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REVIEW

Heart failure with recovered ejection fraction and the utility of defibrillator therapy: a review

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Abstract

Heart failure with recovered ejection fraction (HFrecEF) involves those who have previously had reduced cardiac function that has subsequently improved. However, there is not a single definition of this phenomenon and recovery of cardiac function in terms of left ventricular ejection fraction (LVEF) itself does not necessarily correlate with remission from the detrimental physiology of heart failure (HF) and its consequences. There is also the question of the utility of defibrillators in these patients, and whether they should be replaced at the time of battery depletion. To address this, several studies have shown specific predictors of ensuing LVEF recovery, including patient demographics, co-morbidities, and medication use, as well as predictors of ventricular arrhythmias (VA) following LVEF recovery. Recent studies have also shown novel imaging parameters that may aid in predicting which patients would have a higher risk of these arrhythmias. Additional data describe a small, yet appreciable risk of VA, in addition to appropriate shocks as well. In this review, we describe predictors of LVEF recovery, carefully analyse and characterize the continued risk for VA and appropriate shocks following LVEF recovery, and explore additional novel modalities that may aid in decision-making.

Keywords Heart failure; Recovered; Ejection fraction; Sudden cardiac death; Defibrillator

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Introduction

Heart failure (HF) is a complex clinical syndrome involving either an impairment with ventricular filling or ejection.[1] The current HF guidelines have defined three major categories including heart failure with reduced ejection fraction (HFrEF) with left ventricular ejection fraction (LVEF) $\leq$ 40%, heart failure with preserved ejection fraction (HFpEF) with LVEF $\geq$ 50%, and heart failure with mid-range ejection fraction (HFmrEF) with LVEF 41–49%.[2] However, there is a growing recognition of the concept of heart failure with recovered ejection fraction (HFrecEF) in the literature, as well as in the guidelines. This condition is ambiguously defined as those who have had previous HFrEF with subsequent improvement in their LVEF. This condition appears to truly be an emerging clinical entity with respect to its aetiologies, risk factors, consequences, and clinical outcomes.[1,3,4]

Implantable cardioverter-defibrillators (ICDs) certainly improve mortality in those with HFrEF, specifically with an LVEF $\leq$ 35%.[5] However, while risk reduction is clear in HFrEF, less is known about how to appropriately risk stratify the risk of sudden cardiac death (SCD) in HFrecEF. Some studies have shown a significant risk of ventricular arrhythmias (VA), such as ventricular tachycardia or fibrillation, with appropriate shocks in patients with HFrecEF, both before recovery and even as first-time events after recovery.[6–9] Hence, there is a knowledge gap with respect to the exact role of ICDs in the HFrecEF population.

In this review, we have sought to elucidate whether patients with HFrecEF have a significant persistent risk of VA and other adverse clinical outcomes. We will review the

current literature regarding the definition of HFrecEF, incidence, aetiology, and predictive factors as well as the risk of VA in this population. Finally, we will also review the data for or against continuing defibrillator therapy. We introduce the concept of utilizing imaging studies that could potentially assist in answering this question and give more information for having risk–benefit conversations with patients (*Figure 1*).

Definition and aetiologies

Literature defining and determining the outcomes of HFrecEF has grown considerably. As outlined previously, HFrecEF is defined as a clinical entity consisting of patients with prior HFrEF who have, over time, attained recovery of left ventricular (LV) function.[10] The term 'recovery', however, has been defined differently throughout the literature, with some studies defining recovered LVEF at ≥35%, while others at ≥40%, ≥45%, or even >50% (*Table 1*).[1,3,7,9] Some studies use improvement of LVEF by a per cent, such as 10% improvement, to define HFrecEF.[11] Another term used in some studies for the same clinical presentation of HFrecEF is heart failure with improved ejection fraction (HFiEF or HFimpEF).[12] Still, others have divided HFrecEF to partially recovered (LVEF 35–50%) and recovered (LVEF > 50%).[13] One novel paper proposed a universal definition that was accepted by various expert organizations, stated that HFimpEF involves a baseline LVEF of ≤40%, a ≥10 point increase from baseline LVEF, and a second measurement of LVEF of >40%.[14] Few studies had similar criteria to the proposed universal definition,[6,15] while others met only two criteria without defining a per cent change from baseline LVEF.[3,18,20,22]

