribute to disease progression, potentially outweighing any protective effects and explaining the lack of an obesity paradox in studies such as TOPCAT. [11,45] Differences in statistical adjustments may partly explain the differing findings. Unlike other studies, Butt et al., in the PARADIGM-HF analysis, adjusted for NT-proBNP. Given that natriuretic peptide levels are inversely related to BMI and they serve as key markers of HF severity, failing to account for them may overestimate the protective effect of obesity.[47]

The obesity paradox in HF remains a complex and controversial phenomenon. While observational studies consistently report a survival advantage in obese HF patients, multiple confounders, including reverse causality, selection bias and inadequate adjustment for disease severity markers such as NT-proBNP, challenge its validity.

Weight Loss and Therapeutic Interventions for Obesity-related Heart Failure

Despite extensive research on the obesity paradox, given the above inconsistencies, uncertainty remains regarding the impact of intentional reduction in fat mass in obese patients.

Dietary Intervention

A 2019 meta-analysis of obese individuals without HF found that weight loss, achieved through either dietary interventions or bariatric surgery, led to favourable haemodynamic improvements, including reductions in mean arterial pressure, resting oxygen consumption and pulmonary capillary wedge pressure.[48] In a cohort of 170 overweight and obese patients, Haufe et al. reported that both reduced-fat and reduced-carbohydrate hypocaloric diets reduced LV mass, although no significant changes in LV systolic or diastolic function were observed.[49] In a separate study of 13 obese patients, a supervised weight loss programme led to reductions in LV mass and improvements in myocardial energetics and diastolic function.[50]

Among HF patients, non-surgical weight loss has demonstrated mixed results. Alpert et al. reported that weight loss in 24 morbidly obese HF patients was associated with reductions in LV mass and improved LV systolic and diastolic filling.[12] The SECRET trial showed that caloric restriction and aerobic exercise training in obese HFpEF patients independently improved exercise capacity, despite no significant change in quality of life.[51] More recently, a 15-week weight management programme improved 6-minute walking test (6MWT) performance and Minnesota Living with Heart Failure Questionnaire (MLHFQ) scores in 41 HFpEF patients, but did not significantly affect LVEF.[52] However, a similarly sized study of predominantly HFrEF patients did report a significant improvement in LVEF and New York Heart Association (NYHA) class with a mean body weight reduction of 4.4 kg. This was after a longer, 6-month dietary intervention.[53] Improvements in LVEF, NYHA classification and congestive index were higher when the cohort was

subdivided into 'non-responder' and 'responder' patients with weight loss of at least 3 kg.[53]

Despite promising findings, most studies evaluating weight loss in HF are limited by small sample sizes, short follow-up durations and heterogeneity in weight loss interventions. Larger, well-designed trials are needed to clarify the long-term impact of weight loss on cardiac function and outcomes in HF. Three randomised clinical trials (NCT05942287, NCT06455878 and NCT05878912) evaluating the role of structured dietary weight loss in HFrEF and HFpEF patients with obesity are currently under way.

Bariatric Surgery

Due to the often transient and variable success of dietary weight loss, bariatric surgery has emerged as a significant intervention for weight reduction, with strong evidence supporting its cardioprotective effects in both non-HF and HF populations. Large-scale studies have demonstrated that bariatric surgery is associated with a lower incidence of HF and beneficial cardiac remodelling in individuals without pre-existing HF. A meta-analysis of 39 studies found that bariatric surgery significantly reduced the risk of HF, MI and cardiovascular mortality.[54] Similarly, a Swedish nationwide study of 39,000 obese individuals found that gastric bypass surgery was associated with nearly a 50% lower incidence of HF compared to intensive lifestyle modification.[55]

Beyond HF prevention, bariatric surgery also induces favourable cardiac structural changes in a non-HF population. Sorimachi et al. showed that weight loss after bariatric surgery in 213 obese individuals led to reductions in visceral adipose tissue, epicardial adipose thickness and beneficial LV remodelling.[56] Additionally, a smaller 2008 study of 13 severely obese patients found that post-bariatric surgery, patients exhibited significant improvements in myocardial function, namely reduced left atrial dimensions, LV mass and LV end-diastolic volume.[57]

In patients with established HF, bariatric surgery is shown to improve symptoms, cardiac function and clinical outcomes. In a study of 12 patients with HFpEF, bariatric surgery led to significant improvements in MLHFQ scores, reversal of adverse LV remodelling and favourable lipidomic changes.[58] In an equivalently sized HFrEF cohort, bariatric surgery caused significant improvements in LVEF and NYHA class, alongside reducing hospital readmission rates.[13]

