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Prognostic factors associated with In-Hospital Mortality Among Heart Failure Patients in a Private Tertiary Hospital in Sierra Leone: A Five-Year Cohort Study

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Abstract

Background

Evidence on in-hospital mortality among adults with heart failure (HF) in Sierra Leone is limited. We evaluated prognostic factors associated with all-cause in-hospital mortality and described mortality patterns during admission over five years.

Methods

This retrospective study analysed decompensated HF patients admitted to a private hospital in Sierra Leone from 2021 to 2025. Only the index admission per patient was included to summarize baseline characteristics, estimate in-hospital survival, and examine associations with mortality using Cox regression. Covariates violating the proportional hazards assumption were modelled with linear time interactions.

Results

Among the 765 patients studied [median age 53 years (IQR 42–61); 55.3% male], all 88 deaths occurred within the first week of admission, resulting in a mortality rate of 11.5% (95% CI 9.3–13.9). The annual mortality rate decreased from 19.7% in 2021 to 7.1% in 2025 (overall heterogeneity p = 0.008). A cold-wet haemodynamic profile was present in 77.3% of patients who died and 6.4% of survivors (unadjusted comparison, p < 0.001). In the final model, underweight [aHR = 2.44, (95% CI 1.46–4.07)], overweight [aHR = 1.93, (95% CI 1.05–3.57)], recurrent HF [aHR = 1.71, (95% CI 1.14–2.58)], and dilated cardiomyopathy (DCM) in women [aHR = 15.25, (95% CI 6.66–34.93)] were associated with higher mortality, whereas hypertensive heart disease (HHD) was associated with a lower hazard [aHR = 0.20, (95% CI 0.07–0.52)]. Baseline anaemia, chronic kidney disease (CKD) and Atrial Fibrillation (AF) showed non-proportional associations with mortality. Estimates from the complex final model are exploratory because 88 deaths supported 22 model degrees of freedom.

Conclusions

The in-hospital mortality rate among HF patients was relatively high but gradually decreased over the study period. Most deaths were reported within the first seven days and were associated with several comorbidities. These findings underscore the importance of early risk assessment and targeted management to reduce mortality in under-resourced settings.

Introduction

Heart failure (HF) constitutes a major public health concern, affecting over 64 million people globally [1, 2]. In Africa, limited healthcare facilities, late presentation, and other health conditions, compounded by infectious diseases, can worsen clinical outcomes of HF in hospitals [3–5]. Despite progress in diagnosing and treating HF, especially in sub-Saharan Africa, mortality rates remain high. This is especially true for patients admitted to hospitals, who often have advanced disease and other health issues [6, 7]. In-hospital mortality of HF reflects disease severity, care quality, and health system functioning, especially in resource-limited settings [4].

HF shows different patterns in industralized and developing countries [8–11]. In sub-Saharan Africa, HF patients are usually younger than those in developed countries and often present with advanced symptoms and complications [12–14]. Hypertensive heart disease (HHD), rheumatic valve disease, dilated cardiomyopathy (DCM), and infections are common causes, unlike ischemic heart disease (IHD) in Western countries [9, 14, 15]. Limited access to specialised cardiovascular care, inadequate diagnostic facilities, delayed presentation, and difficulties in implementing guideline-directed medical therapy (GDMT) contribute to poorer clinical outcomes [8, 16–18].

Patients admitted to the hospital with HF face a high risk of poor outcomes, including death. Research shows that being older, having low blood pressure, kidney issues, anaemia, low ejection fraction, atrial fibrillation, electrolyte problems, diabetes, and severe congestion all raise this risk [8, 9, 14]. But the extent to which these factors matter can vary across populations and health systems. Existing modelling tools were developed in Europe, North America, and Asia, where patients and healthcare systems differ markedly from those in sub-Saharan Africa [8, 15, 16, 19].

Evidence on factors associated with mortality among patients hospitalised with HF in West Africa remains limited, with particularly scarce data from Sierra Leone [8, 20]. Sierra Leone is experiencing an increasing burden of cardiovascular disease in the context of ongoing health system challenges [21, 22]. Identifying locally relevant markers may support risk stratification and allocation of constrained monitoring resources, while recognising that prognostic associations do not establish causality. The study aimed to evaluate prognostic factors associated with all-cause in-hospital mortality during the first eligible HF admission and described mortality patterns over the hospital course and across calendar years.

Methods

Study Design and Setting

This retrospective study analysed adult HF patients who were admitted to Choithrams Memorial Hospital (CMH) between January 2021 and December 2025. CMH is a private tertiary referral hospital with 60 beds and is affiliated with the University of Sierra Leone Teaching Hospital Complex. It provides tertiary-level healthcare services to the urban community, with a particular focus on advanced diagnostic evaluations and specialized medical treatment. The hospital offers 24-hour emergency and inpatient

care, as well as specialist cardiovascular services, and handles an average of 40 cardiovascular-related patients each month. While most referrals came from the Western Area, the hospital also receives patients from district hospitals and other private health facilities nationwide, indicating its extensive referral network.

Study Population

Patients aged 18 years and above, whose clinical diagnosis on admission was acute or decompensated heart failure, were evaluated. The admitting physician made the diagnosis, and the cardiologist verified it using documented signs, symptoms, echocardiographic and other clinical investigations, in accordance with the European Society of Cardiology (ESC) 2021 guidelines [23]. Variables with more than 20% missing data, primary conditions unrelated to heart failure, and cases transferred to another facility before an outcome could be determined were excluded. This study included only one hospital admission per heart failure (HF) patient. If a patient was hospitalized more than once during the study, only their first eligible admission was used in the survival analysis. As a result, each patient contributed a single observation.

Classification of Heart Failure:

HF was classified into three heart failure phenotypes: HF with reduced ejection fraction (HFrEF): LVEF ≤ 40%; HF with mildly reduced ejection fraction (HFmrEF): LVEF 41%- 49%; and HF with preserved ejection fraction (HFpEF) - LVEF ≥ 50%. HFpEF required additional documentation of structural heart disease, diastolic dysfunction and or elevated natriuretic peptide criteria. We classified the underlying HF aetiology according to prespecified clinical and echocardiographic criteria as hypertensive heart disease, ischemic heart disease, dilated cardiomyopathy, valvular heart disease, and other causes.

Data collection and quality assurance:

The following data were extracted using a standardised collection form. Variables included sex, age, occupation, body mass index (BMI), comorbidities, family history of heart failure, and specific cardiovascular family history. Medical findings included blood pressure, mean arterial pressure, heart rate, medical history of hypertension, coronary artery disease, left ventricular ejection fraction (LVEF), left ventricular end-diastolic diameter (LVDD), echocardiographic findings, laboratory test results, length of hospitalization, in-hospital outcomes, and classifications of heart failure (reduced, mildly reduced, and preserved). We also recorded the clinical presentation (self-presentation or referral) of heart failure patients and its aetiology, cardiovascular risk factors, NYHA (II-IV) classification, and hemodynamic profile (warm-dry, warm-wet, cold-wet, cold-dry). Serial codes were assigned to all data, while the extracted data were processed with strict confidentiality. Two trained investigators independently reviewed all collected data and resolved any discrepancies by consensus with the cardiologist. Logical

consistency and range checks were conducted before analysis. To assess the extent and pattern of missingness and minimise bias, we used complete-case analysis and performed sensitivity analyses.

Outcomes:

The primary outcome was all-cause in-hospital mortality during the index HF admission. Time-to-event analysis started at admission and ended at death or discharge alive. Discharge alive ended observation and was treated as censoring in the cause-specific Cox analyses. Length of stay was calculated from admission to death or discharge.

Ethical Consideration:

The study was approved by the Sierra Leone Ethics and Scientific Review Committee. Since the study used a retrospective design and analysed existing medical records, the need for individual informed consent was waived. Patient confidentiality was preserved throughout the study by restricting data access to authorised investigators only.

Statistical Analysis

All analyses were performed using Stata MP version 19.5 (StataCorp). Results were considered statistically significant if the two-tailed alpha was 0.05. Continuous variables were reported as median and interquartile range (IQR) while categorical variables were reported as frequencies and percentages. Normality was assessed visually and statistically before modelling. Baseline characteristics were compared by using Wilcoxon rank-sum tests for continuous variables and chi-squared or Fisher's exact tests for categorical variables. To assess trends in mortality over time, the Wald chi-squared test was used. The Kruskal-Wallis test was also used to compare median length of stay across years, while Kaplan-Meier curves were used to show in-hospital survival rates, treating discharged patients as non-informative censoring. Log-rank tests compared survival among different LVEF phenotypes, NYHA classes, hemodynamic profiles, sexes, patient types, age groups, and main cardiac causes, including dilated cardiomyopathy, hypertensive heart disease, valvular heart disease, and ischaemic heart disease. Multivariable Cox regression was used to identify independent prognostic factors for in-hospital mortality, following a stepwise approach that comprised demographic, clinical, and comorbidity factors. Variables were selected using stepwise elimination, and interaction terms for sex with age and DCM were included. We added calendar year to account for changes over time.

We evaluated the proportional hazards (PH) assumption for each covariate using Schoenfeld residuals and graphical inspection of log-log survival plots. Variables violating the PH assumption (anaemia, atrial fibrillation, and CKD) were modelled as time-varying covariates (TVCs), allowing their hazard ratios to vary linearly with time in hospital. The TVC approach models the total effect of a variable at time t as:

$$log\, HR\,(t) = \beta_{main} + \beta_{tvc} * t$$

where β_{main} is the baseline coefficient and β_{tvc} is the time-interaction coefficient.

Time-varying hazard ratios were plotted over days 1–8 of hospitalisation with 95% CIs. Different sequential multivariable Cox models were used to arrive at the final model. A sex-stratified subgroup multivariable Cox proportional hazards regression model was assessed using variables included in the final model. Univariable Cox hazard ratios with 95% confidence intervals and two-sided p-values are presented for all prognostic factors, along with the bivariate comparisons. All multivariable results are presented as adjusted hazard ratios (aHR) with 95% CIs.

