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ORIGINAL ARTICLE

Predicting persistent organ failure on admission in patients with acute pancreatitis: development and validation of a mobile nomogram

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Abstract

Background: Early prediction of persistent organ failure (POF) is important for triage and timely treatment of patients with acute pancreatitis (AP).

Methods: All AP patients were consecutively admitted within 48 h of symptom onset. A nomogram was developed to predict POF on admission using data from a retrospective training cohort, validated by two prospective cohorts. The clinical utility of the nomogram was defined by concordance index (C-index), decision curve analysis (DCA), and clinical impact curve (CIC), while the performance by post-test probability.

Results: There were 816, 398, and 880 patients in the training, internal and external validation cohorts, respectively. Six independent predictors determined by logistic regression analysis were age, respiratory rate, albumin, lactate dehydrogenase, oxygen support, and pleural effusion and were included in the nomogram (web-based calculator: https://shina.shinyapps.io/DynNomapp/). This nomogram had reasonable predictive ability (C-indexes 0.88/0.91/0.81 for each cohort) and promising clinical utility (DCA and CIC). The nomogram had a positive likelihood ratio and post-test probability of developing POF in the training, internal and external validation cohorts of 4.26/31.7%, 7.89/39.1%, and 2.75/41%, respectively, superior or equal to other prognostic scores.

Conclusions: This nomogram can predict POF of AP patients and should be considered for clinical practice and trial allocation.

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Introduction

Acute pancreatitis (AP) has a spectrum of severity ranging from mild to critical.[1] The early prediction of the severity of AP informs clinical decisions about triage, transfer, and intervention.[2] Early prediction is also important in the research setting, where the accurate allocation of patients into trial arms based on predicted severity is important for testing treatments for AP. The key determinants of AP severity are organ failure and infected pancreatic necrosis.[3] Persistent organ failure (POF) is the most important determinant for mortality,[4–6] the leading cause of death from AP[7] and is the basis for the severe grade of AP in the revised Atlanta classification (RAC)[8]).

Given the importance of predicting severity and since Ranson defined his score in 1974,[9] there have been more than 20 prognostic scores have been studied for AP severity prediction.[10] However, their clinical utility is limited by an accuracy of predicting POF of around 75% and many are cumbersome to use.[11] Recent systematic reviews and meta-analyses conclude that current early predictors of POF,[12] infected pancreatic necrosis,[12] and mortality[10] are not sufficiently accurate for decision making in individual patients. The ideal predictor of POF would be applied on patient admission and within 24 h of the onset of symptoms. It would be cost-effective, easy to use and have an accuracy between 95 and 100%. Considerable progress has been made in identifying serum biomarkers to stratify early risk and severity in patients with AP.[13] However, due to their rapid time-course changes in serum concentration, non-specificity, cost, complexity, and suboptimal accuracy, none of these biochemical biomarkers have been adopted into routine clinical practice. In recent years, different nomograms have been applied for predicting severity and mortality,[14–17] splanchnic vein thrombosis,[18] abdominal infection,[19] oral refeeding intolerance,[20] success of catheter drainage in necrotizing AP,[21,22] and risk of new-onset diabetes after AP,[23] as well as computed tomography index for assessing AP outcomes[24] (Table 1). Of 4 studies[14–17] that used a nomogram to predict severity and mortality, 3 studies used online Critical Care Database (Medical Information Mart for Intensive Care III database [MIMIC-III],[14,16] eICU Collaborative Research Database [eICU-CRD][15]) and the fourth used retrospective data.[17] None of the 4 studies reported the duration of symptoms prior to hospital admission. No study to date has used training and validation cohorts to develop a nomogram to predict POF in AP patients.

The aim of this study was (1) to develop and validate a nomogram for predicting POF on admission in patients with AP, and (2) to compare the performance of the nomogram with conventional prognostic scores for predicting POF.

Methods

Study design and ethics

This study followed the STROBE guidelines[25] for observational studies. The study protocol was approved by respective Institutional Review Board in two tertiary hospitals. Data were obtained from two AP cohorts in West China Hospital of Sichuan University (WCH/SCU; Chengdu). The *training cohort* was from a retrospective dataset from 1st July 2009 to 30th June 2013.[6] The *internal validation cohort* was from a prospective dataset from 1st January 2016 and 31st August 2017.[26] The *external validation cohort* was from a prospective dataset from The First Affiliated Hospital of Nanchang University (Nanchang) from 1st January 2005 to 31st December 2012.[27]

Inclusion and exclusion criteria

The inclusion criteria were (1) diagnosis of AP diagnosed by the RAC criteria,[8] (2) age >18 and ≤80 years old, and (3) duration of abdominal pain from onset to admission ≤48 h, (4) data to calculate conventional prognostic scores within 6 h of admission.

The exclusion criteria were (1) patients admitted to another hospital first and then transferred, (2) patients re-admitted during the same episode of AP, (3) patients with an etiology related to chronic pancreatitis, pancreatic neoplasia, pancreatic trauma, or pregnancy, (4) advanced pulmonary, cardiac, renal diseases (chronic kidney disease stage 4–5), liver cirrhosis (modified Child–Pugh grade 2–3) or malignancy, and (5) if the nomogram could not be completed because of missing data.

Definitions, variables, and outcome measures

Data collection: Experienced doctoral students and resident doctors specializing in management of AP were stringently trained for data collection according to pre-defined *proforma* and standard operating procedures in both centers. Each *proforma* was checked and signed off by attending or more senior doctors.

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Table 1 Summarize of the present developed nomograms in AP

Nomograms for predicting SAP or mortality										
	Predicted outcome	Study design	Symptom onset time	Data collection time	Indicators of the Nomogram	Training cohort		Validation cohort		Main findings
						N	C-index	N	C-index	
Jiang *et al*. 2019 [14]	Mortality 30-day	MIMIC-III	NA	Within 24 hWithin 24 h	age, ALT, RDW, BUN	228	0.751	114	0.875	Nomogram > BISAP > SOFA > SIRS for long-term mortality both in cohorts.
	180-day/ 1-year				age, WBC, RDW, SCR		0.758		0.856	
Li *et al*. 2020 [15]	In-hospital mortality	eICU-CRD	NA	Within 24 h	age, BUN and lactate	378	0.896	123	0.892	The nomogram and APACHE IV demonstrated comparable power in predicting in-hospital mortality.
Xu *et al*. 2020 [16]	SAP	MIMIC-III	NA	On admissionOn admission	SOFA, hemoglobin, albumin, TBIL, BUN	708	0.855	477	0.879	Nomogram > SOFA / OASIS for SAP and mortality in both cohorts.
	In-hospital mortality				Age, SOFA, WBC, TBIL, albumin		0.821		0.822	
Cao *et al*. 2021 [17]	SAP	Retrospective	NA	Within 24 h	Sex, Ca^{2+}, SCR, NE%, LYMPH%, EO%	571	0.69	150	0.71	No comparison with others.