Larger cohort studies also support these findings. Vest et al. demonstrated that patients with LVEF <50% showed improvements in LVEF post-surgery.[59] Shimada et al. found that among 524 HF patients, those who underwent bariatric surgery had significantly lower rates of emergency department visits or HF hospitalisations for 13–24 months post-surgery.[60] Further, a retrospective analysis of 2,810 patients admitted with HF reported that previous bariatric surgery was associated with an almost 50% reduction in in-hospital mortality and a shorter length of stay.[61]

For obese HF patients, pharmacological and exercise-based interventions are being increasingly investigated as alternatives or complementary strategies to bariatric surgery and dietary restriction.

GLP-1 Receptor Agonists

GLP-1 receptor agonists (GLP1RA) have emerged as a promising therapeutic option for weight loss and metabolic regulation, with growing evidence supporting their benefits in HF populations. These agents facilitate weight loss by suppressing appetite, slowing gastric emptying and enhancing insulin sensitivity. Beyond metabolic effects, GLP1RA have demonstrated cardioprotective properties, including reduced systemic inflammation, improved endothelial function and favourable LV unloading.[62]

The STEP-HFpEF trial evaluated semaglutide in 529 patients with HFpEF and a BMI ≥30 kg/m^2. After 52 weeks of treatment, semaglutide resulted in a 10.7% body weight reduction compared to placebo. The intervention group had significant improvements in Kansas City Cardiomyopathy Questionnaire (KCCQ) scores, increased 6MWT distances and a reduced composite endpoint of HF hospitalisations and urgent HF visits.[63]

The landmark SELECT trial subsequently investigated semaglutide in 17,604 patients with pre-existing cardiovascular disease and a BMI ≥27 kg/m^2, without a history of diabetes. Semaglutide resulted in an 8.5% placebo-adjusted body weight loss and reduced the risk of HF-related events (HR 0.82; 95% CI [0.71–0.96]).[64] Notably, semaglutide benefited both HFpEF and HFrEF patients, with no significant heterogeneity in outcomes between these groups.[65] In SELECT, early separation in event rates between semaglutide and placebo emerged by 3–6 months, before most participants had reached target dose or achieved substantial weight loss. This suggests that the benefits of semaglutide may not be solely weight-mediated, but could reflect early improvements in inflammation, blood pressure, glycaemic control or direct cardiometabolic effects. However, the trial raised concerns about the drug's long-term tolerability due to a high discontinuation rate due to gastrointestinal side-effects, particularly in HF patients (14.7%).[64] Although, in a 2025 meta-analysis of 922 patients with or without diabetes, once-weekly semaglutide therapy had comparable adverse gastrointestinal effects to once-daily liraglutide, but a greater reduction in body weight and HbA_{1c}.[66]

Tirzepatide, a dual GLP-1 and glucose-dependent insulinotropic polypeptide receptor agonist, was recently evaluated in the SUMMIT trial. Among 731 obese patients with HFpEF, tirzepatide treatment significantly reduced the risk of cardiovascular death or a worsening HF event, while also improving KCCQ scores. A secondary analysis offered mechanistic insights, demonstrating reductions in estimated blood volume (−0.58 l), systolic blood pressure, troponin T, and NT-proBNP levels, alongside improved eGFR.[67] Cardiac MRI also revealed decreases in LV mass and paracardiac adipose tissue, supporting potential direct cardiovascular and renal benefits beyond weight loss.[68]

By contrast, trials of liraglutide and albiglutide in a HFrEF population yielded smaller weight reductions (typically 2–4 kg), with no detectable impact on cardiac function, myocardial glucose use or meaningful change in 6MWT.[69,70] It is important to note that all three trials did not specify obesity as an inclusion criterion. It is therefore possible that it is the weight loss conferred by GLP1RA, rather than the agents themselves, which confers cardiac and HF benefit.