Results

Descriptive characteristics of the study population

A total of 765 patients were evaluated with a median age of 53 years (IQR: 42–61), and a male predominance of 423 (55.3%). Approximately half of the cohort (49.0%) were aged 40–59 years, and the median Body Mass Index (BMI) was 22 kg/m² (IQR:19–25). BMI subgroup classification and clinical vital signs are presented in Table 1a-c. On admission, 389 patients (50.8%) had NYHA IV. Recurrent HF accounted for 463 admissions (60.5%), while de novo HF accounted for 302 (39.5%). The most prevalent haemodynamic profile was warm-wet (65.1%), followed by cold-wet (14.5%), warm-dry (11.6%), and cold-dry (8.8%). (Table 1). HFrEF (61.3%) was the most common phenotype diagnosed, while HFmrEF and HFpEF accounted for 13.6% and 25.1%, respectively. DCM (37.0%) was the most common cardiac aetiology, followed by HHD (31.2%), VHD (17.1%), IHD (6.3%), and other etiologies (7.1%). The leading precipitating factors were infections (37.1%), uncontrolled hypertension (24.6%), anaemia (16.2%), and atrial fibrillation (15.7%). Table 1a-c presents several cardiovascular risk factors. The comorbidity burden was high, with more than half of patients having two or more comorbidities. The findings of all laboratory parameters are also presented in Table 1a-c.

In-Hospital Mortality, Temporal Trends in Mortality and length of stay

Eighty-eight patients (88) died during hospitalisation, resulting in an in-hospital mortality proportion of 11.5% (95% CI: 9.3–13.9%). Patients who died had a shorter median observed hospital stay than those discharged alive [4 days (IQR: 3–5) versus 8 days (IQR: 4–12); χ^2 $p < 0.001$]. All observed in-hospital deaths occurred within the first seven days of admission. A total of 136 patients (17.8%) experienced adverse outcomes, which is a composite of death (n = 88) and prolonged hospitalization exceeding 14 days (n = 48) (Table 1). The crude incidence rate of both in-hospital deaths and discharge alive, as competing events, was 1.53 deaths per 100 patient-days of hospitalisation (95% CI: 1.24–1.89). Figure 1 shows the survival probability during the first 14 days post-admission and the Kaplan-Meier survival curve for the first 14 days after admission. Over the five-year study period, annual in-hospital death rates decreased each year (Fig. 2A), and this decrease was statistically significant ($\chi^2 = 13.70$; $p = 0.008$). In

contrast, the median length of stay increased from 5 days in 2021 to 8 days in 2025, but the difference was not statistically significant ($\chi^2 = 6.04$; $p = 0.196$). (Fig. 2B)

Table 1

a: Baseline characteristics, investigations, bivariate and univariable Cox proportional hazards association between characteristics of participants and in-hospital mortality

Characteristics	Total	Survived	Died		Univariable cox-proportional model	
	N = 765	N = 677	N = 88	P-value	cHR [95% CI]	P-value
Age in years, median (IQR)	53 (42, 61)	53 (42, 60)	63 (45, 72)	< 0.001[w]	1.03 [1.02–1.05]	< 0.001
Age group in years, n (%)				< 0.001		
20–39	162 (21.2)	152 (22.5)	10 (11.4)		1.00 [reference]	
40–59	375 (49.0)	347 (51.3)	28 (31.8)		1.10 [0.53–2.25]	0.804
60+	228 (29.8)	178 (26.3)	50 (56.8)		3.61 [1.83–7.12]	< 0.001
Sex, n (%)				< 0.001		
Females	342 (44.7)	318 (47.0)	24 (27.3)		1.00 [reference]	
Males	423 (55.3)	359 (53.0)	64 (72.7)		2.22 [1.40–3.52]	0.001
BMI, median (IQR)	22 (19, 25)	22 (20, 25)	21 (17, 27)	0.010 w	0.96 [0.91–1.02]	0.181
BMI category, n (%)				< 0.001		
Underweight (< 18.5kg/m2)	152 (19.9)	115 (17.0)	37 (42.0)		1.00 [reference]	
Normal (18.5-24.9kg/m2)	397 (51.9)	371 (54.8)	26 (29.5)		0.24 [0.15–0.39]	< 0.001
Overweight (25.0-29.9kg/m2)	118 (15.4)	107 (15.8)	11 (12.5)		0.35 [0.18–0.68]	0.002
Obese (> 29.9kg/m2)	98 (12.8)	84 (12.4)	14 (15.9)		0.56 [0.31–1.01]	0.056
SBP (mmHg), median (IQR)	119 (97, 144)	122 (101, 148)	82 (73, 87)	< 0.001 w	0.94 [0.93–0.95]	< 0.001
DBP (mmHg), median (IQR)	77 (66, 92)	79 (69, 93)	52 (48, 58)	< 0.001 w	0.88 [0.86–0.90]	< 0.001

Characteristics	Total	Survived	Died		Univariable cox-proportional model	
	N = 765	N = 677	N = 88	P-value	cHR [95% CI]	P-value
MAP, median (IQR)	90 (80, 112)	91 (83, 114)	61 (58, 67)	< 0.001 w	0.90 [0.88–0.92]	< 0.001
Heart Rate, median (IQR)	84 (74, 95)	83 (73, 93)	105 (87, 119)	< 0.001 w	1.03 [1.02–1.04]	< 0.001
Patient type, n (%)				0.180		
De novo	302 (39.5)	273 (40.3)	29 (33.0)		1.00 [reference]	
Recurrent	463 (60.5)	404 (59.7)	59 (67.0)		1.33 [0.86–2.06]	0.201
Circumstance of presentation, n (%)				0.330		
Self-presentation	169 (22.1)	146 (21.6)	23 (26.1)		1.00 [reference]	
Referral	596 (77.9)	531 (78.4)	65 (73.9)		0.77 [0.48–1.22]	0.268
NYHA Functional Classification, n (%)				< 0.001		
NYHA-II	130 (17.0)	130 (19.2)	0 (0.0)		-	
NYHA-III	246 (32.2)	246 (36.3)	0 (0.0)		-	
NYHA-IV	389 (50.8)	301 (44.5)	88 (100.0)		-	
Hemodynamic profile, n (%)				< 0.001		
Warm-dry	89 (11.6)	89 (13.1)	0 (0.0)		-	
Warm-wet	498 (65.1)	478 (70.6)	20 (22.7)		0.15 [0.09–0.24] †	< 0.001
Cold-wet	111 (14.5)	43 (6.4)	68 (77.3)		23.09 [14.27–37.34] †	< 0.001
Cold-dry	67 (8.8)	67 (9.9)	0 (0.0)		-	

Characteristics	Total	Survived	Died		Univariable cox-proportional model	
	N = 765	N = 677	N = 88	P-value	cHR [95% CI]	P-value
Aetiology						
Dilated Cardiomyopathy (DCM), n (%)	283 (37.0)	233 (34.4)	50 (56.8)	< 0.001	2.45 [1.62–3.69]	< 0.001
Hypertensive heart disease (HHD), n (%)	239 (31.2)	234 (34.6)	5 (5.7)	< 0.001	0.14 [0.06–0.34]	< 0.001
Valvular Heart Disease (VHD), n (%)	131 (17.1)	127 (18.8)	4 (4.5)	< 0.001	0.23 [0.09–0.64]	0.005
Ischemic Heart Disease (IHD), n (%)	48 (6.3)	45 (6.6)	3 (3.4)	0.240	0.52 [0.16–1.67]	0.275
Other Aetiology, n (%)	54 (7.1)	54 (8.0)	0 (0.0)	0.006	-	

n (%): Frequency (column percentage). IQR: interquartile range presented as (25th percentile, 75th percentile). W: p-value from Wilcoxon rank sum test for equality of medians between 2 groups. All other p-value are from the Pearson chi-square test of association.

Table 1

b: Baseline characteristics, investigations, bivariate and univariable Cox proportional hazards association between characteristics of participants and in-hospital mortality

Characteristics	Total	Survived	Died		Univariable cox-proportional model	
	N = 765	N = 677	N = 88	P-value	cHR [95% CI]	P-value
Precipitating factors						
Infection, n (%)	284 (37.1)	223 (32.9)	61 (69.3)	< 0.001	3.66 [2.34–5.71]	< 0.001
Acute Myocardial Infarction, n (%)	20 (2.6)	20 (3.0)	0 (0.0)	0.100	-	
Infective Endocarditis, n (%)	17 (2.2)	17 (2.5)	0 (0.0)	0.130	-	
Atrial Fibrillation, n (%)	120 (15.7)	104 (15.4)	16 (18.2)	0.490	1.33 [0.77–2.29]	0.309
Anaemia, n (%)	124 (16.2)	59 (8.7)	65 (73.9)	< 0.001	15.90 [10.03–25.21]	< 0.001
Uncontrolled Hypertension, n (%)	188 (24.6)	188 (27.8)	0 (0.0)	< 0.001	-	
Thyrotoxicosis, n (%)	11 (1.4)	5 (0.7)	6 (6.8)	< 0.001	5.64 [3.01–10.55]	< 0.001
Cardiovascular risk factors						
Smoking history, n (%)	154 (20.1)	132 (19.5)	22 (25.0)	0.230	1.20 [0.76–1.90]	0.432
Alcohol intake history, n (%)	82 (10.7)	77 (11.4)	5 (5.7)	0.100	0.50 [0.20–1.25]	0.139
Hypertension history, n (%)	446 (58.3)	445 (65.7)	1 (1.1)	< 0.001	0.01 [0.00–0.06]	< 0.001
Diabetes Mellitus history, n (%)	170 (22.2)	163 (24.1)	7 (8.0)	< 0.001	0.30 [0.14–0.65]	0.002
Dyslipidaemia history, n (%)	78 (10.2)	60 (8.9)	18 (20.5)	< 0.001	2.48 [1.56–3.96]	< 0.001
Heart failure history, n (%)	482 (63.0)	404 (59.7)	78 (88.6)	< 0.001	4.71 [2.44–9.08]	< 0.001
Family history of heart disease, n (%)	178 (23.3)	128 (18.9)	50 (56.8)	< 0.001	4.21 [2.80–6.32]	< 0.001
Comorbidities						

