The Journal of Emergency Medicine, Vol. 67, No. 1, pp. e1–e9, 2024

https://doi.org/10.1016/j.jemermed.2024.02.011
0736-4679/$ - see front matter

Original Contributions

Analysis of Risk Factors for Severe Acute Pancreatitis in the Early Period (<24 h) After Admission

Qian Yang,* Yunhan Gao,* Zhongfu Li,* Jiang Zheng,† Hong Fu,† and Yu Ma‡

*Department of General Surgery, Chongqing Emergency Medical Center, Chongqing University Central Hospital, Chongqing, 400014, China, †Department of Anesthesiology, Chongqing Emergency Medical Center, Chongqing University Central Hospital, Chongqing, 400014, China, and ‡Department of Intensive Care Unit, Chongqing Emergency Medical Center, Chongqing University Central Hospital, Chongqing, 400014, China

Reprint Address: Hong Fu, MD, Department of Anesthesiology, Chongqing Emergency Medical Center, Chongqing University Central Hospital, Chongqing, 400014, China.

□ **Abstract—Background: Severe acute pancreatitis (SAP) has high mortality. Early identification of high-risk factors that may progress to SAP and active intervention measures may improve the prognosis of SAP patients. Objective: Clinical data within 24 h after admission were retrospectively analyzed to provide an evidence for early screening of high-risk factors in patients with SAP. Methods: A review of clinical data of acute pancreatitis patients from January 1, 2018, to December 31, 2022, was conducted. We compared the clinical data of SAP and non-SAP patients, and a multivariable logistic regression model was used to identify the independent predictors of SAP. The receiver operating characteristic (ROC) curve of SAP was drawn for continuous numerical variables to calculate the optimal clinical cutoff value of each variable, and the predictive value of each variable was compared by the area under the ROC curve. Results: Based on the multivariate logistic regression analysis of Age (odds ratio (OR), 1.032;95% confident interval (CI),1.018–1.046, $p < 0.001$), body mass index (BMI) (OR, 1.181; 95% CI,1.083–1.288, $p < 0.001$), Non-HTGAP (nonhypertriglyceridemic acute pancreatitis) (OR, 2.098; 95% CI,1.276–3.45, $p = 0.003$), white blood cell count (WBC) (OR,1.072; 95% CI,1.034–1.111, $p < 0.001$), procalcitonin (PCT) (OR, 1.060; 95% CI, 1.027–1.095, $p < 0.001$), serum calcium (Ca) (OR,0.121; 95% CI, 0.050–0.292, $p < 0.001$), computed tomography severity index (CTSI) ≥4 (OR,12.942;95% CI,7.267–23.049, $p < 0.001$) were identified as independent risk factors for SAP. The area under the ROC curve (AUC) and optimal CUT-OFF values of continuous numerical variables for predicting SAP were Age (0.6079,51.5), BMI (0.6,23.25), WBC (0.6701,14.565), PCT (0.7086, 0.5175), Ca (0.7787,1.965), respectively. Conclusion: Age, BMI, non-HTGAP, WBC, PCT, serum Ca and CTSI≥4 have good predictive value for SAP.**

□ **Keywords—acute pancreatitis; severity; risk factors; prognosis**

Introduction

Acute pancreatitis is a common acute abdomen (1). It has a global incidence of 30–40 cases per 100,000 people per year and has an overall mortality rate of 1% to 5% (2–4). Its treatment costs are high, especially in severe cases, with an average cost of approximately €10,000 per acute pancreatitis patient reported (5,6). Although most patients with acute pancreatitis have mild acute pancreatitis, approximately 13.1% to 30% develop severe disease (7–9). Despite great advances in the diagnosis and treatment of acute pancreatitis has been made in recent years, the mortality rate of severe acute pancreatitis (SAP) is still high, reportedly as high as 14.3% to 40% (8–11). According to the revised Atlanta classification of severity (12), SAP has

Received: 17 October 2023; Final submission received: 25 January 2024;
Accepted: 2 February 2024

persistent organ damage (>48 h), but this classification leads to a delay in the diagnosis of severity and cannot provide help for the early treatment of acute pancreatitis. Early identification of risk factors that may progress to SAP and early selection of patients who may benefit from interventions are crucial in order to hopefully reduce the incidence of progression to SAP and improve the prognosis of acute pancreatitis patients (13). This study retrospectively analyzed the demographic, etiologic, clinical, and laboratory data of acute pancreatitis patients and explored the early risk factors for SAP to provide evidence for early prediction of high-risk factors for SAP.

