The prognostic impact of hyperglycemia on clinical outcomes of acute heart failure: Insights from the heart function assessment registry trial in Saudi Arabia

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Background: **The prognostic impact of hyperglycemia (HG) in acute heart failure (AHF) is controversial. Our aim is to examine the impact of HG on short- and long-term survival in AHF patients.**

Methods: **Data from the Heart Function Assessment Registry Trial in Saudi Arabia (HEARTS) for patients who had available random blood sugar (RBS) were analyzed. The enrollment period was from October 2009 to December 2010. Comparisons were performed according to the RBS levels on admission as either <11.1 mmol/L or ≥11.1 mmol/L. Primary outcomes were hospital adverse events and short- and long-term mortality rates.**

Results: **A total of 2511 patients were analyzed. Of those, 728 (29%) had HG. Compared to non-HG patients, hyperglycemics had higher rates of hospital, 30-day, and 1-year mortality rates (8.8% vs. 5.6%; $p = 0.003$, 10.4% vs. 7.2%; $p = 0.007$, and 21.8% vs. 18.4%; $p = 0.04$, respectively). There were no differences between the two groups in 2- or 3-year mortality rates. After adjustment for relevant confounders, HG remained an independent predictor for hospital and 30-day mortality [odds ratio (OR) = 1.6; 95% confidence interval (CI) 1.07–2.42; $p = 0.021$, and OR = 1.55; 95% CI 1.07–2.25; $p = 0.02$, respectively].**

Conclusion: **HG on admission is independently associated with hospital and short-term mortality in AHF patients. Future research should focus on examining the impact of tight glycemic control on outcomes of AHF patients.**

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1. Introduction

Acute heart failure (AHF) continues to be a burdensome problem to healthcare systems and is a leading cause of frequent hospitalizations and long-term medical care [1]. Multiple illnesses coexist with HF and influence its prognosis [2–4]. Diabetes mellitus (DM) is known as one of the most commonly associated comorbidities in HF patients with a prevalence ranging from 25% to 40% [5,6]. Data from major HF registries indicate that DM worsens hospital outcomes and increases short-term mortality rates [6–11]. Although the impact of DM on HF outcomes is known, the role of hyperglycemia (HG), whether new-onset or in the context of a preexisting DM, remains controversial [12–20]. Several reports have suggested a negative impact of HG on AHF mainly affecting hospital outcomes and overall survival [12–18], yet others have not shown similar findings [19,20].

HG in acute coronary syndromes (ACS) has been widely investigated. The Diabetes Mellitus Insulin-Glucose Infusion in Acute Myocardial Infarction (DIGAMI) trial showed a survival benefit in ACS patients with tight glycemic control [21]. This was later confirmed in other major trials [22–24]. Currently, the 2013 American Heart Association/American College of Cardiology guidelines recommend targeting sugar levels <180 mg/dL [25]. Glycemic control has become an integral part of the standard management of ACS, however the impact of extrapolating this evidence across the spectrum of all cardiovascular diseases is yet to be determined.

We sought to determine the relationship between HG and hospital adverse outcomes, as well as short- and long-term mortality rates in AHF patients using data from the Heart Function Assessment Registry Trial in Saudi Arabia (HEARTS).

Abbreviations

ACS	Acute Coronary Syndrome
AHF	Acute Heart Failure
BMI	Body Mass Index
DBP	Diastolic Blood Pressure
DLD	Dyslipidemia
DM	Diabetes Mellitus
EF	Ejection Fraction
eGFR	Estimated Glomerular Filtration Rate
HEARTS	Heart Function Assessment Registry Trial In Saudi Arabia
HG	Hyperglycemia
HR	Heart Rate
HTN	Hypertension
IHD	Ischemic Heart Disease
IQR	Interquartile Range
RBS	Random Blood Sugar
SBP	Systolic Blood Pressure
SD	Standard Deviation

2. Materials and methods

HEARTS protocol has been described previously [26,27]. Briefly, HEARTS is a prospective registry that enrolled 2609 consecutive patients with a primary admission diagnosis of AHF. Eighteen tertiary care centers in different regions of Saudi Arabia participated in this registry. Enrollment took place between October 2009 and December 2010, with clinical follow-up until January 2013. The definition of HF was according to the European Society of Cardiology guidelines for the diagnosis and treatment of acute and chronic HF [28]. The study was approved by the institutional review board at each participating hospital and complied with the Declaration of Helsinki.

