

Managing patients with heart failure: A contemporary practice tool for pharmacists

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Introduction

Over the past decade, new pharmacologic agents have been shown to reduce mortality and hospitalizations and improve symptoms and quality of life in patients with heart failure (HF). Despite these advancements, the prognosis of patients with HF remains poor. Few Canadians have access to specialized care, such as clinicians with advanced HF training or dedicated multidisciplinary HF clinics. Thus, much of the management of HF is the responsibility of primary care, including pharmacists in community-based practices. The objective of this practice tool is to provide pharmacists with a succinct, contemporary, and stepwise approach to managing ambulatory patients with HF.

A practical approach for pharmacists

This stepwise approach informed the creation of an infographic to aid pharmacists in managing patients with HF (Figure 1).

Does my patient have heart failure?

Heart failure is a complex clinical syndrome where impaired heart function results in signs and symptoms of reduced cardiac output.¹ Hallmark symptoms include dyspnea, either on exertion, when lying flat (orthopnea), or awakening suddenly at night (paroxysmal nocturnal dyspnea [PND]), fatigue, exercise intolerance, and peripheral edema. Some signs of HF include low blood pressure (BP), respiratory crackles, weight gain, and elevated jugular venous pressure or serum natriuretic peptide levels.

What type of heart failure does my patient have?

Classification of HF is commonly based on a patient's left ventricular ejection fraction (LVEF), typically measured on a cardiac ultrasound (echocardiogram). A "normal" LVEF is considered to be >55%. Classification falls under 1 of 3 categories: HF with reduced ejection fraction (HFrEF, pronounced as "hef-ref"), HF with preserved ejection fraction (HFpEF, pronounced as "hef-pef"), or HF with mildly reduced ejection fraction (Table 1).² In 2025, a new HF guideline from the Canadian Cardiovascular Society (CCS) recommended combining HFpEF and HF with mildly reduced ejection fraction into 1 category termed "heart failure with non-reduced ejection fraction," as the pharmacological management is similar between these 2 groups.³ However, this term is not universally accepted, and there are nuances among these groups. A fourth category—HF with improved ejection fraction (HFimpEF)—is where a patient's LVEF improves by $\geq 10\%$ to be >40% compared to baseline. Some patients may have an abnormal LVEF, but do not have signs or symptoms of HF. These patients are referred to as having left ventricular dysfunction, for which management is beyond the scope of this paper.

