Int J Heart Fail. 2026 Jan;8(1):43-55
https://doi.org/10.36628/ijhf.2025.0061
pISSN 2636-154X·eISSN 2636-1558

International Journal of Heart Failure
IJHF

Original Article

Acute Heart Failure Across the Ejection Fraction Spectrum: Phenotypes, Management, and Outcomes From Nationwide KorHF III Registry

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Received: Aug 14, 2025
Revised: Sep 17, 2025
Accepted: Oct 13, 2025
Published online: Nov 26, 2025

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ABSTRACT

Background and Objectives: Clinical characteristics and outcomes in acute heart failure (AHF) vary by phenotype. We assessed phenotype-specific features, treatment patterns, and outcomes in a nationwide Korean cohort.

Methods: The Korean Heart Failure III registry prospectively enrolled 7,351 AHF admissions at 47 hospitals. Among 6,777 patients with available left ventricular ejection fraction (EF), phenotypes were defined as heart failure with reduced EF (HFrEF, ≤40%), mildly reduced EF (HFmrEF,

41–49%), or preserved EF (HFpEF, ≥50%). The primary endpoint was a 12-month composite of all-cause death or heart transplantation, evaluated from index admission and, among hospital survivors, from discharge. We used inverse probability weighting (multinomial generalized boosted models with stabilized, trimmed weights) and weighted Cox proportional-hazards models to estimate hazard ratios (HRs).

Results: Phenotype distribution was 58.9% HFrEF, 13.6% HFmrEF, and 27.5% HFpEF. Crude 12-month composite rates from index admission were 13.4% (HFrEF), 12.7% (HFmrEF), and 16.8% (HFpEF). After weighting, from index admission, HFmrEF (HR, 0.892; 95% confidence interval [CI], 0.731–1.088) and HFpEF (HR, 1.101; 95% CI, 0.939–1.291) did not differ from HFrEF; from discharge, HFpEF had modestly higher risk (HR, 1.207; 95% CI, 1.008–1.445) whereas HFmrEF did not (HR, 1.039; 95% CI, 0.844–1.279). Hyponatremia and chronic kidney disease were consistent adverse markers, while angiotensin-converting enzyme inhibitor/angiotensin II receptor blocker use at discharge was protective.

Conclusions: Across the EF spectrum, phenotypes showed distinct profiles and risk. Post-discharge risk was modestly higher in HFpEF, supporting phenotype-tailored care and systematic discharge optimization in Korean patients with AHF.

Trial Registration: ClinicalTrials.gov Identifier: NCT04329234

Keywords: Heart failure; Acute disease; Hospitalization; Phenotype; Guideline adherence

INTRODUCTION

Heart failure (HF) is a growing global health burden, particularly among older adults. In Korea, the prevalence of HF rose from 0.77% in 2002 to 2.24% in 2018, with the highest rates in individuals over 80 years of age.[1,2] Acute HF (AHF)—new-onset HF or decompensation of chronic HF—is associated with substantial in-hospital and post-discharge mortality.[3]

Although key HF medications improve outcomes in HF with reduced ejection fraction (HFrEF), the evidence base remains comparatively limited for HF with mildly reduced (HFmrEF) or preserved ejection fraction (HFpEF). Marked heterogeneity in clinical presentation, etiology, and treatment response across phenotypes further complicates risk stratification and care.[4,5] Understanding these differences is critical for developing tailored therapeutic strategies.

The recently established Korean Heart Failure III (KorHF III) registry, a large contemporary cohort whose overall design and initial findings have been described elsewhere,[6,7] provides a unique opportunity to address this gap. The present study is a pre-specified secondary analysis of the KorHF III data, specifically designed to characterize the distinct clinical features, in-hospital management, and outcomes across the full spectrum of HF phenotypes. By applying advanced statistical methods, including inverse probability of treatment weighting (IPTW), we aim to identify phenotype-specific predictors of adverse events and inform tailored management strategies.

METHODS

Patient enrollment and data collection procedures

The design and methodology of the KorHF III registry have been reported in detail previously.[6,7] In brief, KorHF III is a prospective, multicenter cohort study that enrolled patients hospitalized for AHF from March 2018 through December 2022 at 47 tertiary hospitals across South Korea, with follow-up planned through 2027. Eligible patients presented with signs or symptoms of HF accompanied by either pulmonary congestion on chest radiography or physical examination, or structural/functional cardiac abnormalities on echocardiography. Written informed consent was obtained from all participants. The study was approved by the Institutional Review Boards (IRBs) of all participating centers (Seoul National University Hospital IRB, IRB No. 1812-077-995) and registered at ClinicalTrials.gov (NCT04329234).

Study oversight and data quality assurance

The registry is overseen by a steering committee and supported by the Korean Society for Heart Failure, with data integrity ensured by an independent monitoring team and a Clinical Event Committee, as detailed in the primary cohort publication.[6,7]

Outcome definition

The primary endpoint was a composite of all-cause mortality or heart transplantation. This outcome was evaluated over two distinct follow-up periods: 1) from the date of index admission up

to 12 months, and 2) from the date of discharge up to 12 months among hospital survivors. Heart transplantation was included as an event, as it represents a progression to end-stage (stage D) HF requiring advanced therapy.[8,9]

Definition of HF subtypes

Left ventricular ejection fraction (LVEF) was ascertained from transthoracic echocardiography. In accordance with the 2021 European Society of Cardiology guidelines,[9] patients were grouped as follows: HF with preserved ejection fraction (HFpEF, LVEF ≥50%), HF with mildly reduced ejection fraction (HFmrEF, LVEF 41–49%), and HF with reduced ejection fraction (HFrEF, LVEF ≤40%). Patients without sufficient echocardiographic data to estimate LVEF were excluded from phenotype-based analyses (**Figure 1**).

Statistical analysis

Baseline characteristics are summarized as mean ± standard deviation for normally distributed continuous variables and median (interquartile range) for non-normally distributed variables; categorical variables as counts (percentages). Group differences across the three phenotypes were compared using one-way analysis of variance or Kruskal–Wallis tests for continuous variables, and χ^2 or Fisher's exact tests for categorical variables, as appropriate.

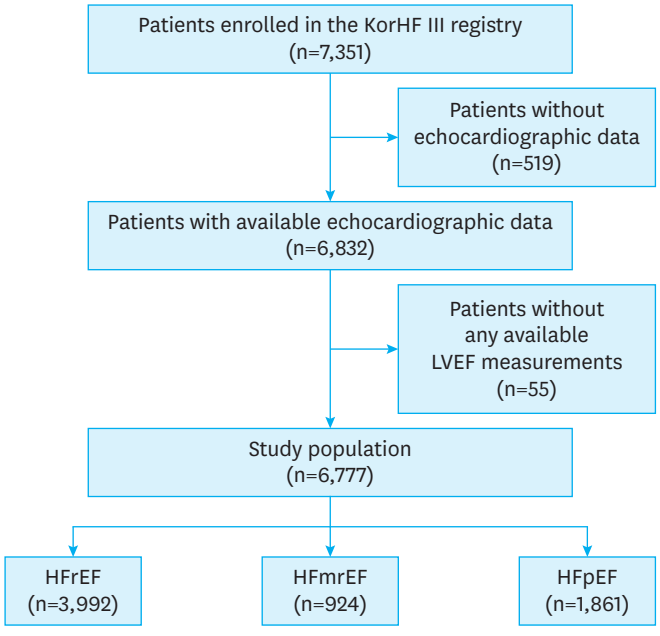


Figure 1. Study flow and phenotype classification.
This flow diagram summarizes enrollment, exclusions, and the analytic cohort with available LVEF, followed by classification into HFrEF, HFmrEF, and HFpEF. It details the number of patients assessed, included, and assigned to each phenotype for subsequent analyses.
LVEF = left ventricular ejection fraction; HFrEF = heart failure with reduced ejection fraction; HFmrEF = heart failure with mildly reduced ejection fraction; HFpEF = heart failure with preserved ejection fraction; KorHF III = Korean Heart Failure III.

