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Association of serum albumin-to-creatinine ratio with in-hospital mortality in patients with severe acute pancreatitis: a retrospective study

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Abstract

Background The serum albumin-to-serum creatinine ratio (sACR) is independently associated with the prognosis of multiple diseases. However, its relationship with in-hospital mortality of patients with severe acute pancreatitis (SAP) remains unclear.

Methods Patients diagnosed with SAP between April 2016 and December 2023 were collected. These patients were categorized into low and high sACR groups based on an optimal cut-off value calculated using Youden's index. Multivariate logistic regression analysis was utilized to examine the relationship between sACR levels and the in-hospital mortality. Additionally, a limited restricted cubic spline (RCS) method was employed to evaluate the nonlinear relationship between sACR values and the risk of in-hospital mortality. The potential for unmeasured confounders between sACR levels and in-hospital mortality was also explored through the calculation of the E value.

Results A total of 114 eligible patients were included in this sutdy. The multivariate logistic regression analysis indicated an independent association between sACR levels and in-hospital mortality ($p < 0.001$). The RCS analysis demonstrated a linear correlation between sACR values and the risk of in-hospital mortality (P for non-linearity > 0.05), where the risk increased as the sACR value decreased.

Conclusions The research findings suggest that sACR levels are independently associated with in-hospital mortality of patients with SAP, providing a means for early identification of those at high risk of in-hospital mortality. This early identification may facilitate the optimizing and strengthening of treatments, ultimately leading to improved outcomes.

Keywords Severe acute pancreatitis, Serum albumin-to-serum creatinine ratio, In-hospital mortality

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Background

Acute pancreatitis (AP) is a condition characterized by inflammation of the pancreas, leading to significant morbidity and mortality [1]. Research indicates that the global incidence of AP has been steadily increasing at a rate of 3.07% per year over the past 56 years [2]. While most cases of AP are mild and self-limited, a subset of patients, approximately 15-20%, progress to severe acute pancreatitis (SAP) [3, 4]. SAP is marked by the development of persistent organ failure, with a mortality rate as high as 36-50% [5, 6]. Identifying SAP patients at risk of death early on is crucial for providing intensive treatment and improving outcomes.

The excessive inflammatory response is a key factor in the pathogenesis of SAP, impacting serum albumin levels by disrupting liver protein metabolism and causing capillary leakage [7, 8]. Clinical studies have linked inflammatory factors to the development of acute kidney injury in AP patients [9, 10]. Some basic studies suggest that the infiltration of inflammatory cells and the release of pro-inflammatory factors contribute to acute kidney injury, leading to elevated serum creatinine levels [11–13]. High serum creatinine levels in AP patients have been associated with poorer clinical outcomes in previous researches [14–16]. The serum albumin-to-serum creatinine ratio (sACR), a novel inflammation-based marker, has shown promise in predicting prognosis in various conditions such as liposarcoma, myocardial infarction, heart failure, and soft tissue sarcoma [17–20]. However, no studies have examined the relationship between sACR and in-hospital mortality in SAP patients.

Methods

Data source and study population

Patients diagnosed with SAP were collected from Yijishan Hospital of Wannan Medical College between April 2016 and December 2023. The diagnostic criteria for SAP were based on the 2012 revised Atlanta Classification [6]. Exclusion criteria were as follows: (1) a history of chronic or recurrent pancreatitis; (2) admission more than 48 h after symptom onset; (3) chronic kidney disease; (4) prior malignancy; (5) incomplete data.

Data collection

The sACR is defined as the serum albumin divided by the serum creatinine [17]. The outcome in this study was in-hospital mortality. Clinical characteristics and laboratory measurements at admission were recorded, including sex, age, etiology, hypertension (no/yes), diabetes (no/yes), white blood cells count (WBC), platelets count (PLT), hemoglobin level (Hb), total bilirubin level (TBIL), albumin level (Alb), creatinine level (Cr), blood urea nitrogen level (BUN), prothrombin time (PT), calcium level (Ca), fasting blood glucose level (FBG), and Acute Pancreatitis Bedside Severity Index Score (BISAP score). Acute Physiology and Chronic Health Evaluation (APACHE) II score and Sequential Organ Failure Assessment (SOFA) score were acquired during their hospital stay in the intensive care unit (ICU). Additionally, systemic and local complications and patients' survival status during hospitalization were documented.

