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Management and Outcomes of Patients With Acute Heart Failure in Relation to Systolic Blood Pressure at Presentation: A Single-Center Prospective Observational Study From Pakistan

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Abstract

Background

Several studies have investigated the clinical characteristics, management, and outcomes of acute heart failure (AHF) in South Asian populations. However, the prognostic relevance of admission systolic blood pressure (SBP), particularly its association with in-hospital mortality and acute supportive care requirements, remains insufficiently characterized across different healthcare settings in the region. Evidence from predominantly high-income healthcare settings suggests an inverse relationship between admission SBP and mortality. This study evaluated the association between admission SBP, clinical management, and in-hospital outcomes among patients hospitalized with AHF in Pakistan.

Methods

This single-center prospective observational study included 135 consecutive adults enrolled at emergency department (ED) presentation with AHF at a tertiary-care hospital in Lahore, Pakistan, between January 1, 2025, and January 1, 2026. Patients were categorized by admission SBP as ⩽100, 101-140, or >140 mmHg. Baseline characteristics, acute management, length of stay, and in-hospital mortality were compared using the Kruskal-Wallis test and the Fisher-Freeman-Halton exact test. Two comparisons were made: SBP ⩽100 versus >100 mmHg, and SBP >140 versus ⩽140 mmHg. Because no deaths occurred above 140 mmHg, Firth's penalized logistic regression was used; the >140 mmHg comparison was adjusted for age only, and the ⩽100 mmHg comparison was unadjusted.

Results

The mean age was 65.9 ± 13.9 years, and 68/135 (50.4%) patients were male. Thirty-one (23.0%) patients had SBP ⩽100 mmHg, 70 (51.9%) had SBP 101-140 mmHg, and 34 (25.2%) had SBP >140 mmHg. Patients with SBP >140 mmHg had a higher median left ventricular ejection fraction (LVEF) and lower median N-terminal pro-B-type natriuretic peptide (NT-proBNP) than those in the lower SBP groups. Inotrope use (58.1% versus 12.9% versus 5.9%; p<0.001) and invasive mechanical ventilation (35.5% versus 11.4% versus 5.9%; p=0.004) were most frequent at SBP ⩽100 mmHg; inotrope use reflects the clinical indication for treatment. Median length of stay was six, four, and four days, respectively (p=0.045). In-hospital mortality was 9/31 (29.0%), 10/70 (14.3%), and 0/34 (0%), respectively (p=0.001). Compared with SBP >100 mmHg, SBP ⩽100 mmHg was associated with higher odds of death (9/31 versus 10/104; unadjusted odds ratio (OR): 3.80, 95% confidence interval (CI): 1.40-10.33). No deaths occurred at SBP >140 mmHg (0/34 versus 19/101 at ⩽140 mmHg; age-adjusted OR: 0.07, 95% CI: <0.01-0.52); because there were no events in this group, the estimate is imprecise and does not show that the true risk is zero. Models were adjusted for age at most, so the mortality ORs are essentially unadjusted for LVEF, NT-proBNP, comorbidity, and illness severity, and residual confounding is likely to be large.

Conclusions

In this ED-enrolled AHF cohort, an admission SBP of 100 mmHg or lower identified patients with about three times the in-hospital mortality and a much greater need for inotropes and invasive ventilation, which may support closer monitoring and early escalation of care for these patients. The absence of deaths above 140 mmHg should not be read as absence of risk, and the associations may be substantially confounded. These findings require confirmation in larger, multicenter studies with fuller adjustment.

Categories: Cardiology, Emergency Medicine, Internal Medicine
Keywords: acute systolic heart failure, in-hospital mortality, inotropic support, pakistan, systolic blood pressure (sbp)

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Introduction

Heart failure (HF) is a major global public health challenge, affecting an estimated 64 million people worldwide [1]. Hypertension is an important modifiable risk factor for the development and progression of HF and a common precipitant of acute HF (AHF) [2,3]. Systolic blood pressure (SBP) at presentation may also provide an early indication of hemodynamic status and short-term prognosis in patients hospitalized with AHF.