To control for confounding by baseline covariates, we performed IPTW. Covariates were first screened using the Boruta algorithm (R package Boruta), and variables confirmed as important were entered into a multinomial propensity-score model estimated by generalized boosted models (R package twang). Stabilized weights were derived and trimmed at the 1st/99th percentiles. Covariate balance before/after weighting was evaluated using absolute standardized mean differences (target <0.1), summarized in tables and Love plots (R package cobalt).

Primary comparative analyses used IPTW-adjusted Kaplan–Meier curves and weighted Cox proportional-hazards models to estimate hazard ratios (HRs) and 95% confidence intervals (CIs) from two time origins: index admission (12-month follow-up) and, among hospital survivors, discharge (12-month follow-up).

In addition, to identify phenotype-specific predictors, we fit Cox models within each phenotype and for each time origin: 1) univariable (univariate) Cox models for each candidate predictor, and 2) multivariable (multivariate) Cox models including prespecified covariates (demographics, comorbidities, admission vitals/laboratories, and discharge medications).

All statistical tests were two-sided, and a p value <0.05 was considered statistically significant. Analyses were conducted in R 4.5.1 (https://cran.r-project.org/bin/windows/base/).

RESULTS

Phenotype-specific clinical, etiologic, and laboratory features at presentation

A total of 6,777 patients hospitalized with AHF were included: 3,992 (58.9%) with HFrEF, 924 (13.6%) with HFmrEF, and 1,861 (27.5%) with HFpEF (**Figures 1** and **2A**). Comprehensive baseline demographic, clinical, and laboratory data for the full cohort are presented in the initial report of the KorHF III registry.[7] In summary, there were significant differences across phenotypes. HFpEF patients were the oldest (mean age 74.7 vs. 65.4 years for HFrEF), more often female (60.7% vs. 33.8% for HFrEF), and had a higher prevalence of hypertension and atrial fibrillation (**Table 1**). Conversely, HFrEF patients exhibited higher natriuretic peptide concentrations (median N-terminal pro–B-type natriuretic peptide 9,366 vs. 5,399 pg/mL for HFpEF) and worse renal indices (**Table 1**). The distribution of etiologies and aggravating factors also differed significantly by phenotype (**Figure 2B and C**). In HFrEF, dilated and ischemic cardiomyopathy were the most common etiologies. In HFmrEF, ischemic cardiomyopathy was most common, while in HFpEF, valvular and hypertensive cardiomyopathy were more frequent.

Laboratory profiles varied by HF phenotype: HFrEF had higher liver enzymes, uric acid, hemoglobin, and slightly worse renal

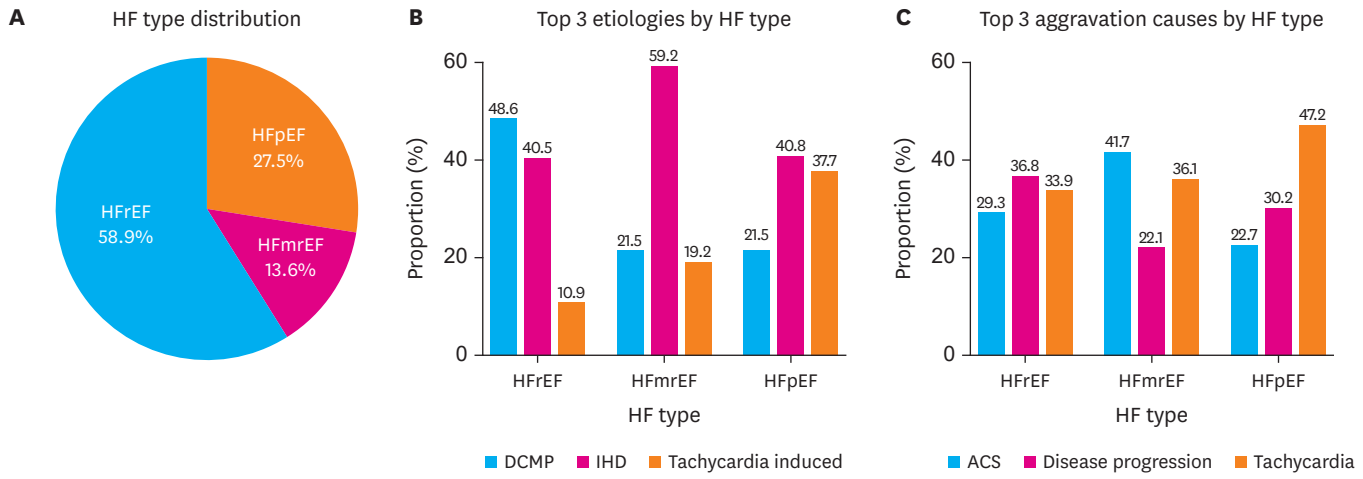


Figure 2. Phenotype-specific features at presentation.
Panel (A) shows the distribution of HF phenotypes in the analytic cohort. Panels (B) and (C) display etiologic and aggravating factors by phenotype, highlighting more dilated/ischemic causes in HFrEF and greater valvular/hypertensive and tachycardia-related patterns in HFpEF, with acute coronary syndrome frequently precipitating decompensation in HFrEF/HFmrEF.
HF = heart failure; HFrEF = heart failure with reduced ejection fraction; HFpEF = heart failure with preserved ejection fraction; HFmrEF = heart failure with mildly reduced ejection fraction; DCMP = dilated cardiomyopathy; IHD = ischemic heart disease; ACS = acute coronary syndrome.

Table 1. Baseline characteristics, clinical management, and in-hospital outcomes by HF phenotype

Variables	Total (n=6,777)	HFrEF (n=3,992)	HFmrEF (n=924)	HFpEF (n=1,861)	p value
Demographics					
Age (years)	68.8±15.3	65.4±16.1	71.0±13.3	74.7±12.3	<0.001
Male (%)	56.9	66.2	52.6	39.3	<0.001
BMI (kg/m^2)	24.6±4.8	24.6±4.9	24.3±4.1	24.7±4.8	0.114
BMI (<18.5 kg/m^2) (%)	6.1	6.2	5.9	5.9	0.879
BMI (>25 kg/m^2) (%)	39.9	38.9	38.1	42.9	0.012
Co-morbidities (%)					
Hypertension	59.7	54.9	65.8	66.9	<0.001
Diabetes mellitus	40.0	40.8	39.8	38.3	0.190
Ischemic heart disease	27.0	28.7	31.5	21.3	<0.001
Atrial fibrillation	33.1	28.3	33.1	43.6	<0.001
Chronic lung disease	7.8	7.0	5.9	10.6	<0.001
Cerebrovascular disease	10.5	9.3	12.2	12.4	0.002
Chronic renal failure	16.1	15.1	17.1	17.8	0.025
Etiology					<0.001
Ischemic CMP	27.7	29.8	38.7	17.8	
Dilated CMP	24.9	34.7	14.3	9.4	
Tachycardia induced CMP	11.0	8.0	12.7	16.4	
Valvular heart disease	8.9	5.7	8.8	15.6	
Hypertensive CMP	6.8	5.7	9.3	8.4	
Clinical status on admission					
De novo HF (%)	56.6	56.5	60.9	54.7	0.009
Lung congestion	42.7	40.3	45.7	46.4	<0.001
SBP at admission (mmHg)	134.3±28.7	131.5±27.8	139.8±30.0	139.1±29.0	<0.001
Low SBP at admission (<100 mmHg) (%)	7.8	9.2	5.4	6.0	<0.001
DBP at admission (mmHg)	80.8±19.3	82.4±19.9	81.9±19.9	78.5±17.8	<0.001
Heart rate at admission (/min)	91.1±25.0	94.6±24.5	90.2±24.8	85.7±24.8	<0.001
NYHA class III–IV (%) at admission	64.6	65.2	59.9	65.6	0.010
Admission route (%)					0.015
Outpatient clinic	29.8	30.3	25.7	30.5	
Emergency department	70.2	69.7	74.3	69.5	
Laboratory test					
BNP (pg/mL)	1,719.1±2,852.2	2,085.8±3,494.3	1,446.5±1,559.6	1,003.7±1,034.2	<0.001
NT-proBNP (pg/mL)	8,148.6±10,728.6	9,366.4±11,466.6	7,857.2±11,487.3	5,398.5±7,482.7	<0.001

