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Association between serum uric acid to serum creatinine ratio and in-hospital mortality in patients with severe acute pancreatitis: a retrospective study

Qing Wang[1,2†], Yanchun Peng[1,2†], Jianfu Jiang[1], Yinghuan Liang[1], Yaqin Chen[3], Liangwan Chen[4,5*] and Yanjuan Lin[2,4*]

Abstract

Objective Serum uric acid/creatinine (SUA/Cr) can reflect the metabolic status of the body. However, the effect of SUA/Cr on the prognosis of severe acute pancreatitis (SAP) patients is not clear. This study aimed to investigate the impact of SUA/Cr on in-hospital mortality in SAP patients.

Methods This study was a retrospective study involving 224 SAP patients admitted to the ICU of Fujian Medical University Union Hospital from January 2020 to April 2024. The primary outcome for patient inclusion was in-hospital mortality, and the secondary outcome include acute kidney injury (AKI), liver dysfunction, sepsis, septic shock, hemorrhagic shock, bacteremia, septicemia, respiratory failure, multiple organ dysfunction syndrome (MODS), etc. The data on serum uric acid and serum creatinine were extracted from the electronic medical record system. Data analysis was performed using SPSS 26.0 software. Receiver operating characteristic (ROC) curve analysis was employed to determine the cutoff value of SUA/Cr. Multivariate logistic regression analysis was conducted to assess the association between SUA/Cr and in-hospital mortality in SAP patients.

Results A total of the 224 SAP patients included in the study, 88 (39.3%) had low SUA/Cr, while 136 (61.7%) had high SUA/Cr. There were statistically significant differences between the two groups in survival, AKI, sepsis, septic shock, septicemia, MODS, ICU days, and length of hospital stay ($P<0.001$). Models I, II and III of in-hospital mortality in SAP patients adjusted for age, body mass index (BMI), sex, diabetes mellitus and nonalcoholic fatty liver disease, respectively. The results of model I showed that the OR of SUA was 1.002 ($P=0.601$), and SCR was 1.009 ($P=0.027$). In Model II, compared with the high SUA/Cr group, the OR of the low group was 8.027 (95%CI:1.447–14.511, $P=0.017$). In Model III, SUA/Cr OR was 0.456 (95%CI: 0.239–0.872, $P=0.018$). Meanwhile, age, BMI, APACHE-II scores, and mechanical ventilation were independent risk factors for death in SAP patients.

†Qing Wang and Yanchun Peng first co-authors.

*Correspondence:
Liangwan Chen
fjxhlwc@163.com
Yanjuan Lin
fjxhyjl@163.com

Full list of author information is available at the end of the article

Conclusion This study indicated that SAP patients in the low SUA/Cr group have worse prognosis and higher in-hospital mortality, and low SUA/Cr is independently associated with increased in-hospital mortality. SUA/Cr may serve as a prognostic biomarker for SAP patients.

Keywords Severe acute pancreatitis, In-hospital mortality, Serum uric acid, Serum creatinine, Ratio

Introduction

Acute Pancreatitis (AP) is a common acute abdominal condition, with an incidence of approximately 30–40 cases per 100,000 people globally each year. With the development of society, the incidence rate of this disease is increasing year by year [1, 2]. 13.1%–30% of AP patients may progress to severe acute pancreatitis (SAP), which is characterized by an abrupt onset, rapid progression, and severe condition [3, 4]. Despite significant progress in the diagnosis and treatment of SAP, the mortality rate remains high, ranging from 14.3% to 40.0%, leading to higher healthcare expenditures [4–7]. Currently, there are no specific drugs for the treatment of AP. Therefore, actively exploring predictive factors for in-hospital mortality in SAP patients and identifying high-risk individuals is crucial for determining treatment strategies. However, existing research focuses more on early identification of risk factors that may progress to SAP, with limited research on the prognosis after progression to SAP.

Serum uric acid (SUA) is the end product of purine metabolism and has antioxidant properties, which can clear free radicals in the body and reduce oxidative stress damage [8, 9]. Serum creatinine (SCR) is a byproduct of muscle metabolism, mainly derived from creatine and phosphocreatine in muscles, and its accumulation in the human body enhances inflammatory response [10, 11]. In recent years, as a novel biomarker, the SUA/Cr has been shown to be associated with the progression and prognosis of type 2 diabetes mellitus [12], acute ischemic stroke [13], hypertension [14], and acute myocardial infarction [15], but the results are still controversial. Excessive inflammatory response is a key factor in the pathogenesis of SAP [16]. Studies have shown that elevated levels of serum uric acid and creatinine can increase the release of inflammatory factors (such as tumor necrosis factor-α and interleukin-6), playing a significant role in the progression of AP [17, 18]. There are few studies on the relationship between SUA/Cr and the prognosis of patients with digestive diseases, and no studies have specifically examined its relationship with in-hospital mortality in SAP patients. Therefore, this study retroactively collected data from SAP patients to investigate the impact of the SUA/Cr on in-hospital mortality.

Materials and methods

Study population

This study was a retrospective study. Select SAP patients (ICD-10 code: K85. X55) admitted to the ICU of Fujian Medical University Union Hospital from January 2020 to April 2024. Inclusion criteria: age ≥ 18 years old, complete laboratory parameters within 24 h of admission. Exclusion criteria: traumatic pancreatitis, pregnancy, malignant tumor, history of pancreatic surgery.

Ethical consideration

This study follows the Helsinki Declaration and has been approved by the Medical Ethics Review Committee of Fujian Medical University Union Hospital (number: 2024KY209), and has obtained exemption from informed consent.