Patients were eligible for this analysis if baseline random blood sugar (RBS) values were available. The diagnosis of DM was based on medical records documentation, patient self-reporting, or if the patient was taking diabetic medications. Patients were labeled as having HG if their RBS was $\geq$11.1 mmol/L, according to the American Diabetes Association guidelines [29]. We described patients' baseline characteristics, therapies, hospital course, and hospital mortality rates. Additionally, we obtained the vital status after 30 days, 1 year, 2 years, and 3 years following hospital discharge by a telephone interview and verified these data as needed using hospital records.

2.1. Statistical analysis

Categorical data were summarized with absolute numbers and percentages. Numeric data were summarized with mean and standard deviation (SD) or median and interquartile range (IQR). Comparisons between different groups were performed using Chi-square test or Fisher's exact for categorical variables and independent sample *t* test or Mann–Whitney *U* test for continuous variables. Kaplan–Meier analysis was applied to plot the cumulative survival and differences between curves were assessed using the log-rank test. We used logistic regression models to estimate unadjusted and adjusted odds ratios (OR) for mortality rates. We adjusted for age, sex, estimated glomerular filtration rate (eGFR), ACS, body mass index (BMI), systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate (HR), AHF type, ejection fraction (EF), dyslipidemia (DLD), anemia, hypertension (HTN), and DM. Logistic regression with interaction terms was used to test the statistical significance of the interaction between HG and other baseline factors. To estimate the strength of association in subgroups we used OR with 95% confidence intervals (CI). A two-sided p value <0.05 was considered statistically significant. All analyses were performed using SAS/STAT software, version 9.2 (SAS Institute Inc., Cary, NC, USA.) and R software (R Foundation for Statistical Computing, Vienna, Austria).

3. Results

Of the 2609 patients enrolled in HEARTS, 2511 (96.2%) patients were eligible for the present analysis. A total of 1783 (71%) were in the non-HG group, while 728 (29%) had HG at baseline. HG patients were generally older and had higher BMIs. Further, they were more likely to be diabetic, hypertensive, and dyslipidemic ($p < 0.001$ for all comparisons; Table 1).

Compared with patients with HG, non-HG patients were more likely to have a history of HF, valvular heart diseases (rheumatic and

Table 1. Baseline characteristics.

	Overall 2511	Non-HG 1783 (71%)	HG 728 (29%)	*p*
Demographics				
Age	61.34 ± 15	60.65 ± 15.6	63.38 ± 12.4	<0.001
Saudi	2135 (85)	1521 (85.3)	614 (84.3)	0.539
Male	1653 (65.8)	1207 (67.7)	446 (61.3)	0.002
Body mass index	29.2 ± 6.7	29 ± 6.7	29.7 ± 6.7	0.028
Risk factors				
Diabetes mellitus	1629 (65.1)	955 (53.8)	674 (92.7)	<0.001
Smoker/ex-smoker	844 (33.6)	614 (34.4)	230 (31.6)	0.171
Hypertension	1781 (71.4)	1195 (67.4)	586 (81.3)	<0.001
Dyslipidemia	870 (36.8)	550 (32.5)	320 (47.8)	<0.001
History of cardiovascular diseases				
Heart failure	1607 (64.2)	1179 (66.3)	428 (59)	<0.001
Ischemic heart disease	1342 (54)	908 (51.6)	434 (59.9)	<0.001
TIA/stroke	241 (9.6)	156 (8.8)	85 (11.7)	0.025
PAD	97 (3.9)	60 (3.4)	37 (5.1)	0.044
PCI	326 (13)	222 (12.5)	104 (14.3)	0.219
CABG	257 (10.3)	175 (9.8)	82 (11.3)	0.282
RHD	172 (6.9)	137 (7.7)	35 (4.8)	0.010
Other VHD	359 (14.4)	271 (15.3)	88 (12.2)	0.045
Atrial fibrillation	390 (15.6)	313 (17.6)	77 (10.6)	<0.001
VT/VF	60 (2.4)	50 (2.8)	10 (1.4)	0.033
ICD	216 (8.6)	179 (10.1)	37 (5.1)	<0.001
CRT	81 (3.2)	67 (3.8)	14 (1.9)	0.018
History of other chronic medical illnesses				
Anemia	1116 (44.6)	781 (44)	335 (46.2)	0.308
CKD on dialysis	70 (9.5)	45 (8.7)	25 (11.4)	0.256
CKD not on dialysis	668 (90.5)	473 (91.3)	195 (88.6)	
Chronic lung disease	179 (7.1)	131 (7.4)	48 (6.6)	0.505

