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RESEARCH ARTICLE

Sex- and phenotype-specific prognostic implications of body mass index in acute heart failure

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Abstract

Introduction This study aimed to determine whether the prognostic implications of body mass index (BMI) differ according to heart failure (HF) phenotype and sex.

Methods From the Korean HF III registry ($n = 7351$), we analyzed 5271 patients hospitalized for acute heart failure (AHF) with available data. BMI was categorized as low (<18.5 kg/m^2), normal (18.5–24.9), or high (≥25.0) using cut-off values consistent with Asia-Pacific criteria. The primary outcome was a composite of 2-year all-cause mortality or heart transplantation. Kaplan–Meier analyses and multivariable Cox proportional hazards models, including interaction terms for BMI, sex, and HF phenotype, were performed.

Results In HF with reduced ejection fraction (HFrEF), lower BMI was consistently associated with worse outcomes in both men and women. In contrast, in HF with preserved ejection fraction (HFpEF), BMI was prognostic in women but not in men: survival differed by BMI category in Kaplan–Meier analyses for all subgroups except men with HFpEF. In multivariable analyses, higher BMI was independently associated with lower risk in women with HFrEF [hazard ratio (HR) 0.66, 95% confidence interval (CI) 0.45–0.96, $P = .032$], whereas lower BMI in women with HFpEF showed a borderline association with higher risk (HR 1.56, 95% CI 0.99–2.47, $P = .057$).

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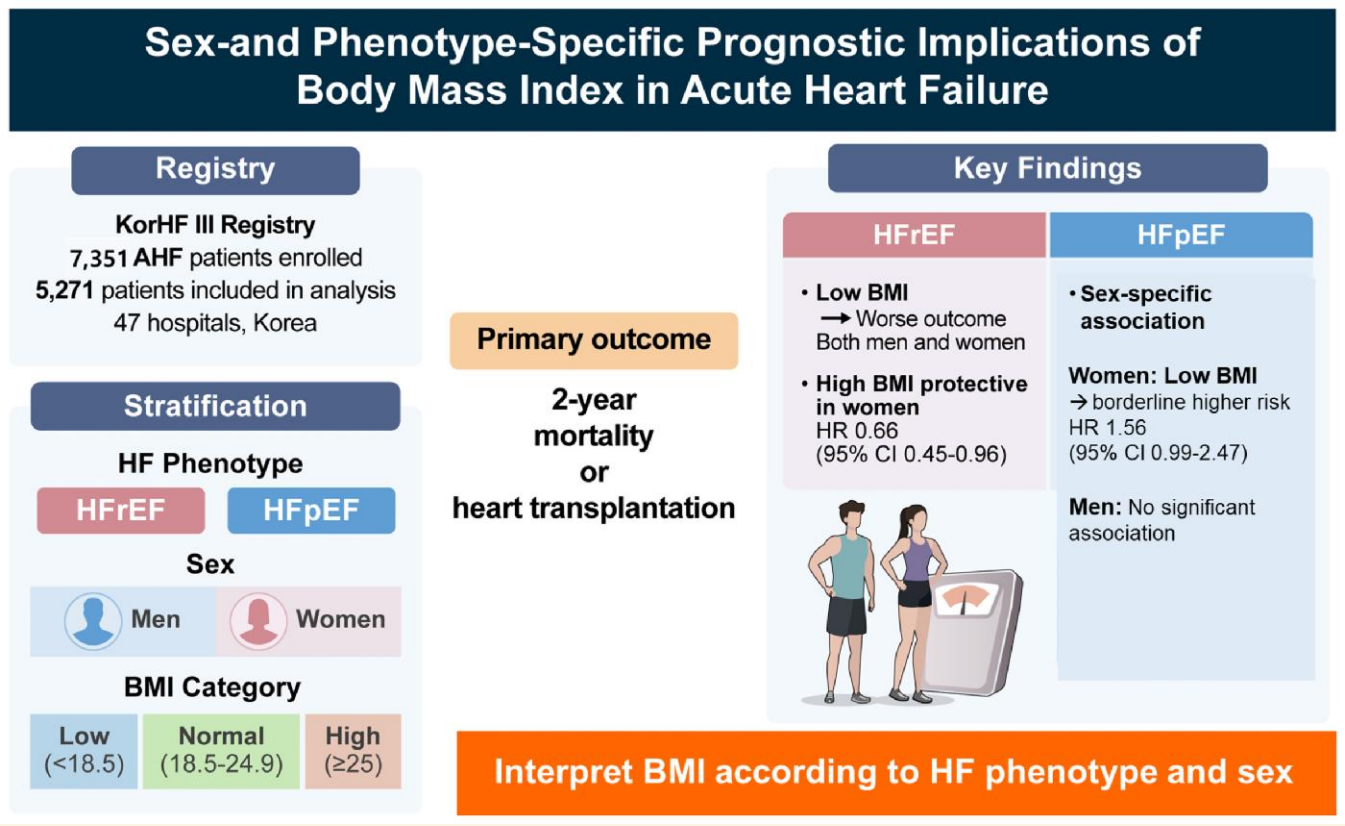
Conclusion The prognostic implications of BMI in AHF differ according to HF phenotype and sex. Lower BMI is a consistent adverse marker in HFrEF in both sexes and shows a borderline adverse association in women with HFpEF, whereas BMI is not prognostic in men with HFpEF. These findings highlight the importance of sex- and phenotype-specific interpretation of BMI in risk assessment.

Graphical Abstract



Graphical abstract of the sex- and phenotype-specific prognostic implications of body mass index in patients with acute heart failure.