Statistical analysis

Continuous variables with a normal distribution were presented as mean±standard deviation, while non-normally distributed variables were shown as median with interquartile range. Categorical variables were represented as numbers and percentages. Statistical analysis of comparisons between variables included the Mann–Whitney U test, independent sample t-test, chi-square test, and Fisher's exact test [21]. The analysis of prediction accuracy for sACR was conducted using the area under the receiver operating characteristic (ROC) curve (AUC) [22]. The optimal cutoff values of sACR and Cr were identified based on Youden's index, and patients were then classified into low and high groups using this optimal cutoff values. The Delong test was utilized to assess the significance of differences in the AUC [23]. Univariate logistic analysis was employed to ascertain the associations between variables and in-hospital mortality. Variables with significant associations ($p<0.1$) were subsequently considered in the multivariate analysis. Multivariate logistic regression analysis was then conducted to determine the independent association between sACR levels and in-hospital mortality. Variance inflation factor values were used to evaluate multicollinearity, VIF>10 considered indicative of multicollinearity [24]. Restricted cubic spline (RCS) analysis was utilized to explore the nonlinear relationship between sACR values and the risk of in-hospital mortality [25]. Furthermore, an exploration into the potential presence of unmeasured confounders between sACR levels and in-hospital mortality was conducted by determining E value [26]. Statistical significance was set at $p<0.05$. Software packages SPSS (version 25.0), R (version 4.0.2), and MedCalc (Version 15.2) were used for data analysis.

Results

Baseline characteristics

This study included a total of 114 patients with SAP who met the specified criteria (Fig. 1). Among these patients, the majority were female, with an average age close to 60 years. Throughout the study, 38.5% patients died during the hospitalization. In comparison to the nonsurvivors group, the survivors group had a longer total length of hospital stay and duration of stay in the ICU. Notably, old age was more common among nonsurvivors compared to survivors. In addition, the BISAP score, APACHE II

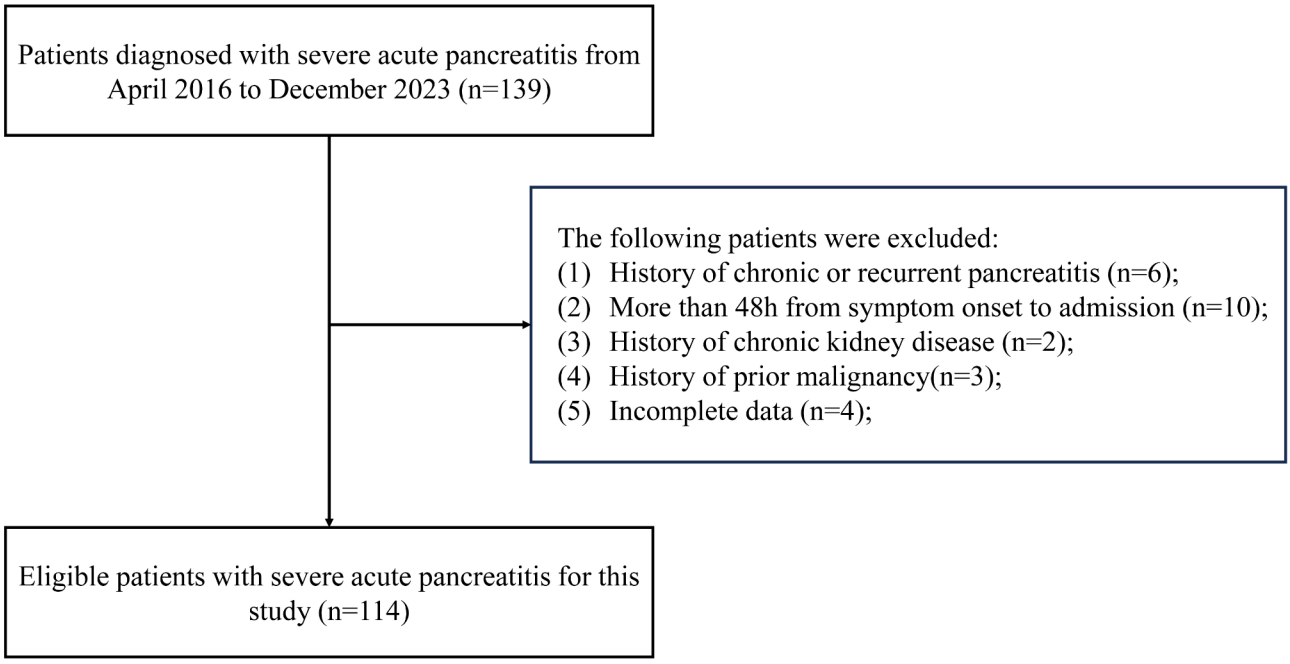

Fig. 1 Flowchart depicting the patient selection process

score, and SOFA score were significantly higher in the nonsurvivors group compared to the survivors group. Furthermore, significant differences were observed in TBIL, BUN, PT, and Cr between survivors and nonsurvivors (Table 1).

Baseline characteristics comparison between high and low sACR groups

A ROC curve analysis demonstrated an AUC of 0.769, with a sensitivity of 75.0% and specificity of 71.4% (Fig. 2). The optimal sACR and Cr cutoff values were 0.206 and 180.3 respectively, leading to the division of the study cohort into two distinct groups based on the optimal sACR or Cr cutoff values. Notably, patients in the low sACR group exhibited significantly elevated levels of age, TBIL, BUN, PT, Cr, BISAP score, APACHE II score, SOFA score and in-hospital mortality compared to those in the high sACR group, while PLT and Alb were notably lower in the low sACR group. There were no significant differences in terms of sex, etiology, length of stay, hypertension status, diabetes status, WBC, Hb, FBG, and Ca between the two groups (Table 2). Further ROC analysis demonstrated that the predictive ability of sACR was significantly higher than that of Ca or BISAP score (both Delong test, $P<0.05$), while comparable to that of Cr (Delong test, $P>0.05$) (Supplementary Fig. 1).