Data are presented as *n* (%) or mean ± SD.
CABG = coronary artery bypass grafting; CKD = chronic kidney disease; CRT = cardiac resynchronization therapy; HG = hyperglycemia; ICD = implantable cardioverter defibrillator; PAD = peripheral arterial disease; PCI = percutaneous coronary intervention; RHD = rheumatic heart disease; SD = standard deviation; TIA = transient ischemic attack; VF = ventricular fibrillation; VHD = valvular heart disease; VT = ventricular tachycardia.

nonrheumatic), arrhythmias (both atrial and ventricular), and to have undergone cardiac device implantation. However, vascular comorbidities such as ischemic heart disease (IHD), strokes/-transient ischemic attacks, and peripheral arterial disease were significantly higher among patients with HG (Table 1).

Table 2 demonstrates the types, etiologies, and exacerbating factors of AHF. HG patients were more likely to present with acute *de novo* HF while non-HG patients were more likely to present with acute on chronic HF ($p < 0.001$ for group comparison). IHD was the prime etiology for AHF in patients with HG, while nonischemic etiologies of AHF were seen more often in non-HG patients ($p < 0.001$ for group comparison). ACS and uncontrolled HTN were the main reasons for AHF exacerbation among HG patients, and had occurred more frequently compared to the non-HG group.

Patients with HG had a higher mean baseline SBP (134.5 vs. 126.6, $p < 0.001$), higher rates of positive troponin levels (51.3% vs. 32.6%, $p < 0.001$), and a higher proportion of low eGFR defined as <60 mL/min/1.73 m^2 (60.2% vs. 51.0%, $p < 0.001$). Non-HG patients were more likely to have severe left ventricular systolic dysfunction (50.6% vs. 39.7%; $p < 0.001$). Among the patients who underwent coronary angiogram during the same admission ($n = 720$), significant left main, three-vessel, and double-vessel disease were more frequently seen in patients with HG. Further comparisons in clinical presentations and baseline investigations are depicted in Table 3.

Hospital therapies and discharge medications are shown in Fig. 1. β-blockers and aldosterone antagonists use was higher in non-HG patients, both prior to hospital admission, and upon discharge, while aspirin and statin therapy were prescribed more frequently in HG patients upon discharge.

Hospital procedures, complications, as well as hospital, short-, and long-term mortality rates are shown in Table 4. Compared to HG patients, the non-HG group were more likely to receive device therapies (implantable cardioverter defibrillators and cardiac resynchronization therapy) and were less likely to require mechanical ventilation. Apart from a higher rate of hospital recurrence of AHF in patients with non-HG (33.1% vs. 28.2%; $p = 0.015$), there were no differences in the rate of hospital complications between the two groups.

The observed hospital, 30-day, and 1-year mortality rates were significantly higher in patients with HG (8.8% vs. 5.5%; $p = 0.003$, 10.4% vs. 7.2%; $p = 0.007$, and 21.8 vs. 18.4; $p = 0.049$, respectively). There were no differences in the 2- and 3-year mortality rates between the two groups. After adjusting for important confounders, HG remained an independent predictor for hospital and 30-day mortality (OR = 1.61; 95% CI 1.07–2.42, $p = 0.022$, and OR = 1.55; 95% CI 1.07–2.25, $p = 0.021$, respectively), Table 5. A Kaplan–Meier plot comparing survival rates between the groups showed that patients with HG had significantly lower survival rates compared with patients with non-HG (log-rank test $p = 0.038$), Fig. 2.