Keywords Body mass index • Heart failure phenotype • Sex differences • Acute heart failure • Registry

Introduction

Heart failure (HF) is an increasing public health concern. In Korea, the prevalence of HF has risen steadily since the early 2000s, accompanied by parallel increases in hospitalizations, comorbidity burden, and healthcare utilization, largely driven by rapid population aging.[1] Obesity contributes to the development of HF through metabolic, haemodynamic, and inflammatory pathways.[2–4] Paradoxically, a higher body mass index (BMI) has been associated with lower mortality in patients with established HF—a phenomenon known as the 'obesity paradox'.[5–8] However, this paradox has not been consistently observed and appears to vary according to HF phenotype, sex, and regional patterns in body composition.[9,10]

HF with preserved ejection fraction (HFpEF) and HF with reduced ejection fraction (HFrEF) differ in pathophysiology, comorbidity profiles, and response to treatment. Accumulating evidence suggests that excess adiposity plays a more prominent role in the development and progression of HFpEF, particularly among women.[4,7,9] In contrast, low BMI, which is more common in East Asian populations,[11–13] may reflect frailty, malnutrition, or cardiac cachexia and has been consistently associated with adverse outcomes in HF.[3,4,14] These observations underscore the possibility that BMI may not confer uniform prognostic implications across HF phenotypes.

Biological differences between men and women further complicate this relationship. Sex-specific patterns of adipose tissue distribution, hormonal regulation, inflammatory and neurohormonal activation, and susceptibility to sarcopenia suggest that BMI may capture distinct pathophysiologic states in men and women with HF.[15–17] Nevertheless, whether sex modifies the association between BMI and outcomes within each HF phenotype remains uncertain, as prior studies have yielded inconsistent results and have often been underpowered for sex-stratified or phenotype-specific analyses.[6,9,18]

Moreover, most studies examining BMI and HF prognosis have been conducted in Western populations and have rarely evaluated sex and HF phenotype concurrently. This leaves a substantial evidence gap in

Asian populations, in whom BMI distribution, body composition, and the prevalence of low BMI differ markedly from those observed in Western cohorts.[10,19,20]

Based on these biological and epidemiological considerations, we hypothesized that the prognostic association between BMI and clinical outcomes in acute HF (AHF) differs according to both sex and HF phenotype. The Korean Heart Failure III (KorHF III) registry captures contemporary nationwide AHF care and provides a unique opportunity to address these gaps.[21,22] Using data from KorHF III, we sought to test two prespecified hypotheses: (i) that the association between BMI and clinical outcomes differs between HFpEF and HFrEF, and (ii) that these associations are further modified by sex.

Methods

Study design and population

This study analyzed data from the KorHF III registry, a nationwide, multicenter prospective cohort that enrolled 7351 patients hospitalized for AHF across 47 tertiary hospitals in Korea between March 2018 and December 2022.[21,22] Patients were eligible if they presented with signs and symptoms of HF along with either radiographic evidence of pulmonary congestion or echocardiographic structural or functional abnormalities, as defined by the 2021 European Society of Cardiology guidelines.[23] For the present analysis, we included 5271 patients with available data on echocardiography and BMI data. HFpEF was defined as a left ventricular ejection fraction (LVEF) ≥50%, and HFrEF as an LVEF <40%. Patients with HF with mid-range ejection fraction (HFmrEF), defined as an LVEF of 40%–49%, were excluded from the primary analyses because HFmrEF represents a heterogeneous transitional phenotype. However, descriptive and outcome analyses for patients with HFmrEF were additionally performed and are presented in Supplementary Table S1.

The study protocol was approved by institutional review boards (Approval No. GFIRB2019-032) and the KorHF III registry was registered on ClinicalTrials.gov (Identifier: NCT04329234). Adherence to the principles outlined in the Declaration of Helsinki was ensured throughout the study process. Funding of this study was provided by the Korean Society of Heart Failure.

Data collection and definitions

Demographic characteristics, comorbidities, laboratory values (e.g. NT-proBNP, haemoglobin), echocardiographic parameters, and medications were recorded using a standardized, web-based case report form.[21,22] BMI was calculated as weight in kilograms divided by height in metres squared and categorized into three groups using cut-off values consistent with the 2022 guidelines of the Korean Society for the Study of Obesity and the World Health Organization Asia-Pacific classification: low (<18.5 kg/m^2), normal (18.5–24.9 kg/m^2), and high (≥25.0 kg/m^2).[24] Because BMI measured at admission may be influenced by acute fluid overload, we additionally performed Cox proportional hazards regression analyses using BMI measured at discharge—when volume status was considered clinically stabilized—to examine whether similar associations were observed (Supplementary Table S2).[19,25]

Patients were followed for up to 60 months after discharge. The primary endpoint was 2-year all-cause mortality, including heart transplantation. In Korea, heart transplantation is performed almost exclusively in patients with end-stage HFrEF and accounts for <1% of events in HFpEF;[26] therefore, a unified mortality-and-transplantation endpoint was used to ensure consistency across heart failure phenotypes. Secondary endpoints included short-term (30-day), intermediate-term (1-year), and long-term (≥3-year) outcomes encompassing all-cause mortality, cardiovascular mortality and cardiovascular readmission.

Statistical analysis

Continuous variables are presented as mean ± standard deviation, and categorical variables as frequencies and percentages. Between-group comparisons were performed using ANOVA for continuous variables, and the chi-squared test for categorical variables. Survival analyses were conducted using Kaplan–Meier curves with log-rank tests.

Multivariable Cox proportional hazards models were used to assess the association between BMI and clinical outcomes, adjusting for age, sex, systolic blood pressure (SBP), comorbidities (including diabetes mellitus and chronic kidney disease), NT-proBNP levels, and the use of guideline-directed medical therapy. To explore potential non-linear associations, BMI was additionally modelled as a continuous variable using restricted cubic splines, with three knots placed at the 10th, 50th, and 90th percentiles of the BMI distribution. Interaction terms between sex and BMI and between the HF phenotype and BMI were tested. Statistical significance was defined as a two-sided *P*-value <.05. All analyses were conducted using R version 4.3.3 (R Foundation for Statistical Computing, Vienna, Austria). To evaluate potential confounding by treatment differences, additional multivariable Cox models further adjusted for HF medications at admission and at discharge were performed as sensitivity analyses (Supplementary Table S3).