Observational studies and clinical trial analyses have demonstrated an inverse association between admission SBP and adverse outcomes. In an analysis of individual patient data from four randomized clinical trials, each 10 mmHg increase in SBP was associated with a 7% lower risk of 180-day mortality (adjusted hazard ratio: 0.93 per 10 mmHg increase) [4]. These findings suggest that admission SBP may reflect differences in cardiac function, systemic perfusion, and the severity of acute decompensation.

Regional evidence also highlights the prognostic relevance of admission blood pressure in South Asian patients with HF. The prospective Trivandrum Heart Failure Registry (THFR), which enrolled 1,205 patients across 18 hospitals in Kerala, India, reported a mean admission SBP of 129.3 ± 32.4 mmHg and an overall in-hospital mortality rate of 8.5%, although its principal outcome analyses focused on HF characteristics and other established prognostic factors rather than detailed SBP-stratified acute management [5]. Similarly, the Kolkata-Coventry comparative acute HF registry demonstrated that lower admission blood pressure was associated with increased in-hospital mortality in the participating Indian public hospital cohort [6]. Evidence from Pakistan is also available: a cohort of hospitalized patients with new-onset systolic HF at a tertiary care hospital identified lower admission SBP as an independent predictor of subsequent mortality among patients surviving the index hospitalization [7]. However, these studies differ in design, patient selection, follow-up, and outcome assessment. None has reported the use of acute supportive interventions across admission SBP categories in prospectively enrolled emergency department (ED) patients, including those who died in hospital.

Differences in patient characteristics, comorbidity burden, healthcare resources, and clinical management may further influence the applicability of international prognostic findings to individual South Asian healthcare settings. In particular, understanding how admission SBP relates not only to mortality but also to the immediate clinical interventions required during hospitalization may provide additional information for early assessment and resource allocation.

This study therefore evaluated the association between admission SBP and clinical characteristics, acute management, length of hospital stay, and in-hospital mortality among patients hospitalized with AHF at a tertiary care hospital in Lahore, Pakistan. It adds three things to the regional evidence: prospective enrolment of consecutive patients at ED presentation, inclusion of patients who died in hospital, and SBP-stratified use of inotropes, invasive mechanical ventilation, central venous catheter insertion, and intravenous fluids alongside in-hospital outcomes. We examined two clinically defined thresholds: SBP ⩽100 mmHg, as a marker of hypotension, and SBP >140 mmHg, as hypertensive presentation.

Materials And Methods

Study design and setting

This single-center prospective observational study was conducted at Ittefaq Hospital (Trust), a tertiary care teaching hospital in Lahore, Pakistan. Consecutive adults admitted with AHF through the ED between 1 January 2025 and 1 January 2026 were enrolled at presentation and followed until discharge or in-hospital death.

The study followed the Declaration of Helsinki. It was approved by Ittefaq Hospital (Trust). Written informed consent was obtained directly from patients or from next of kin for patients who could not consent at presentation because of their clinical condition. For patients who died or deteriorated before consent could be obtained, consent was taken from next of kin.

Patient selection

Adults aged 18 years or older admitted with a diagnosis of AHF were eligible for inclusion. Both de novo AHF and acute decompensation of previously established HF were considered. Consecutive patients presenting to the ED and subsequently admitted with AHF during the study period were assessed for eligibility.

AHF was diagnosed on the basis of characteristic symptoms and clinical signs, supported by natriuretic peptide testing, chest radiographic findings, and echocardiographic assessment, in accordance with the 2021 European Society of Cardiology guidelines for the diagnosis and treatment of acute and chronic HF [8]. The final diagnosis was established by the treating clinical team based on the overall clinical assessment and available investigations.

Patients younger than 18 years, those admitted primarily for a condition other than AHF, and those with

insufficient clinical documentation to establish the diagnosis or determine the principal study outcomes were not included. Patients with acute coronary syndrome, sepsis, pulmonary embolism, or other acute conditions were considered eligible only when AHF was established as the primary reason for admission rather than an alternative diagnosis. Common cardiovascular and metabolic comorbidities, including hypertension, diabetes mellitus, ischemic heart disease, and chronic kidney disease, were not considered automatic exclusion criteria.

A total of 135 consecutive eligible patients were included in the final analysis.

