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Advances in the pharmacological management of heart failure with reduced ejection fraction: A narrative review

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Abstract

Background: Heart failure with reduced ejection fraction (HFrEF) is a chronic and progressive cardiovascular syndrome associated with high morbidity, mortality, and healthcare utilisation worldwide. Despite significant advances in cardiovascular medicine, patients with HFrEF continue to experience frequent hospitalisations and impaired quality of life. Over the past decade, an improved understanding of the complex pathophysiology of HFrEF, including neurohormonal activation, maladaptive ventricular remodelling, inflammation, and metabolic dysfunction, has driven the development of multiple disease-modifying pharmacotherapies and updated treatment paradigms.

Methods: This narrative review synthesises contemporary evidence published between 2020 and 2025 on the pathophysiology and pharmacological management of HFrEF. Major randomised controlled trials, meta-analyses, and international guideline documents were reviewed to evaluate established and emerging therapies, including angiotensin receptor–neprilysin inhibitors, beta-blockers, mineralocorticoid receptor antagonists, sodium–glucose cotransporter-2 inhibitors, and newer agents such as ivabradine, vericiguat, and omecamtiv mecarbil. Particular emphasis was placed on mechanisms of action, clinical outcomes, and the expanding role of clinical pharmacists and Pharm D professionals in optimising guideline-directed medical therapy.

Conclusion: The management of HFrEF has evolved toward early, multidrug, guideline-directed therapy targeting multiple pathophysiological pathways. Integrating emerging pharmacological agents with pharmacist-led care models offers substantial potential to improve adherence, reduce hospital readmissions, and enhance long-term clinical outcomes in patients with HFrEF.

Keywords: Heart failure with reduced ejection fraction; HFrEF; Guideline-directed medical therapy; Neurohormonal activation; Clinical pharmacy

1. INTRODUCTION

Heart failure (HF) is a complex clinical syndrome characterised by typical symptoms such as dyspnea, fatigue and fluid retention arising from structural or functional abnormalities of the heart. Contemporary definitions from international societies emphasise the presence of objective cardiac dysfunction and supporting biomarkers, such as elevated natriuretic peptides, to confirm the diagnosis. Among the different phenotypes of HF, heart failure with reduced ejection fraction (HFrEF), defined by a left ventricular ejection fraction (LVEF) of 40% or less, remains the most extensively studied and therapeutically actionable form.

Despite advances in cardiovascular care, the prognosis of HFrEF remains guarded, with five-year mortality rates approaching those of many malignancies. Recurrent hospitalisations due to decompensation contribute significantly to

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impaired quality of life and healthcare costs. These challenges have driven intensive research into therapies that not only alleviate symptoms but also modify disease progression and improve survival.[1] [2] [3]

