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Glucose-to-albumin ratio predicts short-term mortality in critically ill patients with acute pancreatitis

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The Glucose-to-Albumin Ratio (GAR) has been identified as a significant predictor among sepsis and nonalcoholic fatty liver disease. However, its utility in predicting all-cause mortality among patients with acute pancreatitis (AP) has not been studied. Consequently, this study aimed to evaluate the predictive value of GAR for 28-day mortality among AP patients. This retrospective cohort study used data from the Medical Information Mart for Intensive Care (MIMIC-IV) database. According to the median GAR value, participants were divided into two cohorts. The primary endpoint was all-cause mortality within 28 days. Kaplan–Meier survival analysis and the log-rank test were employed to compare survival between the two cohorts. Cox proportional hazards models were then applied to estimate the hazard ratio of GAR, with and without adjustment for other significant clinical factors. Subgroup analyses were conducted to evaluate the relationship between GAR and mortality within strata of other significant factors. Finally, receiver operating characteristic (ROC) curves based on a logistic regression model were constructed to assess the predictive capability of GAR using the area under the curve (AUC), which was then compared with other predictors. A total of 459 patients with AP were included in the study. Kaplan–Meier curves and the log-rank test demonstrated that patients with a high GAR (> 48.85) had significantly higher 28-day all-cause mortality than those with a low GAR ($p = 0.026$). Cox proportional hazards models further indicated that high binary GAR was associated with an increased unadjusted hazard ratio (HR = 2.006, $p = 0.029$). When treated as a continuous variable, higher GAR remained significantly associated with an elevated hazard ratio after adjusting for age, gender, race, and BMI (HR = 1.010, $p = 0.007$). This association persisted in the final adjusted model derived through backward selection, where higher GAR continued to predict higher 28-day mortality risk (HR = 1.010, $p = 0.008$) after controlling for age, gender, race, BMI, and prothrombin time (PT). Subgroup analyses revealed no significant interaction between binary GAR and other covariates in predicting 28-day mortality. Compared with glucose, albumin, the Systemic Inflammatory Response Syndrome score, and the Glasgow Coma Scale score, GAR demonstrated superior predictive power, with a higher AUC (0.602) for predicting 28-day mortality. Our study originally identified that elevated GAR was correlated with increased 28-day all-cause mortality among AP patients, and GAR demonstrated strong predictive value for short-term mortality risk.

Keywords Glucose-to-albumin ratio, Acute pancreatitis, All-cause mortality, MIMIC-IV, A cohort study

Acute pancreatitis (AP) is one of the leading causes of hospital admission, and its incidence is on the rise, now reaching approximately 34 cases per 100,000 people each year[1,2]. The two leading etiological factors for acute pancreatitis are gallstone disease and alcohol. Other causes include hypertriglyceridemia, hypercalcemia, familial pancreatitis, viral infections, endoscopic retrograde cholangiopancreatography (ERCP) and various medications. These factors initiate pathological cellular pathways and lead to organelle dysfunction, ultimately resulting in the hallmark features of acute pancreatitis, including acinar cell death and local and systemic inflammation[3]. The clinical course varies by severity. About 80% of patients experience mild to moderately severe disease and could typically be managed by supportive care and intravenous fluid administration[4]. In contrast, roughly 20% progress to severe disease characterized by pancreatic or peripancreatic necrosis and, in some cases, multi-organ failure, with a mortality rate of approximately 20%. Because of this wide variability in

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outcomes, AP remains a challenging condition, and identifying accurate biomarkers for early risk stratification is of significant clinical importance.

Among the biomarkers studied, glucose and albumin are key indicators of metabolic and inflammatory status in AP. Elevated blood glucose levels and dysregulated glucose metabolism have been correlated with the severity and clinical outcomes of AP patients[5–7]. High blood glucose level serves as an independent risk factor for predicting prognosis in AP patients[8]. However, glucose levels are affected by several elements, including nutritional status, hepatic dysfunction, and medication use, limiting its reliability when used alone.

Albumin, a negative acute-phase protein, also serves an important role in modulating inflammatory responses[9,10]. Synthesized in the liver, it contributes to antioxidative and anti-inflammatory defense. In severe inflammatory states, albumin levels often decline due to reduced hepatic synthesis and redistribution caused by increased vascular permeability. Hypoalbuminemia has been associated with poorer outcomes in patients with AP[11,12]. Nonetheless, serum albumin levels are also affected by chronic illnesses, inflammation, and malnutrition, reducing the prognostic value of a single measurement.

To address the limitations of using either marker alone, the glucose-to-albumin ratio (GAR) has emerged as a novel biomarker that integrates both metabolic and inflammatory information. GAR has shown predictive value in diseases including non-alcoholic fatty liver disease, sepsis, and intracerebral hemorrhage[13–16]. However, its role in forecasting prognosis among AP patients has not yet been elucidated. Consequently, in the present study, we aim to assess the predictive value of GAR for 28-day mortality in patients with AP utilizing data from the Medical Information Mart for Intensive Care IV (MIMIC-IV) version 3.1 database.

