Chen *et al.*
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Establishment and validation of a prediction model for acute kidney injury in moderate severe and severe acute pancreatitis patients

Wen-Jie Chen[1†], Qin-Yue Su[1,2†], Ming Zhong[3†], Yan-Jun Zheng[1], Xiao-Feng Wang[1], Hong-Ping Qu[3], En-Qiang Mao[1], Zhi-Tao Yang[1], Er-Zhen Chen[1,4*] and Ying Chen[1,5*]

Abstract

Purpose This study aimed to develop a nomogram for predicting acute kidney injury (AKI) in patients with moderate severe acute pancreatitis (MSAP) and severe acute pancreatitis (SAP).

Methods This study enrolled a total of 1,077 patients with MSAP and SAP, categorizing them into three groups: training ($n=646$), internal validation ($n=278$), and external validation ($n=153$). In the training cohort, logistic regression analysis identified independent predictors of AKI in patients with MSAP and SAP. A nomogram was developed based on these independent predictors. The model's performance was assessed using the receiver operating characteristics (ROC) curve, precision–recall (PR) curve, calibration curve, and decision curve analysis (DCA).

Results The incidence rates of AKI in the training set, internal validation set, and external validation set were 32.82%, 32.01%, and 27.45%, respectively. Independent predictors of AKI in patients with MSAP and SAP included: shock index (odds ratio [OR] = 7.42, 95% confidence interval [CI] 2.18–25.19), blood urea nitrogen (OR = 1.32, 95% CI 1.22–1.43), uric acid (OR = 1.002, 95% CI 1.000–1.003), serum calcium (OR = 0.38, 95% CI 0.18–0.79), triglycerides (OR = 1.02, 95% CI 1.004–1.041), hematocrit > 0.5 (OR = 3.24, 95% CI 1.10–9.59), serum sodium < 135 mmol/L (OR = 2.01, 95% CI 1.15–3.49), creatine kinase isoenzyme > 4 ng/mL (OR = 2.61, 95% CI 1.48–4.61), and thrombin time < 14 s (OR = 2.83, 95% CI 1.28–6.27). In the training, internal validation, and external validation sets, the areas under the ROC curves for the nomogram were 0.841, 0.789, and 0.853, respectively. Similarly, the areas under the PR curves were 0.807, 0.733, and 0.770. The calibration curves demonstrated that the predicted outcomes were well-aligned with the actual results. The decision curve analysis (DCA) indicated that the model had satisfactory clinical applicability.

Conclusions Nine indicators have been identified as independent predictors of AKI in patients with MSAP and SAP. The developed nomogram exhibits robust predictive capability and shows promise for clinical application.

Keywords Acute pancreatitis, Acute kidney injury, Predictor, Nomogram

[†]Wen-Jie Chen, Qin-Yue Su and Ming Zhong have contributed equally to this work.

*Correspondence:
Er-Zhen Chen
chenerzhen@hotmail.com
Ying Chen
bichatlion@163.com
Full list of author information is available at the end of the article

Introduction

Acute pancreatitis (AP) is a prevalent gastrointestinal disorder often encountered in emergency departments. According to the latest Atlanta classification, AP is categorized into three forms: mild acute pancreatitis (MAP), moderate severe acute pancreatitis (MSAP), and severe acute pancreatitis (SAP) [1]. Among the complications associated with AP, acute kidney injury (AKI) poses a significant challenge, particularly in patients with MSAP and SAP. Research indicates that the incidence of AKI in AP patients can reach up to 7.9%, with mortality rates ranging from 25 to 75%, depending on the severity of the condition [2, 3]. These alarming statistics underscore the urgent need for early prediction of AKI in AP patients, as prompt identification and intervention could significantly improve clinical outcomes.

To effectively predict AKI in patients with AP, it is essential to identify independent risk factors associated with its development. Clinical studies have identified chronic alcohol consumption and hypertriglyceridemia as significant independent risk factors contributing to the onset of AKI in AP patients [4, 5]. Beyond these traditional risk factors, the investigation of novel biomarkers presents a promising opportunity to improve the predictive accuracy for AKI in AP cases. Biomarkers such as serum cystatin C and urinary neutrophil gelatinase-associated lipocalin have demonstrated potential in predicting the occurrence of AKI in this population [6, 7]. In addition, kidney injury molecule-1 and the systemic inflammatory index (SII) are emerging as valuable indicators that could assist in the early detection of AKI among AP patients [8, 9].

Imaging modalities, especially computed tomography (CT), play a crucial role in diagnosing AP and offer valuable predictive insights into the risk of AKI. Various CT scoring systems have been developed to assess pancreatic inflammation and related complications. Notably, the modified renal rim grade and the extra-pancreatic inflammation CT score have shown promise in predicting the simultaneous occurrence of AKI in patients with SAP [10]. These imaging scores offer significant insights into the severity of pancreatitis, which may correlate with the risk of renal impairment.