Data collection

Data were recorded prospectively on a structured proforma (see Appendices). Variables were age, sex, admission SBP, diastolic BP, heart rate, atrial fibrillation at presentation, previous HF, hypertension, diabetes, ischemic heart disease, previous stroke or transient ischemic attack (TIA), asthma or chronic obstructive pulmonary disease, smoking, and baseline HF medications. Admission SBP was the first reading at presentation, taken with a calibrated automated sphygmomanometer; diastolic BP and heart rate came from the same assessment. Patients were grouped by admission SBP as ⩽100, 101-140, or >140 mmHg.

Laboratory and echocardiographic variables included estimated glomerular filtration rate (eGFR), serum lactate, natriuretic peptide levels, and left ventricular ejection fraction (LVEF). N-terminal pro-B-type natriuretic peptide (NT-proBNP) levels were measured using a Siemens automated immunoassay analyzer in the hospital's central laboratory. Blood samples were collected during the initial hospital assessment, preferably within the first 24 hours of admission, and NT-proBNP concentrations were reported in ng/L. Only NT-proBNP measurements were included in the corresponding analyses; BNP measurements, if available, were not considered equivalent to NT-proBNP. eGFR and serum lactate were taken at the initial assessment, and lactate is reported as a baseline variable. LVEF came from echocardiography reports (midpoint where a range was given) and was classified as reduced (<40%), mildly reduced (40%-49%), or preserved (⩾50%).

Management variables were inotrope use, intravenous nitrates, intravenous fluids, central venous catheter insertion, invasive ventilation, and dialysis during admission. Outcomes were in-hospital mortality and length of stay (discharge date minus admission date, in days). Inotrope use, as a treatment decision, was not analyzed as an outcome.

Missing observations were assessed separately for each variable. Available case denominators were used when calculating percentages and summarizing continuous variables. Missing data were handled using pairwise deletion without imputation, and the number of available observations and missing values was reported for each variable.

Statistical analysis

Continuous variables are given as mean ± SD or median (IQR). Categorical variables are given as counts and percentages. Groups were compared with the Kruskal-Wallis test (continuous variables) and the Fisher-Freeman-Halton exact test (categorical variables, because many cells were small). Two dichotomized comparisons were made: SBP ⩽100 versus >100 mmHg, for the clinical effect of hypotension, and SBP >140 versus ⩽140 mmHg, for hypertensive presentation. No deaths occurred above 140 mmHg, so ordinary logistic regression would fail from complete separation; unadjusted and age-adjusted odds ratios (ORs) were therefore estimated with Firth's penalized likelihood logistic regression, with profile likelihood 95% confidence intervals (CIs) and penalized likelihood-ratio p-values. Age was the only covariate because there were few events. Tests were two-sided with $p<0.05$ taken as significant, and no correction was made for multiple comparisons. Analyses were performed using IBM SPSS Statistics version 20.0 (IBM Corp., Armonk, NY), while Firth's penalized logistic regression models were fitted using R version 4.4.2 with the logistf package (R Foundation for Statistical Computing, Vienna, Austria).

Data quality and validation

Two investigators independently reviewed the data for completeness and consistency, and outliers were checked against patient records.

Results

A total of 135 patients were included. The mean age was 65.9 ± 13.9 years, and 68 (50.4%) were male. At presentation, 31 (23.0%) patients had SBP ⩽100 mmHg, 70 (51.9%) had SBP 101-140 mmHg, and 34 (25.2%) had SBP >140 mmHg.

Age, sex, comorbidities, previous HF, atrial fibrillation, stroke/TIA, smoking, and baseline HF medications did not differ significantly across the three SBP groups (all $p>0.05$). LVEF and NT-proBNP did differ ($p=0.049$ and $p=0.042$). Median LVEF was 38% (IQR: 28-42) in the ⩽100 group, 32% (28-42) in the 101-140 group, and 48% (29-56) in the >140 group; LVEF ⩾50% was present in 7 (22.6%), 14 (20.0%), and 16 (47.1%) patients.

Median NT-proBNP was 1,511 ng/L (810-2,840), 1,593 ng/L (850-2,690), and 904 ng/L (510-1,620), respectively. Admission serum lactate (moved here from the management table) did not differ between groups (p=0.592). No correction was made for multiple comparisons (Table 1).