Figure 1 Heart failure with recovered ejection fraction as a distinct clinical entity and continued benefit from defibrillator therapy. Heart failure with recovered ejection fraction is a clinical entity that is distinct from other types of heart failure with its unique biomarkers and outcomes. Patients start with having a low EF, defined at <40%, but then there is great heterogeneity in defining recovery. Certain patient demographics and co-morbidities have been identified as either positively or negatively predictive of recovery. It is a frequent occurrence for ICD to be placed when EF ≤ 35%, but it is unclear the benefit of reducing SCD when it is time to replace the generator in those with defibrillators with HFrecEF. There is mortality benefit from ICDs in HFrecEF, and new technology may be able to guide these decisions further. ACEi, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; BB, beta-blocker; BNP, brain natriuretic peptide; EF, ejection fraction; HFrecEF, heart failure with recovered ejection fraction; HFrEF, heart failure with reduced ejection fraction; ICD, implantable cardioverter-defibrillator; NYHA, New York Heart Association; SCD, sudden cardiac death.

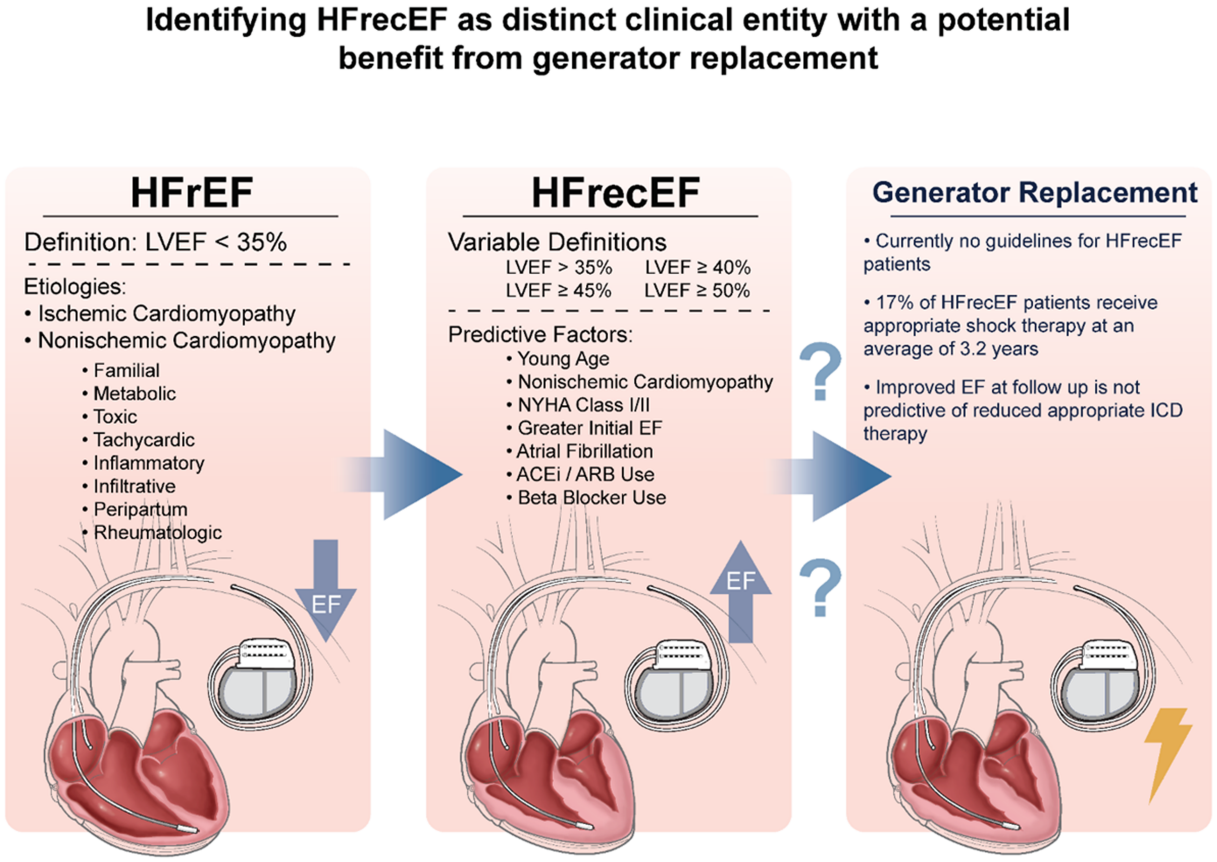


Table 1 Varied definitions of recovered ejection fraction

Source	Year of study	*n*	Definition of recovered LVEF
Madhavan *et al.*[7]	2016	253	>35%
Naksuk *et al.*[6]	2013	91	>35%
Berthelot-Richer *et al.*[32]	2016	944	>35%
Adabag *et al.*[35]	2017	1273	>35%