Characteristics	Total	Survived	Died		Univariable cox-proportional model	
	N = 765	N = 677	N = 88	P-value	cHR [95% CI]	P-value
Hypertensive, n (%)	297 (38.8)	261 (38.6)	36 (40.9)	0.670	1.13 [0.74–1.71]	0.569
Diabetes Mellitus (DM), n (%)	162 (21.2)	132 (19.5)	30 (34.1)	0.002	1.87 [1.21–2.87]	0.005
Stroke comorbidity, n (%)	73 (9.5)	28 (4.1)	45 (51.1)	< 0.001	11.55 [7.84–17.02]	< 0.001
Chronic Obstructive Pulmonary Disease (COPD), n (%)	141 (18.4)	125 (18.5)	16 (18.2)	0.950	0.88 [0.53–1.48]	0.636
Chronic Kidney Disease (CKD), n (%)	197 (25.8)	131 (19.4)	66 (75.0)	< 0.001	9.46 [5.88–15.22]	< 0.001
Anaemia, n (%)	201 (26.3)	124 (18.3)	77 (87.5)	< 0.001	21.30 [11.36–39.95]	< 0.001
Atrial Fibrillation, n (%)	146 (19.1)	122 (18.1)	24 (27.3)	< 0.001	0.11 [0.03–0.43]	0.002
Number of comorbidities				< 0.001		
None	77 (10.1)	76 (11.2)	1 (1.1)		1.00 [reference]	
1	274 (35.8)	268 (39.6)	6 (6.8)		1.66 [0.20–13.96]	0.641
2	414 (54.1)	333 (49.2)	81 (92.0)		15.19 [2.09–110.54]	0.007
Length of hospital stay (days), median (IQR)	7 (4, 11)	8 (4, 12)	4 (3, 5)	< 0.001 **w**	-	
LOS category				< 0.001	-	
Short (< 7 days)	358 (46.8)	270 (39.9)	88 (100.0)		-	
Intermediate (7–14)	359 (46.9)	359 (53.0)	0 (0.0)		-	
Prolonged (> 14 days)	48 (6.3)	48 (7.1)	0 (0.0)		-	
Year of presentation				0.006		

Characteristics	Total	Survived	Died		Univariable cox-proportional model	
	N = 765	N = 677	N = 88	P-value	cHR [95% CI]	P-value
2021	122 (15.9)	98 (14.5)	24 (27.3)		1.00 [reference]	
2022	134 (17.5)	114 (16.8)	20 (22.7)		0.66 [0.37–1.17]	0.159
2023	159 (20.8)	143 (21.1)	16 (18.2)		0.47 [0.25–0.87]	0.015
2024	166 (21.7)	151 (22.3)	15 (17.0)		0.42 [0.23–0.80]	0.008
2025	184 (24.1)	171 (25.3)	13 (14.8)		0.32 [0.16–0.62]	0.001

n (%): Frequency (column percentage). IQR: interquartile range presented as (25th percentile, 75th percentile). W: p-value from Wilcoxon rank sum test for equality of medians between 2 groups. All other p-value are from the Pearson chi-square test of association.

Table 1

c: Baseline characteristics, investigations, bivariate and univariable Cox proportional hazards association between characteristics of participants and in-hospital mortality

Characteristics	Total	Survived	Died		Univariable cox-proportional model	
	N = 765	N = 677	N = 88	P-value	cHR [95% CI]	P-value
Laboratory Results						
Hb g/dL, median (IQR)	12 (10, 14)	12 (10, 14)	8 (8, 9)	< 0.001 w	0.37 [0.31–0.45]	< 0.001
Na+ mmol/L, median (IQR)	139 (135, 141)	139 (135, 141)	127 (124, 130)	< 0.001 w	0.76 [0.74–0.78]	< 0.001
K+, mmol/L, median (IQR)	4 (4, 5)	4 (4, 5)	6 (5, 7)	< 0.001 w	8.16 [5.48–12.15]	< 0.001
Creatinine, mg/dL, median (IQR)	2 (1, 2)	2 (1, 2)	2 (2, 2)	< 0.001 w	2.79 [1.88–4.15]	< 0.001
BUN, mg/dL, median (IQR)	23 (21, 25)	23 (21, 25)	25 (24, 27)	< 0.001 w	1.43 [1.29–1.59]	< 0.001
BNP, pg/mL, median (IQR)	980 (947, 995)	980 (964, 995)	947 (921, 956)	< 0.001 w	0.97 [0.97–0.98]	< 0.001
Hba1c, median (IQR)	5 (5, 6)	5 (5, 5)	5 (5, 6)	< 0.001 w	2.72 [1.76–4.21]	< 0.001
RBS mmol/l, median (IQR)	5 (4, 5)	5 (4, 5)	5 (4, 5)	0.029 w	0.75 [0.68–0.82]	< 0.001
Echocardiogram assessment						
LVEDD (cm), median (IQR)	6 (5, 6)	6 (5, 6)	6 (6, 6)	< 0.001 w	4.99 [3.76–6.64]	< 0.001

n (%): Frequency (column percentage). IQR: interquartile range presented as (25th percentile, 75th percentile). W: p-value from Wilcoxon rank sum test for equality of medians between 2 groups. All other p-value are from the Pearson chi-square test of association.

Characteristics	Total	Survived	Died		Univariable cox-proportional model	
	N = 765	N = 677	N = 88	P-value	cHR [95% CI]	P-value
LVESD (cm), median (IQR)	5 (5, 5)	5 (5, 5)	5 (5, 5)	0.890 w	1.15 [0.79–1.67]	0.471
LAD (cm), median (IQR)	5 (4, 5)	5 (4, 5)	5 (4, 5)	0.008 w	0.41 [0.25–0.67]	< 0.001
E/e', median (IQR)	22 (21, 23)	22 (21, 23)	23 (20, 24)	< 0.001 w	1.51 [1.18–1.93]	0.001
RVSP (mmHg), median (IQR)	48 (40, 57)	48 (40, 57)	46 (42, 57)	0.960 w	1.00 [0.98–1.02]	0.934
MR (> mod), n (%)	223 (29.2)	199 (29.4)	24 (27.3)	0.680	0.90 [0.57–1.44]	0.670
MS, n (%)	12 (1.6)	12 (1.8)	0 (0.0)	0.210	-	
AR (mod), n (%)	56 (7.3)	3 (0.4)	53 (60.2)	< 0.001	26.08 [17.95–37.89]	< 0.001
AS, n (%)	10 (1.3)	4 (0.6)	6 (6.8)	< 0.001	5.23 [2.65–10.32]	< 0.001
TR, n (%)	451 (59.0)	400 (59.1)	51 (58.0)	0.840	0.94 [0.62–1.42]	0.755
Electrocardiogram assessment						
At. Fib, n (%)	266 (34.8)	226 (33.4)	40 (45.5)	< 0.001	-	
LBBB, n (%)	45 (5.9)	45 (6.6)	0 (0.0)	0.013	-	
PVC, n (%)	54 (7.1)	54 (8.0)	0 (0.0)	0.006	-	
LVEF Phenotype, n (%)				0.001		
HFrEF	469 (61.3)	425 (62.8)	44 (50.0)		1.00 [reference]	
HFmrEF	104 (13.6)	81 (12.0)	23 (26.1)		2.49 [1.53–4.05]	< 0.001

n (%): Frequency (column percentage). IQR: interquartile range presented as (25th percentile, 75th percentile). W: p-value from Wilcoxon rank sum test for equality of medians between 2 groups. All other p-value are from the Pearson chi-square test of association.

Characteristics	Total	Survived	Died		Univariable cox-proportional model	
	N = 765	N = 677	N = 88	P-value	cHR [95% CI]	P-value
HFpEF	192 (25.1)	171 (25.3)	21 (23.9)		1.12 [0.67–1.86]	0.674

n (%): Frequency (column percentage). IQR: interquartile range presented as (25th percentile, 75th percentile). W: p-value from Wilcoxon rank sum test for equality of medians between 2 groups. All other p-value are from the Pearson chi-square test of association.

Comparison of Survivors and Non-Survivors

The clinical profiles of survivors and non-survivors differed across nearly all major clinical domains (Table 1). Compared with survivors, those who died were older (median age 63 years [IQR: 45–72] vs 53 years [42–60]; $p < 0.001$), most frequently male (72.7% vs 53.0%; $p < 0.001$), and were underweight in nutritional status (42.0% vs 17.0%; $p < 0.001$). Those who died presented with substantially lower median systolic blood pressure (82 mmHg [IQR: 73–87] vs 122 mmHg [101–148]; $p < 0.001$), lower median diastolic pressure (52 vs. 79 mmHg; $p < 0.001$), and a higher heart rate (105 vs 83 beats per minute; $p < 0.001$). (Table 1). All 88 deaths (100%) occurred in patients presenting with NYHA functional class IV, compared with 44.5% of survivors ($p < 0.001$). Hemodynamic clinical profiling showed that the “cold-wet phenotype” was present in 77.3% of non-survivors, compared with only 6.4% of survivors ($p < 0.001$). Among LVEF phenotypes, HFmrEF was disproportionately represented among non-survivors compared with survivors (26.1% vs 12.0%; $p = 0.001$). (Table 1).

