

Heart Failure With Improved Ejection Fraction (HFimpEF): Current Evidence, Clinical Implications, and Future Directions

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ABSTRACT

Heart failure with improved ejection fraction (HFimpEF) has emerged in recent years as a distinct clinical phenotype within the spectrum of heart failure subtypes. Current evidence supports the hypothesis that HFimpEF represents a state of functional remission rather than a definitive cure, with important clinical implications, particularly for the continuation of guideline-directed medical therapy (GDMT) and for decision-making regarding device therapies. Withdrawal or de-escalation of treatment is associated with high rates of recurrent systolic function deterioration, justifying the indefinite continuation of disease-modifying therapy. A descriptive narrative review of the available literature was performed to summarise current

evidence on the definition, epidemiology, pathophysiological mechanisms, left ventricular ejection fraction (LVEF) trajectories, advanced diagnostic tools, and therapeutic considerations in HFimpEF, and to discuss the main knowledge gaps and future research directions. A better understanding of this phenotype will enable more effective risk stratification and progress towards more individualised management of patients with heart failure, with potential benefits for long-term patient outcomes and care.

Introduction

Heart failure with improved ejection fraction (HFimpEF) is observed in up to half of patients with heart failure with reduced ejection fraction (HFrEF) and represents a heterogeneous phenotype in terms of aetiology, pathophysiological mechanisms, durability of recovery, and prognosis [1]. Although patients with HFimpEF have substantially reduced mortality and hospitalisations for heart failure (HHF) compared with those with persistent HFrEF, improvement in left ventricular ejection fraction (LVEF) does not indicate complete myocardial recovery [2,3]. The presence of subclinical structural, molecular, and functional alterations leads to a significant risk of relapse and adverse long-term cardiovascular events, especially in those with transient improvement in LVEF [4]. This incomplete recovery leaves uncertainty regarding long-term prognosis and optimal management strategies in this population.

Definition

HFimpEF is characterised by an improvement in LVEF of at least 10% from a baseline LVEF $\leq 40\%$ after optimal therapy in patients previously diagnosed with HFrEF [1,2,5]. This improvement may result from guideline-directed medical therapy (GDMT), such as neurohormonal blockers and newer agents, specifically angiotensin receptor-neprilysin inhibitors (ARNI, sacubitril/valsartan) and sodium–glucose cotransporter 2 (SGLT2) inhibitors [2,4–6]. Less frequently, LVEF might improve spontaneously, reflecting intrinsic myocardial reserve and favourable remodelling. This definition highlights the dynamic course of systolic function and its potential for recovery, challenging the traditional view of heart failure as a progressive and irreversible disease.

Epidemiology and pathomechanisms

Observational cohorts and contemporary registries demonstrate that HFimpEF is a frequent outcome among patients initially diagnosed with HFrEF, with reported prevalence ranging from approximately 20% to over 50%, depending on population characteristics, underlying HF aetiology, intensity of medical therapy, as well as the timing and frequency of LVEF reassessment [1,7]. Most LVEF improvement occurs early, typically within the first 12–24 months after diagnosis or initiation of optimised therapy, underscoring the importance of longitudinal evaluation rather than reliance on a single echocardiographic measurement [1,8].

Despite apparent functional recovery, HFimpEF represents a heterogeneous phenotype. Not all patients sustain improvement in LVEF over time; only approximately 70% maintain LVEF $>40\%$ on subsequent imaging, defining persistent HFimpEF, whereas the remainder experience transient improvement followed by recurrent systolic dysfunction [1,9].

The heterogeneity within the HFimpEF group is further shaped by demographic, etiological, and structural factors. Female sex, younger age, shorter HF duration, higher baseline systolic blood pressure, smaller ventricular volumes, and fewer comorbidities are associated with a higher likelihood of recovery [1,4,10]. On the other hand, adverse structural remodelling, larger left ventricular end-diastolic dimensions, renal dysfunction, diabetes, and atrial fibrillation predict less prevalent improvement [6,11,12]. Large registries, including IMPROVE HF and SwedeHF, consistently support these associations and further validate the clinical profile associated with LVEF recovery. Beyond clinical characteristics, genetic background, imaging parameters, and circu-

lating biomarkers further modulate HFimpEF trajectories [2,4].