Other nomograms in AP							
	Predicted outcome	Statistical methods	Indicators of the Nomogram	Training cohort		Validation cohort	
				N	C-index	N	C-index
Hollemans *et al*. 2016 [21]	Success of catheter drainage in infected necrotizing pancreatitis	Univariate and multivariate logistic regression	Sex, percentage of pancreatic necrosis, density collection, multiple organ failure	130	0.76	NA	NA
Zhou *et al*. 2016 [18]	Symptomatic splanchnic vein thrombosis in necrotizing acute pancreatitis	Univariate and multivariate logistic regression	Balthazar's CT score, intra-abdominal pressure and superior mesenteric vein thrombosis	104	0.842	NA	NA
Bevan *et al*. 2017 [20]	Oral feeding intolerance	Univariate and multivariate logistic regression	Day 2 GCSI nausea/ vomiting subs-core, etiology	217	NA	NA	NA
Ma *et al*. 2019 [23]	New-onset diabetes mellitus after the first attack of acute pancreatitis	Univariate and multivariate logistic regression	Age, BMI, glucose, triglyceride, and LDL-C	616	0.686	NA	NA
Bellam *et al*. 2019 [22]	Success of percutaneous catheter drainage in patients with acute pancreatitis having acute fluid collection	Univariate and multivariate logistic regression	volume of collection after PCD and organ failure resolution after PCD	51	0.915	NA	NA

(*continued on next page*)

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Table 1 (*continued*)

Other nomograms in AP							
	Predicted outcome	Statistical methods	Indicators of the Nomogram	Training cohort		Validation cohort	
				N	C-index	N	C-index
Gupta *et al*. 2021 [24]	Mortality	Binomial logistic regression	Pancreatic necrosis, ascites, pleural effusion	103	0.79	20	0.74
	ICU stay		Number of collection, pleural effusion		0.66		0.70
	hospital stay≥4 weeks		Pancreatic necrosis, number of collection, amount PE		0.75		0.77
	Readmission		Number of collection, coeliac artery		0.70		0.52
	ICU stay≥2 weeks		Pancreatic necrosis, largest dimension of collection, pleural effusion		0.83		0.45
	SAP		Number of collection, liver steatosis		0.64		0.69
Zhu *et al*. 2021 [19]	Intra-abdominal infection	LASSO regression	Intra-abdominal pressure, APACHE II score, CTSI, ICU admission, and severity grade	417	0.99	294	0.98

SAP, severe acute pancreatitis; MIMIC-III, Medical Information Mart for Intensive Care III; NA, not available; ALT, alanine aminotransferase; RDW, red cell distribution width; BUN, blood urea nitrogen; WBC, white blood cell count; SCR, serum creatinine; BISAP, Bedside Index of Severity in Acute Pancreatitis; SOFA, Sequential Organ Failure Assessment; SIRS, Systemic Inflammatory Response Syndrome; ICU, intensive care unit; eICU-CRD, eICU Collaborative Research Database; APACHE, Acute Physiology, Age, and Chronic Health Evaluation; TBIL, total bilirubin; OASIS, Outcome and Assessment Information Set; NE%, neutrophil ratio; LYMPH%, lymphocyte Ratio; eosinophil ratio, EO%; GCSI, Gastroparesis Cardinal Symptom Index; LDL-C, low density lipoprotein-cholesterol; PCD, percutaneous catheter drainage; CTSI, computed tomography severity index.

The data collected on admission included age, gender, data for Charlson co-morbidity index, and duration of abdominal pain prior to admission. Vital signs, laboratory parameters (indices of routine blood, liver and renal function, myocardial enzymes, blood lipids and glucose, electrolyte levels and etc.), details of treatment (especially use of oxygen support, vasopressors, dialysis, antibiotic, and steroids), surgical interventions, and clinical outcomes were also recorded.

Presence of pleural effusion that were mostly obtained from nonenhanced computed tomography scan on admission. Patients without overt symptoms of pleural effusion (dyspnea, cough, and occasionally sharp, non-radiating, pleuritic chest pain) were not referred to having X-ray or CT scan in up to 10% of overall patients and these patients were considered as pleural effusion free.

Etiology

Hypertriglyceridemia as the etiology was defined as admission serum triglyceride (TG) level >11.3 mmol/l (1000 mg/dl) or TG > 5.65 mmol/l (500 mg/dl) with lipemic serum when other causes have been ruled out.[28–30] Biliary etiology was considered if gallstones or biliary sludge was present on radiological imaging or had an admission alanine aminotransferase level of over three times the upper limit of normal.[31] Alcohol excess was diagnosed if with drinking history >35 standard drinks per week for >5 years[32] or was deduced according to our center's own practice according to the drinking history after ruling out other etiologies.[6]

Organ failure, complications, and mortality

POF was defined as at least one organ systems (respiratory, circulatory, and renal) with a SOFA score of ≥2 and lasting ≥48 h.[1] Acute necrotic collection and acute peripancreatic fluid collection were defined as per RAC criteria.[8] Major infection included infected pancreatic necrosis, bacteremia, or pneumonia alone or in combination.[33] Mortality was recorded during the index admission or at 3 months of follow up for those who were automatically discharged with persistent multiple organ failure and had high possibility of death. Length of hospital stay were recorded for the index hospitalization.

Prognostic scores

The prognostic scores that were compared with the nomogram in this study were National Early Warning Score (NEWS),[34] Systemic Inflammatory Response Syndrome (SIRS),[11] Bedside Index for Severity in Acute Pancreatitis (BISAP),[11] modified

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Glasgow criteria,[11] Acute Physiology and Chronic Health Examination (APACHE) II,[11] and Sequential Organ Failure Assessment (SOFA).[11] These were all calculated on admission (or within 6 h of admission in rare cases).

Statistical analysis

Continuous variables were expressed as median with 25th-75th percentile and were compared by Mann–Whitney *U* test (2 groups) or Kruskal–Wallis *H* test (3 groups). Categorical data were reported as number with percentage and were compared by means of χ^2 or Fisher's exact test.