Results

Baseline characteristics

Among the 5271 patients hospitalized for AHF, 3599 (68.3%) met the criteria for HFrEF and 1672 (31.7%) met the criteria for HFpEF. In both HF phenotypes, the mean age decreased with increasing BMI. Among patients with HFrEF, mean age was 72.7 ± 13.9 years in the low-BMI group and 58.8 ± 16.3 years in the high-BMI group ($P < .001$). A similar trend was observed in HFpEF, with mean age decreasing from 77.0 ± 13.8 years to 73.4 ± 12.6 years across BMI groups ($P < .001$) (*Table 1*).

The proportion of men also decreased with increasing BMI, falling from 46.0% to 30.2% in the HFrEF group ($P < .001$) and from 76.0% to 61.5% in the HFpEF group ($P < .001$). The prevalence of hypertension and diabetes increased steadily across BMI strata in both phenotypes (all $P < .05$).

Ischaemic cardiomyopathy predominated among patients with HFrEF, though its prevalence decreased from 29.5% in the low-BMI group to 25.4% in the high-BMI group ($P < .001$), whereas the prevalence of dilated cardiomyopathy increased from 19.3% to 29.0% ($P < .001$). In HFpEF, ischaemic cardiomyopathy was observed in 18.5% of patients with high BMI compared with 10.0% in those with low BMI ($P < .001$), whereas valvular heart disease was reported in 23.0% and 13.1%, respectively ($P = .019$). Patients with HFmrEF demonstrated intermediate clinical characteristics and outcomes between those with HFpEF and HFrEF (Supplementary Table S1).

Clinical status at admission and in-hospital course

SBP at admission increased modestly with BMI in both phenotypes (*Table 1*). Among patients with HFpEF, outpatient clinic admissions were more frequent in the high BMI group than in the low BMI group (35.1% vs. 21.0%, $P = .014$), whereas emergency department admissions followed the opposite pattern (64.9% vs. 79.0%, $P = .014$). Similar trends were observed in HFrEF.

Among patients with HFrEF, the use of ACE inhibitors/ARBs, β-blockers, and SGLT2 inhibitors increased with BMI (all $P < .05$). In HFpEF, the overall use of these agents did not differ significantly across BMI groups, although SGLT2 inhibitors were more commonly used in patients with low BMI (3.0% vs. 0.6%).

In-hospital death or heart transplantation occurred in 4.5% of patients with HFrEF and low BMI, compared with 1.8% of those with high BMI ($P = .038$). In HFpEF, the corresponding rates ranged between 2% and 3% without a significant gradient. Median length of stay did not vary by BMI in men; however, women

Table 1 Baseline characteristics, clinical management, and in-hospital outcomes

	HFrEF				HFpEF			
	Low BMI ($n = 224$)	Normal BMI ($n = 1976$)	High BMI ($n = 1399$)	*P*-value	Low BMI ($n = 100$)	Normal BMI ($n = 857$)	High BMI ($n = 715$)	*P*-value
Dermographics								
Age (years)	72.7 ± 13.9	68.0 ± 13.3	58.8 ± 16.3	.001	77.0 ± 13.8	74.8 ± 11.7	73.4 ± 12.6	<.001
Male (%)	46.0	32.1	30.2	<.001	76.0	58.5	61.5	<.001
Body weight (kg)	44.2 ± 6.6	59.3 ± 8.4	79.5 ± 16.1	<.001	41.5 ± 5.7	55.9 ± 8.2	71.1 ± 12.6	<.001
Height (cm)	158.1 ± 10.7	163.1 ± 9.4	164.8 ± 10.3	<.001	154.6 ± 9.4	158.9 ± 9.6	157.5 ± 10.2	.060
Wrist Circumference (cm)	71.0 ± N/A	80.5 ± 14.9	99.4 ± 7.7	<.001	73.0 ± 7.0	78.5 ± 10.6	101.6 ± 8.9	<.001
Hip Circumference (cm)	78.0 ± N/A	86.8 ± 21.7	102.0 ± 2.8	N/A	95.0 ± N/A	N/A	111.5 ± 2.1	<.001
Co-morbidities (%)								
Hypertension	45.9	53.1	57.5	.002	59.0	63.7	72.6	<.001
Diabetes mellitus	26.4	41.6	42.0	<.001	23.0	34.8	44.1	<.001
Ischaemic heart disease	26.8	31.2	25.4	.001	12.0	21.9	21.7	.068
Atrial fibrillation/atrial flutter	27.7	32.3	32.0	.388	44.0	42.3	47.8	.094
Chronic lung disease	19.1	6.9	5.0	<.001	16.0	10.7	9.1	.091
Chronic renal failure	0.5	6.4	3.4	.002	0.3	2.9	2.5	.826
Etiology								
Ischecmic CMP	29.5	31.8	25.4	<.001	10.0	18.4	18.5	<.001
Dilated CMP	19.3	22.1	29.0	<.001				
Tachycardia induced CMP	9.2	8.1	7.3	.549	13.1	17.3	15.4	.405
Valvular heart disease	4.9	5.9	5.4	.766	23.0	16.5	13.1	.019
Hypertensive CMP	4.1	3.5	8.5	<.001	8.1	7.0	10.3	<.001
Clinical status on admission								
De novo HF (%)	51.8	54.1	60.8	<.001	49.0	57.2	55.0	.258
Lung congestion (%)	64.5	65.8	60.6	.007	66	63.8	68.3	.169
SBP at admission (mmHg)	129.3 ± 27.5	128.8 ± 27.1	134.8 ± 27.9	.694	134.4 ± 30.0	137.0 ± 29.5	141.8 ± 27.4	.226
Low SBP at admission (<100 mmHg) (%)	11.6	10.4	8.0	.035	9.0	7.4	3.6	.003
DBP at admission (mmHg)	80.0 ± 18.3	79.7 ± 18.4	86.1 ± 21.0	.219	77.8 ± 17.1	77.0 ± 17.8	80.8 ± 18.0	.863
Heart rate at admission (/min)	95.8 ± 23.3	93.1 ± 24.3	96.1 ± 24.1	.087	86.0 ± 23.2	86.4 ± 25.5	84.7 ± 23.8	.315
NYHA class III-IV (%)	62.6	66.4	64	.263	66.7	65.4	64.3	.860
Admission route (%)								
Outpatient clinic	24.9	29.0	37.5	<.001	21.0	31.7	35.1	.014
Emergency department	75.1	71.0	62.5	<.001	79.0	68.3	64.9	.014
Laboratory test								
BNP (pg/mL)	10 217.8 ± 12 114.1	8112.1 ± 10 971.2	5429.4 ± 8427.9	.108	6549.3 ± 8392.9	4447.8 ± 7128.6	3416.6 ± 5753.4	.140
NT-proBNP (pg/mL)	13 570.2 ± 12 933.8	10 553.4 ± 12 257.0	6686.7 ± 9231.0	.008	8044.8 ± 8794.4	5601.4 ± 7938.3	4393.2 ± 6486.2	.288