From a pathophysiological perspective, emerging evidence implicates inflammatory pathways in myocardial injury, reverse remodelling, and ventricular functional recovery, potentially contributing to both sustained improvement and relapse [1,5]. Current evidence suggests the improvement in LVEF reflects reverse remodelling driven by neurohormonal blockade, hemodynamic unloading, improved myocardial energy, and recovery of stunned or hibernating myocardium [9]. However, persistent abnormalities at the molecular and cellular level, including fibrosis, inflammation, and altered excitation-contraction coupling, are likely to remain the basis for continued vulnerability to relapse [13]. Inflammatory pathways are increasingly recognised as potential contributors to both myocardial injury and recovery, influencing remodelling dynamics and long-term stability. Genetic predisposition, metabolic factors, and differences in myocardial viability further contribute to the heterogeneity of HFimpEF trajectories [4].

Together, these observations highlight the biological and clinical heterogeneity of HFimpEF and support ongoing investigation into advanced imaging techniques and circulating biomarkers to improve risk stratification, guide therapeutic de-escalation strategies, and justify decisions regarding the long-term need for implantable devices. A comprehensive understanding of these mechanisms remains a critical research priority.

Assessment and predictors of LVEF improvement

Across studies, non-ischemic etiologies of heart failure consistently demonstrate a greater potential for reverse remodelling compared with ischemic heart disease, likely due to a lower scar burden and greater myocardial viability, as increasingly characterised by advanced imaging techniques [2,4]. In this context, LVEF recovery may partly reflect the natural course of the underlying disease process rather than the effects of HF therapy alone. This is particularly relevant in inflammatory cardiomyopathy and acute myocarditis, where spontaneous recovery of ventricular systolic function has been reported [14], indicating that improvement may not always be

attributable to GDMT. Similarly, alcohol-induced cardiomyopathy represents a potentially reversible form of myocardial dysfunction, in which substantial LVEF recovery may occur after cessation or marked reduction of alcohol exposure [15]. These observations highlight the importance of considering disease-specific mechanisms when interpreting reverse remodelling in non-ischaemic HF.

In specific clinical settings, such as acute myocardial infarction, additional determinants, including smaller infarct size, lower peak cardiac biomarker levels, preserved diastolic function, and lower E/e' ratios, have been shown to predict subsequent systolic recovery, highlighting the prognostic importance of early diastolic assessment [16]. Advanced imaging markers, such as global longitudinal strain (GLS), provide additional prognostic information: GLS above the median is associated with lower rates of HF events and LVEF deterioration, whereas lower values indicate a higher risk of relapse [2,4]. Longitudinal echocardiographic trajectories further differentiate HFimpEF from persistent HFrEF. HFimpEF is characterised by early LVEF improvement, favourable changes in left ventricular geometry, improved right ventricular function (as reflected by increased TAPSE), and declining pulmonary pressures, consistent with reverse remodelling. In contrast, persistent HFrEF is associated with progressive adverse remodelling [4,10]. These findings reinforce the concept that LVEF is a dynamic parameter and that HF classification should be based on repeated imaging assessments rather than a single echocardiographic measurement to accurately assess recovery, anticipate relapse, and avoid inappropriate de-escalation of therapy.

Accurate diagnosis of HFimpEF is limited in clinical practice because echocardiographically measured LVEF varies with image quality, physiological fluctuations, and observer interpretation. The lack of rigorous longitudinal follow-up may lead to patient misclassification, categorising them as having heart failure with mildly reduced ejection fraction (HFmrEF) or preserved ejection fraction (HFpEF), rather than correctly recognising HFimpEF. Finally, improvements in LVEF may not always correlate with symptomatic improvement. Patients may experience symptomatic relief even without a significant change in LVEF [2].

In addition to echocardiography, cardiac magnetic resonance (CMR) is a valuable complementary modality for evaluating heart failure, particularly when echocardiographic data are limited or inconclusive. It enables accurate quantification of cardiac volumes, mass, and ejection fraction in both ventricles and provides a comprehensive assessment of systolic and diastolic function. Beyond functional evaluation, CMR offers superior tissue characterisation, enabling detection of myocardial fibrosis, inflammation, and injury through techniques such as late gadolinium enhancement and T1- and T2-mapping. These markers of adverse remodelling confer additional prognostic value in patients with HFimpEF. Although CMR has not been specifically established for longitudinal tracking of HFimpEF in most studies, it remains a valuable tool for defining aetiology, assessing scar burden, and evaluating disease progression [2].

