Research Square

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Development and validation of a predictive model for predicting acute kidney injury in patients with acute pancreatitis

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Keywords: acute kidney injury, acute pancreatitis, risk factor, nomogram

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Abstract

Background: Acute kidney injury is a serious complication of acute pancreatitis and significantly increases the risk of mortality. Early identification of acute kidney injury is important for medical interventions and care options. The aim of this study is to develop a predictive model that could rapidly identify high-risk population of acute kidney injury in patients with acute pancreatitis.

Methods: Totally 808 patients with acute pancreatitis admitted to our center from January 2015 to May 2019 were included in the study and were divided into the training (n =566) and validation (n = 242) cohorts randomly, in a 2:1 ratio. The least absolute shrinkage and selection operator (LASSO) regression was used for data dimension reduction and feature selection; then, multivariable logistic regression analysis was used to develop the prediction model. The performance of this nomogram was evaluated with calibration and validated in the validation set. Decision curve analysis was applied to evaluate the clinical usefulness of this model.

Results: Five potential predictors (BMI, RANSON score, serum uric acid, triglycerides and lactate) from 53 high-dimensional clinical variables were incorporated to develop the prediction model of acute kidney injury. The nomogram demonstrated valuable prediction performance with AUROC of 0.994 and 0.996 in the training and validation cohorts, respectively. Individual risk probability was visually scored. The nomogram achieved fine calibration and good clinical usefulness.

Conclusions: The proposed nomogram can help to identify high-risk population of acute kidney injury in patients with acute pancreatitis and facilitate timely individualized clinical decision-making.

Keywords: acute kidney injury; acute pancreatitis; risk factor; nomogram

Background

Acute pancreatitis is the most frequent gastrointestinal cause for hospitalization and one of the leading causes of in-hospital deaths [1, 2]. Acute kidney injury (AKI) is a common complication of acute pancreatitis, occurs in almost 70% of cases of severe acute pancreatitis[3]. The development of acute kidney injury in patients with acute pancreatitis significantly increases the risk of death[4]. Accurate prediction of acute kidney injury in the early stage of acute pancreatitis is important for individualized clinical decision-making and a favorable prognosis.

The exact mechanism of acute kidney injury in acute pancreatitis is complex and the pathophysiology is still incompletely understood[5]. A series of factors have been shown to be associated with acute kidney injury including intra-abdominal hypertension[6], renal hypoperfusion[7, 8], systemic inflammatory response[9], disturbed microcirculation[10] and endothelial dysfunction[11].

To our knowledge, there is still no effective method to predict the occurrence of acute kidney injury in patients with acute pancreatitis. Conventional laboratory examinations have their own limitations and

cannot provide sufficient information for timely diagnosis. A nomogram, derived from predictive models, is considered as a reliable tool for predicting the probability of risk by incorporating comprehensive clinical factors. Therefore, we developed and validated a nomogram for the prediction of acute kidney injury in the patients with acute pancreatitis.

Patients And Methods

Patients with acute pancreatitis admitted to the First Affiliated Hospital of Dalian Medical University (Dalian City, Liaoning Province, China) within 72 h of symptom onset from January 2015 to May 2019 were retrospectively included in the study. The diagnosis and the severity of acute pancreatitis were defined based on the 2012 revision of the Atlanta classification and definitions by international consensus[12], where patients must meet two of the following three criteria: (a) abdominal pain consistent with acute pancreatitis, (b)level of serum lipase or amylase at least three times greater than the upper limit of normal, and (c) characteristic findings of acute pancreatitis on abdominal ultrasonography and/or computerized tomography (CT) scan. The diagnosis of acute kidney injury (AKI) is according to the Kidney Disease Improving Global Outcomes criteria,defined as an absolute increase ($\geq$ 26.4 μmol/L or $\geq$ 50% baseline serum creatinine) in serum creatinine and a decrease in urine output (documented oliguria of < 0.5 mL/kg per hour for > 6 h) within 48 h.

Exclusion criteria were as follows: recurrent pancreatitis, post-ERCP pancreatitis, chronic pancreatitis, malignant gastrointestinal tumor, chronic renal disease, liver disease, pregnancy, intoxication and death during admission. Patients who had developed organ failure before data collection were also excluded.

The patients were randomly assigned into two groups in a 2:1 ratio, using a permuted randomization protocol. Two thirds of the patients were assigned to the training cohort for the nomogram development, and one-third of them were assigned to the validation cohort for the validation of the nomogram.

Data Collection

Data collected included personal characteristics (age, sex, BMI, etiology, and alcohol intake), laboratory studies (blood biochemical indicators and complete blood counts), and abdominal computed tomography (CT) scan.

The etiology of acute pancreatitis mainly includes biliary pancreatitis, hyperlipidemic pancreatitis, alcoholic pancreatitis and idiopathic pancreatitis. Biliary pancreatitis was diagnosed if imaging studies (computed tomography, magnetic resonance imaging, or ultrasonography) showed gallstones or choledocholithiasis. Hyperlipidemic pancreatitis was defined as triglyceride levels greater than 11.3 mmol/L or triglyceride levels greater than 5.65 mmol/L with grossly lipaemic serum. Alcoholic pancreatitis was diagnosed if the patient had a history of heavy alcohol consumption before the onset of acute pancreatitis and a history of alcohol consumption of over 50 g/d for longer than 5 years.

Several multi-factorial scoring systems such as Ranson score[13], Acute Physiology and Chronic Health Evaluation (APAC HE II) [14], computed tomography severity index (CTSI)[15] and the bedside index for severity in acute pancreatitis (BISAP) [16] have been used for estimating the severity of AP. Hematological data for every variable of BISAP, APACHE II, CTSI, Ranson score were collected if available.

BISAP and APACHE II scores were calculated within first 24 hours after admission. Ranson score was evaluated within the first 48 hours. CTSI was obtained from the report of the CT scan which was done after 72 hours and within 7 days. The reporting of the scans was done by same set of radiologists.

Statistical analysis

Categorical variables were described using frequencies and proportions and compared using $\chi 2$ tests. Continuous values were expressed using mean ± standard deviation (SD), or median and interquartile range (IQR) and compared using Student's t test or the nonparametric Mann–Whitney test. The logistic regression model was used to estimate the odds ratio (OR) and 95% CI of the risk of AP-AKI.

