Research Square

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Association of the ALBI Score with Acute Kidney Injury in Patients with Acute Myocardial Infarction: A Retrospective Analysis Drawing on MIMIC-IV and eICU-CRD

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Research Article

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Abstract

Background: This study systematically examined the associations of the ALBI score with the risks of AKI and IHM among patients with AMI using data from two large critical care databases, MIMIC-IV and eICU-CRD.

Methods: Data were derived from eICU-CRD and MIMIC-IV. All subjects were grouped into four categories (Q1–Q4) by quartiles of the ALBI score. The primary outcome was AKI, and the secondary outcome was IHM. Multivariable logistic regression models were applied to estimate the strength of the associations of the ALBI score with AKI and IHM. Restricted cubic splines (RCS) were applied to investigate dose–response associations of the ALBI score with the outcome measures. Subgroup analyses were implemented to verify the stability of the findings.

Results: 2,084 subjects with AMI were included from MIMIC-IV and 4,309 subjects with AMI from eICU-CRD. Multivariable logistic regression indicated that the ALBI score was significantly and positively linked to the risk of AKI (MIMIC-IV: OR [95% CI]: 1.989 [1.504–2.644]; eICU-CRD: 1.464 [1.249–1.719]; both $P < 0.001$). RCS regression indicated linear positive associations of the ALBI score with AKI and IHM.

Conclusions: The ALBI score demonstrates independent, linear positive associations with the risk of AKI and IHM among patients with AMI.

1 Introduction

Acute myocardial infarction (AMI), contributing to roughly 7 million deaths per year, constitutes a principal cause of mortality and disability globally [1]. With the widespread adoption and refinement of reperfusion therapy, short-term survival among patients with AMI has improved substantially; however, acute kidney injury (AKI) remains a prevalent and devastating complication [2]. AKI occurs in 15%–45% of patients with AMI. The precise figure varies according to diagnostic criteria, study populations, and treatment strategies [3, 4]. AKI prolongs hospital stay, increases medical costs, elevates the risk of in-hospital mortality (IHM) 2- to 4-fold, and is closely associated with chronic kidney disease and long-term adverse cardiovascular events [5, 6]. Currently available risk assessment tools for post-AMI AKI predominantly rely on renal function indicators measured at a single time point (e.g., baseline creatinine) or fail to adequately integrate composite biomarkers reflecting multi-organ functional status, thereby yielding limited predictive performance [7, 8]. The development of AKI engages complex pathophysiological mechanisms encompassing inflammatory responses, microcirculatory disturbances, oxidative stress, and inter-organ crosstalk. A single indicator, therefore, rarely achieves precise risk stratification [4]. Identifying additional indicators for early detection of high-risk individuals is important for optimizing perioperative management and improving prognosis.

As an objective, readily calculable metric of hepatic function, the albumin-bilirubin (ALBI) score is derived from the concentrations of total bilirubin (TBIL) and serum albumin (SA). First proposed by Johnson et al. [9] in 2015 for grading hepatic function in individuals with hepatocellular carcinoma, this score has subsequently been extended to the prognostic evaluation of liver cirrhosis, liver transplantation, and various malignancies [10]. Recently, the ALBI score has garnered growing attention in the cardiovascular domain. According to a systematic review and meta-analysis, this score is significantly associated with the mortality risk and major adverse cardiovascular events across multiple cardiovascular diseases [11]. An investigation by Zhao et al. utilizing the Medical Information Mart for Intensive Care IV (MIMIC-IV) demonstrates that each one-unit increment in the ALBI score corresponds to a 32% heightened risk of 28-day mortality in individuals receiving percutaneous coronary intervention (PCI) [12]. Among individuals with heart failure (HF), Gao et al. observe a nonlinear association of the ALBI score with 30-, 90-, and 360-day mortality rates (threshold: −1.92) [13]. Kishihara et al. corroborate that subjects with a discharge ALBI score ≥ − 2.25 face an 82% elevated risk of the composite endpoint [14]. Among individuals receiving implantation of a transcatheter aortic valve, Balaban et al. report that the risk of long-term mortality is independently predicted by the ALBI score (hazard ratio [HR]: 1.86) [15]. Yildirim et al. further confirm that the strongest predictor of 30-day mortality is the ALBI score [16]. Nevertheless, investigations into the associations of this score with AKI and IHM specifically among patients with AMI remain sparse, and whether the ALBI score can serve as an effective tool for early risk stratification has yet to be determined.

Accordingly, the present investigation utilized the large-scale public MIMIC-IV and eICU Collaborative Research Database (eICU-CRD) to systematically examine the associations of the ALBI score with the occurrence of AKI and IHM in individuals with AMI and to explore the dose–response relationships and possible effect modifiers. It seeks to offer new evidence-based support for early risk stratification and clinical decision-making among these individuals.

2 Methods

2.1 Data Sources

Data originated from two databases: MIMIC-IV and eICU-CRD.

MIMIC-IV, a single-center, large-scale critical care repository, comprises clinical data from 65,366 adult individuals admitted to intensive care units (ICUs) at the Beth Israel Deaconess Medical Center (Boston, USA) between 2008 and 2022. It encompasses detailed physiological parameters, laboratory measurements, medication records, and clinical outcomes [17]. eICU-CRD, spanning 208 hospitals throughout the continental United States, comprises 139,367 adult ICU admissions from 2014 to 2015. This database offers broader geographic and hospital-type distributions [18].

The investigators acquired the Collaborative Institutional Training Initiative after completing training in human subjects protection and passing the required examination. Both databases received approval from their respective institutional ethical review boards. Since all information about subjects was anonymized, informed consent was not required in this investigation.

2.2 Study Design and Population

The current retrospective cohort investigation enrolled adult individuals from eICU-CRD and MIMIC-IV with a first ICU admission and a diagnosis of AMI.

Identical inclusion criteria were applied across both databases: (i) age ≥ 18 years; (ii) first ICU admission; (iii) documented diagnosis of AMI. Exclusion criteria comprised: (i) survival time < 24 h or length of ICU stay (LOS ICU) < 24 h; (ii) absent measurement of SA or TBIL in the initial 24 h after ICU admission. Figure <link rid="fig1">1</link>-A and 1-B depict the screening process.

Following screening, 4,309 (eICU-CRD) and 2,084 (MIMIC-IV) subjects were ultimately enrolled. Based on quartiles of the ALBI score, subjects from each database were assigned to four groups: in MIMIC-IV, Q1 comprised 520 subjects, Q2 515, Q3 525, and Q4 524; in eICU-CRD, Q1 comprised 1,054 subjects, Q2 1,097, Q3 1,077, and Q4 1,081.

2.3 Data Extraction

Structured query and data extraction from both databases were performed using PostgreSQL 17.0. Extracted variables covered: (i) demographics: sex, race, age; (ii) vital signs: respiratory rate; diastolic blood pressure (DBP), heart rate, systolic blood pressure (SBP); (iii) comorbidities: diabetes, coronary atherosclerosis, atrial fibrillation, HF, hypertension, chronic obstructive pulmonary disease, rheumatic disease, sepsis; (iv) therapeutic interventions: anticoagulants, antiplatelet agents, loop diuretics, beta-blockers, vasoactive agents, statins, renin-angiotensin system inhibitors, mechanical ventilation; (v) laboratory parameters: albumin, anion gap, alanine aminotransferase, calcium, red cell distribution width (RDW), bicarbonate, creatinine, blood urea nitrogen (BUN), hemoglobin, chloride, hematocrit, mean corpuscular volume, potassium, sodium, white blood cell (WBC) count, TBIL; (vi) disease severity score: Sequential Organ Failure Assessment; (vii) clinical outcomes: IHM, LOS ICU.

For each laboratory parameter, the first measurement in the first 24 h after ICU admission was employed to guarantee that all measurements were completed before admission and clinical interventions. Missing data were handled according to the following strategy: variables with missing data exceeding 20% were removed; for those with less than 20% missing, multiple imputation by chained equations was conducted. Five imputed datasets were produced, and analytic results were pooled. In the present investigation, the missingness for all included variables remained below 20%; consequently, no variables were eliminated.

2.4 Outcome Measures

AKI was the primary outcome. IHM was the secondary outcome.

AKI was determined according to the Kidney Disease: Improving Global Outcomes guidelines [19]. The specific diagnostic criteria were: a ≥ 1.5-fold elevation from baseline in the preceding 7 days or an elevation in serum creatinine ≥ 0.3 mg/dL (≥ 26.5 μmol/L) in 48 h. IHM was ascertained as in-hospital death, irrespective of cause, encompassing deaths occurring in the ICU or on general wards.

