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ORIGINAL RESEARCH

Construction and Validation of a Nomogram Based on Preoperative Inflammatory Indicators for Predicting Acute Kidney Injury After Pancreaticoduodenectomy

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Background: Acute kidney injury (AKI) after pancreaticoduodenectomy is common and early identification of such patients is critical. Inflammation contributes significantly to the onset of postoperative acute kidney injury. We aimed to construct and evaluate a predictive nomogram based on preoperative inflammatory indicators for postoperative AKI in patients undergoing pancreaticoduodenectomy.

Methods: In the current retrospective cohort study, we included 844 adult patients who underwent pancreaticoduodenectomy between December 2016 and June 2020. All enrolled patients were randomly assigned to the training and validation cohorts in a 7:3 ratio. We utilized least absolute shrinkage and selection operator (LASSO) regression for feature selection and multivariable logistic regression analyses to identify key risk factors in the training cohort. These selected factors were subsequently used to construct a nomogram. The nomogram's performance was assessed using various metrics such as the receiver operating characteristic (ROC) curve, calibration curves, Hosmer-Lemeshow goodness of fit, and decision curve analysis (DCA).

Results: In this cohort, AKI was observed in 98 out of 844 patients, representing an incidence rate of 11.6%. LASSO regression and multivariable logistic analysis showed that monocyte-to-lymphocyte ratio (MLR), red blood cell distribution width (RDW), and alkaline phosphatase (ALP) were independent influencing factors of postoperative AKI. The nomogram, which integrated the three identified factors, demonstrated an area under the curve (AUC) of 0.799 in both the training and validation cohorts, indicating moderate discriminative ability. The Hosmer-Lemeshow goodness of fit test and the calibration curve demonstrate good agreement between predicted and observed values. The DCA indicated a positive net clinical benefit.

Conclusion: We developed and validated a nomogram based on preoperative MLR that could help identify individuals at risk of AKI following pancreaticoduodenectomy. This model may help clinicians optimize perioperative management for these patients.

Keywords: acute kidney injury, pancreaticoduodenectomy, inflammatory indicators, nomogram

Introduction

Pancreaticoduodenectomy is currently the primary treatment for pancreatic and ampullary lesions, both benign and malignant. Despite significant improvements in surgical techniques and perioperative care reducing mortality rates for this procedure from over 20% to less than 2%,[1] complication rates remain high, occurring in 29% to 50% of cases.[2–4] Notably, acute kidney injury (AKI) constitutes a significant proportion of these complications, occurring in 5.3–19.7% of cases.[5–10] AKI is closely associated with adverse clinical outcomes. Evidence indicates that AKI following pancreaticoduodenectomy is correlated with higher Clavien-Dindo grades, an elevated risk of intensive care unit admission, and increased 30- and 90-day mortality.[9,10]

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Therefore, early identification and prediction of AKI after pancreaticoduodenectomy represent a critical strategy for optimizing patient prognosis.

Inflammatory reaction mediated by infiltration and activation of lymphocytes and immune cells is an important feature in the occurrence and progress of AKI. Kidney injury triggers resident immune cells to swiftly launch an inflammatory response exert a recruitment effect.[11] This recruitment prompts circulating immune cells to traverse the injured endothelium, infiltrate the renal tissue, and, upon activation, to mediate inflammatory processes that drive tissue destruction and fibrosis.[11] Recent studies indicate that inflammatory markers such as the monocyte-to-lymphocyte ratio (MLR), neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), systemic immune-inflammation index (SII), and systemic inflammation response index (SIRI) are significantly associated with the occurrence of postoperative AKI in patients undergoing various surgical procedures.[12–15] Nevertheless, the value of inflammation-related indicators in AKI subsequent to pancreaticoduodenectomy has not been sufficiently explored. Moreover, clinical prediction models for this surgical complication are still lacking. Only one prior model exists, but it was not specific to pancreaticoduodenectomy.[7]

In summary, there is still a lack of inflammatory predictors or predictive models for the risk of AKI in patients after pancreaticoduodenectomy. Therefore, based on clinical data, the present study aimed to establish a nomogram incorporating inflammatory indicators for predicting the likelihood of secondary AKI in patients undergoing pancreaticoduodenectomy. This tool could help surgeons stratify patients based on the risk of AKI, allowing them to implement appropriate prevention and treatment methods.