(continued to the next page)

Table 1. (Continued) Baseline characteristics, clinical management, and in-hospital outcomes by HF phenotype

Variables	Total (n=6,777)	HFrEF (n=3,992)	HFmrEF (n=924)	HFpEF (n=1,861)	p value
ECG rhythm (%)					<0.001
Atrial fibrillation	29.8	24.7	29.9	40.9	
Atrial flutter	2.3	2.4	2.7	1.8	
Others	5.4	4.9	6.7	5.9	
Pacing rhythm	2.5	2.7	2.0	2.2	
HF medication at admission (%)					
ACEIs/ARBs at admission	57.8	59.0	60.2	54.0	<0.001
ARNI at admission	15.3	23.0	8.1	2.0	<0.001
Beta-blockers at admission	64.6	70.6	63.9	52.2	<0.001
MRAs at admission	55.0	63.1	46.8	41.6	<0.001
Ivabradine at admission	5.4	8.4	1.5	0.8	<0.001
SGLT-2 inhibitor at admission	13.3	17.1	9.7	6.8	<0.001
Other management (%)					
Mechanical ventilation	4.7	4.9	4.5	4.3	0.499
CRRT	1.4	1.4	0.9	1.5	0.358
ECMO	0.9	1.0	0.4	0.7	0.153
LVAD	0.7	1.1	0.0	0.0	<0.001
Outcomes					
Length of hospital stay (days)	12.3±21.7	13.0±21.2	10.3±15.8	11.4±24.5	0.001
Total costs for hospital care* (USD)	8,882.9±21,464.5	9,658.3±21,218.1	8,013.4±20,389.7	7,728.2±21,870.2	0.046
Patient liability costs* (USD)	1,448.5±3,066.1	1,474.3±2,975.7	1,428.3±2,187.8	1,529.4±3,893.0	0.837
Heart transplantation at index admission	0.7	0.8	0.3	0.6	0.242
12-month primary composite outcome from index admission (%)	14.3	13.4	12.7	16.8	0.001
12-month post-discharge primary composite outcome (%)	12.6	11.6	12.3	15.0	0.002

Baseline demographics, comorbidities, etiologies, admission status, admission route, laboratory markers, ECG rhythm, *medications at admission* (chronic outpatient prescriptions confirmed on the day of presentation; not therapies initiated for the index AHF episode), other in-hospital management, and outcomes are summarized by HF phenotype (HFrEF, HFmrEF, HFpEF). Continuous variables are shown as mean±standard deviation and compared with one-way analysis of variance or Kruskal–Wallis tests; categorical variables are number (%) and compared with χ^2 or Fisher's exact tests; p values reflect differences across phenotypes. "Low SBP at admission" is defined as SBP <100 mmHg; heart rate is in beats/min. Costs are presented in US dollars. In-hospital mortality, including heart transplantation denotes a composite endpoint; heart transplantation at index admission is also reported separately.
HF = heart failure; HFrEF = heart failure with reduced ejection fraction; HFmrEF = heart failure with mildly reduced ejection fraction; HFpEF = heart failure with preserved ejection fraction; BMI = body mass index; CMP = cardiomyopathy; SBP = systolic blood pressure; DBP = diastolic blood pressure; NYHA = New York Heart Association; BNP = B-type natriuretic peptide; NT-proBNP = N-terminal pro–B-type natriuretic peptide; ECG = electrocardiography; ACEi = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker; ARNI = angiotensin receptor–neprilysin inhibitor; MRA = mineralocorticoid receptor antagonist; SGLT2i = sodium–glucose cotransporter-2 inhibitor; CRRT = continuous renal replacement therapy; ECMO = extracorporeal membrane oxygenation; LVAD = left ventricular assist device; USD = United States dollar; KRW = Korean won.
*One US dollar is equivalent to 1,335 Korean won.

function; HFpEF showed lower hemoglobin/albumin and a more favorable lipid profile. high-sensitivity C-reactive protein was higher in HFmrEF and HFpEF, while electrolytes, iron parameters, and troponin were similar across groups (**Table 2**).

Use of key HF medications by HF phenotype at admission and discharge

Overall use of key HF medications increased from admission to discharge (**Figure 3A**). In HFrEF, discharge uptake was highest (renin–angiotensin–aldosterone system [RAAS] inhibitors 81.3%, beta-blockers [BBs] 76.3%, mineralocorticoid receptor antagonists [MRAs] 65.4%), and sodium–glucose cotransporter-2 inhibitors [SGLT2i] use rose from 17.1% to 20.3% (**Figure 3B**). HFmrEF showed similar but more modest gains (**Figure 3C**). In contrast, HFpEF changed little, and SGLT2i use remained low (**Figure 3D**). **Supplementary Figure 1** shows year-by-year prescription rates of key HF therapies (angiotensin-converting enzyme inhibitor/angiotensin II receptor blocker [ACEi/ARB], angiotensin receptor–neprilysin inhibitor [ARNI], BB, MRA, SGLT2i) by phenotype (HFrEF, HFmrEF, HFpEF) at admission and discharge.

In-hospital management according to HF phenotype

In-hospital management differed significantly across phenotypes (**Table 3**). Loop diuretics were common overall (68.4%) and most frequently used in HFrEF (71.0%). Inotropes were administered more often in HFrEF than in HFmrEF or HFpEF (25.2% vs. 19.5% and 12.8%; p<0.001). IV diuretics were most commonly used in HFpEF, whereas IV vasodilators were more frequent in HFmrEF.

As shown in **Table 1**, mechanical ventilation, continuous renal replacement therapy, and extracorporeal membrane oxygenation were uncommon and did not differ meaningfully across phenotypes, whereas left ventricular assist device (LVAD) use—though rare—was largely confined to HFrEF.