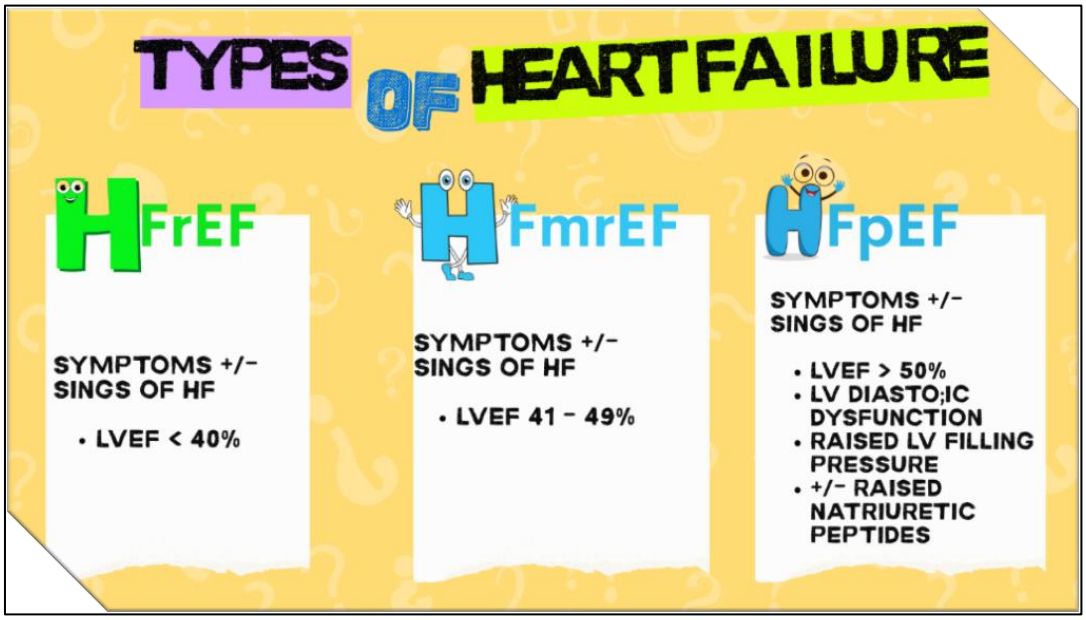

Figure 1 Types of heartfailure

2. CLASSIFICATION AND GLOBAL BURDEN OF HFREF

HF is commonly categorised according to LVEF into HFrEF (≤40%), HF with mildly reduced EF (41–49%), and HF with preserved EF (≥50%). Patients with HFrEF account for approximately half of all HF cases globally and experience the highest rates of mortality and hospitalisation. The growing prevalence of HFrEF is largely attributable to population ageing, improved survival after acute myocardial infarction, and the rising incidence of cardiometabolic risk factors such as hypertension, diabetes and obesity. Low- and middle-income countries are experiencing a particularly rapid increase in HF burden, often compounded by delayed diagnosis and limited access to advanced therapies. [3][4]