Table 2. Heart failure types, etiologies, and exacerbating factors for acute heart failure.

	Overall 2511	Non-HG 1783 (71%)	HG 728 (29%)	*p*
Acute heart failure type				
Acute *de novo* HF	904 (36)	604 (33.9)	300 (41.2)	<0.001
Acute on Chronic HF	1607 (64)	1179 (66.1)	428 (58.8)	
Etiology				
Ischemic	1419 (56.5)	937 (52.5)	482 (66.2)	<0.001
Nonischemic	1092 (43.5)	846 (47.4)	246 (33.8)	
HF exacerbation factors				
NSTACS	702 (28)	440 (24.7)	262 (36)	<0.001
STEMI	266 (10.6)	164 (9.2)	102 (14)	<0.001
Uncontrolled hypertension	506 (20.1)	332 (18.6)	174 (23.9)	0.003
Noncompliance to HF medications	523 (20.8)	403 (22.6)	120 (16.5)	<0.001
Noncompliance to diet	628 (25)	493 (27.6)	135 (18.5)	<0.001
Worsening renal failure	443 (17.6)	341 (19.1)	102 (14)	0.002
Arrhythmia	275 (10.9)	210 (11.8)	65 (8.9)	0.038
Infections	524 (20.9)	363 (20.4)	161 (22.1)	0.326
COPD exacerbation	94 (3.7)	74 (4.1)	20 (2.7)	0.093

Data are presented as *n* (%).
COPD = chronic obstructive pulmonary disease; HF = heart failure; HG = hyperglycemia, NSTACS = non-ST elevation myocardial infarction; STEMI = ST-elevation myocardial infarction.

Table 3. Clinical presentation and investigations.

	Overall 2511	Non-HG 1783 (71%)	HG 728 (29%)	*p*
Hemodynamic parameters				
Systolic blood pressure	128.7 ± 31.3	126.6 ± 30.8	134.5 ± 32.5	<0.001
Diastolic blood pressure	74.1 ± 17.9	73.8 ± 18.2	75.5 ± 17.6	0.030
Heart rate	88.8 ± 21	87.9 ± 20.9	91.4 ± 21.3	<0.001
Lab results				
RBS (mmol/L), median (IQR)	8 (6)	7 (3)	15 (6)	<0.001
Sodium (mmol/L)	135.1 ± 5.3	135.5 ± 5.2	134.2 ± 5.6	<0.001
BUN (μmol/L)	11.9 ± 9.1	11.7 ± 9.2	12.2 ± 8.8	0.288
Hemoglobin (g/dL)	12.4 ± 2.2	12.5 ± 2.21	12.4 ± 2.3	0.521
Creatinine (μmol/L)	109 (70)	108 (69)	118 (58)	0.197
Pro BNP (pmol/L), median (IQR)	675 (668)	705 (650)	664 (1095)	0.632
Troponin positive	848 (38)	516 (32.6)	332 (51.3)	<0.001
eGFR < 60	1346 (53.7)	909 (51)	437 (60.2)	<0.001
Electrocardiography				
Wide QRS duration	361 (14.4)	272 (15.3)	89 (12.3)	0.049
Left bundle branch block	288 (11.5)	197 (11.1)	91 (12.5)	0.300
LV systolic function by echocardiography				
Normal (EF > 55%)	332 (13.8)	224 (13.1)	108 (15.6)	<0.001
Mild (EF = 40–55%)	321 (13.3)	202 (11.8)	119 (17.2)	
Moderate (EF = 30–39.9%)	611 (25.4)	420 (24.5)	191 (27.6)	
Severe (EF < 30%)	1141 (47.4)	866 (50.6)	275 (39.7)	
Coronary angiography (n = 720)				
LMD/TVD	255 (34.7)	166 (31.2)	89 (44.1%)	0.001
Significant double VD	113 (15.4)	70 (13.2)	43 (21.3)	0.006
Significant single VD	97 (13.2)	74 (13.9)	23 (11.4)	0.367
Nonsignificant CAD	79 (10.8)	60 (11.3)	19 (9.4)	0.465
Normal	176 (24)	153 (28.8)	23 (11.4)	<0.001

Data are presented as *n* (%) or mean ± SD, unless otherwise indicated.
BNP = brain natriuretic peptide; BUN = blood urea nitrogen; CAD = coronary artery disease; EF = ejection fraction; eGFR = estimated glomerular filtration rate; HG = hyperglycemia; IQR = interquartile range; LMD = left main disease; RBS = random blood sugar; SD = standard deviation; TVD = three vessel disease; VD = vessel disease.