Variable	Total (n=135)	SBP ≤100 (n=31)	SBP 101-140 (n=70)	SBP >140 (n=34)	p-value
Age (years), mean ± SD	65.9 ± 13.9	64.1 ± 13.9	64.9 ± 13.3	69.6 ± 14.7	0.138
Male sex, n (%)	68 (50.4)	18 (58.1)	37 (52.9)	13 (38.2)	0.226
LVEF (%), median (IQR)	38 (28-51)	38 (28-42)	32 (28-42)	48 (29-56)	0.049
NT-proBNP (ng/L), median (IQR)	1,380 (720-2,510)	1,511 (810-2,840)	1,593 (850-2,690)	904 (510-1,620)	0.042
Serum lactate (mmol/L), median (IQR)	2.1 (1.1-3.4)	2.7 (1.1-5.0)	1.9 (1.1-2.8)	2.2 (1.1-2.8)	0.592
Diabetes mellitus, n (%)	87 (64.4)	18 (58.1)	48 (68.6)	21 (61.8)	0.556
Hypertension, n (%)	101 (74.8)	19 (61.3)	54 (77.1)	28 (82.4)	0.124
Ischemic heart disease, n (%)	74 (54.8)	16 (51.6)	42 (60.0)	16 (47.1)	0.432
Previous heart failure, n (%)	51 (37.8)	11 (35.5)	31 (44.3)	9 (26.5)	0.218
Atrial fibrillation at presentation, n (%)	21 (15.6)	5 (16.1)	11 (15.7)	5 (14.7)	1
Acute stroke/TIA, n (%)	15 (11.1)	1 (3.2)	9 (12.9)	5 (14.7)	0.26
Previous stroke/TIA, n (%)	16 (11.9)	2 (6.5)	10 (14.3)	4 (11.8)	0.603
Asthma/COPD, n (%)	18 (13.3)	6 (19.4)	8 (11.4)	4 (11.8)	0.56
Current/ex-smoker, n (%)	25 (18.5)	7 (22.6)	12 (17.1)	6 (17.6)	0.802
ACEi/ARB, n (%)	32 (23.7)	6 (19.4)	15 (21.4)	11 (32.4)	0.412
Beta-blocker, n (%)	56 (41.5)	11 (35.5)	30 (42.9)	15 (44.1)	0.78
MRA, n (%)	35 (25.9)	6 (19.4)	20 (28.6)	9 (26.5)	0.66
Loop diuretic, n (%)	53 (39.3)	11 (35.5)	32 (45.7)	10 (29.4)	0.258
SGLT2 inhibitor, n (%)	27 (20.0)	4 (12.9)	17 (24.3)	6 (17.6)	0.45
ARNI, n (%)	12 (8.9)	3 (9.7)	7 (10.0)	2 (5.9)	0.852

TABLE 1: Baseline characteristics by admission SBP group

ACEi/ARB, angiotensin-converting enzyme inhibitor/angiotensin receptor blocker; ARNI, angiotensin receptor-neprilysin inhibitor; COPD, chronic obstructive pulmonary disease; IQR, interquartile range; LVEF, left ventricular ejection fraction; MRA, mineralocorticoid receptor antagonist; NT-proBNP, N-terminal pro-B-type natriuretic peptide; SBP, systolic blood pressure; SD, standard deviation; SGLT2, sodium-glucose cotransporter 2; TIA, transient ischemic attack.

Continuous variables were compared with the Kruskal-Wallis test and categorical variables with the Fisher-Freeman-Halton exact test. Missing data were handled by pairwise deletion.

Statistically significant p-value (p<0.05)

Lower admission SBP was associated with more intensive support (Table 2). Inotropes were given to 18/31 (58.1%) patients with SBP ⩽100 mmHg, 9/70 (12.9%) with SBP 101-140 mmHg, and 2/34 (5.9%) with SBP >140 mmHg (p<0.001). Invasive ventilation (35.5%, 11.4%, 5.9%; p=0.004), central venous catheter insertion (22.6%, 4.3%, 2.9%; p=0.007) and intravenous fluids (19.4%, 2.9%, 2.9%; p=0.008) followed the same gradient. Intravenous nitrates (p=0.225) and dialysis (p=0.338) did not differ significantly.