Punnoose *et al.*[3]	2011	358	≥40%
Kalogeropoulos *et al.*[18]	2016	2166	≥40%
Agra Bermejo *et al.*[20]	2018	449	>40%
Lupón *et al.*[21]	2017	1057	>40%
Park *et al.*[22]	2019	5625	>40%
Nadruz *et al.*[15]	2016	286	>40%
Basuray *et al.*[1]	2014	1821	≥50%
[a]Thomas *et al.*[13]	2018	26 355	≥50%

LVEF, left ventricular ejection fraction.
[a]Thomas *et al.* defined recovered LVEF as ≥50% and 'partially recovered' LVEF as ≥35%.

The aetiology of HF varies considerably, including ischaemic cardiomyopathy (ICM) and non-ischaemic cardiomyopathy (NICM), such as hypertensive, diabetic, and valvular heart disease (*Figure 2*).[16] It is known that some causes of HF, such as stress-induced (Takotsubo), peri-partum cardiomyopathy, or thyroid disease, often have LVEF recovery without medical therapy besides treatment of the underlying condition.[17] Therefore, the incidence of HFrecEF varies by the aetiology.

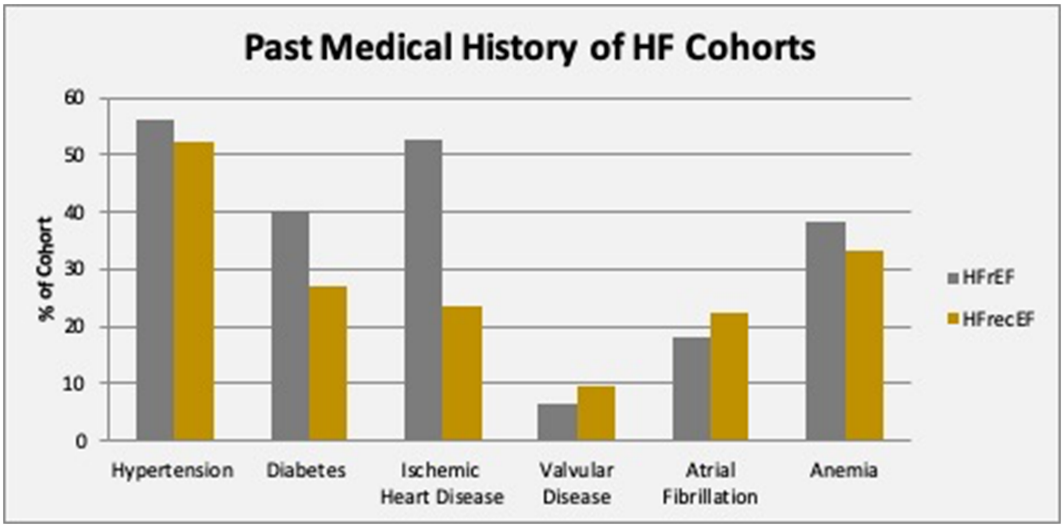
Incidence and predictive factors

As a consequence of the varying definitions and aetiologies of HFrecEF, there is a wide range of reported incidence. In a meta-analysis of patients with cardiac resynchronization therapy (CRT), the reported incidence of LVEF recovery ranged anywhere from 7% to 86%.[9] However, in the specific study in question, 7% of the participants recovered to LVEF > 50% and 86% were >35%.[8] In studies with more modest definitions of HFrecEF, the incidence of recovery appeared to have a bit of a narrower range, from 14% to 33%.[3,9,18]