The interaction between HG and mortality was assessed in several patient subgroups. Subgroups assessed included patients stratified by age (≥70 years vs. <70 years), sex (males vs. females), prior diagnosis of DM, use of insulin, HF etiology (ischemic vs. nonischemic), type of AHF (*de novo* vs. acute on chronic), eGFR (≥60 mL/min/1.73 m^2 vs. <60 mL/min/1.73 m^2), EF (≥40% vs. <40%), SBP (≥90 mmHg vs. <90 mmHg), and history of anemia. A significant interaction between HG and EF was observed, where the negative impact of HG on 30-day mortality was worse in patients with an EF < 40% (EF < 40%, OR = 1.69; 95% CI 1.18–2.42, $p = 0.003$, vs. EF ≥ 40%, OR = 0.72; 95% CI 0.37–1.39, $p = 0.331$, p value for interaction = 0.025). This interaction between HG and EF was not seen in hospital or 1-year mortality. Additionally, a strong interaction was observed between HG and anemia. Anemic patients with HG had a higher hospital mortality compared with nonanemic patients (anemia present, OR = 2.69; 95% CI 1.62–4.46, $p < 0.001$ vs. anemia absent, OR = 1.10; 95% CI 0.71–1.73, $p = 0.66$, p for interaction = 0.01). This interaction between anemia and HG also impacted short- and long-term mortality (data not shown).

4. Discussion

To our knowledge, this is the first report from the Arab Middle East examining the impact of glycemic status on the outcomes of patients with AHF. We found that almost 30% of our patients had HG upon hospital admission. Irrespective of their diabetic status and other comorbidities, these patients had a worse prognosis.

Data on the impact of HG on AHF outcomes are inconsistent [12–20]. Some reports have suggested that HG is independently associated with hospital [12–16], 30- [17,18], and 60-day [14] mortality. However, this association with mortality was less robust in the long term [12,13,19,20]. Conversely, other reports did not show an association between HG and short-term mortality [19,20] but rather an

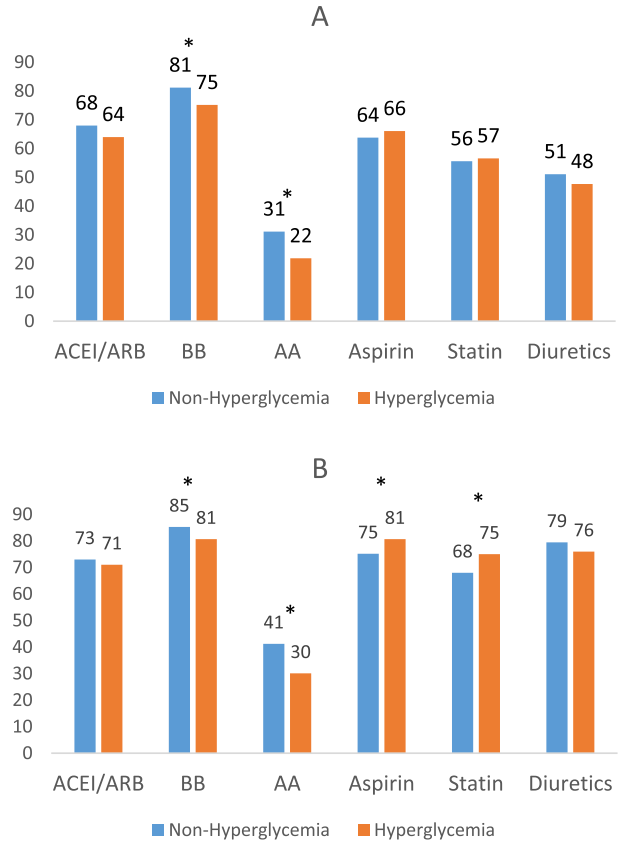

*Figure 1. Differences in evidence-based medical therapies used before admission (A) and at discharge (B). *$p < 0.05$. AA = aldosterone antagonists; ACEI = angiotensin converting enzyme inhibitors; ARB = angiotensin receptors blockers; BB = β-blockers.*

