# Addressing Comorbidities in Heart Failure

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**KEYWORDS**

• Heart failure • Comorbidities • Management • Hypertension • Diabetes
• Atrial fibrillation • Chronic kidney disease • Anemia

**KEY POINTS**

- Comorbidities in heart failure (HF) are almost universal with 85% or greater of patients with HF having two or more chronic conditions.
- HF comorbidities complicate management and are associated with worse outcomes, impacting mortality, hospitalization, and quality of life.
- Hypertension is the most common HF comorbidity and serves as an opportunity to optimize guideline-directed medical therapy.
- HF management should be tailored to the individual patient to address comorbidities and improve outcomes.

## INTRODUCTION

There are approximately 6.7 million Americans with heart failure (HF), which is expected to rise to 8.7 million by 2030.[1] As the population with HF grows, comorbidities are increasingly common. Multimorbidity is extremely widespread with 85% or greater of patients with HF having two or more chronic conditions and greater than 50% with 5 or more, which has increased over the past two decades.[2,3] Additionally, the proportion of patients with HF with preserved ejection fraction (HFpEF) is increasing; patients with HFpEF on average have one additional comorbidity than those with HF with reduced ejection fraction (HFrEF) (4.5 vs 3.7 chronic conditions, respectively).[2]

Both cardiac and noncardiac comorbidities are common in HF. Among HF Medicare beneficiaries, the two most common are hypertension and coronary artery disease (CAD). Additional frequent comorbidities include hyperlipidemia, anemia, diabetes,

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| Abbreviations | |
|---|---|
| ACE | angiotensin-converting enzyme |
| AF | atrial fibrillation |
| ARB | angiotensin-receptor blocker |
| ARNI | receptor–neprilysin inhibitor |
| ASV | adaptive servo-ventilation |
| BP | blood pressure |
| CABG | coronary artery bypass grafting |
| CAD | coronary artery disease |
| CCB | calcium channel blocker |
| CKD | chronic kidney disease |
| CPAP | continuous positive airway pressure |
| CRT | cardiac resynchronization therapy |
| CSA | central sleep apnea |
| DOAC | Direct oral anticoagulant |
| eGFR | estimated glomerular filtration rate |
| GDMT | guideline-directed medical therapy |
| GLP-1RA | glucagon-like peptide-1 receptor agonists |
| HF | heart failure |
| HFpEF | HF with preserved ejection fraction |
| HFrEF | HF with reduced ejection fraction |
| IV | intravenous |
| LVEF | left ventricular ejection fraction |
| MRA | mineralocorticoid receptor antagonist |
| OSA | obstructive sleep apnea |
| PCI | percutaneous coronary intervention |
| QoL | quality of life |
| RAAS | renin–angiotensin–aldosterone system |
| RV | right ventricle |
| SDB | sleep disordered breathing |
| SGLT2i | sodium–glucose cotransporter-2 inhibitors |

arthritis, chronic kidney disease (CKD), atrial fibrillation (AF), chronic obstructive pulmonary disease (COPD)/asthma, dementia, sleep apnea, and depression.[4]

Chronic conditions complicate HF management, resulting in increased prescriptions and adverse drug effects, declining functional status, and increased health care utilization.[2] Additionally, HF comorbidities negatively affect mortality, hospitalization, and quality of life (QoL).[5,6] Therefore, an understanding of the management of common comorbidities is needed to provide optimal care for patients with HF and prevent adverse outcomes. Selected comorbidities that significantly impact HF management are discussed in this review (**Fig. 1**).

## MANAGEMENT PRINCIPLES OF HEART FAILURE COMORBIDITIES

### *Hypertension*

Hypertension is the most common comorbid condition in HF, present in over 80% of patients.[4] Hypertension and HF are closely linked, with hypertension preceding HF in over 90% of Framingham Heart Study patients and independently accounting for greater than 40% of the risk of HF in men and 60% in women.[7]

Sustained pressure overload from hypertension leads to concentric left ventricular hypertrophy and diastolic dysfunction. Left uncontrolled, diastolic dysfunction can progress to HFpEF, and even further to left ventricular dilation and HFrEF.[8] To prevent cardiac dysfunction, stringent blood pressure (BP) control is recommended; the SPRINT trial demonstrated a lower risk of HF with systolic BP less than 120 mm Hg