Data collection

The general data, clinical data and outcome indicators of patients were recorded by the electronic medical record system. General information included the patient's age, sex, body mass index (BMI), smoking history, drinking history, previous medical history [hypertension, diabetes mellitus (DM), heart disease, cerebrovascular disease, gallbladder or bile duct disease, nonalcoholic fatty liver disease (NAFLD)], and etiology of SAP (biliary, hyperlipidemia, alcohol, others or unknown). Clinical data included nutrition risk screening (NRS2002) score, acute physiology and chronic health evaluation-II (APACHE-II) score on the first day of ICU admission, laboratory test indicators within 24 h of admission [white blood cell (WBC) counts, neutrophil counts, lymphocytes, monocytes, red blood cell (RBC) counts, hemoglobin, platelets, C-reactive protein (CRP), triglycerides (TG), total cholesterol (TC), high-density lipoprotein (HDL), low-density lipoprotein (LDL), SUA, SCR, blood glucose, lactic acid], treatment modalities [surgery, continuous renal replacement therapy (CRRT), plasma exchange, blood perfusion]. Record patients' mechanical ventilation (MV) time, ICU stay, length of hospital stay, acute kidney injury (AKI), liver dysfunction, sepsis, septic shock, hemorrhagic shock, bacteremia, septicemia, respiratory failure, (multiple organ dysfunction syndrome) MODS, and mortality through the electronic medical record system.

The primary outcome of this study was in-hospital mortality, with secondary outcomes including AKI, liver dysfunction, sepsis, septic shock, hemorrhagic shock, bacteremia, septicemia, respiratory failure, MODS, ICU stay, and length of hospital stay. The definition of AKI was based on the 2012 KDIGO guidelines [19], which state that SCR levels increase to ≥ 1.5 times baseline within 7 days, or the SCR level increases by ≥ 0.3 mg/dl (>26.5 μ mol/l) within 48 h, or the urine output remains

below 0.5 ml/kg/h for 6 h. Liver dysfunction was defined as a peak serum total bilirubin >7 mg/dl in the ICU [20]. Determine whether the patient has AKI or liver dysfunction based on the SCR value and serum total bilirubin in the electronic medical record system. MODS was defined as dysfunction of at least two organs during the ICU period.

Statistical analysis

SPSS26.0 software was used to analyze the data. Normally distributed data are expressed as mean ± standard deviation and compared with the Student's t-test; non-normally distributed data are reported as median and interquartile range and compared using the Mann-Whitney U test. Categorical data are presented as frequency (%), with comparisons made by Chi-square or adjusted Chi-square test. The SUA/Cr cut-off value was determined as 2.56 by ROC analysis, dividing patients into low and high SUA/Cr groups. Predictive ability of indicators was evaluated by comparing AUC, sensitivity, and specificity. Indicators with $P<0.05$ in the univariate analysis of SAP mortality were included in multivariate regression analysis to identify independent risk factors for mortality in SAP patients. Model I includes the factors: age, BMI, sex, DM, NAFLD, platelets, hemoglobin, APACHE-II, SUA, SCR, and MV. Model II includes the factors: age, BMI, sex, DM, NAFLD, platelets, hemoglobin, APACHE-II, SUA/Cr (categorical variable), and MV. Model III includes the factors: age, BMI, sex, DM, NAFLD, platelets, hemoglobin, APACHE-II, SUA/Cr (continuous variable), and MV. Figures 2 and 3 were plotted using graphpad prism X8. P value of <0.05 was considered statistically significant.

Result

From January 2020 to April 2024, this study included 251 subjects. 27 cases were excluded, resulting in a final analysis of 224 subjects. All enrolled patients completed the collection of general and clinical data. The specific flowchart for patient inclusion is shown in Fig. 1.

There were 88 cases (39.3%) in the low SUA/Cr group, with an age of (53.44 ± 15.56) years, and 136 cases (61.7%) in the high SUA/Cr group, with an age of (44.05 ± 15.10) years, the difference between the two groups was statistically significant ($P<0.001$). There was no significant difference in sex, BMI, smoking history, drinking history, hypertension, heart disease and cerebrovascular disease between the two groups ($P>0.05$). In the low SUA/Cr group, there were 20 cases (22.7%) of DM and 22 cases (25.0%) of NAFLD. In the high SUA/Cr group, there were 50 cases (36.8%) of DM and 68 cases (50.0%) of NAFLD. The differences between the two groups for these conditions were statistically significant ($P<0.05$). (Table 1)

As shown in Table 2, among the etiology of SAP, hyperlipidemia was present in 32 cases (55.4%) in the low SUA/Cr group and in 81 cases (59.6%) in the high SUA/Cr group, with a statistically significant difference in etiology between the two groups ($P=0.005$). There were no

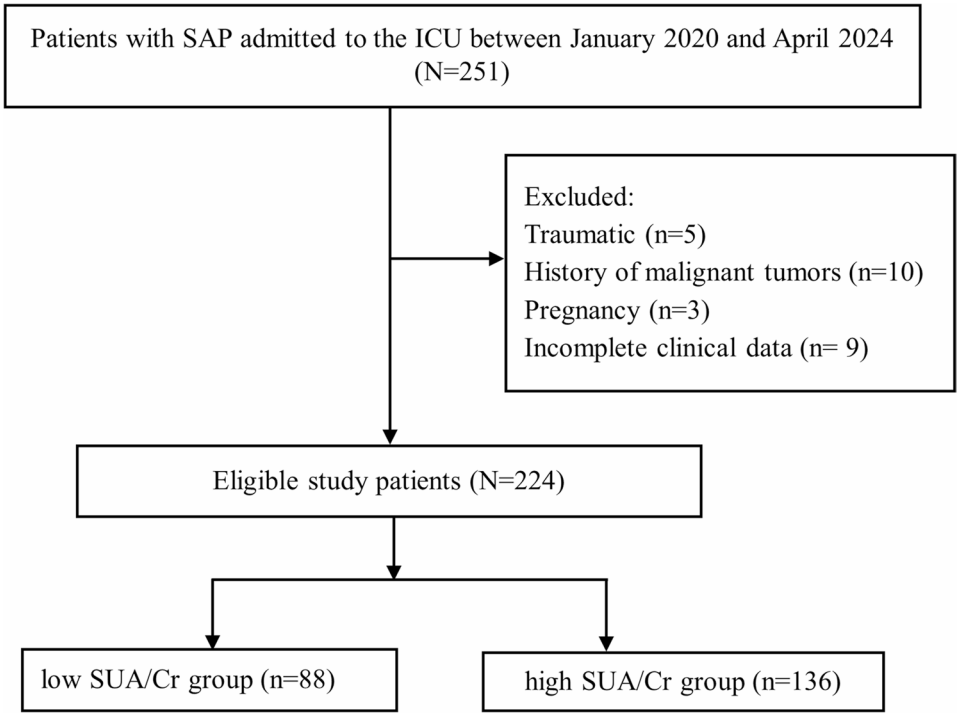

Fig. 1 Flow chart of patient enrollment. SAP, severe acute pancreatitis; SUA/Cr, serum uric acid to serum creatinine ratio