Materials and Methods

Eligibility Criteria

This retrospective cohort study was conducted at the Chongqing Emergency Medical Center, a 1200-bed hospital in Chongqing, China. We reviewed the data of patients aged 18–95 years who were hospitalized for acute pancreatitis from January 1, 2018 to December 31, 2022. Specifically, the inclusion and exclusion criteria in our experience were as follows: inclusion criteria: 1) complete medical records; 2) meet the diagnostic criteria for acute pancreatitis; and 3) abdominal enhancement and chest computed tomography (CT) scan performed within 24 h after admission. Exclusion criteria: 1) the time from onset to admission was more than 72 h; 2) the patient had been treated in other hospitals before entering this hospital; 3) chronic pancreatitis; 4) acute pancreatitis during pregnancy; 5) previous history of pancreatic surgery; 6) pancreatic cancer; 7) previous history of pleural effusion or organ failure; 8) pancreatitis caused by trauma and iatrogenic injury. This study follows the principles outlined in the Declaration of Helsinki. Due to the nature of the retrospective study, the identity and data of the patients were anonymous, so the informed consent was waived by the institutional Review Board. The study was exempted from Ethical approval by the institutional Review Board.

Definitions

Diagnosis (12):The diagnosis of acute pancreatitis meets two of the following three characteristics: 1) abdominal pain similar to acute pancreatitis; 2) serum amylase activity (or lipase activity) was at least three times higher than the upper limit of normal; and 3) abdominal imaging characteristic findings of acute pancreatitis consistent with changes in acute pancreatitis.

Severity (12): Mild acute pancreatitis (MAP): No organ failure; No local or systemic complications. Moderately severe acute pancreatitis (MSAP): transient organ failure (<48 h); or local or systemic complications. SAP: persistent organ failure (>48 h); single or multiple organ failure.

Etiology (13): Biliary acute pancreatitis (BAP) is defined as acute pancreatitis caused by pancreatic duct obstruction caused by various diseases of the biliary tract, such as stones, ascaris lumbricoides, infection, scar stenosis, and inflammatory edema. Hypertriglyceridemic acute pancreatitis (HTGAP) was defined as acute pancreatitis with serum triglycerides (TGs) above 11.3 mmol/L (1000 mg/dL) or TGs between 5.65 and 11.3 mmol/L at the time of onset, and other causes, such as cholelithiasis and alcohol abuse, were excluded. Alcoholic pancreatitis is defined as a history of heavy drinking (≥50 g/d) for more than 5 years or excessive alcohol intake prior to onset without other known causes of acute pancreatitis.

Data Collection

Data were collected through the hospital's electronic medical record system and archived medical records. The imaging data of all patients were re-evaluated by the same radiologist, the results were interpreted according to the same standard, and the computed tomography severity index (CTSI) was scored. Patient general information included age, sex, body mass index (BMI), comorbidities (type 2 diabetes mellitus(T2DM), hypertension, fatty liver), etiology (biliary acute pancreatitis (BAP) vs. nonbiliary acute pancreatitis (non-BAP)/hypertriglyceridemic acute pancreatitis (HTGAP) vs. nonhypertriglyceridemic acute pancreatitis (non-HTGAP)) and disease episode (recurrence vs. first attack). Laboratory parameters included serum white blood cell count (WBC), hematocrit (HCT), procalcitonin (PCT), calcium (Ca), amylase and lipase at admission. Radiographic parameters included CTSI and pleural effusion within 24 h after admission. Treatment factors included the use of prophylactic antibiotics and exclusive enteral nutrition (EEN) within 24 h after admission. The clinical outcomes included death and length of stay (LOS).

Statistical Analysis

SPSS 27.0 software was used to analyze the data. Continuous numerical variables are expressed as the mean ± SD, normally distributed data were compared by one-way analysis of variance, and skewed distribution data were compared by the Mann–Whitney U test. Categorical variables were expressed as the number of cases and percentages and analyzed with the X^2 test or Fisher's exact test. Multivariate logistic regression analysis was used to identify the independent risk factors for SAP. The receiver operating characteristic (ROC) curve of SAP was drawn for continuous numerical variables to calculate the

