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Research Article

Prognostic Accuracy of Albumin Bilirubin Score for Outcomes of In-Hospital Patients with Heart Failure

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ABSTRACT
Background: Hepatic congestion and hypoperfusion are integral to heart-failure (HF) pathophysiology and lead to hyper-bilirubinaemia and hypo-albuminaemia. The logarithmic Albumin–Bilirubin (ALBI) score, already validated in hepatology, may provide an inexpensive prognostic marker in HF, but data for acute, in-hospital outcomes remain sparse.
Objective: To compare the prognostic accuracy of ALBI with N-terminal pro-B-type natriuretic peptide (NT-proBNP) and with a composite (ALBI + NT-proBNP) for predicting all-cause in-hospital mortality among adults admitted with acute HF.
Methods: In this 18-month prospective cohort at a tertiary centre in New Delhi (n = 100, Framingham criteria), serum albumin, total bilirubin and NT-proBNP were assayed within 24 h of admission; ALBI was calculated as $-0.0852 \times$ albumin (g L^{-1}) + $0.66 \times \log_{10}$(bilirubin µmol L^{-1}). Primary end-point was in-hospital death. Discrimination was examined with ROC curves.
Results: Mean age 52.6 ± 16.4 y; 63 % male. Mortality occurred in 39 %. Median ALBI −2.20 (IQR −2.68 to −1.59). An ALBI threshold ≥ −2.08 yielded 90 % sensitivity and 79 % specificity; AUROC = 0.889 versus 0.776 for NT-proBNP (cut-off ≥ 10 242 pg mL^{-1}). The composite marker achieved AUROC = 0.929 (p = 0.001 vs NT-proBNP). Across HF phenotypes, ALBI ≥ −2.27 identified 93.5 % of deaths in HFrEF and 100 % in HFmrEF.
Conclusions: A single ALBI measurement on admission is non-inferior to NT-proBNP and markedly enhances prognostic accuracy when combined with NT-proBNP. Given its negligible cost and bedside availability, ALBI offers a practical risk-stratification tool, particularly where natriuretic-peptide assays are inaccessible.

Keywords: Albumin–Bilirubin Score, Heart Failure, NT-Probnp, In-Hospital Mortality, Prognostic Biomarker, Hepatic Congestion.

INTRODUCTION

Heart failure (HF) affects an estimated 64 million people worldwide, imposing a growing clinical and economic burden (1). In India alone, HF now contributes more than 14 % of disability-adjusted life-years lost to cardiovascular disease (2). Contemporary risk stratification relies heavily on natriuretic peptides such as N-terminal pro-B-type natriuretic peptide (NT-proBNP); however, assay cost and the confounding influence of renal dysfunction restrict their universal applicability (3, 4).

Systemic venous congestion and forward hypoperfusion in HF commonly impair hepatic function, resulting in hypo-albuminaemia and hyper-bilirubinaemia (5). Each abnormality has been linked to adverse outcomes, yet individually they capture only a single dimension of hepatic reserve. The Albumin–Bilirubin (ALBI) score combines both variables in a simple logarithmic equation originally validated for hepatocellular carcinoma. Emerging evidence suggests that ALBI also predicts long-term mortality in chronic and critically ill HF cohorts (6). A Japanese study further indicates that an admission ALBI cut-off improves discrimination for in-hospital death when added to NT-proBNP (7).

Nonetheless, the incremental prognostic value of ALBI over natriuretic peptides during an acute HF admission—and its performance across ejection-fraction phenotypes—remains poorly defined, particularly in lower-resource settings where inexpensive bedside markers are most needed. We therefore conducted a prospective cohort study of adults hospitalised with acute HF to (i) evaluate the discriminative ability of ALBI for all-cause in-hospital death, (ii) compare ALBI with NT-proBNP, and (iii) determine whether a composite ALBI + NT-proBNP model improves prediction. We additionally explored ALBI performance within HF phenotypes classified by left-ventricular ejection fraction (LVEF).

METHODS

Study Design and Setting

We performed a prospective, single-centre cohort study from January 2023 to June 2024 in the Departments of General Medicine and Cardiology at Vardhman Mahavir Medical College & Safdarjung Hospital, New Delhi. The protocol received institutional ethics-committee approval, and written informed consent was obtained from all participants.

Participants

Adults (> 18 years) admitted with acute HF fulfilling Framingham criteria were eligible. Exclusion criteria comprised acute myocardial infarction, chronic liver disease, pregnancy, malignancy, or other causes of hypo-albuminaemia. Using an expected ALBI sensitivity of 63 % for in-hospital death (8), 95 % confidence and 10 % absolute precision, a minimum sample of 97 was required; we enrolled 100 consecutive patients to allow for attrition.

Data Collection

Within 24 h of admission the following were recorded: demographics, co-morbidities, New York Heart Association (NYHA) class, vital signs, electrocardiogram, chest radiograph and two-dimensional echocardiography. Four millilitres of venous blood yielded:

- Serum albumin (bromocresol-green, ADVIA-200 auto-analyser).
- Total bilirubin (Jendrassik–Grof, ADVIA-200).
- NT-proBNP (STANDARD-F fluorescent immuno-assay).