2.5 Computation of ALBI Score

The ALBI score, derived from the concentrations of SA and TBIL, is an objective, readily calculable measure of hepatic function. The present investigation employed the equation proposed by Johnson et al. [9]:

$$\text{ALBI} = (\log_{10} \text{TBIL}\ [\mu\text{mol/L}] \times 0.66) + (\text{albumin}\ [\text{g/L}] \times -0.0852)$$

Albumin in g/dL was expressed as g/L (1 g/dL = 10 g/L). TBIL in mg/dL was converted to μmol/L (1 mg/dL = 17.1 μmol/L). Subjects were grouped into four categories (Q1–Q4) by quartiles of the ALBI score.

2.6 Statistical Analysis

Statistical analyses were carried out in R (v4.3.2) employing two-sided tests, with $P < 0.05$ considered statistically significant. Continuous variables exhibiting normality were described as mean ± standard deviation. One-way analysis of variance was employed for between-group

comparisons. Skewed data were reported as median (interquartile range). The Kruskal-Wallis rank sum test was utilized for comparisons. Categorical data were summarized as frequencies and percentages. The *Pearson* chi-squared test or *Fisher*'s exact test was adopted for between-group comparisons.

Prior to multivariable logistic regression, multicollinearity diagnostics were conducted on all included variables utilizing the variance inflation factor (VIF). All included variables had VIF values below 5, indicating no substantial collinearity (Supplementary Tables S1, S2).

The strength of the associations of the ALBI score with AKI and IHM was estimated via binary logistic regression. Odds ratios (ORs) and 95% confidence intervals (CIs) were computed to quantify these associations. Three sequentially adjusted regression models were established to account for potential confounders. Model 1 was crude. Model 2 accounted for race, sex, and age. Model 3 additionally accounted for heart rate, SBP, respiratory rate, DBP, diabetes, hypertension, anticoagulants, BUN, beta-blockers, chloride, antiplatelet agents, anion gap, loop diuretics, bicarbonate, statins, creatinine, vasoactive agents, calcium, hemoglobin, WBC count, potassium, mean corpuscular volume, and RDW.

Restricted cubic splines (RCS) were incorporated to examine the dose–response association of the ALBI score, analyzed as a continuous variable, with the outcome measures. The ALBI score distribution was modeled using four knots positioned at the 95th, 65th, 35th, and 5th percentiles. RCS regression was conducted on the basis of Model 3 while concurrently accounting for other covariates.

To examine the stability of the associations of the ALBI score with the outcome measures and to probe possible effect modifiers, the following subgroup variables were prespecified for stratified analyses: sex (female vs. male), hypertension (absent vs. present), race (White vs. other), age (< 70 vs. ≥ 70 years), and diabetes (absent vs. present). All subgroup analyses were performed employing Model 3. Interaction tests were applied to assess the effect modification of each subgroup variable on the associations of interest. Specifically, the product term between the ALBI score and the subgroup variable was incorporated into the regression model. The significance of the interaction was evaluated via the likelihood ratio test. An interaction term $P < 0.05$ suggested a marked effect modification.

3 Results

3.1 Baseline Characteristics of Study Population

This investigation incorporated 2,084 (MIMIC-IV) and 4,309 (eICU-CRD) subjects with AMI.

In MIMIC-IV (Table 1), the median age was 70.96 (61.48, 80.96) years. Males accounted for 61.56% (1,283/2,084). With ascending ALBI score, heart rate gradually increased (median heart rate from Q1 to Q4: 84, 86, 90, 90 beats/min; $P < 0.001$), while SBP and DBP progressively declined ($P < 0.001$). Regarding comorbidities, the prevalence of coronary atherosclerosis, sepsis, HF, and hypertension, together with the rates of vasoactive agent use and mechanical ventilation, all rose markedly as the ALBI score rose ($P < 0.001$). Laboratory results demonstrated that, as the ALBI score elevated, the concentrations of albumin progressively decreased (median from Q1 to Q4: 3.90, 3.40, 3.00, 2.50 g/dL; $P < 0.001$), whereas TBIL, BUN, creatinine, RDW, and WBC count progressively rose ($P < 0.001$). With respect to clinical outcomes, the respective incidence of AKI across Q1 to Q4 was 77.88%, 81.94%, 85.52%, and 89.50% ($P < 0.001$). Respective IHM rates were 9.62%, 12.43%, 17.33%, and 31.87% ($P < 0.001$). As the ALBI score rose, the LOS ICU was prolonged ($P < 0.001$).

Table 1
Baseline characteristics of subjects with AMI by quartiles of the ALBI score in MIMIC-IV

Variables	All (N = 2,084)	Q1 (N = 520)	Q2 (N = 515)	Q3 (N = 525)	Q4 (N = 524)	P-value[1]
Demographics						
Sex, n(%)						0.207
Female	801(38.44%)	184(35.38%)	200(38.83%)	219(41.71%)	198(37.79%)	
Male	1,283(61.56%)	336(64.62%)	315(61.17%)	306(58.29%)	326(62.21%)	
Race, n(%)						0.406
Others	801(38.44%)	213(40.96%)	200(38.83%)	200(38.10%)	188(35.88%)	
White	1,283(61.56%)	307(59.04%)	315(61.17%)	325(61.90%)	336(64.12%)	
Age, years	71(61,81)	69(59,79)	72(62,82)	73(63,81)	70(61,80)	0.002
Vital signs						
HR(beats/min)	87(75,101)	84(72,95)	86 (74,99)	90 (76,102)	90 (78,105)	< 0.001
SBP(mmHg)	120 (105,140)	127 (111,146)	122 (107,140)	119 (104,137)	112 (99,131)	< 0.001
DBP(mmHg)	68 (58,82)	74 (62,86)	69 (57,83)	67 (57,79)	65 (55,79)	< 0.001
RR(breaths/min)	20 (16,24)	19 (16,22)	20 (16,24)	20 (16,24)	20 (16,25)	< 0.001
Comorbidity						
Diabetes, n(%)	964(46.26%)	239(45.96%)	237(46.02%)	263(50.10%)	225(42.94%)	0.141
AF, n(%)	907(43.52%)	218(41.92%)	211(40.97%)	239(45.52%)	239(45.61%)	0.297
COPD, n(%)	372(17.85%)	89(17.12%)	94(18.25%)	98(18.67%)	91(17.37%)	0.903
CAS, n(%)	1,510(72.46%)	438(84.23%)	399(77.48%)	359(68.38%)	314(59.92%)	< 0.001
HF, n(%)	1,361(65.31%)	312(60.00%)	352(68.35%)	372(70.86%)	325(62.02%)	< 0.001
Hypertension, n(%)	1,715(82.29%)	428(82.31%)	439(85.24%)	446(84.95%)	402(76.72%)	< 0.001
Rheumatic diseases, n(%)	369(17.71%)	66(12.69%)	113(21.94%)	105(20.00%)	85(16.22%)	< 0.001
Sepsis, n(%)	1,325(63.58%)	237(45.58%)	293(56.89%)	371(70.67%)	424(80.92%)	< 0.001
Treatment						
Anticoagulant, n(%)	1,608(77.16%)	429(82.50%)	416(80.78%)	401(76.38%)	362(69.08%)	< 0.001
Antiplatelet, n(%)	1,095(52.54%)	352(67.69%)	297(57.67%)	273(52.00%)	173(33.02%)	< 0.001
Beta-blocker, n(%)	707(33.93%)	254(48.85%)	208(40.39%)	134(25.52%)	111(21.18%)	< 0.001
Loop diuretics, n(%)	723(34.69%)	170(32.69%)	197(38.25%)	210(40.00%)	146(27.86%)	< 0.001
RAS drugs, n(%)	130(6.24%)	64(12.31%)	34(6.60%)	22(4.19%)	10(1.91%)	< 0.001
Statins, n(%)	979(46.98%)	318(61.15%)	281(54.56%)	243(46.29%)	137(26.15%)	< 0.001
Vasoactive drugs, n(%)	863(41.41%)	135(25.96%)	177(34.37%)	240(45.71%)	311(59.35%)	< 0.001
Ventilation, n(%)	916(43.95%)	173(33.27%)	207(40.19%)	247(47.05%)	289(55.15%)	< 0.001
Lab test results						
Albumin (g/dL)	3.3(2.8,3.7)	3.9(3.7,4.1)	3.4(3.3,3.6)	3.0(2.9,3.2)	2.5(2.2,2.8)	< 0.001

Abbreviations: HR, Heart rate; CAS, Coronary atherosclerosis; SBP, Systolic blood pressure; AF, Atrial fibrillation; DBP, Diastolic blood pressure; HF, Heart failure; RAS drugs, Renin–angiotensin system (RAS) drugs; BUN, Blood urea nitrogen; RR, Respiratory rate; AST, Aspartate aminotransferase; AKI, Acute kidney injury; MCV, Mean corpuscular volume; ALT, Alanine aminotransferase; RDW, Red cell distribution width; SOFA, Sequential Organ Failure Assessment; LOS ICU, Length of ICU stay; WBC, White blood cell count; ALBI, Albumin-bilirubin grade; RBC, Red blood cell count.