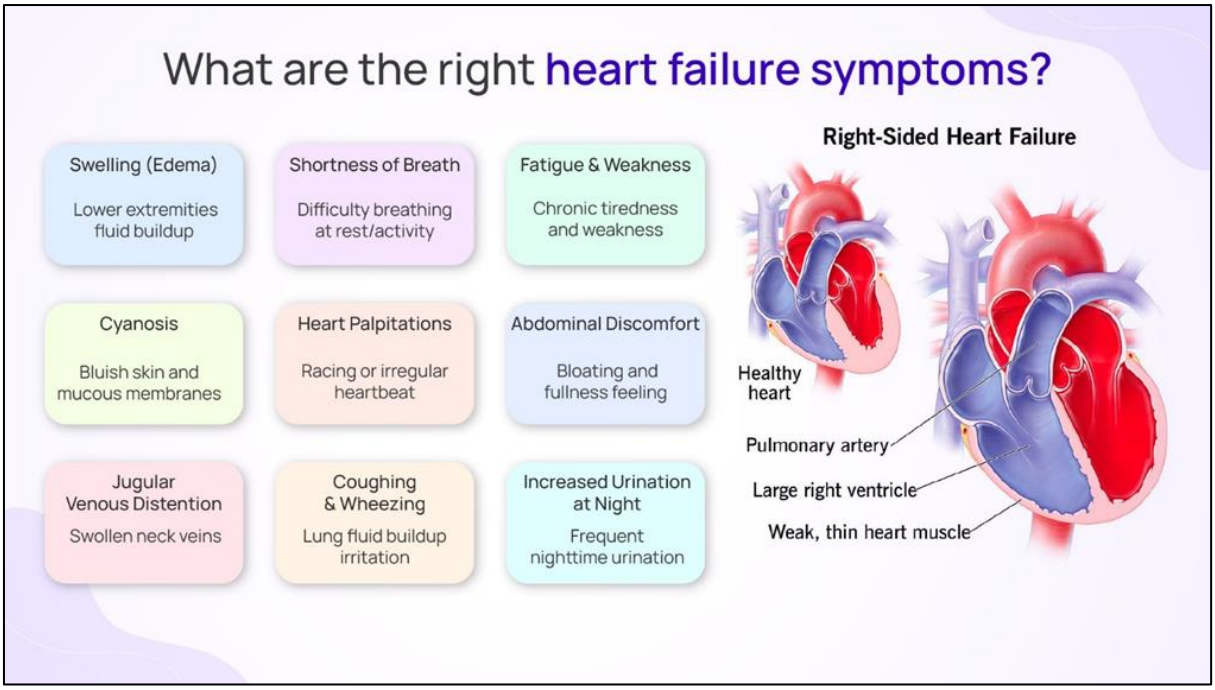

Figure 2 Symptoms of heartfailure

3. PATHOPHYSIOLOGY OF HFREF

The pathophysiology of HFrEF is multifactorial and involves a dynamic interplay between myocardial injury, neurohormonal activation, inflammation, and metabolic derangements. Initial insults such as ischemic injury, cardiomyopathies, or toxic exposures lead to loss of functional cardiomyocytes and reduced contractility. Compensatory mechanisms, including activation of the Renin Angiotensin Aldosterone System (RAAS) and Sympathetic Nervous System (SNS), initially maintain cardiac output but ultimately promote maladaptive ventricular remodelling, fibrosis and progressive systolic dysfunction.

Natriuretic peptide systems, which normally counterbalance RAAS and SNS activity, become insufficient due to increased degradation and reduced receptor responsiveness. Concurrently, chronic inflammation, oxidative stress, mitochondrial dysfunction and impaired myocardial energetics further compromise cardiac performance. These mechanisms provide the biological rationale for multi-target pharmacological strategies in HFrEF.[5][6][7][8][9][10]

Table Comparison of Mechanisms of Action of Advanced Pharmacologic Agents in HFvEF

Table 1 Comparison of Mechanisms of Action of Advanced Pharmacologic Agents in HFvEF

Drug Class / Agent	Mechanism of Action	Key Physiological Effects
ARNI (Sacubitril/Valsartan)	Sacubitril: Inhibits neprilysin → ↑ natriuretic peptides (ANP, BNP), bradykinin, adrenomedullin. Valsartan: Blocks AT1 receptors → inhibits RAAS.	- Vasodilation - Natriuresis, diuresis - ↓ Sympathetic activity - ↓ Fibrosis/remodeling - ↓ Preload & afterload
SGLT2 Inhibitors (Dapagliflozin, Empagliflozin, Sotagliflozin)	Inhibit SGLT2 in proximal renal tubule → ↓ glucose & sodium reabsorption.	- Osmotic diuresis & natriuresis - ↓ Congestion & preload - Improved cardiac metabolism (↑ ketone use) - ↓ Inflammation & oxidative stress - Renal protection
sGC Stimulator (Vericiguat)	Direct stimulation of soluble guanylate cyclase → ↑ cGMP production even in low NO states.	- Vasodilation - Improved myocardial relaxation - ↓ Fibrosis & remodeling - ↓ Preload & afterload
Cardiac Myosin Activator (Omecamtiv Mecarbil)	Selectively binds cardiac myosin → ↑ actin–myosin interaction & prolongs systolic ejection time without ↑ intracellular Ca^{2+}.	- ↑ Contractility safely - ↑ Stroke volume - ↑ Pump efficiency - No ↑ oxygen demand or arrhythmia risk
If Channel Inhibitor (Ivabradine)	Selectively inhibits the If (funny) current in SA node → ↓ heart rate without affecting contractility.	- ↑ Diastolic filling time - ↓ O_2 demand - ↓ Hospitalizations
MRAs (Spironolactone, Eplerenone)	Block aldosterone receptors in the kidney & myocardium.	- Natriuresis - ↓ Fibrosis & remodelling - Improved survival
ACE Inhibitors (e.g., Enalapril)	Inhibit ACE → ↓ angiotensin II & aldosterone.	- Vasodilation

		- ↓ Preload & afterload - ↓ Remodeling
ARBs (e.g., Valsartan, Losartan)	Block AT1 receptor → inhibit angiotensin II effects.	- ↓ Vasoconstriction - ↓ Aldosterone - ↓ Remodeling

Table Dosing, Monitoring Parameters and Contraindications of Key HFvEF Drugs.

