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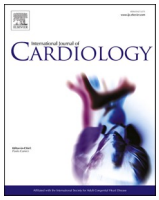

Long-term outcomes of hospitalised patients with de novo and acute decompensated heart failure

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ABSTRACT

Aims: Hospital admission for heart failure (HF) is associated with increased mortality risk. Patients admitted with HF can be divided into those with a known previous diagnosis of HF and de novo cases. However, few studies have compared these groups. We compared long-term outcomes of patients with de novo versus acute decompensated HF (ADHF).

Methods and results: We included data from two London hospitals, King's College Hospital and Princess Royal University Hospital. Data from all admissions were collected from the National Institute for Cardiovascular Outcomes and Research (NICOR) National Heart Failure Audit (NHFA) between 2020 and 2021. The outcome measure was all-cause mortality.

A total of 561 patients were included in the study. One third (29 %) were de novo hospitalisations. Over a median follow-up of 15 (interquartile range 4–21) months, 257 (46 %) patients died. Hospitalisation for ADHF was associated with higher all-cause mortality during follow-up (51 % vs 34 %, $p < 0.001$). In adjusted models, hospitalisation for ADHF remained independently associated with higher all-cause mortality during follow-up (HR 0.60, 95 % CI 0.38–0.96; $p = 0.03$).

Conclusion: Amongst patients hospitalised for HF, having a history of HF is associated with a higher risk of all-cause mortality than de novo cases. This may have implications for randomised studies that do not routinely document patients' HF history. Prospective studies are needed to elucidate the risk profiles of these two distinct populations for better risk stratification.

1. Introduction

Hospitalisation for heart failure (HF) is associated with increased mortality risk [1,2]. Acute HF may present either as a new diagnosis following admission to hospital (termed 'de novo') or after decompensation of previously diagnosed HF ('acute decompensated HF', ADHF). Approximately a quarter of de novo patients experience a readmission for ADHF within a year post-discharge [3]. While some studies suggest that following such repeat admissions, patients are at increased mortality risk with every subsequent hospitalisation [4], there remains a need to elucidate whether the risk for adverse events differs when hospitalised with de novo or decompensated HF.

Often in the literature, de novo and ADHF are not investigated as distinct groups despite potential dissimilarities amongst them and the influence this would have on outcomes. Although previous studies have supported an association between ADHF and worse outcomes [5–10], long-term outcomes have not been robustly investigated using clear classifications of each group. Hence, this status is often unaccounted for or ill-defined in clinical studies, which might skew results and be a source of confounding.

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We hypothesise that admissions for acute decompensation for previously known HF has a worse prognosis than those for de novo HF. To test this hypothesis, our study aims to compare long-term outcomes of patients with de novo versus ADHF.

2. Methods

2.1. Study design, population, and definitions

This was a retrospective cohort study of consecutive, unselected patients hospitalised with HF, across the spectrum of left ventricular ejection fractions (LVEF), between February 2020 and March 2021. We included patients older than 18 years at the time of admission who were admitted with a primary diagnosis of HF in two hospitals from the same Trust, King's College Hospital and Princess Royal University Hospital, London, United Kingdom (UK). Data was collected from the National Institute for Cardiovascular Outcomes and Research (NICOR) National Heart Failure Audit (NHFA) for England and Wales. This collects data on acute HF hospitalisations from NHS Trusts in England and Health Boards in Wales. The NHFA reports data on HF hospitalisations from NHS Trusts in England and Health Boards in Wales. This has been reported as a case ascertainment of more than 80 % for the 2020/21 audit cycle [11]. Data entry is mandatory for all NHS trusts admitting patients with acute HF. Patients were entered into the audit if they had a discharge diagnosis of HF in the primary diagnostic position.