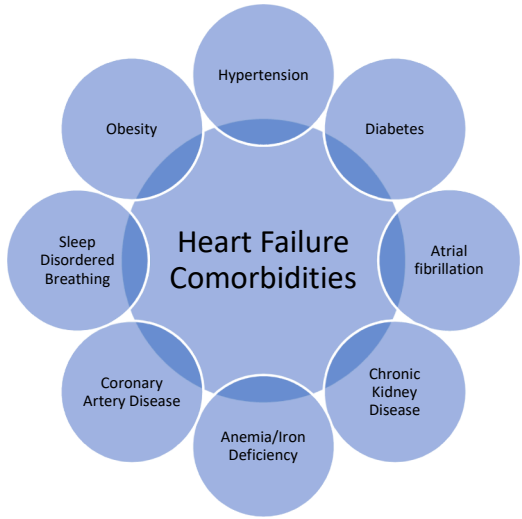


**Fig. 1.** Common HF comorbidities which impact management.

compared to 140 mm Hg.[9] Guidelines recommend a BP goal of less than 130/80 mm Hg for most patients without preexisting HF.[10]

Despite the widespread nature of hypertension, the optimal goal BP in HF is unknown as clinical trials are lacking.[4] There is a J-shaped relationship between systolic BP and mortality in patients with HF. Both high and low BP are associated with increased mortality, although it is unclear whether low BP is harmful or a marker of poor health.[8] In general, guidelines recommend titrating guideline-directed medical therapy (GDMT) to a goal BP of less than 130/80[4,10] (**Table 1**).

For patients at risk for HF, thiazide-diuretics are associated with a lower risk of developing HF than calcium channel blockers (CCB) and angiotensin-converting enzyme (ACE) inhibitors.[11,12] Alpha-blockers are associated with double the risk of HF than thiazide-diuretics.[13] In patients with HFrEF, there are no randomized trials

**Table 1**
**Management of blood pressure in HFrEF versus HFpEF**

| HFrEF | HFpEF |
|---|---|
| Goal Blood Pressure < 130/80 (optimal goal unknown, general goal for most HF patients) | |
| *GDMT Optimization*:<br>ACEi/ARB/ARNI<br>Beta-blockers[a]<br>MRA | *First line:*<br>ARB<br>Beta-blockers<br>MRA<br>Diuretic |
| Avoid Non-dihydropyridine CCBs | *Limited data*:<br>Alpha-blockers<br>CCBs<br>Avoid Nitrates |
| *Lifestyle Modifications:*<br>Sodium restriction, weight reduction, exercise, moderation of alcohol intake, and a heart-healthy diet | |

[a] Evidence-based beta blockers for HFrEF include metoprolol succinate, carvedilol, and bisoprolol.

to determine which medications are best, but optimizing GDMT is the key in management, using ACE inhibitors, angiotensin-receptor blockers (ARBs), angiotensin receptor–neprilysin inhibitors (ARNIs), mineralocorticoid receptor antagonists (MRAs), and evidence-based beta-blockers.[10] Hydralazine/isosorbide dinitrate can be added in black patients with class III or IV HF who remain hypertensive.[14] Nondihydropyridine CCB, such as diltiazem and verapamil, should be avoided in patients with HFrEF, as they result in negative inotropy and worse outcomes.[10,14] In HFpEF patients with hypertension and volume overload, diuretics should be used.[10] Nitrates are associated with a signal of harm in HFpEF.[10]

In addition to medical therapy, lifestyle modifications are necessary for optimal hypertension treatment. Sodium restriction, weight reduction, exercise, moderation of alcohol, and a heart-healthy diet are recommended.[14]

### *Diabetes Mellitus*

Diabetes is highly prevalent among patients with HF, ranging from 20% to 40% in various studies, and more common in HFpEF. Diabetes in HF also carries an increased risk of mortality and hospitalization.[15] Diabetic cardiomyopathy refers to ventricular hypertrophy and dysfunction in patients with diabetes without other risk factors/causes. It is characterized by cardiac hypertrophy, fibrosis, and impaired microvascular perfusion. Insulin resistance is at the root of these changes, contributing to increased myocardial fatty acid oxidation, lipotoxicity, impaired mitochondrial function, inflammation, and upregulation of the renin–angiotensin–aldosterone system (RAAS).[16]

Guidelines recommend the use of sodium–glucose cotransporter-2 inhibitors (SGLT2i) in patients with HF and type 2 diabetes.[4] Four key trials regarding SGLT2i in HF are summarized in **Table 2**. All demonstrated a decrease in the primary outcome in the SGLT2i group, consistent across patients with both diabetes and nondiabetes.[17–20]