The least absolute shrinkage and selection operator (LASSO) method, which is suitable for the regression of high-dimensional data[17], was used to select the most useful predictive features from the training data set in this study. All variables were reduced to limited potential predictors on the basis of 566 patients in the development cohort using the LASSO regression model. We selected the optimal λ in the LASSO model by using 10-fold cross-validation via minimum criteria and one standard error of the minimum criteria (the 1-SE criterion). Then, the model was refit by using all

of the nonzero coefficients, which were selected by LASSO method. The AP-AKI risk score was calculated for each patient as a linear combination of selected features that were weighted by their respective coeffcients.

Nomogram was developed according to the logistic regression. The performance of this model was tested in an independent validation cohort. To quantify the discrimination performance of this model, the receiver operating characteristics (ROC) curve and the area under the curve (AUROC) were calculated[18]. A predictor with an AUC above 0.7 was considered to be useful, while an AUC between 0.8 and 0.9 indicated good diagnostic accuracy. Calibration curves were generated to evaluate the agreement between the predicted probabilities based on the nomogram and the actual values, accompanied with Hosmer-Lemeshow (H-L) chi-square statistics ($P > 0.05$ supports the goodness of calibration). Decision curve analysis was performed to quantify the net benefits at different risk threshold probabilities.

All statistical analyses were implemented using R statistical software version 3.4.2. LASSO logistic regression was performed using the "glmnet" package. The ROC curves were plotted using the "pROC" package. Logistic regression, nomogram construction and calibration plots were performed with the "rms" package. A two-sided P value < 0.05 was considered significant. Decision curve analysis was conducted by using R package "rmda".

Results

Baseline characteristics

Overall, 808 eligible patients were included in the analysis(Fig. 1). There were 566 patients (mean age 59.3 ± 17.9, 54.9% male) in the training set and 242 patients (mean age 61.5 ± 16.8, 61.5% male) in the validation set. The baseline characteristics of the training set and the validation set are listed in Table 1. Ninety-two patients(16.2%) in the training set developed acute kidney injury while 33 patients（13.6%）in the validation set developed acute kidney injury.

Table 1

Baseline characteristics of patients in the Training and validation cohorts

variables	Training Cohort(n = 566)	Validation Cohort(n = 242)
AKI		
yes,n(%)	92(16)	33(14)
no,n(%)	474(84)	209(86)
Gender		
male,n(%)	311(55)	149(62)
female,n(%)	255(45)	93(38)
Smoking		
yes,n(%)	147(26)	93(38)
no,n(%)	419(74)	149(62)
Alcohol intake		
yes,n(%)	223(39)	109(45)
no,n(%)	343(61)	133(55)
HBP		
yes,n(%)	169(30)	80(33)
no,n(%)	397(70)	162(67)
Diabetes		
yes,n(%)	123(22)	55(23)
no,n(%)	443(78)	187(77)
CHD		
yes,n(%)	129(23)	75(31)
no,n(%)	437(77)	167(69)
Etiology		
Biliary,n(%)	77(14)	38(16)
Alcohol,n(%)	384(68)	153(63)
Hypertriglyceridemia,n(%)	89(16)	40(17)
Idiopathic,n(%)	14(2)	11(4)

variables	Training Cohort(n = 566)	Validation Cohort(n = 242)
Bisap		
0,n(%)	9(2)	5(2)
1,n(%)	187(33)	75(31)
2,n(%)	238(42)	106(44)
3,n(%)	71(12)	23(10)
4,n(%)	43(8)	30(12)
5,n(%)	18(3)	3(1)
CTSI		
0,n(%)	46(8)	23(10)
1,n(%)	145(26)	56(23)
2,n(%)	203(35)	80(33)
3,n(%)	99(17)	50(21)
4,n(%)	50(9)	20(8)
5,n(%)	3(1)	6(2)
6,n(%)	17(3)	4(2)
7,n(%)	3(1)	3(1)
Ranson score		
1,n(%)	259(46)	117(48)
2,n(%)	118(21)	48(19)
3,n(%)	65(11)	24(10)
4,n(%)	60(11)	19(8)
5,n(%)	27(5)	23(10)
6,n(%)	29(5)	7(3)
7,n(%)	8(1)	4(2)
Age, years (mean ± SD)	59.32 17.97	61.59 16.81
SBP,mmHg (mean ± SD)	134.92 12.41	136.26 12.96

variables	Training Cohort(n = 566)	Validation Cohort(n = 242)
DBP,mmHg (mean ± SD)	73.68 8.78	75.43 8.30
BMI, kg/m2 (mean ± SD)	25.72 1.06	25.67 1.07
APACHEII (mean ± SD)	7.50 5.53	7.52 5.43
Na, mmol/L (mean ± SD)	138.39 4.89	138.30 4.68
K, mmol/L	3,98 0.58	4.01(3.70,4.32)
Hct,% (mean ± SD)	40.65 6.91	41.06 5.67
WBC, 109/L (mean ± SD)	12.32 5.36	12.58 5.56
BUN, mmol/L (mean ± SD)	6.47 4.97	6.41 4.10
BG, mmol/L (mean ± SD)	9.09 5.02	9.47 5.76
AST, U/L (IQR)	70(30,175)	75.50(29.00,165.75)
Ca, mmol/L	2.06 0.25	2.12(2.00,2.22)
D-Dimer, μg/L (IQR)	917(640,1880)	871.00(577.25,1500.00)
Amy, U/L (mean ± SD)	958.15 869.45	870.64 806.85
LPS,U/L	2040(1124,3390)	2774.61 2591.72
CK-MB, μg/L (IQR)	0.66(0.26,1.71)	0.66(0.30,1.60)
TnI, μg/L (IQR)	0.02(0.006,0.058)	0.02(0.01,0.04)
PT,s	12.3(11.5,13.3)	12.64 2.52
TT,s (mean ± SD)	18.76 13.92	17.02 2.87
APTT,s(mean ± SD)	27.54 13.29	27.08 9.97
ALT, U/L (IQR)	84(27.75,248.50)	81.25(28.75,242.50)