If a single patient had multiple admissions between February 2020 and March 2021 in the NHFA, we analysed only the first admission in this timeframe. The NHFA classified patients as having a new or known diagnosis of HF, which was determined by the admitting physicians based on clinical history. We mitigated any potential inaccuracies in their classification by interrogating audit data from 2003 to 2020 for previous admissions in the contributing hospitals. In our analysis, patients hospitalised for HF who did not have a clinical history prior of HF or no identifiable prior HF admission were considered de novo patients and were compared to those with an established HF diagnosis (ADHF). We characterised specialist input as inpatient management being guided by a HF specialist or cardiologist.

2.2. Baseline characteristics

Parameters routinely collected by the NHFA include age, sex, ethnicity, comorbidities, risk factors associated with cardiovascular disease, and blood pressure. These were used for our analysis alongside other variables listed in the standard dataset obtainable from NICOR (https://www.nicor.org.uk/national-cardiac-audit-programme/datasets/).

2.3. Outcome measures

We investigated all-cause mortality during follow-up as our primary outcome of interest, collected from the NHS Digital platform. Our secondary outcome of interest was in-hospital mortality, obtainable from the NHFA.

2.4. Sensitivity analysis

To investigate the possible confounding effect of elapsed time from diagnosis, we performed a sensitivity analysis using the latest admission, rather than first, of each patient included in the February 2020 and March 2021 NHFA.

2.5. Statistical analysis

Baseline characteristics were presented as either median (interquartile range [IQR]), mean ± standard deviation (SD), or count (percentage). All continuous variables were non-parametric and so comparisons between the de novo and readmission groups were made using the Mann-Whitney *U* test. Chi-squared and Fisher's exact tests were used for categorical variables with counts above and below five, respectively. Survival analysis was used to evaluate all-cause mortality. Patients were grouped by whether their admission was do novo HR or ADHF and compared using the log-rank test.

We used univariable and multivariable Cox proportional hazard models to assess the association between baseline variables and mortality. Initially, we evaluated the association between de novo and ADHF status with all-cause mortality. We then adjusted this in a multivariable model with baseline variables if their association with all-cause mortality met the significance threshold for inclusion ($p < 0.1$). The threshold for statistical significance was set as $p < 0.05$ for all analyses, which was performed using the Statistical Package for Social Sciences (SPSS), for Windows, Version 28, (IBM Corp, Armonk, New York, USA), in tandem with the R software (version 4.2.3, R Foundation for Statistical Computing, Vienna, Austria. URL https://www.r-project.org/).

Table S1 gives the 'Strengthening the Reporting of Observational Studies in Epidemiology' (STROBE) and 'REporting of studies Conducted using Observational Routinely-collected Data' (RECORD) checklists [12,13].

3. Results

3.1. Study population

561 patients with HF were included in our study. Of these, 400 (71 %) had ADHF and 161 (29 %) had a de novo admission. Baseline characteristics are shown in Table 1. The two groups shared similar baseline characteristics with regards to sex and ethnicity, although patients with de novo HF were younger compared to those with known HF (76 vs 80 years, $p < 0.01$). Additionally, fewer in the de novo group had ischaemic heart disease (29 % vs 43 %, $p < 0.01$), respiratory disease (22 % vs 31 %, $p = 0.03$), or atrial arrhythmias (45 % vs 58 %, $p = 0.01$). Most patients received specialist input from cardiology services, with the same representation in the two groups.

In those with de novo HF, fewer patients had HF with preserved ejection fraction (HFpEF) than in the ADHF group (26 % vs 39 %, respectively, $p < 0.01$). There was greater representation of HF with reduced ejection fraction (HFrEF) in the de novo group compared to the ADHF group (60.9 % vs 44.1 %, $p < 0.01$).

More patients with de novo HF had marked limitation of their physical activity (76 % vs 67 %, $p = 0.04$), placing them in either the third or fourth functional classes of the New York Heart Association (NYHA) system. In the de novo group, more patients were discharged on beta blocker therapy (84 % vs 73 %, $p < 0.001$), and either angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARB) or angiotensin receptor/neprilysin inhibitors (ARNI) (72 % vs 59 %, $p < 0.001$). This was also the case for triple therapy (combination of beta blocker therapy, mineralocorticoid receptor antagonist and ACE inhibitor/ARB/ARNI), which was more common on discharge in the de novo group (45 % vs 31 %, $p < 0.01$).