There is increasing evidence for glucagon-like peptide-1 receptor agonists (GLP-1RAs) in HFpEF, but less so for patients with both HF and diabetes. About 10% to 20% of the patients enrolled in diabetes trials with GLP-1RAs had HF, but their HF was poorly characterized.[21] Recently, the STEP-HFpEF trial demonstrated reduced HF symptoms in patients with HFpEF, obesity, and diabetes with semaglutide compared to placebo.[22]

Other antihyperglycemics have fewer benefits in HF. Metformin is safe, but there is not strong evidence for beneficial HF effects. Thiazolidinediones are contraindicated due to increased HF risk. DPP-4 inhibitors have not demonstrated benefit and saxagliptin may increase HF symptoms and hospitalizations. Insulin is considered neutral.[16]

### *Atrial Fibrillation*

There is a complex interplay between AF and HF, as HF increases AF risk and AF worsens HF. They often coexist, with AF occurring in more than a half of individuals with HF and a third of patients with AF developing HF.[23] AF is more common in patients with HFpEF, but patients with HFrEF have higher mortality with coexisting AF.[23]

The pathophysiology of both AF and HF are intricately linked. Both conditions result in neurohormonal activation.[24] AF can worsen HF by decreasing cardiac output through loss of the atrial kick, irregular and rapid ventricular rate, and tachycardia-induced cardiomyopathy. In turn, HF can trigger AF through left atrial enlargement, increased left atrial pressure, and functional mitral regurgitation.[24]

**Table 2**
**Results from major sodium–glucose cotransporter-2 inhibitor in heart failure trials**

| Trial (Year) | Population | Intervention | Primary Outcome | Follow-up | Result |
|---|---|---|---|---|---|
| DAPA-HF[17] (2019) | LVEF ≤40% | Dapagliflozin 10 mg daily | Composite of worsening HF (hospitalization or an urgent visit resulting in IV therapy for HF) or cardiovascular death | 18.2 mo | Reduction in primary outcome in dapagliflozin group (HR 0.74; 95% CI, 0.65–0.85; *P*<.001) |
| EMPEROR-Reduced[18] (2020) | LVEF ≤40% | Empagliflozin 10 mg daily | Composite of cardiovascular death or hospitalization for HF | 16 mo | Reduction in primary outcome in empagliflozin group (HR 0.75; 95% CI, 0.65–0.86; *P*<.001) |
| DELIVER[19] (2022) | LVEF >40% | Dapagliflozin 10 mg daily | Composite of worsening HF (unplanned hospitalization or urgent visit for HF) or cardiovascular death | 2.3 y | Reduction in primary outcome in dapagliflozin group (HR 0.82; 95% CI, 0.73–0.92; *P*<.001) |
| EMPEROR- Preserved[20] (2021) | LVEF >40% | Empagliflozin 10 mg daily | Composite of cardiovascular death or hospitalization for HF | 26.2 mo | Reduction in primary outcome in empagliflozin group (HR 0.79; 95% confidence interval [CI], 0.73–0.92; *P*<.001) |

Management of AF in patients with HF is complex and should be tailored to each patient, with an emphasis on anticoagulation to prevent thromboembolism and catheter ablation to restore sinus rhythm in patients with symptomatic HFrEF. HF is a hypercoagulable state and an independent risk factor for stroke, with patients with lower ejection fractions having higher thromboembolic risk.[25] HF guidelines suggest patients with HF and AF with a CHA2DS2-VASc score 2 or greater in men and 3 or greater in women should be anticoagulated.[4] Direct oral anticoagulants (DOACs) are preferred over warfarin in eligible patients.[4]

Rapid ventricular rates (>100 bpm) in AF are associated with increased mortality in patients with HFrEF, and beta-blocker use for rate control improved outcomes.[26] A heart rate goal less than 110 bpm is recommended for patients with HF, like non-HF patients with AF.[27] Nondihydropyridine CCBs should be avoided for rate control as they can cause negative inotropy. For rhythm control in patients with HF, class III antiarrhythmics can be used, with careful monitoring for QT prolongation.[28] Class IC antiarrhythmics should be avoided in patients with HF. Studies have not shown a benefit of rate versus rhythm control with medications in HF.[4,29]

However, rhythm control with catheter ablation has been shown to be beneficial in HF. The CASTLE-AF trial showed lower rates of death and HF hospitalization in patients with LVEF less than 35% and NYHA class II–IV symptoms randomized to ablation versus standard medical therapy.[30] Ablation is also more likely to maintain sinus rhythm than antiarrhythmic medications in patients with HF.[31] Therefore, guidelines recommend ablation for symptomatic HFrEF patients with AF to improve symptoms, QoL, ventricular function, and outcomes.[4,32]

