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Review Article

Predictors of major adverse cardiac events in post-myocardial infarction patients with heart failure with reduced ejection fraction

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Abstract

Background: Patients who survive myocardial infarction but develop heart failure with reduced ejection fraction remain at substantial risk for recurrent major adverse cardiac events (MACE), despite advances in reperfusion and guideline-directed therapy.

Objective: This review synthesizes evidence on clinical, biochemical, imaging, and genetic predictors of MACE in post-myocardial infarction heart failure with reduced ejection fraction, and how these predictors inform risk stratification and treatment.

Methods: We reviewed cohort studies, randomized trial substudies, and meta-analyses addressing ejection fraction trajectory, natriuretic peptide and troponin trends, cardiac magnetic resonance infarct characteristics, renal function, arrhythmic markers, and risk-modifying therapy in this population.

Findings: Residual ejection fraction below 35%, rising NT-proBNP, larger infarct size and microvascular obstruction on cardiac magnetic resonance, reduced glomerular filtration rate, and nonsustained ventricular arrhythmia each independently predict MACE, while sacubitril-valsartan, mineralocorticoid receptor antagonists, SGLT2 inhibitors, and resynchronization or defibrillator therapy attenuate this risk.

Conclusions: Integrating clinical, biomarker, and imaging predictors into a longitudinal framework, rather than ejection fraction alone, offers the most promising path toward individualized surveillance and therapy intensification in this population.

Keywords: myocardial infarction; heart failure; ejection fraction; major adverse cardiac events; risk prediction

1. Introduction

Heart failure with reduced ejection fraction (HFrEF) complicates roughly one in five hospitalizations for acute myocardial infarction, turning an otherwise successfully reperfused event into a chronic disease trajectory with substantially elevated risk for death, recurrent infarction, and rehospitalization [1]. The fourth universal definition of myocardial infarction anchors diagnosis in a rise and fall of cardiac troponin with supporting evidence of ischemia, and it is this biomarker-confirmed necrosis, when large or poorly reperfused, that sets the stage for the adverse remodeling from which post-infarction HFrEF emerges [2]. Contemporary registries describe mortality after incident heart failure complicating infarction declining only modestly despite widespread primary percutaneous coronary intervention and guideline-directed therapy, leaving major adverse cardiac events (MACE) -- cardiovascular death, recurrent infarction, and heart failure hospitalization -- as the dominant unmet outcome [3].

Risk in post-infarction HFrEF is not distributed uniformly. Left ventricular ejection fraction (LVEF) alone, while foundational to trial eligibility and guideline thresholds, has repeatedly proven an imprecise discriminator of individual risk, prompting three decades of investigation into complementary clinical, biochemical, imaging, electrophysiological, and genetic predictors [4]. This review synthesizes that evidence and considers how each predictor domain intersects with therapies now available to modify the risk it identifies.

2. Epidemiology and clinical burden

Population-based surveillance from Olmsted County shows that while age- and sex-adjusted heart failure incidence declined over a decade, survival after diagnosis improved only modestly, leaving the absolute burden of heart-failure-related death and hospitalization substantial [5]. A companion community cohort focused specifically on heart failure after myocardial infarction found

markedly higher subsequent mortality than infarction without heart failure, a risk that persisted throughout follow-up rather than being confined to the peri-infarction window [3]. Roger's contemporary synthesis situates these findings within population aging and improved acute-event survival, which together increase the prevalent pool of patients living with post-infarction ventricular dysfunction even as age-specific incidence has flattened [6]. European STEMI guidelines increasingly emphasize systematic pre-discharge assessment of left ventricular function, recognizing the interval between infarction and identification of reduced ejection fraction as a critical window for secondary prevention and early heart failure therapy [7].

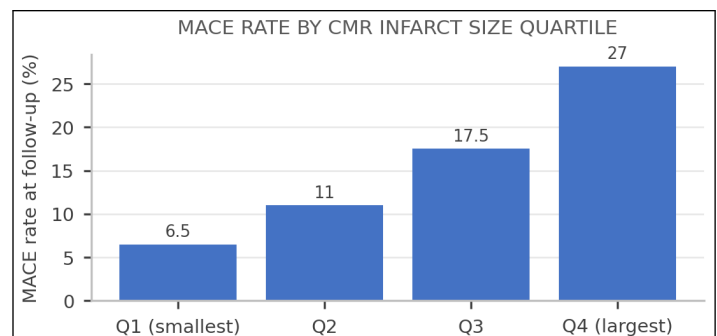


Figure 1: Major adverse cardiac event rate at follow-up by cardiac magnetic resonance infarct size quartile, drawn from patient-level pooled analysis across ten randomized primary percutaneous coronary intervention trials [16].