As previously stated, multiple indicators can predict the occurrence of AKI in patients with AP. In recent years, significant progress has been made in developing prediction models for AP combined with AKI. However, these models still face substantial challenges. First, most AP–AKI prediction models rely on public databases, leading to inconsistent predictive performance and a general lack of external validation. Second, many clinical studies struggle with small sample sizes and complex clinical indicators, which limit the scalability and applicability of these models. To address these issues, this study analyzed data from a large cohort of MSAP and SAP patients admitted to the Emergency Intensive Care Unit (EICU) of Ruijin Hospital, affiliated with Shanghai Jiao Tong University School of Medicine. By using commonly available clinical indicators, we developed a predictive model for AKI in MSAP and SAP patients. The model's performance and clinical utility were systematically evaluated and externally validated, aiming to fill the gaps in existing research.

Materials and methods

Study design

This retrospective observational study encompassed 924 patients diagnosed with MSAP and SAP, who were admitted to the EICU at Ruijin Hospital, School of Medicine of Shanghai Jiao Tong University, between January 1, 2015 and October 31, 2023. The participants were divided into a training set and an internal validation set, following a 7:3 ratio, to facilitate model construction and internal validation. Furthermore, patients with MSAP and SAP who were admitted to the Northern District of Ruijin Hospital from 2021 to 2023 were incorporated for the purpose of external validation. Relevant data were gathered for comprehensive statistical analysis.

Patients

The inclusion criteria were as follows: (1) a diagnosis of MSAP and SAP, with diagnostic criteria for AP involving at least two of the following: persistent upper abdominal pain, elevated amylase and lipase levels exceeding three times the normal limit, and imaging evidence of AP. Patients with AP exhibiting transient (<48 h) or persistent (≥48 h) organ dysfunction, or local or systemic complications of pancreatitis, are diagnosed with MSAP and SAP; (2) age≥18 years; and (3) hospital stay of at least 48 h. The exclusion criteria were as follows: (1) time from onset of symptoms to admission exceeding 7 days; (2) chronic kidney failure; and (3) serum creatinine tested fewer than two times.

Data collection

After identifying the target population, the clinical materials were collected as follows: (1) demographic characteristics: age and gender; (2) vital signs upon admission: body mass index (BMI), respiratory rate (RR), body temperature (T) and shock index (heart rate to systolic blood pressure ratio); (3) laboratory results within 24 h of admission: white blood cell count (WBC), neutrophil, lymphocyte, hemoglobin (HB), hematocrit (HCT), platelet (PLT), NLR (neutrophil to lymphocyte ratio), PLR (platelet to lymphocyte ratio), systemic inflammatory index (SII, neutrophil * platelet to lymphocyte ratio),

C-reactive Protein (CRP), glucose, lactate, alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), γ-glutamyl transpeptidase (γ-GT), albumin (ALB), prealbumin, total bilirubin (TB), direct bilirubin (DB), serum creatinine (SCr), blood urea nitrogen (BUN), uric acid (UA), creatine kinase isoenzyme (CK-MB), myoglobin (Myo), troponin I (TnI), serum sodium (Na^+), serum potassium (K^+), serum chlorine (Cl^-), serum phosphorus (P), serum calcium (Ca^{2+}), pH, base excess (BE), arterial partial pressure of oxygen (PaO_2), arterial partial pressure of carbon dioxide ($PaCO_2$), triglyceride (TG), total cholesterol (TC), activated partial thromboplastin time (APTT), prothrombin time (PT), thrombin time (TT), fibrinogen (Fg), fibrin degradation product (FDP-1), D-dimer, procalcitonin (PCT); and (4) prognosis status: survival status and hospital time.

The primary focus of this study was the incidence of AKI in patients with MSAP and SAP. The diagnosis of AKI adhered to the 2012 kidney disease: improving global outcomes (KDIGO) criteria [11]: an increase in SCr by at least 26.5 μmol/L within 48 h, a rise in SCr to at least 50% above the baseline within 1 week, or urine output of less than 0.5 mL/kg/h sustained for more than 6 h.

Statistical analysis

Patients with MSAP and SAP who met the inclusion criteria in the EICU of Ruijin Hospital were divided into training and internal validation sets in a 7:3 ratio. Patients from the Northern District of Ruijin Hospital who met the same criteria constituted the external validation set. The training set was used to develop a predictive model for AKI, while the internal and external validation sets were used to verify the model's effectiveness. In the data collected from the EICU at Ruijin Hospital, variables with over 20% missing data were excluded, and those with less than 20% missing data were supplemented with either median or mean values, depending on their distribution. For data from the Northern District of Ruijin Hospital, missing data were handled using multiple imputation. Continuous variables were presented as medians with quartiles, while categorical variables were expressed as percentages. Continuous variables were compared using the Mann–Whitney *U* test, and categorical variables were compared using Pearson's chi-squared or Fisher's exact tests. All variables were initially analyzed through univariate logistic regression, and those with a p value < 0.05 were further analyzed using multivariate logistic regression. Variables that maintained a p value < 0.05 in the multivariate analysis were identified as independent predictors of AKI in MSAP and SAP patients, which were subsequently used to construct the nomogram. The area under the curve (AUC) of the receiver operating characteristic (ROC) curve assessed the nomogram's discriminatory power. DeLong's test was employed to determine the statistical significance of differences between the ROC curves of the two cohorts. In addition, the area under the precision–recall (PR) curve (AUC) evaluated the classification performance of the nomogram. Calibration curves were drawn to assess the alignment between observed results and predicted outcomes. Decision curve analysis (DCA) was performed to explore the clinical applicability of the predictive model.