3.2. Outcomes

During a median follow-up of 15 months (IQR 4–21), 257 (46 %) patients died, 55 (34 %) in the ADHF group and 202 (51 %) in the de novo group. In univariable Cox regression analysis, hospitalisation for de novo HF was associated with a lower risk of death during follow-up compared to ADHF (HR 0.59, 95 % CI 0.44–0.80, <0.001). This association remained after adjustment for baseline variables that were associated with mortality on univariable analyses, including comorbidities, and medication (HR 0.60, 95 % CI 0.38–0.96, $p = 0.03$) (Table 2). Similarly, these findings are corroborated by Kaplan-Meier analysis, demonstrating better survival probability with de novo HF status versus ADHF ($p = 0.00046$) (Fig. 1).

During hospitalisation, 51 (9.1 %) patients died, 42 (11 %) in the

Table 1
Demographic characteristics of study group.

		De novo	ADHF	*P*-value
Number of patients, *n* (%)		161 (29)	400 (71)	–
Age at admission, years, median (IQR)		76 (63–86)	80 (71–87)	<0.01
Male, sex, *n* (%)		87 (54)	225 (56)	0.63
Ethnicity, *n* (%)	White	105 (73)	234 (66)	0.30
	Black	27 (19)	80 (23)	
	Other	12 (8.3)	41 (12)	
NYHA III/IV, *n* (%)		122 (75.8)	264 (66.8)	0.04
Peripheral oedema, *n* (%)	None	26 (16.1)	46 (11.6)	0.1
	Mild	37 (23.0)	130 (32.7)	
	Moderate	54 (33.5)	124 (31.2)	
	Severe	44 (27.3)	97 (24.4)	
Clinical presentation (on admission), median (IQR)	HR, bpm	89 (78–106)	79 (68–92)	<0.001
	BMI, kg/m^2	27.3 (23.1–32.5)	27.8 (23.3–32.4)	0.81
	Systolic blood pressure, mmHg,	130 (117–147)	129 (113–148)	0.26
Left ventricular ejection fraction, *n* (%)	Normal (>50 %)	42 (26.1)	156 (39.1)	<0.01
	Mildly reduced (40–50 %)	21 (13.0)	67 (16.8)	
	Moderately reduced (35–40 %)	19 (11.8)	41 (10.3)	
	Severely reduced (<35 %)	79 (49.1)	135 (33.8)	
Biochemistry at discharge, median (IQR)	Haemoglobin, g/L	120 (103–136)	115 (101−131)	0.04
	Serum sodium, mmol/L	138 (136–141)	138 (135–141)	0.53
	Serum potassium, mmol/L,	4.3 (4.0–4.7)	4.2 (3.9–4.6)	0.09
Length of stay, days, median (IQR)		8 (5–16)	7 (4–13)	0.12
HF nurse follow-up, *n* (%)		80 (49.7)	167 (42.5)	0.12
Atrial fibrillation or atrial flutter, *n (%)*		68 (45.0)	224 (58.2)	0.01
Comorbidities, *n* (%)	IHD	47 (29.4)	171 (43.0)	<0.01
	Valve disease	72 (45.6)	201 (50.8)	0.27
	Hypertension	99 (61.9)	271 (68.1)	0.16
	Diabetes	54 (33.8)	148 (37.3)	0.43
	Cardiomyopathy	36 (25.9)	93 (26.6)	0.87
	Congenital heart disease	3 (2.1)	1 (0.3)	0.07
	Cerebrovascular accident	18 (11.4)	64 (16.2)	0.15
	Respiratory disease	33 (22)	127 (31)	0.03
Medications (at discharge), *n* (%)	ACE-I, ARNI, or ARB	104 (72.2)	216 (59.3)	<0.001
	Beta blocker	124 (84.4)	278 (72.6)	<0.001
	Loop diuretic	127 (83.0)	345 (88.9)	0.06
	MRA	75 (54.7)	164 (46.5)	0.01
	Digoxin	27 (19.6)	75 (20.3)	0.86
	Thiazide or Metolazone	4 (3.0)	16 (4.4)	0.48
	Oral nitrates	8 (7.3)	33 (11.0)	0.27
	Hydralazine	4 (3.9)	15 (5.0)	0.79
	Warfarin	13 (8.1)	50 (12.6)	0.13
	Other oral anticoagulant	63 (39.1)	184 (46.2)	0.13
	SGLT2 inhibitors	2 (1.3)	6 (1.5)	1.00
	Optimal medical therapy	63 (45)	111 (31)	<0.01
HF specialist care, *n* (%)		150 (93)	359 (90)	0.21
All-cause hospital mortality, *n* (%)		55 (34.2)	202 (50.5)	<0.001
In-hospital mortality, *n* (%)		9 (5.6)	42 (10.5)	0.07