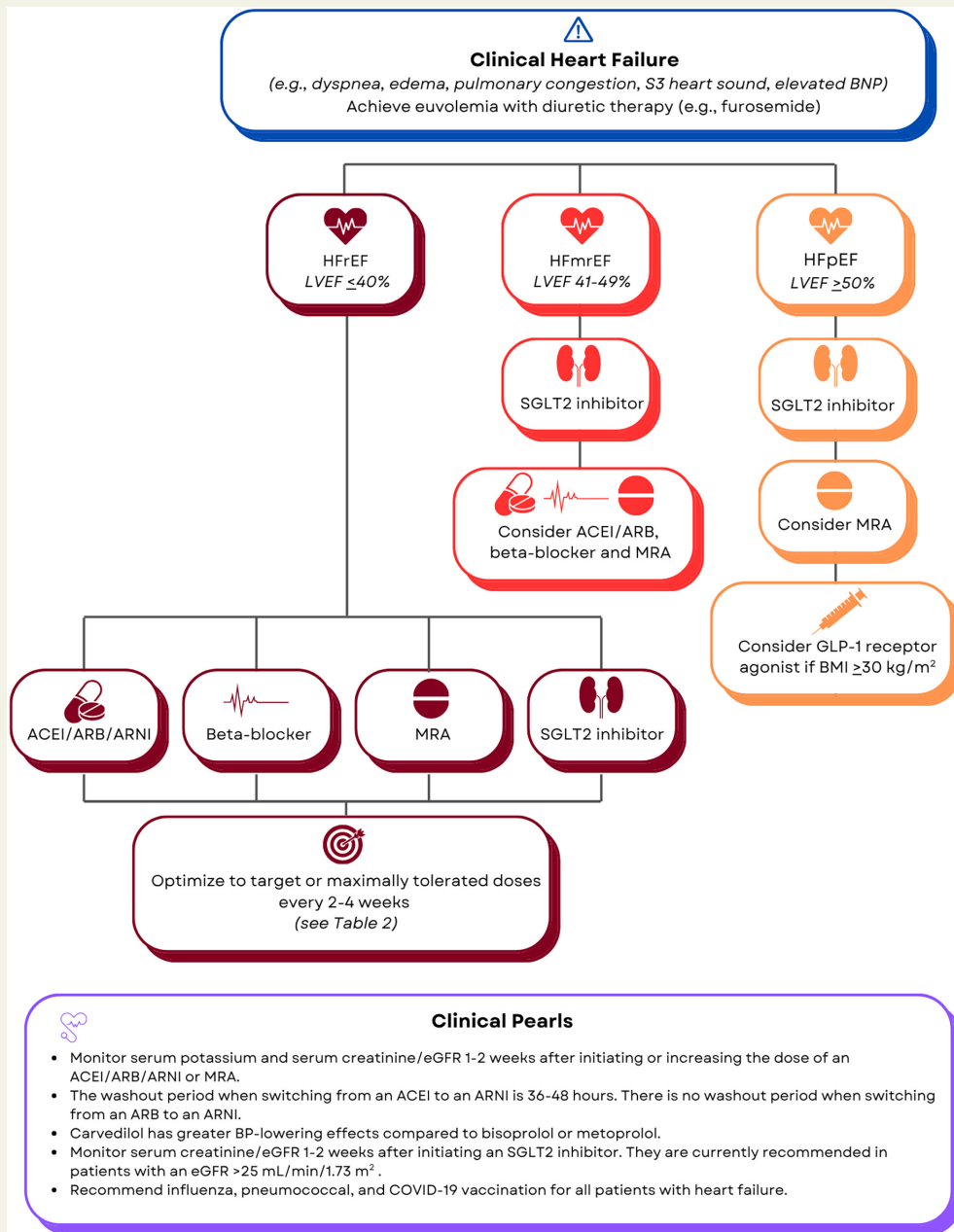
Does my patient have congestion? If so, how do I manage it?

Congestion in HF is secondary to impaired forward blood flow, decreased kidney perfusion, and neurohormonal effects, causing sodium and water retention. Congestion from the left side of the heart presents as dyspnea, orthopnea, and PND. Congestion from the right side of the heart causes leg edema and abdominal distention/ascites. Patients can have

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Figure 1 Flow diagram regarding the pharmacologic management of patients with heart failure

ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin II receptor blocker; ARNI, angiotensin receptor-neprilysin inhibitor; BMI, body mass index; BNP, B-type natriuretic peptide; BP, blood pressure; eGFR, estimated glomerular filtration rate; GLP-1, glucagon-like peptide-1; HFmrEF, heart failure with mildly reduced ejection fraction; HFpEF, heart failure with preserved ejection fraction; HFrEF, heart failure with reduced ejection fraction; LVEF, left ventricular ejection fraction; MRA, mineralocorticoid receptor antagonist; SGLT2, sodium-glucose cotransporter 2.

variable congestion, including no congestion. If a patient has excess fluid, the goal of therapy is to decongest them to improve symptoms. If a patient is acutely volume overloaded, decongestion may temporarily take priority over implementing disease-modifying therapy.