Association between sACR and in-hospital mortality

The results of the univariate logistic analysis showed that Age, Platelet count, Blood urea nitrogen, prothrombin time, Creatinine (both as continuous and categorical variables), and sACR (both as continuous and categorical variables) are significantly associated with in-hospital mortality (Table 3). Following adjustment for covariates, multivariate logistic analysis indicated that sACR (continuous variable) was independently associated with in-hospital mortality, while Cr (continuous variable) was not independently associated with in-hospital mortality; meanwhile, elevated sACR (categorical variables) was associated with a favorable prognosis, whereas elevated Cr (categorical variables) was associated with an unfavorable prognosis (Table 4). As depicted in Fig. 3, there was a linear negative association between sACR levels and the risk of in-hospital mortality (P for non-linearity>0.05), highlighting the escalating risk of in-hospital mortality with diminishing sACR values. Moreover, sensitivity analysis utilizing E value was conducted to evaluate the potential influence of unmeasured confounders. The calculated E value of 3.53 surpassed the relative risk of sACR and unmeasured confounders (RR=2.06), suggesting that the probable impact of unmeasured confounders on the correlation between sACR levels and in-hospital mortality was likely insignificant.

Discussion

In this study, we investigated the relationship between sACR and in-hospital mortality in patients with SAP. Our findings revealed that a lower sACR level was independently associated with higher in-hospital mortality. Moreover, we observed a linear relationship between sACR values and the risk of in-hospital mortality, indicating that the risk of in-hospital mortality decreased with

Table 1 Baseline characteristics

Characteristics	All patients (*n* = 114)	Survivors (*n* = 70)	Nonsurvivors (*n* = 44)	*P* value
Age, years	59.6 ± 16.1	55.5 ± 16.7	66.0 ± 12.9	0.001
Sex, n (%)				0.834
Female	61 (53.5%)	38 (54.3%)	23 (52.3%)	
Male	53 (46.5%)	32 (45.7%)	21 (47.7%)	
Etiology				0.050
Biliary	74 (64.9%)	47 (67.1%)	27 (61.4%)	
Alcohol	11 (9.6%)	6 (8.6%)	5 (11.4%)	
Hyperlipemia	18 (15.8%)	14 (20.0%)	4 (9.1%)	
Other	11 (9.6%)	3 (4.3%)	8 (18.2%)	
Total length of stay, days	19.0 (10.8, 30.0)	23.5 (16.8, 37.0)	8.0 (4.0, 22.3)	< 0.001
Length of ICU stay, days	9.0 (5.0, 18.3)	10.5 (7.0, 17.5)	6.5 (1.3, 19.5)	0.014
Hypertension, n (%)				0.172
Yes	53 (46.5%)	29 (41.4%)	24 (54.5%)	
No	61 (53.5%)	41 (58.6%)	20 (45.5%)	
Diabetes (n,%)				0.712
Yes	34 (29.8%)	20 (28.6%)	14 (31.8%)	
No	80 (70.2%)	50 (71.4%)	30 (68.2%)	
Laboratory parameters				
White blood cell count, 10^9/L	13.8 (10.3, 18.0)	13.5 (10.2. 17.3)	14.7 (11.0, 19.3)	0.503
Platelet count, 10^9/L	163.3 ± 60.3	171.4 ± 62.5	150.3 ± 54.8	0.069
Hemoglobin, g/L	134.5 ± 30.1	131.1 ± 31.9	140.1 ± 26.4	0.120
Total bilirubin, μmol/L	23.4 (14.7, 39.0)	21.0 (12.3, 37.9)	27.3 (18.4, 39.6)	0.042
Blood urea nitrogen, mmol/L	12.1 (8.1, 17.3)	9.2 (6.2, 15.9)	15.6 (11.8, 21.8)	< 0.001
Prothrombin time, s	13.9 (12.6, 15.5)	13.6 (12.2, 14.4)	14.8 (13.6, 16.8)	0.001
Fasting blood glucose, mmol/L	9.9 (7.5, 13.2)	10.5 (7.6, 13.6)	9.4 (7.4, 12.5)	0.430
Calcium, mmol/L	1.8 ± 0.3	1.8 ± 0.3	1.8 ± 0.4	0.595
Albumin, g/L	31.3 ± 6.2	31.4 ± 6.6	31.1 ± 5.7	0.821
Creatinine, μmol/L	125.8 (70.6, 238.2)	83.8 (57.3, 170.0)	224.4 (123.3, 333.5)	< 0.001
sACR	0.2 (0.1, 0.5)	0.4 (0.2, 0.5)	0.1 (0.1, 0.2)	< 0.001
BISAP score	3.0 (2.0, 4.0)	3.0 (2.0, 4.0)	4.0 (3.0, 4.0)	< 0.001
APACHE II score	16.0 (11.0, 22.0)	12.0 (10.0, 17.0)	22.5 (19.0, 25.75)	< 0.001
SOFA score	8.0 (6.0, 10.0)	7.0 (6.0, 9.25)	10.0 (8.0, 11.0)	< 0.001
Systemic complications				
Gastrointestinal bleeding, n (%)	17 (14.9%)	3 (4.3%)	14 (31.8%)	
Persistent respiratory failure, n (%)	75 (65.8%)	35 (50%)	40 (90.9%)	
Persistent circulatory failure, n (%)	40 (35.1%)	9 (12.9%)	31 (70.5%)	
Persistent renal failure, n (%)	34 (29.8%)	13 (18.6%)	21 (47.7%)	
Local complications				
APFC, n (%)	52 (45.6%)	31 (44.3%)	21 (47.7%)	
PPC, n (%)	4 (3.5%)	4 (5.7%)	0 (0.0%)	
ANC, n (%)	29 (25.4%)	19 (27.1%)	10 (22.7%)	
WON, n (%)	8 (7.0%)	7 (10.0%)	1 (2.3%)	