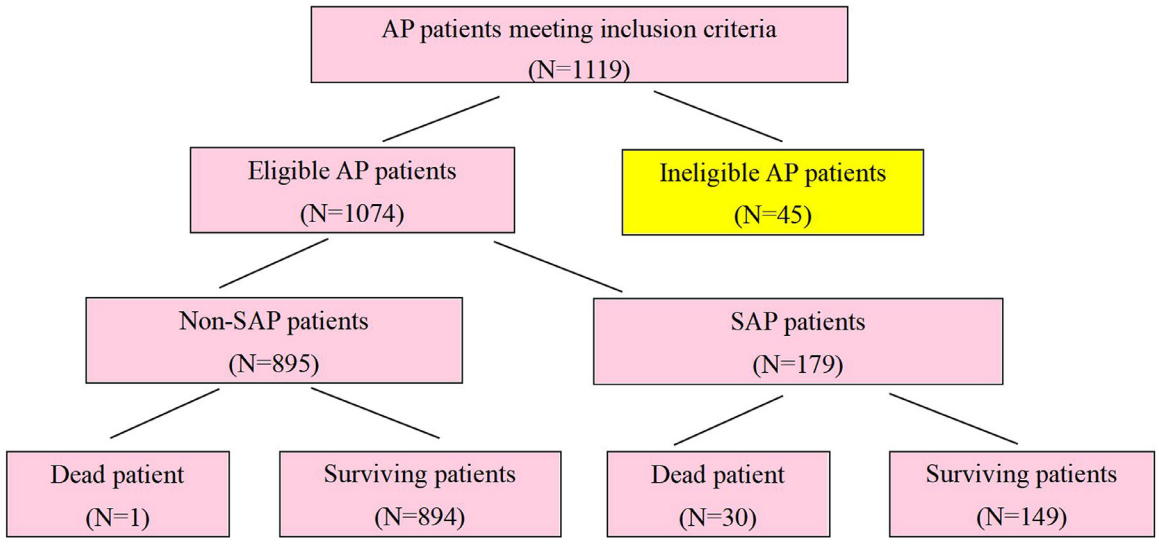

Figure 1. Study flowchart. AP = acute pancreatitis; SAP = severe acute pancreatitis.

optimal clinical cutoff value of each variable, and the predictive value of each variable was compared by the area under the ROC curve (AUC). $P<0.05$ was considered statistically significant.

Results

The Clinical Characteristics of the Acute Pancreatitis Patients

A total of 1074 patients were enrolled according to the established criteria (Figure 1). There were 634 males and 440 females, with a mean age of 51.7 (SD±17.3) years. There were 669 cases (62.3%) of MAP, 226 cases (21%) of MSAP and 179 cases (16.7%) of SAP. According to the etiology classification, there were 441 cases of BAP, 405 cases of HTGAP, 11 cases of alcoholic acute pancreatitis, and 217 cases of other types of acute pancreatitis. Alcoholic acute pancreatitis accounted for a small proportion, so it was not analyzed separately. BAP is still the most common type of acute pancreatitis, followed by HTGAP.

The Clinical Characteristics of the SAP and Non-SAP Group Patients

The comparative results of the clinical characteristics of the patients with SAP and non-SAP are reported in Table 1. These data, including age, BMI, WBC, PCT, amylase, lipase, and LOS, were significantly higher in the SAP group than in the non-SAP group within 24 h after admission ($p < 0.05$), whereas the serum concentrations of Ca were significantly lower in the SAP group ($p < 0.05$). The proportions of patients with non-HTGAP, CTSI≥4, pleural effusion, antibiotic use, and death were significantly higher in the SAP group than in the non-SAP group within 24 h after admission ($p < 0.05$). No significant difference was found in sex, hypertension, T2DM, fatty liver, etiology (BAP vs. non-BAP), HCT, or episode (recurrence vs. first attack) between the two groups ($p > 0.05$).

Mortality: The mortality of the whole group was 2.9% (31/1074), and it was different according to the severity of acute pancreatitis. There were no deaths in the MAP group, 1 death in the MSAP group (1/226, 0.4%), and 30 deaths in the SAP group (30/179, 16.8%). LOS: The median LOS was 13.53 (SD±13.006) days for the whole group, 29.98 (SD±24.625) days in the SAP group and 10.24 (SD±4.159) days in the non-SAP group. The main cause of death in this study was MODS, and other causes included acute respiratory distress syndrome, sepsis, coronary atherosclerotic heart disease, and sudden cardiac death. One patient with MSAP died of coronary atherosclerotic heart disease and sudden cardiac death.

The Clinical Characteristics of the Patients in the Death and Survival Groups

The comparative results of the clinical characteristics of the patients in the death and survival groups are reported in Table 2. These data, including age, BMI, WBC, PCT, amylase, and lipase, were significantly higher in the death group than in the survival group ($p < 0.05$), whereas the serum concentrations of Ca were significantly lower in the death group ($p < 0.05$). The proportions of patients with non-HTGAP, CTSI≥4, pleural effusion, and antibi-