variables	Training Cohort(n = 566)	Validation Cohort(n = 242)
γ-GGT, U/L (IQR)	138.50(53.00,377.00)	156.50(49.75,384.25)
ALP, U/L (mean ± SD)	126.30 91.96	130.96 121.48
T-BIL, μmol/L (mean ± SD)	25.94 17.64	26.51 16.64
D-BIL, μmol/L	7.65(4.50,12.63)	10.20 8.65
LDH,U/L (mean ± SD)	187.04 50.79	188.47 60.90
LDL-C, mmol/L (IQR)	2.07(1.59,2.85)	2.05(1.57,2.85)
HDL-C, mmol/L (mean ± SD)	1.30 0.35	1.36 0.34
UA, μmol/L (mean ± SD)	329.59 109.87	325.38 115.15
TC, mmo/L (IQR)	4.21(3.65,4.87)	4.19(3.65,4.82)
TG, mmo/L (IQR)	1.26(0.89,1.76)	1.21(0.96,1.75)
ALB, g/L (mean ± SD)	36.61 7.18	35.78 8.53
ALB/GLO(IQR)	1.40(1.17,1.70)	1.30(1.12,1.60)
Lac, mmol/L	1.74 1.17	1.42(0.92,1.82)
RBC,1012/L (mean ± SD)	4.55 0.83	4.50 0.89
PLT, 109/L (mean ± SD)	208.39 86.77	197.28 77.98
Hemoglobin,g/L (mean ± SD)	134.02 24.30	133.67 34.47
PaO2,mmHg (mean ± SD)	80.14 12.20	80.48 13.58
PH (mean ± SD)	7.48 2.80	7.37 0.14
PaCO2,mmHg (mean ± SD)	39.09 15.94	40.83 23.06
SaO2,% (mean ± SD)	93.82 8.98	93.31 11.30

variables	Training Cohort(n = 566)	Validation Cohort(n = 242)
SB, mmol/L (mean ± SD)	23.49 10.67	24.52 13.62
BE, mmol/L (IQR)	-1.60(-3.82,1.1)	-1.60(-4.23,1.33)
BNP, pg/ml (IQR)	48.65(32.29,70.40)	46.80(31.90,70.40)
AaDO2 (IQR)	3.77(3.25,4.29)	3.68(3.13,4.22)
PCT, ng/ml(IQR)	0.086(0.030,0.398)	0.06(0.03,0.43)
HBP = high blood pressure; DM = diabetes mellitus; CHD = coronary heart disease; BISAP = bed-side index for severity of acute pancreatitis; CTSI = computed tomography severity index; SBP = systolic blood pressure; DBP = diastolic blood pressure;		
BMI = Body Mass Index; APACHEII = acute physiology and chronic health evaluation II; PaO2 = arterial partial pressure of oxygen; PH = Pondus Hydrogenii; Na = sodium; K = Potassium; Hct = hematocrit; WBC = white blood cell; BUN = blood urea nitrogen;		
BG = blood glucose; AST, aspartate amino-transferase; Ca = calcium; Amy = amylase; LPS = lipase; TnI = troponin I; PT = prothrombin time; TT = thrombin time; APTT = activated partial thromboplastin time; ALT = alanine amino-transferase; γ-GGT = γ-glutamyltranspeptadase; ALP = alkaline phosphatase; T-BIL = total bilirubin; D-BIL = direct bilirubin; LDH = lactate dehydrogenase; LDL-C = Low-density lipoprotein cholesterol; HDL-C = High-density lipoprotein cholesterol; UA = Uric Acid; TC = total cholesterol; TG = triglyceride; ALB = albumin; ALB/GLO = albumin/globulin; Lac = lactic acid; RBC = red blood cell; PLT = Platelets; Hb = hemoglobin; PaCO2 = partial pressure of carbon dioxide in artery; SaO2 = arterial oxygen saturation; SB = standard bicarbonate; BE = bases excess; BNP = B-type natriuretic peptide; PCT = procalcitonin; AaDO2 = alveolar-arterial oxygen tension difference; IQR = inter-quartile range		

Development of the prediction model

We used LASSO binary logistic regression for building the model. A total of 53 high-dimensional clinical variables were incorporated in the LASSO regression and nine potential predictors were selected on the basis of the training set (Fig. 2).The ten candidate predictors were included into multivariate logistic regression analysis, five variables entered the risk prediction model, including BMI, RANSON score, serum uric acid, triglycerides and lactate. Based on these results, a predictive nomogram was developed to predict probability of acute kidney injury after the first-attack of pancreatitis (Table 2 and Fig. 3).

Table 2
Multivariate logistic regression of acute kidney injury

variables	Odds Ratio(95%CI)	P
BMI	31.20(3.91, 573.82)	0.005
Ranson score	14.46 (5.60, 67.19)	< 0.001
UA	33.18 (5.48, 357.66)	0.001
TG	20.84 (1.55, 529.22)	0.035
Lac	71.55 (8.91, 1149.50)	< 0.001
BMI = Body Mass Index; UA = Uric Acid; TG = triglyceride; Lac = lactic acid;		

Validation of the AKI nomogram

The nomogram had good performance in acute kidney injury prediction. Figure 4 showed the validation of the nomogram by the receiver operating characteristic (ROC) curves. The area under the curve (AUC) for the training and validation set was 0.994 (95% confidence interval [CI]: 0.9906–0.9983) and 0.996 (95% confidence interval [CI]: 0.9903-1.0000), respectively. The calibration curves indicate a perfect model in which the predicted probabilities are identical to the actual outcomes (Fig. 5). The Hosmer-Lemeshow test demonstrated a non-significant statistic in each set (P > 0.05) which suggested that the model was well calibrated. Decision curve analysis (DCA) was applied in this study and showed that, if the threshold probability is between 1% and 99%, using the nomogram for prediction of acute kidney injury added more benefit than treating either all or no patients (Fig. 6).

Discussion

Acute kidney injury is a well-known complication of acute pancreatitis. It occurs in almost 70% of cases of severe acute pancreatitis [3].The development of acute kidney injury in patients with severe acute pancreatitis significantly increases the risk of hospital mortality [3, 19, 20] and carries a very poor prognosis[21, 22]. In recent years, blood purification treatment plays a significant role in the comprehensive treatment of acute pancreatitis[23, 24]. It has been reported that early hemofiltration could improve the treatment efficacy and decrease mortality rate in patients with pancreatitis complicated by acute renal failure[25, 26]. Therefore, early identification of acute kidney injury in the course of acute pancreatitis would be of great value in helping clinicians triage patients to the appropriate level of care and guide clinical practice in the management of acute pancreatitis.