Furosemide is the most commonly used loop diuretic to manage a patient's volume status. Furosemide is only used to

maintain euvolemia (it has no disease-modifying benefit) and should be used at the lowest effective dose (which may be zero). A typical oral dose of furosemide ranges from 20 to 120 mg daily to twice daily, though higher doses may be given to refractory patients. If dosed twice daily, the second dose of furosemide should be taken in the early afternoon (13:00–14:00) to minimize bothersome nocturia. In cases where a

Table 1 Classification of heart failure

Category	LVEF
Heart failure with reduced ejection fraction (HFrEF)	≤40%
Heart failure with non-reduced ejection fraction (HFnrEF)	>40%
Heart failure with mildly reduced ejection fraction (HFmrEF)	41%–49%
Heart failure with preserved ejection fraction (HFpEF)	≥50%
Heart failure with improved ejection fraction (HFimpEF)	Increased by ≥10% from baseline to be >40%

LVEF, left ventricular ejection fraction.

patient has a blunted response to a loop diuretic (termed “diuretic resistance”), a thiazide diuretic (e.g., metolazone) may be added to increase diuresis. This combination is used sparingly (once or twice weekly) with frequent monitoring and for the shortest possible duration, as it can cause severe electrolyte abnormalities. If a patient does not respond to oral therapy, intravenous therapy may be required. Also, patients with abdominal congestion may require short-term intravenous furosemide due to impaired absorption of oral loop diuretic therapy.

Diuretic therapy should be monitored frequently, as overdiuresis may contribute to hypotension and renal dysfunction, and may limit the initiation and uptitration of disease-modifying therapy. Patients should measure their weight on a daily basis (typically first thing in the morning after emptying their bladder), as rapid weight gain (about 2 kg in 48 hours or 2.5 kg in 1 week) may be indicative of fluid retention and worsening HF. Recommendations to restrict sodium intake to <2000 mg per day to minimize fluid retention are commonplace; however, data to support these recommendations are weak. A recent trial demonstrated that sodium restriction did not reduce death or hospitalization in patients with HF.⁴ Fluid restriction (<2 litres per day) is typically only recommended for patients who are unable to manage their fluid status with diuretics.

What disease-modifying therapy should I recommend for my heart failure patients?

Heart failure with reduced ejection fraction: There are 4 standard “guideline-directed medical therapies” (GDMTs) for all patients with HFrEF (Table 2). Each of these classes of medications has been demonstrated to reduce death and/or HF hospitalizations and improve quality of life in patients with HFrEF. The most recent iteration of the CCS HFrEF guidelines recommends that all patients with HFrEF should be treated with: (i) renin–angiotensin–aldosterone system (RAAS) inhibitor (angiotensin-receptor neprilysin inhibitor [ARNI], angiotensin-converting enzyme inhibitor

[ACEI], or angiotensin receptor blocker [ARB]); (ii) beta-blocker; (iii) mineralocorticoid receptor antagonist (MRA); and (iv) sodium-glucose cotransporter-2 (SGLT2) inhibitor.⁵

For RAAS inhibitors, an ARNI is preferred over an ACEI/ARB based on the results of the PARADIGM-HF trial, which showed that sacubitril/valsartan was superior to enalapril at lowering the risk of cardiovascular death and HF hospitalization in patients with HFrEF (number needed to treat [NNT] 22).⁶ However, cost and insurance coverage are common barriers to accessing sacubitril/valsartan in practice. Additionally, there was more symptomatic hypotension in the sacubitril/valsartan group compared to enalapril, so this may also limit initiation. ARNIs are contraindicated in patients with any history of angioedema, the risk of which is increased when combined with an ACEI. Therefore, when switching from an ACEI to ARNI (or vice versa), the manufacturer recommends a 36-hour washout period to reduce the risk of angioedema, though 48 hours may be easier for some patients. There is no washout period when switching from an ARB to sacubitril/valsartan (or vice versa). ARBs are typically reserved for patients with an ACEI-induced cough. However, some clinicians may preferentially start an ARB to facilitate a future switch to sacubitril/valsartan. Sacubitril/valsartan (as well as SGLT2 inhibitors) have diuretic-sparing effects, so a patient's diuretic dose should be reassessed prior to initiation.

The addition of beta-blockers may initially worsen HF by decreasing cardiac output due to their negative inotropic effect, which may result in patients feeling worse and retaining fluid. For this reason, they must be started at low doses and gradually uptitrated to mitigate these effects (start low and go slow). Specific agents studied in HF are preferred (Table 2).