Materials and Methods

Ethical Statement

This retrospective single-center study was conducted at Henan Provincial People's Hospital with approval from the ethics committee (Approval No. 2021-Lunshen-77). Due to the study's retrospective design, informed consent was waived by ethics committee.

Study Population and Enrollment Criteria

The study analyzed 844 adult patients who had pancreaticoduodenectomy from December 2016 to June 2020, a population also used in our previously published study.[16] All participants had documented preoperative serum creatinine levels and at least one postoperative measurement within the first 7 days after surgery. Exclusion criteria included: (1) a history of urologic procedures such as nephrectomy, renal transplantation, or urinary obstruction relief, due to potential confounding effects on postoperative creatinine levels; (2) preoperative AKI; (3) chronic kidney disease, indicated by an eGFR below 60 mL/min/1.73 m^2; and (4) a need for dialysis.

Data Collection

Data extracted from the electronic medical record database encompassed demographic details, comorbidities, medication history prior to surgery, preoperative lab results, intraoperative information, and postoperative factors. Demographic characteristics encompassed both age and sex. Comorbidities included hypertension, coronary heart disease, diabetes mellitus and biliary obstruction. The preoperative medication history comprised contrast agents, diuretics, non-steroidal anti-inflammatory drugs (NSAIDs), angiotensin-converting enzyme inhibitors (ACEI), and angiotensin receptor blockers (ARB). Preoperative laboratory data encompassed measurements of white blood cells (WBC), red blood cells (RBC), hemoglobin (HGB), platelets (PLT), red blood cell distribution width (RDW), neutrophil-to-lymphocyte ratio (NLR= neutrophil/lymphocyte), monocyte-to-lymphocyte ratio (MLR= monocyte/lymphocyte), platelet-to-lymphocyte ratio (PLR= platelet/lymphocyte), systemic immune inflammation index [SII= (neutrophil*platelet)/lymphocyte], systemic inflammation response index [SIRI= (neutrophil*monocyte)/lymphocyte], aspartate aminotransferase (AST), alanine aminotransferase (ALT), albumin (ALB), total bilirubin (TBIL), alkaline phosphatase (ALP), total bile acids (TBA), blood urea nitrogen (BUN), serum creatinine (SCr), eGFR, uric acid (UA), retinol-binding proteins (RBP), cystatin C (CysC), prothrombin time (PT), prothrombin time activity (PTA), international normalized ratio (INR), activated partial thromboplastin time (APTT), fibrinogen (FBG), and thrombin time (TT). Preoperative interventions refer to percutaneous

transhepatic cholangial drainage (PTCD). Intraoperative variables encompassed vasopressor use, blood transfusion, blood loss, urine output, operative duration, and hypotension. Intraoperative hypotension was characterized by a mean arterial pressure below 65 mmHg for over 10 cumulative minutes during surgery.[17] Finally, we also included pathological results. The eGFR was determined using the Chronic Kidney Disease Epidemiology Collaboration Group formula.[18]

Dealing with Missing Data

Visualize missing data through the plot_missing function in the "DataExplorer" package. Missing data were handled using multiple imputation with the predictive mean matching method via the "mice" package in R. A total of 5 imputed datasets were generated, and the 3rd dataset was used for subsequent analysis. All candidate variables were included in the imputation model to maintain the natural correlations among variables and minimize bias in the following LASSO regression. All patients' clinical data were collected, and it was found that the missing ratio of CysC and RBP was 15.28% and 15.05%, respectively, and the missing ratio of other clinical data was less than 2.61%, or even not (Figure S1).

Outcome

The main outcome was postoperative AKI, defined by KDIGO criteria as either a rise in SCr of ≥0.3 mg/dL (≥26.5 μmol/L) within 48 hours post-surgery or an increase in SCr to ≥1.5 times the baseline within 7 days post-surgery.[19] Since the urine volume of most postoperative patients was not available, the KDIGO urine output standard was not used in this study.