ACE-I, angiotensin-converting enzyme inhibitors; ADHF, acute decompensated heart failure; ARB, angiotensin receptor blockers; ARNI, angiotensin receptor-neprilysin inhibitor; BMI, body mass index; HF, heart failure; HR, heart rate; IHD, ischaemic heart disease; IQR, interquartile range; MRA, mineralocorticoid receptor antagonists; NYHA, New York Heart Association; SGLT2, sodium-glucose cotransporter-2.

ADHF group and 9 (5.6 %) in the de novo group. Type of HF admission was not associated with inpatient mortality in either unadjusted or adjusted analyses (Table 3), and statistically insignificant survival probability differences ($p = 0.071$) (Fig. 2).

3.3. Sensitivity analysis

The results were also confirmed in the sensitivity analysis using the last available admission for each patient from the NHFA. Consistent with our primary analysis, there remained a significant association with worse survival in the ADHF group ($p < 0.0001$, Fig. 3).

4. Discussion

This was a retrospective observational study comparing long-term outcomes in patients with de novo HF, using established definitions [14], and those with ADHF. This comparison provides valuable insights into the prognosis of these distinct groups and may contribute to more effective study designs.

Our results show that, amongst 561 patients, being readmitted for ADHF was associated with a higher incidence of all-cause mortality over follow-up compared to de novo HF. As expected, patients with de novo HF presented at an earlier age and were more likely to be discharged on a triple therapy of beta blockers, mineralocorticoid receptor antagonists, and either ACE inhibitors, ARB, or ARNI. In keeping with an older demographic, the decompensated HF group had more comorbidities and higher NHYA class. The frailty of this group may have limited the uptake of guideline-directed medical therapy, given that most of these medications require adequate renal function. Also, more time since diagnosis in the ADHF group may account for the development of ischaemic heart disease in addition to more opportunity for investigations such as cardiac angiograms. Overall, our results reinforce traditional prognosticators of long-term mortality, including age, male sex, peripheral oedema severity, increased systolic blood pressure, and loop diuretic therapy [15–17].

After adjusting for relevant variables, experiencing a readmission for ADHF was independently associated with poor prognosis. In addition to the accumulation of comorbidities, repeated admissions for ADHF results in congestion or hypoperfusion that injures vital organs including the heart, lungs, and kidneys [18]. It may be that the detrimental effect of these is additive with every admission. Therefore, ADHF patients would express a lower myocardial reserve in withstanding future haemodynamic insults, manifesting in adverse long-term outcomes. For patients with longstanding heart failure, the risk of an adverse event is cumulative and somehow synergistic with every repeat acutisation of HF. Often an episode of acute decompensation is the result of multiple precipitating factors leading to haemodynamic instability. This heterogeneity is rarely captured in studies and represents an independent prognostic factor. However, randomised trials often do not account for the independent association of ADHF or de novo HF status with worse all-cause mortality and instead include all patients regardless of their disease chronicity. Although the randomisation process can minimise this effect, it cannot eliminate it. Hence, future studies should consider the distinctive profiles of these two groups in their design. Additionally, our results underscore the need for better resource allocation in managing and supporting ADHF patients in their first HF presentation to prevent readmission, and resultant worsening of their risk profile.