Table 2 Dosing, Monitoring Parameters, and Contraindications of Key HFvEF Drugs

Drug Class / Agent	**Typical Dosing**	**Monitoring Parameters**	**Contraindications / Precautions**
ARNI (Sacubitril/Valsartan)	Start: 24/26 mg or 49/51 mg BID (based on BP & renal function) Target: 97/103 mg BID	- BP (risk of hypotension) - Serum potassium - Renal function (SCr, eGFR) - Signs of angioedema	- Concomitant ACE inhibitor use (36-hour washout required) - History of angioedema - Pregnancy - Severe hepatic impairment
SGLT2 Inhibitors (Dapagliflozin, Empagliflozin, Sotagliflozin)	Dapagliflozin: 10 mg once daily, Empagliflozin: 10 mg once daily, Sotagliflozin: 200–400 mg once daily	- Renal function (eGFR) - Volume status - Blood glucose (in diabetics) - Signs of UTI or genital infections	- eGFR < 20–25 mL/min/1.73 m^2 (varies by drug) - Type 1 diabetes (risk of ketoacidosis) - Recurrent genital infections - Severe dehydration
sGC Stimulator (Vericiguat)	Start: 2.5 mg once daily. Titrate: 5 mg → 10 mg once daily	- BP (hypotension risk) - Pregnancy test if applicable - Symptoms of dizziness or syncope	- Concomitant nitrates or PDE-5 inhibitors - Pregnancy (teratogenic) - Symptomatic hypotension
Cardiac Myosin Activator (Omecamtiv Mecarbil)	25 mg BID → titrated to 50 mg BID using plasma level monitoring (if available)	- Cardiac function (EF, stroke volume) - Troponin (rare ↑ may occur) - Heart rate & rhythm	- Severe hepatic impairment - Use with strong CYP3A4 inhibitors - Ventricular arrhythmia history (caution)
If Channel Inhibitor (Ivabradine)	Start: 5 mg BID Target: 7.5 mg BID (aim HR 50–60 bpm)	- Heart rate - BP - ECG for atrial fibrillation	- HR <70 bpm at baseline - Acute decompensated HF - Conduction abnormalities (SA/AV block) - Severe hepatic impairment - Pregnancy
MRAs (Spironolactone, Eplerenone)	Spironolactone: 12.5–25 mg/day → 25–50 mg/day Eplerenone: 25 mg/day → 50 mg/day	- Serum potassium (risk of hyperkalemia) - Renal function (SCr, eGFR)	- Hyperkalemia (K^+ > 5.0) - Renal failure (eGFR < 30 mL/min)

		- BP	- Addison's disease - Concomitant potassium supplements
ACE Inhibitors (Enalapril, Ramipril)	Enalapril: 2.5 mg BID → 10–20 mg BID Ramipril: 2.5 mg/day → 10 mg/day	- BP - Serum potassium - Renal function - Cough/angioedema	- Pregnancy - History of angioedema - Bilateral renal artery stenosis

4. Established pharmacological therapies

4.1. Angiotensin Converting Enzyme Inhibitors and Angiotensin Receptor Blockers

Angiotensin-converting enzyme inhibitors (ACEIs) reduce the formation of angiotensin II and attenuate aldosterone secretion, leading to vasodilation, reduced preload and afterload and inhibition of pathological remodelling. Landmark trials such as CONSENSUS and SOLVD established ACEIs as the first agents to confer a survival benefit in HFrEF. Angiotensin receptor blockers (ARBs) provide an alternative for patients intolerant to ACEIs and exert similar hemodynamic and anti-remodelling effects through selective blockade of the angiotensin II type-1 receptor.