Table 1. The Clinical Characteristics of SAP and Non-SAP Group Patients

	Non-SAP $N = 895$	SAP $N = 179$	p value
Age(years)	50.6 ± 17.0	57.2 ± 17.8	<0.001
Male	538(60.1%)	96(53.6%)	0.108
BMI(kg/m^2)	22.7 ± 2.8	23.6 ± 2.2	<0.001
Hypertension	228(25.5%)	51(28.5%)	0.401
T_2DM	286(32%)	50(27.9%)	0.289
Fatty liver	239(26.7%)	40(22.3%)	0.225
Etiology			
BAP vs. Non-BAP	360/535	81/98	0.212
HTGAP vs. Non-HTGAP	353/542(60.6%)	52/127(70.9%)	0.009
Other	182	46	
WBC($\times$ 10^9/L)	13.0 ± 5.3	16.3 ± 5.6	<0.001
HCT(L/L)	0.4 ± 0.06	0.4 ± 0.08	0.416
PCT(ng/ml)	1.5 ± 3.6	5.1 ± 10.9	<0.001
Ca(mmol/L)	2.0 ± 0.2	1.8 ± 0.3	<0.001
Amylase(U/L)	586.0 ± 723.4	903.4 ± 852.7	<0.001
Lipase(U/L)	674.6 ± 755.2	979.3 ± 1081.3	<0.001
CTSI≥4	293(32.7%)	161(89.9%)	<0.001
Pleural effusion	129(14.4%)	65(36.3%)	<0.001
Episode(Recurrence VS First attack)	111/784(87.6%)	28/151(84.4%)	0.238
Antibiotics use	533(59.6%)	179(100%)	<0.001
Death	1(0.1%)	30(16.8%)	<0.001
LOS(d)	10.2 ± 4.2	30.0 ± 24.6	<0.001

Severe acute pancreatitis (SAP):Persistent organ failure (>48 h); single or multiple organ failure. Non-Severe acute pancreatitis (Non-SAP): Including mild acute pancreatitis (MAP) and moderate severe acute pancreatitis (MSAP). MAP: No organ failure; No local or systemic complications. MSAP: Transient organ failure (<48 h); or local or systemic complications. Biliary acute pancreatitis (BAP): acute pancreatitis caused by pancreatic duct obstruction caused by various biliary tract diseases such as stones, ascaris lumbricoides, infection, scar stenosis, inflammatory edema, etc.Hypertriglyceridemic acute pancreatitis (HTGAP): Acute pancreatitis with a serum triglyceride level higher than 11.3 mmol/L (1000 mg/dL) or a triglyceride level between 5.65 and 11.3 mmol/L at the time of onset, and other causes, such as cholelithiasis and alcohol abuse, were excluded. CTSI = Computed tomography severity index;LOS = Length of stay; BMI = body mass index; T_2DM = type 2 diabetes mellitus; WBC = white blood cell count; HCT = hematocrit; PCT = procalcitonin; Ca = calcium .

otic use were significantly higher in the death group than in the survival group ($p < 0.05$), whereas the recurrence was significantly lower in the death group ($p < 0.05$). No significant difference was found in sex, hypertension, T2DM, fatty liver, etiology (BAP vs. non-BAP), HCT, or LOS between the two groups ($p > 0.05$).

All 31 patients in the death group had their first attack of acute pancreatitis. The LOS of the 31 deceased patients ranged from 1 to 117 days, with a median of 22.7742 (SD±31.0373) days. Five of the 31 patients died within 1 day, 18 died within 14 days, and 22 died within 30 days. Nine patients died after 30 days of hospitalization, and 2 died after 100 days (114 and 117 days).

Analysis of Early Risk Factors for SAP

The univariate analysis of SAP is shown in Table 3. The risk factors for SAP included age, BMI, etiology (non-HTGAP), WBC count, PCT, Ca, CTSI≥4, and pleural effusion. These variables were included in the multivariate analysis. Figure 2 displays the results of the multivariate analysis for SAP. Age (OR,1.032;95% CI,1.018–1.046, $p < 0.001$), BMI (OR,1.181;95% CI,1.083–1.288, $p < 0.001$), non-HTGAP (OR,2.098;95% CI,1.276–3.45, $p = 0.003$), WBC (OR,1.072;95% CI,1.034–1.111, $p < 0.001$), PCT(OR,1.060;95% CI,1.027–1.095, $p < 0.001$), serum Ca (OR,0.121;95% CI,0.050–0.292, $p < 0.001$),

Table 2. The Clinical Characteristics of Death and Survival Group Patients

	Death Group $N = 31$	Survival Group $N = 1043$	p Value
Age(years)	63.2 ± 18.0	51.3 ± 17.1	<0.001
Male	15(48.4%)	619(59.3%)	0.221
BMI(kg/m^2)	23.9 ± 2.2	22.8 ± 2.7	0.025
Hypertension	9(29%)	270(25.9%)	0.694
T_2DM	8(25.8%)	328(31.4%)	0.504
Fatty liver	4(12.9%)	275(26.4%)	0.092
Etiology			
BAP vs. Non-BAP	15/16	426/617	0.400
HTGAP vs. Non-HTGAP	6/25 (80.6)	399/644 (61.7)	0.032
Other	10	218	
WBC(× 10^9/L)	17.3 ± 6.2	13.5 ± 5.4	<0.001
HCT(L/L)	0.4 ± 0.07	0.4 ± 0.06	0.442
PCT(ng/mL)	10.4 ± 17.6	1.8 ± 4.7	<0.001
Ca(mmol/L)	1.8 ± 0.4	2.0 ± 0.2	<0.001
Amylase(U/L)	1189.0 ± 1046.3	622.6 ± 739.4	<0.001
Lipase(U/L)	1181.0 ± 1227.3	711.8 ± 807.9	0.002
CTSI≥4	31(100%)	423(40.6%)	<0.001
Pleural effusion	12(38.7%)	182(17.4%)	0.002
Episode (Recurrence vs. First attack)	0/31(100%)	139/904(86.7%)	0.026
Antibiotics use	31(100%)	681(65.3%)	<0.001
LOS(d)	22.8 ± 31.0	13.3 ± 12.0	0.477