Independent risk factors for POF were identified and assessed significance of each by univariate logistic regression analysis in the training cohort. All variables significantly associated with POF were candidates for stepwise multivariate analysis, the results of which were used to formulate a nomogram. The predictive performance of the nomogram was measured by concordance index (C-index) and calibration with 1000 bootstrap samples to decrease the overfit bias.[35] The clinical utility of the nomogram was assessed by decision curve analysis (DCA) and clinical impact curve (CIC).[36] DCA is a simple, novel method of evaluating predictive models with decision analyses of diagnostic and prognostic tests by using a risk-benefit ratio.[37] CIC shows the estimated number considered at high risk for each risk threshold and depicts the proportion of those that are true positives.[36]

The area under curve (AUC) of the receiver operating characteristic (ROC) curves with 95% confidence intervals (CI) was calculated for potential predictors. An AUC of 0.5–0.7 was taken to represent no significant discrimination, 0.7 to 0.8 acceptable, 0.8 to 0.9 excellent, and >0.9 outstanding.[38] The AUC of two predictors were compared by method of DeLong.[39] The optimum cut-off for each predictor was determined by the ROC curve. The sensitivity, specificity, positive likelihood ratio (PLR), and negative likelihood ratio (NLR) were calculated by clinical decision analysis. A positive likelihood ratio is the probability that a positive test would be expected in a patient divided by the probability that a positive test would be expected in a patient without the endpoint of interest (e.g. POF). A negative likelihood ratio is the converse and if < 0.1 effectively rules out the endpoint. The post-test probabilities for each predictor were derived from the PLR (and NLR), using the pre-test probability of the particular endpoint from the relevant cohort and the standard likelihood ratio nomogram.[40]

The analyses were performed using IBM SPSS Statistics V26.0 software (SPSS, IBM Corp; Armonk, New York, USA) and R software version 4.0.4 (http://www.Rproject.org). Two-tailed *P* value with statistical significance set at < 0.05 was used for all tests.

Results

Patient characteristics

There were 816, 398, and 880 eligible patients in the training, internal validation and external validation cohorts, respectively.

Table 2 Baseline characteristics and clinical outcomes in training and validation cohorts

Variables	Training cohort	Validation cohorts	
	n = 816	(Internal) *n* = 398	(External) *n* = 880
Demographics			
Age, years*	45 (37–55)	45 (38–51)	50 (41–62)
Male	568 (69.6)	273 (68.6)	533 (60.6)
Charlson comorbidity index*	1 (0–1)	1 (0–2)	0 (0–0)
Etiology			
Biliary	230 (28.2)	78 (19.6)	506 (57.5)
Hypertriglyceridemia	267 (32.7)	167 (42.0)	184 (20.9)
Alcohol	30 (3.7)	31 (7.8)	61 (6.9)
Unknown or others	289 (35.4)	122 (30.7)	129 (14.7)
Time to admission, hours*	12 (7–24)	15 (9–24)	NA
Clinical outcomes			
Persistent organ failure	80 (9.8)	30 (7.5)	178 (20.2)
Respiratory	77 (9.4)	30 (7.5)	169 (19.2)
Circulatory	14 (1.7)	2 (0.5)	12 (1.4)
Renal	10 (1.2)	3 (0.8)	28 (3.2)
Need for HDU/ICU	59 (7.2)	29 (7.3)	168 (19.1)
Local complication			
Acute peripancreatic fluid collection	163 (20.0)	119 (29.9)	332 (37.7)
Acute necrotic collection	59 (7.2)	55 (13.8)	204 (23.2)
Major infection	45 (5.5)	20 (5.0)	53 (6.0)
Infected pancreatic necrosis	12 (1.5)	9 (2.3)	36 (4.1)
Bacteremia	15 (1.8)	6 (1.5)	13 (1.5)
Lung	35 (4.3)	9 (2.3)	18 (2.0)
Necrosectomy	14 (1.7)	13 (3.3)	17 (1.9)
Mortality	10 (1.2)	4 (1.0)	20 (2.3)
RAC			
Mild	508 (62.3)	190 (47.7)	328 (37.3)
Moderately severe	228 (27.9)	178 (44.7)	374 (42.5)
Severe	80 (9.8)	30 (7.5)	178 (20.2)
Length of hospital stay (days)*	9 (6–13)	8 (6–11)	8 (6–13)

NA, not available; HDU, high dependency unit; ICU, intensive care unit; RAC, revised Atlanta classification.
Values in parentheses are percentages unless indicated otherwise
*values are median with 25th-75th percentile.

The demographic and clinical outcome profiles are outlined in Table 2 and **Online** Supplementary Tables S1a–c. These results are similar to previous published studies.[6,26,27] The median age was 45 years old and about 70% patients were male in both training and internal validation cohorts, while median age was 50 years old and 61% patients were male in the external validation cohorts. Hypertriglyceridemia (32.7% and 42%) was the most common etiology in the training and internal validation cohorts. Biliary (57.5%) was the most common etiology in the external validation cohort.

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Developing the prediction nomogram

Candidate variables for the nomogram were drawn from demographic, vital signs, blood and biochemical tests, on admission. The independent prognostic factors for POF after univariate and multivariate logistic regression analysis were age (OR 1.03 [95% CI 1.01–0.05]; $P = 0.01$), respiratory rate (1.25 [1.10–1.42]; $P = 0.001$), albumin (0.92 [0.87–0.98]; $P = 0.013$), lactate dehydrogenase (LDH; 1.002 [1.000–1.003]; $P = 0.036$), oxygen support (5.17 [2.91–9.20]; $P < 0.001$), and pleural effusion (3.61 [1.97–6.61]; $P < 0.001$) (Table 3). These variables were then used to construct the predictive nomogram (Fig. 1a). The total score was calculated as: ([age × 0·3224] – 4.8358]) + ([respiratory rate × 2.5] – 25]) + (43.518 – [0·7912 × albumin]) + (LDH × 0.0196) + 15.521 (if oxygen support) + 12.551 (if exist of pleural effusion). To facilitate the clinical application of our findings, we developed a mobile terminal-based calculator (https://shina.shinyapps.io/DynNomapp/) of the predictive nomogram (Fig. 1b and c).