Table 1 Baseline data for the two groups of SAP patients according to the SUA/Cr

Variables	Total *N* = 224	Low group *n* = 88	High group *n* = 136	*P* value
Age, years	47.74 ± 15.92	53.44 ± 15.56	44.05 ± 15.10	< 0.001
Male	155 (69.2)	64 (72.7)	91 (66.9)	0.357
BMI, kg/m^2	23.86 (22.37, 25.95)	24.22 (22.86, 26.01)	23.39 (22.04, 25.90)	0.102
Smoking history	86 (38.4)	30 (34.1)	56 (41.2)	0.287
Drinking history	63 (28.1)	22 (25.0)	41 (30.1)	0.403
Hypertension	73 (32.6)	34 (38.6)	39 (28.7)	0.120
DM	70 (31.3)	20 (22.7)	50 (36.8)	0.027
Heart disease	12 (5.4)	6 (6.8)	6 (4.4)	0.546
Cerebrovascular disease	6 (2.7)	3 (3.4)	3 (2.2)	0.682
Gallbladder or bile duct disease	81 (36.2)	35 (39.8)	46 (33.8)	0.365
NAFLD	90 (40.2)	22 (25.0)	68 (50.0)	< 0.001

Abbreviations: *SAP* severe acute pancreatitis, *SUA/Cr* serum uric acid to serum creatinine ratio, *BMI* body mass index, *DM* diabetes mellitus, *NAFLD* nonalcoholic fatty liver disease. Values are expressed as mean ± SD, median (interquartile range) or *n* (%)

Table 2 Clinical situation and laboratory indicator data for the two groups of SAP patients according to the SUA/Cr

Variables	Total *N* = 224	Low group *n* = 88	High group *n* = 136	*P* value
Etiology				0.005
Biliary	70 (31.3)	38 (43.2)	32 (23.5)	
Hyperlipidemia	113 (50.4)	32 (55.4)	81 (59.6)	
Alcohol	12 (5.4)	6 (6.8)	6 (4.4)	
Others or unknown	29 (12.9)	12 (13.6)	17 (12.5)	
NRS 2002, scores	4.0 (3.0, 4.0)	4.0 (3.0, 5.0)	4.0 (3.0, 4.0)	0.222
APACHE-II, scores	12.0 (9.0, 15.0)	14.0 (11.0, 16.0)	11.0 (8.0, 14.0)	< 0.001
WBC, ×10^9/L	13.04 ± 5.35	13.00 ± 5.40	13.07 ± 5.34	0.923
Neutrophils, ×10^9/L	12.05 ± 9.08	12.05 ± 9.08	11.92 ± 8.41	0.498
lymphocytes, ×10^9/L	1.00 (0.62, 1.40)	0.90 (0.58, 1.53)	1.04 (0.70, 1.33)	0.323
Monocytes, ×10^9/L	0.62 ± 0.45	0.61 ± 0.39	0.62 ± 0.49	0.843
RBC, ×10^9/L	4.00 ± 1.13	3.63 ± 1.27	4.25 ± 0.97	< 0.001
Hemoglobin, g/L	123.07 ± 34.95	111.68 ± 37.45	130.43 ± 31.23	< 0.001
Platelets, ×10^9/L	189.0 (137.0, 253.50)	167.00 (102.75, 240.50)	203.00 (156.00, 265.75)	0.002
CRP, mg/L	172.0 (78.46, 236.56)	188.10 (112.94, 248.75)	160.0 (63.65, 223.37)	0.027
TG, mmlo/L	3.17 (1.24, 7.53)	2.55 (1.17, 5.70)	3.85 (1.28, 9.20)	0.055
TC, mmol/L	4.00 (2.39, 6.80)	2.56 (1.92, 4.59)	4.69 (3.14, 7.68)	< 0.001
HDL, mmol/L	0.69 (0.43, 0.94)	0.48 (0.29, 0.81)	0.76 (0.56, 0.97)	< 0.001
LDL, mmol/L	1.72 (1.07, 2.53)	1.13 (0.85, 1.85)	2.05 (1.45, 2.79)	< 0.001
SUA, ummol/L	282.5 (193.0, 395.5)	227.0 (141.50, 335.0)	304.0 (225.0, 401.0)	< 0.001
SCR, ummol/L	173.0 (58.0, 139.5)	178.5 (79.0, 263.5)	64.0 (51.08, 80.50)	< 0.001
Blood sugar, mmol/L	11.33 ± 4.81	11.53 ± 5.53	11.20 ± 4.29	0.620
Lactic acid, mmol/L	2.76 ± 2.76	3.86 ± 3.74	2.00 ± 1.35	< 0.001
Treatment methods				
Operation	77 (34.4)	44 (50.0)	33 (24.3)	< 0.001
CRRT	105 (46.9)	61 (69.3)	44 (32.4)	< 0.001
Plasma exchange	75 (33.5)	20 (22.7)	55 (40.4)	< 0.001
Blood perfusion	32 (14.3)	19 (21.6)	13 (9.6)	< 0.001
MV	78 (34.8)	51 (58.0)	27 (19.9)	< 0.001
MV duration, hours	222.0 (92.15, 379.5)	255.0 (129.5, 385.0)	173.0 (14.13, 354.5)	0.051