The analyses were conducted using SPSS 25 and R version 4.2.3, with a p value of less than 0.05 being deemed statistically significant.

Results

Baseline characteristics of patients

As shown in Fig. 1, the study enrolled 924 MSAP and SAP patients admitted to the EICU at Ruijin Hospital. These patients were categorized into a training cohort (n = 624, 70%) and a validation cohort (n = 278, 30%). The overall incidence of AKI was 32.6%, with stages 1, 2, and 3 accounting for 53.49%, 16.61%, and 29.9%, respectively (Supplementary Table S1). In addition, 153 patients with MSAP and SAP from the Northern District of Ruijin Hospital were included for external validation, with an AKI incidence of 27.45%. For this group, AKI grades 1, 2, and 3 were 47.62%, 7.14%, and 45.24%, respectively. No statistically significant differences were observed between the training and internal validation sets with respect to basic clinical information, laboratory results, and clinical progression (p > 0.05). However, certain characteristics exhibited statistically significant differences between the training and external validation sets, suggesting the model's strong generalizability, as evidenced by its accurate prediction of AKI during external validation.

Logistic regression analysis between AKI and potential predictors

The results of the univariate logistic regression analysis are detailed in Table 1. Indicators with a p value of less than 0.05 were selected for subsequent multivariate logistic regression analysis. This analysis identified several independent risk factors for AKI in patients with MSAP and SAP. The factors include an elevated shock index (Odds Ratio [OR] = 7.42, 95% Confidence Interval [CI] 2.18–25.19, p = 0.001), increased BUN (OR = 1.32, 95% CI 1.22–1.43, p < 0.001), elevated UA (OR = 1.002, 95% CI 1.000–1.003, p = 0.05), decreased serum calcium (OR = 0.38, 95% CI 0.18–0.79, p = 0.01), increased TG (OR = 1.02, 95% CI 1.004–1.041, p = 0.017), HCT greater than 0.5 (OR = 3.24, 95% CI 1.10–9.59, p = 0.034), serum sodium less than 135 mmol/L (OR = 2.01, 95% CI 1.15–3.49, p = 0.014), CK–MB greater than 4 ng/ml (OR = 2.61,

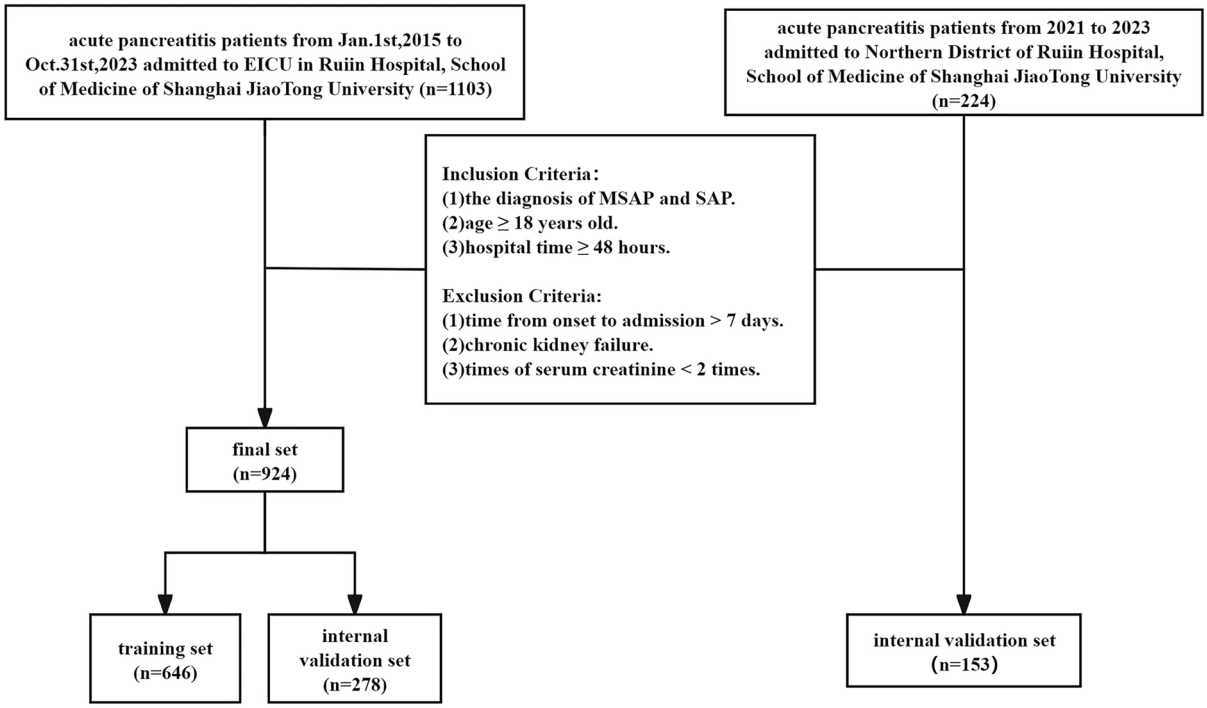

Fig. 1 Flowchart for patient selection

95% CI 1.48–4.61, $p<0.001$), and TT less than 14 s (OR = 2.83, 95% CI 1.28–6.27, $p=0.01$), as reflected in Table 2 with p values less than 0.05.