For HF patients with LVEF $\leq$50% in whom rhythm strategies fail and ventricular rates remain uncontrolled, atrioventricular nodal ablation and cardiac resynchronization therapy (CRT) implantation should be considered for improvement in outcomes and symptoms.[4,33] There is no benefit of RV pacing over CRT in patients with EF greater than 50%.[4]

### *Coronary Artery Disease*

CAD is the most common cause of HF in the United States and is implicated in both HFrEF and HFpEF.[34] Medical management includes statins, antiplatelets, as well as ACE/ARB and beta-blockers.[4,34]

For patients with CAD, coronary artery bypass grafting (CABG) has been shown to be beneficial in patients with HFrEF. The STICH trial compared medical management to CABG plus medical therapy in HFrEF patients with CAD amenable to revascularization. Although there was no significant decrease in mortality in the CABG group over 56 months, at 10 year follow-up, there was a significant reduction in mortality with CABG.[35,36] Surgical revascularization now has a class I recommendation in the AHA/ACC/HFSA HF guidelines in select patients with LVEF 35% or lesser with suitable coronary anatomy.[4]

Percutaneous coronary intervention (PCI), although less invasive, has less compelling data in HF. REVIVED-BCIS2 was the first randomized trial to evaluate PCI in ischemic HF; however, PCI plus medical therapy failed to show a reduction in mortality or HF hospitalizations compared to medical therapy alone.[37] Observational data have been conflicting, with some studies showing neutral effects and others suggesting benefit or even harm.[38] Further data are needed to evaluate PCI in HF, especially in the era of improved PCI techniques allowing complete revascularization.

### *Chronic Kidney Disease*

Heart disease is the leading cause of death in patients with CKD. There is increased risk of HF, especially HFpEF, in patients with CKD.[39] It is unknown whether CKD

drives HF, HF drives CKD, or they share common pathophysiology. Changes caused by CKD (including volume/pressure overload, RAAS activation, oxidative stress, anemia, and the effects of HD) subsequently exacerbate HF. Simultaneously, hemodynamic, neurohormonal, and inflammatory changes in HF promote CKD.[40,41]

Diagnosing HF in patients with CKD is challenging given symptom overlap and difficulty interpreting objective data. For example, brain natriuretic peptide and troponin levels can be unreliable with impaired renal clearance, although elevations in either or both still correlate with HF in CKD.[40]

Patients with CKD and an eGFR less than 30 mL/min/1.73 $m^2$ are typically excluded from clinical trials, so there are limited data on GDMT in patients with advanced CKD.[41] Overactivation of the mineralocorticoid receptor has been implicated in cardiorenal disease, making MRAs an important therapy in HF and CKD.[42] The landmark trials for MRAs included patients with CKD stages 1 to 3 but not advanced CKD.[43] Data suggest that HF benefits of spironolactone remain in patients with CKD, but the risk of hyperkalemia and worsening renal function increases.[44] Less is known about MRA use with advanced CKD and dialysis patients.[43] SGLT2i decrease glomerular hyperfiltration and can be used with eGFR as $\geq$20 mL/min/1.73 $m^2$. A meta-analysis demonstrated that allocation to a SGLT2i reduced the risk of CKD progression by 37%.[45] **Table 3** summarizes evidence for GDMT in patients with CKD.

### *Anemia and Iron Deficiency*

Anemia, defined as hemoglobin less than 13.0 g/dL in men and less than 12.0 g/dL in women, is present in approximately 30% of stable and 50% of hospitalized patients with HF, compared to less than 10% in the general population.[46] Anemia in HF is associated with increased mortality, hospitalization, and worsening symptoms/functional status.[47] Approximately 30% of anemia in HF is due to iron deficiency.[48] Iron deficiency in HF is defined as ferritin less than 100 μg/L or if ferritin is between 100 and 299 μg/L, a transferrin saturation of less than 20%.[46] Independent of anemia, iron deficiency is associated with reduced exercise tolerance, QoL, increased hospitalization, and increased mortality in patients with HF.[46]