Continued

Table 1 *Continued*

	HFrEF				HFpEF			
	Low BMI (*n* = 224)	**Normal BMI (*n* = 1976)**	**High BMI (*n* = 1399)**	***P*-value**	**Low BMI (*n* = 100)**	**Normal BMI (*n* = 857)**	**High BMI (*n* = 715)**	***P*-value**
HF medication at admission								
ACEIs/ARBs	53.6	57.5	61.5	.017	45.0	54.3	56.8	.078
ARNI	20.8	23.4	24.9	.337	2.0	2.4	2.0	.869
Beta-blocker	61.5	69.6	74.5	<.001	51.0	51.9	54.0	.656
MRAs	62.4	62.2	66.2	.055	47.0	41.9	41.0	.521
Ivabradine	10.4	8.9	8.3	.579	2.0	0.9	0.4	.188
SGLT-2 inhibitor	49.1	41.1	51.0	.001	50.0	17.0	24.6	.001
Other management								
Mechanical ventilation	4.1	5.2	4.7	.727	3.0	4.6	4.0	.672
CRRT	2.3	1.6	1.2	.343	0.0	1.8	1.1	.266
ECMO	0.9	1.4	0.8	.270	0.0	1.0	0.4	.319
LVAD	1.4	1.3	0.9	.630	0.0	0.0	0.0	N/A
Outcome								
Length of hospital staty (days)	14.7 ± 16.6	13.6 ± 23.2	11.7 ± 19.7	.324	12.9 ± 12.2	11.6 ± 28.1	10.7 ± 22.7	.362
Total costs for hospital care (US dollars)[a]	9466.2 ± 17 550.0	10 603.3 ± 23 453.0	8723.0 ± 18 573.2	.279	6250.2 ± 6849.0	8243.6 ± 20 397.0	7227.5 ± 26 004.3	.694
Patient liability costs (US dollars)	1566.6 ± 2616.5	1598.2 ± 3522.4	1331.4 ± 2348.0	.955	1585.7 ± 3511.1	1578.1 ± 2976.2	1432.6 ± 4870.9	.703
Heart transplantation at index administration	1.3	1.2	0.6	.177	0.0	0.1	0.0	.621
In-hospital death, including heart transplantation (%)	4.5	2.2	1.8	.038	3.0	2.5	2.2	.885

AHF, acute heart failure; ACEi, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; BMI, body mass index; BNP, B-type natriuretic peptide; CKD, chronic kidney disease; CMP, cardiomyopathy; CRRT, continuous renal replacement therapy; DBP, diastolic blood pressure; ECMO, extracorporeal membrane oxygenation; GDMT, guideline-directed medical therapy; HF, heart failure; HFpEF, heart failure with preserved ejection fraction; HFrEF, heart failure with reduced ejection fraction; LVAD, left ventricular assist device; MRAs, mineralocorticoid receptor antagonists; NT-proBNP, N-terminal pro–B-type natriuretic peptide; NYHA, New York Heart Association; SBP, systolic blood pressure; SGLT2i, sodium-glucose cotransporter 2 inhibitor.

[a]One US dollar is equivalent to 1335 Korean won.

with HFpEF and low BMI remained in hospital for a mean of 13.0 ± 11.0 days compared with 10.8 ± 23.5 days in the high-BMI group ($P = .033$).

Sex-stratified clinical outcomes by follow-up period

Table 2 summarizes the outcomes by BMI, sex, and follow-up period. All-cause mortality is presented using a unified definition that incorporates heart transplantation, reflecting standard clinical practice in Korea.[26] Separate mortality categories (cardiovascular and non-cardiovascular) are additionally reported to facilitate interpretability. Thirty-day mortality, transplantation, and readmission rates did not differ significantly by BMI in either HF phenotype. At one year, patients with HFpEF and low BMI experienced a higher incidence of all-cause mortality compared with those with high-BMI counterparts (29.2% vs. 11.3% in men, $P = .024$; 23.7% vs. 9.5% in women, $P < .001$). By two years, women with HFpEF and low BMI had the highest absolute event rate (35.5% vs. 14.8% in the high BMI group, $P < .001$), whereas the difference in men (29.2% vs. 17.1%) did not reach statistical significance ($P = .064$).

In HFrEF, low BMI was consistently associated with worse outcomes across follow-up intervals in both sexes. At more than three years of follow-up, all-cause mortality occurred in 35.5% of men and 35.9% of women with low BMI, compared with 13.7% and 17.5%, respectively, in the high BMI group (both $P < .001$). Overall, lower BMI was associated with worse outcomes in both HF phenotypes, with the effect most pronounced in women with HFpEF.

Survival and multivariable analysis by BMI, sex, and HF phenotype

Kaplan–Meier survival analysis demonstrated significant differences in 2-year outcomes across BMI groups within all sex and HF phenotype subgroups (*Figure 1*). Among women with HFpEF, survival declined steadily from high to low BMI (log-rank $P < .0001$). A similar trend was observed in the men with HFpEF, although the between-group differences were smaller, likely due to the limited number of patients in the low BMI group ($n = 24$). Nevertheless, the overall difference was statistically significant ($P = .0072$). Both men and women with HFrEF showed stepwise declines in survival with decreasing BMI. The separation between survival curves became more pronounced after the first year of follow-up (log-rank $P < .0001$ in men, $P = .004$ in women). In supplementary analyses modelling BMI as a continuous variable using restricted cubic splines, the risk tended to increase at lower BMI values across phenotypes, consistent with the patterns observed in the categorical analyses (Supplementary Figure S1).