For MRAs, spironolactone is most commonly used, primarily due to its low cost. Eplerenone is often considered an alternative to spironolactone in patients with sex hormone-related adverse effects (breast tenderness, gynecomastia).

Use of SGLT2 inhibitors in HF includes those with and without diabetes. In patients with concurrent diabetes, adjustment

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Table 2 Simplified overview of the effect of heart failure pharmacologic therapy

	HFrEF (LVEF ≤40%)	HFmrEF (LVEF 41%–49%)	HFpEF (LVEF ≥50%)
ARNIs	+	+/-	+/-
ACEIs	+	+/-	+/-
ARBs	+	+ (candesartan)	+ (candesartan)
Beta-blockers	+ (bisoprolol, carvedilol, metoprolol)	+/-	+/-
MRAs	+ (eplerenone, spironolactone)	+ (finerenone) +/- (spironolactone)	+ (finerenone) +/- (spironolactone)
SGLT2 inhibitors	+ (dapagliflozin, empagliflozin)	+ (dapagliflozin, empagliflozin)	+ (dapagliflozin, empagliflozin)
Incretin mimetics	?	+/- (semaglutide)	+ (tirzepatide) +/- (semaglutide)

+ denotes a beneficial effect (lower risk of death and/or heart failure hospitalization); +/- denotes an inconclusive or unclear effect; ? denotes unknown effect. ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; ARNI, angiotensin receptor-neprilysin inhibitor; HFmrEF, heart failure with mildly reduced ejection fraction; HFpEF, heart failure with preserved ejection fraction; HFrEF, heart failure with reduced ejection fraction; LVEF, left ventricular ejection fraction; MRA, mineralocorticoid receptor antagonist; SGLT2, sodium-glucose cotransporter-2.

to other antihyperglycemic medications may be required to avoid hypoglycemia if their hemoglobin A1c is well-controlled. While rare, these patients should be cautioned about the risk of euglycemic diabetic ketoacidosis.

It is recommended that patients be initiated on all 4 GDMTs at low starting doses (an “in-parallel” approach, either together or in close succession), which are then slowly uptitrated to the target doses, as tolerated (Table 3). This is a shift from a “strict sequential” approach, in which patients are initiated on 1–2 GDMTs that are uptitrated to target or maximally tolerated doses before additional therapies are started. It is recommended that GDMTs be uptitrated to the target or maximally tolerated dose every 2–4 weeks. Older patients (≥80 years of age) may require slower uptitration (every 4–8 weeks) to minimize the risk of adverse effects. The choice of which agent to uptitrate is often based on patient-specific factors (e.g., BP, heart rate, renal function). A practical tip is to stagger up-titration of the ARNI/ACEI/ARB or MRA and beta-blocker to mitigate overlapping adverse effects (e.g., hyperkalemia, worsening renal function) and minimize the need for bloodwork (serum creatinine and electrolytes). Dapagliflozin and empagliflozin are typically started at the target dose (10 mg daily), so up-titration is not required.

Additional therapies, such as ivabradine, vericiguat, or digoxin, may be added to standard GDMTs based on specific circumstances. Beyond drug therapy, patients with HFpEF may also receive device therapy, such as an implantable cardioverter-defibrillator or biventricular pacemaker.

Heart failure with preserved ejection fraction: Despite HFpEF accounting for approximately half of all patients with HF,

few therapies have demonstrated an improvement in clinical outcomes in patients with HFpEF (Tables 2 and 3). Despite numerous clinical trials, no drug therapy to date has been shown to reduce cardiovascular or all-cause death in patients with HFpEF.⁷

Like HFrEF, SGLT2 inhibitors are recommended in all patients with HFpEF. Both dapagliflozin and empagliflozin were shown to reduce the composite of cardiovascular death and HF hospitalizations in patients with HFpEF and HFmrEF (LVEF >40%) in the DELIVER and EMPEROR-Preserved trials, respectively (NNT 33 and 31, respectively).^{8,9}