In a meta-analysis of studies of HFrecEF, the frequency of LVEF improvement ranged from 9% to 72%.[19] Definitions ranged from LVEF >50/45/40% or ejection fraction (EF) increase by >10%. Of note, the studies that reported higher incidences of HFrecEF consisted of patients with Takotsubo cardiomyopathy and post-partum cardiomyopathy. On the contrary, the studies with lower incidence rates had chronic HFrEF previously.

There is a growing wealth of data regarding predictors of HFrecEF. Several studies have demonstrated commonalities among those who tend to have LVEF recovery, which may help predict who will have recovery. The CARDIOCHUS-CHOP registry is one such study that determined certain predictors of LVEF recovery, which included younger age, lower New York Heart Association (NYHA) functional class, treatment with renin-angiotensin-aldosterone system inhibitors and beta-blockers (BB), absence of defibrillator, and non-ischaemic aetiology.[20] Other studies reaffirmed the aforementioned findings and, in addition, found that their HFrecEF group was less likely to have left bundle branch block (LBBB) on electrocardiogram, more likely to have atrial fibrillation, and had a slightly higher LVEF at time of HF

Figure 2 Differences between heart failure with reduced ejection fraction and heart failure with recovered ejection fraction in select baseline characteristics. Three sources, Agra Bermejo *et al.*, Lupón *et al.*, and Park *et al.*, provided the past medical history of patients in different heart failure cohorts. The conditions that all three papers included were combined to produce average per cent representation of the specific demographics provided in the figure. HF, heart failure; HFrecEF, heart failure with recovered ejection fraction; HFrEF, heart failure with reduced ejection fraction; LVEF, left ventricular ejection fraction.



diagnosis.[21] Furthermore, it appears that patients with overall fewer co-morbidities tend to have a higher incidence of recovered LVEF.[22]

Some factors that may predict worsening LV function after recovery are older age, longer duration from initial presentation to recovery time, presence of diabetes, and increased LV end-diastolic dimension (LVEDD) at initial presentation.[23] Additionally, ICM portends a poorer prognosis and less likelihood of recovering systolic function.[20] One score specifically created to predict reverse remodelling of LVEF recovery is the ST2-R2 score. It utilizes the biomarker soluble ST2 (which was an independent predictor for reverse remodelling) along with five clinical variables (non-ischaemic aetiology, absence of LBBB, HF duration, baseline LVEF, and BB use) to predict worsening LVEF.[24] This was validated in a multicentre study as well.[25]

Heart failure with recovered ejection fraction as an emerging clinical entity

When analysing HFrecEF patients, it is found that they have a distinct biomarker profile and functional characteristics from both HFrEF and HFpEF patients. A study reported that patients with HFrecEF with abnormal global longitudinal strain (GLS) on routine echocardiography had significantly worse outcomes than those with normal GLS, even with recovered LVEF.[26] Similarly, a study demonstrated that a significant amount of HFrecEF patients had abnormal brain natriuretic peptide (BNP) levels and about half had detectable troponin levels at baseline. These same patients continued to experience HF symptoms and hospitalizations for exacerbations, at about the same rate as HFpEF patients.[1] Therefore, it is thought that recovery of EF does not indicate resolution of HF, and these patients may continue to experience oxidative stress, cardiomyocyte injury, and neurohormonal activation leading to clinical disease. From a clinical perspective, while the LVEF may improve, it does not negate the need for close surveillance and monitoring, as the myocardium may still have a pathogenic substrate. The risk of arrhythmias due to underlying scar and myocardial remodelling still exists.