Figure 2 illustrates a nomogram created from the previously identified independent predictors to predict the onset of AKI in patients with MSAP and SAP.

Evaluation and validation of the nomogram

The ROC curves of the prediction model are displayed in Fig. 3a for the training group (AUC = 0.842, 95% CI 0.805–0.878), the internal validation group (AUC = 0.789, 95% CI 0.725–0.852), and the external validation group (AUC = 0.853, 95% CI 0.780–0.926). These results demonstrate the nomogram's robust predictive capability in identifying AKI in MSAP and SAP patients, as evidenced by the AUC values exceeding 0.75. Delong's test results indicate no statistically significant difference between the ROCs of the internal and external validation groups in comparison with the ROC of the training set ($p>0.05$). Figure 3b illustrates the PR curves for the prediction model in the training group (AUC = 0.807), the internal validation group (AUC = 0.733), and the external validation group (AUC = 0.770), underscoring the nomogram's effective ability to distinguish between AKI and non-AKI cases and its robust classification performance (AUCs > 0.70). The calibration curves presented in Fig. 3c for the training, internal validation, and external validation sets reveal that the predicted outcomes align closely with the actual observations, indicating the model's strong calibration capability.

Clinical applicability of the prediction model

The DCA curve evaluated the clinical applicability of the prediction model for AKI in patients with MSAP and SAP. As illustrated in Fig. 3d, the DCA curve for the training group suggested that a positive net benefit would be achieved from this prediction model when the probability threshold surpassed approximately 10%. For the internal validation group, the DCA indicated a positive net benefit with a probability threshold exceeding approximately 12.5%, while thresholds below the previously mentioned level yielded no or negative net benefits. Moreover, the external validation group showed a positive net benefit with a probability threshold exceeding about 5%.

Discussion

Patients with MSAP and SAP often experience transient or persistent organ dysfunction, which frequently requires treatment and management in the intensive care unit (ICU). While MAP is usually self-limiting and rarely results in AKI, MSAP and SAP demand considerable clinical attention. In this study, the incidence of AKI among patients with MSAP and SAP was approximately

Table 1 Results of univariate logistic regression analysis examining the relationship between variables and the occurrence of AKI in patients with MSAP and SAP within the training cohort

Variables	*p* value	Odd ratio	95% Confidence interval
Male	0.131	1.31	0.92–1.87
Age, (years)	0.045	1.01	1.00–1.02
Hypertension	0.012	1.55	1.10–2.19
Diabetes	0.389	1.17	0.82–1.69
Circulatory disease	0.867	0.94	0.47–1.91
CKD	0.204	1.74	0.74–4.10
Cancer history	0.452	0.68	0.24–1.88
Smoking	0.190	1.25	0.90–1.74
Drinking	0.280	1.20	0.86–1.69
AP etiology			
Biliary AP			Reference
Hyperlipidemic AP	0.484	0.87	0.59–1.29
Alcoholic AP	0.921	0.97	0.48–1.94
Other	0.094	0.53	0.25–1.12
Mixed AP	0.067	1.59	0.97–2.59
BMI, (kg/m^2)	0.411	1.02	0.98–1.05
Vital sign			
RR, (bpm)	< 0.001	1.05	1.02–1.07
T, (℃)	0.777	1.03	0.82–1.30
Shock Index	< 0.001	17.63	7.27–42.79
Laboratory result			
WBC, (10^9/L)			
[4, 12]			Reference
< 4 or > 12	0.054	0.72	0.52–1.01
NLR	0.277	1	1.00–1.01
PLR	0.155	1	1.00–1.00
SII	0.264	1	1.00–1.00
Hb, (g/L)			
[120, 160]			Reference
< 120	0.158	1.35	0.89–2.05
> 160	0.04	2.12	1.27–3.53
HCT			
[0.3, 0.5]			Reference
< 0.3	0.005	2.19	1.26–3.82
> 0.5	< 0.001	5.38	2.18–13.32
PLT, (10^9/L)			
[150, 300]			Reference
< 150	< 0.001	2.05	1.45–2.88
> 300	0.449	1.34	0.63–2.85
CRP, (mg/L)	< 0.001	1.004	1.002–1.005
Glucose, (mmol/L)	< 0.001	1.08	1.04–1.12
Lactate, (mmol/L)	0.002	1.24	1.08–1.42
PCT, (ng/mL)	< 0.001	1.11	1.07–1.14
ALT, (IU/L)	0.16	1.001	1.000–1.003
AST, (IU/L)	< 0.001	1.003	1.001–1.005
ALP, (IU/L)	0.62	0.996	0.996–1.003
γ-GT, (IU/L)	0.989	1	0.999–1.001
Alb, (g/L)	0.002	0.95	0.92–0.98
Prealbmin, (mg/L)	0.143	0.998	0.995–1.001
TB, (μmol/L)	0.027	1.01	1.001–1.013
DB, (μmol/L)	0.001	1.02	1.01–1.03
SCr, (μmol/L)	< 0.001	1.36	1.27–1.44
BUN, (mmol/L)	< 0.001	1.005	1.004–1.006
Na^+, (mmol/L)			
[135, 145]			Reference
< 135	0.021	1.61	1.07–2.42
> 145	< 0.001	3.3	1.85–5.88
K^+, (mmol/L)			
[3.5, 5.5]			Reference
< 3.5	0.002	0.49	0.31–0.76
> 5.5	0.003	5.6	1.78–17.62
Cl^-, (mmol/L)			
[96, 108]			Reference
< 96	0.279	1.66	0.66–4.14
> 108	< 0.001	2.33	1.56–3.48
P < 0.8 mmol/L	0.046	0.71	0.51–0.99
Ca^{2+}, (mmol/L)	< 0.001	0.12	0.07–0.23
CK-MB > 4 ng/mL	< 0.001	5.78	3.75–8.90
MYO > 70 ng/mL	< 0.001	7.13	4.84–10.51
TnI > 30 pg/mL	< 0.001	2.58	1.82–3.65
pH			
[7.35, 7.45]			Reference
< 7.35	< 0.001	2.27	1.58–3.28
> 7.45	0.255	0.61	0.26–1.43
PaO_2, (Kpa)	0.472	1.01	0.98–1.05
$PaCO_2$, (Kpa)	0.017	0.8	0.67–0.96
BE, (mmol/L)			
[− 3, 3]			Reference
> 3	0.78	0.8	0.17–3.81
< − 3	< 0.001	2.54	1.77–3.63
TG, (mmol/L)	0.009	1.02	1.01–1.03
TC, (mmol/L)	0.674	1.01	0.97–1.05
APTT, (s)			
[35, 45]			Reference
< 35	0.06	0.69	0.47–1.02
> 45	< 0.001	4.07	1.92–8.65
PT, (s)			
[10, 16]			Reference
> 16	< 0.001	4.53	2.70–7.62
TT, (s)			
[14, 21]			Reference
< 14	0.43	1.32	0.67–2.60
> 21	< 0.001	2.71	1.64–4.47