Use of MRAs in patients with HFpEF has been controversial due to the results of the TOPCAT trial, which showed that spironolactone 45 mg daily versus placebo did not reduce the composite endpoint of death from cardiovascular causes, aborted cardiac arrest, or HF hospitalization in patients with HFpEF (LVEF ≥45%).¹⁰ Yet, some clinicians will still recommend spironolactone based on a posthoc subgroup analysis that demonstrated patients who were enrolled from North and South America had a lower risk of the primary outcome versus those from Russia and Georgia.¹¹ The recently published FINEARTS-HF trial investigated a new non-steroidal MRA finerenone versus placebo in patients with HFpEF or HFmrEF (LVEF ≥40%).¹² Finerenone reduced the primary composite outcome of worsening HF events and cardiovascular death (NNT 31), though it did not result in any mortality benefit. Like other MRAs, renal dysfunction, hyperkalemia, and hypotension were more common with finerenone. As finerenone is non-steroidal, it has not been associated with sex hormone-related adverse effects. Finerenone was ap-

Table 3 Common pharmacologic therapy for patients with heart failure

Drug	Starting dose	Target dose
RAAS inhibitors		
Candesartan	4–8 mg PO daily	32 mg PO daily
Perindopril	2–4 mg PO daily	8 mg PO daily
Ramipril	1.25–2.5 mg PO BID	5 mg PO BID
Sacubitril/valsartan	24/26 mg PO BID	97/103 mg PO BID
Valsartan	40 mg PO BID	160 mg PO BID
Beta-blockers		
Bisoprolol	2.5 mg PO daily	10 mg PO daily
Carvedilol	3.125 mg PO BID	25 mg PO BID (if ≤ 85 kg) 50 mg PO BID (if > 85 kg)
Metoprolol	12.5–25 mg PO BID	100 mg PO BID
MRAs		
Eplerenone	25 mg PO daily	50 mg PO daily
Finerenone*	10–20 mg PO daily	20–40 mg PO daily
Spirolactone	12.5 mg PO daily	25–50 mg PO daily
SGLT2 inhibitors		
Dapagliflozin	10 mg PO daily	10 mg PO daily
Empagliflozin	10 mg PO daily	10 mg PO daily
Incretin mimetics		
Semaglutide	0.25 mg SC weekly	2.4 mg SC weekly
Tirzepatide	2.5 mg SC weekly	15 mg SC weekly

BID, twice a day; MRA, mineralocorticoid receptor antagonist; PO, by mouth; RAAS, renin–angiotensin–aldosterone system; SC, subcutaneously; SGLT2, sodium–glucose cotransporter-2.

*In the FINEARTS-HF trial, the dose of finerenone was started at 10 mg daily and uptitrated to 20 mg daily in patients with an estimated glomerular filtration rate of ≤ 60 mL/min/1.73 m², and started at 20 mg daily and uptitrated to 40 mg daily in patients with an estimated glomerular filtration rate of > 60 mL/min/1.73 m².

proved by Health Canada as an adjunct to standard therapy in adults with HFmrEF/HFpEF (LVEF $\geq 40\%$) in January 2026.

As many patients with HFpEF also have hypertension, use of ARBs is common. Candesartan is the preferred option based on the CHARM-Preserved trial, which showed a lower risk of HF hospital admissions versus placebo (NNT 42), though it was a secondary outcome.¹³ Sacubitril/valsartan is generally not recommended in patients with HFmrEF or HFpEF, as it failed to show a benefit in the PARAGON-HF trial.¹⁴ There are no data supporting the use of beta-blockers as disease-modifying therapy in patients with HFpEF; however, patients may take them for other comorbidities (e.g., atrial fibrillation, angina).