The ALBI score was calculated as $-0.0852 \times$ albumin (g L^{-1}) $+ 0.66 \times \log_{10}$ (total bilirubin μmol L^{-1}). HF phenotypes were defined as heart-failure with reduced ejection fraction (HFrEF ≤ 40 %), mildly reduced (HFmrEF 41–49 %) and preserved (HFpEF ≥ 50 %).

Outcome and Statistical Analysis

The primary endpoint was all-cause in-hospital mortality. Data normality was assessed with the Shapiro–Wilk test. Continuous variables are expressed as mean ± SD or median (inter-quartile range, IQR) and compared using the unpaired t-test or Mann–Whitney U test. Categorical variables were compared with χ^2 or Fisher's exact test. Receiver-operating-characteristic (ROC) curves generated areas under the curve (AUROC), optimal cut-offs (Youden index), sensitivity, specificity, positive-predictive value (PPV) and negative-predictive value (NPV). Pairwise AUROC comparisons employed DeLong's method. Multivariable logistic regression adjusted for Get-With-the-Guidelines-HF risk score, serum urea and creatinine. Two-tailed $p < 0.05$ denoted significance. Analyses were conducted in SPSS v25 (IBM).

RESULTS

Baseline Characteristics

Table 1 summarises baseline data for all 100 participants. Mean age was 52.6 ± 16.4 years; 63 % were male. Median NT-proBNP was 7 861 pg mL^{-1} (IQR 4 420–14 390) and median ALBI −2.20 (IQR −2.68 to −1.59). HFrEF accounted for 64 % of cases, HFmrEF 12 % and HFpEF 24 %. Overall in-hospital mortality was 39 %.

Table 1 — Baseline profile (n = 100)

Variable	Mean ± SD / n (%)
Age, years	52.6 ± 16.4
Male sex	63 (63 %)
NYHA class III–IV	76 (76 %)
Serum albumin, g dL^{-1}	3.44 ± 0.73
Total bilirubin, mg dL^{-1}	1.42 ± 0.85
ALBI score	−2.09 ± 0.68
NT-proBNP, pg mL^{-1}	9 874 ± 7 369

ALBI Categories and Outcome

Mean ALBI among non-survivors was −1.53 ± 0.55 versus −2.48 ± 0.49 in survivors ($p < 0.001$). Distribution of deaths across ALBI strata is shown in Table 2.

Table 2 — ALBI Categories versus In-Hospital Outcome

ALBI category	Survivors (n = 61)	Deaths (n = 39)	χ^2	p-value
≥ −2.27	12 (20 %)	27 (69 %)	44.6	< 0.001
−2.59 to −2.27	17 (28 %)	3 (8 %)		
≤ −2.59	32 (52 %)	9 (23 %)		

Discriminative Performance

ROC analysis demonstrated AUROC 0.889 for ALBI, 0.776 for NT-proBNP, and 0.929 for the composite ALBI + NT-proBNP (Figure 1). Operating characteristics appear in Table 3. DeLong testing indicated non-inferiority of ALBI versus NT-proBNP ($p = 0.076$) and superiority of the composite model ($p = 0.001$).

Table 3 — Roc-Derived Prognostic Metrics

Marker	AUROC (95 % CI)	Optimal cut-off	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
ALBI	0.889 (0.82–0.96)	≥ −2.08	90	79	73	92
NT-proBNP	0.776 (0.68–0.87)	≥ 10 242 pg mL^{-1}	80	80	72	86
ALBI + NT-proBNP	0.929 (0.88–0.98)	≥ 0.524	82	90	84	89

Multivariable Analysis

After adjustment, ALBI remained independently associated with mortality (adjusted OR 3.8, 95 % CI 1.7–8.6; $p = 0.001$).

DISCUSSION

This prospective study demonstrates that the admission ALBI score, derived solely from albumin and bilirubin, accurately identifies patients at risk of in-hospital death from acute HF. The AUROC of 0.889 surpasses that reported by Han et al. for 30-day mortality in a Chinese cohort (8) and approaches the 0.93 achieved when ALBI was combined with NT-proBNP in a Japanese series (7). Our composite model replicated that superiority, underscoring the complementary pathophysiological information provided by hepatic and cardiac biomarkers.

Unlike NT-proBNP, which may be spuriously elevated in chronic kidney disease or sepsis (4), ALBI is unaffected by renal filtration. Its inexpensive reagents (< ₹40 ≈ 0.5 USD) and rapid turnaround make it particularly attractive for low-resource settings where natriuretic-peptide assays remain unavailable. Pathophysiologically, ALBI integrates hepatocellular synthetic reserve (albumin) and cholestatic congestion (bilirubin), both direct correlates of right-sided filling pressures and systemic venous congestion (5). The strong association between higher ALBI strata and NYHA class, worsening renal indices and mortality in our cohort supports this mechanistic link.

Importantly, ALBI maintained prognostic accuracy across HFrEF and HFmrEF phenotypes, and a non-significant trend in HFpEF. This breadth of applicability aligns with data in critically ill HF (6) and in ambulatory HFrEF populations (9), suggesting that ALBI reflects haemodynamic compromise irrespective of LVEF.

CONCLUSION

A single, inexpensive ALBI measurement on admission offers prognostic performance comparable to NT-proBNP and enhances prediction when the two markers are combined. Implementation of ALBI-based triage may improve resource allocation and outcomes, particularly in low-resource environments.

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