Variables	All (N = 2,084)	Q1 (N = 520)	Q2 (N = 515)	Q3 (N = 525)	Q4 (N = 524)	P-value[1]
Demographics						
ALT (U/mL)	34(19,79)	30 (19,58)	31(18,61)	34 (19,82)	44 (21,135)	< 0.001
Anion gap (mmol/L)	15 (13,18)	15(13,18)	15 (13,18)	15 (13,18)	15 (12,19)	0.877
AST (U/mL)	57 (29,153)	55 (26,151)	47 (27,115)	55 (31,143)	78(36,263)	< 0.001
Bicarbonate (mmol/L)	22(19,24)	23(20,25)	22(19,24)	21(19,24)	20(17,23)	< 0.001
BUN (mg/dL)	26(17,45)	21(15,32)	25(16,43)	30(18,48)	31(19,52)	< 0.001
Calcium (mg/dL)	8.4(7.8,8.9)	8.8(8.4,9.2)	8.5(8.1,8.9)	8.3(7.8,8.7)	7.8(7.3,8.4)	< 0.001
Chloride (mmol/L)	103(99,107)	103(99.,105)	102(98,106)	103(99,107)	104(99,109)	< 0.001
Creatinine (mg/dL)	1.3(0.9,2.1)	1.1(0.8,1.6)	1.2(0.9,2.1)	1.4(0.9,2.3)	1.4(1.0,2.4)	< 0.001
Hemoglobin (g/dL)	10.8(9.0,12.6)	12.3(10.5,13.9)	11.1(9.4,12.5)	10.3(8.7,11.9)	9.9(8.3,11.3)	< 0.001
Hematocrit (%)	33.1(28.0,38.1)	37.1(32.2,41.4)	33.5(29.3,37.8)	32.0(26.7,36.5)	29.9(25.5,34.8)	< 0.001
MCV (fL)	92(88,96)	91(87,95)	92(87,96)	92(88,96)	92(88,96)	0.005
Potassium (mmol/L)	4.3(3.9,4.7)	4.2(3.9,4.7)	4.2(3.8,4.7)	4.3(3.9,4.7)	4.3(3.8,4.8)	0.497
RBC (10^{12}/mL)	3.6(3.0,4.2)	4.0(3.5,4.6)	3.7(3.2,4.2)	3.5(2.9,4.0)	3.3(2.8,3.8)	< 0.001
RDW (%)	14.5(13.5,16.0)	13.8(13.1,14.9)	14.3(13.2,15.6)	14.7(13.8,16.2)	15.4(14.2,17.2)	< 0.001
Sodium (mmol/L)	138(135,141)	138(136,140)	138(135,140)	138(135,141)	138(134,141)	0.105
Total bilirubin (mg/dL)	0.6(0.4,1.0)	0.5(0.3,0.6)	0.6(0.4,0.8)	0.6(0.4,1.0)	1.1(0.6,2.3)	< 0.001
WBC (10^9/mL)	11.8(8.4,16.4)	11.4(8.6,14.8)	11.4(8.2,15.7)	11.8(8.5,15.6)	13.1(8.5,19.1)	< 0.001
Illness severity score						
SOFA	5(3,9)	4(2,6)	5(2,7)	6(3,9)	8(6,11)	< 0.001
Clinical outcomes						
LOS ICU (day)	3.2(1.9,6.0)	2.6(1.8,4.3)	3.0(1.9,5.5)	3.7(2.1,6.5)	4.1(2.1,8.3)	< 0.001
In-hospital mortality (%)	372(17.85%)	50(9.62%)	64(12.43%)	91(17.33%)	167(31.87%)	< 0.001
AKI, n(%)	1,745(83.73%)	405(77.88%)	422(81.94%)	449(85.52%)	469(89.50%)	< 0.001
ALBI	-2.1(-2.5,-1.7)	-2.7(-2.9,-2.6)	-2.3(-2.4,-2.2)	-1.9(-2.0,-1.8)	-1.3(-1.5,-1.0)	< 0.001

[1] Pearson's Chi-squared test; Kruskal-Wallis rank sum test

Abbreviations: HR, Heart rate; CAS, Coronary atherosclerosis; SBP, Systolic blood pressure; AF, Atrial fibrillation; DBP, Diastolic blood pressure; HF, Heart failure; RAS drugs, Renin–angiotensin system (RAS) drugs; BUN, Blood urea nitrogen; RR, Respiratory rate; AST, Aspartate aminotransferase; AKI, Acute kidney injury; MCV, Mean corpuscular volume; ALT, Alanine aminotransferase; RDW, Red cell distribution width; SOFA, Sequential Organ Failure Assessment; LOS ICU, Length of ICU stay; WBC, White blood cell count; ALBI, Albumin-bilirubin grade; RBC, Red blood cell count.

In eICU-CRD (Table 2), the median age was 67.00 (57.00, 77.00) years. Males accounted for 62.24% (2,682/4,309). Consistent with the trends observed in MIMIC-IV, heart rate progressively accelerated from Q1 to Q4 with rising ALBI score (median: 79, 80, 85, 88 beats/min; $P < 0.001$), whereas SBP and DBP progressively declined ($P < 0.001$). Concerning comorbidities, the prevalence of diabetes, HF, chronic obstructive pulmonary disease, atrial fibrillation, and sepsis elevated markedly with rising ALBI score ($P < 0.001$). In terms of therapeutic interventions, the utilization rates of anticoagulants, antiplatelet agents, beta-blockers, and statins decreased with increasing ALBI score ($P < 0.001$), while the rates of vasoactive agent use and mechanical ventilation increased ($P < 0.001$). Laboratory findings indicated that the concentrations of albumin declined from 4.10 (4.00, 4.30) g/dL in Q1 to 2.70 (2.40, 2.90) g/dL in Q4 ($P < 0.001$), and BUN, creatinine, RDW, and WBC count all rose in parallel with the ALBI score ($P < 0.001$). Regarding clinical outcomes, the respective incidence of AKI across Q1 to Q4 was 53.70%,

63.72%, 73.07%, and 74.56% ($P < 0.001$). Respective IHM rates were 3.51%, 6.84%, 10.40%, and 21.37% ($P < 0.001$). With the ALBI score escalating, the LOS ICU was substantially prolonged ($P < 0.001$).

Table 2
Baseline characteristics of subjects with AMI by quartiles of the ALBI score in eICU-CRD

Variables	All (N = 4,309)	Q1 (N = 1,054)	Q2 (N = 1,097)	Q3 (N = 1,077)	Q4 (N = 1,081)	P-value[1]
Demographics						
Sex, n(%)						< 0.001
Female	1,627(37.76%)	350(33.21%)	401(36.55%)	416(38.63%)	460(42.55%)	
Male	2,682(62.24%)	704(66.79%)	696(63.45%)	661(61.37%)	621(57.45%)	
Race, n(%)						0.082
Others	997(23.14%)	231(21.92%)	257(23.43%)	231(21.45%)	278(25.72%)	
White	3,312(76.86%)	823(78.08%)	840(76.57%)	846(78.55%)	803(74.28%)	
Age, years	67(57,77)	61(53,71)	66(57,76)	70(60,80)	71(61,80)	< 0.001
Vital signs						
HR(beats/min)	83 (71,97)	79 (69,92)	80 (70,95)	85 (73,98)	88 (75,102)	< 0.001
SBP(mmHg)	127 (109,147)	132(116,152)	130 (112,150)	126(109,145)	118(101,139)	< 0.001
DBP(mmHg)	73 (61,86)	78 (67,91)	74 (63,87)	71(60,84)	67(55,81)	< 0.001
RR(breaths/min)	19 (16,23)	18.00(15.00,21.00)	19(16,23)	20(16,24)	21(17,25)	< 0.001
Comorbidity						
Diabetes, n(%)	708(16.43%)	103(9.77%)	177(16.13%)	198(18.38%)	230(21.28%)	< 0.001
AF, n(%)	456(10.58%)	56(5.31%)	102(9.30%)	132(12.26%)	166(15.36%)	< 0.001
COPD, n(%)	319(7.40%)	53(5.03%)	70(6.38%)	88(8.17%)	108(9.99%)	< 0.001
CAS, n(%)	184(4.27%)	37(3.51%)	45(4.10%)	48(4.46%)	54(5.00%)	0.384
HF, n(%)	742(17.22%)	105(9.96%)	166(15.13%)	210(19.50%)	261(24.14%)	< 0.001
Hypertension, n(%)	728(16.89%)	165(15.65%)	210(19.14%)	190(17.64%)	163(15.08%)	0.045
Rheumatic diseases, n(%)	16(0.37%)	1(0.09%)	5(0.46%)	6(0.56%)	4(0.37%)	0.305
Sepsis, n(%)	519(12.04%)	48(4.55%)	81(7.38%)	130(12.07%)	260(24.05%)	< 0.001
Treatment						
Anticoagulant, n(%)	1,934(44.88%)	519(49.24%)	508(46.31%)	482(44.75%)	425(39.32%)	< 0.001
Antiplatelet, n(%)	2,731(63.38%)	760(72.11%)	744(67.82%)	686(63.70%)	541(50.05%)	< 0.001
Beta-blocker, n(%)	2,193(50.89%)	620(58.82%)	614(55.97%)	545(50.60%)	414(38.30%)	< 0.001

Abbreviations: HF, Heart failure; DBP, Diastolic blood pressure; RR, Respiratory rate; AF, Atrial fibrillation; SBP, Systolic blood pressure; CAS, Coronary atherosclerosis; HR, Heart rate; RAS drugs, Renin–angiotensin system (RAS) drugs; RDW, Red cell distribution width; AST, aspartate aminotransferase; WBC, White blood cell count; BUN, Blood urea nitrogen; ALT, alanine aminotransferase; MCV, Mean corpuscular volume; AKI, Acute kidney injury; LOS ICU, Length of ICU stay; SOFA, Sequential Organ Failure Assessment; ALBI, albumin-bilirubin grade; RBC, red blood cell count.