Results

Baseline characteristics

A total of 459 ICU patients with AP were ultimately included in the study (Fig. 1). Their demographic and clinical characteristics are detailed in Table 1. The median age was 56.6 years, with 211 females (46.0%) and 248 males (54.0%). Most patients were White ($n = 285$, 62.09%).

Participants were categorized into two groups according to the median GAR value: a low GAR group (≤ 48.85) and a high GAR group (> 48.85). Compared with those in the low GAR group, patients in the high GAR group showed significantly elevated weight, BMI, and Systemic Inflammatory Response Syndrome (SIRS) scores. The GCS score is not significantly different between two GAR groups. They also had higher rates of vasopressin use, invasive ventilation, continuous renal replacement therapy (CRRT), and renal replacement therapy (RRT) (all $p < 0.05$). When addressing comorbidities, acute kidney injury (AKI) and diabetes were significantly more common among patients with high GAR ($p < 0.05$).

Laboratory data also showed notable differences between the groups. Those with high GAR showed significantly elevated levels of white blood cell count (WBC), red blood cell count (RBC), platelets (Plt), blood urea nitrogen (BUN), and serum potassium, along with lower levels of serum calcium and alkaline phosphatase (ALP) (all $p < 0.05$).

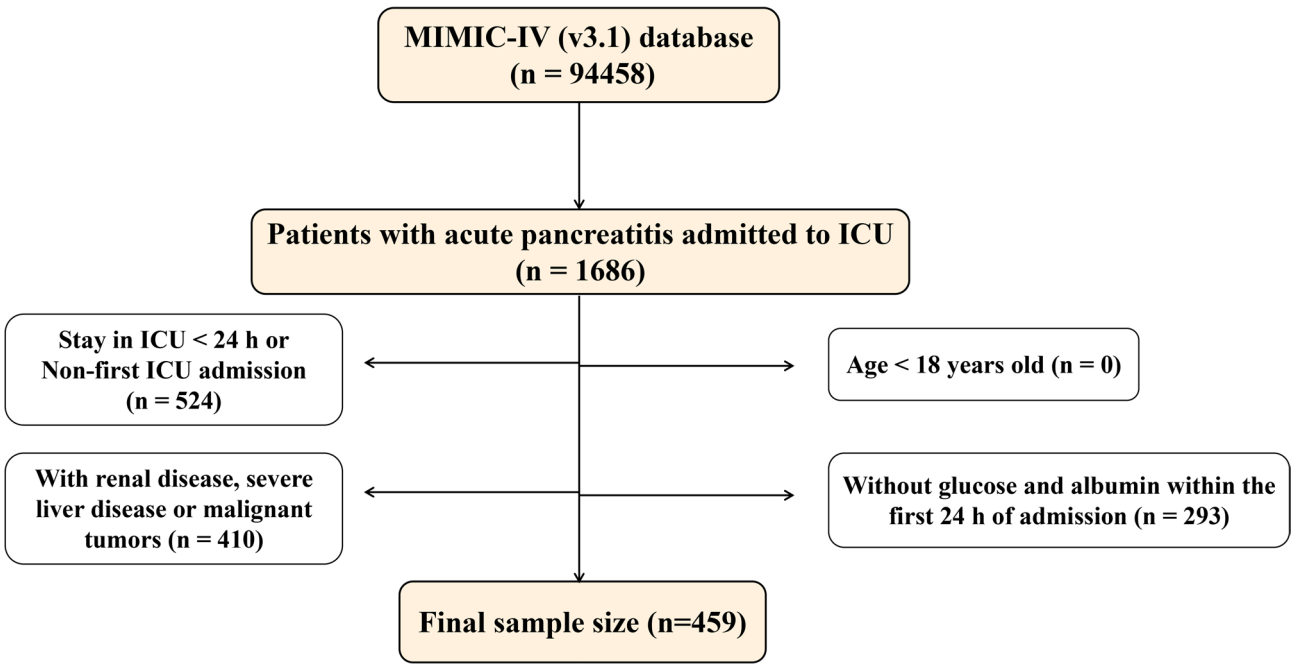

Fig. 1. Flowchart for participants from the MIMIC-IV (v 3.1). AP, acute pancreatitis; MIMIC-IV, Medical Information Mart for Intensive Care IV; ICU, intensive care unit.