Abbreviations: *SAP* severe acute pancreatitis, *SUA/Cr* serum uric acid to serum creatinine ratio, *NRS* nutritional risk screening, *APACHE* acute physiology and chronic health evaluation, *WBC* white blood cell, *RBC* red blood cell, *CRP* C-reactive protein, *TG* triglycerides, *TC* total cholesterol, *HDL* high-density lipoprotein, *LDL* low-density lipoprotein, *CRRT* continuous renal replacement therapy, *MV* mechanical ventilation. Values are expressed as mean ± SD, median (interquartile range) or *n* (%)

Table 3 Clinical outcomes for the two groups of SAP patients according to the SUA/Cr

Variables	Total $N=224$	Low group $n=88$	High group $n=136$	*P* value
In-hospital mortality	40 (17.9)	35 (39.8)	5 (3.7)	< 0.001
AKI	34 (15.2)	25 (28.4)	9 (6.6)	< 0.001
Liver dysfunction	56 (25.0)	26 (29.5)	30 (22.1)	0.206
Sepsis	30 (13.4)	23 (26.1)	7 (5.1)	< 0.001
Septic shock	27 (12.1)	23 (26.1)	4 (2.9)	< 0.001
Hemorrhagic shock	5(2.2)	4 (4.5)	1 (0.7)	0.079
Bacteremia	5 (2.2)	4 (4.5)	1 (0.7)	0.079
Septicemia	10 (4.5)	9 (10.2)	1 (0.7)	0.001
Respiratory failure	49 (21.9)	25 (28.4)	24 (17.6)	0.057
MODS	33 (14.7)	29 (33.0)	4 (2.9)	< 0.001
ICU days, days	7.00 (4.00, 15.75)	14.0 (7.0, 23.0)	6.0 (4.0, 9.75)	< 0.001
Length of hospital stay, days	17.0 (10.0, 28.0)	23.0 (12.0, 33.75)	15.0 (9.0, 25.0)	< 0.001

Abbreviations: *SAP* severe acute pancreatitis, *SUA/Cr* serum uric acid to serum creatinine ratio, *AKI* acute kidney injury, *MODS* multiple organ dysfunction syndrome. Values are expressed as median (interquartile range) or *n* (%)

Table 4 Results of univariate logistic regression analysis of in-hospital mortality in SAP patients

Variables	OR	95% CI	*P*
Age, years	1.039	1.017–1.062	0.001
Male	1.047	0.497–2.206	0.903
BMI, kg/m^2	1.164	1.041–1.302	0.008
Smoking history	1.570	0.751–3.282	0.231
Drinking history	2.062	0.861–4.943	0.104
Hypertension	1.303	0.640–2.656	0.456
DM	0.262	0.098–0.700.098.700	0.008
Heart disease	0.916	0.193–4.350	0.912
Cerebrovascular disease	0.918	0.104–8.078	0.938
NAFLD	0.258	0.109–0.613	0.002
APACHE-II, scores	12.140	6.876–21.435	< 0.001
RBC, $\times10^9$/L	0.536	0.382–0.752	< 0.001
Hemoglobin, g/L	0.983	0.972–0.993	0.001
Platelets, $\times10^9$/L	0.990	0.986–0.995	< 0.001
CRP, mg/L	1.002	0.998–1.006	0.318
TG, mmlo/L	0.998	0.978–1.019	0.862
TC, mmol/L	0.891	0.798–0.995	0.040
HDL, mmol/L	0.462	0.177–1.203	0.114
LDL, mmol/L	0.255	0.141–0.459	< 0.001
SUA, ummol/L	1.149	1.090–1.212	< 0.001
SCR, ummol/L	1.008	1.005–1.011	< 0.001
SUA/Cr	0.313	0.205–0.479	< 0.001
Low SUA/Cr	17.302	6.430–46.558.430.558	< 0.001
Blood glucose, mmol/L	1.006	0.937–1.080	0.874
Lactate acid, mmol/L	1.452	1.222–1.726	< 0.001
MV	22.953	8.467–42.226	< 0.001

Abbreviations: *SAP* severe acute pancreatitis, *OR* odds ratio, *CI* confidence interval, *BMI* body mass index, *DM* diabetes mellitus, *NAFLD* nonalcoholic fatty liver disease, *APACHE* acute physiology and chronic health evaluation, *RBC* red blood cell, *CRP* C-reactive protein, *TG* triglycerides, *TC* total cholesterol, *HDL* high-density lipoprotein, *LDL* low-density lipoprotein, *SUA/Cr* serum uric acid to serum creatinine ratio, *MV* mechanical ventilation

significant differences in NRS2002 score, WBC counts, neutrophil counts, lymphocytes, monocytes, TG and blood glucose between the two groups at admission ($P>0.05$). However, there were statistically significant differences in APACHE II scores, RBC counts, hemoglobin, platelets, CRP, TC, HDL, LDL, SUA, SCR, and lactate acid, treatment method and duration of MV ($P<0.05$).

As shown in Table 3, there were significant differences in mortality, AKI, sepsis, septic shock, septicemia and MODS between the two groups ($P<0.001$). However, there was no significant difference in hemorrhagic shock, bacteremia and respiratory failure between the two groups ($P>0.05$). The duration of ICU stay for the low SUA/Cr group was 14.0 (7.0, 23.0) days, the high SUA/Cr group it was 6.0 (4.0, 9.75) days. The length of hospital stay in the low SUA/Cr group was 23.0 (12.0, 33.75) days, and in the high SUA/Cr group was 15.0 (9.0, 25.0) days, there were statistically significant difference in ICU stay and length of hospital stay between the two groups ($P<0.001$).