Few studies have explored the long-term outcomes of de novo versus ADHF [5–10]. Focusing on survival after 30 days post-discharge,

Table 2
Univariable and multivariable analyses for all-cause mortality using Cox proportional hazard models.

Characteristics	Univariable HR (95 % CI)	*p*-values for univariable HR	Multivariable HR (95 % CI)	p-values for multivariable HR
Male	1.24 (0.96–1.58)	0.10	1.61 (1.08–2.38)	0.02
Race				
White	–		–	–
Black	0.72 (0.52–1.01)	0.06	1.12 (0.73–1.71)	0.60
Other	0.69 (0.44–1.09)	0.11	0.84 (0.47–1.51)	0.56
Age (per year)	1.04 (1.03–1.05)	<0.001	1.03 (1.01–1.04)	0.01
De novo	0.59 (0.44–0.80)	<0.001	0.60 (0.38–0.96)	0.03
NYHA III/IV	0.94 (0.72–1.23)	0.65	–	–
Peripheral oedema				
None	–		–	–
Mild	1.29 (0.84–2.00)	0.25	2.03 (1.07–3.86)	0.03
Moderate	0.99 (0.63–1.53)	0.95	1.54 (0.80–2.95)	0.18
Severe	1.65 (1.08–2.54)	0.02	2.92 (1.52–5.59)	0.001
HF nurse follow-up	0.44 (0.34–0.57)	<0.001	0.55 (0.37–0.83)	0.004
Atrial fibrillation or atrial flutter	1.40 (1.08–1.81)	0.01	1.28 (0.85–1.94)	0.24
Left ventricular ejection fraction				
Normal	–	–	–	–
Mild	0.82 (0.57–1.19)	0.30	–	–
Moderate	1.07 (0.71–1.59)	0.76	–	–
Severe	0.80 (0.60–1.07)	0.13	–	–
HF specialist care	1.26 (0.80–2.00)	0.31	–	–
Clinical presentation (on admission)				
Heart rate (bpm)	0.99 (0.99–1.00)	0.003	1.00 (0.99–1.01)	0.84
Systolic blood pressure (per 10 mmHg)	0.91 (0.86–0.96)	<0.001	0.86 (0.79–0.93)	<0.001
BMI (kgm-2)	0.97 (0.95–0.99)	<0.001	0.97 (0.95–1.00)	0.04
Biochemistry				
Haemoglobin	0.99 (0.99–1.00)	<0.001	0.99 (0.98–1.00)	0.01
Serum sodium	0.98 (0.95–1.01)	0.22	–	–
Serum potassium	1.14 (0.90–1.45)	0.27	–	–
Medications (at discharge)				
ACE-I, ARNI, or ARB	0.49 (0.38–0.64)	<0.001	0.87 (0.60–1.27)	0.48
Beta blocker	0.51 (0.39–0.68)	<0.001	0.76 (0.51–1.23)	0.17
Loop diuretic	0.61 (0.43–0.86)	0.01	0.46 (0.27–0.77)	0.004
MRA	0.64 (0.48–0.83)	0.001	1.06 (0.72–1.56)	0.78
Digoxin	0.91 (0.66–1.27)	0.59	–	–
Thiazide or Metolazone	1.34 (0.73–2.45)	0.35	–	–
Oral nitrates	1.02 (0.62–1.69)	0.94	–	–
Hydralazine	0.92 (0.43–1.97)	0.84	–	–
Warfarin	1.02 (0.70–1.49)	0.92	–	–
Other oral anticoagulant	0.71 (0.55–0.91)	0.007	0.48 (0.32–0.73)	<0.001
SGLT2 inhibitors	0.22 (0.03–1.58)	0.13	–	–
Comorbidities				
Ischaemic heart disease	1.08 (0.84–1.38)	0.56	–	–
Valve disease	1.50 (1.17–1.92)	0.001	1.07 (0.73–1.57)	0.73
Hypertension	0.94 (0.72–1.21)	0.61	–	–
Diabetes	1.00 (0.77–1.29)	0.98	–	–
Congenital heart disease	1.28 (0.32–5.15)	0.73	–	–
Cerebrovascular accident	1.29 (0.93–1.79)	0.13	–	–
Cardiomyopathy	0.73 (0.53–1.01)	0.06	0.81 (0.53–1.25)	0.34
Respiratory disease	1.00 (0.76–1.32)	0.98	–	–