Characteristic	Total ($N=459$)[1]	GAR		p-value[2]
		≤48.85 ($N=230$)[1]	>48.85 ($N=229$)[1]	
Demographics				
Age (year)	56.6 (17.5)	56.9 (19.0)	56.3 (15.9)	0.715
Gender				0.280
Female	211 (46.0%)	112 (48.7%)	99 (43.2%)	
Male	248 (54.0%)	118 (51.3%)	130 (56.8%)	
Race				0.286
Black	30 (6.54%)	13 (5.65%)	17 (7.42%)	
White	285 (62.09%)	148 (64.35%)	137 (59.83%)	
Other	60 (13.07%)	24 (10.43%)	36 (15.72%)	
Unknown	84 (18.30%)	45 (19.57%)	39 (17.03%)	
Height (cm)	169.0 (10.1)	168.0 (10.5)	169.8 (9.7)	0.156
Weight (kg)	87.2 (23.4)	82.6 (22.4)	91.7 (23.6)	**<0.001**
BMI	31.3 (7.7)	29.7 (7.0)	32.7 (8.0)	**0.001**
GCS score	12.0 (3.7)	12.2 (3.5)	11.8 (3.9)	0.185
SIRS score	3.1 (0.8)	3.0 (0.8)	3.2 (0.7)	**0.008**
Clinical treatments				
Octreotide	34 (7.4%)	16 (7.0%)	18 (7.9%)	0.848
Vasopressin	94 (20.5%)	32 (13.9%)	62 (27.1%)	**<0.001**
InvasiveVent	230 (50.1%)	100 (43.5%)	130 (56.8%)	**0.006**
CRRT	49 (10.7%)	15 (6.5%)	34 (14.8%)	**0.006**
RRT	63 (13.7%)	22 (9.6%)	41 (17.9%)	**0.014**
Acute and chronic complications				
AKI	340 (74.1%)	156 (67.8%)	184 (80.3%)	**0.003**
Sepsis	293 (63.8%)	141 (61.3%)	152 (66.4%)	0.301
Myocardial infarct	40 (8.7%)	21 (9.1%)	19 (8.3%)	0.88
Congestive heart failure	66 (14.4%)	33 (14.3%)	33 (14.4%)	>0.999
Mild liver disease	109 (23.7%)	56 (24.3%)	53 (23.1%)	0.847
Chronic pulmonary disease	89 (19.4%)	53 (23.0%)	36 (15.7%)	0.062
Cerebrovascular disease	28 (6.1%)	17 (7.4%)	11 (4.8%)	0.335
Hypertension	265 (57.7%)	131 (57.0%)	134 (58.5%)	0.808
Diabetes	140 (30.5%)	38 (16.5%)	102 (44.5%)	**<0.001**
Laboratory parameters				
WBC (10^9 /mL)	15.0 (8.2)	14.0 (8.5)	16.0 (7.8)	**0.010**
RBC (10^{12}/mL)	3.9 (0.9)	3.8 (0.8)	4.0 (1.0)	**0.015**
Plt (10^9 /mL)	239.8 (130.6)	227.0 (124.5)	252.7 (135.5)	**0.035**
Hb (g/dL)	11.7 (2.6)	11.6 (2.4)	11.9 (2.8)	0.160
Hct (%) (hematocrit)	35.7 (7.6)	35.0 (7.0)	36.3 (8.2)	0.055
PT (s)	16.1 (8.5)	15.9 (6.9)	16.2 (9.9)	0.707
Cr (mg/dL)	1.4 (1.4)	1.3 (1.5)	1.5 (1.4)	0.245
BUN (mg/dL)	24.1 (20.1)	21.9 (19.8)	26.3 (20.1)	**0.018**
Calcium (mg/dL)	7.8 (1.2)	8.0 (1.2)	7.7 (1.2)	**0.008**
Potassium (mmol/L)	4.2 (0.9)	4.1 (0.8)	4.3 (1.0)	**0.050**
Sodium (mmol/L)	138.2 (6.3)	138.3 (5.6)	138.0 (7.0)	0.633
ALP (U/mL)	138.3 (133.1)	153.1 (152.4)	124.0 (109.6)	**0.021**
ALT (U/mL)	184.9 (665.1)	229.3 (860.8)	141.4 (383.5)	0.167
AST (U/mL)	304.9 (1,324.6)	364.4 (1,488.8)	247.1 (1,142.7)	0.352
TB (mg/dL)	2.0 (3.4)	2.2 (3.7)	1.7 (3.0)	0.120
RDW	14.9 (2.0)	14.8 (1.9)	14.9 (2.2)	0.681
Anion gap	15.8 (5.4)	15.6 (5.2)	16.0 (5.5)	0.376
Continued				

Characteristic	Total ($N=459$)[1]	GAR		p-value[2]
		≤48.85 ($N=230$)[1]	>48.85 ($N=229$)[1]	
LD (IU/L))	636.0 (1,174.1)	587.7 (1,151.0)	688.9 (1,200.4)	0.446
Clinical outcomes				
LOS Hospital (day)	20.0 (20.8)	18.1 (20.3)	21.9 (21.1)	**0.047**
LOS ICU (day)	7.6 (10.1)	6.7 (10.1)	8.5 (10.0)	0.069
28-day mortality	44 (9.6%)	15 (6.5%)	29 (12.7%)	**0.038**

Table 1. Baseline characteristics in patients with acute pancreatitis. [1] Mean (SD); n (%) [2] Welch Two Sample t-test; Pearson's Chi-squared test. BMI, Body Mass Index; GCS, Glasgow Coma Scale; SIRS, Systemic Inflammatory Response Syndrome; CRRT, continuous renal replacement treatment; RRT, Renal Replacement Therapy; AKI, acute kidney injury; WBC, white blood cell count; RBC, red blood cell count; Plt, platelet; Hb, hemoglobin; Hct, hematocrit; PT, prothrombin time; Cr, creatinine; BUN, blood urea nitrogen; ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; TB, total bilirubin; RDW, red blood cell distribution width; LD, lactate dehydrogenase; LOS Hospital, length of hospital stay; LOS ICU, length of ICU stay. Significant values are in bold.