Variables	All (N = 4,309)	Q1 (N = 1,054)	Q2 (N = 1,097)	Q3 (N = 1,077)	Q4 (N = 1,081)	P-value[1]
Demographics						
Loop diuretics, n(%)	1,434(33.28%)	279(26.47%)	355(32.36%)	416(38.63%)	384(35.52%)	< 0.001
RAS drugs, n(%)	911(21.14%)	319(30.27%)	269(24.52%)	201(18.66%)	122(11.29%)	< 0.001
Statins, n(%)	1,624(37.69%)	497(47.15%)	445(40.57%)	407(37.79%)	275(25.44%)	< 0.001
Vasoactive drugs, n(%)	727(16.87%)	112(10.63%)	162(14.77%)	207(19.22%)	246(22.76%)	< 0.001
Ventilation, n(%)	1,576(36.57%)	259(24.57%)	365(33.27%)	412(38.25%)	540(49.95%)	< 0.001
Lab test results						
Albumin (g/dL)	3.5(3.1,3.9)	4.1(4.0,4.3)	3.7(3.5,3.8)	3.3(3.2,3.5)	2.7(2.4,2.9)	< 0.001
ALT (U/mL)	30(20,49)	29(20,42)	29(19,45)	30(20,49)	33(21,67)	< 0.001
Anion gap (mmol/L)	12(9,15)	13(10,15)	12(9,15)	12(9,15)	12(9,15)	< 0.001
AST (U/mL)	39(24,91)	31(22,55)	34(22,79)	42(25,106)	55(30,139)	< 0.001
Bicarbonate (mmol/L)	24(21,27)	25(22,27)	24(22,27)	24(21,26)	24(20,27)	< 0.001
BUN (mg/dL)	20(15,31)	17(13,22)	18(14,25)	21(15,34)	27(18,43)	< 0.001
Calcium (mg/dL)	8.9(8.5,9.4)	9.3(9.0,9.7)	9.0(8.7,9.4)	8.8(8.4,9.2)	8.4(7.9,8.9)	< 0.001
Chloride (mmol/L)	103(99,106)	102(100,105)	103(100,106)	103(99,106)	102(98,107)	0.025
Creatinine (mg/dL)	1.1(0.9,1.6)	1.0(0.9,1.3)	1.1(0.9,1.4)	1.2(0.9,1.7)	1.4(1.0,2.4)	< 0.001
Hemoglobin (g/dL)	13.2(11.4,14.8)	14.5(13.2,15.6)	13.6(12.0,14.9)	12.8(11.0,14.4)	11.6(9.9,13.4)	< 0.001
Hematocrit (%)	39.7(34.5,43.6)	42.8(39.2,45.9)	40.7(36.3,44.0)	38.4(33.9,42.6)	35.1(30.5,40.3)	< 0.001
MCV (fL)	90(87,94)	90(87,93)	90(87,94)	91(87,95)	91(86,94)	< 0.001
Potassium (mmol/L)	4.1(3.7,4.5)	4.0(3.7,4.3)	4.0(3.7,4.4)	4.1(3.7,4.5)	4.2(3.7,4.7)	< 0.001
RBC (10^{12}/mL)	4.4(3.8,4.9)	4.8(4.4,5.1)	4.5(4.0,4.9)	4.3(3.7,4.8)	3.9(3.4,4.5)	< 0.001
RDW (%)	14.00(13.20,15.20)	13.50(13.00,14.40)	13.80(13.20,14.80)	14.10(13.30,15.20)	14.90(13.80,16.50)	< 0.001
Sodium (mmol/L)	138(135,140)	138(136,141)	138(135,140)	137(135,140)	137(133,140)	< 0.001
Total bilirubin (mg/dL)	0.6(0.4,0.8)	0.5(0.3,0.7)	0.5(0.4,0.7)	0.6(0.4,0.9)	0.7(0.5,1.1)	< 0.001

Abbreviations: HF, Heart failure; DBP, Diastolic blood pressure; RR, Respiratory rate; AF, Atrial fibrillation; SBP, Systolic blood pressure; CAS, Coronary atherosclerosis; HR, Heart rate; RAS drugs, Renin–angiotensin system (RAS) drugs; RDW, Red cell distribution width; AST, aspartate aminotransferase; WBC, White blood cell count; BUN, Blood urea nitrogen; ALT, alanine aminotransferase; MCV, Mean corpuscular volume; AKI, Acute kidney injury; LOS ICU, Length of ICU stay; SOFA, Sequential Organ Failure Assessment; ALBI, albumin-bilirubin grade; RBC, red blood cell count.

Variables	All (N = 4,309)	Q1 (N = 1,054)	Q2 (N = 1,097)	Q3 (N = 1,077)	Q4 (N = 1,081)	P-value[1]
Demographics						
WBC (10^9/mL)	11.0(8.4,14.5)	10.7(8.4,13.6)	10.7(8.4,13.9)	10.9(8.2,14.6)	11.8(8.7,16.2)	< 0.001
Illness severity score						
SOFA	5(3,8)	4(2,5)	4(3,7)	5(3,8)	7(5,10)	< 0.001
Clinical outcomes						
LOS ICU (day)	53 (37,95)	43 (32,68)	50(35,87)	57(38,98)	74(44,139)	< 0.001
In-hospital mortality (%)	455(10.56%)	37(3.51%)	75(6.84%)	112(10.40%)	231(21.37%)	< 0.001
AKI, n(%)	2,858(66.33%)	566(53.70%)	699(63.72%)	787(73.07%)	806(74.56%)	< 0.001
ALBI	-2.3(-2.7,-1.9)	-2.9(-3.0,-2.8)	-2.5(-2.6,-2.4)	-2.2(-2.3,-2.1)	-1.6(-1.8,-1.3)	< 0.001

[1] Pearson's Chi-squared test; Kruskal-Wallis rank sum test

Abbreviations: HF, Heart failure; DBP, Diastolic blood pressure; RR, Respiratory rate; AF, Atrial fibrillation; SBP, Systolic blood pressure; CAS, Coronary atherosclerosis; HR, Heart rate; RAS drugs, Renin–angiotensin system (RAS) drugs; RDW, Red cell distribution width; AST, aspartate aminotransferase; WBC, White blood cell count; BUN, Blood urea nitrogen; ALT, alanine aminotransferase; MCV, Mean corpuscular volume; AKI, Acute kidney injury; LOS ICU, Length of ICU stay; SOFA, Sequential Organ Failure Assessment; ALBI, albumin-bilirubin grade; RBC, red blood cell count.

3.2 Associations of the ALBI Score with Clinical Outcomes in Subjects with AMI

3.2.1 Association of the ALBI Score with AKI

The ALBI score, treated as a continuous variable, exhibited a significant positive link to the risk of AKI in Model 1 (OR [95% CI]: 1.817 [1.472–2.254], $P < 0.001$). This association remained stable after adjusting for age, sex, and race (Model 2) (1.834 [1.485–2.278], $P < 0.001$). After further adjustment for all confounders (Model 3), the score remained significantly related to AKI (1.989 [1.504–2.644], $P < 0.001$) (Table 3). When this score was analyzed as a categorical variable grouped by quartiles (with Q1 serving as the reference), Model 3 yielded ORs for Q2, Q3, and Q4 of 1.269 (95% CI: 0.907–1.780, $P = 0.166$), 1.611 (1.109–2.349, $P = 0.013$), and 2.617 (1.678–4.117, $P < 0.001$), respectively, revealing a pronounced dose–response pattern (P for trend < 0.001).