As shown in Table 4, the results of univariate logistic regression analysis for in-hospital mortality of ASP patients indicate that age (OR = 1.039, $P=0.001$), BMI (OR = 1.164, $P=0.008$), DM (OR = 0.262, $P=0.008$), NAFLD (OR = 0.258, $P=0.002$), APACHE-II score (OR = 1.039, $P=0.001$), RBC counts (OR = 0.536, $P<0.001$), hemoglobin (OR = 0.983, $P=0.001$), platelets (OR = 0.990, $P<0.001$), TC (OR = 0.891, $P=0.040$), LDL (OR = 255, $P<0.001$), SUA (OR = 1.149, $P<0.001$), SCR (OR = 1.008, $P<0.001$), SUA/Cr (OR = 0.313, $P<0.001$), low SUA/Cr group (OR = 17.303, $P<0.001$), lactic acid (OR = 1.452, $P<0.001$), and MV (OR = 22.953, $P<0.001$) were all factors influencing mortality in SAP patients ($P<0.005$).

Table 5 shows the multivariate logistic regression analysis of in-hospital mortality of SAP patients. The results of model I showed that the OR of SUA was 1.002 (95%CI: 0.994–1.010, $P=0.601$), and SCR was 1.009 (95%CI: 1.001–1.018, $P=0.027$). In Model II, the OR of the low SUA/Cr group was 8.027 (95% CI: 1.447–14.511, $P=0.017$) compared to the high SUA/Cr group. In Model III, SUA/Cr entered the multivariate model as a

Table 5 Results of multivariate logistic regression analysis of in-hospital mortality in SAP patients

Variables	Model I		Model II		Model III	
	OR (95% CI)	*P* value	OR (95% CI)	*P* value	OR (95% CI)	*P* value
Age, years	1.081 (1.015–1.152)	0.015	1.050 (1.005–1.098)	0.028	1.050 (1.005–1.098)	0.029
BMI, kg/m^2	1.424 (1.051–1.930)	0.023	1.291 (1.017–1.639)	0.036	1.290 (1.005–1.655)	0.045
APACHE-II, scores	1.351 (1.078–1.693)	0.009	1.203 (1.011–1.431)	0.037	1.222 (1.024–1.458)	0.026
SUA, ummol/L	1.002 (0.994–1.010)	0.601	-		-	
SCR, ummol/L	1.009 (1.001–1.018)	0.027	-		-	
SUA/Cr	-		-		0.456 (0.239–0.872)	0.018
SUA/Cr (low group)	-	-	8.027 (1.447–14.511)	0.017	-	
MV	15.777 (2.803–38.805)	< 0.001	25.185(5.116–52.777)	< 0.001	21.338 (4.409–43.279)	< 0.001

Model I, II, III adjustment factors: sex, diabetes mellitus, nonalcoholic fatty liver disease, platelets, hemoglobin

Abbreviations: *SAP* severe acute pancreatitis, *OR* odds ratio, *CI* confidence interval, *BMI* body mass index, *APACHE* acute physiology and chronic health evaluation, *SUA/Cr* serum uric acid to serum creatinine ratio, *MV* mechanical ventilation

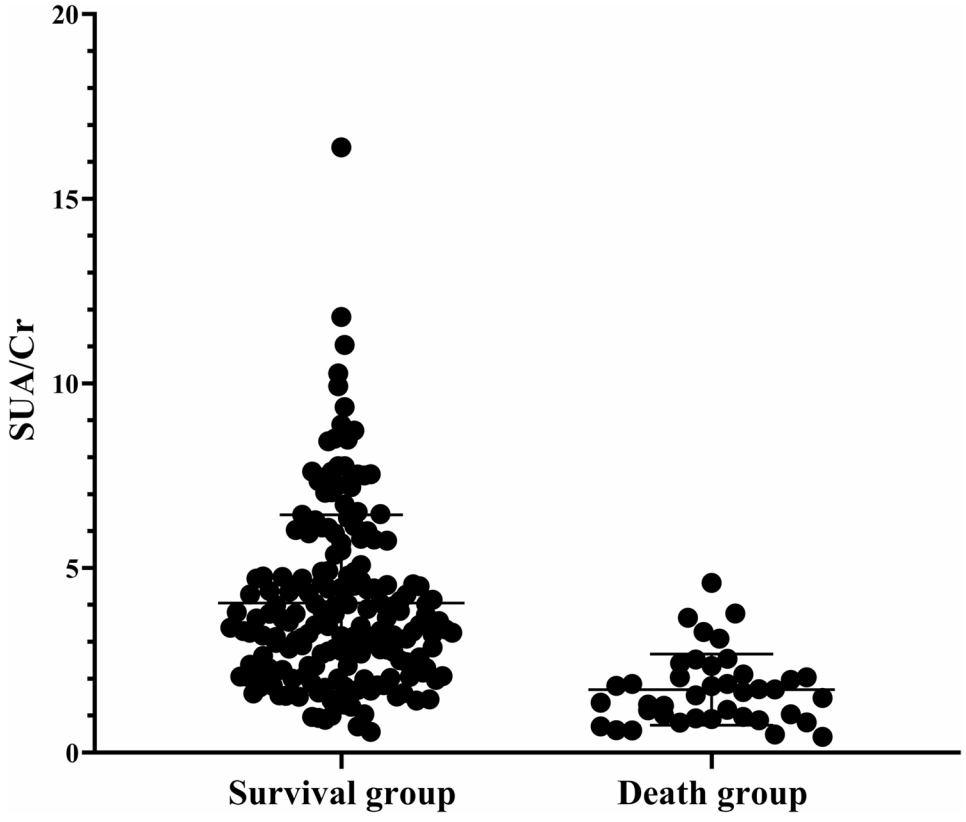

Fig. 2 The distribution of SUA/Cr in the survival and death groups of ASP patients. SAP, severe acute pancreatitis; SUA/Cr, serum uric acid to serum creatinine ratio

continuous variable, OR = 0.456 (95%CI: 0.239–0.872, *P* = 0.018). Among the three models, age, BMI, APACHE-II scores, and MV were independent risk factors for mortality in SAP patients.