Table 3 Univariate and multivariate logistic regression analysis with stepwise variable selection

Variables	Univariate analysis		Multivariate analysis	
	OR (95% CI)	*P* value	OR (95% CI)	*P* value
Demographics				
Age, years	1.036 (1.019–1.053)	**<0.001**	1.030 (1.007–1.053)	**0.01**
Gender	1.263 (0.777–2.053)	0.346		
Charlson comorbidity index	0.96 (0.684–1.346)	0.811		
Etiology	0.861 (0.711–1.043)	0.127		
Time to admission, hours	1.005 (0.984–1.026)	0.658		
Admission vital signs				
Body temperature	1.377 (0.902–2.102)	0.138		
Respiratory rate	1.352 (1.207–1.514)	**<0.001**	1.249 (1.099–1.420)	**0.001**
Heart rate	1.029 (1.016–1.042)	**<0.001**	1.006 (0.989–1.023)	0.499
Admission laboratory parameters				
Amylase, IU/l	1.000 (1.000–1.001)	**0.001**	1.000 (1.000–1.000)	0.448
Lipase, IU/l	1.000 (1.000–1.000)	0.096		
White blood cell, 10^9/l	1.112 (1.062–1.165)	**<0.001**	1.085 (0.854–1.379)	0.505
Neutrophils, 10^9/l	1.115 (1.063–1.169)	**<0.001**	0.974 (0.755–1.257)	0.839
Lymphocytes, 10^9/l	0.958 (0.709–1.293)	0.777		
NLR	1.007 (0.996–1.018)	0.186		
Platelet, 10^9/l	1.000 (0.997–1.003)	0.868		
Hematocrits, l/l	2.973 (0.023–377.4)	0.659		
Hemoglobin, g/l	1.011 (1.000–1.022)	0.057		
ALT, IU/l	1.001 (0.999–1.002)	0.247		
AST, IU/l	1.001 (1.000–1.002)	0.125		
Albumin, g/l	0.884 (0.844–0.925)	**<0.001**	0.924 (0.868–0.984)	**0.013**
Glucose, mmol/l	1.063 (1.021–1.106)	**0.003**	1.003 (0.945–1.066)	0.915
Urea, mmol/l	1.219 (1.124–1.321)	**<0.001**	1.105 (0.973–1.256)	0.123
Creatinine, μmol/l	1.012 (1.005–1.018)	**<0.001**	1.001 (0.992–1.009)	0.904
Triglycerides, mmol/l	1.017 (0.993–1.041)	0.162		
Cholesterol, mmol/l	1.041 (0.977–1.109)	0.211		
Lactate dehydrogenase, IU/l	1.003 (1.002–1.004)	**<0.001**	1.002 (1.000–1.003)	**0.036**
Calcium, mmol/l	0.093 (0.033–0.263)	**<0.001**	0.719 (0.174–2.970)	0.648
Oxygen supporting	5.722 (3.486–9.393)	**<0.001**	5.170 (2.905–9.202)	**<0.001**
Pleural effusion	3.802 (2.318–6.237)	**<0.001**	3.611 (1.973–6.610)	**<0.001**

NLR, neutrophil to lymphocyte ratio; LDH, lactate dehydrogenase; ALT, alanine aminotransferase; AST, aspartate aminotransferase.

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Concordance and clinical utility of the nomogram

In the training cohort there was good concordance (C-index) between predicted POF and actual POF at 0.88 (Fig. 2a) and shown by the calibration curve (Fig. 2b). The C-index was 0.91 for the internal validation cohort (Figure 2c and d) and 0.81 for the external validation cohort (Figure 2e and f) compared with the training cohort.

The nomogram of training cohort (Fig. 3a and b), internal validation cohort (Fig. 3c and d), and external validation cohort (Fig. 3e and f) exhibited better or equal clinical utility (DCA) with the other prognostic scores and had good clinical net benefits (CIC) for the identification of SAP patients. Of particular note, the nomogram outperformed all other indices in the internal validation cohort suggesting that it could help clinicians to obtain maximum benefit when making clinical decisions as it showed more benefit than the extreme situation of diagnosing POF in all patients or none.

Prediction of POF, major infection, and mortality by nomogram

Prediction of POF

The pre-test probability of POF in the training, internal validation and external validation cohorts was 9.8% (80/816), 7.5% (30/398) and 20.2% (178/880) patients, respectively (Table 2).

The nomogram had a positive likelihood ratio and post-test probability of developing POF in the training, internal validation and external validation cohorts of 4.26 and 31.7% (259/816), PLR 7.89 and 39.1% (156/398) and PLR 2.75 and 41% (361/880) patients, respectively (Table 4).