3. Left ventricular function and remodeling

Pooled analysis from the CHARM program demonstrated a graded relationship between lower ejection fraction and higher cardiovascular death and heart failure hospitalization, with risk not plateauing above the conventional 40% threshold [4]. This underlies the emphasis on serial rather than single-timepoint ejection fraction assessment, since many patients with early post-infarction LVEF below 40% recover function over subsequent months while others decline despite optimal therapy, and trajectory carries more prognostic information than any single measurement [8]. Adverse

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remodeling -- progressive dilation and sphericity independent of further ejection fraction decline -- is mechanistically distinct from ejection fraction itself; patients who fail to show reverse remodeling by three to six months carry substantially higher subsequent MACE risk [8]. The VALIANT trial, enrolling over 14,000 patients with left ventricular dysfunction, heart failure, or both after myocardial infarction, confirmed that lower ejection fraction and more extensive wall motion abnormality independently predicted the composite of death, reinfarction, and heart failure hospitalization [9].

4. Biomarkers: natriuretic peptides and troponin

De Lemos and colleagues established B-type natriuretic peptide (BNP) as an independent prognostic marker across acute coronary syndromes and chronic heart failure, with higher concentrations tracking worse outcomes regardless of ejection fraction [10]. In the PEACE trial substudy of stable coronary artery disease, Omland and colleagues found BNP and NT-proBNP predicted cardiovascular death and heart failure hospitalization even in ambulatory patients without overt heart failure, after adjustment for ejection fraction [11]. The trajectory of NT-proBNP after treatment initiation adds further value: Januzzi and colleagues showed that early NT-proBNP reduction after starting sacubitril-valsartan correlated with subsequent improvement in ventricular structure, while patients without meaningful biomarker decline more often showed adverse remodeling on follow-up imaging [12]. Consensus guidance from the Heart Failure Association of the ESC formalizes age-, renal-function-, and body-mass-index-adjusted interpretation of these concentrations [13].

5. Cardiac MRI: infarct size and microvascular obstruction

Cardiac magnetic resonance (CMR) is the reference standard for quantifying infarct size and identifying microvascular injury that persists after successful

reperfusion. Eitel and colleagues showed that myocardial salvage index and the extent of microvascular obstruction each independently predicted MACE, with low salvage plus extensive obstruction identifying a high-risk subgroup despite comparable ejection fraction to lower-risk patients [14]. A pooled individual-patient-data analysis across seven randomized reperfusion trials confirmed microvascular obstruction independently predicted mortality, heart failure hospitalization, and reinfarction, additive to infarct size itself [15]. Stone and colleagues' patient-level pooled analysis across ten primary PCI trials, encompassing more than 2,600 patients, found infarct size above roughly 15-20% of left ventricular mass identified a markedly higher-risk stratum independent of resulting ejection fraction [16].

6. Renal function and comorbidity

Renal impairment is among the most consistent independent predictors of adverse outcome across the heart failure literature. Hillege and colleagues showed estimated glomerular filtration rate was a stronger independent predictor of mortality in chronic heart failure than either ejection fraction or functional class [17]. Damman and colleagues' updated meta-analysis confirmed that both baseline renal impairment and worsening renal function during treatment independently predicted mortality [18]. This cardiorenal interaction is bidirectional and clinically consequential: reduced cardiac output and congestion impair renal perfusion, while renal impairment limits tolerated doses of angiotensin-neprilysin inhibition, mineralocorticoid receptor antagonism, and diuretics -- the very treatments shown to modify MACE risk. Structured outpatient intravenous diuretic protocols have been developed specifically to navigate this constraint [19].

7. Arrhythmic risk and electrical instability

Sudden cardiac death from ventricular arrhythmia accounts for a substantial proportion of MACE in post-infarction HFrEF. Huikuri and colleagues found that

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reduced heart rate variability, abnormal heart rate turbulence, and microvolt T-wave alternans added incremental predictive value for sudden death beyond ejection fraction alone, particularly in patients not meeting conventional defibrillator eligibility [20]. The earlier MUSTT trial established that inducible sustained ventricular tachyarrhythmia identified markedly elevated arrhythmic-death risk, providing mechanistic grounding for the later shift toward broader, ejection-fraction-based defibrillator eligibility that superseded electrophysiological testing in routine practice [21]. Analysis of sudden death timing within the VALIANT cohort showed hazard was highest in the first thirty days after infarction complicated by left ventricular dysfunction or heart failure, declining gradually thereafter, informing timing conventions for deferred defibrillator implantation [22].

Table 1: Independent predictor domains for MACE in post-myocardial infarction heart failure with reduced ejection fraction.		
Domain	Representative marker	Key evidence
Ventricular function/remodeling	LVEF trajectory, reverse remodeling	[4,8]
Natriuretic peptide biomarkers	NT-proBNP level and trend	[10,11,12]
CMR infarct characterization	Infarct size, microvascular obstruction	[14,15,16]
Renal function	Estimated glomerular filtration rate	[17,18]
Electrical instability	Heart rate variability, inducible VT	[20,21]

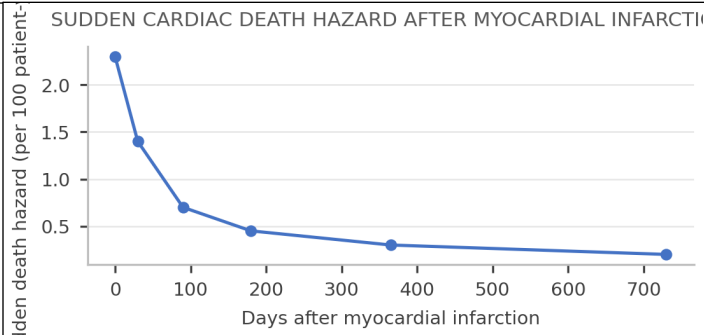


Figure 2: Cumulative sudden cardiac death hazard by interval after myocardial infarction complicated by left ventricular dysfunction or heart failure, showing the highest hazard within the first 30 days and gradual decline thereafter [22].