ACE-I, angiotensin-converting enzyme inhibitors; ARB, angiotensin receptor blockers; ARNI, angiotensin receptor-neprilysin inhibitor; BMI, body mass index; CI, confidence interval; HF, heart failure; HR, hazard ratio; MRA, mineralocorticoid receptor antagonists, NYHA, New York Heart Association; SGLT2, sodium-glucose cotransporter-2.

Parenica et al. showed de novo HF status to be associated with better outcomes [5]. A meta-analysis exploring de novo onset HF versus acute decompensated congestive HF compared 15 studies on this topic. Despite high heterogeneity, mortality at three months and one year was lower for the de novo cohort and there was no significant association with in-hospital mortality, both consistent with our findings [7].

However, many of these studies only adjusted for a limited number of medications, or none, thereby excluding their potential therapeutic effect in the analyses. Indeed, guideline-directed medical therapy has shown to improve survival outcomes in the HFrEF population [19,20], who represented 64.5 % of our cohort. In this group, pharmacological therapy of sodium-glucose cotransporter-2 (SGLT2) inhibitors, ACE-I/ARNI, beta blockers, and MRA (four pillars), have been promoted as mainstay management [17]. Indeed, approximately a third of our cohort were on triple therapy at discharge reflecting the guideline directed medical therapy contemporary at the time of admission. Our results show ADHF status to be an independent prognosticator of all-cause mortality even after accounting for these post-discharge medications. Hence, previous studies may have not accounted for the full effect of guideline-directed medical therapy when comparing survival in the de novo and ADHF populations.

Furthermore, our study adds to the existing literature for several other reasons. First, several registries only include admissions from cardiology units, which does not reflect the real-world and may create a selection bias for more aggressively managed patients. In our cohort we included HF patients from wards not limited to cardiology in UK tertiary settings. Hence, this study represents a contemporary real-world cohort of patients managed across different specialities. Furthermore, previous nationwide studies demonstrated that HF specialist care is independently associated with increased implementation of medical therapy and better long-term outcomes across the LVEF spectrum [21,22]. Our results strengthen this evidence by highlighting the importance of HF specialist involvement at first presentation and after an acute decompensation. This also underscores the need for appropriate resource

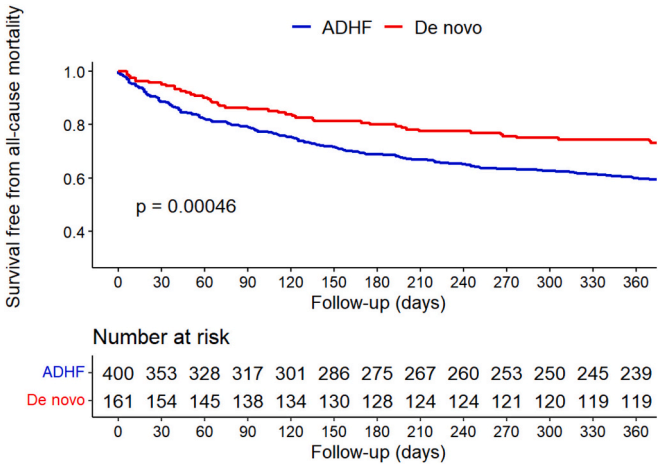

Fig. 1. Kaplan-Meier for all-cause mortality in patients with de novo versus acute decompensated heart failure.

allocation to improve the management of patients with HF throughout the stages of their disease. Second, studies that limit their cohort to patients who received intravenous therapy creates a selection bias for patients with worse illness severity [9,10]. Finally, in keeping with the South London population, this was an ethnically diverse cohort as close to a third (29 %) identified their ethnicity as part of the Black or Other categories. Therefore, we report results of a well-represented and demographically diverse cohort of patients that may have been historically misrepresented in the literature.