Table 2. Laboratory tests on admission by HF phenotype

Variables	Total (n=6,777)	HFrEF (n=3,992)	HFmrEF (n=924)	HFpEF (n=1,861)	p value
WBC (10^3/μL)	8.3±3.6	8.5±3.6	8.6±3.7	7.8±3.7	<0.001
Neutrophil (%)	67.8±12.3	67.6±12.2	68.3±12.7	68.5±12.4	0.021
Lymphocyte (%)	21.7±10.6	22.1±10.6	21.5±11.1	20.8±10.4	<0.001
Hb (g/dL)	12.4±2.5	13.0±2.4	12.1±2.4	11.5±2.4	<0.001
Platelet (10^3/μL)	215.9±83.5	221.7±84.0	217.9±84.2	206.4±82.3	<0.001
Total bilirubin (mg/dL)	1.0±1.0	1.1±1.0	1.0±1.1	0.9±0.7	<0.001
Albumin (g/dL)	3.9±0.5	3.9±0.5	3.9±0.5	3.8±0.6	0.002
AST (IU/L)	69.5±262.6	81.0±311.2	66.8±182.7	53.2±208.0	0.001
ALT (IU/L)	54.1±175.1	66.0±208.2	46.3±143.5	36.8±117.7	<0.001
Uric acid (mg/dL)	7.0±2.7	7.3±2.9	6.8±2.4	6.5±2.5	<0.001
BUN (mg/dL)	26.2±15.7	26.0±15.6	26.1±15.5	25.9±15.1	0.953
Creatinine (mg/dL)	1.4±1.3	1.5±1.3	1.5±1.4	1.3±1.1	0.002
Sodium (mmol/L)	137.9±4.4	137.9±4.3	138.0±4.3	137.9±4.6	0.827
Hyponatremia (<135 mmol/L, %)	17.4	17.1	16.3	18.8	0.182
Hyponatremia (<130 mmol/L, %)	4.6	4.3	4.1	5.3	0.176
Potassium (mmol/L)	4.3±0.7	4.3±0.6	4.3±0.7	4.3±0.7	0.004
Hyperkalemia (>5.5 mmol/L, %)	0.9	1.2	0.5	0.6	0.031
Hypokalemia (<3.5 mmol/L, %)	2.0	2.2	2.2	1.5	0.146
Total cholesterol (mg/dL)	144.9±45.7	147.2±46.1	147.0±46.9	140.3±44.3	<0.001
Triglyceride (mg/dL)	108.7±69.2	111.6±73.7	109.5±61.5	102.3±63.5	0.001
HDL (mg/dL)	42.5±13.9	41.6±13.2	43.4±14.9	44.2±14.6	<0.001
LDL (mg/dL)	88.5±39.0	91.7±39.8	88.4±39.4	82.0±36.1	<0.001
Iron (ug/dL)	53.6±42.1	54.6±38.8	47.6±32.7	53.5±50.0	0.196
TIBC (mcg/dL)	284.1±85.4	284.3±87.1	274.3±83.8	285.7±83.3	0.382
Ferritin (ng/mL)	237.8±535.6	240.4±550.8	274.3±666.7	219.9±431.6	0.489
BNP (pg/mL)	1,719.1±2,852.2	2,085.8±3,494.3	1,446.5±1,559.6	1,003.7±1,034.2	<0.001
NT-proBNP (pg/mL)	8,148.6±10,728.6	9,366.4±11,466.6	7,857.2±11,487.3	5,398.5±7,482.7	<0.001
TnI (ng/mL)	13.7±249.2	14.1±244.8	12.7±116.3	3.4±24.6	0.367
TnT (ng/mL)	1.8±12.4	1.5±8.5	2.6±21.8	1.0±4.8	0.108
hs-CRP (mg/dL)	3.1±9.6	2.7±7.0	4.2±13.4	3.7±12.7	0.012

Admission laboratory measurements are summarized by HF phenotype (HFrEF, HFmrEF, HFpEF). Continuous variables are presented as mean ± standard deviation; categorical variables (e.g., hyponatremia, hyperkalemia, hypokalemia) are shown as number (%). Labs were obtained at emergency department presentation or within 24 hours of admission, using local hospital platforms. Hyponatremia was defined a priori as sodium <135 mmol/L (and <130 mmol/L for severe hyponatremia); hyperkalemia as potassium >5.5 mmol/L; hypokalemia as potassium <3.5 mmol/L. Group comparisons used one-way analysis of variance or Kruskal–Wallis tests for continuous variables and χ^2 or Fisher's exact tests for categorical variables; p values indicate differences across phenotypes. Totals may not equal 100% due to rounding; missing data were not imputed.
HF = heart failure; HFrEF = heart failure with reduced ejection fraction; HFmrEF = heart failure with mildly reduced ejection fraction; HFpEF = heart failure with preserved ejection fraction; WBC = white blood cell; Hb = hemoglobin; AST = aspartate aminotransferase; ALT = alanine aminotransferase; BUN = blood urea nitrogen; HDL = high-density lipoprotein; LDL = low-density lipoprotein; TIBC = total iron-binding capacity; BNP = B-type natriuretic peptide; NT-proBNP = N-terminal pro–B-type natriuretic peptide; TnI = troponin I; TnT = troponin T; hs-CRP = high-sensitivity C-reactive protein.

IPTW-adjusted Kaplan–Meier survival curves by HF phenotype

After IPTW, baseline characteristics were well balanced across the three phenotypes, with all absolute standardized mean differences <0.1 (**Supplementary Tables 1** and **2**, **Supplementary Figure 2**).

The IPTW-adjusted survival analyses are shown in **Figure 4**. From the index admission (**Figure 4A**), relative to HFrEF, the 12-month primary composite outcome was not different for HFmrEF (HR, 0.892; 95% CI, 0.731–1.088; p=0.260) and not different for HFpEF (HR, 1.101; 95% CI, 0.939–1.291; p=0.235). For context, the unadjusted 12-month composite rates in **Table 1** were 13.4% (HFrEF), 12.7% (HFmrEF), and 16.8% (HFpEF); overall 14.3%.

Among hospital survivors, the post-discharge analysis over 12 months (**Figure 4B**) likewise showed no difference for HFmrEF versus HFrEF (HR, 1.039; 95% CI, 0.844–1.279; p=0.717), whereas HFpEF had a modestly higher risk (HR, 1.207; 95% CI, 1.008–1.445; p=0.040). As a crude reference after discharge, **Table 1** reports 11.6% for HFrEF, 12.3% for HFmrEF, and 15.0% for HFpEF over 12 months (overall 12.6%).

Predictors of in-hospital and 12-month post-discharge primary composite outcome by HF phenotype

In multivariable Cox models fit separately by phenotype (**Figure 5** and **Supplementary Figure 3**), predictors of the primary composite outcome differed by the time origin.

For the 12-month primary composite endpoint with index admission as the time origin (**Supplementary Figure 3**): In HFrEF, higher 12-month risk was associated with chronic kidney disease (CKD), hyponatremia, acute decompensated HF (ADHF),