There are some preliminary data regarding the use of incretin mimetics (e.g., semaglutide, tirzepatide) in patients with HFpEF (with and without diabetes). The STEP-HFpEF and STEP-HFpEF DM trials investigated subcutaneous semaglutide in patients with HFpEF (LVEF $\geq 45\%$) and obesity (body mass index [BMI] ≥ 30 kg/m²).^{15,16} Compared to placebo, semaglutide significantly improved clinical symptoms and achieved greater weight loss in both trials. However, they were not designed to evaluate HF hospitalization or cardiovascular death. Tirzepatide was compared to a placebo in patients with HFpEF (LVEF $\geq 50\%$) and obesity (BMI ≥ 30 kg/m²) in the recently published SUMMIT trial.¹⁷ Tirzepatide reduced the composite outcome of any worsening HF event and cardiovascular death (NNT 19) and improved clinical symptoms,

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Table 4 Key monitoring parameters for heart failure pharmacologic therapy

Drug class	BP	HR	Serum potassium	SCr	Other
	Monitor for low systolic BP (<90 mmHg), orthostatic hypotension (systolic BP drop of ≥20 mmHg with postural change), and symptoms of hypotension (e.g., lightheadedness)	Monitor for marked bradycardia (HR <50 bpm)	Monitor renal function (SCr) and serum potassium 1–2 weeks after initiating or increasing the dose		
RAAS inhibitors*	✓	–	✓	✓	Cough (ACEI > ARNI)
Beta-blockers	✓	✓	–	–	Fatigue, exercise intolerance
MRAs	✓ (less)	–	✓	✓	Gynecomastia (spironolactone)
SGLT2 inhibitors	✓ (minimal)	–	–	✓	Genital infection (yeast infection, UTI)
Incretin mimetics	–	–	–	–	GI effects (nausea, vomiting, diarrhea, abdominal pain)

✓/known adverse effect—monitor closely.
ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; ARNI, angiotensin receptor-neprilysin inhibitor; BP, blood pressure; bpm, beats per minute; HR, heart rate; GI, gastrointestinal; MRA, mineralocorticoid receptor antagonist; RAAS, renin–angiotensin–aldosterone system; SBP, systolic blood pressure; SCr, serum creatinine; SGLT2, sodium-glucose cotransporter-2; UTI, urinary tract infection.
*In the PARADIGM-HF trial, hypotension was more common with ARNI vs ACEI, while elevated serum creatinine (≥221 μmol/L) and severe hyperkalemia (>6.0 mmol/L) were less common with ARNI vs ACEI.

but more patients discontinued therapy due to gastrointestinal adverse events. Neither of these agents has indications for HF in Canada at present.

Heart failure with mildly reduced ejection fraction: The management of patients with HFmrEF (LVEF 41%–49%) is not well-understood. There are no landmark randomized controlled trials evaluating drug therapy specifically in patients with HFmrEF; however, several trials have included this subgroup. The SGLT2 inhibitors (dapagliflozin and empagliflozin) are generally recommended in patients with HFmrEF based on the results of the DELIVER and EMPEROR-Preserved trials.^{8,9} Also, some clinicians recommend MRA therapy in patients with HFmrEF based on the TOPCAT (LVEF ≥45%) and FINEARTS-HF (LVEF ≥40%) trials.^{10,12} Thus, the 2025 CCS guidelines recommend both an SGLT2 inhibitor and MRA in patients with “heart failure with non-reduced ejection fraction.”³ Posthoc subgroup and

meta-analyses have generally found a benefit with ACEI/ARBs and beta-blockers in patients with HFmrEF.¹⁸ Therefore, these patients could be considered for standard GDMTs. The 2025 CCS “heart failure with non-reduced ejection fraction” guidelines recommend candesartan as the preferred agent for blood pressure control in patients with hypertension, but do not recommend the routine use of beta-blockers in this population.

Heart failure with improved ejection fraction: Patients with HFimpEF are often continued to be managed as those with HFrEF. One small trial demonstrated that continuing pharmacologic therapy in patients with HFimpEF (after being diagnosed with dilated cardiomyopathy), as opposed to withdrawing therapy, resulted in a lower rate of HF relapse within 6 months.¹⁹ Therefore, it is generally recommended to continue standard therapy in patients with HFimpEF. It is important to note that LVEF is dynamic and can change over time. Thus,

HFimpEF could be thought of as HFrEF in remission, rather than HFmrEF or HFpEF.

What are the key monitoring parameters for patients on HF medications?