association with long-term mortality [18]. Our data agree with the general pattern of these reports where HG is more likely to be related to short-term mortality. The discrepancy in the findings of these studies could be explained by the diverse methods and inclusion and exclusion criteria that were used, such as the exclusion of diabetic patients [14,16,19], using different blood sugar measurements (random and/or fasting) and cut-offs, or selecting patients under special circumstances such as AHF patients admitted to the intensive care only [15,19].

Our subgroup analysis suggests an interaction between HG and an anemic status as well as with EF. The test of interaction is hypothesis generating and may suggest colinearity between HG and anemia on one hand, and HG and an EF < 40% on the other hand. Alternatively, anemia and a low EF such as HG simply reflect disease severity. Therefore, a risk score for AHF that combine all potential risk factors for worse prognosis is essential for targeted therapy and hospital disposition. In addition, the high readmission and mortality rates in AHF patients further necessitate conducting trials focusing on risk score designing and validation [30]. Indeed, there have been many proposed risk scores that correlate with hospital and postdischarge mortality [31–33]. However, none of them is implemented as a standard-of-care in current clinical practice.

Whether HG in AHF serves as a marker of disease severity or a direct cause for adverse outcomes remains unclear. Some have suggested that chronic elevation of blood sugar as evident by an elevated HbA1c could cause direct injury to the myocardium [34]. In addition, persistent hyperglycemia (e.g., Type I DM) may lead to an insulin-resistant state [35] and impaired glucose uptake by the myocardium shifting the energy generation pathway towards utilization and oxidization of free fatty acids by the myocardium [36,37] which in turn may promote arrhythmogenesis [38]. Finally, HG may impair the cardiac function through various mechanisms such as oxidative stress [39,40], endothelial atherogenesis, and vascular inflammation [41]. However, HG in AHF can simply be stress-induced. The normal physiological response to stress insults leads to high glucose levels as a result of sympathetic nervous system activation and/or excessive release of stress hormones such as cortisol [42,43]. The fact that HG seemed to be an independent predictor of short- rather than long-term mortality might support the premise that HG is merely a marker of severity rather than a direct cause of mortality.

The clinical implications of our findings are numerous. Firstly, the measurement of RBS in the Emergency Department is simple and provides very useful information in predicting the hospital course and prognosis of AHF. Therefore, it can potentially be used as a tool amongst other tools for risk stratification in AHF patients. Secondly, HG in the context of AHF was found to be predictive of the development of new-onset DM [18]. Similar findings were observed in critically ill patients [44], and patients with ACS [45]. This should encourage treating physicians to screen patients with abnormal glucose levels for DM following the acute phase of HF. Finally, as HG is an independent predictor of short-term adverse outcomes in the context of AHF, this should raise interest in studies examining the efficacy of aggressive glycemic control on the outcomes of AHF patients. Despite the general recommendation by the American Diabetes Association to aim for strict glycemic control in any hospital admission regardless of the primary diagnosis [46], the evidence for this practice in AHF is weak.

Table 4. Adverse hospital outcomes, 30-day, 1-, 2-, and 3-year mortality rates.