4.1. Limitations

We included patients from only two South London Hospitals, limiting the sample size and generalisability of our findings to other healthcare services. Additionally, the robustness of our data collection was limited to the clinical audit data available, which may have introduced selection bias. However, NICOR data provides a comprehensive list of clinical characteristics, input by clinicians who were blinded to our analysis. Despite using audit data from previous years alongside contemporaneous documentation of HF history, we may have missed prior HF diagnoses when identifying previous admissions. This study precedes the evidence and guidelines on quadruple therapy. Therefore, we reported the data on guideline directed medical therapy at the time of admission. Finally, our study analysis cannot prove a causal relationship as it is restricted to only reporting associations between the variables of interest.

5. Conclusion

ADHF is independently associated with higher all-cause mortality, demonstrated in an ethnically diverse population with substantial guideline-directed medical therapy usage. This may bias randomised cohorts, where HF history is not routinely collected. Further prospective studies are warranted to characterise the risk profile of this population and identify factors that might be important to mitigate adverse outcomes.

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CRediT authorship contribution statement

Layla Badawy: Writing – original draft, Formal analysis, Data curation, Conceptualization. **Anawinla Ta Anyu:** Data curation, Conceptualization. **Matthew Sadler:** Formal analysis. **Aamir Shamsi:** Writing – original draft, Visualization, Data curation. **Hannah Simmons:** Validation, Methodology, Data curation. **Mohammad Albarjas:** Data curation, Conceptualization. **Susan Piper:** Data curation, Conceptualization. **Paul A. Scott:** Data curation, Conceptualization. **Theresa A. McDonagh:** Writing – original draft, Supervision, Conceptualization. **Antonio Cannata:** Writing – original draft, Supervision, Formal analysis, Conceptualization. **Daniel I. Bromage:** Writing – review & editing, Writing – original draft, Supervision, Conceptualization.

Declaration of competing interest

None.

Table 3
Univariable cox proportional hazard model analyses for in-hospital mortality.

Characteristics	Univariable HR (95 % CI)	p-value
Male	1.48 (0.83–2.6)	0.18
Race		
White	–	
Black	0.5 (0.21–1.20)	0.12
Other	0.98 (0.38–2.51)	0.96
Age	1.03 (1.01–1.06)	0.01
De novo	0.52 (0.25–1.08)	0.08
NYHA III/IV	0.59 (0.33–1.05)	0.07
Peripheral oedema		
None	–	
Mild	4.37 (1.00–19.1)	0.05
Moderate	3 (0.69–13.1)	0.15
Severe	2.28 (0.52–10.0)	0.27
HF nurse follow-up	0.06 (0.01–0.23)	<0.001
Atrial fibrillation or atrial flutter	1.43 (0.80–2.56)	0.22
Left ventricular ejection fraction		
Normal	–	
Mild	1 (0.36–2.79)	1.00
Moderate	1.57 (0.60–4.09)	0.36
Severe	1.4 (0.73–2.67)	0.31
HF specialist care	0.32 (0.11–0.92)	0.03
Clinical presentation (on admission)		
Heart rate (bpm)	0.99 (0.98–1.01)	0.41
Systolic blood pressure (mmHg)	0.99 (0.97–1.00)	0.03
BMI (kgm-2)	0.96 (0.92–1.00)	0.05
Biochemistry		
Haemoglobin	1 (0.99–1.01)	0.96
Serum sodium	1.01 (0.95–1.06)	0.81
Serum potassium	1.85 (1.19–2.88)	0.01
Medications (at discharge)		
ACE-I, ARB or ARNI	0.22 (0.10–0.50)	<0.001
Beta blocker	0.17 (0.08–0.33)	<0.001
Loop diuretic	0.14 (0.07–0.29)	<0.001
MRA	0.19 (0.08–0.45)	<0.001
Digoxin	0.42 (0.17–1.04)	0.06
Thiazide or Metolazone	0.96 (0.29–3.19)	0.95
Oral nitrates	0.52 (0.12–2.27)	0.39
Hydralazine	0.86 (0.19–3.91)	0.84
Warfarin	0.53 (0.16–1.70)	0.28
Other oral anticoagulant discharge	0.09 (0.03–0.29)	<0.001
SGLT2 inhibitors	0 (0.00-Inf)	1.00
Comorbidities		
Ischaemic heart disease	1.81 (1.03–3.19)	0.04
Valve disease	1.84 (1.00–3.38)	0.05
Hypertension	0.94 (0.53–1.65)	0.82
Diabetes	0.94 (0.52–1.67)	0.82
Congenital heart disease	4.72 (0.64–34.9)	0.13
Cerebral vascular accident	1.12 (0.52–2.41)	0.77
Respiratory disease	0.83 (0.43–1.59)	0.57
Cardiomyopathy	1.06 (0.56–2.01)	0.86