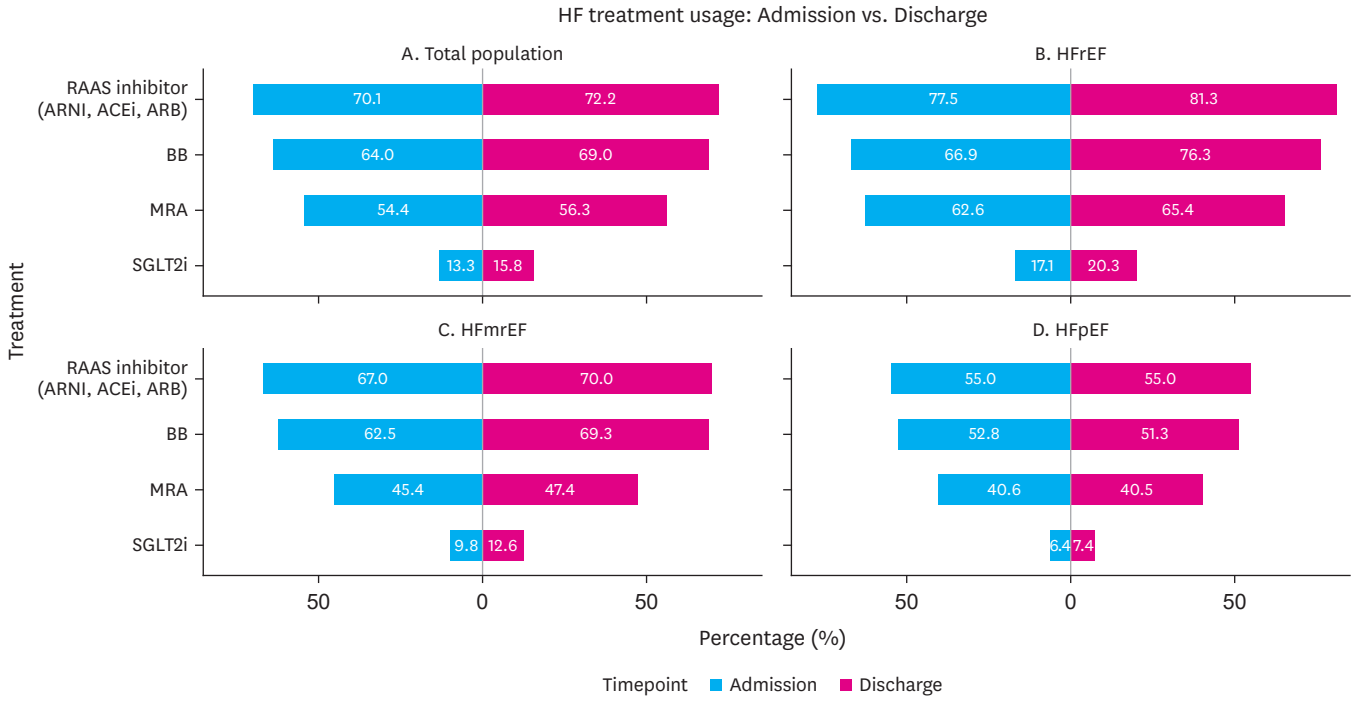
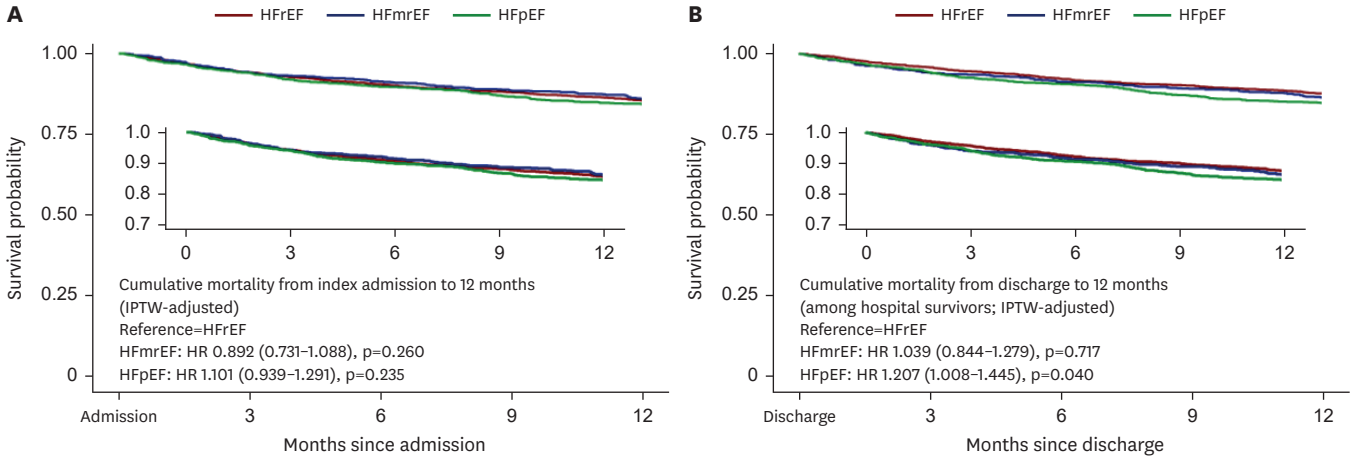


Figure 3. Use of key HF medications at admission and discharge by phenotype.
Stacked bars depict the use of key heart failure medications admission and discharge, showing overall increases driven mainly by HFrEF, modest gains in HFmrEF, and minimal change in HFpEF. SGLT2i uptake remained low compared with RAAS inhibitors, beta-blockers, and MRAs, particularly in HFpEF.
HF = heart failure; HFrEF = heart failure with reduced ejection fraction; HFmrEF = heart failure with mildly reduced ejection fraction; HFpEF = heart failure with preserved ejection fraction; SGLT2i = sodium–glucose cotransporter-2 inhibitor; RAAS = renin–angiotensin–aldosterone system; MRA = mineralocorticoid receptor antagonist; ARNI = angiotensin receptor–neprilysin inhibitor; ACEi = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker; BB = beta-blocker.



IPTW-adjusted No. at risk					
HFrEF	2,363	2,129	2,006	1,919	1,372
HFmrEF	2,219	2,009	1,892	1,803	1,287
HFpEF	2,195	1,967	1,835	1,725	1,182

IPTW-adjusted No. at risk					
HFrEF	2,372	2,160	2,045	1,964	1,661
HFmrEF	2,217	1,992	1,885	1,807	1,483
HFpEF	2,042	1,816	1,710	1,597	1,335

Figure 4. IPTW-adjusted KM survival curves for the primary composite outcome.
KM curves show the cumulative incidence of the primary composite outcome (all-cause mortality or heart transplantation) according to heart failure phenotype after IPTW adjustment. HRs and 95% confidence intervals were derived from weighted Cox proportional-hazards models with HFrEF as the reference group.
IPTW = inverse probability of treatment weighting; KM = Kaplan-Meier; HR = hazard ratio; HFrEF = heart failure with reduced ejection fraction; HFmrEF = heart failure with mildly reduced ejection fraction; HFpEF = heart failure with preserved ejection fraction; HF = heart failure.

Table 3. In-hospital management by heart failure phenotype

Variables	Total (n=6,777)	HFrEF (n=3,992)	HFmrEF (n=924)	HFpEF (n=1,861)	p value
Pharmacological treatments					
Nitrates	18.9	18.8	22.8	17.0	0.001
Loop diuretics	68.4	71.0	62.6	65.6	<0.001
Amiodarone	10.5	12.1	8.6	8.0	<0.001
Digoxin	14.2	15.4	11.7	12.8	0.002
Warfarin	7.4	7.5	6.1	7.7	0.253
NOAC	28.3	25.3	27.5	35.3	<0.001
Antiplatelet agent (aspirin)	38.9	41.1	47.1	30.1	<0.001
Statins/ezetimibe	56.5	57.1	61.1	53.2	<0.001
Insulin	29.3	30.0	33.1	25.5	0.036
Oral diabetes medication	36.6	38.9	34.5	32.5	<0.001
Parenteral medications					
Diuretics (IV)	84.4	84.6	80.7	85.9	0.007
Vasodilators (IV)	37.4	37.1	41.9	35.5	0.014
Inotropes (any)	21.2	25.2	19.5	12.8	<0.001
Dobutamine	81.7	85.6	60.4	77.8	<0.001
Dopamine	47.5	47.2	37.5	59.5	0.024
Milrinone	11.5	14.2	1.6	13.6	0.020
Epinephrine	26.8	28.2	15.7	35.8	0.033
Norepinephrine	77.5	74.9	79.3	83.6	0.149
Vasopressin	8.7	9.9	4.6	9.3	0.415
Heparin (IV)	28.1	29.8	31.6	22.3	<0.001
Non-pharmacological treatments					
Blood transfusion	8.3	7.6	7.9	10.2	0.004
Intermittent HD	2.7	2.9	2.4	2.5	0.651
CRT	1.0	1.6	0.1	0.2	
ICD	1.7	2.3	1.0	0.6	<0.001
PCI	13.9	13.2	24.6	10.0	<0.001
CABG	2.2	3.0	1.5	0.7	<0.001
Valve operation (surgical)	0.9	0.7	1.1	1.2	0.138
Percutaneous valve intervention	0.3	0.1	0.3	0.7	0.001