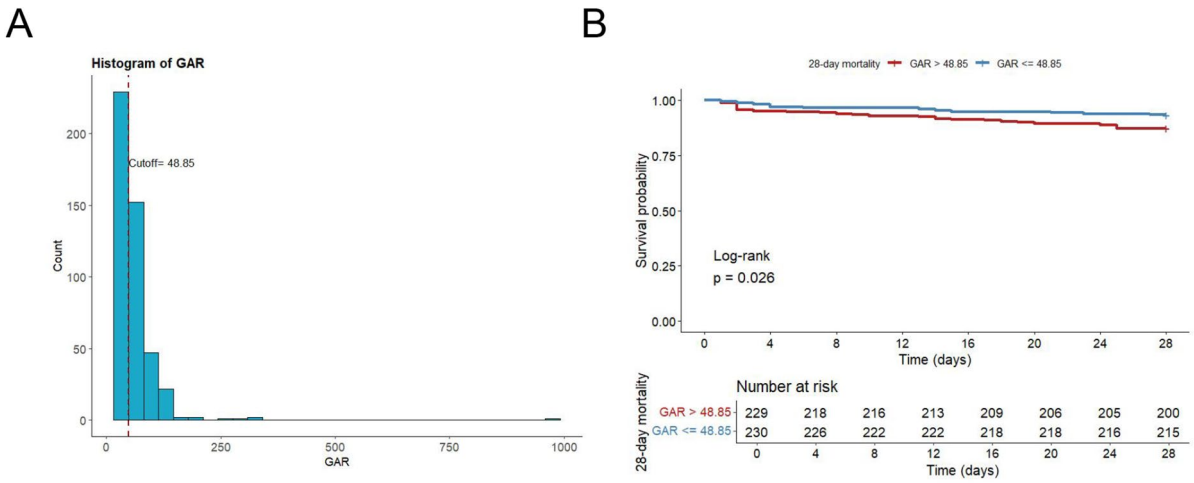

Fig. 2. The cutoff point and distribution of GAR (**A**) and Kaplan-Meier survival analysis curves illustrating all-cause mortality among AP patients at 28-d (**B**) of hospital admission.

These differences corresponded with worse clinical outcomes. Patients in the high GAR group experienced longer hospital stays (21.9 vs. 18.1 days, $p=0.047$), marginally longer ICU stays (8.5 vs. 6.7 days, $p=0.069$), and elevated 28-day mortality (12.7% vs. 6.5%, $p=0.038$) compared with those in the low GAR group.

Kaplan-Meier survival analysis

The primary outcome was all-cause mortality within 28 days. Kaplan–Meier survival curves and the log-rank test were employed to compare outcomes between two groups stratified by the median GAR value (Fig. 2). Patients with a high GAR (>48.85) had significantly higher 28-day mortality and poorer short-term survival than those having a low GAR (≤48.85) (log-rank $p=0.026$). To examine the impact of commonly studied biomarkers—including glucose, albumin, SIRS, and GCS—patients were dichotomized into high vs. low groups using their median values. KM survival curves with log-rank tests (Supplementary Fig. 1) demonstrated no significant differences in 28-day mortality for glucose (log-rank $p=0.737$), SIRS (log-rank $p=0.791$), and GCS (log-rank $p=0.067$). In contrast, albumin showed a significant association: patients with low albumin (≤2.9 g/dL) had higher 28-day mortality and poorer short-term survival compared to those with higher albumin levels (>2.9 g/dL) (log-rank $p=0.038$).

The hazard ratios of GAR on short-term survival

Cox proportional hazards models were applied to evaluate the association between GAR and short-term survival, defined as 28-day all-cause mortality, both prior to and following adjustment for pertinent clinical factors (Table 2). In the unadjusted model, when GAR was treated as a binary variable, those in the high GAR group exhibited significantly poorer short-term survival, with an increased risk of 28-day mortality (HR 2.006; 95% CI 1.075–3.741; $p=0.029$). When GAR was evaluated as a continuous variable in Model 1, adjusting for age, gender, race, and BMI, it remained significantly correlated with an elevated risk of 28-day mortality (HR 1.010; 95% CI: 1.003–1.017; $p=0.007$). Furthermore, in the final adjusted Model 2—derived through backward selection

GAR	Unadjusted		Model 1		Model 2	
	HR (95% CI)	*p*-value	HR (95% CI)	*p*-value	HR (95% CI)	*p*-value
Continuous	1.002 (0.999, 1.005)	0.144	1.010 (1.003, 1.017)	**0.007**	1.010 (1.002, 1.017)	**0.008**
Binary						
Ratio < = 48.85	Ref		Ref		Ref	
Ratio > 48.85	2.006 (1.075, 3.741)	**0.029**	2.018 (0.904, 4.506)	0.087	2.649 (0.934, 7.513)	0.067

Table 2. Cox proportional hazard (PH) regression models of GAR with mortality among AP patients. Significant values are in bold. Model 1: adjustment for age, gender, race and BMI. Model 2: 28-day mortality for continuous GAR: adjusted for age, gender, race, BMI, and PT. 28-day mortality for binary GAR: adjusted for age, gender, race, BMI, and LD.

and including only significant covariates not correlated with GAR—this association persisted. Continuous GAR continued to demonstrate a significant raised short-term mortality risk (HR = 1.010; 95% CI: 1.002–1.017; $p = 0.008$) after controlling for age, gender, race, BMI, and PT.