DCM (56.8% vs 34.4%; $p < 0.001$), infection (69.3% vs 32.9%; $p < 0.001$), anaemia (73.9% vs 8.7%; $p < 0.001$), CKD (75.0% vs 19.4%; $p < 0.001$), and previous stroke (51.1% vs 4.1%, $p < 0.001$), were more prevalence among patients who died. Figure 3 shows Kaplan-Meier survival curves for the different aetiologies among the study participants. Notably, comorbid hypertension, atrial fibrillation, and a history of diabetes were significantly more common among survivors. (Table 1)

Multivariable Cox Proportional Hazards Analysis

The six-stage Cox model showed better discrimination at each step, starting from C = 0.768 with only demographics in stage 1 and reaching C = 0.984 in the full model at stage 4. The parsimonious Stage 3 model (C = 0.968) had slightly lower discrimination but greater parsimony. At stage 4, global proportional hazards testing found strong time-varying effects ($p < 0.0001$), mainly due to anaemia, AF, and CKD. When the analysis was split by sex, there were interactions for age ≥ 60 and DCM, so interraction terms were added to Stage 5 and time-varying covariate (TVC) modelling was used in Stage 6. Three variables did not meet the proportional hazards assumption at Stage 5 and were modelled as TVC in Stage 6, which showed changing hazard patterns revealing dynamic hazard trajectories throughout the hospitalisation period (Supplementary Table 1). The final model (N = 765, events = 88; Wald chi^2 (22) =

483.67, p < 0.0001) included demographics, HF phenotype, aetiology, and key comorbidities. (Supplementary Table 2).

In the sequential hierarchical Cox model, the final model included demographics, HF phenotype, aetiology, and key comorbidities (Fig. 4). Underweight (aHR = 2.44, 95% CI 1.46–4.07; p = 0.001), overweight (aHR = 1.93, 95% CI 1.05– 3.57), and obesity (aHR = 1.83, 95% CI 0.98–3.43; p = 0.058) were associated with increased mortality, although obesity did not reach statistical significance. Recurrent heart failure increased adjusted mortality hazard by 71% (aHR = 1.71, 95% CI 1.14–2.58; p = 0.010) relative to de novo presentation (Fig. 4). Sex (aHR = 1.16; p = 0.674) and age ≥ 60 years (aHR = 0.80; p = 0.632) were not significant, but their interaction (aHR = 3.90; 95% CI 1.36–11.21; p = 0.011) showed that older age greatly increased mortality in males but not in females. In males ≥ 60 years, the hazard was approximately 3.6-fold higher than in females 20–59 years. (Fig. 4)

In 2024 (aHR 0.49, 95% CI 0.27–0.91; p = 0.023) and 2025 (aHR 0.57, 95% CI 0.32–1.00; p = 0.048), mortality was significantly lower compared to 2021. (Fig. 4). LVEF phenotype remained a strong independent risk factor for mortality. Both HFrEF (aHR = 0.24, 95% CI 0.14–0.40; p < 0.001) and HFpEF (aHR = 0.34, 95% CI 0.19–0.61; p < 0.001) were associated with substantially lower adjusted mortality hazard. Male sex and DCM interaction reduced mortality risk by 87.5% (aHR = 0.125, 95% CI 0.05–0.31; p < 0.001). In females, DCM increased risk by 15.25 (95% CI 6.66–34.93; p < 0.001); but in males, the effect was not significant. Further evaluation of the sex-stratified analysis of the estimated DCM in males (15.25 × 0.125 ≈ 1.91, p = 0115) was not statistically significant. Hypertensive heart disease (HHD) was associated with a lower adjusted mortality hazard (aHR = 0.20, 95% CI 0.07–0.52; p = 0.001). (Fig. 4). The DCM main effect was statistically significant in females [15.25 (95% CI 6.66–34.93; p < 0.001)], representing the hazard of DCM in females with DCM versus females without. The estimated DCM effect in males (15.25 × 0.125 ≈ 1.91) was not statistically significant in sex-stratified analysis (p = 0.115). (Fig. 4)

Anaemia was associated with a higher risk of mortality hazard for each extra day in admission (TVC coefficient 1.54 per day, 95% CI 1.14–2.10; p = 0.005). The cumulative time-varying HR on day 1 was 3.48 (95% CI, 1.17–10.31), increasing to 8.30 on day 3, 19.82 on day 5, and 73.06 on day 8. For each additional day, unresolved anaemia increased the prevailing mortality hazard by approximately 1.54 times. CKD demonstrated a similar progressive increase (TVC coefficient of 1.40 per day; 95% CI, 1.02–1.93; p = 0.037).

Figure 5 shows the time-varying adjusted hazard ratios for the effects of anaemia (Fig. 5A), chronic kidney disease (Fig. 5B), and atrial fibrillation (Fig. 5C) over the first 8 days of admission. The time-specific HR increased from 1.34 at day 1 (not specific) to 2.65 on day 3, 5.22 on day 5, and 14.45 on day 8. In contrast, atrial fibrillation had a time-varying protective effect, with a TVC coefficient of 0.51 per day (95% CI: 0.39–0.66; p < 0.001). The time-specific HR declined from 0.89 on day 1 (NS) to 0.23 on day 3, 0.06 on day 5, and 0.008 on day 8. The baseline coefficient (aHR 1.77; p = 0.516) was non-significant, confirming that the AF effect is entirely time-dependent.

Discussion

This five-year retrospective cohort study provided evidence for adults hospitalised with HF in Sierra Leone. The all-cause mortality rate was 11.5%, with all deaths occurring within the first seven days, highlighting the early admission period as a vulnerable window. From 2021 to 2025, annual mortality gradually decreased, but because this is an observational design, the changes related to treatment or health-system improvements should be met with caution. The strongest unadjusted bedside marker was a cold-wet haemodynamic profile. Multivariable analyses identified associations involving BMI, recurrent HF, LVEF phenotype and aetiology, with exploratory evidence of effect modification by sex and non-proportional associations for baseline anaemia, CKD and AF.

The observed in-hospital mortality is within the range reported in several recent sub-Saharan African studies, including 8.6% in central Ethiopia, 14.9% in a multicentre Ethiopian cohort, and 8.7% in the contemporary THESUS-HF II registry [24–26]. However, considerable study heterogeneity exists, with some recent African cohorts reporting in-hospital mortality below 5% [27, 28]. Mortality nevertheless remains higher than the approximately 4%–6% reported in many contemporary registries from high-income or better-resourced settings [9, 11, 29]. These differences may reflect later presentation, greater haemodynamic compromise, differences in HF aetiology and comorbidity burden, limited access to diagnostic and therapeutic resources, less implementation of guideline-directed medical therapy, and broader health-system constraints [8, 14, 17]. The concentration of deaths during the first week of admission is particularly relevant for resource-constrained hospitals. It supports prioritising clinical surveillance, haemodynamic assessment and monitoring resources during the initial days of hospitalisation.

The all-cause in-hospital mortality dropped steadily from 19.7% in 2021 to 7.1% in 2025. Even after adjusting for other confounding factors, the progressive decrease in mortality over the study period suggests genuine improvements in clinical care rather than differences in the types of HF patients treated. Similar improvements in survival have been reported in longitudinal HF cohorts and hospitalisation studies from other healthcare settings [30–33]. The favourable trend observed in our cohort may reflect a combination of earlier recognition and referral, improved multidisciplinary management, better identification and treatment of precipitating factors and comorbidities, greater implementation of evidence-based therapy, and broader improvements in hospital quality of care [14, 33, 34]. Nevertheless, additional prospective multicentre studies incorporating detailed process-of-care indicators are required to determine the relative contributions of patient characteristics, clinical interventions and health-system improvements to this encouraging trend.

The strong association observed between the cold-wet haemodynamic profile and in-hospital mortality in our study is clinically plausible. This phenotype reflects the coexistence of congestion and peripheral hypoperfusion and therefore represents advanced haemodynamic and circulatory compromise [35–37]. However, because the association was derived from unadjusted analysis, it should not be interpreted as an independent prognostic effect. In settings where invasive haemodynamic monitoring is unavailable,

systematic bedside assessment of congestion and perfusion may provide a pragmatic means of identifying patients requiring urgent clinical review, closer monitoring and prompt phenotype-directed management [23, 35, 36]. Management should include appropriate decongestive therapy and correction of precipitating factors, while intravenous inotropes should be reserved for carefully selected patients with persistent hypoperfusion, hypotension or cardiogenic shock despite adequate filling status [23, 35, 37].

In this cohort, HFmrEF was associated with a higher adjusted mortality hazard than HFrEF and HFpEF. This finding contrasts with much of the published literature, in which HFmrEF generally has a lower mortality risk than HFrEF and a prognosis similar to or intermediate between HFrEF and HFpEF [38, 39]. However, this should be interpreted cautiously because of the relatively small HFmrEF subgroup, potential imprecision and interobserver variability in LVEF measurement, particularly around phenotype-defining thresholds, with possible residual differences in aetiology, comorbidity burden, treatment, and disease severity [40]. Recent South African evidence found no significant difference in in-hospital mortality among the three LVEF phenotypes, further indicating that the present association may be cohort-specific [41]. These findings therefore do not establish that HFmrEF is intrinsically associated with greater mortality, and replication in larger, prospectively characterized multicentre African cohorts is required before this observation influences clinical practice.

DCM emerged as the most significant aetiology-specific prognostic factor for in-hospital mortality. A significant interaction between DCM and sex was also observed, with a strong association between DCM and mortality among women but no statistically significant association in men. The high prevalence of DCM-related deaths likely reflects advanced ventricular remodelling, severe systolic dysfunction, unmeasured aetiological heterogeneity, arrhythmia burden, and limited myocardial reserve, all of which are plausible contributors to poor prognosis in DCM. 42,43] [45, 46] Previous studies have also identified sex-related differences in the clinical presentation, progression, and outcomes of DCM, although the magnitude of disparity observed in our cohort appears greater than that reported in other populations [44, 45]. Further prospective studies are warranted to elucidate the underlying sex-specific differences in DCM outcomes in Africa.