Sample Size

Following the approach by Riley et al,[20] the minimum sample size for developing a nomogram prediction model was estimated using the "pmsampsize" package in R. The calculation assumed an incidence of postoperative AKI of 5.3% and an expected C-statistic of 0.79, as reported in previous studies of pancreatic surgery.[7] The model was planned to include 4 variables because previous studies reported 3–4 risk factors for AKI following pancreaticoduodenectomy.[9,10] We hypothesized that these established risk factors would be highly likely to be retained in our final prediction model. Using these parameters, the minimum required sample size was estimated to be 576 patients. The training set in this study included 590 patients, meeting this requirement.

Statistical Analysis

Grouping of Study Population

Patients were randomly assigned to two groups using simple randomization: 70% for the training cohort and 30% for the validation cohort. Create the nomogram using the training cohort, then utilize the validation cohort for internal validation.

Baseline Data Analysis

Continuous variables with a normal distribution are presented as mean ± standard deviation, non-normally distributed variables as median (interquartile range), and categorical variables as percentages of the total. An independent sample *t*-test was used to compare the two groups for measurements with a normal distribution and equal variance. Data with non-normal distribution or non-equal variance will be compared between groups using the Mann–Whitney *U*-test. The counting data for the two groups were analyzed using Pearson's chi-square test or Fisher's exact test. Comparison between two groups using "CBCgrps" package.[21] Multicollinearity was evaluated using the variance inflation factor (VIF) calculated with the vif() function in the "car" package in R. A VIF value ≥ 10 was considered indicative of severe multicollinearity.

Screening Variables

We employed least absolute shrinkage and selection operator (LASSO) regression analysis using the "glmnet" package to filter the variables, and the optimal penalty parameter λ was determined through 10-fold cross-validation combined with the 1-SE criterion. Then, multivariable logistic regression analysis was conducted to identify independent influencing factors.

Development, Validation and Evaluation of Nomograms

The "rms" package's nomogram function is used to built a nomogram model from the selected independent influencing factors. The nomogram's discrimination ability was evaluated by calculating the area under the curve (AUC). The pROC function from the "rms" package was utilized to create a receiver operating characteristic (ROC) curve. The Hosmer-Lemeshow goodness of fit test was conducted using the "ResourceSelection" package, while the "rms" package facilitated the calibration curve to assess the nomogram model's fit. To assess clinical practicability, the "rmda" package was used to generate a decision curve analysis (DCA) curve.

Furthermore, the model underwent internal validation using 1000 bootstrap resamples. Statistical analyses were performed using R software (version 4.2.1), with significance defined as $p<0.05$, except for the Hosmer-Lemeshow goodness of fit test, where $p>0.05$ was considered significant.

Results

Baseline Characteristics of the Study Participants

The study included 844 patients (Figure 1), and 11.6% of them experienced postoperative AKI. In a 7:3 ratio, we assigned 590 patients to the training cohort and 254 patients to the validation cohort. Table 1 presents the baseline data for both the training and validation cohorts. The two groups showed no significant differences in demographic characteristics, laboratory data, or surgery-related factors. In the training cohort, 11.5% (68/590) of patients developed