Percentages indicate the proportion of patients who received each therapy during the index hospitalization, stratified by phenotype (HFrEF, HFmrEF, HFpEF). "Parenteral medications" denote IV use. Inotropes (any) indicates receipt of ≥1 inotropic/vasoactive agent; individual agent rows (dobutamine, dopamine, milrinone, epinephrine, norepinephrine, vasopressin) are expressed among patients who received any inotrope, and may exceed 100% in total because combinations were permitted. Device/procedure variables (e.g., ICD, PCI, CABG) were recorded during hospitalization and summarized at discharge. Group comparisons used χ^2 tests (or Fisher's exact test when appropriate); p values reflect differences across phenotypes. Totals may not equal 100% due to rounding; missing cells indicate data not applicable or not displayed.
HFrEF = heart failure with reduced ejection fraction; HFmrEF = heart failure with mildly reduced ejection fraction; HFpEF = heart failure with preserved ejection fraction; NOAC = non–vitamin K oral anticoagulant; IV = intravenous; HD = hemodialysis; CRT = cardiac resynchronization therapy; ICD = implantable cardioverter-defibrillator; PCI = percutaneous coronary intervention; CABG = coronary artery bypass graft.

lung congestion at admission, and low systolic blood pressure (<100 mmHg), whereas ACEi/ARB, BB, and ARNI at discharge were protective. In HFmrEF, ADHF and CKD were significant adverse predictors, while ACEi/ARB at discharge was protective. In HFpEF, low body mass index (BMI <18.5 kg/m^2), hyponatremia, and CKD were associated with higher risk; ACEi/ARB at discharge was protective and ARNI at discharge showed a protective trend.

For the 12-month primary composite endpoint with discharge as the time origin among hospital survivors (**Figure 5**): In HFrEF, the adverse profile persisted: CKD, hyponatremia, ADHF, lung congestion at admission, low systolic blood pressure, and low BMI were associated with higher 12-month risk, while ACEi/ARB and BB at discharge were protective (ARNI showed a protective direction). In HFmrEF, ADHF and CKD remained significant adverse predictors; medication effects did not reach statistical significance. In HFpEF, low BMI, hyponatremia, and CKD were again linked to higher risk, and ACEi/ARB at discharge was protective.

Univariate predictors of the primary composite outcome were assessed for two follow-up periods (from index admission to 12 months and from discharge to 12 months), stratified by HF phenotype (**Supplementary Tables 3-8**).

DISCUSSION

Of the 7,351 patients enrolled in KorHF III, 6,777 were included in the analytic cohort; we observed marked phenotype-related heterogeneity in presentation, treatment, and outcomes. At baseline, HFpEF patients were older and more often female, with

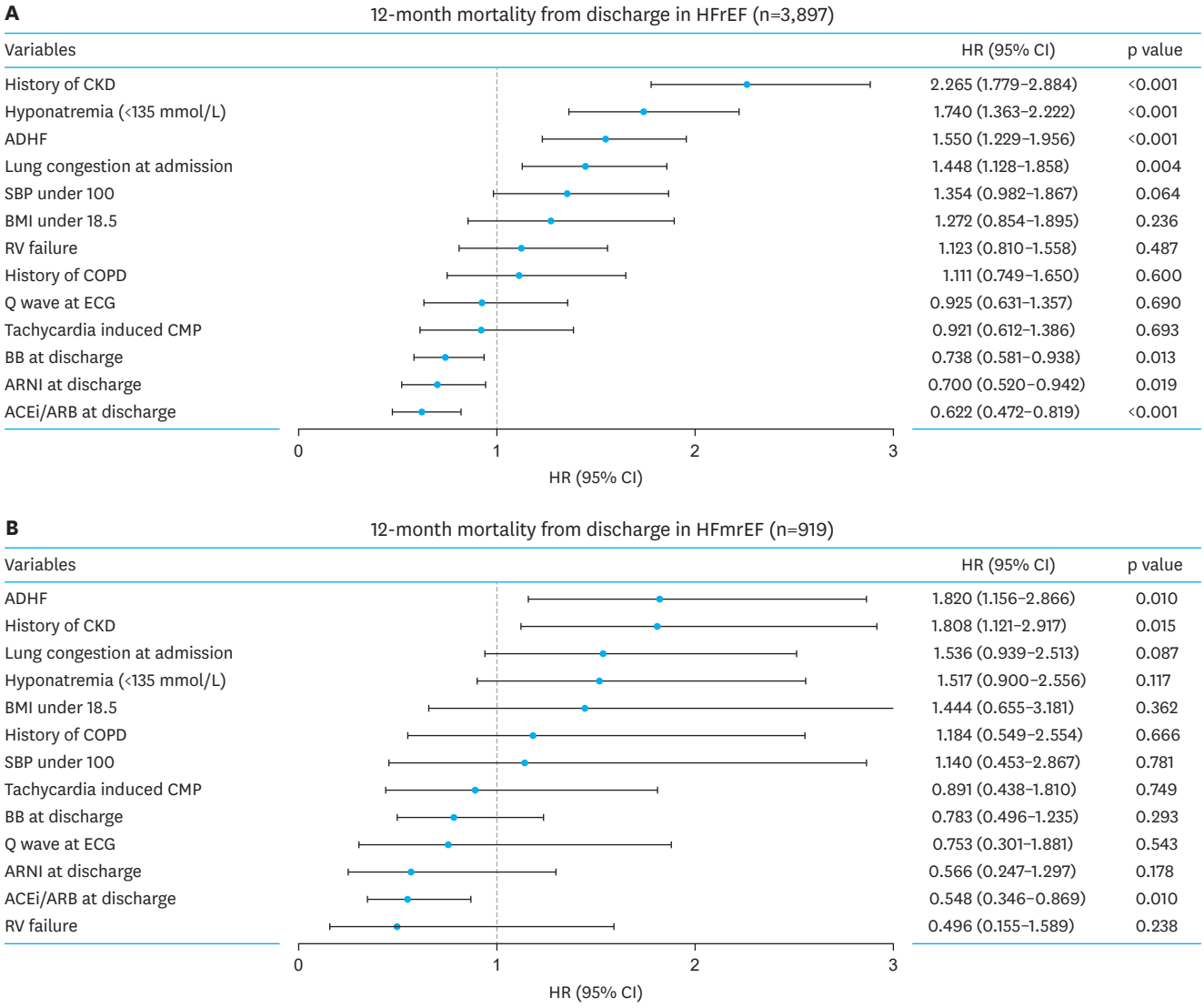


Figure 5. Phenotype-specific multivariable predictors for 12-month post-discharge primary composite outcome.
Forest plots display adjusted associations for in-hospital and 12-month post-discharge outcomes, modeled separately by phenotype. Adverse markers (e.g., hyponatremia, CKD, congestion, low BMI in HFpEF) contrast with protective associations for discharge therapies (ACEi/ARB, ARNI, BBs), with effect sizes shown as HRs with 95% CIs.
CKD = chronic kidney disease; BMI = body mass index; HFpEF = heart failure with preserved ejection fraction; ACEi = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker; ARNI = angiotensin receptor–neprilysin inhibitor; BB = beta-blocker; HR = hazard ratio; CI = confidence interval; HFrEF = heart failure with reduced ejection fraction; ADHF = acute decompensated heart failure; SBP = systolic blood pressure; RV = right ventricular; COPD = chronic obstructive pulmonary disease; ECG = electrocardiogram; CMP = cardiomyopathy; HFmrEF = heart failure with mildly reduced ejection fraction. (continued to the next page)