4.2. Evidence-Based Beta-Blockers

Beta-blockers counteract chronic SNS activation, reduce heart rate, improve myocardial efficiency and decrease arrhythmic risk. Large trials including CIBIS-II, MERIT-HF and COPERNICUS demonstrated significant reductions in mortality and hospitalisation with bisoprolol, metoprolol succinate, and carvedilol, respectively. These agents are now foundational components of GDMT in clinically stable patients with HFrEF.

4.3. Mineralocorticoid Receptor Antagonists

MRAs such as spironolactone and eplerenone inhibit aldosterone-mediated sodium retention and myocardial fibrosis. Trials including RALES, EPHESUS, and EMPHASIS-HF showed substantial reductions in mortality and HF hospitalisations across a broad spectrum of disease severity. Careful monitoring of renal function and serum potassium is essential to ensure safety.[1][2][13]

5. BREAKTHROUGH AND EMERGING THERAPIES

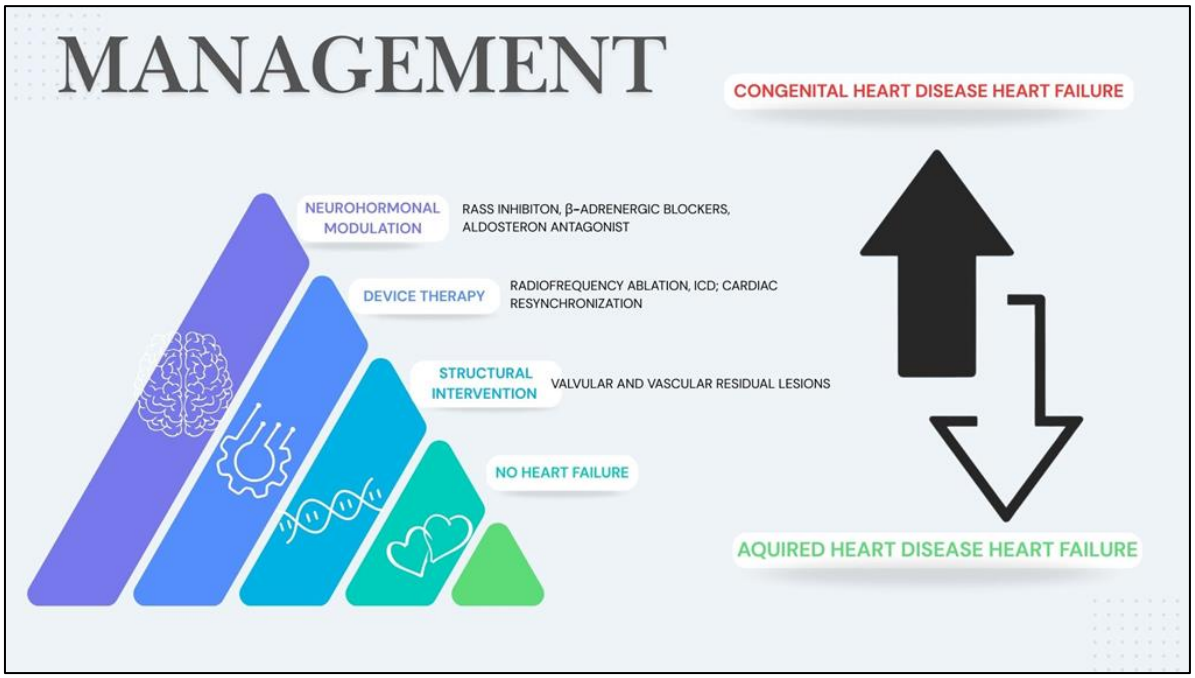

Figure 3 management of heartfailure

5.1. Angiotensin Receptor Neprilysin Inhibitors

Sacubitril / valsartan combines neprilysin inhibition with angiotensin receptor blockade, enhancing endogenous natriuretic peptide activity while suppressing RAAS. In the PARADIGM-HF trial, this agent significantly reduced cardiovascular mortality and HF hospitalisation compared with enalapril, leading to its preferential recommendation in contemporary guidelines.