Data are presented as numbers, mean ± standard deviation, median (25th–75th percentiles), or frequency [percentage (%)]

Abbreviations: sACR, serum albumin-to-serum creatinine ratio; ICU, intensive care unit; BISAP, bedside index for severity in acute pancreatitis; APACHE II, acute physiology and chronic health evaluation; SOFA, sequential organ failure assessment; APFC, acute peripancreatic fluid collection; PPC, pancreatic pseudocyst; ANC, acute necrotic collection; WON, walled-off necrosis

increasing sACR values. To the best of our knowledge, this is the first study to explore the association between sACR and in-hospital mortality in patients with SAP.

Previous studies have demonstrated that sACR is a simple and cost-effective indicator that is associated with the prognosis of various diseases [27–29]. For instance, Zhao et al. reported a negative association between low sACR levels and mortality of AP patients who underwent debridement [27]. Chen et al. also found an inverse correlation between low sACR levels and the 28-day mortality of burn patients [28]. Additionally, Turkyilmaz et al. noted a negative association between low sACR and in-hospital mortality in patients with ST-segment elevation myocardial infarction following percutaneous coronary

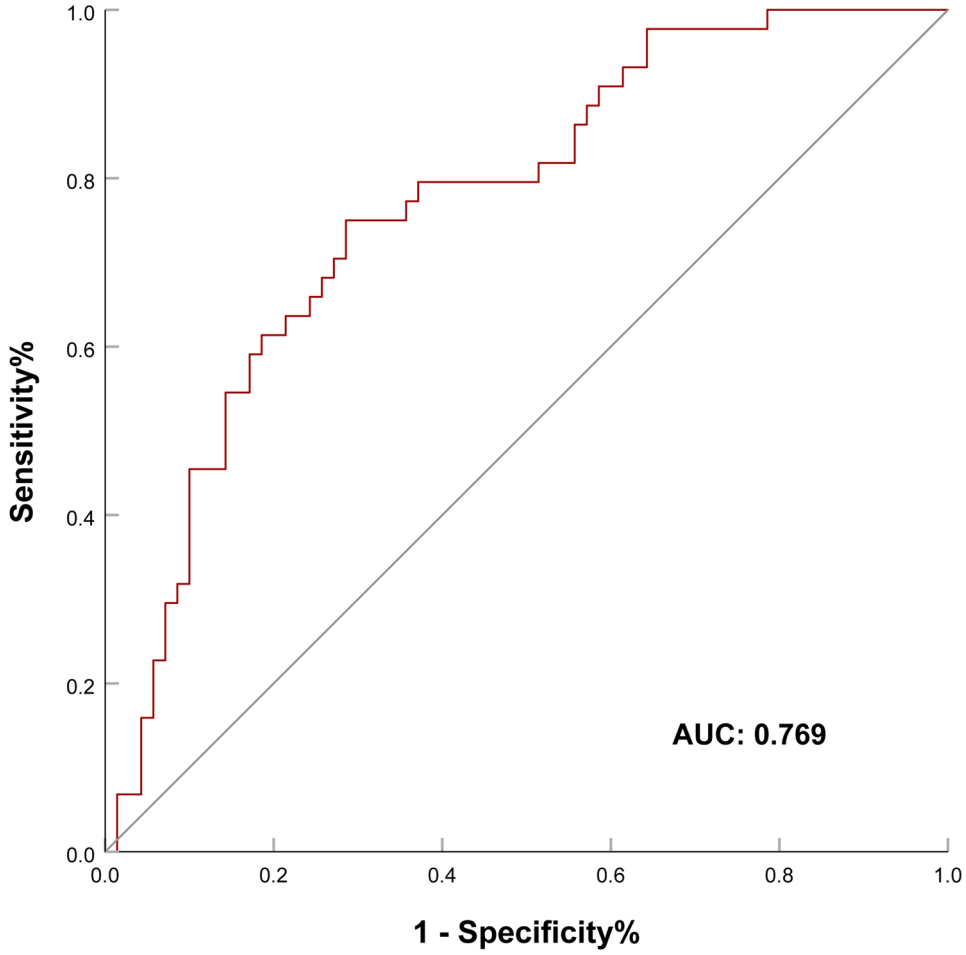

Fig. 2 Receiver operating characteristic curve of sACR for predicting in-hospital mortality in patients with severe acute pancreatitis

interventions [29]. Similarly, our study demonstrated an inverse association between low sACR levels and in-hospital mortality in SAP patients.