	Overall 2511	Non-HG 1783 (71%)	HG 728 (29%)	*p*
Hospital procedures				
Dialysis	119 (4.7)	79 (4.4)	40 (5.5)	0.255
Ventilation	282 (11.2)	186 (10.4)	96 (13.2)	0.047
Intraaortic balloon pumps	84 (3.35)	54 (3)	30 (4.1)	0.167
Pacing	35 (1.4)	20 (1.1)	15 (2.1)	0.069
Hospital ICD	145 (5.8)	127 (7.1)	18 (2.5)	<0.001
Hospital CRT	65 (2.6)	56 (3.1)	9 (1.2)	0.006
Hospital complications				
Recurrent CHF	796 (31.7)	591 (33.1)	205 (28.2)	0.015
Sepsis	186 (7.4)	124 (6.9)	62 (8.5)	0.175
Shock	218 (8.7)	147 (8.2)	71 (9.7)	0.223
Cardiogenic	162 (74.3)	113 (76.9)	49 (69)	0.271
Noncardiogenic	21 (9.6)	11 (7.5)	10 (14.1)	
Both	35 (16.1)	23 (15.6)	12 (16.9)	
AF requiring therapy	150 (6)	113 (6.3)	37 (5.1)	0.229
VT/VF	106 (4.2)	67 (3.8)	39 (5.4)	0.071
Major bleeding	35 (1.4)	26 (1.5)	9 (1.2)	0.667
TIA/stroke	45 (1.8)	29 (1.6)	16 (2.2)	0.328
All-cause mortality				
In-hospital mortality	163 (6.5)	99 (5.5)	64 (8.8)	0.003
30-d mortality	204 (8.1)	128 (7.2)	76 (10.4)	0.007
1 y mortality	487 (19.4)	328 (18.4)	159 (21.8)	0.049
2 y mortality	589 (23.5)	406 (22.8)	183 (25.1)	0.209
3 y mortality	607 (24.2)	416 (23.4)	191 (26.2)	0.127

Data are presented as *n* (%).
AF = atrial fibrillation; CHF = congestive heart failure; CRT = cardiac resynchronization therapy; HG = hyperglycemia; ICD = implantable cardioverter defibrillator; TIA = transient ischemic attack; VF = ventricular fibrillation. VT = ventricular tachycardia.

Table 5. Crude and adjusted odds ratios and 95% confidence intervals for short- and long-term mortality in acute heart failure.

All-cause mortality	Crude OR (95% CI)	*p*	Adjusted OR (95% CI)	*p*
Hospital mortality	1.64 (1.18–2.27)	0.003	1.61 (1.07–2.42)	0.022
30-d mortality	1.50 (1.11–2.02)	0.008	1.55 (1.07–2.25)	0.021
1 y mortality	1.24 (1.00–1.53)	0.049	1.25 (0.96–1.63)	0.100
2 y mortality	1.14 (0.93–1.39)	0.209	1.13 (0.88–1.45)	0.354
3 y mortality	1.17 (0.96–1.42)	0.127	1.17 (0.91–1.50)	0.209

CI = confidence interval; OR = odds ratio.

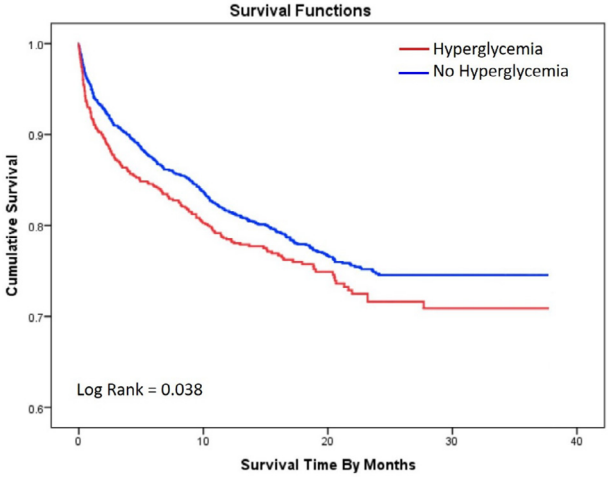

Figure 2. Survival analysis using Kaplan–Meier plots in hyperglycemics versus nonhyperglycemics.

This study suffered from several limitations. Data on hospital readmission rates were not collected in the HEARTS registry, therefore, the impact of HG on AHF readmissions rates and postdischarge disease deterioration could not be assessed. In addition, the registry only recorded all-cause mortality, and thus we are unable to comment on the rates of cardiovascular mortality. Finally, HbA1c data were not collected systematically. Hence, we could not determine if HG is a new event or simply a reflection of an undiagnosed DM.

5. Conclusion

Our study highlights the deleterious short-term prognostic impact of HG in AHF patients. Our

findings should prompt the design of clinical trials addressing the impact of tight glycemic control in AHF on clinical outcomes.

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