Conversely, HHD was associated with a lower adjusted mortality hazard. Although HHD is a predominant cause of HF in African cohorts, evidence that it consistently confers lower mortality than other HF aetiologies remains limited and heterogeneous [46, 47]. The observed association should therefore not be interpreted as evidence that HHD is biologically protective; rather, it may reflect differences in disease stage, ventricular function and myocardial reserve, comorbidity burden, treatment exposure, or residual case-mix differences [48]. HHD develops along a continuum of pressure-overload remodelling, initially characterised by compensatory left ventricular hypertrophy but potentially progressing to myocardial fibrosis, diastolic dysfunction, chamber dilatation, and systolic failure [49]. Patients classified as having HHD in this cohort may consequently have had less advanced myocardial dysfunction than those with DCM or other severe aetiologies. Nevertheless, this explanation remains speculative because the extent of fibrosis and remodelling was not directly measured. These findings emphasise the importance of

considering the underlying HF aetiology alongside clinical severity when estimating prognosis and prioritising management.

Underweight was independently associated with higher in-hospital mortality, consistent with evidence linking low BMI, malnutrition, frailty and cardiac cachexia to poor HF outcomes [50–52] [53–55]. The higher adjusted hazard among overweight patients was less expected and may reflect congestion-related distortion of admission weight, residual confounding, sparse events or multiple comparisons. Conversely, the increased adjusted hazard observed among overweight patients requires cautious interpretation, particularly because admission weight in decompensated HF can be influenced by congestion, as it does not differentiate adipose tissue from lean mass. Although numerous studies have documented an "obesity paradox" in heart failure, recent evidence suggests that this apparent protective effect may diminish or disappear after rigorous adjustment for clinical confounders and the use of more precise measures of adiposity, implying that excess adiposity could still contribute to adverse outcomes. [50, 53].

Patients admitted with recurrent heart failure experienced a higher risk of in-hospital mortality compared with de novo presentation, underscoring recurrent hospitalisation as a marker of disease progression and vulnerability [25]. These findings are consistent with previous studies demonstrating a stepwise increase in all-cause mortality with successive heart failure hospitalisations [53–56]. This evidence emphasizes the necessity for aggressive optimization of guideline-directed medical therapy and intensified post-discharge follow-up in this high-risk population [36, 57]. Furthermore, our study highlights the importance of robust post-discharge follow-up programs to minimise rehospitalisations.

23,

Baseline anaemia and CKD showed time-dependent associations with mortality, indicating that the strength of their association varied over time rather than progressively worsening during admission. These findings indicate that their prognostic effects were not constant over the hospital course and should not be interpreted as evidence of progressive daily deterioration, as serial haemoglobin and renal-function trajectories were not modelled. Declining renal function contributes to neurohormonal activation, chronic inflammation, endothelial dysfunction, anaemia, and fluid overload, all of which exacerbate cardiovascular deterioration and increase mortality risk [23, 58–60]. In patients with heart failure, the coexistence of CKD, referred to as cardiorenal syndrome, further amplifies the risk, particularly with prolonged hospitalisation and worsening renal impairment [59, 60].

The declining time-dependent association between AF and mortality was unexpected and contrasts with most published evidence, which generally associates concomitant AF with higher mortality and HF-related hospitalisation [61, 63]. Several plausible explanations may account for this finding, including sparse late events, survivor selection, competing discharge, and heterogeneity in how AF was classified, as a precipitating factor, pre-existing comorbidity or ECG finding. Differences in HF phenotype, AF burden, and treatment-related confounding might also have contributed. This association should not be

interpreted as evidence that AF was protective, but rather as an exploratory finding requiring confirmation in a prespecified and adequately powered analysis.

Our cohort observed that increasing age was significantly associated with higher in-hospital mortality among men. The observed age-by-sex interaction suggests that the association between advancing age and mortality may be stronger in men than in women, whereas this sex-related difference appears less pronounced at younger ages. This finding is compatible with evidence that HF phenotype, comorbidity burden and outcomes vary across age and sex [64, 65]. Ageing-related changes in cardiovascular structure, ventricular–arterial coupling, comorbidities and sex-specific physiological adaptations may contribute to these differences [66]. However, the interaction should be considered hypothesis-generating because the present study was not designed to identify the biological or clinical mechanisms underlying it. Although previous literature supports important sex differences in HF phenotypes and outcomes, confirmation in larger, adequately powered cohorts is required.

Clinical implications

These findings suggest that pragmatic, cost-effective approaches can support early risk assessment for patients admitted with HF in Sierra Leone and similar West African regions. At admission, it is important to evaluate haemodynamic profiling for congestion and perfusion, and to assess blood pressure, haemoglobin, renal function, electrolytes, and nutritional status. Patients with recurrent HF or haemodynamic compromise may require intensified monitoring during early admission. At the health-system level, stronger referral pathways, access to echocardiography and essential laboratory testing, evidence-based treatment protocols, discharge planning, and early follow-up are reasonable priorities. The findings about women with DCM and patients with HFmrEF need to be confirmed by further studies before being used to guide specific clinical decisions.

Strengths and limitations:

The strengths of the study are its five-year cohort, inclusion of an index admission per patient, detailed clinical records, and robust laboratory and echocardiographic data. It also analyses event timing in a country with limited HF outcome data. Limitations include the retrospective single-centre private-hospital design; referral and selection bias; possible exposure and aetiologic misclassification; unreported variable-level missingness and complete-case selection. The small number of deaths relative to model complexity increases risk of overfitting. Lack of internal or external validation; and limited generalisability to public and district hospitals.

Conclusion

In this five-year cohort of adults hospitalized with heart failure in Sierra Leone, in-hospital mortality was 11.5%, with all deaths occurring within seven days of admission. Mortality proportions declined across calendar years. Several admission characteristics were associated with mortality, including a cold-wet haemodynamic profile, BMI, recurrent HF, phenotype, and aetiology, while baseline anaemia, CKD, and AF

showed non-proportional associations. The complex interaction and time-varying estimates are exploratory because of sparse events, competing discharge and overfitting risk. The findings support early clinical and laboratory assessment but require confirmation in larger multicentre cohorts.

Declarations

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References

1. GBD 2021 Diseases and Injuries Collaborators. Global incidence, prevalence, years lived with disability (YLDs), disability-adjusted life-years (DALYs), and healthy life expectancy (HALE) for 371 diseases and injuries in 204 countries and territories and 811 subnational locations, 1990–2021: a systematic analysis for the Global Burden of Disease Study 2021. Lancet. 2024;403(10440):2133–61.
2. Savarese G, Lund LH. Global Public Health Burden of Heart Failure. Card Fail Rev. 2017;3(1):7–11.
3. Sliwa K, Mayosi BM. Recent advances in the epidemiology, pathogenesis and prognosis of acute heart failure and cardiomyopathy in Africa. Heart. 2013;99(18):1317–22.
4. Damasceno A, Mayosi BM, Sani M, Ogah OS, et al. The causes, treatment, and outcome of acute heart failure in 1006 Africans from 9 countries. Arch Intern Med. 2012;172(18):1386–94.
5. Bloomfield GS, Barasa FA, Doll JA, Velazquez EJ. Heart failure in sub-Saharan Africa. Curr Cardiol Rev. 2013;9(2):157–73.

6. Dokainish H, Teo K, Zhu J, et al. Global mortality variations in patients with heart failure: results from the INTER-CHF study. Lancet Glob Health. 2017;5(7):e665–72.
7. Ponikowski P, Anker SD, AlHabib KF, Cowie MR, Force TL, Hu S, Jaarsma T, Krum H, Rastogi V, Rohde LE, Samal UC, Shimokawa H, Budi Siswanto B, Sliwa K, Filippatos G. Heart failure: preventing disease and death worldwide. ESC Heart Fail. 2014;1(1):4–25.
8. Mbanze I, Spracklen TF, Jessen N, Damasceno A, Sliwa K. Heart failure in low-income and middle-income countries. Heart. 2025;111(8):341–51. 10.1136/heartjnl-2024-324176.
9. Khan MS, Shahid I, Bennis A, Rakisheva A, Metra M, Butler J. Global epidemiology of heart failure. Nat Rev Cardiol. 2024;21(10):717–34. 10.1038/s41569-024-01046-6.
10. Yogeswaran V, Hidano D, Diaz AE, et al. Regional variations in heart failure: a global perspective. Heart. 2024;110(1):11–8. 10.1136/heartjnl-2022-321295.
11. Shahim B, Kapelios CJ, Savarese G, Lund LH. Global public health burden of heart failure: an updated review. Card Fail Rev. 2023;9:e11. 10.15420/cfr.2023.05.
12. Cherukupalli R, Matharu A, Akwanalo C, Bagha S, Bundhoo K, Jevallee H, Essackjee S, Sarr SA, Kimambo D, Chiwanza M, Bulaya T, Muni SK. Heart Failure Management from African Perspective: A consensus statement on the urgent need for action. Cardiovasc J Afr. 2025;36(3):353–62. Epub 2025 Sep 2. PMID: 41790485.
13. Ogah OS, Sani MU, et al. Geographic variations, peculiarities, and management of heart failure in sub-Saharan Africa. Curr Heart Fail Rep. 2025;22(1):29.
14. Siddikatou D, Mandeng Ma Linwa E, Ndobo V, et al. Heart failure outcomes in sub-Saharan Africa: a scoping review of recent studies conducted after the 2022 AHA/ACC/HFSA guideline release. BMC Cardiovasc Disord. 2025;25(1):302. 10.1186/s12872-025-04756-y.
15. Tromp J, Teng THK. Regional differences in the epidemiology of heart failure. Korean Circ J. 2024;54(10):591–602. 10.4070/kcj.2024.0199.
16. Wauye VM, Ahadzi D, Udayakumar K, Ngeno GT. Heart failure in sub-Saharan Africa: systems of care. J Asian Pac Soc Cardiol. 2025;4:e16. 10.15420/japsc.2024.37.
17. Satheesh G, Dhurjati R, Alston L, et al. Use of guideline-recommended heart failure drugs in high-, middle-, and low-income countries: a systematic review and meta-analysis. Glob Heart. 2024;19(1):74.
18. Agarwal A, Husain MJ, Datta B, Kishore SP, Huffman MD. Access to heart failure medicines in low- and middle-income countries: an analysis of essential medicines lists, availability, price, and affordability. Circ Heart Fail. 2022;15(4):e008971.
19. Walli-Attaei M, Joseph P, Johansson I et al. Characteristics, management, and outcomes in women and men with congestive heart failure in 40 countries at different economic levels: an analysis from the Global Congestive Heart Failure registry. Lancet Glob Health. 2024;12(3).
20. Agbor VN, Essouma M, Ntusi NAB, Nyaga UF, Bigna JJ, Noubiap JJ. Heart failure in sub-Saharan Africa: a contemporaneous systematic review and meta-analysis. Int J Cardiol. 2018;257:207–15.