Variable	Total (n=135)	SBP ≤100 (n=31)	SBP 101-140 (n=70)	SBP >140 (n=34)	p-value
Inotrope use, n (%)	29 (21.5)	18 (58.1)	9 (12.9)	2 (5.9)	<0.001
Intravenous nitrates, n (%)	10 (7.4)	1 (3.2)	4 (5.7)	5 (14.7)	0.225
Invasive ventilation, n (%)	21 (15.6)	11 (35.5)	8 (11.4)	2 (5.9)	0.004
Central venous catheter insertion, n (%)	11 (8.1)	7 (22.6)	3 (4.3)	1 (2.9)	0.007
Intravenous fluids, n (%)	9 (6.7)	6 (19.4)	2 (2.9)	1 (2.9)	0.008
Dialysis, n (%)	10 (7.4)	4 (12.9)	5 (7.1)	1 (2.9)	0.338
Length of stay (days), median (IQR)	4 (3-7)	6 (4-8)	4 (2-7)	4 (2-6)	0.045
In-hospital mortality, n (%)	19 (14.1)	9 (29.0)	10 (14.3)	0 (0.0)	0.001

TABLE 2: Acute management and outcomes by admission SBP group

IQR, interquartile range; SBP, systolic blood pressure.

Categorical variables were compared with the Fisher-Freeman-Halton exact test and length of stay with the Kruskal-Wallis test.

Statistically significant p-value (p<0.05)

Median length of stay was six days (IQR: 4-8) at SBP ⩽100 mmHg and four days in both the 101-140 group (IQR: 2-7) and the >140 group (IQR: 2-6); the difference was statistically significant (p=0.045). Length of stay was available for 133 of 135 patients.

In total, 19/135 (14.1%) patients died in hospital: 9/31 (29.0%) at SBP ⩽100 mmHg, 10/70 (14.3%) at 101-140 mmHg, and 0/34 (0.0%) at >140 mmHg (p=0.001). Because the >140 group is small, the absence of deaths there does not establish an absence of mortality risk (Table *2*).

The 140-mmHg threshold was prespecified to define hypertensive presentation. In this dichotomized analysis, 101/135 (74.8%) patients had SBP ⩽140 mmHg and 34/135 (25.2%) had SBP >140 mmHg. Firth's penalized logistic regression was used because no deaths occurred in the higher SBP group; p-values are from penalized likelihood-ratio tests of the age-adjusted models. Age was the only covariate because of the small number of events.

Patients with SBP >140 mmHg had lower age-adjusted odds of in-hospital death (OR: 0.07, 95% CI: <0.01-0.52; p=0.004), with 0/34 versus 19/101 deaths. With no events in the higher SBP group and adjustment for age alone, this estimate is imprecise, is likely confounded by LVEF, NT-proBNP, comorbidity, and illness severity, and does not show that the true risk is zero. Estimates for invasive ventilation, central venous catheter insertion, intravenous fluids, and dialysis also favored the higher SBP group, but all CIs included 1 (Table *3*).

Outcome	SBP ≤140 (n=101), n (%)	SBP >140 (n=34), n (%)	Unadjusted OR (95% CI)	Age-adjusted OR (95% CI)	p-value
In-hospital mortality	19 (18.8)	0 (0.0)	0.06 (<0.01-0.47)	0.07 (<0.01-0.52)	0.004
Invasive ventilation	19 (18.8)	2 (5.9)	0.33 (0.06-1.10)	0.36 (0.07-1.22)	0.107
Central venous catheter insertion	10 (9.9)	1 (2.9)	0.39 (0.04-1.76)	0.45 (0.05-2.05)	0.331
Intravenous fluids	8 (7.9)	1 (2.9)	0.49 (0.05-2.31)	0.28 (0.03-1.49)	0.148
Dialysis	9 (8.9)	1 (2.9)	0.44 (0.05-2.00)	0.49 (0.05-2.30)	0.402
Treatment decision (reflects clinical practice; not an outcome)					
Inotrope use	27 (26.7)	2 (5.9)	0.21 (0.04-0.69)	0.23 (0.04-0.76)	0.014

TABLE 3: Association of admission SBP >140 mmHg (versus ≤140 mmHg) with in-hospital outcomes

CI, confidence interval; OR, odds ratio; SBP, systolic blood pressure.