In addition, we evaluated the risk effects of other commonly studied biomarkers—glucose, albumin, SIRS, and GCS—using multivariable Cox regression models. After adjusting for major confounders (age, race, gender, and BMI), higher glucose levels were significantly associated with an increased risk of 28-day mortality (HR = 1.003; 95% CI: 1.001–1.006; $p = 0.019$). On the contrary, for albumin, higher levels were consistently associated with lower mortality risk in 28 days in both continuous (HR = 0.565; 95% CI: 0.343–0.928; $p = 0.024$) and binary formats (HR = 0.517; 95% CI: 0.274–0.975; $p = 0.042$) in the unadjusted model, in agreement with the KM analysis results. After adjustment for confounders, this protective effect remained statistically significant for binary albumin (HR = 0.439; 95% CI: 0.193–1.000; $p = 0.050$). Among the remaining biomarkers, only continuous GCS demonstrated a protective association in the unadjusted model, with higher scores associated with decreased short-term mortality risk (HR = 0.931; 95% CI: 0.869–0.999; $p = 0.046$). However, this association was no longer significant after controlling for confounders.

The effect of GAR on LOS at hospital and ICU

In univariate linear regression, higher GAR was associated with longer hospital stay (Coefficient = 3.85; SE = 1.93; $p = 0.047$). However, after adjusting for age, race, gender, and BMI, this association was no longer statistically significant. GAR was not significantly associated with ICU stay in either univariate or multivariable models (Supplementary Table 2).

Subgroup analysis

To evaluate whether the correlation between GAR and 28-day all-cause mortality varied across patient characteristics, subgroup analyses were performed based on essential clinical and demographic factors. Stratified analyses were performed according to age, gender, race, BMI, acute kidney injury (AKI), sepsis, myocardial infarction, congestive heart failure, chronic pulmonary disease, hypertension, and diabetes. Across subgroups stratified by same factor, the hazard ratios for GAR did not differ significantly, indicating no meaningful interactions between GAR and any of these factors. This consistency suggests that the correlation between GAR and short-term mortality is stable across diverse patient profiles, further supporting the robustness of our findings (Fig. 3).

Receiver operating characteristic (ROC) analysis for all-cause mortality

We generated ROC curves for five biomarkers—GAR, glucose, albumin, SIRS, and GCS—to assess their capability in predicting 28-day all-cause mortality among AP patients. As shown in Table 3; Fig. 4, GAR demonstrated the highest AUC (0.602; 95% CI: 0.508–0.696), outperforming glucose (0.542; 95% CI: 0.450–0.634), albumin (0.601; 95% CI: 0.502–0.699), SIRS (0.554; 95% CI: 0.482–0.626), and GCS (0.565; 95% CI: 0.467–0.662). These findings indicate that GAR has comparatively better predictive performance. Overall, GAR showed strong diagnostic ability for 28-day mortality and is beneficial in forecasting short-term survival.

Discussion

The present study shows that GAR serve as a significant predictor of short-term survival among patients with AP. Kaplan–Meier survival curves, log-rank tests, and Cox regression analyses consistently indicated that patients with a GAR > 48.85 showed elevated 28-day all-cause mortality than patients with a GAR ≤ 48.85, both before and after adjusting for potential confounders. Subgroup analyses further reinforced these findings: the hazard ratio for GAR and 28-day mortality was consistent across subgroups defined by demographic and clinical characteristics, with no evidence of interaction between GAR and other risk factors. Additionally, ROC analyses demonstrated that GAR had better predictive performance than conventional indicators such as glucose, albumin, SIRS score, and GCS, as reflected in its higher AUC. Collectively, these results support GAR as a reliable and clinically valuable marker for early risk stratification in patients with AP.