21. Russell JBW, Koroma TR, Sesay S, et al. Burden of cardiometabolic risk factors and preclinical target organ damage among adults in Freetown, Sierra Leone: a community-based health-screening survey. BMJ Open. 2023;13(5):e067643.
22. Mensah GA, Roth GA, Fuster V. The global burden of cardiovascular diseases and risk factors: 2020 and beyond. J Am Coll Cardiol. 2019;74(20):2529–32.
23. McDonagh TA, Metra M, Adamo M, Gardner RS, Baumbach A, Böhm M, et al. 2023 Focused update of the 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure. Eur Heart J. 2023;44(37):3627–39.
24. Solela G, Yimer YS. Clinical characteristics, treatment patterns, and outcomes of hospitalized patients with acute heart failure in central Ethiopia: a retrospective observational study. BMC Cardiovasc Disord. 2024;24:241. 10.1186/s12872-024-03905-z.
25. Bekele F, Sheleme T, Tsegaye T, et al. Prevalence and risk factors of mortality among heart failure patients in low-resource setting hospitals: a multicentre prospective observational study. Front Cardiovasc Med. 2024;11:1429513. 10.3389/fcvm.2024.1429513.
26. Sliwa K et al. Aetiology, management, and outcomes of acute heart failure in 17 African countries (THESUS-HF II): a prospective, multicentre observational study. Lancet. 2026.
27. Adamu UG et al. Clinical characteristics and outcomes of adults with acute heart failure according to left ventricular ejection fraction phenotype in Johannesburg, South Africa. ESC Heart Fail. 2025.
28. Nkoke C, et al. Contemporary profile and in-hospital outcomes of decompensated heart failure in a semi-rural setting in Cameroon: the Buea Heart Study. Glob Heart. 2025. 10.5334/gh.1442.
29. Ide T, Kaku H, Matsushima S, et al. Clinical characteristics and outcomes of hospitalized patients with heart failure from the large-scale Japanese Registry of Acute Decompensated Heart Failure. Circ J. 2021;85(9):1438–50.
30. Clark KAA, Reinhardt SW, Chouairi F, et al. Trends in heart failure hospitalizations in the United States from 2008 to 2018. J Card Fail. 2022;28(2):171–80.
31. Garred CH, Malmborg M, Malik ME et al. Age-specific mortality trends in heart failure over 25 years: a retrospective Danish nationwide cohort study. Lancet Healthy Longev. 2024;5(5).
32. Bozkurt B, Ahmad T, Alexander KM, et al. HF STATS 2024: heart failure epidemiology and outcomes statistics—an updated 2024 report from the Heart Failure Society of America. J Card Fail. 2025;31:66–116.
33. Mebazaa A, Davison B, Chioncel O, et al. Safety, tolerability and efficacy of up-titration of guideline-directed medical therapies for acute heart failure (STRONG-HF): a multinational, open-label, randomised trial. Lancet. 2022;400(10367):1938–52.
34. Sokos G, Kido K, Panjrath G, et al. Multidisciplinary care in heart failure services. J Card Fail. 2023;29(6):943–58.
35. Chioncel O, Mebazaa A, Maggioni AP, et al. Acute heart failure congestion and perfusion status—impact of the clinical classification on in-hospital and long-term outcomes: insights from the ESC-EORP-HFA Heart Failure Long-Term Registry. Eur J Heart Fail. 2019;21(11):1338–52.

36. López-Vilella R, et al. Clinical profiling of patients admitted with acute heart failure: a comprehensive survival analysis. Front Cardiovasc Med. 2024;11:1381514.
37. Masip J, Peacock WF, Arrigo M, et al. Acute heart failure in the 2021 ESC heart failure guidelines: a scientific statement from the Association for Acute Cardiovascular Care of the European Society of Cardiology. Eur Heart J Acute Cardiovasc Care. 2022;11(2):173–85.
38. Raja DC, Samarawickrema I, Das S, et al. Long-term mortality in heart failure with mid-range ejection fraction: systematic review and meta-analysis. ESC Heart Fail. 2022;9(6):4088–99.
39. Kapłon-Cieślicka A, Benson L, Chioncel O, et al. A comprehensive characterization of acute heart failure with preserved versus mildly reduced versus reduced ejection fraction: insights from the ESC-HFA EORP Heart Failure Long-Term Registry. Eur J Heart Fail. 2022;24(2):335–50.
40. Vancheri F, Longo G, Henein MY. Left ventricular ejection fraction: clinical, pathophysiological, and technical limitations. Front Cardiovasc Med. 2024;11:1340708.
41. Adamu UG, Maseko M, Tsabedze N, et al. Clinical characteristics and outcomes of adults with acute heart failure in a South African teaching hospital. ESC Heart Fail. 2025;12(5):3643–54.
42. Arbelo E, Protonotarios A, Gimeno JR, et al. 2023 ESC Guidelines for the management of cardiomyopathies. Eur Heart J. 2023;44(37):3503–626.
43. Fairweather D, Beetler DJ, Musigk N, et al. Sex and gender differences in myocarditis and dilated cardiomyopathy: an update. Front Cardiovasc Med. 2023;10:1129348. 10.3389/fcvm.2023.1129348.
44. Owen R, Buchan RJ, Frenneaux M, et al. Sex differences in the clinical presentation and natural history of dilated cardiomyopathy. JACC Heart Fail. 2024;12(2):352–63. 10.1016/j.jchf.2023.10.009.
45. Stroeks SLVM, Oko-Osi S, Arasu A, et al. Sex differences in dilated cardiomyopathy: evidence gaps and future directions. J Am Coll Cardiol. 2026;87(6):723–35.
46. Karaye KM, Dokainish H, ElSayed A, et al. Clinical profiles and outcomes of heart failure in five African countries: results from the INTER-CHF study. Glob Heart. 2021;16(1):50.
47. Masenga SK, Kirabo A. Hypertensive heart disease: risk factors, complications and mechanisms. Front Cardiovasc Med. 2023;10:1205475.
48. Díez J, Butler J. Growing heart failure burden of hypertensive heart disease: a call to action. Hypertension. 2023;80(1):13–21.
49. Ogah OS, Sliwa K, Akinyemi JO, Falase AO, Stewart S. Hypertensive heart failure in Nigerian Africans: insights from the Abeokuta Heart Failure Registry. J Clin Hypertens (Greenwich). 2015;17(4):263–72.
50. Nguyen HTT, Ha TTT, Tran HB, et al. Relationship between BMI and prognosis of chronic heart failure outpatients in Vietnam: a single-center study. Front Nutr. 2023;10:1251601.
51. Liang L, Zhao X, Huang L, et al. Prevalence and prognostic importance of malnutrition, as assessed by four different scoring systems, in elderly patients with heart failure. Nutr Metab Cardiovasc Dis. 2023;33(5):978–86.

52. Osório AF, Ribeiro ÉCT, Parahiba SM, et al. Prognostic value of nutritional screening tools in hospitalized patients with decompensated heart failure: a systematic review and meta-analysis. Nutr Res. 2023;120:1–19.
53. Butt JH, Petrie MC, Jhund PS, et al. Anthropometric measures and adverse outcomes in heart failure with reduced ejection fraction: revisiting the obesity paradox. Eur Heart J. 2023;44(13):1136–53.
54. Lindmark K, Boman K, Stålhammar J, et al. Recurrent heart failure hospitalizations increase the risk of cardiovascular and all-cause mortality in patients with heart failure in Sweden: a real-world study. ESC Heart Fail. 2021;8(3):2144–53.
55. Shakoor A, Abou Kamar S, Malgie J, et al. The different risk of new-onset, chronic, worsening, and advanced heart failure: a systematic review and meta-regression analysis. Eur J Heart Fail. 2024;26(2):216–29.
56. Greene SJ, Bauersachs J, Brugts JJ, et al. Worsening heart failure: nomenclature, epidemiology, and future directions. J Am Coll Cardiol. 2023;81(4):413–24.
57. Heidenreich PA, Bozkurt B, Aguilar D, et al. 2022 AHA/ACC/HFSA guideline for the management of heart failure. Circulation. 2022;145(18):e895–1032.
58. Ru SC, Yu X, Chen M, et al. Incidence, mortality and predictors of acute kidney injury in patients with heart failure: a systematic review. ESC Heart Fail. 2023;10(6):3237–49. 10.1002/ehf2.14520.
59. Banerjee D, Rosano G, Herzog CA. Acute kidney injury in acute heart failure—when to worry and when not to worry? Nephrol Dial Transpl. 2025;40(1):10–9.
60. Cooper L, DeVore AD, Mentz RJ, et al. Patients hospitalized with acute heart failure, worsening renal function and persistent congestion are at high risk for adverse outcomes despite current medical therapy. Clin Cardiol. 2023;46(10):1140–8. 10.1002/clc.24080.
61. Mundisugih J, Franke KB, Tully PJ, Munawar DA, Kumar S, Mahajan R. Prevalence and prognostic implication of atrial fibrillation in heart failure subtypes: systematic review and meta-analysis. Heart Lung Circ. 2023;32(6):666–77. 10.1016/j.hlc.2023.02.009.
62. Kroshian G, Joseph J, Kinlay S, et al. Atrial fibrillation and risk of adverse outcomes in heart failure with reduced, mildly reduced, and preserved ejection fraction: a systematic review and meta-analysis. J Cardiovasc Electrophysiol. 2024;35(4):715–26.
63. Agbor VN, Tianyi FL, Aminde LN, et al. Burden of atrial fibrillation among adults with heart failure in sub-Saharan Africa: a systematic review and meta-analysis. BMJ Open. 2022;12(10):e061618.
64. Qiu W, Wang W, Wu S, Zhu Y, Zheng H, Feng Y. Sex differences in long-term heart failure prognosis: a comprehensive meta-analysis. Eur J Prev Cardiol. 2024;31(17):2013–23.
65. Mogos MF, Muchira JM, Park C, Osmundson S, Piano MR. Age-stratified sex differences in heart failure with preserved ejection fraction among adult hospitalizations. J Cardiovasc Nurs. 2025;40(1):84–93.
66. Rosano GMC, Stolfo D, Anderson L, et al. Differences in presentation, diagnosis and management of heart failure in women: a scientific statement of the Heart Failure Association of the ESC. Eur J Heart Fail. 2024;26(8):1669–86.