Table 3
Association of ALBI score with AKI by multivariable logistic regression in MIMIC-IV

	Model 1			Model 2			Model 3		
AKI									
Characteristics	OR	95% CI	P	OR	95% CI	P	OR	95% CI	P
ALBI score	1.817	1.472, 2.254	< 0.001	1.834	1.485, 2.278	< 0.001	1.989	1.504, 2.644	< 0.001
ALBI type									
Q1	Ref.	Ref.		Ref.	Ref.		Ref.	Ref.	
Q2	1.288	0.950, 1.752	0.104	1.287	0.947, 1.752	0.108	1.269	0.907, 1.780	0.166
Q3	1.678	1.221, 2.316	0.002	1.687	1.225, 2.333	0.001	1.611	1.109, 2.349	0.013
Q4	2.421	1.718, 3.450	< 0.001	2.446	1.733, 3.489	< 0.001	2.617	1.678, 4.117	< 0.001

CI: Confidence Interval, OR: Odds Ratio

Model 1: Crude.

Model 2: Comprising adjustments for sex, race, and age.

Model 3: Encompassing adjustments for HR, sex, SBP, race, DBP, age, RR, diabetes, hypertension, anticoagulant, beta-blocker, antiplatelet, loop diuretics, anion gap, statins, BUN, vasoactive drugs, bicarbonate, calcium, chloride, creatinine, hemoglobin, MCV, potassium, RDW, WBC.

Abbreviations: RR, Respiratory rate; WBC, White blood cell count; DBP, Diastolic blood pressure; BUN, Blood urea nitrogen; RDW, Red cell distribution width; HR, Heart rate; Ref., Reference; SBP, Systolic blood pressure; ALBI, albumin-bilirubin grade

The respective ORs for the ALBI score in Models 1, 2, and 3 were 1.930 (95% CI: 1.712–2.179, $P < 0.001$), 1.788 (1.583–2.024, $P < 0.001$), and 1.464 (1.249–1.719, $P < 0.001$) in the independent external validation cohort from eICU-CRD. These results consistently indicated that an increased ALBI score was significantly associated with an elevated risk of AKI (Table 4). Upon grouping by quartiles, the respective ORs for Q2, Q3, and Q4 were 1.308 (1.084–1.580, $P = 0.005$), 1.714 (1.383–2.110, $P < 0.001$), and 1.620 (1.278–2.054, $P < 0.001$) in Model 3. Consistent effect directions and comparable effect magnitudes were noted in both databases, suggesting favorable robustness of the findings.

Table 4
Association of ALBI score with AKI by multivariable logistic regression in eICU-CRD

	Model 1			Model 2			Model 3		
AKI									
Characteristics	OR	95% CI	P	OR	95% CI	P	OR	95% CI	P
ALBI score	1.930	1.712, 2.179	< 0.001	1.788	1.583, 2.024	< 0.001	1.464	1.249, 1.719	< 0.001
ALBI type									
Q1	Ref.	Ref.		Ref.	Ref.		Ref.	Ref.	
Q2	1.514	1.274, 1.800	< 0.001	1.420	1.192, 1.692	< 0.001	1.308	1.084, 1.580	0.005
Q3	2.340	1.953, 2.807	< 0.001	2.111	1.755, 2.542	< 0.001	1.714	1.393, 2.110	< 0.001
Q4	2.527	2.106, 3.036	< 0.001	2.266	1.879, 2.736	< 0.001	1.620	1.278, 2.054	< 0.001

CI: Confidence Interval, OR: Odds Ratio

Model 1: Crude.

Model 2: Encompassing adjustments for sex, race, and age.

Model 3: Comprising adjustments for HR, sex, RR, SBP, diabetes, DBP, race, hypertension, age, anticoagulant, beta-blocker, antiplatelet, loop diuretics, anion gap, statins, vasoactive drugs, BUN, calcium, creatinine, bicarbonate, chloride, hemoglobin, MCV, potassium, RDW, WBC.

Abbreviations: RR, Respiratory rate; WBC, White blood cell count; DBP, Diastolic blood pressure; RDW, Red cell distribution width; Ref., Reference; SBP, Systolic blood pressure; BUN, Blood urea nitrogen; HR, Heart rate; ALBI, Albumin-bilirubin grade

3.2.2 Association of the ALBI Score with IHM

In MIMIC-IV, the respective ORs for IHM in Models 1, 2, and 3 were 2.633 (95% CI: 2.180–3.190, $P < 0.001$), 2.822 (2.316–3.451, $P < 0.001$), and 2.304 (1.800–2.957, $P < 0.001$; Table 5). Following stratification by quartiles, compared with Q1, the respective ORs for IHM in Q2, Q3, and Q4 were 1.101 (0.731–1.662, $P = 0.646$), 1.363 (0.914–2.046, $P = 0.132$), and 3.169 (2.101–4.827, $P < 0.001$; Table 5).

Table 5
Association of ALBI score with IHM by multivariable logistic regression in MIMIC-IV

	Model 1			Model 2			Model 3		
In-hospital mortality									
Characteristics	OR	95% CI	P	OR	95% CI	P	OR	95% CI	P
ALBI score	2.633	2.180, 3.190	< 0.001	2.822	2.316, 3.451	< 0.001	2.304	1.800, 2.957	< 0.001
ALBI type									
Q1	Ref.	Ref.		Ref.	Ref.		Ref.	Ref.	
Q2	1.334	0.903, 1.981	0.15	1.265	0.852, 1.888	0.245	1.101	0.731, 1.662	0.646
Q3	1.971	1.368, 2.868	< 0.001	1.868	1.291, 2.731	0.001	1.363	0.914, 2.046	0.132
Q4	4.397	3.136, 6.260	< 0.001	4.552	3.229, 6.514	< 0.001	3.169	2.101, 4.827	< 0.001

CI: Confidence Interval, OR: Odds Ratio

Model 1: Crude.

Model 2: Comprising adjustments for sex, race, and age.

Model 3: Encompassing adjustments for HR, race, SBP, anion gap, DBP, age, RR, sex, BUN, diabetes, hypertension, anticoagulant, beta-blocker, antiplatelet, loop diuretics, statins, vasoactive drugs, bicarbonate, calcium, chloride, creatinine, hemoglobin, MCV, potassium, RDW, WBC.

Abbreviations: RR, Respiratory rate; WBC, White blood cell count; DBP, Diastolic blood pressure; RDW, Red cell distribution width; Ref., Reference; SBP, Systolic blood pressure; BUN, Blood urea nitrogen; HR, Heart rate; ALBI, Albumin-bilirubin grade

In eICU-CRD, the respective ORs for IHM in Models 1, 2, and 3 were 3.773 (95% CI: 3.188–4.476, $P < 0.001$), 3.600 (3.019–4.303, $P < 0.001$), and 2.637 (2.154–3.233, $P < 0.001$; Table 6). After stratification by quartiles, the respective ORs for Q2, Q3, and Q4 were 1.607 (1.096–2.388, $P = 0.017$), 1.890 (1.302–2.788, $P = 0.001$), and 3.525 (2.428–5.205, $P < 0.001$).

Table 6
Association of ALBI score with IHM by multivariable logistic regression in eICU-CRD

	Model 1			Model 2			Model 3		
In-hospital mortality									
Characteristics	OR	95% CI	P	OR	95% CI	P	OR	95% CI	P
ALBI score	3.773	3.188, 4.476	< 0.001	3.600	3.019, 4.303	< 0.001	2.637	2.154, 3.233	< 0.001
ALBI type									
Q1	Ref.	Ref.		Ref.	Ref.		Ref.	Ref.	
Q2	2.017	1.357, 3.049	< 0.001	1.745	1.174, 2.635	0.007	1.607	1.096, 2.388	0.017
Q3	3.19	2.199, 4.732	< 0.001	2.512	1.726, 3.731	< 0.001	1.89	1.302, 2.788	0.001
Q4	7.47	5.283, 10.85	< 0.001	5.873	4.141, 8.548	< 0.001	3.525	2.428, 5.205	< 0.001

CI: Confidence Interval, OR: Odds Ratio

Model 1: Crude.

Model 2: Encompassing adjustments for sex, race, and age.

Model 3: Comprising adjustments for HR, SBP, race, DBP, anion gap, RR, diabetes, age, hypertension, sex, anticoagulant, beta-blocker, bicarbonate, antiplatelet, loop diuretics, statins, vasoactive drugs, BUN, calcium, chloride, creatinine, hemoglobin, MCV, potassium, RDW, WBC.

Abbreviations: RR, Respiratory rate; WBC, White blood cell count; HR, Heart rate; DBP, Diastolic blood pressure; RDW, Red cell distribution width; BUN, Blood urea nitrogen; Ref., Reference; SBP, Systolic blood pressure; ALBI, albumin-bilirubin grade

3.3 Nonlinear Relationship of the ALBI Score with Clinical Outcomes

The dose–response relationships of the ALBI score, analyzed as a continuous variable, with AKI and IHM were examined using RCS regression. This score exhibited a linear positive association with AKI in both MIMIC-IV (*P* for overall < 0.001, *P* for nonlinear = 0.773) and eICU-CRD (*P* for overall < 0.001, *P* for nonlinear = 0.099) (Fig. <link rid="fig2">2</link>-A and 2-B).