more hypertension and atrial fibrillation, whereas HFrEF showed higher natriuretic peptide levels. Use of key HF medications rose from admission to discharge, but gains were minimal in HFpEF. On IPTW analyses, 12-month risk from admission was similar for HFmrEF and HFpEF versus HFrEF, whereas from discharge HFpEF showed a modest excess risk and HFmrEF remained comparable to HFrEF. Across phenotypes, hyponatremia, CKD, and low BMI tracked with higher risk; in HFrEF, congestion, ADHF, and low SBP were additional signals. Discharge ACEi/ARB was consistently protective (BB in HFrEF; ARNI directionally protective).

In our cohort, use of RAAS inhibitors, BBs, and MRAs rose most in HFrEF, whereas uptakes in HFmrEF/HFpEF were modest and SGLT2i use remained low.[10] These gaps likely reflect a mix of patient factors (advanced age, hypotension, renal dysfunction, polypharmacy) and system barriers (coverage, order-set inertia, fragmented transitions).[11] A pragmatic approach is a phenotype-tailored discharge algorithm: 1) HFrEF—early ARNI/ACEi or ARB, BBs, MRA, and SGLT2i with rapid uptitration and

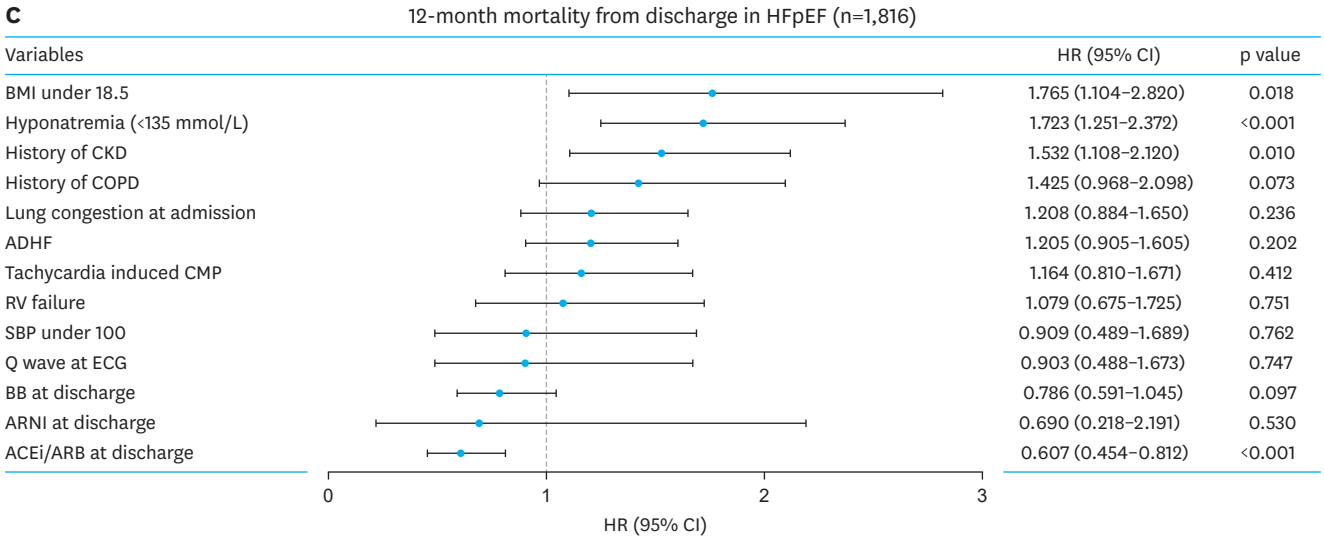


Figure 5. (Continued) Phenotype-specific multivariable predictors for 12-month post-discharge primary composite outcome.
Forest plots display adjusted associations for in-hospital and 12-month post-discharge outcomes, modeled separately by phenotype. Adverse markers (e.g., hyponatremia, CKD, congestion, low BMI in HFpEF) contrast with protective associations for discharge therapies (ACEi/ARB, ARNI, BBs), with effect sizes shown as HRs with 95% CIs.
CKD = chronic kidney disease; BMI = body mass index; HFpEF = heart failure with preserved ejection fraction; ACEi = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker; ARNI = angiotensin receptor–neprilysin inhibitor; BB = beta-blocker; HR = hazard ratio; CI = confidence interval; HFrEF = heart failure with reduced ejection fraction; ADHF = acute decompensated heart failure; SBP = systolic blood pressure; RV = right ventricular; COPD = chronic obstructive pulmonary disease; ECG = electrocardiogram; CMP = cardiomyopathy; HFmrEF = heart failure with mildly reduced ejection fraction.

labs; 2) HFmrEF—apply the same four drug classes when tolerated; 3) HFpEF—prioritize SGLT2i as first-line disease-modifying therapy, add RAAS blockade/ARNI for blood-pressure or congestion phenotypes, and address comorbidity clusters (atrial fibrillation, obesity, hypertension).[10,12] Implementation should pair a discharge checklist (med start/targets, labs, education), pharmacy-nursing titration protocols, and early post-discharge follow-up (≤7–14 days) with telemonitoring to narrow inertia and safety-check renal/electrolytes.[10,13]

Across phenotypes, we observed heterogeneous prognostic profiles in which hyponatremia, CKD, ADHF at index admission, low BMI (<18.5 kg/m^2), and clinical lung congestion conferred risk with differing magnitudes by subtype.[14,15] Many of these factors are at least partially modifiable, suggesting room for targeted, multidisciplinary intervention: systematic decongestion guided by symptoms, exam, and labs; proactive correction of hyponatremia (judicious diuresis, fluid strategy, and selective use of adjuncts when appropriate); kidney-protective care (dose adjustment, avoidance of nephrotoxins, hemodynamic monitoring); and structured nutritional support for underweight patients.[16-18] Embedding these components into phenotype-aware care pathways—and operationalizing them in a phenotype-weighted risk score for discharge and early follow-up—may help translate risk identification into risk reduction.[14,19]

The higher 12-month post-discharge mortality for HFpEF on IPTW analysis highlights transitional-care vulnerabilities that extend beyond inpatient stabilization.[20] A structured post-discharge care pathway should prioritize: early clinic follow-up within 7 days; pharmacist- and nurse-led medication optimization bundles (diuretic titration, blood-pressure control, initiation/maintenance of SGLT2i and other phenotype-appropriate therapies); remote monitoring for weight, symptoms, and blood pressure with rapid-response protocols; and coordinated rehabilitation and comorbidity management (atrial fibrillation, obesity, hypertension, CKD).[21] Embedding these elements within multidisciplinary HF clinics, supported by standardized discharge checklists and clear escalation thresholds, can reduce therapeutic inertia and detect deterioration earlier. Programs should be evaluated against hard performance metrics—30-day and 90-day readmissions and 12-month mortality—with feedback loops to iteratively refine workflows and resource allocation for the HFpEF population.[20]

Strengths

This nationwide, contemporary prospective cohort across 47 tertiary hospitals used standardized eCRFs, independent monitoring, and blinded adjudication, enabling granular characterization of phenotype-specific presentation, in-hospital care, discharge therapy, and 12-month outcomes. Analytically, we prespecified phenotype-based comparisons and applied multivariable modeling alongside IPTW with stabilized, trimmed weights (TWANG) and Boruta-informed covariate selection to mitigate

confounding, thereby linking real-world treatment patterns to risk across HFrEF, HFmrEF, and HFpEF.