Figures

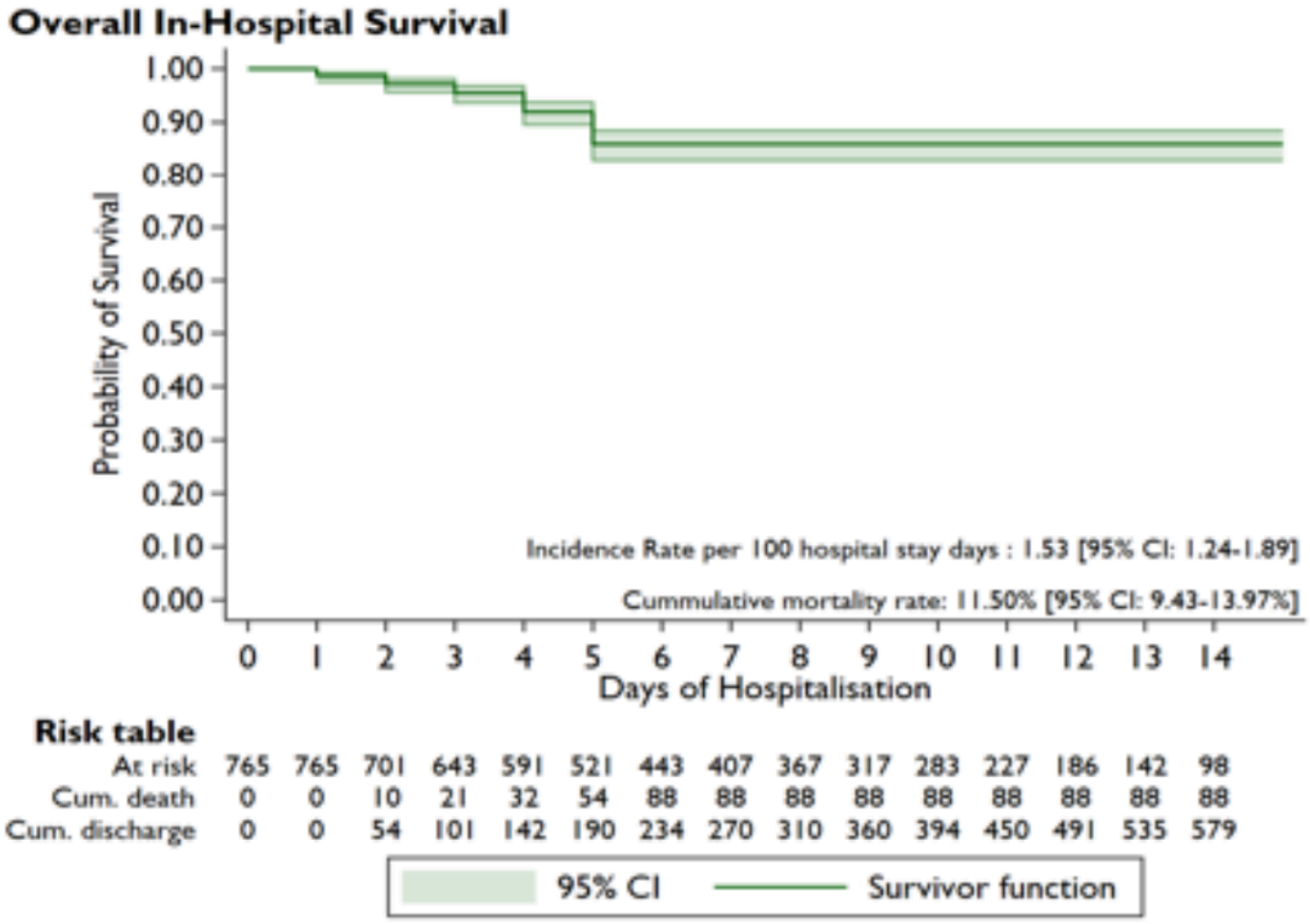


Figure 1: Kaplan Meier Survival rate curve among all study participants

Figure 1

See image above for figure legend.

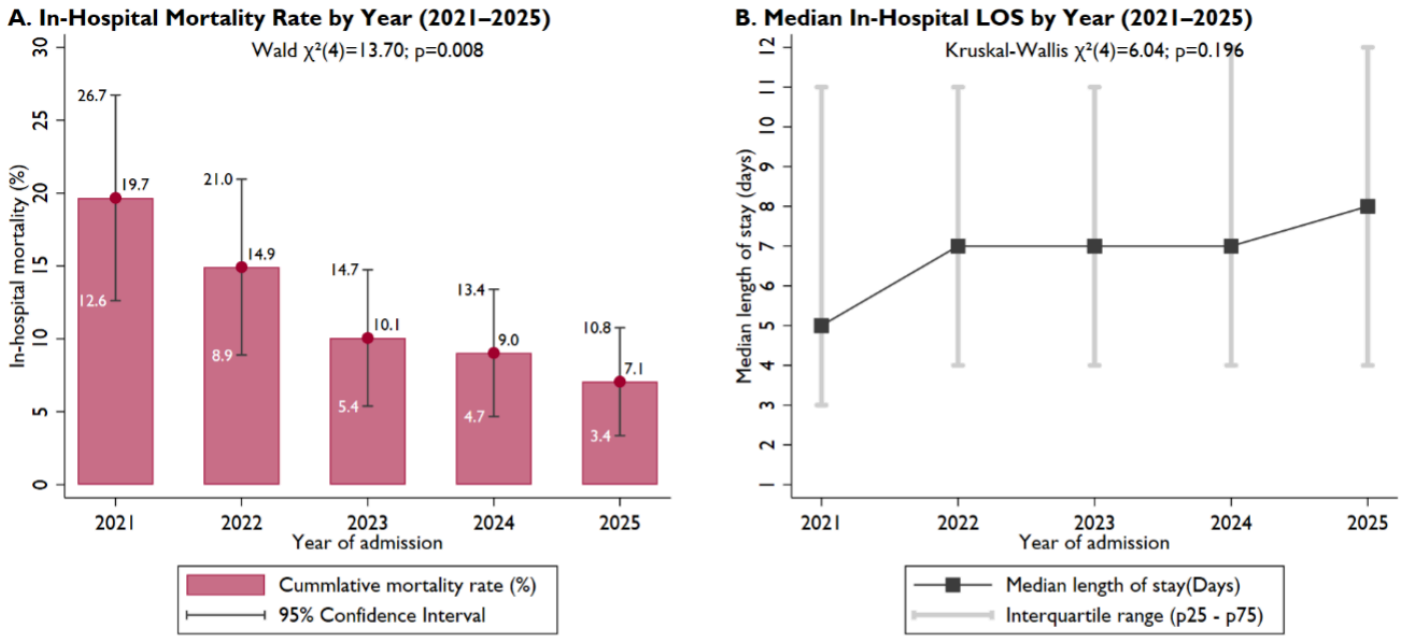


Figure 2

Trend of in-hospital mortality rate and length of hospital stay between 2021 and 2025 among study participants