Reference group: SBP ≤140 mmHg. All odds ratios are from Firth's penalized logistic regression; p-values are penalized likelihood-ratio tests of the age-adjusted models.

Statistically significant p-value (p<0.05)

Inotrope use is a treatment decision rather than an outcome. Its lower frequency at higher SBP (2/34, 5.9% versus 27/101, 26.7%; age-adjusted OR: 0.23, 95% CI: 0.04-0.76) reflects clinical practice, since inotropes are given for hypotension, and is not an independent risk relationship; it is shown separately in Table *3* for completeness.

Because the clinical question concerns hypotension, we also compared SBP ⩽100 mmHg (n=31) with SBP >100 mmHg (n=104), a contrast with events in both groups (Table *4*). Patients with SBP ⩽100 mmHg had higher odds of in-hospital death (9/31, 29.0% versus 10/104, 9.6%; OR: 3.80, 95% CI: 1.40-10.33; p=0.010), invasive ventilation (OR: 5.05, 1.93-13.48), central venous catheter insertion (OR: 6.84, 2.01-25.91), and intravenous fluids (OR: 7.39, 1.96-32.91). The estimate for dialysis was imprecise and included 1 (OR: 2.48, 0.65-8.80). The higher inotrope use (OR: 11.14, 4.49-29.25) again reflects the indication for treatment. These ORs are unadjusted; mean age was similar in the two groups (64.1 versus 66.4 years), and confounding by LVEF, NT-proBNP, comorbidity, and illness severity cannot be excluded (Table *4*).

Outcome	SBP >100 (n=104), n (%)	SBP ≤100 (n=31), n (%)	Unadjusted OR (95% CI)	p-value
In-hospital mortality	10 (9.6)	9 (29.0)	3.80 (1.40-10.33)	0.01
Invasive ventilation	10 (9.6)	11 (35.5)	5.05 (1.93-13.48)	0.001
Central venous catheter insertion	4 (3.8)	7 (22.6)	6.84 (2.01-25.91)	0.002
Intravenous fluids	3 (2.9)	6 (19.4)	7.39 (1.96-32.91)	0.003
Dialysis	6 (5.8)	4 (12.9)	2.48 (0.65-8.80)	0.176
Treatment decision (reflects clinical practice; not an outcome)				
Inotrope use	11 (10.6)	18 (58.1)	11.14 (4.49-29.25)	<0.001

TABLE 4: Association of admission SBP ≤100 mmHg (versus >100 mmHg) with in-hospital outcomes

CI, confidence interval; OR, odds ratio; SBP, systolic blood pressure.

Reference group: SBP >100 mmHg. Odds ratios are from Firth's penalized logistic regression for consistency with Table 3; p-values are penalized likelihood-ratio tests.

Statistically significant p-value (p<0.05)

Discussion

This prospective observational study evaluated the relationship between admission SBP, clinical management, and in-hospital outcomes among patients hospitalized with AHF in Pakistan. Three principal findings emerged. First, patients presenting with SBP ⩽100 mmHg had the greatest use of inotropes, invasive mechanical ventilation, central venous catheter insertion, and intravenous fluids; the high inotrope use reflects the clinical indication for treatment and is not an independent risk relationship. Second, in-hospital mortality was highest at SBP ⩽100 mmHg (29.0%) and lowest at >140 mmHg (0%). The ⩽100 versus >100 mmHg comparison, which has events in both groups, showed higher odds of death at ⩽100 mmHg (unadjusted OR: 3.80, 95% CI: 1.40-10.33). Third, length of stay was longest at ⩽100 mmHg (median 6 versus 4 days; p=0.045), a modest difference. These findings support the potential role of admission SBP as an early marker of clinical severity, although the observational design does not establish causality.

The inverse relationship between admission SBP and adverse outcomes is consistent with previous studies. The Organized Program to Initiate Lifesaving Treatment in Hospitalized Patients with Heart Failure (OPTIMIZE-HF) registry demonstrated that lower admission SBP was associated with greater in-hospital and post-discharge mortality [9]. Risk increased as SBP declined, with the lowest mortality among patients presenting with higher SBP [10]. Similarly, a study of older patients hospitalized with HF found that lower admission SBP was associated with increased one-year mortality, independent of other clinical characteristics [11].