This is consistent with the findings in an open-label, randomized trial that examined the effect of discontinuation of pharmacologic treatment in patients with dilated cardiomyopathy with recovered LVEF. Even though at the beginning of the study the patients had an LVEF $\geq$ 50% along with normalized LV end-diastolic volume and BNP, in the first 6 months of the trial, 44% of patients relapsed and required restarting medications.[27] Additionally, data from Lupón *et al*. showed that the clinical course of HF patients with reduced ejection varied according to aetiology, duration, and patient gender. Patients with ischaemic HF had improvement in their LVEF within 1 year of diagnosis, which was followed by a plateau for approximately 10 years and then declined gradually afterwards.[28]

An analysis of the CARDIOCHUS-CHOP registry demonstrated that patients with HFrecEF had a better overall prognosis and outcomes than both HFrEF and HFpEF patients, but once again, the overall survival was worse than healthy individuals.[20] Similar results were found in a cohort of 1057 HF patients; all-cause mortality, cardiovascular-related mortality, HF hospitalization, HF-related mortality, and sudden death were significantly lower in the HFrecEF group than HFrEF and HFpEF, but higher than healthy controls.[21]

Another retrospective study with 1821 patients illustrated that nearly 20% of patients with HFrecEF (LVEF $\geq$ 50%) suffered from death, cardiac transplantation, or ventricular assist device (VAD) placement by 8 years of follow-up, even when data were adjusted for clinical and demographic criteria.[1] Therefore, these patients continued to have clinical and physiologic characteristics of HF and required continued therapy.

This compiled information is evidence that HFrecEF patients are different than HFrEF, HFpEF, and healthy patients with respect to demographics, biomarkers, symptoms, hospitalizations, and prognosis. Thus, it is necessary to have a novel algorithm for the management of this separate entity. The Journal of the American College of Cardiology (JACC) Scientific Expert Panel recently published initial guidelines for defining, diagnosing, and managing patients with HFrecEF.[29] The focus consisted of a newly proposed definition, continuation of guideline-directed medical therapy, and close follow-up. A topic that is briefly discussed in that review is the controversy of VA risk in HFrecEF patients and the use of defibrillators in this patient population, which we will address moving forward.

Left ventricular ejection fraction recovery while implantable cardioverter-defibrillator present

While it is appropriate for ICDs to be placed in those with an EF of $\leq$35% for primary prevention,[30] a dilemma arises when one is faced with patients with recovered EF. A recent retrospective analysis of the American College of Cardiology's National Cardiovascular Data Registry examined patients with ICDs for primary prevention at the time of generator change.[13] The study showed that among 26 355 patients undergoing replacement, 17.4% had partially recovered EF ($>$35% and $\leq$50%) and 7.3% had recovered EF ($>$50%). About 42.5% of patients with recovered EF had a dual-chamber ICD, and 40.3% had a CRT-D. Another multicentre trial evaluated 253 patients who had ICDs placed for primary prevention.[7] They showed that, at the time of generator change, EF had recovered to $>$35% in 28% of the patients. Likewise, another

study demonstrated that 27% of the patients who had ICD for primary prevention had recovered EF to >35% at the time of generator change.[6]

Therefore, it is evident that LVEF recovery occurs in a major proportion of patients who had an ICD placed for primary prevention. The outcomes, including risk of VA and SCD, impact the decision to replace the generator at the time of the battery expiring.

Ventricular arrhythmias in heart failure with recovered ejection fraction patients

While limited, there are data characterizing the risk of VA (ventricular tachycardia/fibrillation) and appropriate ICD shocks in patients with recovered LVEF. In retrospective studies, as many as 2–36% of patients with recovered EF (>35%) receive appropriate ICD shocks following generator exchange (*Table 2*).[6,7,31–34] Of the aforementioned studies, one demonstrated that 36% of the patients with HFrecEF (EF > 35%) had appropriate ICD shocks, which was statistically similar to the HFrEF group both before and after generator replacement. Of these patients with HFrecEF, 12% had their first appropriate ICD shock after generator replacement.[6]

Another study that analysed a primarily ischaemic population displayed that although patients with HFrEF had a higher incidence per year of VA after generator change than those with HFrecEF, patients with HFrecEF still continued to have an appreciable 5% risk of VA per year and the overall mortality was similar between the two groups.[7] The rate of appropriate ICD shock therapy following generator change for HFrecEF patients at 3 years was 14%. To compare, 27% of the entire cohort of patients, regardless of LVEF, experienced appropriate ICD therapy at 3 years after generator change.