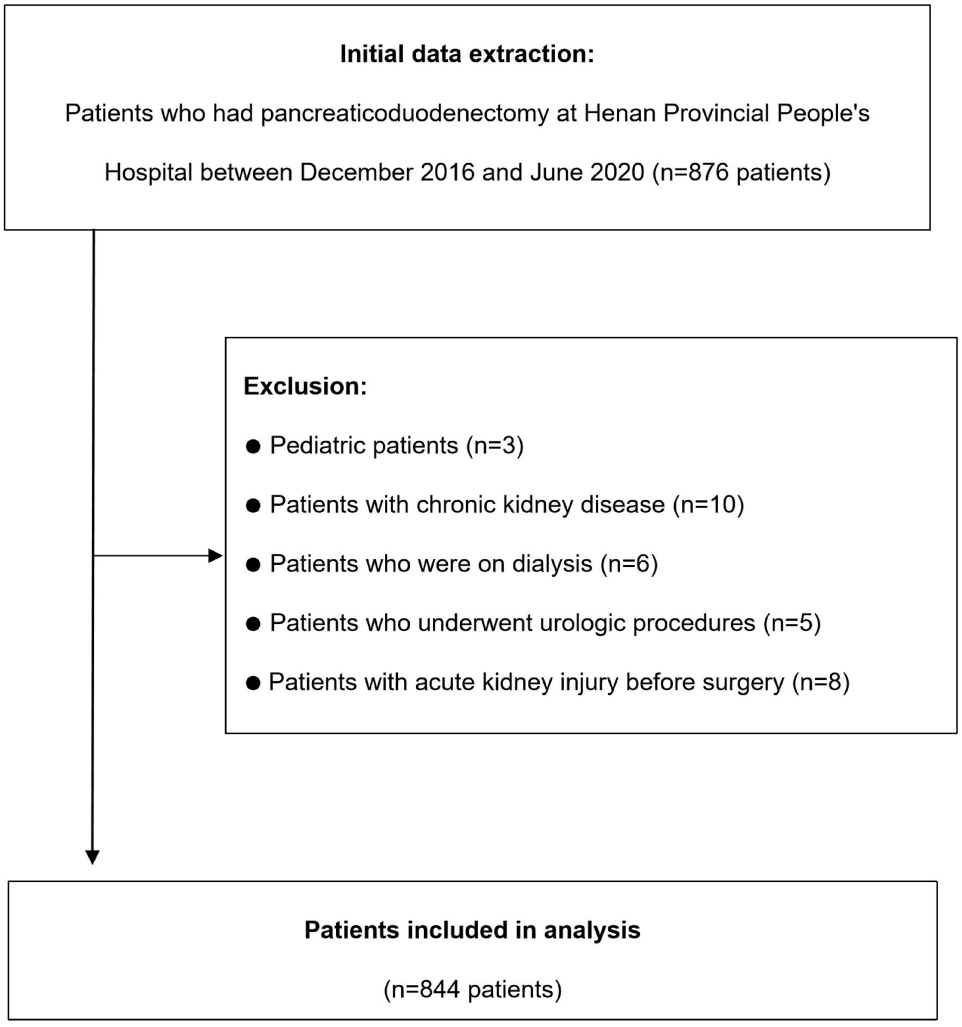

Figure 1 Flow chart of patients' exclusion process.

Table 1 Demographic, Clinical Characteristics and Surgery-Related Factors of the Training and Validation Cohorts