For IHM, the ALBI score likewise exhibited a linear positive link to mortality risk in both MIMIC-IV (*P* for overall < 0.001, *P* for nonlinear = 0.074) and eICU-CRD (*P* for overall < 0.001, *P* for nonlinear = 0.521) (Fig. <link rid="fig3">3</link>-A and 3-B).

3.4 Subgroup Analyses

The stability of the associations between the ALBI score and clinical outcomes was evaluated through subgroup analyses stratified by diabetes, race, sex, hypertension, and age.

3.4.1 Association of the ALBI Score with AKI

In MIMIC-IV (Fig. 4-A), the association of the ALBI score with AKI remained significantly positive across all subgroups, and no significant interactions were detected (all *P* for interaction > 0.05). This suggested that the discriminative capacity of the ALBI score for AKI remained stable across different population subgroups.

In eICU-CRD (Fig. 4-B), the link between the ALBI score and AKI similarly remained significant across all subgroups. However, significant interaction effects emerged. The age subgroup (*P* for interaction = 0.014) revealed that the strength of the link was more pronounced among individuals < 70 years (OR [95% CI]: 1.63 [1.32–2.01]) than among those ≥ 70 years (1.29 [1.01–1.66]); the sex subgroup (*P* = 0.017) indicated a stronger link in males (1.55 [1.25–1.91]) relative to females (1.29 [1.00–1.66]); the race subgroup (*P* = 0.039) showed a more pronounced link among individuals of 'other race' (2.02 [1.26–3.29]) than among White individuals (1.33 [1.11–1.59]).

3.4.2 Association of the ALBI Score with IHM

In MIMIC-IV (Fig. 5-A), the association of the ALBI score with IHM was marked across all subgroups. The hypertension subgroup (*P* for interaction = 0.029) demonstrated a stronger link in subjects with hypertension (OR [95% CI]: 2.28 [1.64–3.21]) than in those without

hypertension (1.35 [0.69–2.66]); the diabetes subgroup (*P* = 0.022) revealed a stronger link in subjects with diabetes (2.52 [1.59–4.08]) than in those without diabetes (1.73 [1.20–2.52]).

In eICU-CRD (Fig. 5-B), the link between the ALBI score and IHM was marked across all subgroups. The hypertension subgroup (*P* for interaction = 0.039) showed a stronger link in subjects without hypertension (OR [95% CI]: 2.84 2.28–3.54) than in those with hypertension (1.60 [0.88–2.90]); the diabetes subgroup (*P* = 0.044) revealed a stronger link in subjects without diabetes (2.74 [2.18–3.44]) than in those with diabetes (2.11 [1.22–3.65]).

4 Discussion

4.1 Principal Findings

The current investigation incorporated 6,393 subjects with AMI drawn from two databases (eICU-CRD and MIMIC-IV) and systematically evaluated the associations of the ALBI score with AKI and IHM. This score, whether treated as a categorical or continuous variable, exhibited a significant positive association with AKI and IHM. The association remained consistent across the two independent databases. RCS regression further revealed linear positive relationships of the score with both AKI and IHM. These observations suggest that the ALBI score, a straightforward and low-cost metric of hepatic function, may facilitate early risk stratification in patients with AMI.

4.2 Interpretation of Results

4.2.1 Comparison with Prior Investigations

The current findings corroborate and extend the existing literature. Yildirim et al. [20] identify the ALBI score as a robust independent predictor of the no-reflow phenomenon (OR = 12.10) in individuals with non-ST-elevation acute coronary syndrome receiving PCI. This indicates that the ALBI score may reflect oxidative stress and systemic inflammation, which in turn participate in myocardial microcirculatory impairment. Among patients with end-stage HF receiving implantation of a left ventricular assist device, Niklewski et al. [21] report that each one-unit increment in the ALBI score corresponds to a 4.98-fold elevated risk of all-cause mortality (HR = 4.98), representing one of the largest effect sizes documented to date. Horio et al. [22] construct a multi-biomarker model integrating the ALBI score in individuals with acute coronary syndrome and demonstrate that this model markedly enhances the predictive capacity of the Global Registry of Acute Coronary Events score for IHM. Among patients with HF, Gao et al. [13], drawing on MIMIC, observe a nonlinear association of the ALBI score with 30-, 90-, and 360-day mortality (threshold: −1.92). Kishihara et al. [14] confirm that subjects with a discharge ALBI score ≥ − 2.25 face an 82% elevated risk of the composite endpoint. Additionally, the present investigation found that compared with Q1 (most favorable hepatic function), subjects in Q4 (most impaired hepatic function) exhibited a substantially elevated incidence of AKI and IHM rate, and these elevated risks persisted after comprehensive adjustment for confounding factors. Yang et al. [23], in a study of critically ill subjects with AKI, similarly report that a high grade of ALBI is markedly linked to mortality and demonstrates a linear relationship in RCS analysis, consistent with the present findings. Building upon the aforementioned evidence, this investigation focused specifically on a population with AMI and simultaneously evaluated the associations of the ALBI score with both AKI and IHM. Thus, it may offer new reference data for the application of the ALBI score in the cardiovascular domain.

4.2.2 Exploration of Potential Mechanisms

The potential mechanisms underlying the link between an elevated ALBI score (i.e., worsening hepatic function) and adverse outcomes in patients with AMI may involve multiple dimensions. Several speculative interpretations are proposed below on the basis of the existing literature. First, hypoalbuminemia, a critical component of the ALBI score, reflects the nutritional status and systemic inflammatory burden of the patient [24]. An investigation by Yang et al. [25] employing MIMIC-III demonstrates that early concentration of albumin serves as an independent predictor of AKI occurring within 48 h following PCI in patients with AMI. Hypoalbuminemia contributes to AKI possibly by diminishing osmotic pressure of plasma colloids leading to renal interstitial edema, reducing renal perfusion, and compromising albumin-mediated antioxidant and anti-inflammatory protective functions [26]. Second, hyperbilirubinemia represents another integral component of the ALBI score. Research by Vítek et al. [27] indicates that markedly elevated levels of bilirubin may signify impaired hepatic function or cholestasis. Cholestasis can facilitate endotoxemia and systemic inflammatory responses via the gut-kidney axis, thereby precipitating or aggravating renal injury. Furthermore, the liver and kidneys engage in intimate 'liver-kidney crosstalk' across multiple dimensions, including hemodynamic regulation, metabolic clearance, and inflammatory responses [28]. Under critical illness, multi-organ interactions may further amplify inflammatory cascades, oxidative stress, and microcirculatory dysfunction, thereby facilitating the development of AKI [29]. Against the pathological backdrop of AMI, myocardial ischemia-reperfusion injury can trigger systemic inflammatory response syndrome, further exacerbating hepatic and renal dysfunction and establishing a vicious 'cardio-hepatic-renal axis' [30]. Notably, the mechanisms outlined above rest primarily on inferences drawn from the available literature. The current investigation did not directly validate these pathways. Additional basic research and prospective cohort investigations are required to substantiate them.

4.3 Interpretation of Subgroup Analysis Findings

Based on subgroup analyses, the association of the ALBI score with AKI remained stable across most subgroups, suggesting that the discriminative capacity of this score may exhibit favorable robustness across diverse populations. Notably, within eICU-CRD, age, sex, and race exerted significant interaction effects on the link between the ALBI score and AKI. The link was more pronounced in males, subjects aged < 70 years, and those of 'other race'. The underlying reasons for this phenomenon remain unclear. Speculatively, younger patients may possess greater compensatory capacity for hepatic impairment, such that once hepatic function deteriorates, it signals a more severe pathological state, while racial differences may be attributable to genetic background, lifestyle, or healthcare accessibility. In MIMIC-IV, hypertension and diabetes exhibited marked interaction effects on the association of the ALBI score with IHM [31]. Conversely, within eICU-CRD, the association of this score with IHM was more pronounced among subjects without hypertension and without diabetes. These subgroup discrepancies between the two databases may be attributable to differences in population characteristics, such as illness severity and comorbidity burden. These subgroup findings should be regarded as exploratory and await confirmation through additional external investigations.

4.4 Clinical Implications

The clinical relevance resides in the fact that the ALBI score, a straightforward indicator sourced from routine laboratory tests (SA and TBIL), requires no additional expenditure or specialized equipment. The score can be conveniently integrated into the admission assessment workflow for patients with AMI. This may help identify individuals at heightened risk for AKI and IHM early, thereby guiding clinicians to implement closer renal function monitoring and preventive interventions [32]. Prior investigations have indicated that early recognition and management of AMI-associated AKI hold considerable significance for reducing IHM rate and improving long-term renal outcomes [33]. Relative to the traditional Child-Pugh score, the ALBI score requires only two objective indicators—TBIL and albumin—avoiding the bias inherent in subjective judgment and offering superior reproducibility and clinical operability [10].