Evidence from more recent studies also supports this association. An individual patient data analysis of four randomized clinical trials found that higher SBP was associated with lower 180-day mortality and fewer adverse clinical outcomes [12]. The Kyoto Congestive Heart Failure (KCHF) registry reported higher one-year mortality among patients with lower admission SBP across predefined blood pressure strata [13]. A systematic review and meta-analysis likewise identified an association between lower blood pressure and adverse cardiovascular outcomes in patients with HF [14].

Regional data provide the closest comparison. THFR (1,205 patients, Kerala) reported in-hospital mortality of 8.5% and a median stay of six days [5]; our mortality was higher (14.1%) and our median stay shorter (four days, IQR: 3-7), although differences in case mix and outcome definitions prevent direct comparison. That registry documented admission SBP but did not compare acute interventions across SBP categories [5]. The Kolkata-Coventry registry found that lower admission blood pressure was associated with higher in-hospital mortality in an Indian public hospital cohort, in line with our result at ⩽100 mmHg [6]. In Pakistan, Jafary et al. found that lower admission SBP independently predicted later mortality in new-onset systolic HF but excluded patients who died during the index admission [7]. Our study adds prospective enrolment of consecutive ED patients, inclusion of in-hospital deaths, and a description of acute organ support (inotropes, ventilation, central venous catheters, intravenous fluids) by SBP stratum, using two clinically defined thresholds. Mortality rates across these cohorts cannot be compared directly because populations, recruitment methods, and outcome definitions differ.

The overall in-hospital mortality of 14.1% in the present study highlights the clinical burden of AHF in this hospital population. Direct comparisons with international registries should be interpreted cautiously because differences in case mix, disease severity, referral patterns, treatment availability, and outcome definitions may influence reported mortality [15]. Nevertheless, the observed concentration of deaths in the lower SBP groups supports further evaluation of admission SBP as a practical marker for early clinical assessment in resource-constrained settings.

The association between low SBP and greater use of supportive interventions is physiologically plausible. In patients with AHF, low SBP may reflect impaired cardiac output, systemic hypoperfusion, or advanced ventricular dysfunction [11]. Such patients may be less able to tolerate therapies that reduce preload or afterload and may require more intensive hemodynamic monitoring. Conversely, some patients presenting with higher SBP may have acute pulmonary congestion driven predominantly by increased afterload and fluid redistribution, with relatively preserved systemic perfusion.

Our findings are compatible with these differing clinical presentations. Patients with SBP >140 mmHg had a higher median LVEF and lower median NT-proBNP levels than patients in the lower SBP groups [16]. These differences were nominal (p=0.049 and p=0.042) and were not corrected for multiple comparisons. The study did not directly characterize hemodynamic phenotypes, and the differences should not be interpreted as proof of distinct underlying mechanisms. Moreover, LVEF alone does not fully characterize cardiac output or the severity of congestion.

Inotrope use is a treatment decision, so its strong association with low SBP largely restates the indication for treatment rather than an independent risk relationship. The greater use of inotropes among patients with low admission SBP is consistent with the clinical role of these agents in patients with hemodynamic compromise. Previous studies have also reported associations between inotrope use and adverse outcomes in AHF. The Korean Acute Heart Failure (KorAHF) registry described an association between inotrope use and adverse clinical outcomes, while analyses from the Italian Network on Heart Failure (IN-HF) Outcome registry found that inotrope-treated patients had a particularly high risk of death [17,18]. These associations must be interpreted in the context of confounding by indication: patients receiving inotropes are often more severely ill, and the observed relationship does not establish that treatment itself caused the adverse outcomes.

The findings have several potential clinical implications. Admission SBP is routinely measurable and can be incorporated into the initial assessment of patients with suspected AHF. A low reading should prompt careful evaluation of perfusion, congestion, renal function, and other indicators of clinical deterioration. Where appropriate, it may support decisions regarding monitoring intensity and escalation of care. Conversely, a higher SBP should not be considered sufficient evidence of low risk or used alone to justify early discharge. Treatment decisions should incorporate the complete clinical assessment, response to therapy, renal function, oxygenation, and other prognostic indicators.