Multivariable Cox regression models were used to assess the association between BMI and the risk of 2-year mortality or heart transplantation after adjusting for relevant clinical covariates (*Table 3*). In HFrEF, high BMI was independently associated with a lower risk of adverse outcomes in women (HR 0.66, 95% CI 0.45–0.96; $P = .032$), while BMI was not a significant predictor in men. In HFpEF, low BMI showed a trend toward higher risk in women, although this association did not reach statistical significance (HR 1.56, 95% CI 0.99–2.47; $P = .057$). No significant associations were observed between BMI and outcomes in men with HFpEF.

Across all models, age and NT-proBNP levels were consistently associated with worse prognosis. In men with HFrEF, hypertension (HR 1.27, $P = .039$) and ischaemic heart disease (HR 1.33, $P = .012$) were additional independent risk factors. In women with HFpEF, diabetes was associated with an increased risk (HR 1.40; $P = .035$), whereas in men with HFpEF, severe anaemia (haemoglobin <8 g/dL) conferred a particularly high-risk (HR 3.09; $P < .001$).

Discussion

Prognostic implications of BMI by HF phenotype and sex

In this nationwide cohort of Korean patients hospitalized for AHF, the prognostic implications of BMI differed according to sex and HF phenotype. Low BMI was consistently associated with worse outcomes in both men and women with HFrEF. In patients with HFpEF, the adverse association of low BMI appeared more pronounced among women; however, this trend did not reach statistical significance and warrants cautious interpretation. In contrast, BMI was not independently associated with outcomes in men with HFpEF. These findings suggest the presence of sex- and phenotype-specific modifiers in the phenomenon commonly referred to as the 'obesity paradox,' underscoring the importance of individualized interpretation of BMI in the clinical care of patients with HF.

Interpretation of the obesity paradox and sex-phenotype differences

The 'obesity paradox,' wherein patients with overweight or obesity and HF demonstrate better survival than those with normal or low BMI, has been widely reported but remains controversial due to population heterogeneity and methodological differences across studies.[20,27,28] In our study, the inverse association between BMI and mortality was most evident in women with HFrEF, aligning with prior findings that suggest the paradox is more robust in HFrEF populations.[28,29]

Among patients with HFpEF, Kaplan–Meier analyses suggested an association between low BMI and poorer outcomes, particularly among women. Although this association did not reach statistical significance in multivariable models, the observed trend may remain clinically relevant and is consistent with prior studies linking low lean mass, sarcopenia, and metabolic vulnerability to increased mortality risk in women with HFpEF.[4,7,29,30] Given that this association approached but did not achieve statistical significance, these findings warrant cautious interpretation and should be considered hypothesis-generating. These observations should also be interpreted in the context of prior multinational data demonstrating lower mortality rates among women with AHF compared with men, with the most pronounced survival advantage reported in Northeast Asia.[31,32] This broader epidemiologic pattern may partly account for the relatively lower absolute event rates observed among women in our Korean cohort.[32,33]

In contrast, BMI was not significantly associated with outcomes in men with HFpEF. This finding may reflect the limitations of BMI as a surrogate for body composition in this subgroup. Compared with women, men may have greater metabolic reserves, and BMI does not distinguish between fat and lean mass. In addition, phenotypes such as sarcopenic obesity or central adiposity—conditions that may be under-recognized in East Asian populations—can be present even among individuals with a normal BMI, thereby limiting the utility of BMI as a marker of cardiometabolic risk.[19,27,29,34,35]

Several biological mechanisms may underlie the stronger prognostic impact of low BMI observed in women with HFpEF. Women—particularly those who are postmenopausal—may be more susceptible to sarcopenia, frailty, and adverse shifts in body composition, which could amplify the clinical consequences of low BMI.[36,37] Declining oestrogen levels have also been associated with reductions in skeletal muscle mass and impaired metabolic regulation.[38] By contrast, men may retain greater skeletal-muscle reserves at comparable BMI levels, potentially attenuating the prognostic relevance of BMI in men with HFpEF.[39,40] Taken together, these sex-related differences in muscle mass, adipose distribution, and hormonal milieu may contribute to the divergent associations observed in our study.