Figure 2 illustrates the distribution of the SUA/Cr between the survival group and the death group. In Fig. 3, the AUC for SUA/Cr was 0.849 (95% CI: 0.788–0.910, *P* < 0.001; sensitivity: 87.5%, specificity: 71.2%). The area under the ROC curve for SCR was 0.782 (95% CI: 0.705–0.858, *P* < 0.001; sensitivity: 77.5%, specificity: 67.9%). The AUC for SUA was 0.553 (95% CI: 0.463–0.643, *P* = 0.296; sensitivity: 63.9%, specificity: 54.9%).

Discussion

At present, there are numerous studies on the risk factors associated with the severity of illness in patients with SAP, but fewer focus on the risk factors for in-hospital mortality in this group. In this study, we investigated the association between SUA/Cr and in-hospital mortality in ICU patients with SAP, which to our knowledge is the first study to examine this relationship. The results

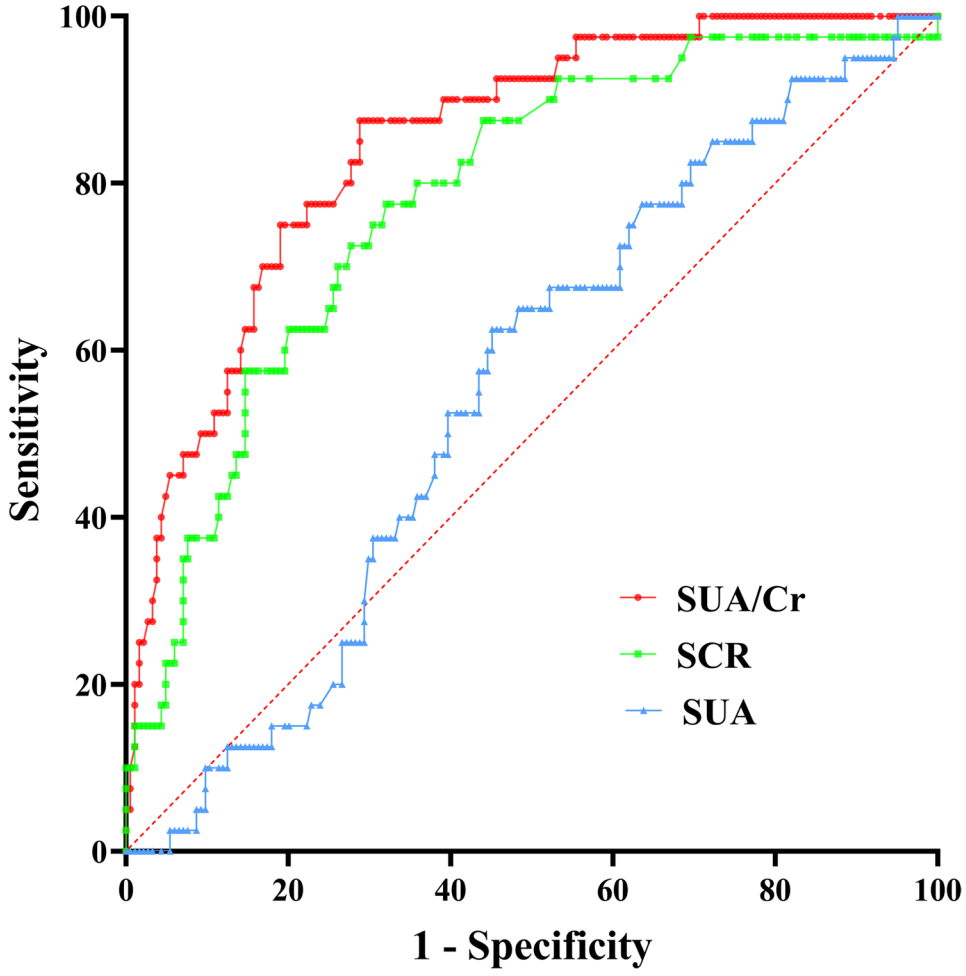

Fig. 3 Receiver operating characteristic curves of SUA, SCR, SUA/Cr for predicting in-hospital mortality in SAP patients. SAP, severe acute pancreatitis; SUA/Cr, serum uric acid to serum creatinine ratio

indicated that low SUA/Cr levels were are independently associated with an increased risk of in-hospital mortality among SAP patients. Additionally, we found that patients in the lower SUA/Cr group had a higher proportion of septicemia, MODS, and respiratory failure, as well as longer durations of ICU stay and hospital stay.

A retrospective study showed that high SUA levels at admission were independently associated with mortality and other adverse cardiovascular events during hospitalization [21]. Recently, a study involving 231 patients with chronic coronary syndrome showed no correlation between SUA and coronary artery disease [22]. The study by Hefei Tang et al. [23] on stroke patients showed that although there was a significant difference in the incidence of adverse outcomes (male < female), the association between low SUA and adverse outcomes was more significant in male patients and not significant in female patients. Additionally, studies have shown that even within the normal range, elevated SUA levels are significantly associated with hyperlipidemia and atherosclerosis [24]. In the univariate analysis of mortality in SAP patients in this study, the OR for SUA was 1.149 ($P < 0.001$), but in the multivariate analysis, adjusted for age, BMI, APACH-II and other confounding factors, the OR for SUA was 1.002 ($P = 0.601$), and the AUC was only 0.553, indicating poor predictive value of SUA for SAP patients.

SCR as a biomarker, it has been shown to be associated with the prognosis of various diseases. In the study of 5198 patients with acute heart failure, Cho YH et al. [25] found that there was a J-shaped relationship between SCR level and mortality, and both low and high levels were associated with increased mortality. In a multicenter study, 486 SAP patients were investigated, and SCR (OR = 1.003, $P = 0.017$) was determined to be an independent risk factor for 90 days death of patients, and also a biomarker of pancreatic necrosis [26]. However, Lankisch et al. found that any increase in SCR concentration within 48 h after admission is not a sign of pancreatic necrosis during the first episode of AP and is not

related to prognosis [27]. In this study, after adjusting for multiple confounders, the OR of SCR for mortality in SAP patients was 1.009 (P = 0.027), with an AUC of 0.782, indicating that SCR had a certain predictive value in patients with SAP.