The absence of in-hospital deaths among patients with SBP >140 mmHg should be interpreted cautiously. This subgroup comprised 34 patients, and an absent event does not show that the true risk of death is zero. The age-adjusted OR of 0.07 (95% CI: <0.01-0.52) is imprecise, and because adjustment was limited to age, it is probably confounded by LVEF, NT-proBNP, comorbidity, and illness severity; patients with SBP >140 mmHg had higher LVEF and lower NT-proBNP. Larger prospective studies are needed to test whether the association persists after adjustment for these factors.

Limitations

This study has several limitations. First, it was conducted at a single tertiary care hospital with a small sample, which limits generalizability and the precision of estimates. Second, deaths were few and none occurred in the highest SBP group; Firth's penalized regression addresses separation, but the estimates remain imprecise. Third, the >140 versus ⩽140 mmHg models were adjusted for age only, and the ⩽100 versus >100 mmHg comparison was unadjusted, so the mortality ORs are essentially unadjusted for LVEF, NT-proBNP, comorbidity, and illness severity, and residual confounding is likely to be large. Fourth, p-values were not corrected for the many comparisons made, and some variables had missing values, as shown in Table *1*.

Fifth, admission SBP was categorized into groups, which may obscure differences within categories and does not establish a continuous dose-response relationship. Sixth, the study assessed in-hospital outcomes only and did not evaluate post-discharge mortality, readmissions, or longer-term functional status. Finally, the single-center observational design precludes causal inference, and treatment decisions varied with clinical severity. Future multicenter prospective studies should evaluate SBP alongside other markers of perfusion, congestion, renal function, and cardiac function, and larger datasets would permit more robust multivariable adjustment and analysis of continuous SBP.

Conclusions

In this ED-enrolled AHF cohort, an admission SBP of 100 mmHg or lower identified patients with about three

times the in-hospital mortality (29.0% versus 9.6%; unadjusted OR: 3.80, 95% CI: 1.40-10.33) and a much greater need for inotropes (58.1% versus 10.6%) and invasive ventilation (35.5% versus 9.6%), which may support closer monitoring and early escalation of care for these patients. No deaths occurred at SBP >140 mmHg (0/34), but this estimate is imprecise and does not show that the risk is zero. Because adjustment was limited to age, residual confounding by LVEF, NT-proBNP, comorbidity, and illness severity is likely.

Admission SBP is readily available at presentation and should be interpreted alongside other clinical and laboratory findings rather than used as an isolated prognostic measure. Multicenter studies with larger sample sizes and longer follow-up are needed to validate these findings and clarify their implications for AHF management in resource-limited healthcare settings.

Appendices

Data collection proforma

Section 1: Patient Demographics

Name: _____

MR/IHT Number: _____

Age (years): _____

Sex (M/F): _____

Hospital Stay/Date of Admission: _____

Patient Location: ____

Section 2: Medical History

History of heart failure: ____

Hypertension: _____

Cigarette smoking: ____

Ischemic heart disease: ____

Atrial fibrillation or flutter: ____

Previous TCI/stroke: ____

Diabetes: ____

Asthma/COPD: ___

Section 3: Clinical Parameters at Admission

Systolic blood pressure (mmHg): _____

Atrial fibrillation: ____

Diastolic blood pressure (mmHg): _____

Ejection fraction (%): _____

Heart rate, beats per minute: ____

New York Heart Association class before admission (Class I-IV): ____

Section 4: Laboratory Investigations

ProBNP, ng/L: _____

eGFR, mL/min per 1.73 m^2: ____

Section 5: Medications at baseline

Angiotensin-converting enzyme inhibitors/ARBs: ____

Beta-blockers: ____

Aldosterone antagonists: ____

Loop diuretics: ____

SGLT-2 Inhibitors: ____

ARNI: ____

Section 6: In-Hospital Intervention

Inotrope usage: ____

Invasive ventilation: ____

Serum lactate: ____

Dialysis: ____

CV Insertion: ____

IV Fluid: ____

Section 7: Outcome

Length of Hospital Stay (days): ____

Mortality: ____

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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