A nomogram is accepted as a reliable tool to predict risk by illustrating important predictors for clinical events. To our knowledge, there are still no studies on the prediction of acute kidney injury in acute pancreatitis at present. We therefore conducted this study to develop and validate a nomogram for the

prediction of acute kidney injury associated with acute pancreatitis, which can visually score individual risk and allow early identification of patients at high risk.

As is shown in our study, the overall prevalence of acute kidney injury is 15.5%, in which the incidence of the training set is 16.3%, and the incidence of the validation set is 13.6%, in line with the literature reports [27, 28]. This nomogram includes five readily available indices, including BMI, RANSON score, serum uric acid, triglycerides and lactate. The proposed model achieved sufficient accuracy and good clinical usefulness.

The mechanisms underlying acute kidney injury complicating acute pancreatitis have not been completely understood,but appears to result from initial hypovolemia followed by complex interactions between inflammatory, vascular, and humoral factors. Many studies have identified that obesity is associated with an increased risk of kidney failure, local complications and mortality in patients with acute pancreatitis[29]. The body mass index (BMI) is a measure of obesity and a higher BMI was independently associated with increased risks of acute kidney injury[30]. Hyperlipidemia, especially high circulating concentrations of triglycerides, can lead to the development of severe and systemic complications in patients with acute pancreatitis[31, 32]. Hyperuricemia is linked to metabolic syndrome[33] and has been shown to predict kidney disease onset and progression[34, 35]. A recent study has shown that an elevated lactate level is closely related to persistent organ failure in acute pancreatitis[36]. Over the past few decades, several multi-factorial scoring systems based on clinical and biochemical data have been used for assessing the severity of acute pancreatitis[37, 38]. These include Ranson's score, BISAP, CTSI and APACHE II to name a few[39]. In agreement with these observations, which are based on extensive clinical data, this nomogram incorporated five factors as predictors of acute kidney injury: BMI, RANSON score, serum uric acid, triglycerides and lactate.

To develop a simple but efficient predictive model, we utilized the LASSO method to data dimension reduction and screen the optimized predictors. This method surpasses the methods using the strength of univariate differences with outcome and enhance the accuracy and interpretability of the predictive model [17, 40]. Validation of the nomogram is important to avoid overfitting and to determine generalizability. In this study, the AUROC in the training and validation cohorts demonstrated adequate discrimination power (0.994 and 0.996, respectively). Calibration curve showed optimal agreement between predicted and actual observations, which suggested that the nomogram was quite predictive. Decision curve analysis was performed to determine the clinical usefulness and demonstrated that, if the threshold probability is between 1% and 99%, using the nomogram for prediction of acute kidney injury added more benefit than treating either all or no patients. The clinical value of the nomogram is to make a comprehensive evaluation of risk and provides insights into personalized decision-making, especially for high-risk population.

Our analysis has a few advantages. First, the nomogram is practical because all the variables included are easily and routinely collected in clinical practice and it may take less time to calculate individual risk score. Second, the data was collected on a relatively large population of acute pancreatitis cohort and

candidate risk factors included were very comprehensive, which improves the application value of the prediction model. In addition, the discrimination and calibration validation of the model ensured our model of strong evidence to predict the acute kidney injury in acute pancreatitis. Decision curve analysis demonstrated our prediction model have good clinical usefulness.

As the first study of this kind, there is no similar model for reference, the current study also has several limitations. Firstly, as a retrospective study, we cannot avoid potential biases. Secondly, this study was conducted in a single center, with a relatively small sample size and only internal validation, the results may not be widely generalizable in other regions and races. In the next step, we will focus on conducting a prospective multi-center research for enrolling much larger sample cases. Finally, we didn't include novel biomarkers reported in recent studies because they are not yet widely used clinically. The proposed nomogram may be further optimized after incorporating more valuable variables such as serum uromodulin, neutrophil gelatinase-associated lipocalin (NGAL).

Conclusions

We developed a risk prediction model which can help to identify high-risk population of acute kidney injury in patients with acute pancreatitis. This model provides a favorable level of performance and can guide the initiation of optimal treatment strategies at an early stage. Both physicians and patients could perform individualized risk management with this easy-to-use scoring system, which is in line with the current trend toward precision medicine.

Abbreviations

HBP=high blood pressure; DM=diabetes mellitus; CHD=coronary heart disease; BISAP=bed-side index for severity of acute pancreatitis; CTSI=computed tomography severity index; SBP=systolic blood pressure; DBP=diastolic blood pressure; BMI=Body Mass Index; APACHEII=acute physiology and chronic health evaluation II; PaO2=arterial partial pressure of oxygen; PH=Pondus Hydrogenii; Na= sodium; K= Potassium; Hct =hematocrit; WBC=white blood cell; BUN=blood urea nitrogen; BG=blood glucose; AST, aspartate amino-transferase; Ca=calcium; Amy=amylase; LPS=lipase; TnI=troponin I; PT=prothrombin time; TT=thrombin time; APTT=activated partial thromboplastin time; ALT=alanine amino-transferase; γ-GGT=γ-glutamyltranspeptadase; ALP=alkaline phosphatase; T-BIL=total bilirubin; D-BIL=direct bilirubin; LDH=lactate dehydrogenase; LDL-C=Low-density lipoprotein cholesterol; HDL-C=High-density lipoprotein cholesterol; UA=Uric Acid; TC=total cholesterol; TG=triglyceride; ALB=albumin; ALB/GLO=albumin/globulin; Lac=lactic acid; RBC=red blood cell; PLT=Platelets; Hb= hemoglobin; PaCO2=partial pressure of carbon dioxide in artery; SaO2=arterial oxygen saturation; SB=standard bicarbonate; BE=bases excess; BNP=B-type natriuretic peptide; PCT=procalcitonin; AaDO2=alveolar-arterial oxygen tension difference; IQR=inter-quartile range

Declarations

Ethics approval and consent to participate

All patients provided full informed consent before clinical procedures to which they were subjected after study inclusion. This study protocol was approved by the Ethic Committee of the First Affiliated Hospital of Dalian Medical University.