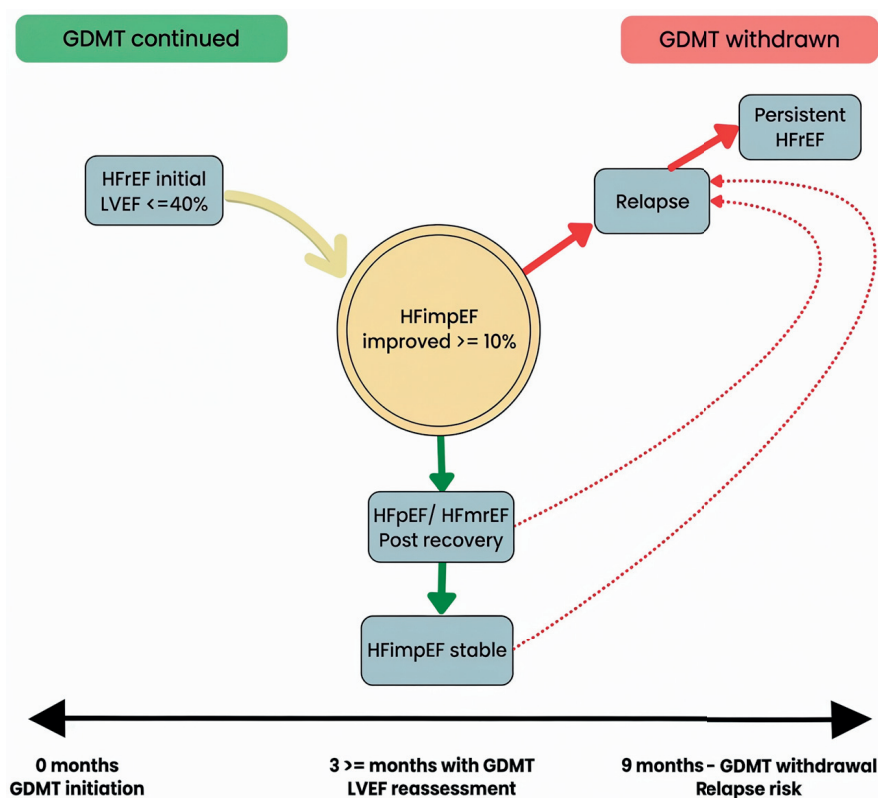
Clinical predictors of sustained improvement include younger age, female gender, non-ischemic aetiology, shorter HF duration, higher baseline systolic blood pressure, and absence of prior myocardial infarction or ICD implantation

[6,10,13]. Favourable imaging features such as smaller LV volumes, preserved LA function, and higher TAPSE and RV GLS further stratify risk, as do biomarker and metabolic profiles, including lower HbA1c, reduced insulin resistance, and favourable trends in BNP and troponin [4,7]. However, natriuretic peptide trajectories should always be interpreted in the appropriate clinical context because elevations may also reflect haemodynamic stress unrelated to LV reverse remodelling [17].

LVEF Trajectories

The growing number of patients with diagnosed HFimpEF has raised important clinical and scientific questions regarding long-term prognosis, risk of recurrent cardiovascular events, durability of LVEF recovery, and the optimal duration and intensity of GDMT.

Longitudinal analyses have identified distinct LVEF trajectories with important prognostic implications (see **Figure 1**). Patients with persistent HFimpEF have the most favourable prognosis



GDMT – guideline-directed medical treatment; HFimpEF heart failure with improved ejection fraction.

Figure 1. The influence of the GDMT on HFimpEF trajectory

sis. In contrast, transient HFimpEF, characterised by initial recovery and followed by recurrent systolic deterioration, is associated with a poor outcome, comparable to that of persistent HFrEF [1,9]. Notably, relapses often occur years after initial recovery, underscoring the need for long-term follow-up.

Beyond LVEF, advanced imaging parameters such as GLS and residual structural remodelling provide additional prognostic value, identifying patients at higher risk despite preserved LVEF [4]. In this context, improvement in LVEF represents functional remission rather than structural myocardial recovery, given the persistence of molecular, structural, and metabolic abnormalities that confer vulnerability to relapse [16]. It is therefore important to distinguish myocardial remission from true recovery, the latter representing a sustained reversal of the underlying myocardial disease and being uncommon.