A sub-analysis from the SCD-HeFT (Sudden Cardiac Death in Heart Failure Trial) supports the continued use of ICD in HFrecEF patients. The study randomized patients with NYHA Class II–III HF with EF ≤ 35% on goal-directed medical therapy to ICD vs. placebo. The mortality benefit of ICD was compared between patients with HFrEF and HFrecEF, and the study found a similar relative reduction in all-cause mortality with the ICD.[5,35]

Another distinct population is patients with HFrecEF who benefited from CRT initiation. The MADIT-CRT (Multicenter Automatic Defibrillator Implantation Trial with Cardiac Resynchronization Therapy) was a prospective, multicentre randomized control trial of 752 patients with CRT. Compared with HFrEF patients, it showed that patients with HFrecEF (with EF > 50% or super-responders) had the risk of VA reduced by 76%, while patients with HFrecEF (EF 36–50%) had the risk of VA decreased by 66% over 2 years.[8] Factors associated with decreased risk of VA were female gender, body mass index < 30 kg/m^2, LBBB, cessation of smoking tobacco products, and no previous VA. Another sub-analysis of the MADIT-CRT similarly showed a low incidence of VA in HFrecEF super-responders, but still had a 12% risk of them in the 2 years following initiation of CRT.[8,36] Therefore, it is low, but arrhythmic risks persist. Some patients may benefit from the continuation of defibrillator therapy more than others according to the aetiology of cardiomyopathy. Another study with an overall rate of VA in 4.4 years of 25% found that patients with NICM and HFrecEF had a low risk of VA (90% risk reduction), but the ICM cohorts did not.[32]

There are important limitations in the aforementioned studies as some of them were retrospective and lacked power. However, overall, the data appear to demonstrate that there is a significant incidence of VA and risk of SCD in patients regardless of LVEF recovery in many patients.

Implantable cardioverter-defibrillator therapy risks

Implantable cardioverter-defibrillators are not without significant risks. Device placement and exchange can expose the patient to peri-procedural and post-procedural complications such as myocardial perforation, device infection, device malfunction, and even death. In the analysis from the REPLACE registry, 8.5% of patients experienced a major device-related complication by 6 months following device

Table 2 Appropriate implantable cardioverter-defibrillator shocks following generator change in patients with left ventricular ejection fraction > 35%

Source	Year	Generator exchange, *n*	ICD shocks following generator exchange and recovered LVEF, *n* (%)	Years to follow-up
Naksuk *et al*.[6]	2016	25	5 (20)	6.2 ± 2.2
[a]Madhavan *et al*.[7]	2014	71	24 (14)	3
Kini *et al*.[31]	2016	59	5 (8.5)	3.5 ± 2.0
[b]Schliamser *et al*.[33]	2017	70	4 (5.7)	2

ICD, implantable cardioverter-defibrillator; LVEF, left ventricular ejection fraction.
[a]Madhavan *et al*. found appropriate ICD shocks at 1, 2, and 3 year intervals in 7%, 9%, and 14% of cumulative patients, respectively.
[b]Schliamser *et al*. found that 17.3% of patients with 'improved' ejection fraction received appropriate defibrillator therapy, while only specifying that 5.7% had an ejection fraction of >35% at the time of shock therapy.

exchange.[37] In the highest risk group, such as those undergoing lead extraction, peri-procedural mortality was as high as 1.1%. Despite this, concerns for peri-procedural events were dispelled for those with HFrecEF in a large retrospective study where these events were noted to be rare (<1%).[13] However, ICD shocks may have a significant impact on patients' well-being and overall quality of life. In a study not specific to HFrecEF, it is estimated that 25% of ICD shocks are inappropriate and result in increased anxiety/depression and led to strict driving restrictions, overall reducing quality of life.[38] Therefore, when having conversations about replacing the ICD in HFrecEF, encouraging clear communication and ensuring shared-decision making will allow for an informed decision.