5.2. Sodium Glucose Cotransporter-2 Inhibitors

Initially developed for type 2 diabetes, SGLT2 inhibitors such as dapagliflozin and empagliflozin have demonstrated robust benefits in HFrEF irrespective of diabetic status. Trials including DAPA-HF and EMPEROR - Reduced showed marked reductions in HF hospitalisations and improved patient-reported outcomes, with favourable renal effects and early onset of benefit.

5.3. Ivabradine

Ivabradine selectively lowers heart rate by inhibiting the sinoatrial node If current. In the SHIFT trial, ivabradine reduced HF hospitalisations in patients with sinus rhythm and persistent tachycardia despite optimised beta-blocker therapy, supporting its role as an adjunctive agent in selected patients.

5.4. Vericiguat

Vericiguat stimulates soluble guanylate cyclase and enhances cyclic guanosine monophosphate signalling, a pathway often impaired in HF. The VICTORIA trial demonstrated a modest reduction in cardiovascular death or HF hospitalisation among high-risk patients with recent decompensation, highlighting its potential role in advanced disease.

5.5. Omecamtiv Mecarbil

Omecamtiv mecarbil is a first-in-class cardiac myosin activator that improves systolic function without increasing intracellular calcium or myocardial oxygen demand. In GALACTIC-HF, it produced a modest but significant reduction in HF events, particularly among patients with severely reduced EF.[11][12][14][15]

6. ROLE OF CLINICAL PHARMACISTS AND PHARM D PROFESSIONALS

Heart failure with reduced ejection fraction (HFrEF) is a complex, multi-drug condition requiring careful initiation, titration, and monitoring of multiple pharmacologic classes. Because optimal outcomes depend heavily on medication safety, adherence, and timely up-titration of GDMT, Pharm D professionals and clinical pharmacists play an indispensable role within multidisciplinary HF care teams. Their involvement has been shown to improve medication optimisation, reduce HF-related hospitalisations, increase GDMT uptake, and enhance patient-reported outcomes in various health systems worldwide.

6.1. Medication Reconciliation

Medication reconciliation is crucial for safe and effective HF management, particularly given the polypharmacy common in HFrEF.

6.1.1. Key responsibilities

- Identifying discrepancies between prescribed medications and actual patient use.
- Detecting inappropriate or harmful combinations (e.g., triple RAAS blockade, NSAIDs causing fluid retention, interacting antiarrhythmics).
- Verifying **ACEi → ARNI washout period** to prevent angioedema risk.
- Reviewing anticoagulants/antiplatelets in HF patients with AF, CAD, or post-MI status.
- Ensuring renal-safe agents for patients with CKD or diabetes.

6.1.2. Impact

- Prevents medication errors during transitions of care (admission, discharge, follow-up).
- Reduces ADEs that could precipitate HF decompensation.
-

6.2. Monitoring of Adverse Effects

HFrEF pharmacotherapy involves drugs with significant renal, metabolic, and hemodynamic effects. Pharmacists are essential in the early identification of adverse events and in adjusting therapy.

6.2.1. Key monitoring domains

- **Electrolytes**: hyperkalaemia with MRAs, renal dysfunction with ACEi/ARB/ARNI, volume depletion with SGLT2 inhibitors.
- **Renal function**: creatinine/eGFR while using RAAS inhibitors and SGLT2i.
- **Blood pressure**: risk of hypotension with ARNI, β-blockers, SGLT2i.
- **Heart rate**: bradycardia with β-blockers or ivabradine threshold evaluation.
- **Volume status**: identifying congestion, dehydration, or diuretic resistance.
- **Drug–drug interactions**: digoxin, amiodarone, DOACs, CYP modulators.

6.2.2. Outcome

- Decreases risk of hospitalisation due to preventable adverse drug reactions.
- Ensures safe up-titration of GDMT toward evidence-based target doses.