Inflammation plays a significant pathological role in SAP [30]. The decrease in albumin levels can be attributed to increased catabolism, reduced synthesis, and extravascular leakage due to edema. In the presence of an underlying inflammatory process, inflammatory mediators can accelerate the breakdown of albumin, cause capillary leakage, and facilitate the transfer of albumin out of the blood vessels, consequently impacting serum albumin levels [31, 32]. Albumin serves as both a robust indicator of inflammation and a predictor of poor prognosis. Li et al. found that low levels of albumin were linked to a higher risk of persistent organ failure in AP patients [33]. Furthermore, a large cohort study revealed that low serum albumin levels were associated with elevated inflammatory indicators and 30-day mortality [8]. Creatinine, which is the most frequently utilized biomarker for measuring kidney function, is often found to be elevated in cases of systemic inflammatory response syndrome [34, 35]. Studies by Erika et al. and Muddana et al. showed a positive correlation between higher serum creatinine levels and mortality and complications in AP, respectively [16, 36]. Additionally, Lipinski et al. suggested that early measurement of elevated creatinine levels in AP can predict pancreatic necrosis and poor outcomes [37]. Therefore, the combined assessment of serum albumin and creatinine levels provides valuable insight into inflammatory status and has the potential to predict in-hospital mortality in SAP patients.

Our study has several limitations. Firstly, the retrospective design may introduce a selection bias, necessitating further validation through prospective multicenter studies with larger sample sizes. Secondly, the specific mechanism underlying in-hospital mortality in SAP patients related to sACR remains unclear and warrants further investigation.

Table 2 Baseline characteristics of the study population with different levels of sACR group

Characteristics	sACR groups ($n=114$)		*P* value
	sACR ≦ 0.206 ($n=53$)	sACR > 0.206 ($n=61$)	
Age, years	65.6 ± 13.6	54.4 ± 16.4	< 0.001
Sex, n (%)			0.206
Female	25 (47.2%)	36 (59.0%)	
Male	28 (52.8%)	25 (41.0%)	
Etiology			0.230
Biliary	34 (64.2%)	40 (65.6%)	
Alcohol	5 (9.4%)	6 (9.8%)	
Hyperlipemia	6 (11.3%)	12 (19.7%)	
Other	8 (15.1%)	3 (4.9%)	
Total length of stay, days	16.0 (6.0, 32.5)	21.0 (15.0, 29.5)	0.060
Length of ICU stay, days	10.0 (4.0, 20.0)	8.0 (5.5, 17.0)	0.509
In-hospital mortality, n (%)	33 (62.3%)	11 (18.0%)	< 0.001
Hypertension, n (%)			0.101
Yes	29 (54.7%)	24 (39.3%)	
No	24 (45.3%)	37 (60.7%)	
Diabetes (n,%)			0.190
Yes	18 (35.8%)	15 (24.6%)	
No	34 (64.2%)	46 (75.4%)	
Laboratory parameters			
White blood cell count, 10^9/L	13.8 (9.7, 20.0)	13.7 (10.4, 17.4)	0.455
Platelet count, 10^9/L	144.6 ± 58.0	179.5 ± 58.0	0.002
Hemoglobin, g/L	131.4 ± 25.0	137.3 ± 33.9	0.296
Total bilirubin, μmol/L	27.6 (20.8, 42.0)	18.2 (12.0, 33.1)	0.002
Blood urea nitrogen, mmol/L	17.2 (14.9, 23.0)	8.4 (5.4, 9.8)	< 0.001
Prothrombin time, s	14.5 (13.3, 16.5)	13.6 (12.2, 14.4)	0.012
Fasting blood glucose, mmol/L	9.4 (7.3, 12.2)	10.7 (7.6, 13.8)	0.458
Calcium, mmol/L	1.7 ± 0.3	1.8 ± 0.3	0.410
Albumin, g/L	29.3 ± 5.2	33.0 ± 6.5	0.001
Creatinine, μmol/L	259.6 (195.7, 249.8)	71.3 (55.6, 92.4)	< 0.001
sACR	0.1 (0.1, 0.2)	0.4 (0.4, 0.6)	< 0.001
BISAP score	4.0 (3.0, 4.0)	3.0 (2.0, 3.5)	< 0.001
APACHE II score	20.0 (15.0, 24.0)	13.0 (10.0, 18.0)	< 0.001
SOFA score	10.0 (8.0, 11.0)	7.0 (6.0, 9.0)	< 0.001
Systemic complications			
Gastrointestinal bleeding, n (%)	12 (22.6%)	5 (8.2%)	
Persistent respiratory failure, n (%)	39 (73.6%)	36 (59.0%)	
Persistent circulatory failure, n (%)	26 (49.1%)	14 (23.0%)	
Persistent renal failure, n (%)	23 (43.4%)	11 (18.0%)	
Local complications			
APFC, n (%)	28 (52.8%)	24 (39.3%)	
PPC, n (%)	0 (0.0%)	4 (6.6%)	
ANC, n (%)	9 (17.0%)	20 (32.8%)	
WON, n (%)	4 (7.5%)	4 (6.6%)	