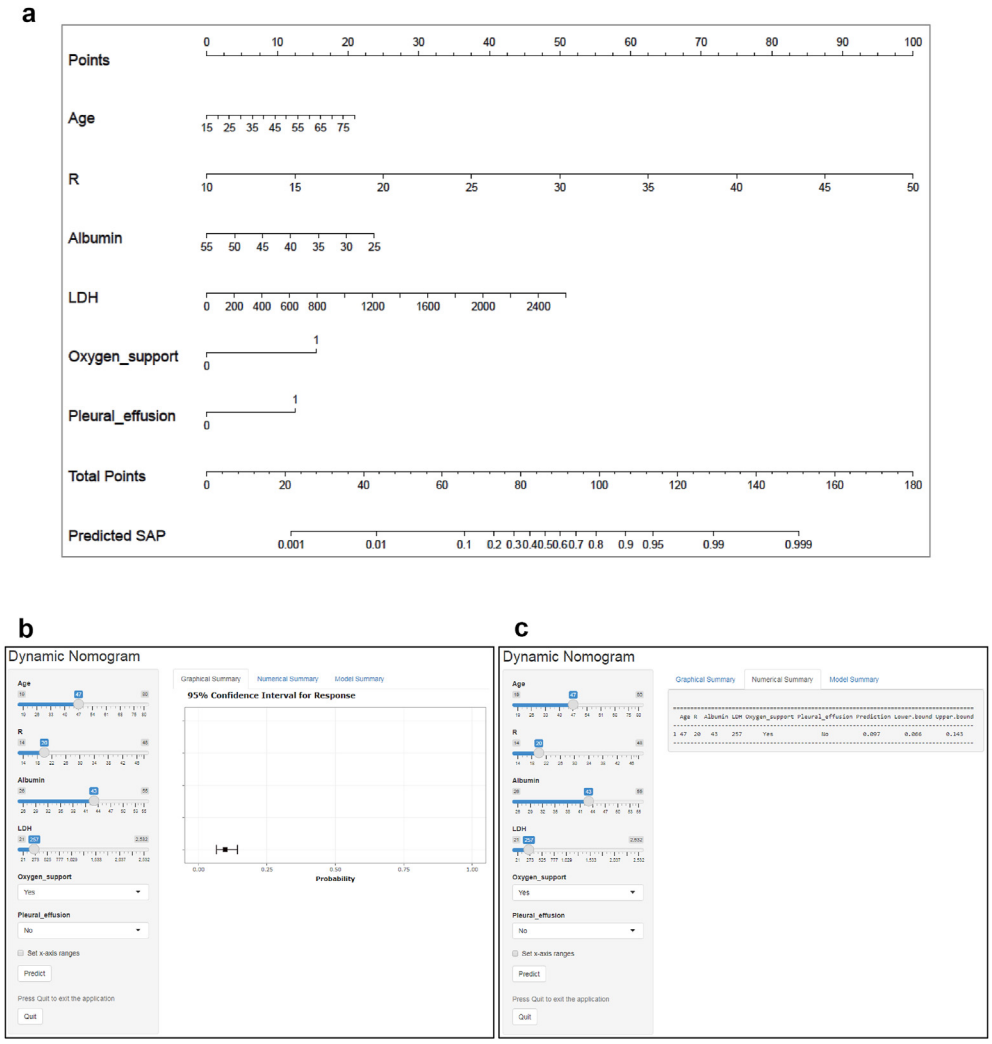

Figure 1 Nomogram for predicting POF in patients with AP and dynamic web-based calculator. Patient prognostic values are located on the axis of each variable; a line is then drawn upwards at a 90° angle to determine the number of points for that particular variable. The sum of these numbers is located on the total points axis, and a line is drawn at a 90° angle downward to the "Predicted SAP" axis to determine the likelihood of POF (a). Alternatively, POF rate can be ascertained from the online calculator with 95% CI in both graphical (b) and numerical (c) summary

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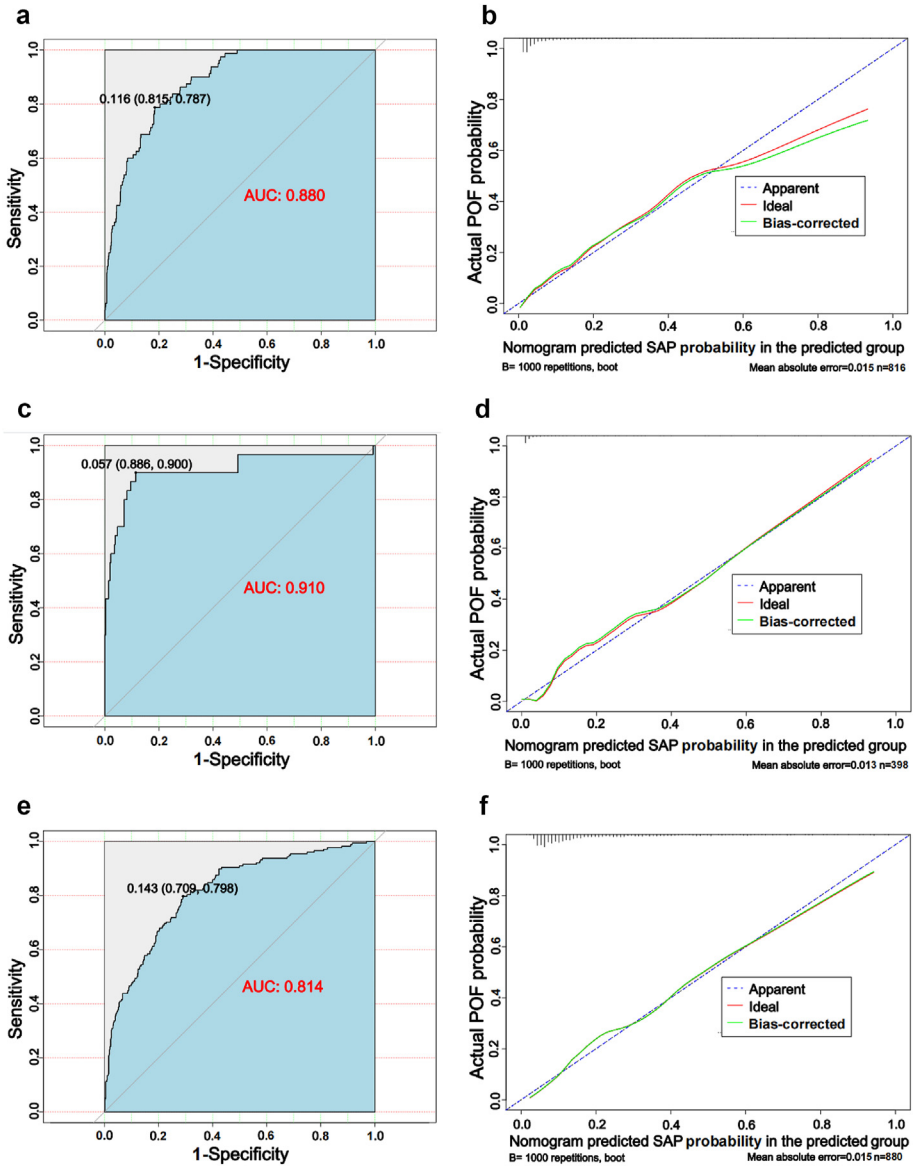

Figure 2 Assessment of the nomogram in all three cohorts. The accuracy of the model was determined using AUC analysis and the distribution of the predicted probabilities presented by calibration curve for cohorts of the training (a, b), internal validation (c, d), and external validation (e, f)

The nomogram had a negative likelihood ratio and post-test probability of not developing POF in the training, internal validation and external validation cohorts of 0.26 and 2.8% (23/816), PLR 0.11 and 0.9% (4/398) and PLR 0.29 and 6.7% (59/880) patients, respectively (Table 4).

Comparison with other prognostic systems for POF

Training Cohort: Comparing with the other prognostic scores, the nomogram had the second highest AUC (AUC 0.88 [0.86–0.91]; the AUC for BISAP was 0.89 [0.87–0.91]. BISAP had the highest predictive value (AUC 0.89 [0.87–0.91], PLR 7.24) for POF compared with the other indices (Table 4; *upper panel*) and followed by the nomogram (AUC 0.88 [0.86–0.91], PLR 4.26), both higher than APACHE II (0.79 [0.76–0.82], PLR 3.61) and Glasgow (0.75 [0.72–0.78], PLR 2.89) ($P < 0.05$, Online Supplementary Table S2) in training cohort.