Biliary acute pancreatitis (BAP): acute pancreatitis caused by pancreatic duct obstruction caused by various biliary tract diseases such as stones, ascaris lumbricoides, infection, scar stenosis, inflammatory edema, etc. Hypertriglyceridemic acute pancreatitis (HTGAP): Acute pancreatitis with a serum triglyceride level higher than 11.3 mmol/L (1000 mg/dL) or a triglyceride level between 5.65 and 11.3 mmol/L at the time of onset, and other causes, such as cholelithiasis and alcohol abuse, were excluded. CTSI = Computed tomography severity index; LOS = Length of stay; BMI = body mass index; T_2DM = type 2 diabetes mellitus; WBC = white blood cell count; HCT = hematocrit; PCT = procalcitonin; Ca = calcium ..

Table 3. Univariate Analysis of Risk Factors for SAP

Variables	Univariate Analysis		
	OR	95%CI	p Value
Age (years)	1.022	1.013–1.031	<0.001
BMI (kg/m^2)	1.136	1.068–1.208	<0.001
Etiology (Non-HTGAP)	1.591	1.122–2.256	0.009
WBC (× 10^9/L)	1.106	1.075–1.138	<0.001
PCT (ng/mL)	1.089	1.056–1.124	<0.001
Ca (mmol/L)	0.021	0.010–0.043	<0.001
CTSI≥4	18.377	11.072–30.503	<0.001
Pleural effusion	3.386	2.369–4.839	<0.001

Non-HTGAP = non-hypertriglyceridemic acute pancreatitis; CTSI = computed tomography severity index. BMI = body mass index; WBC = white blood cell count; PCT = procalcitonin; Ca = calcium ..

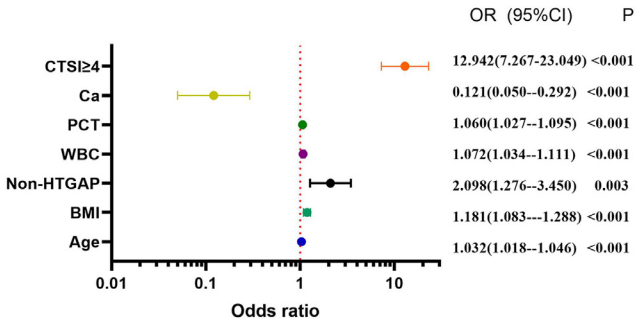

Figure 2. Multivariate analysis of early risk factors for SAP. CTSI = Computed tomography severity index; BMI = body mass index; Non-HTGAP = non-hypertriglyceridemic acute pancreatitis; WBC = white blood cell count; PCT = procalcitonin; Ca = calcium ..

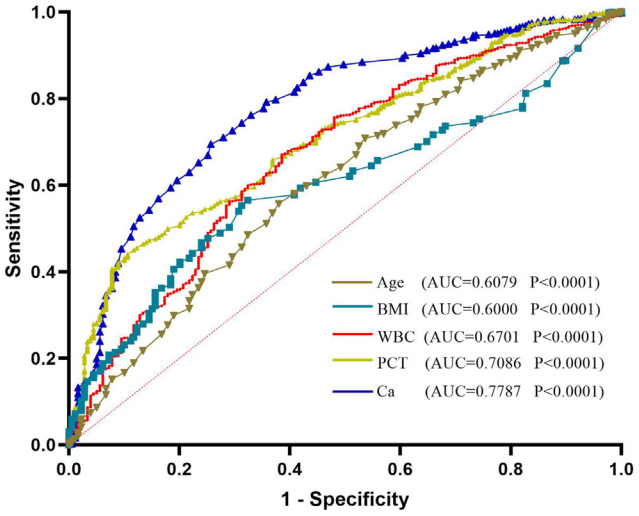

Figure 3. ROC curve and AUC for continuous numerical variable. ROC = receiver operating characteristic ; AUC = area under the ROC curve; BMI = body mass index; WBC = white blood cell count; PCT = procalcitonin; Ca = calcium.

and CTSI≥4 (OR,12.942;95% CI,7.267–23.049, $p < 0.001$) were identified as independent risk factors for SAP.