Data are presented as numbers, mean ± standard deviation, median (25th–75th percentiles), or frequency [percentage (%)]. Abbreviations: sACR, serum albumin-to-serum creatinine ratio; ICU, intensive care unit; BISAP, bedside index for severity in acute pancreatitis; APACHE II, acute physiology and chronic health evaluation; SOFA, sequential organ failure assessment; APFC, acute peripancreatic fluid collection; PPC, pancreatic pseudocyst; ANC, acute necrotic collection; WON, walled-off necrosis

Table 3 Association between variables and in-hospital mortality in patients with severe acute pancreatitis in univariate analyses

Variables	Univariate analyses		
	OR	95% CI	*P* value
Age, years	1.047	1.020–1.077	0.001
Sex, n (%)	0.922	0.432–1.970	0.834
Hypertension, n (%)			
No	Ref		
Yes	1.697	0.795–3.660	0.173
Diabetes (n,%)			
No	Ref		
Yes	1.167	0.508–2.640	0.712
White blood cell count, 109/L	1.001	0.945–1.058	0.974
Platelet count, 109/L	0.994	0.987-1.000	0.072
Hemoglobin, g/L	1.010	0.997–1.024	0.122
Total bilirubin, μmol/L	1.005	0.993–1.017	0.427
Blood urea nitrogen, mmol/L	1.104	1.048–1.172	< 0.001
Prothrombin time, s	1.310	1.104–1.594	0.004
Fasting blood glucose, mmol/L	0.965	0.878–1.057	0.450
Ca2+, mmol/L	1.376	0.428–4.529	0.592
Creatinine, μmol/L (continuous variable)	1.007	1.004–1.011	< 0.001
Creatinine			
Low	Ref		
High	10.456	10.445–26.279	< 0.001
sACR (continuous variable)	0.004	0.0002–0.042	< 0.001
sACR			
Low	Ref		
High	0.133	0.055–0.306	< 0.001

Table 4 sACR was independently associated in-hospital mortality in patients with severe acute pancreatitis in multivariate analyses

Variables	Multivariate analyses		
	OR	95% CI	*P* value
[#]Creatinine, μmol/L (continuous variable)	1.006	1.000-1.012	0.053
[#]Creatinine			
Low	Ref		
High	7.254	2.375–24.166	< 0.001
[*]sACR (continuous variable)	0.009	0.0002–0.269	0.011
[*]sACR			
Low	Ref		
High	0.236	0.075–0.744	0.014

[#]indicates: Age, Platelet count, Blood urea nitrogen, prothrombin time and Creatinine were included in the multivariate logistic analysis. [*]indicates: Age, Platelet count, Blood urea nitrogen, prothrombin time and sACR were included in the multivariate logistic analysis. Variables that did not have a significant effect on outcome ($p > 0.1$) in the univariate analysis were not included in the multivariate logistic analysis

Abbreviations: sACR, serum albumin-to-serum creatinine ratio; OR, odds ratio; CI, confidence interval