Consent for publication

Not applicable.

Availability of data and materials

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

Competing interests

The authors declare that they have no competing interests.

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None.

Authors' contributions

Study concept and design: YLL, JK. acquisition of data, DNG, HHZ, CW, XJZ; Data analysis and interpretation: DLY, LX; drafting of the manuscript, YLL, HW. critical revision, LYL, LJ, JK. All authors read and approved the final manuscript.

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Figures

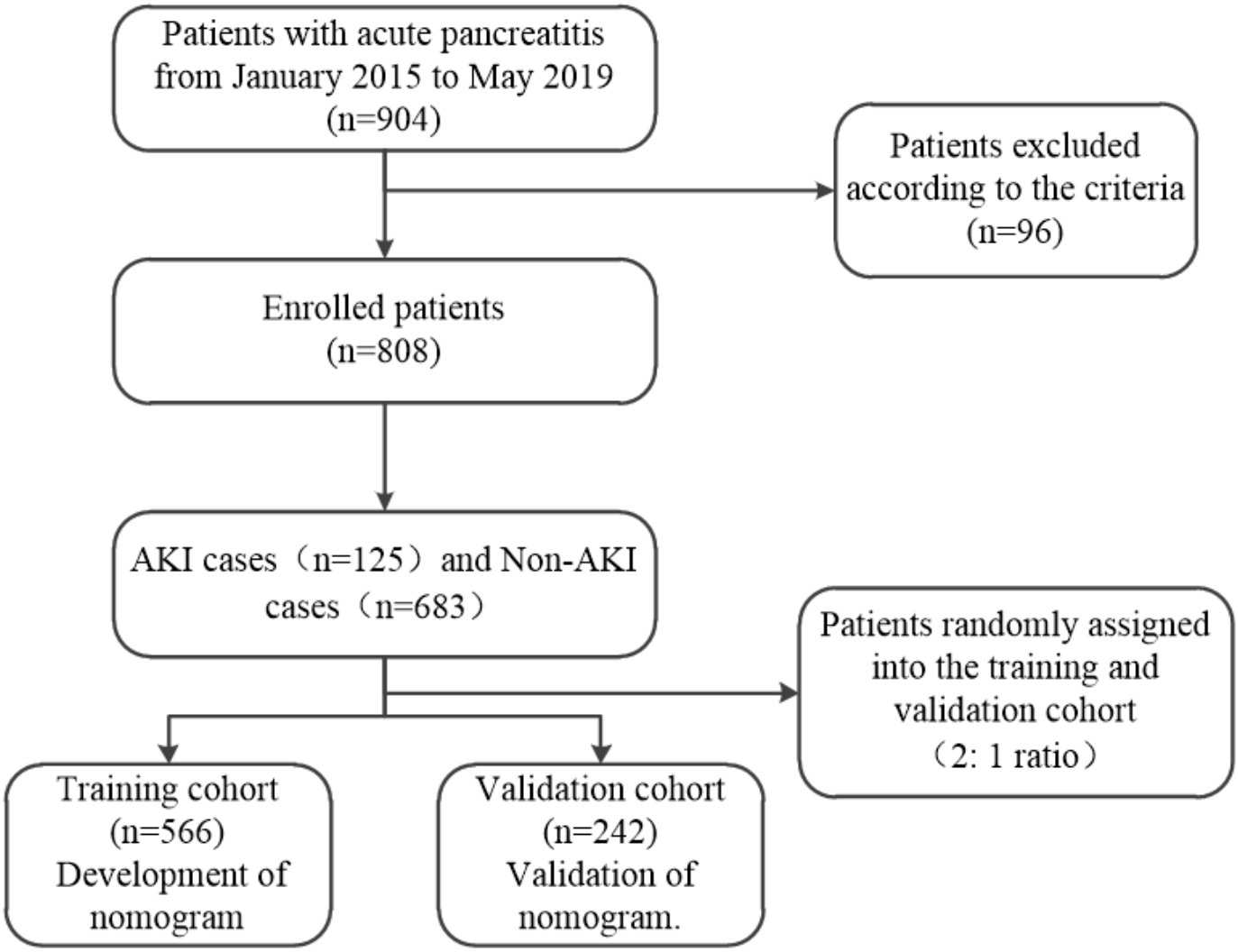


Figure 1

Flowchart of the patients enrollment and study design.