The pathogenesis of anemia and iron deficiency includes impaired erythropoietin synthesis, malabsorption due to gut edema, inflammation leading to altered iron metabolism, fluid retention/hemodilution, and frequent use of anticoagulants/antiplatelets leading to blood loss.[46,49] Both anemia and iron deficiency cause reduced oxygen delivery to metabolizing tissues, which triggers neurohormonal, hemodynamic, and renal blood flow alterations. Left ventricular remodeling and hypertrophy can result.[46]

**Table 3**
**Guideline-directed medical therapy use in chronic kidney disease**

| Class | Recommendations |
|---|---|
| Beta-blockers | There is no significant interaction between CKD status and mortality effect of beta-blockers, suggesting that the effect is similar for patients with HF and CKD compared to patients with HF alone.[69] |
| ACE/ARB/ARNI | Effective in patients with HFrEF and CKD 1–3, but limited data for CKD 4–5.[43] |
| MRAs | MRAs should be limited to patients with creatinine <2.5 mg/dL (or eGFR >30) and normal potassium with close monitoring.[4,41] |
| SGLT2i | Beneficial for both renal and cardiovascular outcomes and can be used in patients with eGFR as low as 20 mL/min/1.73 $m^2$ and continued until initiation of dialysis.[43] |

Given the relationship between anemia/iron deficiency and worsening HF, all patients with HF should have a baseline laboratory assessment screening for anemia/iron deficiency.[4] Several studies have shown improved outcomes with intravenous (IV) iron replacement in patients with HF who are iron deficient, even independent of anemia. FAIR-HF showed improvement in NYHA class, 6 min walk test, and QoL in NYHA class II–III HF patients with EF 40% or lesser receiving IV iron compared to placebo.[50] Findings were similar in CONFIRM-HF, which followed patients over a longer duration (52 weeks compared to 24 weeks).[51] AFFIRM-AHF and HEART-FID both showed reduction in HF hospitalizations with IV iron compared to placebo.[51,52] Despite these improvements, no studies have shown mortality benefit with IV iron. Oral iron was not shown to improve exercise capacity compared to placebo in patients with HF in the IRON-OUT study, which has been attributed to poor oral absorption of iron in HF.[4,53]

There has been interest in erythropoietin-stimulating agents given impaired erythropoietin production in HF; however, studies have shown harm.[54] RED-HF investigated darbepoetin alfa in HFrEF. There was no difference in hospitalization or mortality but higher rates of thromboembolic events and stroke.[54] Meta-analyses have confirmed these findings and erythropoietin-stimulating agents have a class III (harm) recommendation in the AHA/ACC/HFSA HF guidelines.[4,55]

### *Sleep Disordered Breathing*

Sleep disordered breathing (SDB) can be difficult to diagnose in patients with HF, as symptoms such as orthopnea, daytime fatigue, and insomnia overlap between conditions. Obstructive sleep apnea (OSA) is characterized by collapse of the pharynx and central sleep apnea (CSA) by decrease in respiratory drive during sleep. SDB has a high prevalence in patients with HF with one study showing a prevalence of 76% (40% CSA, 36% OSA).[56] In acute HF decompensation prevalence of SDB worsens, especially CSA and associated Cheyne-Stokes respirations.[57] SDB is associated with worse outcomes in patients with HF, with increased mortality in both untreated OSA and CSA.[58] CSA is an independent risk factor for cardiac rehospitalization.[59]

Treatment of SDB varies based on subtype and sleep studies can inform therapeutic strategies.[4] Continuous positive airway pressure (CPAP) therapy has shown benefit in HF patients with SDB. The CANPAP study investigating CPAP in HF patients with CSA showed improvement in ejection fraction, sleep quality, 6 min walk distance, and nocturnal oxygenation, but no improvement in mortality.[60] A meta-analysis showed no improvement in hospitalization or mortality with CPAP but QoL improvements, suggesting CPAP may be considered in selected patients with HF for QoL improvement.[61]

Adaptive servo-ventilation (ASV) is a noninvasive therapy for CSA which delivers servo-controlled inspiratory pressure support on top of expiratory positive pressure.[62] SERVE-HF studied ASV compared to placebo in HF patients with EF 45% or lesser showing increased mortality. This is possibly due to dampening Cheyne-Stokes respirations, which may be a compensatory mechanism in patients with HF, and alterations in hemodynamics.[62] ASV now has a class III (harm) recommendation by the AHA/ACC/HFSA for use in patients with HF.[4]

### *Obesity*

Obesity has a prevalence of 51% and 35% in patients with HFpEF and HFrEF, respectively, according to a retrospective study.[63] HF can be challenging to diagnose in obesity due symptom overlap and difficulty interpreting physical examination and