Internal validation cohort: The nomogram showed the best predictive value (AUC 0.91 [0.88–0.94], PLR 7.89, Table 4; *middle panel*) in internal validation cohort and higher than all other clinical scoring systems ($P < 0.05$; Online Supplementary Table S2), followed by NEWS (0.79 [0.75–0.83], PLR 2.80) and BISAP (0.75 [0.71–0.79], PLR 5.72). In addition, the NLR

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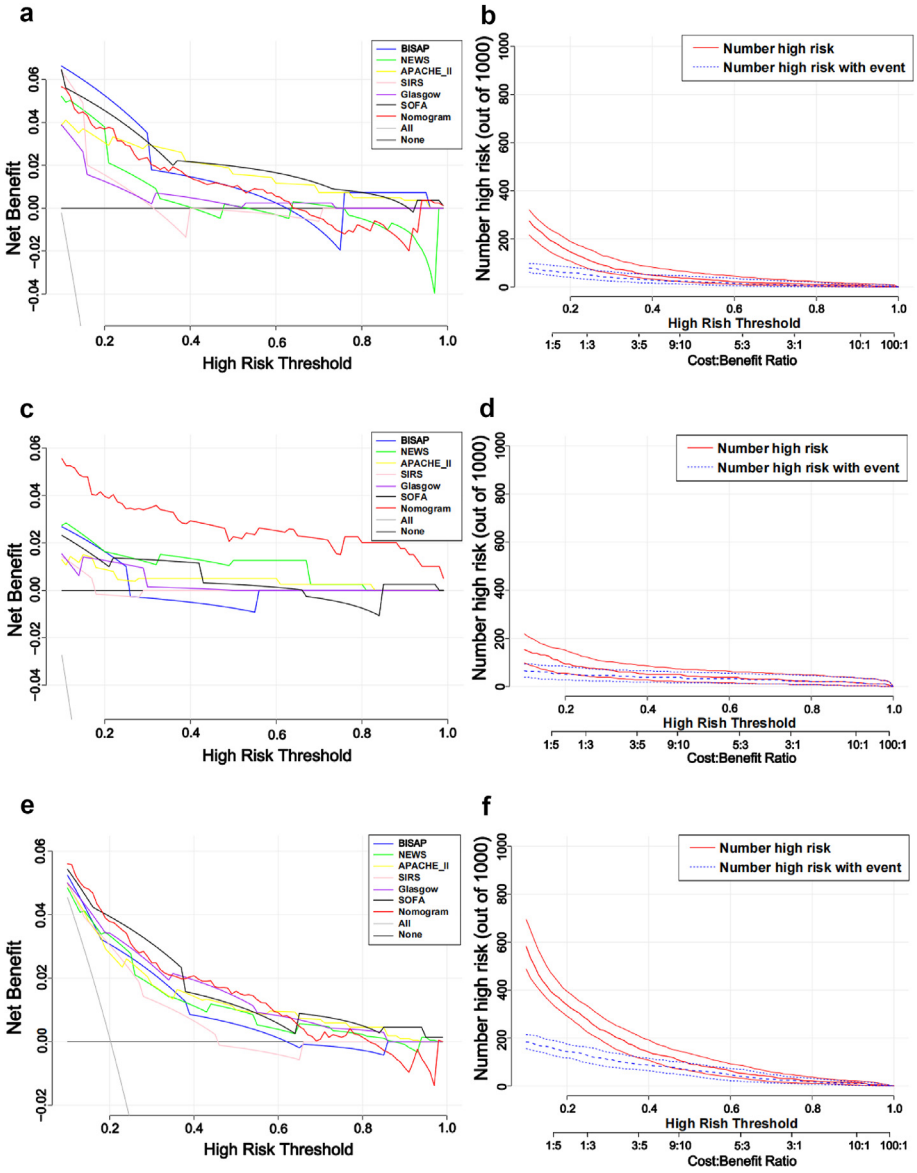

Figure 3 **Clinical utility of the nomogram**. The DCA and CIC of the nomogram for predicting POF in cohorts of training (a, b), internal validation (c, d), and external validation (e, f)

(0.11) of the nomogram ranked the best among all the comparative indices, with the lowest post-test probability of not being POF (0.9%) indicating a high negative prediction value.

External validation cohort: The nomogram also ranked the top AUC (0.81 [0.79–0.84]), lowest NLR (0.29) and post-test probability of not being POF (6.7%) among all the indices in the external validation cohort (Table 4; *lower panel*), with the AUC higher than NEWS, BISAP, APACHE II and SIRS ($P < 0.05$; Online Supplementary Table S2).

Subgroup analyses compared predictive value of the nomogram on POF between different causes of AP patients. The nomogram showed no significant difference in predictive values for predicting POF in both hypertriglyceridemia and non-hypertriglyceridemia (biliary, alcohol and others) patients in all three cohorts (Table 5).

Prediction of major infection

There was 5.5% (45/816), 5.0% (20/398) and 6.0% (53/880) had infection in the training, internal and external validation cohorts, respectively. In the training cohort, the accuracy of these predictors was generally low with BISAP (0.75 [0.72–0.78], PLR 4.29) having the highest predictive value compared with the other indices in training cohort (Online Supplementary Table S3; *upper panel*), but there was no statistical difference between any of them (Online Supplementary Table S2). In both the internal and external validation cohorts, the nomogram showed the best

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Table 4 Predictive value of the nomogram and clinical prognostic scores for persistent organ failure