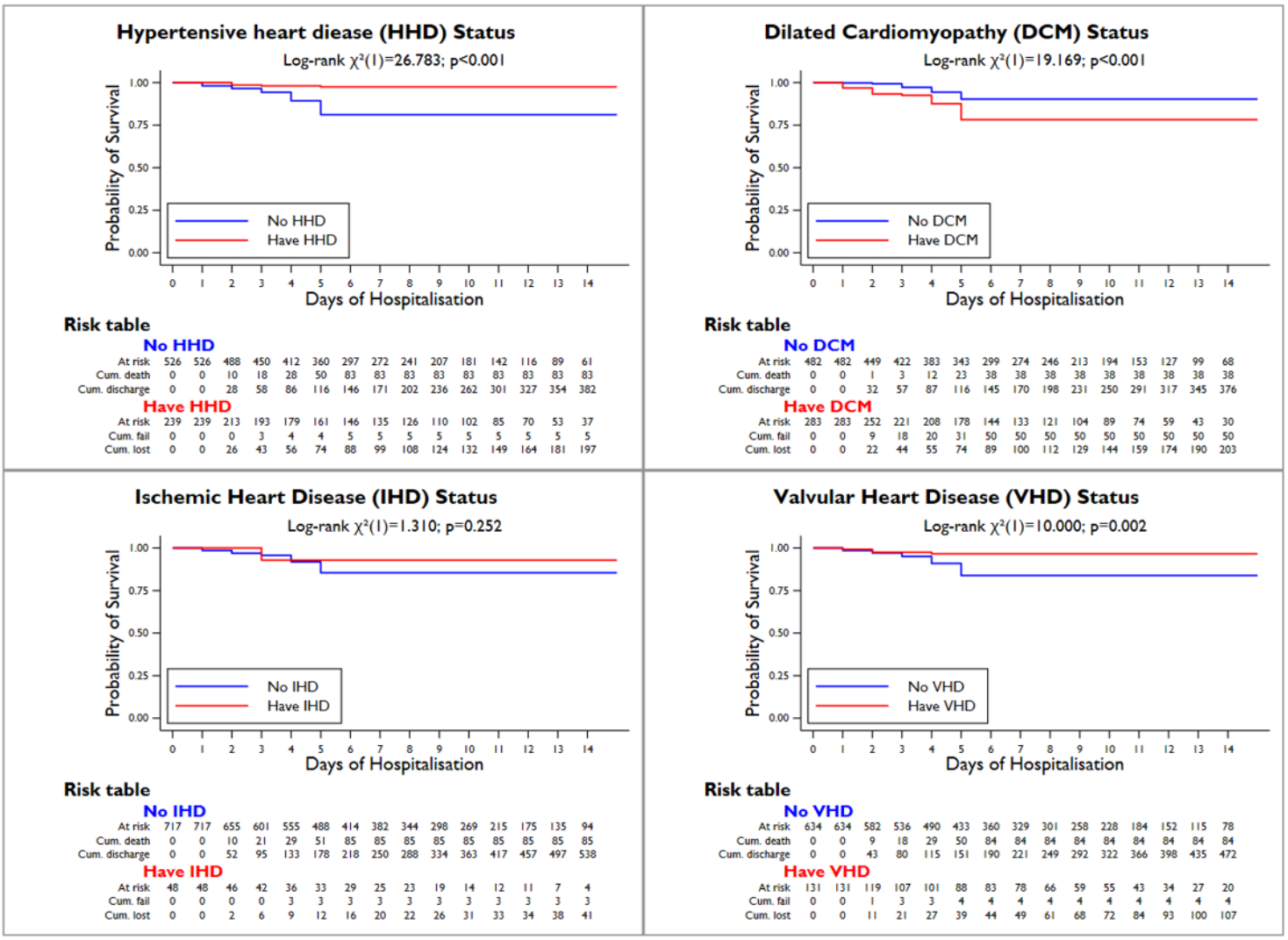


Figure 3

Kaplan Meier Survival rate curve among all study participants

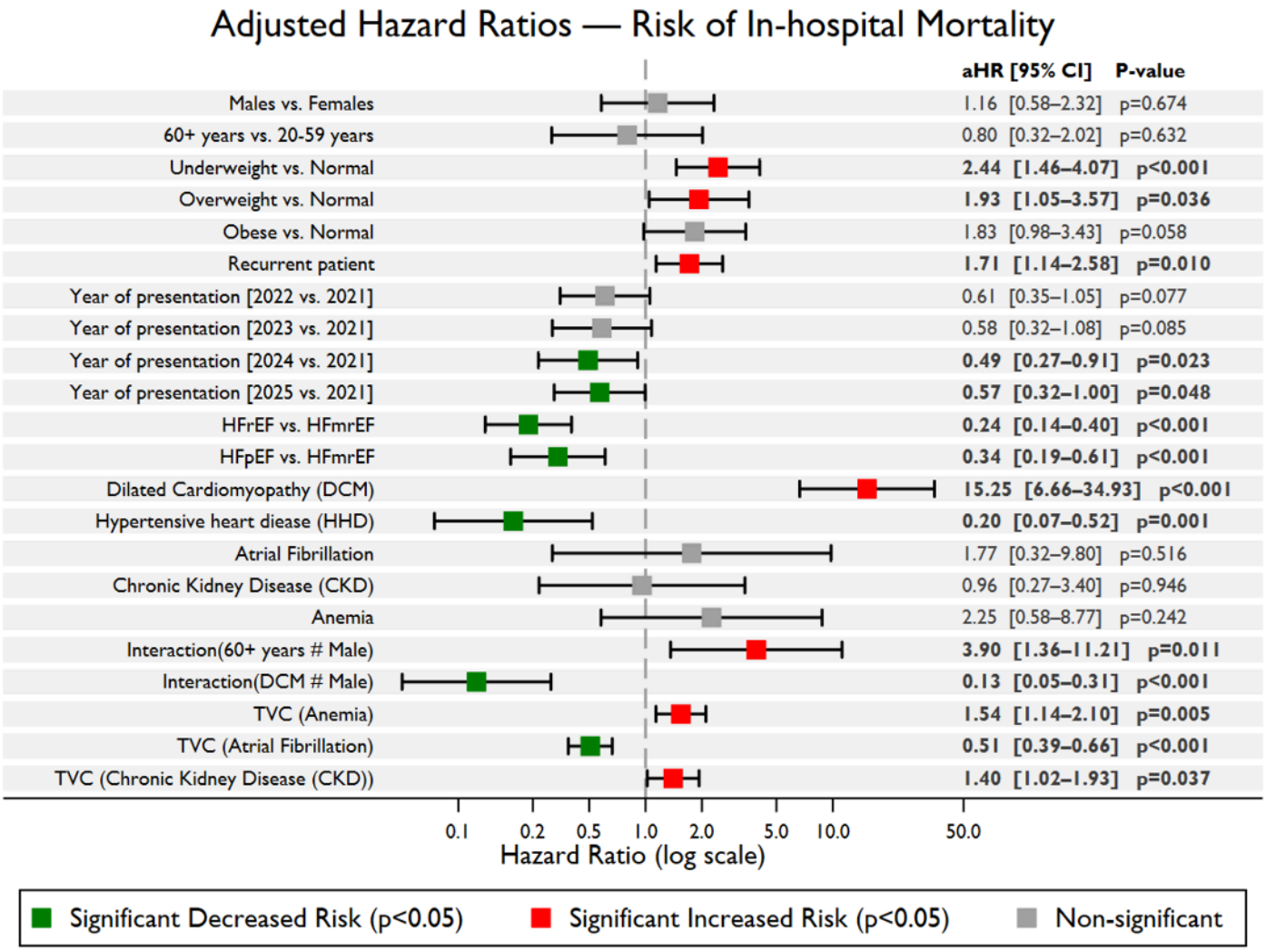


Figure 4

Multivariable Cox proportional hazards regression model of risk factors for in-hospital

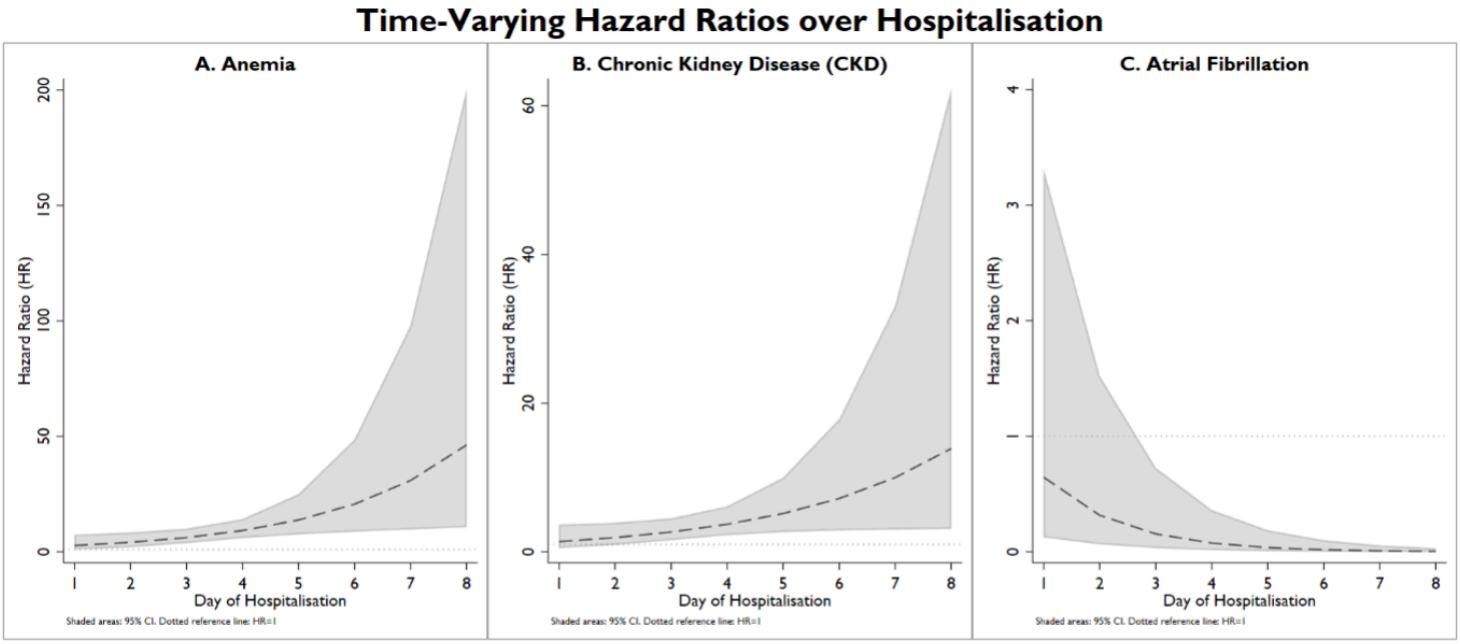


Figure 5

Risk curve of time-varying covariates in the multivariable Cox proportional hazards regression model during the first 8 days

Supplementary Files

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- BMCSupplementaryTables.docx