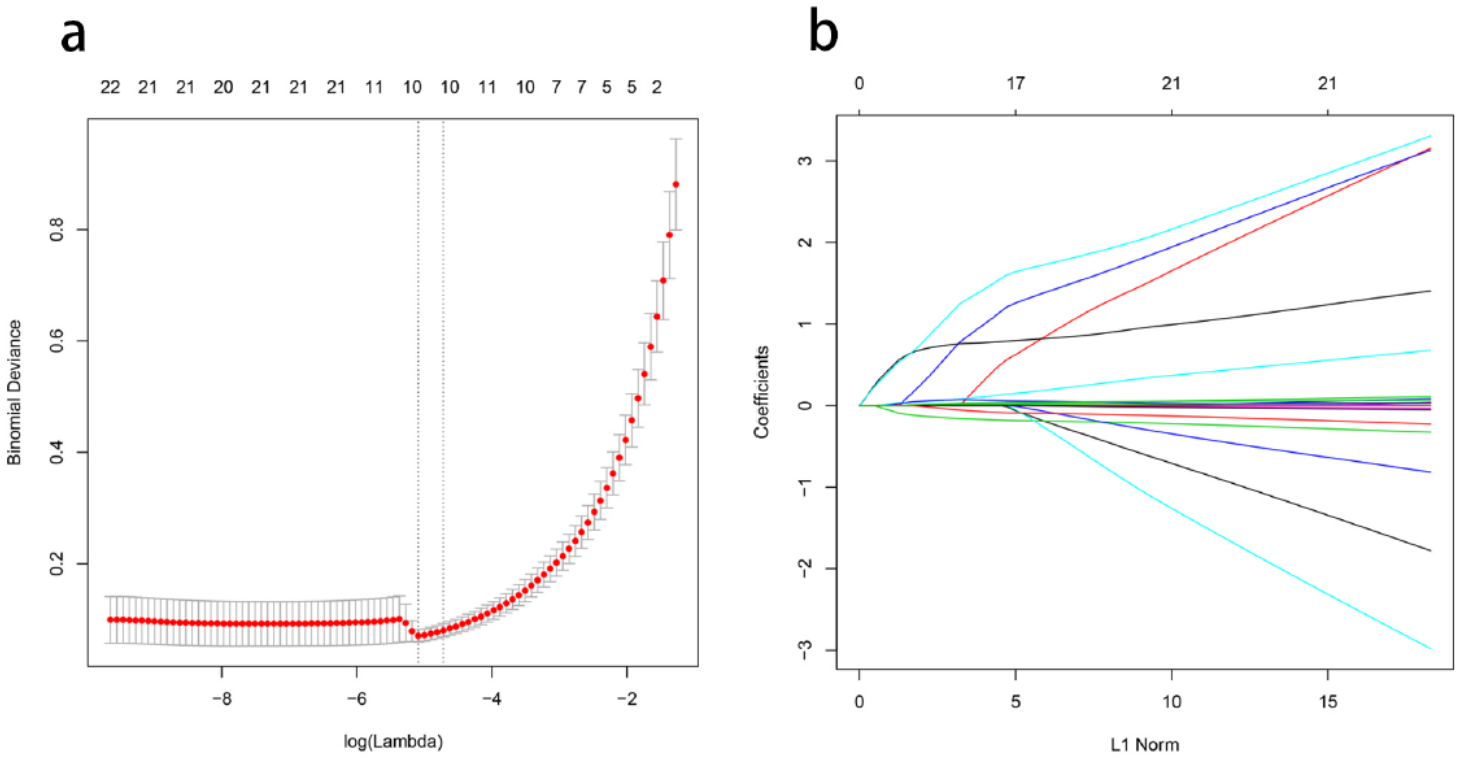


Figure 2

Texture feature selection using the least absolute shrinkage and selection operator (LASSO) logistic regression model. a. Tuning parameter (λ) selection in the LASSO model was conducted by 10-fold cross-validation based on minimum criteria. The binomial deviance was plotted versus log (λ). Dotted vertical lines were drawn at the optimal λ values based on the minimum criteria and the one standard error of the minimum criteria (the 1-SE criteria). b. The coefficients of the selected features are shown by lambda parameter.

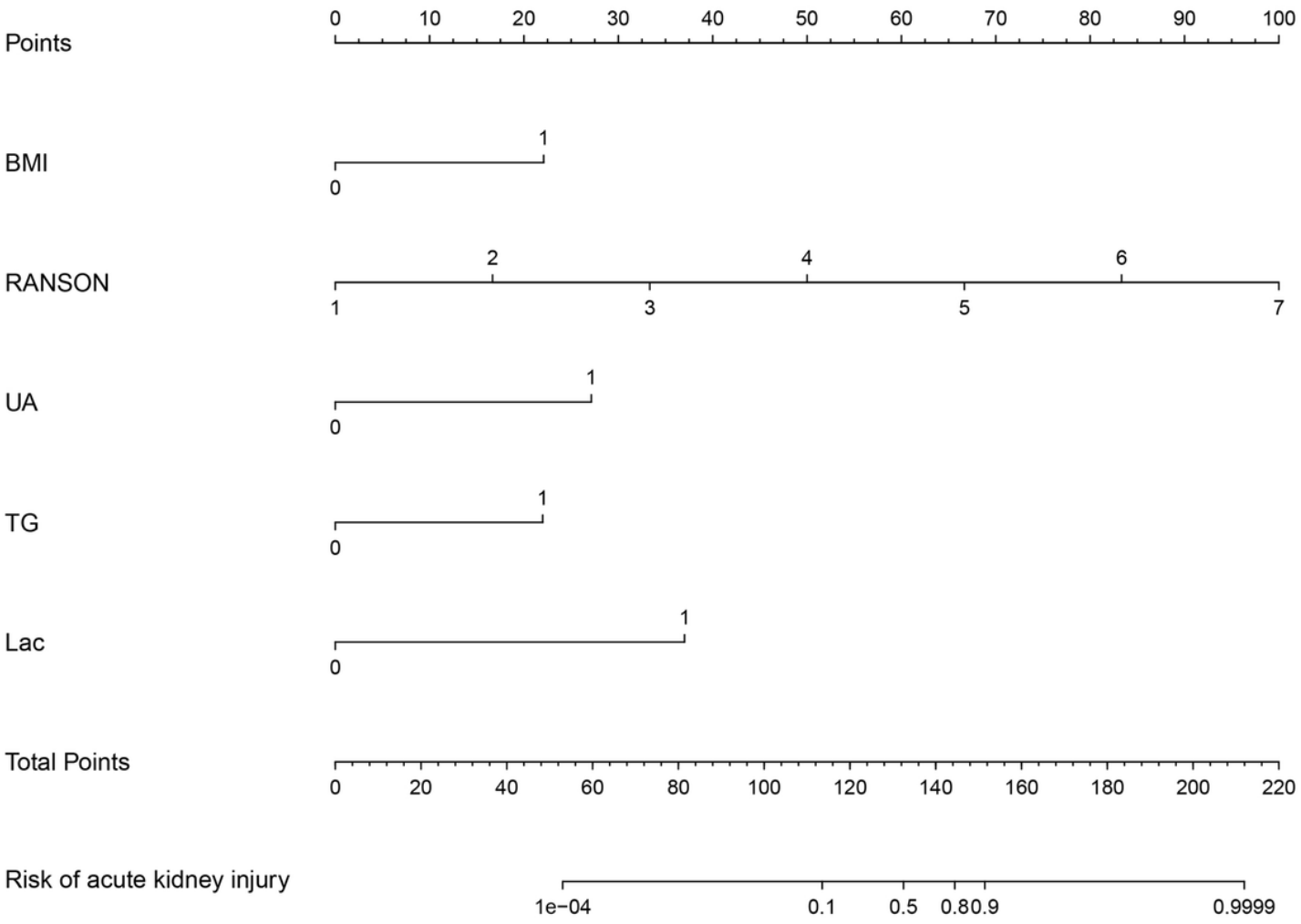


Figure 3

Nomogram to predict acute kidney injury in the patients with acute pancreatitis. The first row: point assignment of the variables; the second to eighth rows: predictors of AP-AKI; the ninth row: total score of seven predictors; the tenth row: prediction of the risk of AP-AKI. Each clinical factor corresponds to a specific point by drawing a line straight upward to the points axis. After summing the points for all variables, locate the sum on the ‘total points’ line to predict the probability of acute kidney injury.