Variables	All (n = 844)	Training Cohorts (n=590)	Validation Cohorts (n = 254)	*P* Value
Baseline variables				
Age (years)	60.00 (51.00, 67.00)	61.00 (51.00, 67.00)	61.00 (53.00, 66.00)	0.325
Sex, n (%)				0.601
Man	492 (58.29)	340 (57.63)	152 (59.84)	
Women	352 (41.71)	250 (42.37)	102 (40.16)	
Coexisting conditions				
Hypertension, n (%)	223 (26.42)	159 (26.95)	64 (25.20)	0.657
Coronary heart disease, n (%)	58 (6.87)	38 (6.44)	20 (7.87)	0.544
Diabetes mellitus, n (%)	141 (16.71)	97 (16.44)	44 (17.32)	0.830
Biliary obstruction, n (%)	304 (36.02)	207 (35.08)	97 (38.19)	0.433
Medication history				
Contrast, n (%)	690 (81.75)	480 (81.36)	210 (82.68)	0.720
Diuretics, n (%)	77 (9.12)	53 (8.98)	24 (9.45)	0.932
NSAIDs, n (%)	144 (17.06)	99 (16.78)	45 (17.72)	0.816
ACEI/ARB, n (%)	206 (24.41)	147 (24.92)	59 (23.23)	0.663
Laboratory parameters				
WBC (10^9/L)	6.14 (4.94, 7.40)	6.20 (5.00, 7.40)	6.03 (4.84, 7.40)	0.702
RBC ($10^{12/}$L)	4.03 ± 0.59	4.02 ± 0.59	4.03 ± 0.60	0.857
HGB (g/L)	124.00 (111.00, 135.00)	123.50 (111.00, 135.00)	126.00 (112.25, 135.00)	0.664
RDW (%)	13.30 (12.30, 14.60)	13.30 (12.30, 14.57)	13.20 (12.30, 14.60)	0.559
PLT (10^9/L)	234.00 (187.00, 293.00)	233.50 (187.00, 292.75)	237.50 (185.25, 292.75)	0.937
NLR	2.64 (1.88, 4.08)	2.58 (1.90, 4.11)	2.75 (1.86, 4.06)	0.918
MLR	0.26 (0.18, 0.36)	0.26 (0.18, 0.36)	0.26 (0.18, 0.35)	0.604
PLR	165.07 (120.32, 227.89)	164.53 (120.68, 227.59)	166.21 (119.85, 228.19)	0.929
SII	628.70 (393.83, 1047.06)	627.20 (400.87, 1049.91)	631.32 (390.48, 1024.08)	0.899
SIRI	0.98 (0.58, 1.72)	1.00 (0.57, 1.73)	0.97 (0.58, 1.67)	0.776
ALT (U/L)	110.60 (32.15, 226.57)	108.25 (32.92, 227.50)	111.75 (29.70, 225.75)	0.853
AST (U/L)	76.35 (29.00, 157.00)	79.50 (29.20, 154.98)	71.55 (28.02, 160.45)	0.985
ALB (g/L)	37.60 (34.10, 40.90)	37.90 (34.30, 41.00)	37.2 (33.8, 40.45)	0.105
TBIL (μmol/L)	60.60 (12.45, 172.12)	57.85 (12.22, 164.30)	69.30 (13.12, 186.15)	0.469
ALP (U/L)	191.50 (88.00, 296.00)	193.50 (89.00, 294.00)	186.50 (86.00, 298.75)	0.713
TBA (μmol/L)	54.90 (4.70, 160.07)	52.05 (4.82, 161.82)	64.55 (3.78, 152.50)	0.504
BUN (mmol/L)	4.60 (3.67, 5.58)	4.60 (3.69, 5.58)	4.60 (3.60, 5.57)	0.998
SCr (μmol/L)	54.00 (45.00, 62.00)	53.00 (45.00, 62.00)	55.00 (46.00, 62.00)	0.336
eGFR [$mL \bullet min^{-1} \bullet (1.73m^2)^{-1}$]	104.90 (96.82, 114.02)	105.55 (96.94, 114.59)	104.45 (95.99, 113.02)	0.225
UA (μmol/L)	215.50 (164.00, 271.25)	214.00 (163.00, 270.75)	216.50 (167.00, 276.00)	0.885
RBP (mg/L)	29.00 (23.00, 35.70)	29.00 (23.00, 36.00)	28.10 (23.00, 33.90)	0.316
CysC (mg/L)	0.88 (0.79, 1.00)	0.88 (0.79, 0.99)	0.88 (0.78, 1.00)	0.889
PT (s)	11.90 (11.20, 12.70)	11.90 (11.10, 12.70)	11.90 (11.30, 12.80)	0.164
PTA (%)	117.00 (99.00, 140.00)	117.00 (100.00, 141.00)	115.00 (99.00, 138.75)	0.215
INR (INR)	0.92 (0.84, 1.00)	0.92 (0.84, 1.00)	0.93 (0.85, 1.01)	0.209
APTT (s)	33.10 (30.20, 36.30)	33.00 (30.20, 36.20)	33.38 (30.10, 36.48)	0.418
FBG (g/L)	3.95 (3.18, 4.80)	3.96 (3.21, 4.77)	3.90 (3.14, 4.87)	0.774
TT (s)	17.10 (16.00, 18.20)	17.10 (16.10, 18.20)	16.95 (15.80, 18.00)	0.238
Preoperative interventions				
PTCD, n (%)	46 (5.45)	31 (5.25)	15 (5.91)	0.828
Intraoperative variables				
Vasopressor use, n (%)	67 (7.94)	43 (7.29)	24 (9.45)	0.354
Blood transfusion, n (%)	393 (46.56)	276 (46.78)	117 (46.06)	0.907
Blood loss (mL)	300 (200, 500)	300 (200, 500)	300 (200, 500)	0.117

(*Continued*)

Table 1 (Continued).