Recently, a variety of serum biomarkers have been explored for predicting outcomes in patients with AP, such as the C-reactive protein-to-albumin ratio (CAR)[17,18], the red blood cell distribution width-to-albumin ratio (RAR)[19], the total bilirubin-to-albumin ratio (TBAR)[20] and the albumin corrected anion gap (ACAG)[21]. The GAR as also emerged as a novel prognostic marker in diseases including sepsis, nonalcoholic fatty liver disease

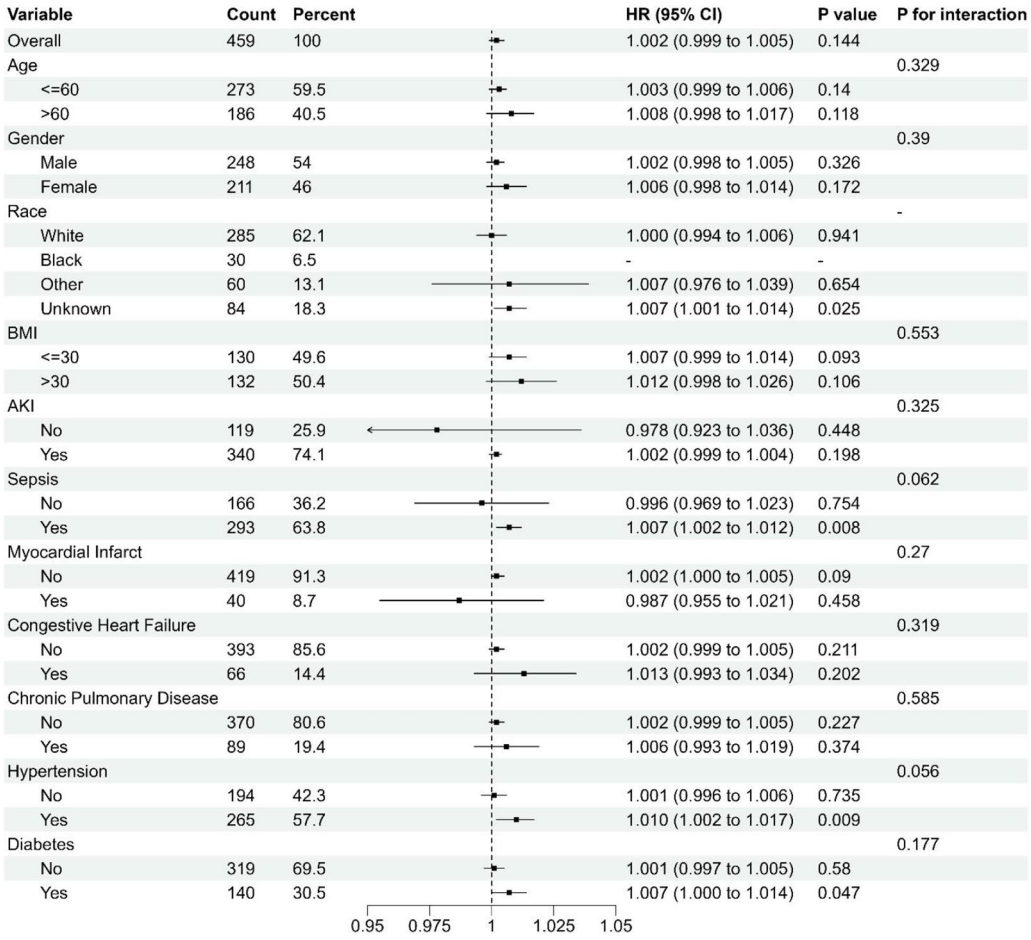

Fig. 3. Forest plots illustrating subgroup analysis of the association between all-cause mortality and GAR among AP patients at 28-d of hospital admission.

Variables	AUC	95%CI
28-day mortality		
GAR	0.602	0.508–0.696
Glucose	0.542	0.450–0.634
Albumin	0.601	0.502–0.699
SIRS	0.554	0.482–0.626
GCS	0.565	0.467–0.662

Table 3. The results of ROC analysis.

(NAFLD), and spontaneous intracerebral hemorrhage, where it has demonstrated good predictive accuracy[13–16]. However, its prognostic utility in AP has not previously been examined.

Glucose dysregulation is common in AP, affecting nearly 40% of patients, and has been demonstrated to associate with both disease severity and clinical outcomes[5–7]. Studies have reported that both hyperglycemia and hypoglycemia are associated with prolonged hospitalization[22]. Elevated glucose levels often indicate more severe acute pancreatitis[8,23,24]. Hyperglycemia in AP may result from reduced insulin production due to pancreatic parenchymal damage. Consistent with prior findings, our study also identified elevated blood glucose as a risk factor for adverse outcomes. However, glucose levels are affected by a wide range of factors—including stress, hepatic dysfunction, dietary habits, and nutritional status—which limits the reliability of glucose alone as a prognostic marker.