Given these combined findings, GLP1RAs are likely to become a key component in managing obesity-related HF. However, despite their recommendation for cardiovascular risk reduction in patients with obesity, GLP1RAs have not yet been endorsed by ESC, the National Institute of Health and Clinical Excellence (NICE) or American College of Cardiology (ACC) guidelines specifically for HF treatment.[71] Concerns have also been raised about the loss of lean body mass with GLP1RA, particularly relevant in HF populations where sarcopenic obesity is being increasingly recognised.[72]

Exercise Training and Cardiac Rehabilitation

Exercise training is a cornerstone of HF management, improving exercise capacity, symptom burden and quality of life. Regular physical activity has been associated with improved peak oxygen consumption (VO_2 max), enhanced endothelial function and reduced HF-related hospitalisations.[73]

The SECRET trial demonstrated that caloric restriction and aerobic exercise independently improved exercise capacity in obese patients with HFpEF. However, there was no significant change in quality of life.[51] A 2024 meta-analysis by Lee et al. found that lifestyle interventions led to an average weight reduction of 5.3 kg, improved 6MWT performance and better NYHA classification in HFpEF patients.[74] Similarly, Fukuta et al. found that exercise training in HFpEF improved exercise tolerance and quality of life (QoL), despite no significant changes in LV systolic or diastolic function.[75]

In HFrEF, the HF-ACTION trial demonstrated that supervised aerobic training improved exercise capacity and led to a 13% risk reduction for cardiovascular mortality or hospitalisation among patients with good adherence.[76] Resistance training may provide additional benefit when integrated with aerobic and dietary strategies, as suggested by the SECRET-II trial.[77]

Despite these findings, uptake of exercise therapy remains low. Barriers include frailty, high symptom burden, fear of exacerbating symptoms and limited access to structured programmes. The REHAB-HF trial demonstrated that tailored rehabilitation in older, frail patients hospitalised for HF (including obese participants) improved global physical function, although not short-term rehospitalisation.[78] These findings nevertheless support the use of supervised, individualised rehabilitation to overcome deconditioning and functional decline for HF patients.

Future Directions

Obesity plays a multifaceted and increasingly recognised role in the pathogenesis and clinical course of HF. Through mechanisms spanning haemodynamic overload, metabolic dysregulation, neurohormonal activation and chronic inflammation, excess adiposity contributes to both HFpEF and HFrEF phenotypes. While the obesity paradox has prompted debate regarding the prognostic impact of higher BMI in established HF, evidence increasingly supports the clinical benefits of intentional weight loss in selected patients.

Surgical weight loss, particularly through bariatric procedures, has shown robust and consistent cardioprotective effects in both HF and non-HF populations. Dietary and lifestyle interventions offer benefits, although trial findings are more heterogeneous. Pharmacological agents, including GLP1RA, have emerged as promising tools in addressing the metabolic and systemic consequences of obesity in HF. Trials such as STEP-HFpEF, SELECT and SUMMIT have demonstrated that semaglutide and tirzepatide can produce meaningful improvements in weight, symptoms and cardiovascular outcomes in HFpEF patients with obesity. It is worth noting that to date, however, no randomised controlled trial has compared the cardiac benefit of bariatric surgery to GLP1RA or dietary intervention in HF patients.

Exercise training and cardiac rehabilitation continue to be underused despite substantial evidence for improved physical function, quality of life and hospitalisation outcomes in HF patients, including those with obesity. Addressing barriers to implementation, such as frailty, deconditioning and access, will be critical to realising the full potential of these interventions.

Further work is required to define how best to sequence and combine available therapies. Results from randomised trials investigating intentional fat loss in both HFrEF and HFpEF are awaited and longer-term data are needed to assess the sustainability of benefits from newer pharmacological agents. It also remains uncertain whether combined use of GLP1RA and sodium-glucose cotransporter-2 (SGLT2) inhibitors confers additive or synergistic cardiovascular benefit in HF, although a recent meta-analysis suggested combination therapy may reduce HF hospitalisations more effectively than monotherapy, alongside greater weight loss.[79]

Moreover, efforts to personalise weight loss strategies based on HF phenotype, adiposity distribution and metabolic profile will be essential. Biomarker profiling and advanced measures of body composition, such as MRI, CT and dual-energy X-ray absorptiometry, may better guide therapy than BMI or anthropometric indices alone, although clinical uptake is limited by cost, accessibility and standardisation challenges.[80]

Future clinical trials should prioritise diversity in HF subtype, sex and ethnicity to address existing disparities in obesity-related HF research and care. While mortality and hospitalisation are critical endpoints used in HF trials, patient-reported outcome measures are equally important and less prevalent in the literature. Improvements in symptoms, function and quality of life may represent meaningful benefits to patients, particularly when increased longevity is not guaranteed.

Conclusion

Ultimately, the increasing burden of obesity-linked HF underscores the need for integrated, multifaceted treatment strategies to address the underlying pathophysiological drivers. As evidence accumulates, intentional weight loss, whether achieved through medical, surgical or lifestyle means, should be viewed not simply as a supportive strategy, but as a potential disease-modifying approach in HF management. ❑

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