Table 2 **Clinical outcomes by BMI category and HF phenotype**

Clinical outcomes (HFpEF)	Male low BMI ($n = 24$)	Male normal BMI ($n = 356$)	Male high BMI ($n = 275$)	*P*-value	Female low BMI ($n = 76$)	Female normal BMI ($n = 501$)	Female high BMI ($n = 440$)	*P*-value
Length of hospital stay, days	12.5 ± 15.8	13.5 ± 40.6	10.6 ± 21.6	.867	13.0 ± 11.0	10.2 ± 13.2	10.8 ± 23.5	.033
Short-term (30-day) outcomes (%)								
All-cause mortality (including transplantation)	2 (8.3)	17 (4.8)	6 (2.2)	.079	4 (5.3)	14 (2.8)	16 (3.6)	.426
Cardiovascular mortality	0 (0.0)	2 (0.6)	1 (0.4)	.869	0 (0.0)	2 (0.4)	2 (0.5)	.848
Non-cardiovascular mortality	1 (4.2)	1 (0.3)	0 (0.0)	.002	0 (0.0)	1 (0.2)	2 (0.5)	.696
All-cause readmission	5 (20.8)	21 (5.9)	15 (5.5)	.012	2 (2.6)	26 (5.2)	22 (5.0)	.630
Intermediate-term (1-year) outcomes (%)								
All-cause mortality (including transplantation)	7 (29.2)	58 (16.3)	31 (11.3)	.024	18 (23.7)	89 (17.8)	42 (9.5)	<.001
Cardiovascular mortality	0 (0.0)	12 (3.4)	4 (1.5)	.222	2 (2.6)	12 (2.4)	12 (2.7)	.949
Cardiovascular readmission	1 (4.2)	9 (2.5)	13 (4.7)	.151	6 (7.9)	31 (6.2)	44 (10.0)	.138
Primary outcomes (2-year, %)								
All-cause mortality (including transplantation)	7 (29.2)	85 (23.9)	47 (17.1)	.064	27 (35.5)	127 (25.3)	65 (14.8)	<.001
All-cause readmission	1 (4.2)	19 (5.3)	12 (4.4)	.723	3 (3.9)	20 (4.0)	15 (3.4)	.717
Cardiovascular readmission	1 (4.2)	7 (2.0)	7 (2.5)	.861	2 (2.6)	10 (2.0)	8 (1.8)	.746
Long-term (≥3-year) outcomes (%)								
All-cause mortality (including transplantation)	9 (37.5)	112 (31.5)	69 (25.1)	.128	33 (43.4)	163 (32.5)	102 (23.2)	<.001
Cardiovascular mortality	0 (0.0)	14 (3.9)	6 (2.2)	.303	2 (2.6)	16 (3.2)	16 (3.6)	.873
All-cause readmission	0 (0.0)	3 (8.8)	3 (8.6)	.909	0 (0.0)	4 (8.5)	11 (9.9)	.540
Clinical outcomes (HFrEF)	**Male low BMI ($n = 24$)**	**Male normal BMI ($n = 356$)**	**Male high BMI ($n = 275$)**	***P*-value**	**Female low BMI ($n = 76$)**	**Female normal BMI ($n = 501$)**	**Female high BMI ($n = 440$)**	***P*-value**
Length of hospital stay, days	14.4 ± 14.8	14.1 ± 23.8	11.6 ± 19.6	.278	15.1 ± 18.6	12.7 ± 21.9	11.9 ± 20.1	.097
Short-term (30-day) outcomes (%)								
All-cause mortality (including transplantation)	7 (5.8)	40 (3.0)	18 (1.8)	.023	6 (5.8)	24 (3.8)	10 (2.4)	.166
Cardiovascular mortality	2 (1.7)	2 (0.1)	2 (0.2)	.006	0 (0.0)	2 (0.3)	1 (0.2)	.841

Continued

Table 2 *Continued*

Clinical outcomes (HFrEF)	Male low BMI ($n = 24$)	Male normal BMI ($n = 356$)	Male high BMI ($n = 275$)	*P*-value	Female low BMI ($n = 76$)	Female normal BMI ($n = 501$)	Female high BMI ($n = 440$)	*P*-value
Non-cardiovascular mortality	0 (0.0)	2 (0.1)	1 (0.1)	.881	0 (0.0)	5 (0.8)	0 (0.0)	.124
All-cause readmission	4 (3.3)	86 (6.4)	56 (5.7)	.359	6 (5.8)	44 (6.9)	16 (3.8)	.088
Cardiovascular readmission	1 (0.8)	67 (5.0)	45 (4.6)	.117	4 (3.9)	31 (4.9)	8 (1.9)	.038
Non-cardiovascular readmission	3 (2.5)	18 (1.3)	10 (1.0)	.415	2 (1.9)	12 (1.9)	7 (1.7)	.971
Intermediate-term (1-year) outcomes (%)								
All-cause mortality (including transplantation)	22 (18.2)	173 (12.9)	69 (7.1)	<.001	18 (17.5)	101 (15.9)	37 (8.7)	.001
Cardiovascular mortality	2 (1.7)	31 (2.3)	17 (1.7)	.602	5 (4.9)	22 (3.5)	9 (2.1)	.264
Cardiovascular readmission	8 (6.6)	60 (4.5)	32 (3.3)	.252	4 (3.9)	31 (4.9)	20 (4.7)	.325
Primary outcomes (2-year, %)								
All-cause mortality (including transplantation)	31 (25.6)	253 (18.9)	95 (9.7)	<.001	28 (27.2)	138 (21.7)	54 (12.8)	<.001
All-cause readmission	6 (5.0)	56 (4.2)	25 (2.6)	.03	2 (1.9)	28 (4.4)	16 (3.8)	.453
Cardiovascular readmission	5 (4.1)	39 (2.9)	18 (1.8)	.055	1 (1.0)	17 (2.7)	10 (2.4)	.570
Long-term (≥3-year) outcomes (%)								
All-cause mortality (including transplantation)	43 (35.5)	338 (25.2)	134 (13.7)	<.001	37 (35.9)	183 (28.8)	74 (17.5)	<.001
Cardiovascular mortality	4 (3.3)	40 (3.0)	20 (2.0)	.34	5 (4.9)	27 (4.3)	12 (2.8)	.418
All-cause readmission	0 (0.0)	26 (13.5)	8 (5.7)	.034	0 (0.0)	8 (9.6)	4 (6.3)	.525

BMI, body mass index; CV, cardiovascular; HF, heart failure; HFpEF, heart failure with preserved ejection fraction; HFrEF, heart failure with reduced ejection fraction.