Our results show that a lower SUA/Cr ratio is associated with worse clinical outcomes in patients with SAP (Table 3). Numerous studies have demonstrated that SUA exhibits direct antioxidant effects [28]. Lower SUA levels may reflect excessive activation of inflammatory responses, potentially increasing the risk of systemic inflammatory response syndrome (SIRS) and MODS [28, 29]. The accumulation of SCR in the human body not only leads to metabolic disturbances but also amplifies a series of inflammatory reactions [30]. In patients with SAP, oxidative stress serves as one of the key mechanisms contributing to pancreatic tissue damage and inflammatory responses [3]. A lower SUA/Cr may indicate insufficient antioxidant capacity in the body, which fails to effectively counteract oxidative stress, thereby exacerbating pancreatic tissue injury and inflammatory reactions. The reduced SUA/Cr may indirectly reflect decreased albumin levels [15], a finding corroborated by our research results presented in Table 2. Albumin possesses antioxidant and anti-inflammatory properties while maintaining plasma colloid osmotic pressure [31]. Its diminished levels could further aggravate inflammatory responses and tissue damage. These mechanisms collectively may explain why a lower SUA/Cr is associated with poorer clinical outcomes in SAP patients.

In a study conducted by D'Elia L et al. [32] involving 20,724 participants, it was shown that when using the first quintile of SUA/Cr as a reference, the other four groups exhibited an increased risk of cardiovascular events during follow-up, and the SUA/Cr was of greater value than uric acid alone in predicting CV risk. Similarly, the findings from Linlin Zhao et al. [33] demonstrated a positive correlation between SUA/Cr and the risk of metabolically unhealthy phenotypes in Chinese subjects, suggesting that it can serve as an independent risk predictor for the development of these phenotypes. In contrast, Tang et al. [14] investigated 15,269 hypertensive patients in the NHANES database and found that a lower SUA/Cr was associated with a higher risk of all-cause mortality and cardiovascular mortality among hypertensive adults. In our study, the mortality rate of patients in the low SUA/Cr group was 39.8%, while the high group was only 3.7%. After adjusting for multiple confounding factors, the Model II showed that the OR of the low SUA/Cr group was 8.027 (P < 0.001). When the SUA/Cr was entered as a continuous variable into model III, the OR = 0.456 (P = 0.018). The AUC for SUA/Cr in the ROC analysis was 0.849 (P < 0.001), indicating a predictive value superior to that of SUA or SCR alone. This study suggest that, in the clinical management of SAP, SUA/Cr can be used as a simple indicator for early prognosis assessment, which can help quickly identify high-risk mortality patients. For low ratio patients, more proactive monitoring measures, antioxidant intervention strategies, adjustment of fluid management, and intervention complications can be adopted to avoid missing the intervention window due to "observation waiting", which is expected to reduce in-hospital mortality and improve overall prognosis.

This study has several limitations. First, it is a single-center, retrospective study, and further prospective, multicenter, large-sample studies are needed to validate our findings. Second, we selected commonly available clinical data that are easily accessible at admission, which limited the clinical data available for analysis. Finally, the proportion of patients with diabetes and non-alcoholic fatty liver disease in our study population was higher. Therefore, the generalizability of the results to the general population is limited. Further investigation in larger populations is needed to confirm these findings.

Conclusion

In summary, our study results indicate that SAP patients in the low SUA/Cr group have worse prognosis and higher in-hospital mortality, and low SUA/Cr is independently associated with increased in-hospital mortality. Meanwhile, age, BMI, and MV are independent risk factors for in-hospital mortality. Therefore, early identification of these risk factors and the implementation of proactive interventions may help reduce mortality in SAP and improve patient outcomes.

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Authors' contributions

Conceptualization, Q.W. and Y.P.; data curation, J.J., and Y.C.; investigation, J.J., Y.P. and Y.L.; methodology, Q.W. and Y.C.; writing—original draft paper, Q.W. and Y.P.; writing—review and editing, Q.W. and Y.P.; supervision, Y.L. and L.C. All authors have read and agreed to the published version of the manuscript.

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Data availability

All data can be obtained from the corresponding author upon reasonable request.

Declarations

Ethics approval and consent to participate

This study adheres to the Helsinki Declaration and has obtained approval from the Medical Ethics Committee of Fujian Medical University Union Hospital, and has obtained exemption from informed consent (Approval No: 2024KY209).

Consent for publication

Patient information was hidden in our study.

Competing interests
The authors declare no competing interests.

Author details
[1]Department of Critical Care Medicine, Fujian Medical University Union Hospital, Fuzhou, Fujian, China
[2]Department of Nursing, Fujian Medical University Union Hospital, No.29 Xinquan Road, Fuzhou, Fujian Province 350001, China
[3]School of Nursing, Fujian Medical University, Fuzhou, Fujian, China
[4]Department of Cardiac Surgery, Fujian Medical University Union Hospital, No.29 Xinquan Road, Fuzhou, Fujian Province 350001, China
[5]Fujian Provincial Special Reserve Talents Laboratory, Fuzhou, Fujian, China