Table 1 (continued)

Variables	*p* value	Odd ratio	95% Confidence interval
Fg, (g/L)			
[1.8, 3.5]			Reference
> 3.5	0.54	1.15	0.74–1.78
< 1.8	0.25	2.31	0.55–9.78
FDP-1, (mg/L)	< 0.001	1.02	1.01–1.04
D-Dimer, (mg/L)	< 0.001	1.09	1.05–1.12

Table 2 Multivariate logistic regression analysis results for variables associated with the occurrence of AKI in MSAP and SAP patients within the training cohort

Variables	*p* value	Odd ratio	95% Confidence interval
Shock Index	0.001	7.42	2.18–25.19
BUN, (mmol/L)	< 0.001	1.32	1.22–1.43
UA, (mmol/L)	0.05	1.002	1.000–1.003
Ca^{2+}, (mmol/L)	0.01	0.38	0.18–0.79
TG, (mmol/L)	0.017	1.02	1.004–1.041
HCT			
[0.3, 0.5]			Reference
< 0.3	0.096	1.93	0.89–4.20
> 0.5	0.034	3.24	1.10–9.59
Na^{+}, (mmol/L)			
[135, 145]			Reference
< 135	0.014	2.01	1.15–3.49
> 145	0.222	1.65	0.74–3.69
CK-MB > 4 ng/mL	0.001	2.61	1.48–4.61
TT, (s)			
[14, 21]			Reference
< 14	0.01	2.83	1.28–6.27
> 21	0.321	1.44	0.70–2.95

30%, and these cases were associated with high mortality rates and extended hospital stays. A multicenter retrospective study found that the incidence of AKI in SAP patients admitted to the ICU can surge to 69.3%, with a mortality rate reaching 55.6%, especially in those diagnosed with AKI stage 3 [12]. Consequently, the early prediction of AKI in patients with MSAP and SAP is crucial, as it can enable clinicians to implement preventive strategies or timely medical interventions. This proactive approach has the potential to reduce mortality rates and alleviate the associated healthcare burden.

Utilizing univariate and multivariate logistic regression analyses, the study identified several independent predictors of AKI in patients with MSAP and SAP. These predictors include shock index, BUN, UA, serum calcium, TG, HCT greater than 0.5, sodium levels less than 135 mmol/L, CK–MB exceeding 4 ng/mL, and TT below 14 s. These indicators were harnessed to develop a predictive model for AKI in patients with MSAP and SAP, which demonstrated commendable predictive performance and clinical applicability.