ROC Curve and the Optimal Clinical Cutoff Value

ROC curves and the respective AUC were calculated for each valuable continuous numerical variable to provide more accurate information about the capacity of the 5 variables in distinguishing SAP from non-SAP. The AUC values of age, BMI, WBC, PCT, and serum Ca for distinguishing SAP were 0.6079, 0.6, 0.6701, 0.7086, and 0.7787, respectively (Figure 3). The clinically optimal cutoff values were determined according to the corresponding sensitivity and specificity. The optimal cutoff values of the above 5 variables were 51.5, 23.25, 14.565, 0.5175, and 1.965, respectively (Table 4). The serum concentrations of Ca (95% CI, 0.7416–0.8159, $p < 0.0001$) had the highest value in predicting the progression of SAP in acute pancreatitis patients. When the serum concentration of Ca on admission is lower than 1.965 mmol/L, the possibility of acute pancreatitis developing into SAP should be considered.

Discussion

Acute pancreatitis is a common and potentially life-threatening acute abdomen. The revised Atlanta classification (12) divides acute pancreatitis into MAP, MSAP and SAP types. MAP is characterized by no organ failure, no local or systemic complications, very rare mortality, and good prognosis. MSAP only causes transient organ failure, most of which have a good prognosis, and the mortality is far less than that of SAP. SAP is characterized by persistent organ failure (>48 h), high mortality and poor prognosis (12). However, this classification method does not provide a clear clinical stratification of severity on admission, nor does it provide an early prediction of which patients are at the highest risk for developing clinically SAP and may require intensive treatment. To identify high-risk factors for SAP, a variety of clinical

Table 4. Diagnostic Value of Each Valuable Continuous Numerical Variables in Predicting SAP

Predictive Marker	Cut-off	Sensitivity	Specificity	AUC	95%CI	*p* Value
Age (years)	51.5	0.62	0.558	0.6079	0.5623–0.6534	<0.0001
BMI (kg/m^2)	23.25	0.676	0.565	0.6000	0.5600–0.6401	<0.0001
WBC ($\times$ 10^9/L)	14.565	0.615	0.67	0.6701	0.6261–0.7141	<0.0001
PCT (ng/mL)	0.5175	0.894	0.44	0.7086	0.6703–0.7469	<0.0001
Ca (mmol/L)	1.965	0.743	0.694	0.7787	0.7416–0.8159	<0.0001

AUC = the area under the receiver operating characteristic (ROC) curve; BMI = body mass index; WBC = white blood cell count; PCT = procalcitonin; Ca = calcium..

scoring systems (14,15) have been used to predict the severity of acute pancreatitis, such as APACHE-II, Ranson, BISAP, Glasgow, and SIRS. However, some clinical scores (16,17) are not available until at least 24 h after hospitalization. It cannot be used to predict the severity of acute pancreatitis on admission (16,17). To our knowledge, few studies have combined demographic, etiologic, clinical, and laboratory data that are easily identified and simply commonly used on admission for early prediction of acute pancreatitis severity. Previous studies have focused on specific clinical scoring systems (14,15) and laboratory serum markers (18–20).

In our study, the overall mortality was 2.9%, and the mortality in the SAP group was 16.8%, which is consistent with previous reports. Age, BMI, non-HTGAP, WBC, PCT, Ca, and CTSI≥4 were independent risk factors for SAP. A retrospective study analyzing the influence of demographic factors and treatment on its outcome showed that the severity of acute pancreatitis and mortality increased with age (8). In a prospective study designed to simply assess acute pancreatitis severity within 72 h after admission, results showed that advanced age (>65 years) was an independent prognostic factor of complex acute pancreatitis (21). A previous prospective study found that the proportion of patients who die of acute pancreatitis is relatively high in elderly individuals, and age is considered to be related to death. However, after careful analysis, it was found that when only the death caused by complications of acute pancreatitis was analyzed, there was no significant difference in mortality between the young group and the elderly group, and the high mortality was related to concomitant diseases with advanced age (22). Our study suggested that age was associated with the severity and mortality of acute pancreatitis, and the regression analysis suggested that age was an independent risk factor for SAP. Further studies on the association of age with the severity and mortality of acute pancreatitis are needed, especially prospective studies.