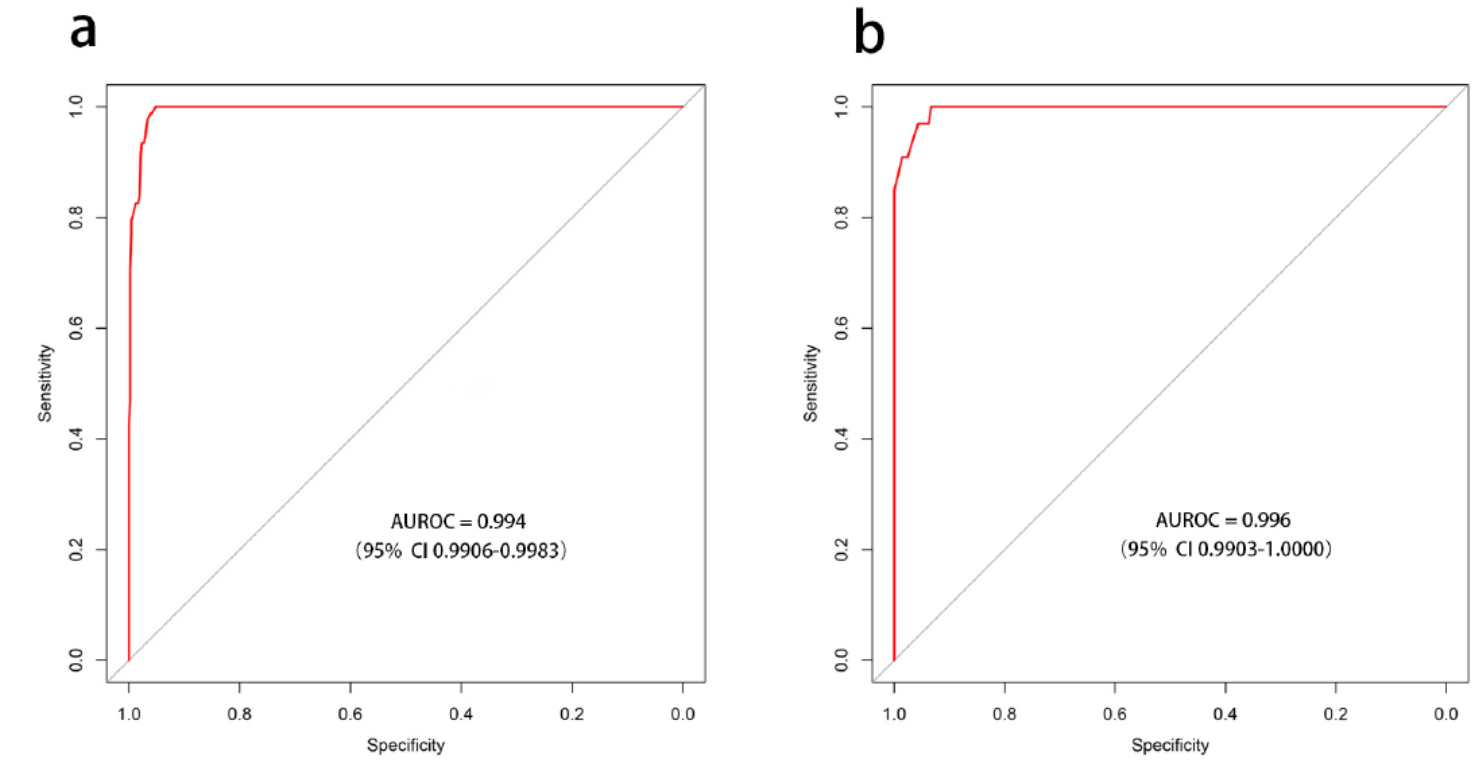


Figure 4

Prediction performance of the model. a, ROC curve in the training set; b, ROC curve in the validation set. AUROC, the area under the receiver operating characteristic, CI, confidence interval