The shock index is a well-regarded indicator for evaluating hemodynamics and gauging the severity of blood volume deficiency [13]. Insufficient blood volume is a frequent cause of AKI in patients with AP [14]. In this study, the shock index proved to be an independent predictor of AKI in these patients. Furthermore, a study by Risinger et al. indicated that the shock index could also predict oliguria in post-traumatic patients with AKI. Beyond forecasting AKI, the shock index serves as an indicator of mortality in critically ill patients, including those with sepsis, trauma, or acute heart failure [15–17]. The potential importance of the shock index should not be underestimated. In the early stages of AP, elevated HCT levels suggest hemoconcentration and a decrease in effective circulating blood volume, which could cause red blood cells to become trapped in the kidneys, leading to renal dysfunction [18]. However, there is ongoing controversy regarding the extent to which changes in HCT can predict the onset of AKI. This study found that patients with HCT levels below 0.3 or above 0.5 were more likely to develop AKI, with HCT > 0.5 identified as an independent predictor for AKI in AP patients. Hemodilution and hypotension during cardiac surgery are common contributors to AKI post-surgery, and decreased hematocrit is associated with an increased risk of AKI [19]. In addition, another study revealed that Dengue patients with HCT levels exceeding 44% were at a higher risk of developing AKI [20].

BUN and UA are traditional yet critical markers of kidney function. BUN is frequently assessed alongside SCr to evaluate renal performance; however, its levels can be influenced by numerous factors. Despite its limited diagnostic value, BUN remains vital for constructing prediction models for AKI. While hyperuricemia is recognized as an independent risk factor for sepsis-associated and contrast-induced AKI [21, 22], its connection to AKI in patients with AP has not been clearly established. This study suggests that elevated uric acid levels may increase the incidence of AKI in individuals with pancreatitis.

Electrolyte imbalances are frequent clinical complications in both AP and AKI. This study identifies hyponatremia as an independent risk factor for MSAP and SAP when accompanied by AKI. Previous research [23, 24] indicates that dysnatremia often results from improper diuretic use and that pre-existing hyponatremia is closely

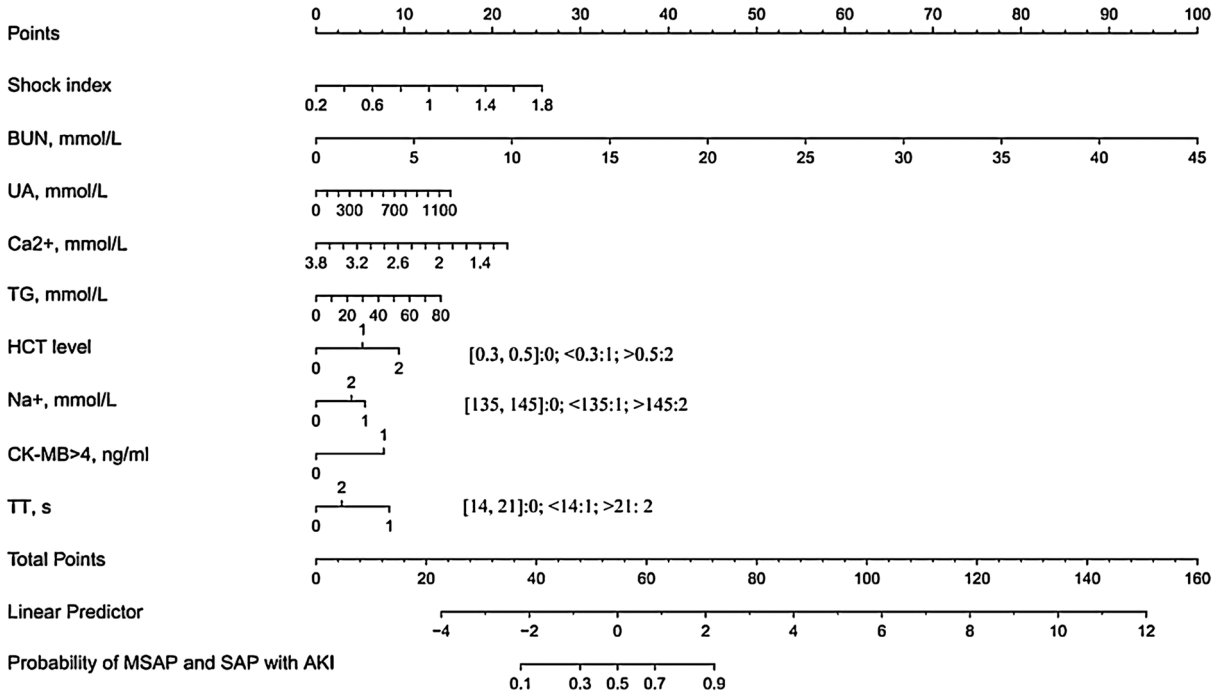

Fig. 2 Nomogram of the predictive model for AKI in patients with MSAP and SAP

linked to the development of hospitalization-related AKI. These findings suggest that certain clinical interventions might contribute to the onset of AKI.

Hypocalcemia is commonly observed in critically ill patients [25], especially those suffering from AP. Although serum calcium levels can indicate the severity of AP [26], there is no substantial evidence to suggest that they predict renal impairment. In this study, hypocalcemia was identified as an independent risk factor in MSAP and SAP with AKI. Both hypocalcemia and hypercalcemia were linked to an increased incidence of AKI in these patients. Charat Thongprayoon et al. demonstrated that abnormal calcium levels at admission elevate the risk of AKI, with persistent hypercalcemia potentially resulting in calcium deposition in the kidneys, thereby causing renal dysfunction [27]. CK–MB, primarily synthesized by cardiomyocytes, acts as a biomarker for the early diagnosis of acute myocardial infarction. In cases of acute pancreatitis, inflammation and coronary hypoperfusion may lead to elevated CK–MB levels, although its connection to renal insufficiency remains uncertain.