A meta-analysis of how BMI affects the outcome of acute pancreatitis confirmed that a BMI higher than 25 increases the risk of severe acute pancreatitis, while BMI>30 increases the risk of mortality (23). Our study suggested that high BMI was associated with the incidence of SAP and mortality of acute pancreatitis and that BMI was an independent risk factor for SAP. Some studies have shown that the incidence of SAP and mortality of acute pancreatitis in HTGAP are higher than those in biliary acute pancreatitis (24,25). A systematic review that attempted to determine whether HTGAP was more severe than acute pancreatitis caused by other etiologies showed heterogeneity among the findings, with no difference in severity in 2 of the 7 reports and possible increases in severity in the remaining 5 studies (26). Our study compared and analyzed the differences in severity and mortality between HTGAP and non-HTGAP, and the results showed that the incidence of SAP and mortality of acute pancreatitis were lower in HTGAP patients, and non-HTGAP was an independent risk factor for SAP. Therefore, it is necessary to further study the relationship between etiology and acute pancreatitis severity and mortality.

An elevated WBC count and PCT are signs of infection, although these may also be associated with systemic inflammatory response syndrome(SIRS) in other patients with noninfectious pancreatitis (27).There was a study to develop a novel prognostic approach that could simply and objectively assess acute pancreatitis severity within 72 h after admission. The results showed that leucocytes >13,000/mm^3 was an independent predictor of complicated acute pancreatitis (21). A systematic review evaluating the value of PCT as a marker of SAP progression showed that serum PCT measurement may be valuable in predicting the severity of acute pancreatitis (28). There was a prospective study designed to determine the value of PCT as an early prognostic marker in acute pancreatitis, and the results showed that PCT has limited added value in the early assessment of acute pancreatitis severity (29). In our statistical model, WBC and PCT elevation within 24 h of admission were associated with SAP incidence and acute pancreatitis mortality, and both univariate and multivariate logistic regression analyses showed that WBC and PCT were independent risk factors for SAP. More studies

are needed to reveal the relationship between WBC, PCT and acute pancreatitis severity and mortality.

One study (30) has reported that the incidence of hypocalcemia in patients with SAP is significantly higher, and it is believed that serum Ca on admission is independently related to persistent organ failure in acute pancreatitis patients. The results of this study showed that the decrease in the serum concentrations of Ca in acute pancreatitis patients was associated with the incidence of SAP and mortality of acute pancreatitis and was an independent risk factor for SAP, which was basically consistent with previous reports.

A study compared the accuracy of CT and clinical scoring systems in predicting acute pancreatitis severity on admission. The results showed that the accuracy of the CT scoring systems in predicting acute pancreatitis severity was similar to that of the clinical scoring systems, and CTSI≥4 is considered to be the optimal cutoff value for predicting the severity and mortality of acute pancreatitis (31). Our study suggested that CTSI≥4 was an independent risk factor for SAP, which is consistent with literature reports.

Advantages and Shortcomings

Our study has some advantages. First, our study included a large sample size; to my knowledge, no larger retrospective study has been performed to screen for early high-risk factors for SAP. Secondly, we selected easily available and commonly used data at admission instead of using some complex scoring systems for early prediction of severity, which has good practical application value. Our study has some shortcomings. First, the fact that this is a retrospective study involving different clinical departments (gastroenterology, general surgery, ICU) could lead to some deviations. Second, we selected commonly used clinical data that could be easily identified on admission, and limited clinical data were available for analysis. A prospective, multicenter study is needed to confirm these findings.

Conclusions

In summary, our results suggest that the use of demographic, etiologic, clinical, and laboratory data on admission of acute pancreatitis patients can help clinicians predict the potential risk of developing SAP, which may drive optimal treatment selection to reduce the incidence and mortality of SAP. Age, BMI, non-HTGAP, WBC count, PCT, serum Ca, and CTSI≥4 were independent risk factors for SAP. Therefore, early identification of these risk factors and active intervention measures may help to reduce the occurrence of SAP and improve the prognosis of acute pancreatitis.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

CRediT authorship contribution statement

Qian Yang: Writing – review & editing, Writing – original draft, Project administration, Investigation, Data curation, Conceptualization. **Yunhan Gao:** Data curation. **Zhongfu Li:** Data curation. **Jiang Zheng:** Investigation, Data curation. **Hong Fu:** Funding acquisition, Data curation, Conceptualization. **Yu Ma:** Formal analysis, Conceptualization.

Acknowledgments

The authors thank all study participants. The authors thank the fund support provided by Chongqing Municipal Health Commission [ZQNYXGDRCGZS2019006].