8. Pharmacologic and device-based risk modification

Each predictor above maps to a therapy capable of modifying the risk it identifies. Angiotensin-neprilysin inhibition, superior to enalapril in chronic HFrEF in PARADIGM-HF, was tested directly after acute myocardial infarction in PARADISE-MI; although the primary outcome narrowly missed significance, benefit concentrated among patients with the highest-risk features reviewed above [23,24]. A related trial in acutely decompensated heart failure, including a substantial post-infarction subgroup, showed early angiotensin-neprilysin inhibition produced greater natriuretic peptide reduction than enalapril [25]. EPHEsus randomized patients with left ventricular dysfunction and heart failure or diabetes after infarction to eplerenone, finding reduced all-cause mortality and cardiovascular hospitalization, most pronounced at the lowest ejection fractions [26], building on the mortality benefit of spironolactone established by RALES in severe chronic heart failure [27]. DAPA-HF and EMPEROR-Reduced each showed sodium-glucose cotransporter-2 inhibitors reduced cardiovascular death and heart failure hospitalization across trial-eligible ejection fractions, with a pooled meta-analysis confirming consistent benefit including in patients with recent myocardial infarction [28,29,30]. MADIT-II established that prophylactic defibrillator implantation reduced

mortality in post-infarction patients with ejection fraction at or below 30% [31], and SCD-HeFT extended this benefit to a broader heart failure population [32]. Cardiac resynchronization therapy, validated in CARE-HF and MADIT-CRT, further reduces hospitalization and mortality in patients with wide QRS and reduced ejection fraction [33,34]. Beta-blockade with carvedilol in post-infarction left ventricular dysfunction, established by CAPRICORN, remains foundational background therapy underlying each of these interventions [35].

Table 2: Landmark trials of pharmacologic and device-based therapy targeting MACE risk in post-myocardial infarction heart failure with reduced ejection fraction.			
Therapy	Trial	Population	Key outcome
Angiotensin-neprilysin inhibition	[23]	Post-MI with reduced EF or pulmonary congestion	Trend toward reduced CV death/HF hospitalization
Mineralocorticoid receptor antagonist	[26]	Post-MI, LV dysfunction + HF or diabetes	Reduced all-cause mortality and CV hospitalization
SGLT2 inhibitor	[28,29]	Chronic HFrEF, including recent MI	Reduced CV death/HF hospitalization
Implantable defibrillator	[31]	Post-MI, EF ≤30%	Reduced all-cause mortality
Cardiac resynchronization therapy	[33,34]	Reduced EF with wide QRS	Reduced HF hospitalization and mortality

9. Genetic and integrated risk prediction

Genome-wide association studies have identified dozens of loci influencing atherosclerotic burden and plaque instability, and polygenic risk scores are increasingly explored as an adjunct to conventional predictors, particularly for identifying patients at highest risk of recurrent ischemic events superimposed on established ventricular dysfunction [36]. The MAGGIC risk score, derived from individual patient data across more than 39,000 patients from 30 studies, incorporates age, ejection fraction, renal function, and natriuretic peptide where available into a single validated mortality prediction tool

used widely in post-infarction and non-ischemic heart failure alike [37]. Registry data from the CHAMP-HF cohort illustrate that even well-characterized, guideline-eligible patients frequently remain on suboptimal doses of therapies shown to modify each predictor reviewed here, indicating that gaps in therapy titration, not only gaps in risk identification, remain a major driver of preventable MACE [38].

10. Conclusion

Predicting MACE in post-myocardial infarction HFrEF requires moving beyond a single ejection fraction threshold toward layered assessment integrating ventricular trajectory, natriuretic peptide biomarkers and their response to therapy, CMR-defined infarct size and microvascular obstruction, renal function, and markers of electrical instability [4,10,14,18,20]. Each domain maps to an evidence-based intervention, from angiotensin-neprilysin inhibition and mineralocorticoid receptor antagonism through SGLT2 inhibition to device-based rhythm and resynchronization therapy, so that risk identification and modification are, in this population, inseparable clinical tasks [23,26,28,31,33]. Integrated tools such as MAGGIC provide a practical starting point, but registry evidence that guideline-directed therapy remains systematically underdosed suggests that the largest immediate gains may come from more completely acting on predictors already well established, even as CMR, high-sensitivity biomarkers, and polygenic scoring mature further [37,38,8,36,7].

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