Hypertriglyceridemia is a common cause of acute pancreatitis. A retrospective study by Wu et al. identified hypertriglyceridemia as an independent risk factor for early AKI in patients with AP, likely due to triglyceride accumulation in the kidneys [28]. While a shortened TT is generally considered clinically insignificant and tied to elevated fibrinogen levels, this study discovered that a TT greater than 21 s was associated with AKI. Meanwhile, a TT less than 14 s emerged as an independent risk factor for MSAP and SAP with AKI. Some studies indicates that an elevated TT is independently linked to AKI in the context of sepsis [29, 30]. Sörensen-Zender et al. [31]emphasized the complex role of fibrinogen in acute ischemic kidney injury, revealing that reducing excess fibrinogen can enhance renal function. This finding may shed light on the current study's results. Consequently, further investigation into fibrinogen's role in AKI among patients with AP is warranted.

Few studies have concentrated on developing predictive models for AKI in patients with AP. Dongliang Yang et al. [32] created a model aimed at predicting MSAP and SAP with AKI, using indicators, such as CRP, intra-abdominal pressure, and Cystatin C, which demonstrated strong predictive capability. However, the necessity for experienced physicians to measure intra-abdominal pressure restricts the model's practicality. Another prediction model for AKI in AP patients [33] failed to show significant improvement in discrimination compared to the nomogram used in this study. The predictors incorporated into this study's nomogram are both objective and readily obtainable, enhancing its excellent predictive performance and applicability in clinical settings.

This study had several limitations. First, it was a single-center, retrospective observational study. AP is a rapidly developing disease, and AKI often occurs in the early stage