References

1. Xiaoping C, Jianping W, Jizong Z. Surgery [M]. Beijing: People's Medical Publishing House; 2018. p. 457–61 Chinese.
2. Petrov MS, Yadav D. Global epidemiology and holistic prevention of pancreatitis. Nat Rev Gastroenterol Hepatol 2019;16(3):175–84.
3. 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.
4. Xiao AY, Tan ML, Wu LM, et al. Global incidence and mortality of pancreatic diseases: a systematic review, meta-analysis, and meta-regression of population-based cohort studies. Lancet Gastroenterol Hepatol 2016;1(1):45–55.
5. Peery AF, Crockett SD, Murphy CC, et al. Burden and cost of gastrointestinal, liver, and pancreatic diseases in the United States: update 2018. Gastroenterology 2019;156(1):254–72.
6. Andersson B, Appelgren B, Sjödin V, et al. Acute pancreatitis—costs for healthcare and loss of production. Scand J Gastroenterol 2013;48(12):1459–65.
7. Boxhoorn L, Voermans RP, Bouwense SA, et al. Acute pancreatitis. Lancet 2020;396(10252):726–34.
8. Berger Z, Mancilla C, Tobar E, et al. Acute pancreatitis in Chile: a multicenter study on epidemiology, etiology and clinical outcome. Retrospective analysis of clinical files. Pancreatology 2020;20(4):637–43.
9. Li W, Ou L, Fu Y, et al. 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).
10. 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.
11. van Santvoort HC, Bakker OJ, Bollen TL, et al. A conservative and minimally invasive approach to necrotizing pancreatitis improves outcome. Gastroenterology 2011;141(4):1254–63.

12. 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(1):102–11.
13. Tenner S, Baillie J, DeWitt J, et al. American College of Gastroenterology guideline: management of acute pancreatitis. Am J Gastroenterol 2013;108(9):1400–15 1416.
14. Mounzer R, Langmead CJ, Wu BU, et al. Comparison of existing clinical scoring systems to predict persistent organ failure in patients with acute pancreatitis. Gastroenterology 2012;142(7):1476–82.
15. Cho JH, Kim TN, Chung HH, et al. Comparison of scoring systems in predicting the severity of acute pancreatitis. World J Gastroenterol 2015;21(8):2387–94.
16. Ong Y, Shelat VG. Ranson score to stratify severity in acute pancreatitis remains valid - Old is gold. Expert Rev Gastroenterol Hepatol 2021;15(8):865–77.
17. Eachempati SR, Hydo LJ, Barie PS. Severity scoring for prognostication in patients with severe acute pancreatitis: comparative analysis of the Ranson score and the APACHE III score. Arch Surg 2002;137(6):730–6.
18. Nieminen A, Maksimow M, Mentula P, et al. Circulating cytokines in predicting development of severe acute pancreatitis. Crit Care 2014;18(3):R104.
19. Maksimow M, Kyhälä L, Nieminen A, et al. Early prediction of persistent organ failure by soluble CD73 in patients with acute pancreatitis*. Crit Care Med 2014;42(12):2556–64.
20. van den Berg FF, de Bruijn AC, van Santvoort HC, et al. Early laboratory biomarkers for severity in acute pancreatitis; a systematic review and meta-analysis. Pancreatology 2020;20(7):1302–11.
21. Gonzálvez-Gasch A, de Casasola GG, Martín RB, et al. A simple prognostic score for risk assessment in patients with acute pancreatitis. Eur J Intern Med 2009;20(3):e43–8.
22. Fan ST, Choi TK, Lai CS, et al. Influence of age on the mortality from acute pancreatitis. Br J Surg 1988;75(5):463–6.
23. Dobszai D, Mátrai P, Gyöngyi Z, et al. Body-mass index correlates with severity and mortality in acute pancreatitis: a meta-analysis. World J Gastroenterol 2019;25(6):729–43.
24. Zheng Y, Zhou Z, Li H, et al. A multicenter study on etiology of acute pancreatitis in Beijing during 5 years. Pancreas 2015;44(3):409–14.
25. Pu W, Luo G, Chen T, et al. A 5-year retrospective cohort study: epidemiology, etiology, severity, and outcomes of acute pancreatitis. Pancreas 2020;49(9):1161–7.
26. Carr RA, Rejowski BJ, Cote GA, et al. Systematic review of hypertriglyceridemia-induced acute pancreatitis: a more virulent etiology? Pancreatology 2016;16(4):469–76.
27. Müller CA, Uhl W, Printzen G, et al. Role of procalcitonin and granulocyte colony stimulating factor in the early prediction of infected necrosis in severe acute pancreatitis. Gut 2000;46(2):233–8.
28. Mofidi R, Suttie SA, Patil PV, et al. The value of procalcitonin at predicting the severity of acute pancreatitis and development of infected pancreatic necrosis: systematic review. Surgery 2009;146(1):72–81.
29. Modrau IS, Floyd AK, Thorlacius-Ussing O. The clinical value of procalcitonin in early assessment of acute pancreatitis. Am J Gastroenterol 2005;100(7):1593–7.
30. Peng T, Peng X, Huang M, et al. Serum calcium as an indicator of persistent organ failure in acute pancreatitis. Am J Emerg Med 2017;35(7):978–82.
31. Bollen TL, Singh VK, Maurer R, et al. A comparative evaluation of radiologic and clinical scoring systems in the early prediction of severity in acute pancreatitis. Am J Gastroenterol 2012;107(4):612–19.