	AUC	*P* value	Cut-off	Sensitivity (%)	Specificity (%)	PLR	NLR	Post_Prob_Pos (%)	Post_Prob_Neg (%)
Training cohort *(9.8% pre-test probability)*									
Nomogram	**0.88** (0.86–0.91)	<0.001	91.2	78.8	81.5	4.26	0.26	31.7	2.8
NEWS	0.85 (0.82–0.87)	<0.001	4	66.3	88	5.54	0.38	37.6	4
BISAP	**0.89** (0.87–0.91)	<0.001	2	78.8	89.1	**7.24**	0.24	44.0	2.5
APACHE II	0.79 (0.76–0.82)	<0.001	6	61.3	83	3.61	0.47	28.2	4.9
SIRS	0.84 (0.81–0.86)	<0.001	2	91.3	74.6	3.59	0.12	28.1	1.3
Glasgow	0.75 (0.72–0.78)	<0.001	2	61.3	78.8	2.89	0.49	23.9	5.1
SOFA	0.87 (0.84–0.89)	<0.001	1	85	81.2	4.53	0.18	33.0	1.9
Validation cohort									
Internal validation (7.5% pre-test probability)									
Nomogram	**0.91** (0.88–0.94)	<0.001	79.2	90	88.6	**7.89**	**0.11**	39.1	**0.9**
NEWS	**0.79** (0.75–0.83)	<0.001	4	70.0	75.0	2.80	0.40	18.5	3.1
BISAP	0.75 (0.71–0.79)	<0.001	2	46.7	91.9	5.72	0.58	31.7	4.5
APACHE II	0.66 (0.61–0.71)	0.005	6	36.7	89.4	3.46	0.71	21.9	5.4
SIRS	0.68 (0.63–0.72)	<0.001	2	70.0	61.4	1.81	0.49	12.8	3.8
Glasgow	0.69 (0.64–0.73)	<0.001	3	46.7	80.7	2.42	0.66	16.4	5.1
SOFA	0.73 (0.68–0.77)	<0.001	2	63.3	75.8	2.62	0.48	17.5	3.7
External validation (20.2% pre-test probability)									
Nomogram	**0.81** (0.79–0.84)	<0.001	69.4	79.8	70.9	2.75	**0.29**	41	**6.7**
NEWS	0.76 (0.73–0.79)	<0.001	5	62.4	78.6	2.92	0.48	42.5	10.8
BISAP	0.75 (0.72–0.78)	<0.001	2	53.4	84.3	3.41	0.55	46.3	12.3
APACHE II	0.75 (0.72–0.78)	<0.001	9	56.2	80.8	2.92	0.54	42.6	12.1
SIRS	0.73 (0.70–0.76)	<0.001	2	68.0	69.2	2.21	0.46	35.9	10.5
Glasgow	0.78 (0.75–0.81)	<0.001	3	59.6	82.5	3.4	0.49	46.3	11.1
SOFA	0.80 (0.77–0.83)	<0.001	2	63.5	85.0	4.24	0.43	51.8	9.8

AUC, area under curve; PLR, positive likelihood ratio; NLR, negative likelihood ratio; Post_Prob_Pos, post-test probability of a positive test; Post_Prob_Neg, post-test probability of a negative test; NEWS, National Early Warning Score; BISAP, Bedside Index for Severity in Acute Pancreatitis; APACHE II, Acute Physiology and Chronic Health Examination II; SIRS, Systemic Inflammatory Response Syndrome; SOFA, Sequential Organ Failure Assessment.

predictive value (highest AUC 0.78 [0.73–0.82]/0.80 [0.77–0.83]; lowest NLR 0.47/0.22; lowest post-test probability of not being major infection 2.4%/1.4%; Online Supplementary Table S3; *middle and lower panels*).

Prediction of mortality

There was 1.2% (10/816), 1.0% (4/398) and 2.3% (20/880) mortality in the training, internal validation and external validation cohorts, respectively. All the deaths were from patients with POF in the three cohorts (12.5%, 13.3% and 11.2%; Online Supplementary Tables S1a–c). In both the training and external cohorts, the nomogram (AUC 0.88 [0.85–0.90], 0.89 [0.87-0.91]) showed the equivalent predictive values for mortality with most of the clinical scoring systems (Online Supplementary Table S4). In the internal validation cohort, the nomogram had the highest predictive values (0.99 [0.98–1.00], PLR 32.8, NLR 0.26) for mortality, followed by BISAP (0.95 [0.92–0.97], PLR 9.85), and NEWS (0.90 [0.86–0.93], PLR 29.6, NLR 0.26), all higher than the remaining clinical scores (Online Supplementary Table S4).

Data integrity

Since there was substantial amount of missing data, we assessed whether the patients included in all cohorts were different from those who were excluded due to missing data. There were no significant differences in age, hypertriglycedemic etiology, persistent organ failure, major infection, and mortality (Online Supplementary Table S5).

Discussion

In this study, we developed and validated a mobile terminal-based nomogram for predicting POF, major infection, and mortality using six independent prognostic factors (age,

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Table 5 Accuracy of nomogram in predicting POF between hypertriglyceridemia and non-hypertriglyceridemia associated-acute pancreatitis patients

Etiology	Pre-test probability	AUC	*P* value	Cut-off	Sensitivity (%)	Specificity (%)	PLR	NLR	Post_Prob_Pos (%)	Post_Prob_Neg (%)	*P* value
Training cohort											
HTG	12.4%	0.89 (0.84–0.92)	<0.001	71.7	100	62.0	2.63	0.0	27.0	0.0	0.998
Non-HTG	8.56%	0.89 (0.86–0.91)	<0.001	91.2	83.0	78.7	3.89	0.22	26.7	2.0	
Validation cohort											
Internal validation cohort											
HTG	8.98%	0.91 (0.86–0.95)	<0.001	82.5	93.3	90.8	10.13	0.07	50.0	0.7	0.970
Non-HTG	6.49%	0.92 (0.87–0.95)	<0.001	79.2	86.7	90.3	8.91	0.15	38.2	1.0	
External validation cohort											
HTG	25%	0.86 (0.80–0.91)	<0.001	76.3	76.1	82.6	4.37	0.29	59.3	8.8	0.115
Non-HTG	19%	0.80 (0.77–0.83)	<0.001	69.6	77.3	70.9	2.66	0.32	38.3	7.0	

POF, persistent organ failure; AUC, area under curve; PLR, positive likelihood ratio; NLR, negative likelihood ratio; Post_Prob_Pos, post-test probability of a positive test; Post_Prob_Neg, post-test probability of a negative test; HTG, hypertriglyceridemia.

respiratory rate, albumin, LDH, oxygenation, and pleural effusion) that readily available on admission. This nomogram was found to be superior with the both internal and external validation cohort compared with other prognostic scores recommended for clinical use for predicting POF and major infection. Subgroup analysis comparing hypertriglyceridemia and non-hypertriglyceridemia etiology did not significantly change the predictive values of the nomogram for predicting POF, indicating its suitability for heterogenous etiologies. As POF is the diagnostic criteria for SAP, this nomogram is recommended for routine clinical use to predict SAP on admission or to rule it out (validation NLR 0.11/0.29). Therefore, these findings encourage the use of the simple-to-use web-based nomogram before new markers are developed and introduced in our settings. The consecutive nature of patient recruitment and short time from onset of pain to admission, stringently applied in two large different Chinese centers, adds strength to these conclusions.