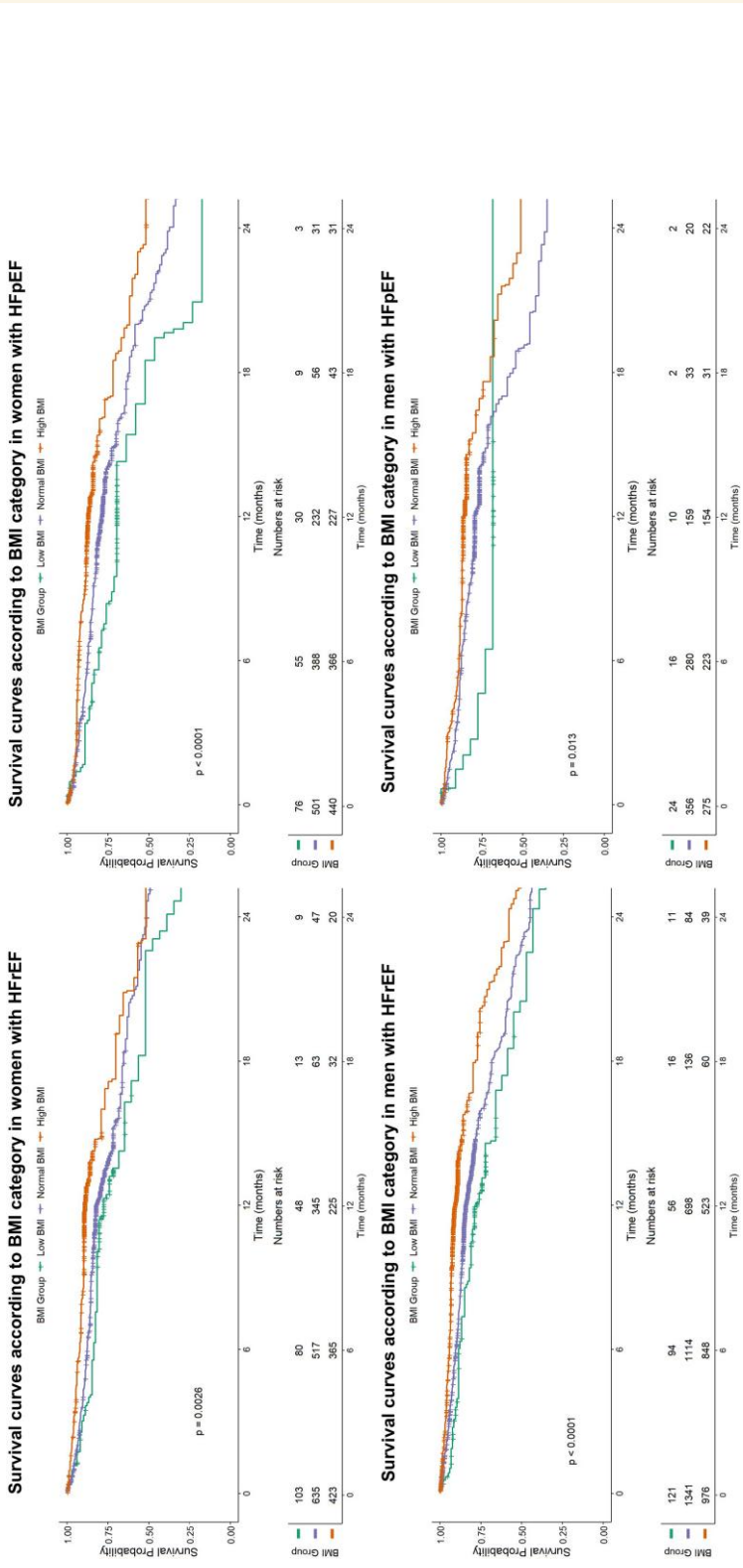


Figure 1 Kaplan–Meier survival curves according to body mass index category in patients hospitalized with acute heart failure, stratified by heart failure phenotype (HFpEF and HFrEF) and sex. Survival probabilities are presented for low, normal, and high body mass index groups. Differences between curves were assessed using the log-rank test, which showed significant survival differences across body mass index groups in all subgroups except males with HFpEF. Numbers at risk are displayed below each panel. BMI, body mass index; HF, heart failure; HFpEF, heart failure with preserved ejection fraction; HFrEF, heart failure with reduced ejection fraction

Table 3 Multivariable cox regression for 2-year death or heart transplantation by sex and BMI

Variables (HFpEF female)	HR (95% CI)	*P*-value	Variables (HFpEF male)	HR (95% CI)	*P*-value
BMI (Ref: Normal)			BMI (Ref: Normal)		
Low BMI	1.56 (0.99–2.47)	.057	Low BMI	0.88 (0.39–2.01)	.768
High BMI	0.85 (0.61–1.19)	.337	High BMI	0.71 (0.47–1.07)	.105
Age (per 1-year increase)	1.03 (1.01–1.04)	.002	Age (per 1-year increase)	1.02 (1.00–1.04)	.013
Menopausal status (Yes vs No)	1.13 (0.65–1.96)	.658	Hypertension (Yes vs No)	0.80 (0.54–1.17)	.251
Hypertension (Yes vs No)	0.75 (0.53–1.06)	.100	Diabetes mellitus (Yes vs No)	1.31 (0.90–1.90)	.163
Diabetes mellitus (Yes vs No)	1.40 (1.02–1.91)	.035	Chronic kidney disease (Yes vs No)	1.25 (0.77–2.01)	.363
Chronic kidney disease (Yes vs No)	1.36 (0.96–1.92)	.084	Ischaemic heart disease (Yes vs No)	0.71 (0.45–1.12)	.139
Ischaemic heart disease (Yes vs No)	0.83 (0.57–1.21)	.330	NT-proBNP (per 1000 pg/mL)	1.00 (1.00–1.00)	<.001
NT-proBNP (per 1000 pg/mL)	1.00 (1.00–1.00)	.001	SBP (per 10 mmHg increase)	1.00 (0.99–1.00)	.196
SBP (per 10 mmHg increase)	1.00 (0.99–1.00)	.323	Haemoglobin <8 g/dL (Yes vs No)	3.09 (1.64–5.82)	<.001
Haemoglobin <8 g/dL (Yes vs No)	1.42 (0.91–2.22)	.123	Hyponatremia <135 mmol/L (Yes vs No)	1.17 (0.75–1.83)	.501
Hyponatremia <135 mmol/L (Yes vs No)	1.33 (0.93–1.89)	.114			
Variables (HFrEF female)	**HR (95% CI)**	***P*-value**	**Variables (HFrEF male)**	**HR (95% CI)**	***P*-value**
BMI (Ref: Normal)			BMI (Ref: Normal)		
Low BMI	0.81 (0.50–1.32)	.392	Low BMI	1.26 (0.80–1.97)	.314
High BMI	0.66 (0.45–0.96)	.032	High BMI	0.90 (0.70–1.15)	.390
Age (per 1-year increase)	1.03 (1.01–1.04)	.001	Age (per 1-year increase)	1.03 (1.03–1.04)	<.001
Menopausal status (Yes vs No)	1.69 (0.89–3.22)	.110	Hypertension (Yes vs No)	1.27 (1.01–1.59)	.039
Hypertension (Yes vs No)	0.87 (0.62–1.23)	.434	Diabetes mellitus (Yes vs No)	1.03 (0.82–1.29)	.799
Diabetes mellitus (Yes vs No)	1.27 (0.92–1.76)	.142	Chronic kidney disease (Yes vs No)	1.05 (0.79–1.41)	.722
Chronic kidney disease (Yes vs No)	1.19 (0.78–1.82)	.416	Ischaemic heart disease (Yes vs No)	1.33 (1.07–1.66)	.012
Ischaemic heart disease (Yes vs No)	0.99 (0.71–1.39)	.976	NT-proBNP (per 1000 pg/mL)	1.00 (1.00–1.00)	<.001
NT-proBNP (per 1000 pg/mL)	1.00 (1.00–1.00)	<.001	SBP (per 10 mmHg increase)	1.00 (0.99–1.00)	.008
SBP (per 10 mmHg increase)	1.00 (0.99–1.00)	.370	Haemoglobin <8 g/dL (Yes vs No)	1.28 (0.59–2.78)	.529
Haemoglobin <8 g/dL (Yes vs No)	0.60 (0.15–2.46)	.480	Hyponatremia <135 mmol/L (Yes vs No)	1.27 (0.97–1.66)	.077
Hyponatremia <135 mmol/L (Yes vs No)	1.30 (0.89–1.88)	.171			