**Table 4**
**Key management strategies for HF comorbidities**

| Comorbidity | Key Management Strategies |
|---|---|
| Hypertension | Optimal BP goal and medications in HF are unknown given lack of clinical trials. For most patients, goal BP is <130/80. In HFrEF, GDMT should be uptitrated to obtain BP goal. Nondihydropyridine CCBs should be avoided. In HFpEF, diuretics should be first-line, in addition to ACE inhibitors/ARBs/MRAs/beta-blockers. |
| Diabetes | SGLT2i should be used first-line in HF patients with hyperglycemia. |
| Atrial Fibrillation | HF patients with AF and CHA2DS2-VASc score $\geq$2 in men and $\geq$3 in women should be anticoagulated with a DOAC if eligible. There is no benefit to rate vs rhythm control. Catheter ablation should be considered for patients with symptomatic HFrEF. If other strategies fail, consider atrioventricular node ablation with CRT implantation. |
| Coronary Artery Disease | CAD is the most common cause of HF. CABG should be considered in HFrEF patients with suitable anatomy for revascularization. Data for PCI are less clear. |
| Chronic Kidney Disease | MRAs and ACE/ARB/ARNI can be carefully used in CKD 1–3; less is known about use in CKD 4–5. SGLT2i can be used with GFR $\geq$20 mL/min/1.73 $m^2$ and has beneficial renal/HF outcomes. No renal adjustments are needed for beta-blockers. |
| Anemia/Iron Deficiency | IV iron improves QoL and decreases hospitalizations but not mortality in HF patients with iron deficiency, even independent of anemia. Oral iron has not been shown to be beneficial. Erythropoietin-stimulating agents should be avoided given increased risk of thrombosis. |
| Sleep Disordered Breathing | For HF patients and sleep apnea, CPAP can improve QoL. ASV is associated with harm in HF patients with CSA and should not be used. |
| Obesity | Diet and exercise are recommended, with consideration of bariatric surgery in select patients. There is increasing evidence of GLP-1RA use in HFpEF, but they may cause harm in HFrEF. |

echocardiography.[64] Additionally, the sensitivity of brain natriuretic peptide is reduced, as levels are lower in obesity.[4]

Obesity itself implicated in HF in addition to the role it plays in causing subsequent risk factors for HF. Expansion of adipose tissue causes inflammation, oxidative stress, adipokine release, and apoptosis, which leads to microvascular endothelial dysfunction, cardiac fibrosis, and structural myocardial changes.[65] However, obesity has been associated with improved HF survival, termed the "obesity paradox". Nevertheless, weight loss has many potential benefits, including improvement in comorbid conditions, reduction in insulin resistance and systemic inflammation, and ability to access advanced therapies such as transplantation.[64]

An individualized nutrition plan, graded exercise, and specialist consultation for patients with HF and obesity are advised. Bariatric surgery is recommended for selected patients.[64] Data on pharmacologic agents for obesity are limited, although there is increasing evidence for GLP-1RAs in HFpEF. The SUMMIT trial comparing tirzepatide to placebo in patients with HFpEF and obesity demonstrated a decrease in death from cardiovascular causes or a worsening HF event with tirzepatide.[66] In a similar population, the STEP-HFpEF trial showed improvement in symptoms and physical limitations

with semaglutide.[67] Pooled analysis of the SELECT, FLOW, STEP-HFpEF, and STEP-HFpEF DM trials demonstrated a reduction of combined endpoints of cardiovascular death or worsening heart failure and worsening heart failure alone with semaglutide in patients with HFpEF.[68] In HFrEF, however, trials suggest a tendency toward increased risk of HF hospitalizations and arrythmias.[21,65]

## SUMMARY

Comorbidities in patients with HF are increasingly common, especially as the population with HF grows and ages. Both cardiac and noncardiac comorbidities affect management and can impact mortality, hospitalization, and QoL in patients with HF. Therefore, clinicians should understand how to address common comorbidities to optimally care for patients with HF and prevent adverse outcomes (**Table 4**).

## CLINICS CARE POINTS

- Comorbidities in HF are almost universal with 85% or greater of patients with HF having two or more chronic conditions.
- HF comorbidities complicate management and affect mortality, hospitalization, and QoL.
- Hypertension is the most common HF comorbidity and serves as an opportunity to optimize GDMT.
- HF management should be tailored to the individual patient to address comorbidities. Key management strategies are detailed in **Table 4**.

## DISCLOSURES

None.

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