Serum albumin, the predominant plasma protein synthesized by the liver, has also been linked to AP severity and prognosis[11,12,25]. During inflammation, albumin contributes to produce anti-inflammatory mediators, including lipoxins and protectins, which increases its consumption. In addition, systemic inflammation increases vascular permeability, promoting albumin extravasation and further reducing serum levels[10]. Despite its clinical importance, albumin is influenced by chronic disease, malnutrition, and inflammation, limiting the predictive

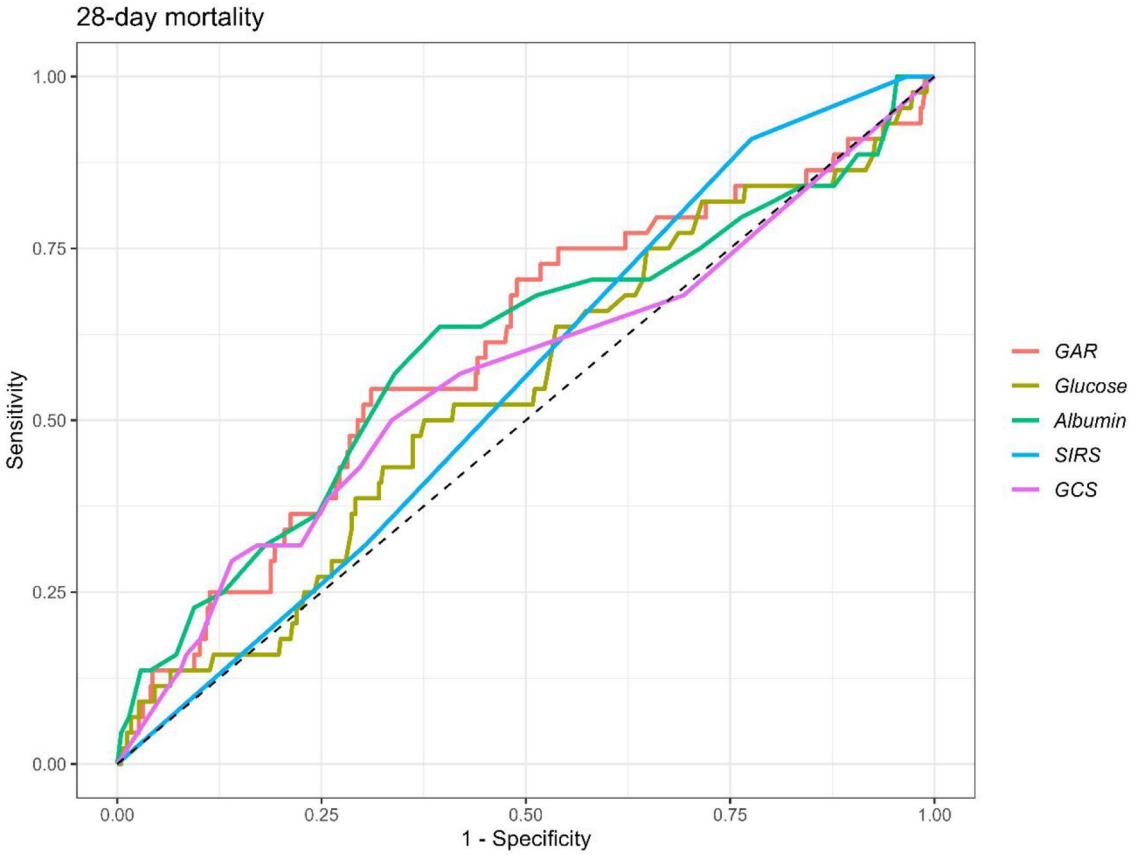

Fig. 4. ROC curves for predicting all-cause mortality among AP patients at 28-d of hospital admission.

value of a single measurement. Therefore, combining glucose and albumin—as achieved with GAR—may reduce the impact of individual confounders and enhance prognostic accuracy.

Prior research has demonstrated the prognostic significance of GAR across various clinical contexts, including sepsis, NAFLD, spontaneous intracerebral hemorrhage, postoperative delirium in geriatric hip fracture patients, and pancreatic neuroendocrine neoplasms (pNENs)[13–16,26,27]. For example, higher GAR has been associated with increased disease progression in NAFLD[13] and elevated mortality in ICU patients with sepsis, where integrating GAR into clinical models improved risk prediction and clinical decision-making[14,15]. Similarly, GAR may serve as a potential prognostic marker in those suffering from intracerebral hemorrhage[16]. In our study, we identified a similar relationship between GAR and prognosis, suggesting that GAR may be a promising biomarker for early risk stratification and management in AP patients.

A key strength of the present study lies in its utilization of a large, real-world critical care database, which enabled the evaluation of GAR across diverse clinical presentations and enhanced the generalizability of the findings. Nonetheless, some limitations need to be considered. First, the study was designed as single-center retrospective, which might limit external validity and introduce potential selection and information biases; thus, prospective multicenter investigations are required to validate the results. Second, only the initial GAR value after ICU admission was analyzed. Whether dynamic changes in GAR over time offer greater prognostic value remains unclear and should be explored in future research. Third, the information were sourced from the MIMIC-IV database, spanning 2008 to 2022—a period during which treatment strategies for AP have evolved. Variations in clinical practice over time may have influenced patient outcomes. Taken together, these limitations highlight the need for further validation using contemporary datasets and rigorously designed prospective studies that account for temporal trends and repeated GAR measurements.

Conclusion

In the present study, we originally identified that a higher GAR was significantly associated with elevated 28-day all-cause mortality in patients with acute pancreatitis. GAR emerged as an independent predictor of short-term survival and demonstrated better prognostic performance than blood glucose, serum albumin, SIRS, or GCS alone. Its strong predictive value suggests that GAR may serve as a practical tool for early risk stratification, helping clinicians initiate timely interventions and optimize treatment planning. Future prospective studies are warranted to confirm these findings and further clarify the relationship between GAR and prognosis in AP.