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References

1. 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(1):122–34.
2. Szatmary P, Grammatikopoulos T, Cai W, et al. Acute pancreatitis: diagnosis and treatment. Drugs. 2022;82(12):1251–76.
3. Boxhoorn L, Voermans RP, Bouwense SA, et al. Acute Pancreat Lancet. 2020;396(10252):726–34.
4. Li W, Ou L, Fu Y, Chen Y, Yin Q, Song H. Risk factors for concomitant infectious pancreatic necrosis in patients with severe acute pancreatitis: a systematic review and meta-analysis. Clin Res Hepatol Gastroenterol. 2022;46(5):101901.
5. Bang JY, Wilcox CM, Arnoletti JP, Varadarajulu S. Superiority of endoscopic interventions over minimally invasive surgery for infected necrotizing pancreatitis: meta-analysis of randomized trials. Dig Endosc. 2020;32(3):298–308.
6. Yang H, Cao R, Zhou F, et al. The role of interleukin-22 in severe acute pancreatitis. Mol Med. 2024;30(1):60.
7. Schepers NJ, Bakker OJ, Besselink MG, et al. Impact of characteristics of organ failure and infected necrosis on mortality in necrotising pancreatitis. Gut. 2019;68(6):1044–51.
8. Ames BN, Cathcart R, Schwiers E, Hochstein P. Uric acid provides an antioxidant defense in humans against oxidant- and radical-caused aging and cancer: a hypothesis. Proc Natl Acad Sci U S A. 1981;78(11):6858–62.
9. Johnson RJ, Bakris GL, Borghi C, et al. Hyperuricemia, acute and chronic kidney disease, hypertension, and cardiovascular disease: report of a scientific workshop organized by the National Kidney Foundation. Am J Kidney Dis. 2018;71(6):851–65.
10. Navaneethan SD, Mitch WE. Does serum creatinine reflect muscle mass in patients with kidney failure? J Am Soc Nephrol. 2024;35(1):1–2.
11. Palevsky PM, Liu KD, Brophy PD, et al. KDOQI US commentary on the 2012 KDIGO clinical practice guideline for acute kidney injury. Am J Kidney Dis. 2013;61(5):649–72.
12. Wang Q, Wang Z, Sun Y, et al. The serum uric acid to serum creatinine ratio is an independent risk factor for type 2 diabetes in community-dwelling elderly: a longitudinal analysis. Nutr Metab Cardiovasc Dis. 2024;34(12):2749–56.
13. Zhang D, Liu Z, Guo W et al. Association of serum uric acid to serum creatinine ratio with 1-year stroke outcomes in patients with acute ischemic stroke: A multicenter observational cohort study. Eur J Neurol. 2024;31(12):e16431.
14. Tang Z, Liu H, Ding Y, Yuan C, Shao Y. Association between serum uric acid to serum creatinine ratio with cardiovascular and all-cause mortality in adults with hypertension. Sci Rep. 2024;14(1):18008.
15. Jiang L, Jin J, He X, et al. The association between serum uric acid / serum creatinine ratio and in-hospital outcomes in elderly patients with acute myocardial infarction. BMC Cardiovasc Disord. 2024;24(1):52.
16. 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(6):713–e7227.
17. Li L, Wu H, Yin H, et al. Serum uric acid concentration in acute pancreatitis is significantly higher than healthy population. Sci Rep. 2024;7(1):18279.
18. Kashani K, Rosner MH, Ostermann M.Creatinine: From physiology to clinical application. Eur J Intern Med. 2020;72:9–14.
19. Kidney Disease: Improving Global Outcomes (KDIGO) Glomerular Diseases Work Group. KDIGOClinical Practice Guideline for the Management of Glomerular Diseases. Kidney Int. 2021;100(4S):S1-S276.
20. Mullen JT, Ribero D, Reddy SK et al. Hepatic insufficiency and mortality in 1,059 noncirrhotic patients undergoing major hepatectomy. J Am Coll Surg. 2007;204(5):854–62.
21. Centola M, Maloberti A, Castini D, et al. Impact of admission serum acid uric levels on in-hospital outcomes in patients with acute coronary syndrome. Eur J Intern Med. 2020;82:62–7.
22. Maloberti A, Bossi I, Tassistro E, et al. Uric acid in chronic coronary syndromes: relationship with coronary artery disease severity and left ventricular diastolic parameter. Nutr Metab Cardiovasc Dis. 2021;31(5):1501–8.
23. Tang H, Mo J, Chen Z, et al. Uric acid contributes to obesity-paradox of the outcome of ischemic stroke. Front Neurol. 2019;10:1279.
24. Lima WG, Martins-Santos MES, Chaves VE. Uric acid as a modulator of glucose and lipid metabolism. Biochimie. 2015;116:17–23.
25. Cho Y-H, Park JJ, Lee H-Y et al. J-shaped relationship between serum creatinine and mortality in Korean patients with acute heart failure. Clin Res Cardiol. 2025;114(10):1280–9.
26. Li J, Gao J, Huang M, Fu X, Fu B. Risk factors for death in patients with severe acute pancreatitis in Guizhou Province, China. Gastroenterol Res Pract. 2024;2024:8236616.
27. Lankisch PG, Weber-Dany B, Maisonneuve P, Lowenfels AB. High serum creatinine in acute pancreatitis: a marker for pancreatic necrosis? Am J Gastroenterol. 2010;105(5):1196–200.
28. Mijailovic NR, Vesic K, Borovcanin MM. The influence of serum uric acid on the brain and cognitive dysfunction. Front Psychiatry. 2022;13:828476.
29. Zhang Z, Huang Y, Zhou S, et al. Effect of serum antioxidants on cognitive dysfunction in first-episode and drug-naïve patients with major depressive disorder. J Affect Disord. 2025;372:533–9.
30. Wyss M, Kaddurah-Daouk R. Creatine and creatinine metabolism. Physiol Rev. 2000;80(3):1107–213.
31. Ikizler TA. Nutrition, inflammation and chronic kidney disease. Curr Opin Nephrol Hypertens. 2008;17(2):162–7.
32. D'Elia L, Masulli M, Cirillo P et al. Serum uric acid/Serum creatinine ratio and cardiovascular mortality in diabetic Individuals-The uric acid right for heart health (URRAH) project. Metabolites. 2024;14(3):164.
33. Zhao L, Qiu X. Higher ratio of serum uric acid to serum creatinine (SUA/SCr) increases the risk of metabolic unhealthy phenotype. Nutr Metab Cardiovasc Dis. 2023;33(10):1981–8.

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