In the ischemic context, a larger infarct size, reflected by elevated cardiac biomarker levels or by persistent ST-segment elevation, limits recovery and increases the risk of relapse. Consistently, biomarkers of congestion, metabolic stress, and myocardial injury (such as higher NT-proBNP) together with bilirubin, uric acid, and urea are associated with a lower probability of LVEF improvement, reflecting incomplete reverse remodelling [1,4,8,11]. Similarly, clinical factors such as male gender, the presence of atrial fibrillation or flutter, ischemic heart disease, previous myocardial infarction, persistent need for loop diuretics or implanted ICD are associated with a decline in LVEF after initial improvement. While early and sustained use of GDMT, favourable diastolic function ($E/e' < 15$), higher baseline LVEF, lower ventricular volumes, and higher HDL levels are associated with more sustained recovery.

Almost half of relapses occur beyond 24 months, highlighting the long-term vulnerability of this population [1]. Patients with transient HFimpEF experience approximately two-fold higher risks of all-cause mortality, cardiovascular death, and heart failure rehospitalisation compared with those with persistent HFimpEF, emphasising that maintenance of reverse remodelling is critical for long-term benefit. Finally, longitudinal trajectory analyses show that patients who die often exhibit an inverted, flattened U-shaped LVEF pattern, characterised by mod-

est initial improvement followed by deterioration, suggesting that dynamic LVEF trajectories may serve as early indicators of poor prognosis [1].

Therapeutic Considerations

A crucial clinical question in managing HFimpEF is whether GDMT can be safely withdrawn or de-escalated once LVEF improves. Although this strategy may appear attractive, current evidence strongly discourages routine treatment reduction [2,5]. Both randomised withdrawal trials and large observational cohorts consistently demonstrate high rates of relapse of LV dysfunction and clinical heart failure after treatment discontinuation, reinforcing the concept of HFimpEF as a state of remission rather than true myocardial recovery. In this context, the randomised TRED-HF trial, which included patients with previously diagnosed dilated cardiomyopathy (DCM) who had recovered LVEF and were asymptomatic on GDMT, showed relapse in approximately 44% of patients within six months following gradual withdrawal of treatment, with no relapses observed in those who continued therapy [18–20]. However, it is worth noting that TRED-HF was a small, open-label pilot study restricted to patients with recovered DCM, limiting its generalisability to the broader HFimpEF population. Moreover, evidence on treatment withdrawal remains limited and should be interpreted cautiously relative to the substantially larger randomised evidence base supporting GDMT.

Among GDMT components, renin–angiotensin system inhibition emerges as the most consistent modifiable factor associated with maintenance of LVEF improvement, while evidence on β -blockers is more heterogeneous, likely reflecting variability in dosage, adherence, and underlying disease severity. Importantly, GDMT confers a prognostic benefit regardless of ejection fraction trajectory, supporting its continued use even in patients with apparent systolic recovery [21].

Recent trials extend these findings to newer therapies. Sodium–glucose cotransporter 2 (SGLT-2) inhibitors and nonsteroidal mineralocorticoid receptor antagonists, such as finerenone, have demonstrated consistent reductions in cardiovascular events in HFimpEF populations, with greater absolute benefit attributed to their

higher baseline risk, as observed in the DELIVER and FINEARTS-HF trials [2,5,22-25]. In DELIVER, dapagliflozin reduced the composite of cardiovascular death or worsening heart failure in patients with previously reduced LVEF, with benefits comparable to those observed in the overall study population. Similarly, finerenone demonstrated consistent efficacy in patients with HFimpEF. In the FINEARTS-HF trial, finerenone reduced the rate of worsening heart failure events and cardiovascular death (RR = 0.84) in patients with HF and LVEF ≥ 40 , primarily by reducing heart failure events [23-25].

Taken together, these findings further reinforce the continuation of multidrug therapy as the foundation of long-term treatment and support the concept of HFimpEF as an active heart failure phenotype, given its consistent association with improved survival and fewer hospitalisations.