Materials and methods

Data source

The current research utilized information from the MIMIC-IV (v 3.1) database, a large, openly available database that was developed and maintained by the Massachusetts Institute of Technology's Laboratory of Computational Physiology[28]. This database encompasses information of all patients who were hospitalized in the Beth Israel Deaconess Medical Center (BIDMC) from 2008 to 2022. To safeguard confidentiality of patients, all personal identifiers have been eliminated and substituted with randomized codes. As a result, informed consent and institutional ethical approval were not required for the present study. Members of the research team completed the required training facilitated by the Collaborative Institutional Training Initiative (CITI), including the "Conflict of Interest" and "Data or Specimens Only Research" modules (Certification ID: 70421177), and were granted full access to the database.

Population selection criteria

According to the International Classification of Diseases, Revision 9 (ICD-9) code 577.0 and International Classification of Diseases, Revision 10 (ICD-10) code K85–K85.92, hospital admission data for patients with AP were retrieved from the database. Participants were excluded if they satisfied any of the following criteria: (1) the individual was under 18 years of age at the initial admission; (2) multiple ICU admissions for AP, in which case only the first admission was included; (3) ICU stay shorter than 24 h; (4) presence of renal disease, severe liver disease, or malignant tumors; or (5) missing blood glucose or albumin measurements within 24 h of admission. After the application of these criteria, 459 patients were incorporated into the final analysis (Fig. 1).

Data extraction

GAR was selected as the principal variable of interest. To minimize treatment-related influence, blood glucose and albumin levels acquired from the initial laboratory measurements conducted upon admission.

All study variables were obtained from the database utilizing Structured Query Language (SQL). Potential confounders were classified into the following primary domains: demographic characteristics, clinical treatments, comorbidities, laboratory indicators, and clinical outcomes.

Endpoint events

The primary outcome was all-cause mortality at 28 days. Secondary outcomes included ICU length of stay and total hospital stay.

Statistical analysis

Summary statistics were first used to describe all variables collected in the study. Based on the median GAR value in the final selected cohort of 459 patients, participants were categorized into two groups: high GAR and low GAR. Continuous variables were reported as mean ± standard deviation (SD) and the comparison between two groups was conducted utilizing the Welch two-sample t-test. Categorical variables were presented as counts and percentages (%) and compared utilizing Pearson's chi-square test or Fisher's exact test, as appropriate.

The Kaplan–Meier (KM) method was then applied to estimate short-term survival, defined as 28-day all-cause mortality, and survival curves were plotted for each GAR group. Differences in survival between the groups were assessed utilizing the log-rank test. To identify additional potential risk factors for 28-day mortality beyond GAR, all other variables were first evaluated utilizing univariate Cox proportional hazards models. Variables with a p-value < 0.05 were subsequently included in a multivariate Cox regression model. Backward elimination, with a significance threshold of 0.05, was applied to select the most relevant predictors while retaining age, gender, race and BMI as forced covariates. Each significant predictor from this process was further assessed for correlation with GAR. Only those not strongly correlated with GAR were included in the final adjusted model. In this model, the HR of GAR for 28-day mortality was estimated while adjusting for other significant risk factors.

Subgroup analyses were conducted to evaluate whether the relationship between GAR and 28-day mortality differed across patient characteristics and to assess potential interaction effects. Within each subgroup, Cox proportional hazards models were employed to estimate the HR of GAR, and interactions between GAR and each stratifying factor were tested using the Wald test. HRs across subgroups were visualized in forest plots, with corresponding p-values for interaction reported.

Finally, ROC analysis using a logistic regression model was constructed to assess the predictive performance of GAR, as measured by the AUC, and to compare it with other biomarkers and established disease severity scores, including GCS and SIRS. All statistical analyses were conducted utilizing R (version 4.4.3) with relevant packages, and all tests were two-sided with a significance level of 0.05.

Data availability

Data are available from the corresponding author upon reasonable request.

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Author contributions

Conceptualization: Xin Lin, Side Liu. Data curation: Xin Lin, Weiwei Ma, Zhengjia Chen. Formal analysis: Xin Lin, Weiwei Ma, Zhengjia Chen. Funding acquisition: Xin Lin, Side Liu. Investigation: Xin Lin, Weiwei Ma, Zhengjia Chen. Methodology: Xin Lin, Weiwei Ma, Zhengjia Chen. Writing – original draft: Xin Lin. Writing – review & editing: Xin Lin, Weiwei Ma, Zhengjia Chen, Side Liu.

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Declarations

Competing interests

The authors declare no competing interests.

Ethical approval

The ethical approval and participation consent followed the Helsinki Declaration guidelines. Massachusetts Institute of Technology and Beth Israel Deaconess Medical Center review committee approved the utilization of the MIMIC-IV database. Given that the data is publicly accessible via the MIMIC-IV database, the need for ethical approval and informed consent was waived.

Additional information

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