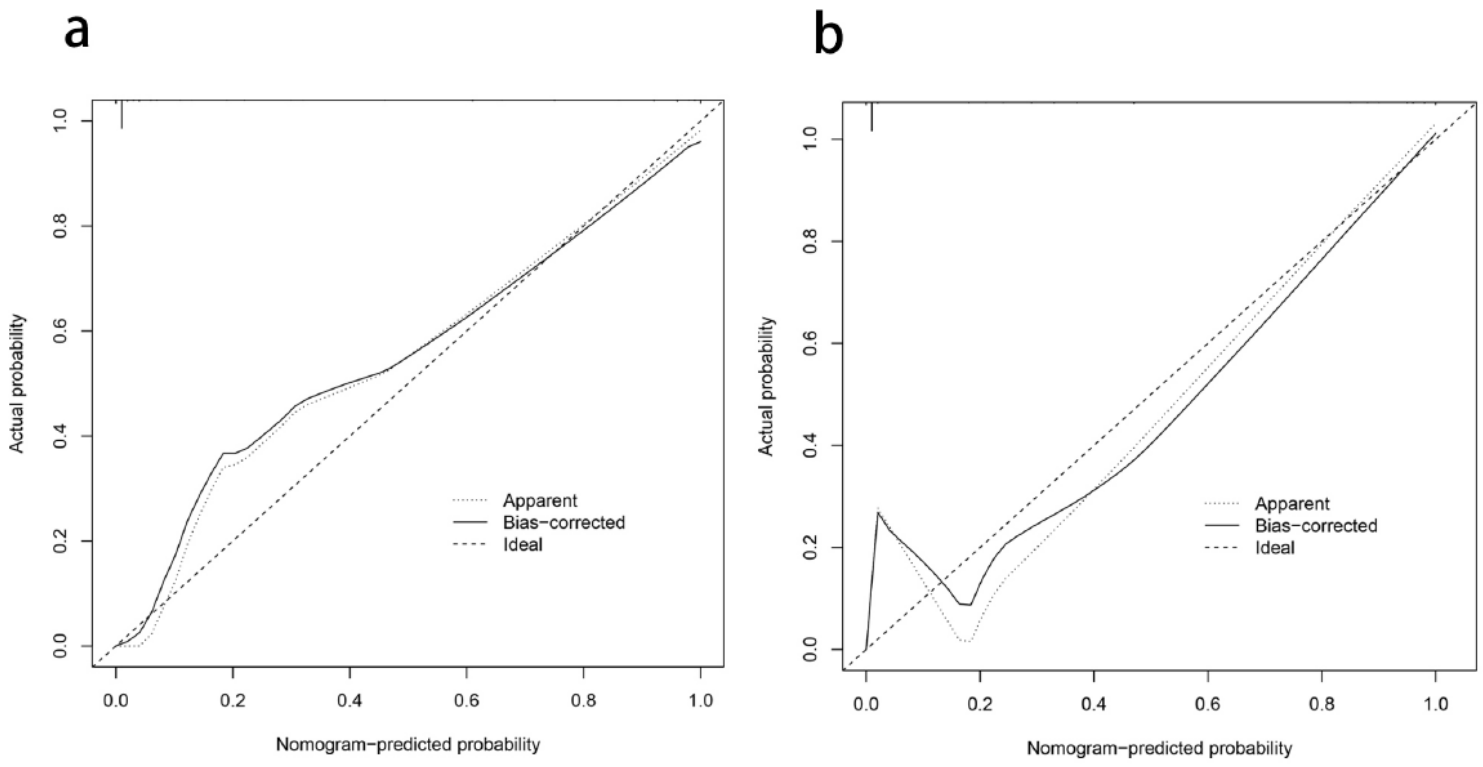


Figure 5

Calibration plots shows the relationship between the predicted probabilities based on the nomogram and the actual values. a, Calibration plot of the training cohort. b, Calibration plot of the validation cohort.

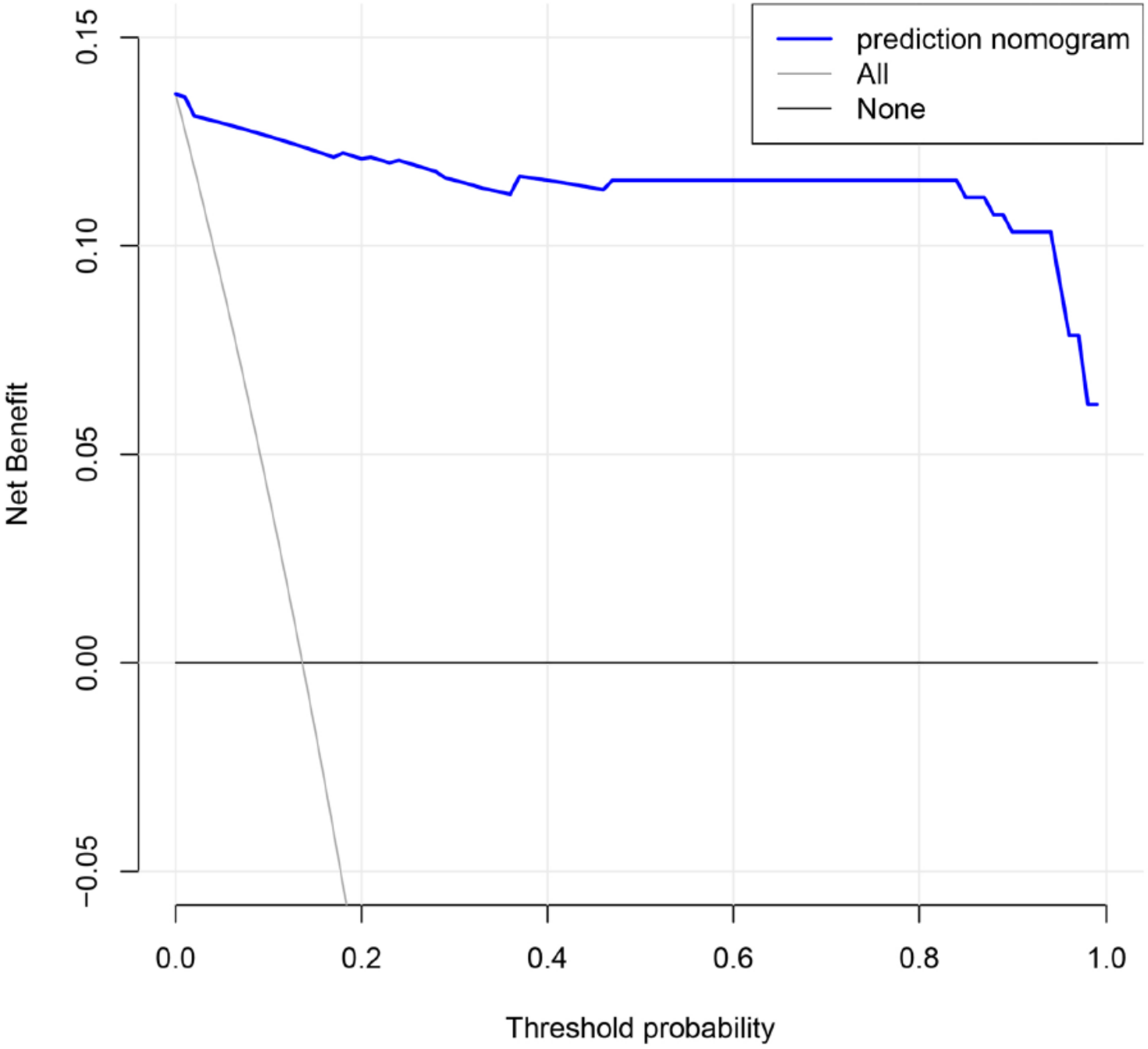


Figure 6

Decision curve analysis for the nomogram. The net benefit was plotted versus the threshold probability. The black and thin grey line represent the assumption that all patients and no patients have acute kidney injury, respectively.