Age is recognized as an individual risk factor for increased severity of AP and has been used by several prognostic scores[10,11] and practice guidelines.[41] To investigate the role of age and comorbidity in the severity of AP, Frey *et al.*[42] carried out a retrospective study in 84,713 patients with a first-attack AP. They found that the 65 to 75 age group, and age >75 are strong predictors of early death with an odds ratio (OR) of 2.6 and 5.2, respectively. Similar findings also applied to patients with two chronic comorbidities (OR: 3.5) or $\geq$ 3 comorbidities (OR: 7.4). Moreover, the mortality rate was only 0.1% (14/14,280) for younger patients (age <55) without chronic comorbidities compared to 5.9% (701/24,852) for elderly patients (age >64) with $\geq$3 comorbidities in the first 14 days. In addition, they showed that recent cancer, heart failure, and renal and liver diseases are strongly correlated with outcomes. Further, in acute interstitial AP, which is known to have low mortality, the Charlson comorbidity index was strongly associated with adverse clinical outcomes.[43] A recent review has concluded that age is associated with frequent cases of severe AP, leading to increased multiorgan failure and high mortality.[44] Because of the significant impact of the degree and number of comorbidities on clinical outcomes, we therefore excluded patients with advanced (end-stage) comorbidities with an emphasizing on assessing the intrinsic prognostic factors for AP severity.

Hypoalbuminemia occur in critically ill patients due to several factors including dilution from resuscitation, increased interstitial loss, altered liver function, and catabolic nutritional state.[45] It is strongly associated with poor clinical outcomes in acutely ill patients and it has also been shown to independently associated with POF and mortality in AP patients.[46] Whitcomb *et al.*[47] has recently found that albumin dropped rapidly in AP patients with multiple organ failure resulting in unregulated capillary leak with continued loss of larger plasma proteins. In contrast, the plasma albumin levels only dropped slightly in patients without multiple organ failure who tended to recover quickly unless they develop complications (infected pancreatic necrosis or sepsis). The therapeutic effect of albumin in inflammatory states is not only by affecting plasma volume dilation, but also by regulating inflammation and oxidative stress.[48] Hypalbuminemia has recently been shown to be associated with severe AP and increased mortality in a cohort of 1149 patients.[49] This is consistent with the inclusion of serum albumin level in a number of prognostic indices, including the Glasgow criteria.[16]

Raised lactate levels has been observed in many critical acute illness situations including sepsis[50] and AP.[51] Elevated lactate may serve as a protective mechanism and has been shown to reduce Toll-like receptor and inflammasome-mediated pancreatic and liver injury via its receptor GPR81. LDH can reversibly catalyze the oxidation of lactate to pyruvate and has been employed by Ranson, Glasgow, Japanese Severity Score,[11] and a nomogram[15] for early AP severity prediction and reported as a

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simple and useful parameter for predicting POF[52] and pancreatic necrosis.[53]

Respiratory rate and oxygen support reflect respiratory status and with respiratory failure as one most common organ dysfunction in AP,[6] the early recognition of respiratory dysfunction is considered important. Our results showed a positive association between the development of POF and an increased respiratory rate or requirement for oxygen support on admission. Therefore, both respiratory rate and oxygen support have been adopted in NEWS[34] for their convenience and prognostic value. The significance of pleural effusion in AP patients has been long reported, and is part of BISAP.[54] Our results showed that pleural effusion (odds ratio: 3.61 95%CI 1.97–6.6, $P < 0.001$) was an independent predictor for SAP, consistent with a previous study.[55]

In our univariate analysis before establishing the predictive nomogram, we also found white blood cell count, glucose, urea (or blood urea nitrogen), creatinine, and ionized calcium were independent individual prognostic factors for POF, consistent with previously published literature.[11] However, when these parameters were fitted into our multiple logistic regression model, they only had negligible impact on the final nomogram. Unlike most previously published studies included high proportion of SAP patients, we used a training consecutive cohort constituted only up to 10% of SAP patients which may partially explain different weights of individual prognostic factors in varied epidemiology situations. For example, three of the four existing predictive nomograms for AP severity were conducted in the ICU settings which cannot be generalizable for emergence departments or general wards where patients are primarily admitted.

Some of the six independent prognostic factors of our nomogram are included as part of NEWS (respiratory rate and oxygen support) and BISAP (age, respiratory rate, and pleural effusion), the two that we found had justifiable good predictive values for POF in both our training and internal validation cohorts. However, the nomogram was simpler than BISAP and more AP-specific than NEWS to quantitatively predict clinical outcomes in a personalized way. In addition, we validated the nomogram in another Chinese tertiary hospital with the etiological composition different from ours. The results showed that the nomogram with stable clinical applicability in both AP cohorts with hypertriglyceridemia (internal validation) or biliary (external validation) as the main etiology, adding strength to its applicability.

Our study also has some limitations. Firstly, excluding missing data may contribute to varied accuracy of the nomogram. However, we found majority of the key parameters of baseline and outcomes, albeit not all (i.e. gender and length of hospital stay), were similar between those included and those with missing data in all cohorts. Secondly, the nomogram model was developed mainly based on the variables that were easy to get in our retrospective sets, but did not include other factors that may influence the precision of the model. For example, the oxygenation index was not included in our analysis because only paucity data were available. In a most recent study,[56] the authors found that oxygenation index had low prognostic power (AUC 55.3%) for acute respiratory distress syndrome. Thirdly, the nomogram did not have specific markers for circulatory and renal failure. The reasons for this may be attributed to low incidence of circulatory and renal failure of the study population and at the early disease stage respiratory failure commonly precedes other organ failures.[4,5] In the last, the lack of international validation may limit the extrapolation and generalizability of the nomogram.

Conclusions

Our nomogram based on six readily available factors accurately predicted POF on admission in patients with AP. This nomogram can be routinely used for early AP severity prediction in our clinical practice if further validated in multiple center studies.

Authors' contributions

QX, WH, and WHe obtained funding, and concepted, designed and supervised the study. NS collected, analyzed all retrospective and prospective data of Chengdu center, and drafted the manuscript. XZ collected and analyzed all retrospective data. WHe, LX, YZ, and NL provided and analyzed external validation datasets. LL, WC, LY, XY (Xinmin Yang) and RZ supported collecting data. PZ assisted data analysis. LD, TJ, ZL and KJ audited data quality. GJ and XY (XiaonanYang) supervised the patients' treatment. WH and QX designed, TW and PS assisted, RS reviewed the proforma and e-database. VKS, RS, and JAW had important intelligence input. WH and JAW critically revised the manuscript. All authors read and approved the final manuscript.

Ethics approval and consent to participate

This study was approved by the Institutional Review Board of West China Hospital of Sichuan University and The First Affiliated Hospital of Nanchang University.

Consent for publication

All authors have provided consent for publication of the manuscript.

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

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Conflicts of interest

None to declare.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.hpb.2022.05.1347.

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