Limitations

First, lactate was not captured, limiting precise identification of cardiogenic shock. Second, although consecutive enrollment was intended, coronavirus disease-era convenience sampling and consent requirements may have introduced selection/survivorship bias—potentially contributing to modest mortality rates and under-representation of the sickest patients. Third, medication data reflect prescriptions at admission and do not capture in-hospital dosing trajectories or uptitration; thus the impact of dynamic therapy during index hospitalization cannot be assessed, and the low observed use of SGLT2 inhibitors likely reflects lack of national reimbursement for non-diabetic heart-failure indications during enrollment (system-level constraint rather than clinical inertia). Fourth, ≤7% lacked baseline echocardiography; defining phenotype by the maximal LVEF from transthoracic echocardiographic methods may have introduced misclassification. Fifth, the cohort's ethnic homogeneity limits generalizability beyond Korea. Sixth, the higher mortality observed in HFpEF should be interpreted cautiously, as residual differences in age and comorbidity (e.g., hypertension, atrial fibrillation) may contribute despite adjustment. Finally, residual and unmeasured confounding remain possible; LVAD numbers were too small for inclusion; and the composite endpoint included heart transplantation, for which access likely varied across centers, complicating interpretation in subgroups with limited access.

In this nationwide, contemporary cohort, HF phenotypes based on EF showed distinct clinical profiles, treatment patterns, and trajectories of risk. Despite overall gains in use of key HF medications, uptake remained modest in HFmrEF and minimal in HFpEF, and IPTW analyses revealed higher 12-month mortality in HFpEF (with HFmrEF approximating HFrEF) alongside phenotype-specific, partly modifiable risk markers (e.g., hyponatremia, congestion, CKD, low BMI). These findings support a phenotype-tailored strategy that standardizes discharge initiation and early uptitration of evidence-based therapies—prioritizing SGLT2 inhibitors in HFpEF—coupled with structured post-discharge care and close follow-up. Implementing such pathways may narrow treatment gaps and translate risk identification into risk reduction across the EF spectrum.

ACKNOWLEDGEMENTS

The authors gratefully acknowledge the contributions of the KorHF III investigators and participating institutions. KorHF III investigators; Seoul National University Bundang Hospital (Dong-Ju Choi), Seongnam, Severance Cardiovascular Hospital (Seok-Min Kang), Wonju Severance Cardiovascular Hospital (Byung-Su Yoo), Wonju, Seoul National University Hospital (Hyun-Jai Cho), Samsung Medical Center (Jin Oh Choi), Kangnam Sacred Heart Hospital (Seonghoon Choi), Gangnam Severance Hospital (Eui-Young Choi), Kangdong Sacred Heart Hospital (Il Suk Sohn), Kangdong Sacred Heart Hospital (Dae-Gyun Park), Kangbuk Samsung Hospital (Ki-Chul Sung), Konkuk University Medical Center (Sung Hea Kim), Kyung Hee University Hospital (Hyemoon Chung), Korea University Guro Hospital (Eung Ju Kim), Korea University Anam Hospital (Seong-Mi Park), Seoul National University Boramae Medical Center (Myung-A Kim), Seoul St. Mary Hospital (Jong-Chan Youn), Asan Medical Center (Junho Hyun), Ewha Womans University Medical Center (Kyung-Jin Kim), Seoul, Dongtan Sacred Heart Hospital (Seong Woo Han), Hwaseong, Bucheon Sejong Hospital (Seok-Geon Hong), Bucheon St. Mary's Hospital (Sang-Hyun Ihm), Bucheon SoonChunHyang University Hospital (Min Gyu Kong), Bucheon, Ilsan Paik Hospital (Sung Woo Cho), National Cancer Center (Jae-Kyung Kim), Goyang, Pyeongchon Sacred Heart Hospital (Sang-Ho Jo), Anyang, Ajou University Hospital (Joon-Han Shin), Uijeongbu St. Mary's Hospital (Hyo-Suk Ahn), Uijeongbu, Kangwon National University Hospital (Dong Ryeol Ryu), Chuncheon Sacred Heart Hospital (Kyeong-Sun Hong), Chuncheon, Gil Medical Center (Mi-Seung Shin), Incheon Sejong Hospital (Kyung-Hee Kim), Incheon St. Mary's Hospital (Mi-Jeong Kim), Inha University Hospital (Sung-Hee Shin), Incheon, Kyungpook National University (Dong Heon Yang), Dongsan Hospital (Hyung-Seop Kim), Yeungnam University Medical Center (Jong-Ho Nam), Deagu, Ulsan University Hospital (Shin-Jae Kim), Kosin University Gospel Hospital (Bong-Joon Kim), Pusan University Hospital (Jung Hyun Choi), Inje University Busan Paik Hospital (Jeong-Sook Seo), Busan, Pusan National University Yangsan Hospital (Yong-Hyun Park), Chungbuk National University Hospital (Myung-Chan Cho), Cheongju, Chungnam National University Hospital (Jin-Ok Jeong), Daejeon, Gyeongsang National University Hospital (Seok-Jae Hwang), Jinju, Chonnam National University Hospital (Kye Hun Kim), Gwangju, Jeonbuk National University Hospital (Sun Hwa Lee), Jeonju, Jeju University Hospital (Seung Jae Joo).

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Trial Registration
ClinicalTrials.gov Identifier: NCT04329234

Funding
This work was supported by the Korean Society of Heart Failure and the Heart Failure Registry TFT.

Conflict of Interest
The authors have no financial conflicts of interest.

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SUPPLEMENTARY MATERIALS

Supplementary Table 1
Balance of baseline covariates before and after inverse probability of treatment weighting for the primary composite outcome analysis from index admission to 12 months

Supplementary Table 2
Balance of baseline covariates before and after inverse probability of treatment weighting for the primary composite outcome analysis from discharge to 12 months

Supplementary Table 3
Univariate predictors of the 12-month primary composite outcome from index admission in patients with HF with reduced ejection fraction

Supplementary Table 4
Univariate predictors of the 12-month primary composite outcome from index admission in patients with HF with mildly reduced ejection fraction

Supplementary Table 5
Univariate predictors of the 12-month primary composite outcome from index admission in patients with HF with preserved ejection fraction

Supplementary Table 6
Univariate predictors of the 12-month post-discharge primary composite outcome in HF with reduced ejection fraction patients

Supplementary Table 7
Univariate predictors of the 12-month post-discharge primary composite outcome in HF with mildly reduced ejection fraction patients

Supplementary Table 8
Univariate predictors of the 12-month post-discharge primary composite outcome in HF with preserved ejection fraction patients

Supplementary Figure 1
Yearly prescription rates of key HF medications (2018–2022): admission vs. discharge.

Supplementary Figure 2
Covariate balance before and after inverse probability of treatment weighting.

Supplementary Figure 3

Phenotype-specific multivariable predictors for the 12-month primary composite outcome from index admission.

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