4.5 Strengths and Limitations

Several strengths should be noted. First, the current research harnessed two independent critical care databases (eICU-CRD and MIMIC-IV), representing single-center and multicenter patient populations, respectively. The findings were consistent across both databases, reinforcing their credibility and generalizability. Employing MIMIC-IV (single-center) as the primary analytic cohort and eICU-CRD (multicenter) as the sensitivity analytic cohort, this dual-center validation effectively mitigated the risk of center-specific bias that a single database might introduce. Second, multiple statistical approaches—including multivariable logistic regression, RCS regression, and subgroup analyses—were applied to systematically evaluate the associations of the ALBI score with clinical outcomes along with potential effect modifiers. Third, the ALBI score is computed from routine laboratory parameters, making it clinically actionable. Additionally, the dose–response analyses clarified the linear relationships of the ALBI score with the outcomes, providing a reference for establishing clinical thresholds.

Nonetheless, several limitations should be acknowledged. First, this retrospective cohort investigation cannot exclude the influence of residual confounding. Although numerous known confounders were accounted for, unmeasured confounding variables may persist. Third, the ALBI score was derived exclusively from the first measurement in the initial 24 h after ICU admission and could not capture dynamic changes during hospitalization, which may provide additional prognostic information. Fourth, the patient populations of the two databases differ to some extent, likely attributable to variations in the types of hospitals and geographic regions covered. Fifth, this investigation included only AMI subjects with a first ICU admission. The applicability of the results to patients with AMI hospitalized in non-ICU settings requires further verification. Finally, although the cohort from eICU-CRD was used for external validation, both databases originate from critical care populations in the United States with a predominantly White racial composition. The generalizability of the findings to Asian, African, and non-ICU hospitalized populations should be further validated.

5 Conclusion

Utilizing data from 6,393 subjects with AMI across MIMIC-IV and eICU-CRD, the current investigation identified independent, linear positive associations of the ALBI score with AKI and IHM among patients with AMI, a finding that remained consistent across the two databases. These results indicate that the score, a straightforward and low-cost indicator of hepatic function, may be effectively applied to early risk stratification in patients with AMI. This may offer clinicians a new reference for identifying individuals at elevated risk of AKI and IHM and for implementing targeted preventive measures. Future prospective investigations are necessary to verify these conclusions and to examine the prognostic utility of dynamic changes in the ALBI score.

Declarations

Clinical trial number

Not applicable.

Ethics approval and consent to participate

The investigators acquired the Collaborative Institutional Training Initiative after completing training in human subjects protection and passing the required examination. Both databases received approval from their respective institutional ethical review boards. Since all information about subjects was anonymized, informed consent was not required in this investigation.

Consent for publication

Not applicable.

Declaration of interests

The authors declare that they have no competing interests.

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Data statement

The datasets used and analysed during the current study are available from the corresponding author on reasonable request.

Declaration of Artificial Intelligence

The author declare that no artificial intelligence was used at the time of writing the paper.

Author Contributions

All authors contributed to the study conception and design. **Writing - original draft preparation:** [Xiezhen Chen, Wenqing Zhou]**; Writing - review and editing:** [Weiye Cao]**; Conceptualization:** [Weiye Cao]**; Methodology:** [Weiye Cao]**; Formal analysis and investigation:** [Xiezhen Chen, Wenqing Zhou]**; Resources:** [Weiye Cao]**; Supervision:** [Hongwei Wu], and all authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

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Not applicable.

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Figures

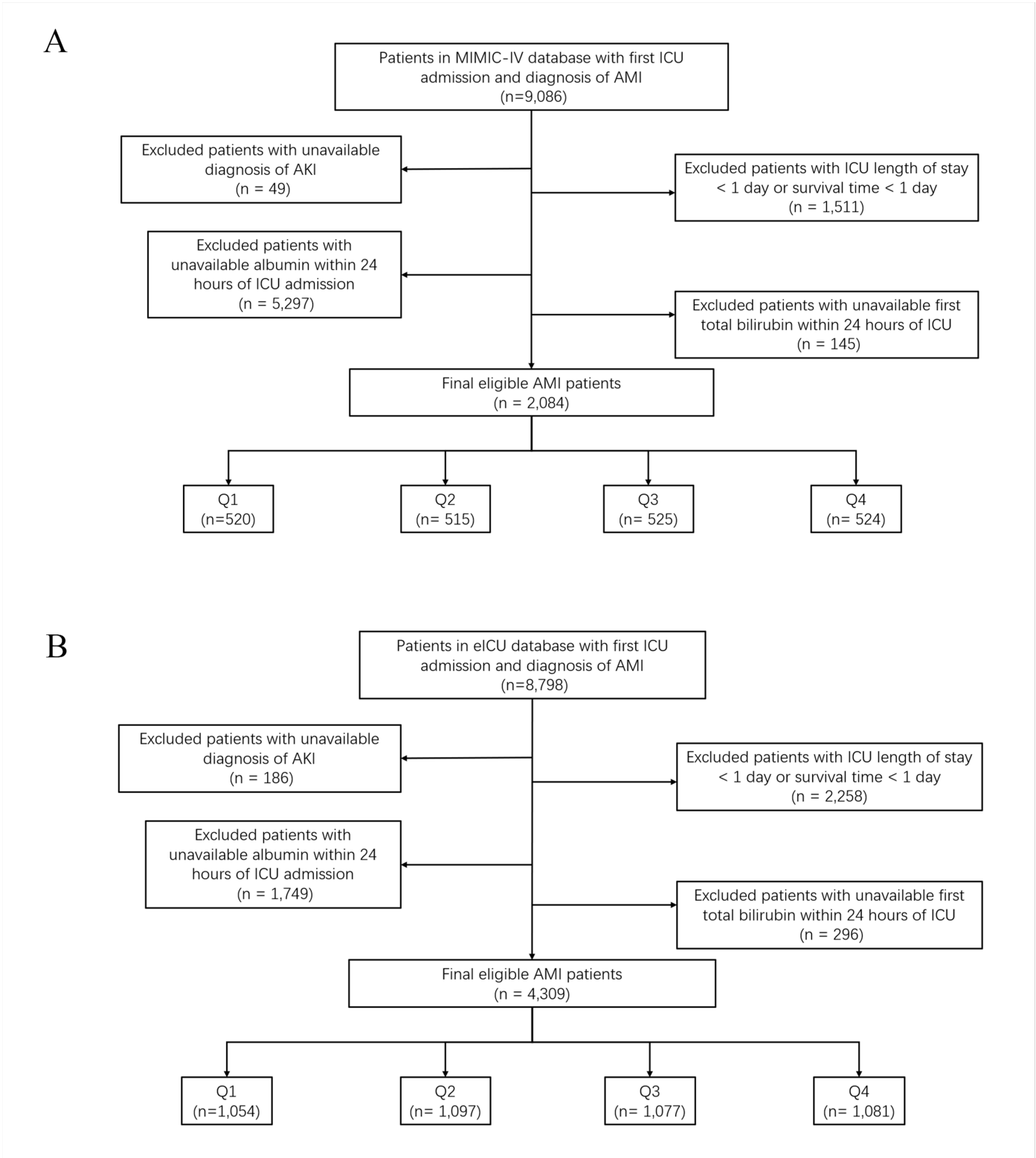


Figure 1

(A). Patient selection flowchart for MIMIC-IV;