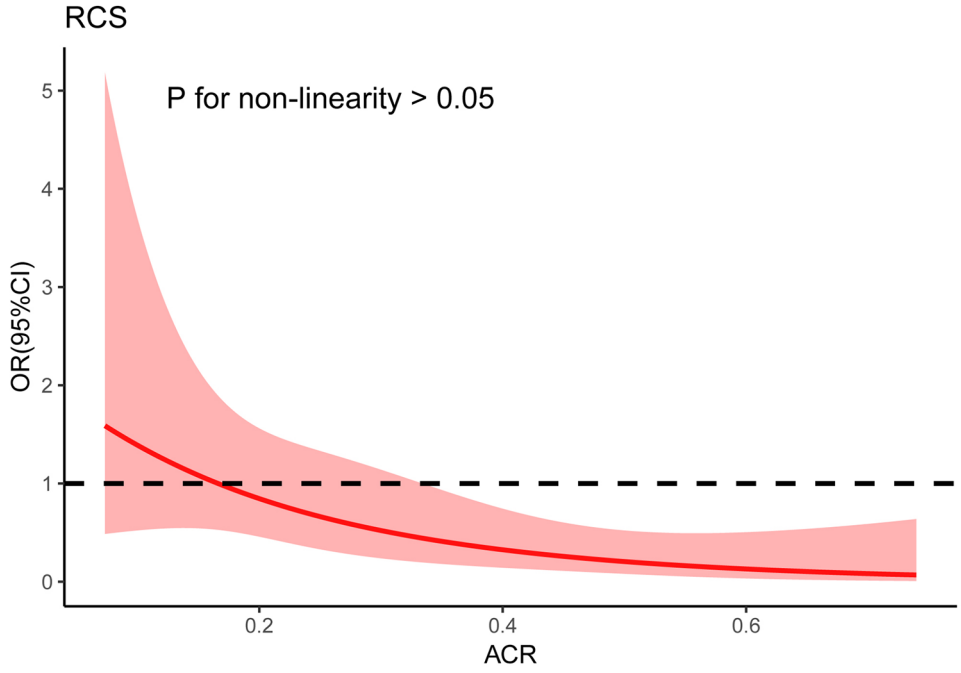

Fig. 3 Associations of the sACR values and the risk of in-hospital mortality in patients with severe acute pancreatitis using restricted cubic splines

Conclusion

In conclusion, sACR serves as a simple and practical clinical indicator with utility in assessing in-hospital mortality in SAP patients. This could aid clinicians in implementing more proactive treatment strategies to reduce in-hospital mortality.

Abbreviations

sACR	Serum albumin-to-serum creatinine ratio
SAP	Severe acute pancreatitis
RCS	Restricted cubic spline
AP	Acute pancreatitis
WBC	White blood cells count
PLT	Platelets count
Hb	Hemoglobin
TBIL	Total bilirubin
Alb	Albumin
Cr	Creatinine
BUN	Blood urea nitrogen
PT	Prothrombin time
Ca	Calcium
FBG	Fasting blood glucose
BISAP	Acute Pancreatitis Bedside Severity Index
ROC	Operating characteristic curve
ICU	Intensive care unit
AUC	Area under the curve

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12876-024-03493-4.

Supplementary Material 1: Supplement Figure 1 Receiver operating curves of the sACR, Cr, Ca, and the BISAP score for prediction in-hospital mortality in patients with severe acute pancreatitis

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Author contributions

Study conception and design by W.W; data acquisition and analysis, and original draft preparation by L.Y, S.C and M.C; Tables 1, 2 and 4 and Supplement Fig. 1 preparation: L.Y; Table 3; Fig. 1 preparation: S.C; Figs. 2 and 3 preparation: M.C; Validation: J.Z, C.H and W.W; all the authors involved in drafting or revising the article and approved of the submitted version.

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Data availability

The data information used and/or analyzed during this study are available from the corresponding author (Wei Wang: wwwy@wnmc.edu.cn) on reasonable request.

Declarations

Ethics approval and consent to participate

Due to retrospective nature of the study, ethics approval and informed consent from patients were waived by the Division of Science and Technology of Yijishan hospital of Wannan Medical college in accordance with national legislation and institutional requirements.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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References