Common adverse effects of HF medications are included in Table 4.

Hypotension is common with most medications used in HF, with MRAs and SGLT2 inhibitors to a lesser degree than RAAS inhibitors and beta-blockers. Diuretic therapy may cause hypotension due to overdiuresis. A low systolic BP (90–100 mmHg) is generally acceptable if the patient is asymptomatic. Practical tips to mitigate symptomatic hypotension include ensuring the patient is not hypovolemic, staggering BP-lowering medications to morning and evening, and, if appropriate, eliminating or reducing non-essential medications that may also lower BP (e.g., amlodipine for hypertension, tamsulosin for benign prostatic hypertrophy).

Renal dysfunction is common with most HF medications except beta-blockers. Notably, renal dysfunction may also represent worsening HF or hypovolemia, so it is important to determine the cause. When initiating a RAAS inhibitor or SGLT2 inhibitor, a rise in serum creatinine up to 30% from baseline is acceptable. Another common reason for renal dysfunction is the use of over-the-counter non-steroidal anti-inflammatory drugs, which should generally be avoided in patients with HF. Finally, any acute illness that causes hypovolemia (e.g., diarrhea, vomiting) may cause renal dysfunction that is exacerbated by HF medications (except beta-blockers). Therefore, educating patients about “sick day management” is key.

Hyperkalemia is common in patients taking RAAS inhibitors and MRAs. Mild hyperkalemia (defined as a serum potassium level of 5.1–5.5 mmol/L) is acceptable in most patients and does not warrant a dose reduction.⁵ Further, it can often be corrected with dietary interventions by avoiding high potassium-containing foods (e.g., bananas, potatoes). Referral to a dietician may be warranted. Patients with moderate hyperkalemia (>5.5 mmol/L) likely require a reduction in the dose of their MRA and/or ARNI/ACEI/ARB.

For beta-blockers, most patients do not experience the worrisome systemic adverse effects (e.g., depression, sleep disturbances, bronchospasm, cold extremities) with the cardioselective beta-blockers commonly used in HF. Carvedilol is a combined alpha-beta blocker, which has a more potent BP-

lowering effect than bisoprolol or metoprolol. A heart rate of 50–60 bpm is typically acceptable if the patient is asymptomatic (absence of lightheadedness, syncope, excessive fatigue).

An uncommon adverse effect (4%–5%) of SGLT2 inhibitors that warrants discussion with patients is genital infections (e.g., yeast infection, urinary tract infection). The risk tends to be higher among women versus men, older adults, and those with diabetes and/or hyperglycemia or a prior history of genital infection. They are often readily treated with antifungals or antibiotics, and usually do not necessitate discontinuation of therapy.

What is the role of the pharmacist in managing patients with heart failure?

Pharmacists in community- or primary care-based practice settings can play a meaningful role in caring for patients with HF, as many patients do not have access to specialized cardiology care. For patients with HFrEF, an easy “win” is to ensure patients are taking all 4 standard GDMTs, as tolerated. It is important to educate patients, as well as other clinicians, that SGLT2 inhibitors are now recommended for most HF patients (regardless of their LVEF), irrespective of having diabetes. As polypharmacy is common among HF patients, pharmacists can assist with streamlining patients’ medication regimens. For patients who have difficulty with twice-daily dosing, a once-daily regimen is possible for all agents with the exception of sacubitril/valsartan. Studies have demonstrated that pharmacist involvement in the care of patients with HF can improve the use of GDMTs and achievement of target doses, as well as reduce all-cause death and hospitalizations.^{20,21} Use of a pharmacist-led stewardship tool increased the use of GDMTs in hospitalized patients with HFrEF, which could potentially be translated to outpatient settings.²²

Conclusion

Management of HF across the spectrum of LVEF has evolved over the past decade. With the increasing complexity of medical management, pharmacists can play a key role in ensuring patients receive contemporary pharmacologic treatment (safely and effectively) tailored to their type of HF, and in educating patients about their therapy goals to ensure they align with their values and preferences. ■

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