ACE-I, angiotensin-converting enzyme inhibitors; ARB, angiotensin receptor blockers; ARNI, angiotensin receptor-neprilysin inhibitor; BMI, body mass index; CI, confidence interval; HF, heart failure; HR, hazard ratio; MRA, mineralocorticoid receptor antagonists, NYHA, New York Heart Association; SGLT2, sodium-glucose cotransporter-2.

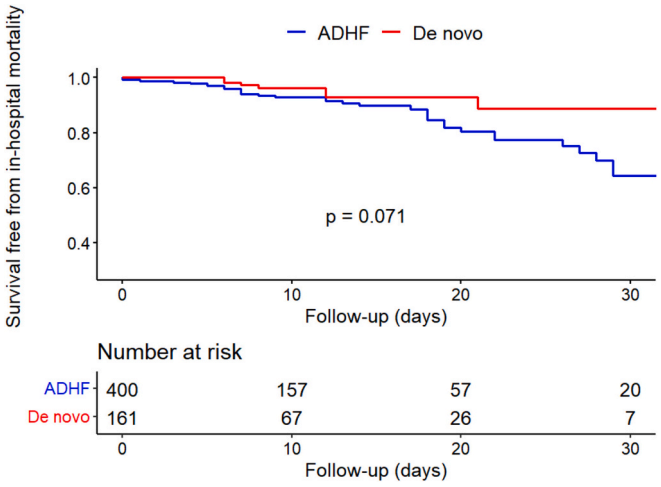

Fig. 2. Kaplan-Meier for in-hospital mortality in patients with de novo versus acute decompensated heart failure.

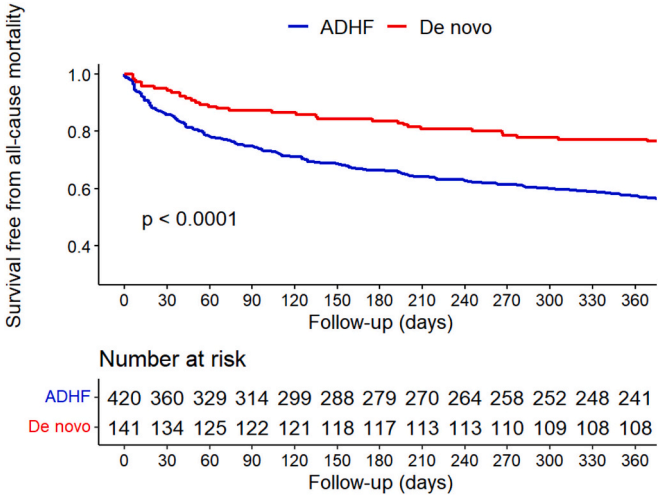

Fig. 3. Kaplan-Meier for all-cause mortality in patients with de novo versus acute decompensated heart failure, analysing only the last admissions of each patient from the NICOR audit dataset.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ijcard.2025.133061.

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