1. Lee PJ, Papachristou GI. New insights into acute pancreatitis. Nat Rev Gastroenterol Hepatol. 2019;16:479–96.

2. Iannuzzi JP, King JA, Leong JH, et al. Global incidence of Acute Pancreatitis is increasing over time: a systematic review and Meta-analysis. Gastroenterology. 2022;162:122–34.
3. Greenberg JA, Hsu J, Bawazeer M, et al. Clinical practice guideline: management of acute pancreatitis. Can J Surg. 2016;59:128–40.
4. Li XY, He C, Zhu Y, et al. Role of gut microbiota on intestinal barrier function in acute pancreatitis. World J Gastroenterol. 2020;26:2187–93.
5. Popa CC, Badiu DC, Rusu OC, et al. Mortality prognostic factors in acute pancreatitis. J Med Life. 2016;9:413–8.
6. Banks PA, Bollen TL, Dervenis C, et al. Classification of acute pancreatitis–2012: revision of the Atlanta classification and definitions by international consensus. Gut. 2013;62:102–11.
7. Kaplan M, Ates I, Akpinar MY, et al. Predictive value of C-reactive protein/albumin ratio in acute pancreatitis. Hepatobiliary Pancreat Dis Int. 2017;16:424–30.
8. Eckart A, Struja T, Kutz A et al. Relationship of nutritional status, inflammation, and serum albumin levels during Acute illness: a prospective study. Am J Med. 2020;133:713 – 22.e7.
9. Chai X, Huang HB, Feng G, et al. Baseline serum cystatin C is a potential predictor for acute kidney Injury in patients with Acute Pancreatitis. Dis Markers. 2018;2018:8431219.
10. Huang HL, Nie X, Cai B, et al. Procalcitonin levels predict acute kidney injury and prognosis in acute pancreatitis: a prospective study. PLoS ONE. 2013;8:e82250.
11. Liu Y, Liu Y, Wang Q, et al. MIF inhibitor ISO-1 alleviates severe acute pancreatitis-associated acute kidney injury by suppressing the NLRP3 inflammasome signaling pathway. Int Immunopharmacol. 2021;96:107555.
12. Nassar TI, Qunibi WY. AKI Associated with Acute Pancreatitis. Clin J Am Soc Nephrol. 2019;14:1106–15.
13. Yang X, Zhang X, Lin Z, et al. Chaiqin chengqi decoction alleviates severe acute pancreatitis associated acute kidney injury by inhibiting endoplasmic reticulum stress and subsequent apoptosis. Biomed Pharmacother. 2020;125:110024.
14. Talamini G, Uomo G, Pezzilli R, et al. Serum creatinine and chest radiographs in the early assessment of acute pancreatitis. Am J Surg. 1999;177:7–14.
15. Wan J, Shu W, He W, et al. Serum creatinine level and APACHE-II score within 24 h of Admission are Effective for Predicting Persistent Organ failure in Acute Pancreatitis. Gastroenterol Res Pract. 2019;2019:8201096.
16. Muddana V, Whitcomb DC, Khalid A, et al. Elevated serum creatinine as a marker of pancreatic necrosis in acute pancreatitis. Am J Gastroenterol. 2009;104:164–70.
17. Panotopoulos J, Posch F, Funovics PT, et al. Elevated serum creatinine and low albumin are associated with poor outcomes in patients with liposarcoma. J Orthop Res. 2016;34:533–8.
18. Huang X, Liu Y, Zhong C, et al. Association between serum albumin-to-creatinine ratio and clinical outcomes among patients with ST-elevation myocardial infarction after percutaneous coronary intervention: a secondary analysis based on Dryad databases. Front Cardiovasc Med. 2023;10:1191167.
19. Li S, Xie X, Zeng X, et al. Association between serum albumin to serum creatinine ratio and mortality risk in patients with heart failure. Clin Transl Sci. 2023;16:2345–55.
20. Willegger M, Posch F, Schieder S, et al. Serum creatinine and albumin predict sarcoma-specific survival in patients with myofibroblastic and fibroblastic sarcomas. J Orthop Res. 2017;35:2815–24.
21. Pintado MC, Trascasa M, Arenillas C, et al. New Atlanta classification of acute pancreatitis in intensive care unit: complications and prognosis. Eur J Intern Med. 2016;30:82–7.
22. Walter SD. The partial area under the summary ROC curve. Stat Med. 2005;24:2025–40.
23. DeLong ER, DeLong DM, Clarke-Pearson DL. Comparing the areas under two or more correlated receiver operating characteristic curves: a nonparametric approach. Biometrics. 1988;44:837–45.
24. Yang L, He C, Wang W. Association between neutrophil to high-density lipoprotein cholesterol ratio and disease severity in patients with acute biliary pancreatitis. Ann Med. 2024;56:2315225.
25. Harrell FE Jr, Lee KL, Pollock BG. Regression models in clinical studies: determining relationships between predictors and response. J Natl Cancer Inst. 1988;80:1198–202.
26. Huang Z, Han Y, Hu H, et al. Triglyceride to high-density lipoprotein cholesterol ratio is associated with regression to normoglycemia from prediabetes in adults: a 5-year cohort study in China. J Transl Med. 2023;21:868.
27. Zhao Z, Yu Y, Xie R, et al. Prognostic value of the creatinine-albumin ratio in acute pancreatitis debridement. BMC Surg. 2020;20:322.
28. Chen WH, Ye HF, Wu YX, et al. Association of creatinine-albumin ratio with 28-day mortality in major burned patients: a retrospective cohort study. Burns. 2023;49:1614–20.
29. Turkyilmaz E, Ozkalayci F, Birdal O, et al. Serum albumin to creatinine ratio and short-term clinical outcomes in patients with ST-Elevation myocardial infarction. Angiology. 2022;73:809–17.
30. Ismail OZ, Bhayana V. Lipase or amylase for the diagnosis of acute pancreatitis? Clin Biochem. 2017;50:1275–80.
31. Fuhrman MP, Charney P, Mueller CM. Hepatic proteins and nutrition assessment. J Am Diet Assoc. 2004;104:1258–64.
32. Jensen GL. Inflammation as the key interface of the medical and nutrition universes: a provocative examination of the future of clinical nutrition and medicine. JPEN J Parenter Enter Nutr. 2006;30:453–63.
33. Li S, Zhang Y, Li M, et al. Serum albumin, a good indicator of persistent organ failure in acute pancreatitis. BMC Gastroenterol. 2017;17:59.
34. Kashani K, Rosner MH, Ostermann M, Creatinine. From physiology to clinical application. Eur J Intern Med. 2020;72:9–14.
35. Chen Y, Wei W, Fu J, et al. Forsythiaside A ameliorates sepsis-induced acute kidney injury via anti-inflammation and antiapoptotic effects by regulating endoplasmic reticulum stress. BMC Complement Med Ther. 2023;23:35.
36. Wilkman E, Kaukonen KM, Pettilä V, et al. Early hemodynamic variables and outcome in severe acute pancreatitis: a retrospective single-center cohort study. Pancreas. 2013;42:272–78.
37. Lipinski M, Rydzewski A, Rydzewska G. Early changes in serum creatinine level and estimated glomerular filtration rate predict pancreatic necrosis and mortality in acute pancreatitis: Creatinine and eGFR in acute pancreatitis. Pancreatology. 2013;13:207–11.

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