The role of magnetic resonance imaging

In order to provide more clarity to the problem, one consideration is to analyse alternative variables in addition to LVEF to further estimate arrhythmogenic risk, especially in those with primary prevention devices and favourable long-term prognosis at the time of generator exchange. One possible method may be via imaging modalities such as cardiac magnetic resonance imaging (CMR); advances in CMR now allow the ability to quantify and qualify myocardial scar. The GAUDI-CRT study used contrast-enhanced cardiac magnetic resonance imaging (ce-CMR) to evaluate scar size and heterogeneity to predict appropriate ICD therapies in patients receiving primary prevention CRT.[39] In this study, patients underwent ce-CMR prior to CRT implantation to measure scar mass, border zone, and border channel mass. Patients were then prospectively monitored for appropriate ICD therapy. The investigators found that increased scar parameters were, in fact, associated with appropriate ICD therapy. Scar mass < 10 g was associated with a 100% negative predictive value for appropriate ICD therapy.

Studies utilizing late gadolinium enhancement (LGE) CMR found that larger scar border zone was associated with VA[40] and larger transmural scar extent scar mass was significantly associated with appropriate ICD therapy after implantation.[41] Another prospective longitudinal study of 472 patients found that the presence and extent of mid-wall fibrosis detected by LGE CMR in dilated cardiomyopathy were both independently associated with an increased risk of VA and mortality.[19]

A unique study utilizing LGE CMR introduced entropy as a novel parameter.[42] Traditionally, areas of scar have been operator dependent and are therefore dependent on predefined thresholds of signal intensity. Rather than self-defining these zones, the authors evaluated the heterogeneity of scar by characterizing the overall variation of signal intensity across the scar. Entropy of 154 patients with myocardial infarction was studied before undergoing primary or secondary prevention ICD implantation, which found a statistically significant association with VA. Therefore, it has been shown by CMR that greater scar mass, increased scar heterogeneity/entropy, higher border zone channel mass, and increased myocardial wall fibrosis may all be associated with increased risk of VA. Of note, this literature is prior to ICD implantation; however, one could consider its utilization in a patient after ICD use when analysing risk of VA.

The role of echocardiography

In addition to CMR, several echocardiographic parameters have been studied to predict arrhythmogenic risk. A recent retrospective study evaluated the association between relative wall thickness (RWT) and VA within the MADIT-CRT population.[43] In the 1260 patients studied with LVEF < 30% and LBBB, the RWT was found to be the most powerful echocardiographic measure to predict VA. Notably, a low RWT < 0.24 was associated with an 83% increased risk of VA. Likewise, every 0.01 unit decrease of RWT was associated with a 12% increase in VA. Implantation of a CRT device was associated with increased RWT over time, and in this population, every 10% increase in RWT demonstrated a 34% and 36% reduction in subsequent VA and SCD, respectively.

In addition to these echocardiographic parameters, some prospective studies have also evaluated mechanical dispersion by strain echocardiography (an indicator of contraction heterogeneity) as a predictor of VA.[44,45] One group of investigators evaluated mechanical dispersion and GLS in patients post-myocardial infarction.[45] Another group evaluated patients undergoing CRT placement with reduced LVEF. Both studies showed that increased mechanical dispersion increased the risk of VA independent of LVEF.[44]

Therefore, assessment of RWT and mechanical dispersion via echocardiography, which is often already done to assess LVEF, may be a cost-effective and low-risk method to give additional information when assessing the risk of VA and if ICD batteries should be replaced in HFrecEF.

The role of biomarkers

While magnetic resonance imaging (MRI) and echocardiograms may help identify patients at risk for VA, the combination of advanced imaging and novel biomarkers may allow for a more granular stratification of these at-risk individuals. In a seminal paper, it was found that ST2 concentrations were predictive of SCD in patients with chronic HF. Notably ele-

vated ST2 levels increased the odds of SCD by nearly 40%. Hence, these data provide complementary information to BNP and N-terminal pro-brain natriuretic peptide (NT-proBNP) levels, which are standard of care in HF patients.[46] Additional studies demonstrated that ST2 in combination with galectin-3 (Gal-3) could further refine risk classification for SCD.[47] And finally, Gal-3 and sST2 have been identified as the fibrosis biomarkers and thus can serve as a powerful risk stratification tool when combined with novel MRI imaging protocols that utilize gadolinium to identify and quantify fibrosis. These biomarkers can enhance the risk stratification process by detecting fibrosis at an early stage[48] and could improve long-term outcomes in HF patients. While LVEF may improve in HFrecEF, it may be more reassuring if normalization of these novel biomarkers is also observed.