(B) Patient selection flowchart for eICU-CRD

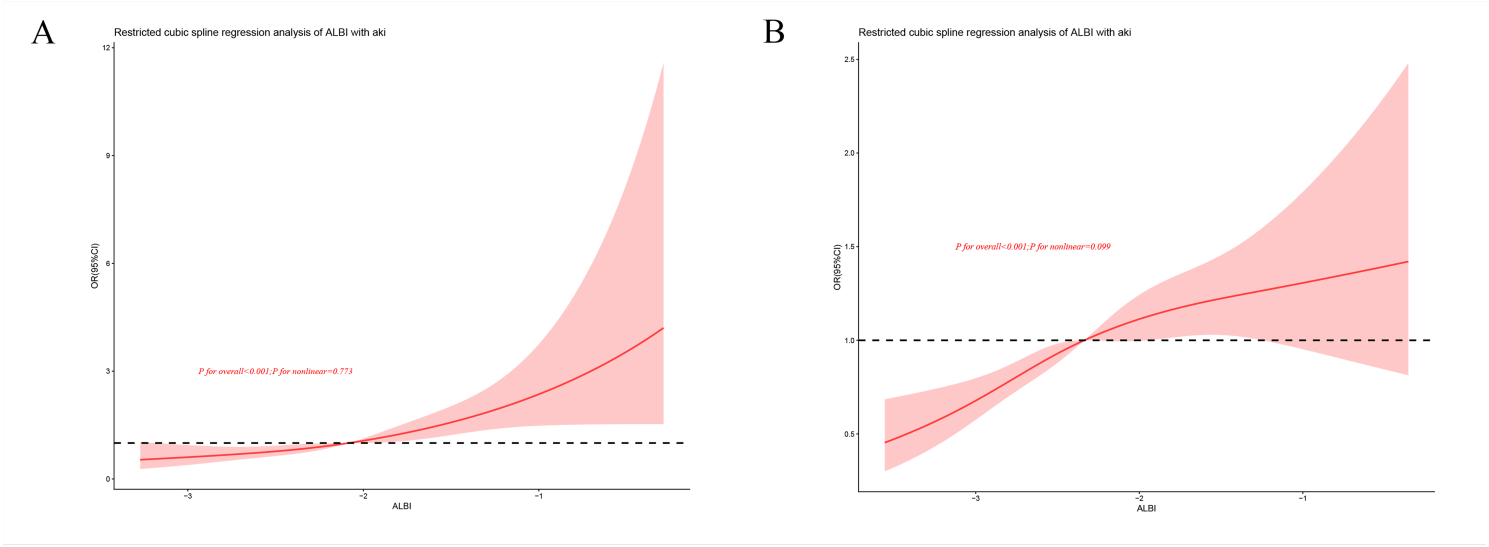


Figure 2

(A). Association of ALBI score with AKI by RCS regression in MIMIC-IV;

(B). Association of ALBI score with AKI by RCS regression in eICU-CRD

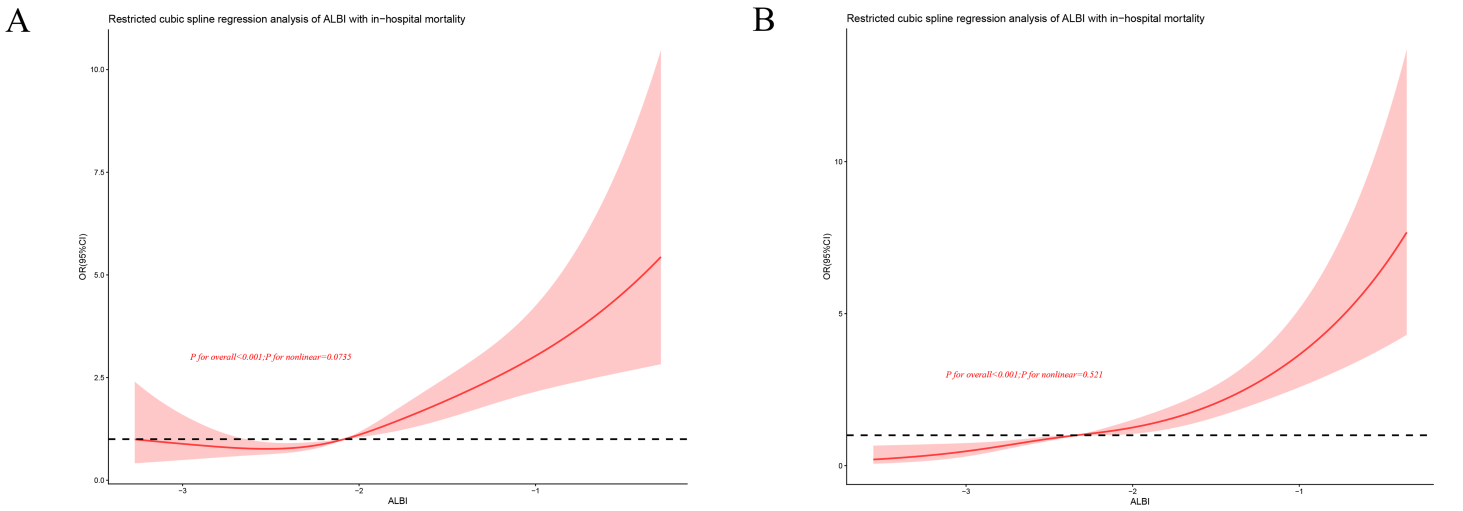


Figure 3

(A). Association of ALBI score with IHM by RCS regression in MIMIC-IV; **(B)**. Association of ALBI score with IHM by RCS regression in eICU-CRD

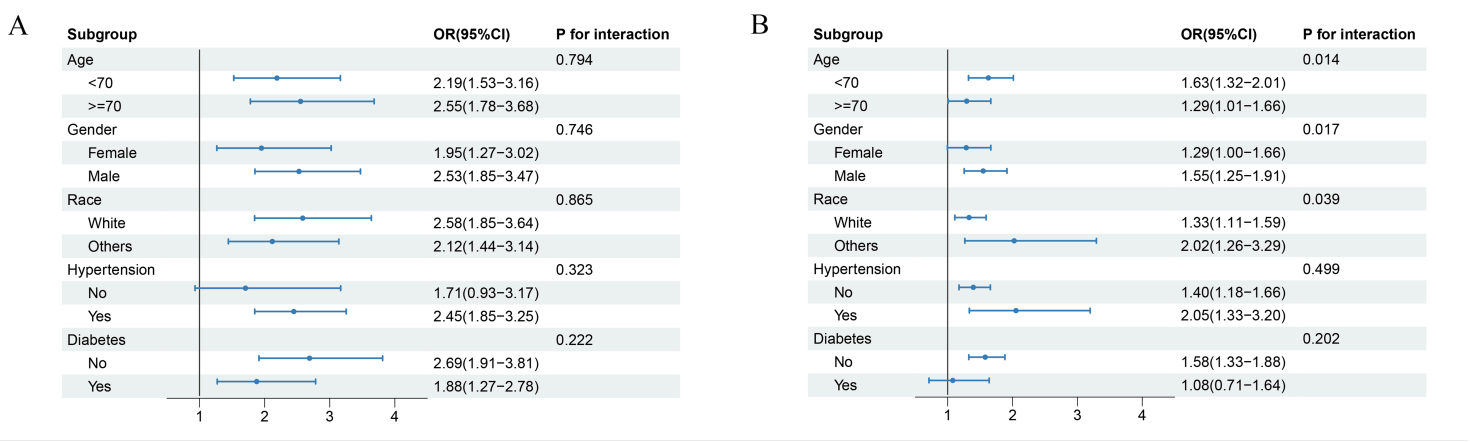


Figure 4

(A). Forest plot of subgroup analyses for ALBI score and AKI in MIMIC-IV; **(B)**. Forest plot of subgroup analyses for ALBI and AKI in eICU-CRD

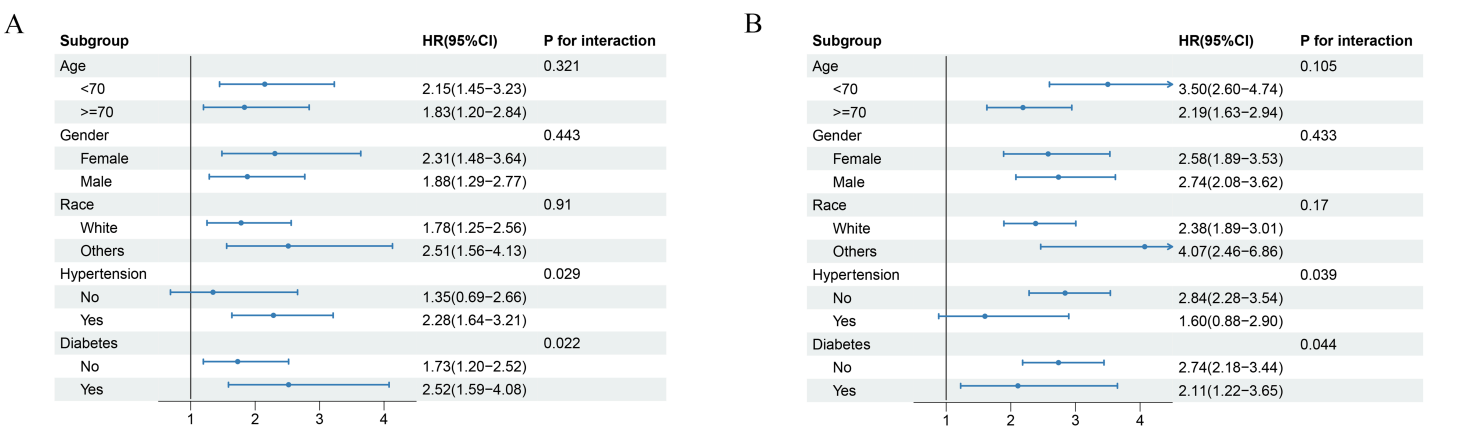


Figure 5

(A). Forest plot of subgroup analyses for ALBI score and IHM in MIMIC-IV; **(B)**. Forest plot of subgroup analyses for ALBI and IHM in eICU-CRD

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