BMI, body mass index; CI, confidence interval; HFpEF, heart failure with preserved ejection fraction; HFrEF, heart failure with reduced ejection fraction; HR, hazard ratio; NT-proBNP, N-terminal pro–B-type natriuretic peptide; SBP, systolic blood pressure.

Moreover, in the setting of AHF, BMI measured at presentation may be influenced by fluid overload, potentially leading to misclassification of true nutritional or metabolic status.[25,41] To address this concern, we performed a sensitivity analysis using BMI measured at discharge, when volume status was considered clinically stabilized. The results were consistent with the primary analyses (Supplementary Table S2), suggesting that fluid-related measurement bias is unlikely to have materially influenced the observed associations.

Clinical implications

Our findings highlight important considerations for HF risk assessment and management. BMI should not be interpreted in isolation, particularly in HFpEF, in which frailty and altered body composition may substantially influence prognosis. Women with low BMI may benefit from further assessment of nutritional status, physical frailty, and muscle mass.[13,30,42] In HFrEF, the consistent association between low BMI and adverse outcomes in both sexes reinforces the prognostic relevance of body size, even in a phenotype with well-established evidence-based therapies.[27,28] Clinical decision-making may benefit from consideration of both sex and HF phenotype. Future studies should explore targeted strategies, such as nutritional support or physical rehabilitation, for high-risk subgroups and incorporate both sex and HF phenotype into their design and analytic frameworks.

Study strengths and limitations

This study leveraged a large, nationwide, contemporary registry with standardized definitions and adjudicated outcomes. The inclusion of both HFpEF and HFrEF populations, together with stratified analyses by sex, enhances the generalizability and clinical relevance of our findings. Patients with HFmrEF were excluded from the primary analyses; however, descriptive data provided in the Supplementary Material indicate that this group occupies an intermediate clinical position between HFpEF and HFrEF. Given that BMI–outcome associations were not formally evaluated in HFmrEF, the applicability of our findings to this phenotype remains uncertain and should be interpreted cautiously.

Several limitations should also be acknowledged. First, BMI was assessed only at the time of admission and therefore does not capture longitudinal changes in body weight or body composition. Although we adjusted for NT-proBNP and major comorbidities, residual confounding by unmeasured factors—such as frailty, nutritional status, inflammatory activity, and sarcopenia—cannot be excluded. Second, direct measures of lean mass, fat distribution, and physical frailty were not available, limiting the interpretation of BMI in populations

susceptible to sarcopenia or cachexia. Although BMI in AHF may be influenced by fluid overload, supplementary analyses using BMI measured at discharge yielded similar results, supporting the robustness of the findings despite this limitation. Third, residual selection bias related to missing BMI or ejection fraction data cannot be fully excluded. Excluded patients generally had more incomplete clinical profiles, and outcome comparisons stratified by these variables were not feasible.

Fourth, the observational design precludes causal inference, and the relatively small sample size in certain subgroups, such as men with low BMI and HFpEF, may have limited statistical power. In addition, because heart transplantation is rarely performed for HFpEF in Korea, the primary endpoint was defined as all-cause mortality including transplantation. Finally, as BMI distribution, adiposity patterns, and HF phenotype prevalence vary across geographic regions, caution is warranted when generalizing these findings beyond East Asian populations.[19,32,43] Nonetheless, the overall direction of the sex- and phenotype-specific associations observed in this study is broadly consistent with international data.

Despite these limitations, this study provides valuable insights into the sex- and phenotype-specific prognostic implications of BMI in AHF and underscores the importance of individualized risk stratification.

Conclusion

Among patients hospitalized for AHF, the prognostic implications of BMI varied according to both sex and HF phenotype. Low BMI was consistently associated with worse outcomes in both men and women with HFrEF and in women with HFpEF, but not in men with HFpEF. These findings suggest that low BMI may reflect frailty or reduced physiological reserve rather than body size alone, particularly among women with HFpEF. Accordingly, prognostic assessment should incorporate both sex and HF phenotype when interpreting BMI. Further studies are warranted to elucidate the underlying mechanisms and to explore targeted interventions for high-risk subgroups.

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Supplementary data

Supplementary data are available at *ESC Heart Failure* online.

Declarations

Conflict of Interest

None declared.

Data Availability

The data underlying this article were obtained from the Korean Heart Failure III (KorHF III) registry. The data are not publicly available due to institutional and ethical restrictions but may be made available from the corresponding author upon reasonable request and with permission of the KorHF III steering committee.

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Ethics Approval

The study protocol was approved by the Institutional Review Board of Gachon University Gil Medical Center (Approval No. GFIRB2019-032) and by the ethics committees of the participating institutions. The study was conducted in accordance with the principles of the Declaration of Helsinki.

Pre-registered Clinical Trial Number

The KorHF III registry was registered at ClinicalTrials.gov (Identifier: NCT04329234).

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