6.3. Optimising GDMT (Guideline-Directed Medical Therapy)

One of the strongest evidence-supported roles for clinical pharmacists lies in optimising GDMT, which includes ARNI, β-blockers, MRAs, and SGLT2 inhibitors.

6.3.1. Pharmacist responsibilities

- Initiating appropriate GDMT pillars early (when allowed by local protocols).
- Titrating to target or maximally tolerated doses using structured schedules.
- Identifying and managing barriers such as hypotension, renal limitation, or cost.
- Switching from ACEi/ARB to ARNI when indicated.
- Ensuring correct sequencing after acute decompensation (e.g., starting SGLT2i soon after stabilisation).
- Monitoring biomarker trends (NT-proBNP) and symptoms.

6.3.2. Impact on outcomes

Evidence shows:

- Higher GDMT uptake rates in pharmacist-led HF clinics.
- Faster titration to target doses.
- Lower all-cause hospitalisations and HF readmissions.
- Improved survival in some real-world analyses.

6.4. Counselling on Adherence and Lifestyle Modification

Adherence in HF is often poor due to pill burden, side effects, and lifestyle demands. Pharmacists have demonstrated a significant impact in improving adherence.

6.4.1. Counselling focus areas:

- **Medication adherence**: timing, missed-dose instructions, expected side effects.
- **Lifestyle**:
  - Sodium restriction
  - Fluid management
  - Weight monitoring ("2 kg in 2 days" rule)
  - Smoking and alcohol reduction
  - Exercise and cardiac rehabilitation
- **Sick-day rules**: withholding diuretics or SGLT2 inhibitors during dehydration/illness.
- **Vaccination advice** (influenza, pneumococcal).
- **Recognising early warning signs of decompensation**.

6.4.2. Impact

- Improved adherence and patient empowerment.
- Fewer exacerbations and better QOL scores.
- Reduced emergency visits.

6.5. Reducing Hospital Readmissions

Hospital readmissions are a major burden in heart failure and are closely linked to medication issues such as nonadherence, inadequate titration, or adverse effects.

6.5.1. Pharmacist contributions to reducing readmission

- HF discharge counselling and post-discharge follow-up (phone calls, telehealth).
- Early outpatient monitoring of BP, HR, K+, and renal function.
- Ensuring correct diuretic plans (spot-dose adjustments).
- Identifying patients at high risk: recent decompensation, polypharmacy, low literacy.
- Coordination with cardiologists and HF nurses for early intervention.

6.5.2. Measured benefits (reported in studies)

- 20–40% reduction in 30-day HF readmissions in pharmacist-led programs.
- Lower ED visits and improved continuity of care.

Optimal HFrEF management requires careful initiation, titration, and monitoring of multiple drug classes. Clinical pharmacists play a critical role in medication reconciliation, prevention of drug-related problems, monitoring of adverse effects, and optimisation of GDMT. Pharmacist-led HF programs have been associated with higher rates of target-dose achievement, improved adherence, reduced readmissions, and better quality of life. Patient education regarding lifestyle modification, self-monitoring, and early recognition of decompensation further enhances outcomes.[16][17]

7. Challenges and future directions

Despite strong evidence, real-world implementation of GDMT remains suboptimal due to cost constraints, polypharmacy, and adherence issues. Future strategies are likely to incorporate precision medicine approaches using biomarkers, genomics, and advanced imaging to individualise therapy. Emerging gene-based and regenerative treatments, along with digital health technologies and remote monitoring, may further transform HF care. Continued interdisciplinary collaboration, particularly involving clinical pharmacists, will be essential to translate innovation into practice. [1][2]

8. Conclusion

The pharmacological management of HFrEF has undergone a paradigm shift, with early combination therapy using multiple disease-modifying agents now recognised as the most effective approach. While significant progress has been achieved, ongoing efforts are required to overcome implementation barriers and to integrate emerging therapies and technologies. Strengthening pharmacist-led care models and embracing personalised strategies will be central to improving long-term outcomes and reducing the global burden of heart failure.

Compliance with ethical standards

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