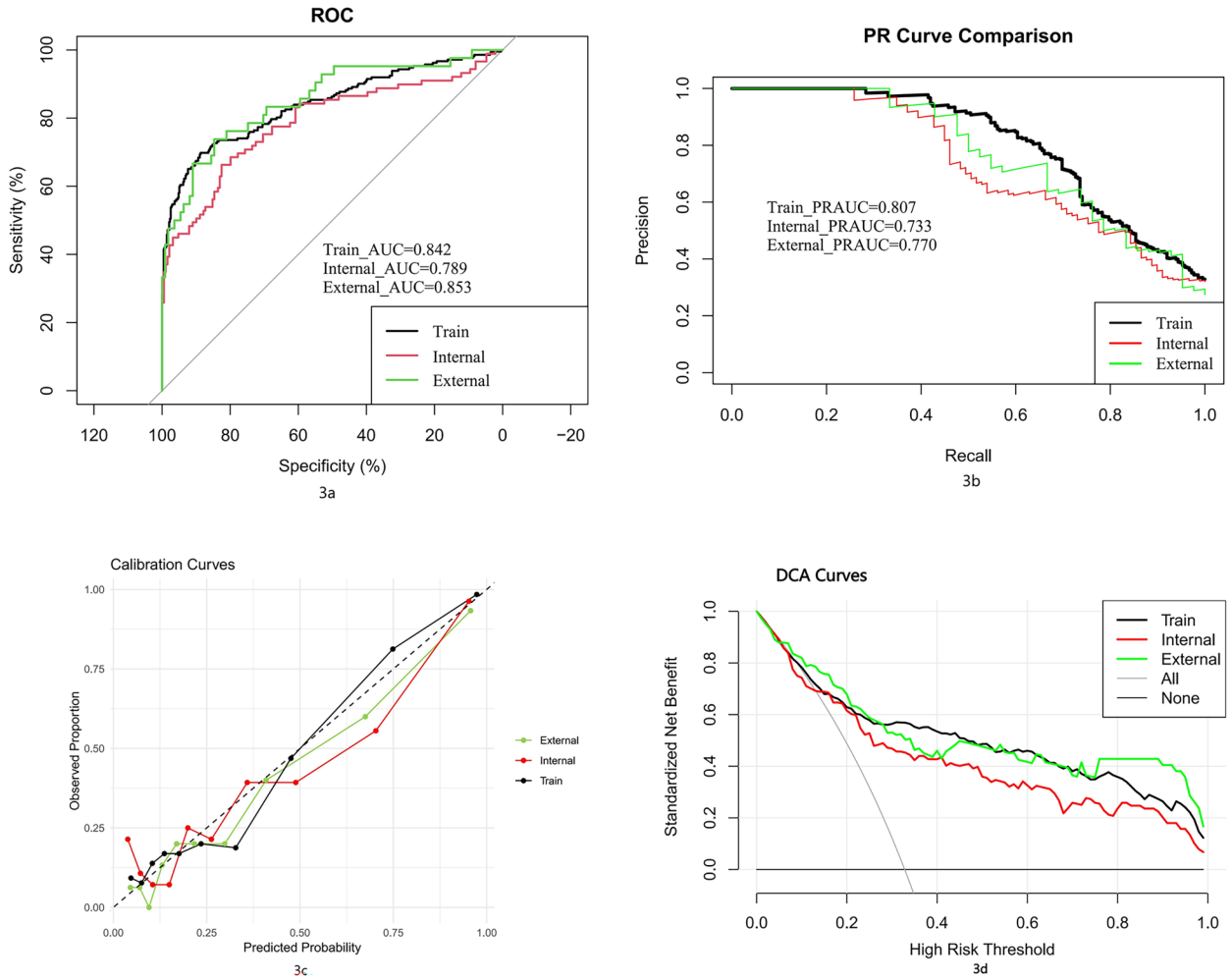

Fig. 3 Evaluation and validation of the predictive model. **a** ROC curves of the predictive model in the training set, internal validation set, and external validation set, all displaying AUCs greater than 0.75. **b** PR curves of the predictive model in the training, internal validation, and external validation sets, all exhibiting AUCs exceeding 0.70. **c** Calibration curves of the predictive model in the training, internal validation, and external validation sets, all demonstrating a high degree of calibration. **d** DCA curves for the predictive model in the training, internal validation, and external validation sets, all indicating robust clinical applicability

of the disease. Patients may have already experienced AKI upon admission and we cannot ignore this part of patients. Consequently, we could only examine the correlation between predictive indicators and AKI, a limitation commonly found in most predictive models for AKI in critically ill patients. Second, we excluded indicators with over 20% missing data, potentially overlooking some relevant factors. Finally, although the external validation showed the model's strong predictive capability, further expanding the sample size is necessary because of the limited number of participants.

Conclusion

Nine indicators have been identified as independent predictors of AKI in patients with MSAP and SAP. The developed nomogram exhibits robust predictive performance for AKI in these patient groups, making it clinically applicable.

Abbreviations

AP	Acute pancreatitis
MAP	Mild acute pancreatitis
MSAP	Moderate severe acute pancreatitis
SAP	Severe acute pancreatitis
AKI	Acute kidney injury
SII	Systemic inflammatory index
CT	Computed tomography
EICU	Emergency intensive care unit
BMI	Body mass index
RR	Respiratory rate
T	Body temperature

WBC	White blood cell count
HB	Hemoglobin
HCT	Hematocrit
PLT	Platelet
NLR	Neutrophil to lymphocyte ratio
PLR	Platelet to lymphocyte ratio
CRP	C-reactive protein
ALT	Alanine aminotransferase
AST	Aspartate aminotransferase
ALP	Alkaline phosphatase
Γ-GT	γ-Glutamyl transpeptidase
ALB	Albumin
TB	Total bilirubin
DB	Direct bilirubin
SCr	Serum creatinine
BUN	Blood urea nitrogen
UA	Uric acid
CK-MB	Creatine kinase isoenzyme
Myo	Myoglobin
TnI	Troponin I
Na^+	Serum sodium
K^+	Serum potassium
Cl^-	Serum chlorine
P	Serum phosphorus
Ca^{2+}	Serum calcium
BE	Base excess
PaO_2	Arterial partial pressure of oxygen
$PaCO_2$	Arterial partial pressure of carbon dioxide
TG	Triglyceride
TC	Total cholesterol
APTT	Activated partial thromboplastin time
PT	Prothrombin time
TT	Thrombin time
Fg	Fibrinogen
FDP-1	Fibrin degradation product
PCT	Procalcitonin
KDIGO	Kidney disease: improving global outcomes
AUC	Area under the curve
ROC	Receiver operating characteristic
PR	Precision–recall
DCA	Decision curve analysis
OR	Odds ratio
CI	Confidence interval
ICU	Intensive care unit

Supplementary Information

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Supplementary Material 1.

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Author contributions
Wenjie Chen and Qinyue Su collected data and wrote the main manuscript. Ming Zhong was in charge of interpretation of data and prepared the relevant figure and table. Yanjun Zheng and Xiaofeng Wang were in charge of statistics analysis. Hongping Qu, Enqiang Mao and Zhitao Yang reviewed and checked the manuscript. Erzhen Chen and Ying Chen proposed the idea, were in charge of the design of work and confirmed the final manuscript.

Availability of data and materials
The datasets in this study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
This study adhered to the principles of the Declaration of Helsinki and was approved by the Ethics Committee of Ruijin Hospital, Shanghai Jiao Tong University School of Medicine (Ethical Number: KY2023-391). Patient data were analyzed with strict adherence to anonymity protocols. Since this was a retrospective study involving no additional interventions, informed consent from patients was exempted.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

Author details
[1]Department of Emergency, Ruijin Hospital, Shanghai Jiaotong University School of Medicine, Ruijin 2# Road No. 197, Shanghai 200025, China. [2]Department of Critical Care Medicine, State Key Laboratory of Complex Severe and Rare Diseases, Peking Union Medical College Hospital, Peking Union Medical College, Beijing, China. [3]Department of Critical Care Medicine, Ruijin Hospital, Shanghai Jiaotong University School of Medicine, Shanghai, China. [4]Shanghai Institute of Aviation Medicine, Ruijin Hospital, Shanghai Jiaotong University School of Medicine, Shanghai, China. [5]Department of Emergency and Critical Care Medicine, Ruijin Hospital Wuxi Branch, Shanghai Jiao Tong University School of Medicine, Wuxi, China.

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