Decisions regarding implantable device therapy remain challenging. CRT promotes reverse remodelling and may facilitate LVEF recovery, whereas ICD provides a survival benefit independent of improvement in systolic function. Although the risk of arrhythmia decreases with LVEF recovery, it does not disappear. Therefore, an individualised assessment based on LVEF trajectory, myocardial substrate, and overall clinical context is required [26,27].

Applying HFimpEF criteria in patients following myocardial infarction provides additional prognostic value and supports early, intensive HF treatment, as sustained inhibition of the renin-angiotensin-aldosterone system has been shown to be the main modifiable factor associated with long-term preservation of LVEF in this group. Similar findings are observed in cancer therapy-related cardiac dysfunction (CTRCD), where treatment discontinuation is associated with poorer outcomes, underscoring the persistence of myocardial vulnerability despite apparent recovery [3,28,29].

In this context, clinical progression, residual risk, and individual patient characteristics should guide therapeutic decision-making, underscoring the need for a personalised and cautious approach. Despite limitations in the data regarding the intensity, adherence, and longitudinal changes in GDMT, current evidence consistently supports its indefinite continuation. Therefore, HFimpEF should be considered a chronic,

high-risk condition requiring lifelong therapy, close monitoring, and individualised multidisciplinary care, rather than a resolved disease state.

Knowledge Gaps and Future Research Directions

Despite rapid advances, several key questions remain unresolved. Reliable biomarkers and imaging tools to predict durable recovery versus relapse are lacking, and studies are needed on the long-term impact of emerging therapies and the optimal duration required to define persistent HFimpEF. In addition, residual myocardial dysfunction frequently persists despite normalisation of LVEF, confirming that this parameter alone is insufficient for risk stratification.

The existing HFimpEF literature has several important methodological limitations. Most published studies are retrospective and observational, often single-centre, which limits causal inference and generalizability. Selection bias is common, particularly because patients with serial echocardiographic follow-up are included, which may overestimate both prevalence and favourable prognosis. Furthermore, considerable heterogeneity in the timing and frequency of imaging assessments, which are often not protocol-based, makes it difficult to identify transient HFimpEF and detect early relapse. The near-exclusive reliance on LVEF for heart failure classification limits the ability to detect residual myocardial dysfunction. Assessment is primarily based on echocardiography, with limited incorporation of advanced techniques such as GLS or CMR, mainly due to limited availability. Consequently, the role of myocardial fibrosis and other tissue markers remains insufficiently characterised.

Another important limitation is the underrepresentation of contemporary therapies and the lack of systematic evaluation of treatment intensity, longitudinal changes, and adherence.

Finally, important knowledge gaps persist, including the absence of randomised clinical trials specifically designed for HFimpEF populations, a lack of clear strategies for therapeutic de-escalation, and limited evidence to guide device management after LVEF recovery. Moreover, the biological mechanisms distinguishing functional remission from true myocardial recovery remain

incompletely understood. Together, these limitations highlight the need for prospective, multicenter, longitudinal studies with deep phenotyping that integrate clinical data, advanced imaging, biomarkers, and genomic information to improve risk stratification and optimise therapeutic management in HFimpEF [2,5].

Conclusions

HFimpEF represents a significant and increasingly prevalent phenotype within the spectrum of heart failure, characterised by reverse remodeling and improved LV function but with persistent vulnerability to relapse. Sustained LVEF improvement provides a substantial survival benefit; however, deterioration remains common and has clinical consequences. Current evidence strongly supports lifelong continuation of comprehensive GDMT and close long-term monitoring. Future research should focus on trajectory-based risk stratification, mechanical understanding, and personalised therapeutic strategies to maximise the prognostic potential of myocardial recovery while minimising residual risk.

Statements

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CRedit authorship contribution statement

Teresa Fernandez Moreno: Writing – review & editing, Writing – original draft, Conceptualisation, Visualisation. Franciszek Kuklinski: Writing – review & editing, Conceptualisation, Visualisation. Malgorzata Knapp: Writing – review & editing, Conceptualisation. Karol Kaminski: Writing – review & editing, Conceptualisation. Jose A. Barrabes Riu: Writing – review & editing, Validation, Supervision, Conceptualisation. Agnieszka Tycinska: Writing – review & editing, Validation, Supervision, Conceptualisation.

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Data availability

No data sets were generated or analysed during the current study.

Conflict of interest statement

The authors declare no conflict of interest.

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