How to have patient-centred conversations

Tying together this information next leads to how to have risk–benefit conversations with patients with HFrecEF whose ICD battery is expiring. Given the improvement in patient outcomes with shared decision-making,[49] it is favourable as a healthcare provider to present the information known to us and make a plan that best fits each individual patient, particularly those who met the initial criteria for a primary prevention ICD. For example, in the first scenario, a patient with HFrecEF has a history of known VA and recent appropriate ICD shocks. In this case, in addition to continued medical management, it would seem most clear that replacement of the ICD battery would have significant benefit to the patient given the risk for recurrent arrhythmias and reducing SCD.

In another case, a patient presents with HFrecEF with an EF $>$ 50% and low-risk features for VA, such as NICM, and has no factors associated with re-worsening of LV function, such as older age, longer duration from initial presentation to recovery time, diabetes, and increased LVEDD at initial presentation.[23] In this second scenario, especially if this patient was experiencing decreased quality of life from their ICD, such as severe anxiety, inappropriate shocks, or had a complication from their current ICD, it may be in their best interest not to replace the battery, particularly if the device was implanted for primary prevention. Risk stratification of patients into low-risk, moderate-risk, and high-risk groups would be beneficial for individual decision-making. Stratification could be based on a combination of HF aetiology, imaging, and biomarkers. For example, stress cardiomyopathy with normal biomarkers and no LGE would be low risk; however, ICM, elevated ST2, and $>$15% LGE would be high risk. A previous history of VT would also be high risk.

However, for most patients with HFrecEF, there may be a mixed picture. They may have borderline improved LVEF, some factors that increase risk of worsening function of EF, or had minor complications from their ICD. By ensuring appropriate communication and discussing the risks and benefits, the patient and provider will be able to come to a conclusion together. In this case, additional imaging could be considered, such as CMR to assess scar mass, entropy, border zone mass, or wall fibrosis, and echocardiogram to assess RWT and mechanical dispersion, for additional information. This approach emphasizes gathering sufficient data to support patient-centred decision-making.

Conclusions

Heart failure with recovered ejection fraction is a medical condition with unique predictive factors, biometric markers, and clinical consequences from HFrEF and HFpEF. It is essential to have specific guidelines on how clinicians should address the management of HFrecEF. One question revolves around the replacement of ICD batteries and risk of SCD in these patients. Although the data present in the literature have its limitations, such as the varying definitions, aetiologies, and reported incidence of HFrecEF in different studies, as well as the retrospective nature of these studies, there is an appreciable risk of VA in patients with HFrecEF; 2–36% of patients with recovered EF receive appropriate ICD shocks following generator exchange.[6,7,31–33] Those with a recovered LVEF that is $<$50% and ischaemic aetiology of HF may be at highest risk for SCD. To better decide if an ICD battery should be exchanged in HFrecEF patients, standardization of the HFrecEF definition and a prospective study categorizing HFrecEF patients according to the aetiology of their cardiomyopathy and risk of VA is needed.

Imaging modalities such as CMR and echocardiography show promise in helping to stratify arrhythmogenic substrate in HFrecEF. Both assessment of scar by CMR as well as LV geometry by echocardiography may aid in identifying patients at high risk for SCD. CMR is growing in availability and echocardiography is widely accessible; thus, both can prove to be useful in providing additional information to guide the question of ICD exchange in HFrecEF. We have made great strides to start to answer this question; however, much work is yet to be done. In the future, we hope to have stronger evidence and promising adjuvant decision-making parameters that can help guide therapy and ensure safety, with or without a defibrillator.

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