Variables	All (n = 844)	Training Cohorts (n=590)	Validation Cohorts (n = 254)	*P* Value
Urine output (mL)	775 (500, 1000)	700 (500, 1000)	800 (500, 1000)	0.133
Operative duration (min)	370.00 (315.00, 455.00)	370.00 (320.00, 450.00)	375.00 (300.50, 465.00)	0.888
Intraoperative hypotension, n (%)	102 (12.09)	71 (12.03)	31 (12.20)	1.000
Pathology, n (%)				0.151
Malignant	800 (94.79)	564 (95.59)	236 (92.91)	
Benign	44 (5.21)	26 (4.41)	18 (7.09)	

Abbreviations: NSAIDs, non-steroidal anti-inflammatorydrugs; ACEI/ARB, angiotensin-converting enzyme inhibitors and angiotensin receptor blockers; WBC, white blood cells; RBC, red blood cells; HGB, hemoglobin; RDW, red blood cell distribution width; PLT, platelets; NLR, neutrophil-to-lymphocyte ratio; MLR, monocyte-to-lymphocyte ratio; PLR, platelet-to-lymphocyte ratio; SII, systemic immune inflammation index; SIRI, systemic inflammation response index; ALT, alanine aminotransferase; AST, aspartate aminotransferase; ALB, albumin; TBIL, total bilirubin; ALP, alkaline phosphatase; TBA, total bile acids; BUN, blood urea nitrogen; SCr, serum creatinine; eGFR, estimated glomerular filtration rate; UA, uric acid; RBP, retinol-binding proteins; CysC, cystatin C; PT, prothrombin time; PTA, prothrombin time activity; INR, international normalized ratio; APTT, partial thromboplastin time; FBG, fibrinogen; TT, thrombin time; PTCD, percutaneous transhepatic cholangial drainage.

AKI, and AKI group exhibited elevated RDW, MLR, SIRI, TBIL, ALP and CysC levels (all $p < 0.05$) (Table 2). In the validation group, 11.8% (30 out of 254) of patients experienced AKI, and those in the AKI group exhibited higher rates of diuretics use, elevated RDW, MLR, SIRI, and ALP levels, shorter TT, and longer operative duration (all $p< 0.05$) (Table 2).

Table 2 Demographic, Clinical Characteristics and Surgery-Related Factors of the Training Cohort and Validation Cohort Stratified by AKI Category

Variables	Training Cohort			Validation Cohorts		
	Non-AKI Group (n = 522)	AKI Group (n = 68)	*P* value	Non-AKI Group (n = 224)	AKI Group (n = 30)	*P* value
Baseline variables						
Age (years)	59.50 (51.00, 67.00)	61.00 (52.00, 69.25)	0.091	61.00 (53.00, 66.00)	60.50 (53.00,67.00)	0.837
Sex, n (%)			0.387			0.539
Man	297 (56.90)	43 (63.24)		132 (58.93)	20 (66.67)	
Women	225 (43.10)	25 (36.76)		92 (41.07)	10 (33.33)	
Coexisting conditions						
Hypertension, n (%)	139 (26.63)	20 (29.41)	0.733	54 (24.11)	10 (33.33)	0.385
Coronary heart disease, n (%)	33 (6.32)	5 (7.35)	0.791	17 (7.59)	3 (10.00)	0.715
Diabetes mellitus, n (%)	82 (15.71)	15 (22.06)	0.248	39 (17.41)	5 (16.67)	1.000
Biliary obstruction, n (%)	183 (35.06)	24 (35.29)	1.000	84 (37.5)	13 (43.33)	0.676
Medication history						
Contrast, n (%)	423 (81.03)	57 (83.82)	0.697	184 (82.14)	26 (86.67)	0.720
Diuretics, n (%)	45 (8.62)	8 (11.76)	0.530	17 (7.59)	7 (23.33)	0.013
NSAIDs,n (%)	87 (16.67)	12 (17.65)	0.975	42 (18.75)	3 (10.00)	0.355
ACEI/ARB,n (%)	129 (24.71)	18 (26.47)	0.868	50 (22.32)	9 (30.00)	0.481
Laboratory parameters						
WBC (10^9/L)	6.18 (5.00, 7.32)	6.67 (4.88, 8.38)	0.214	6.00 (4.87, 7.42)	6.30 (4.80, 7.29)	0.796
RBC (10^{12}/L)	4.03 ± 0.58	3.96 ± 0.63	0.408	4.10 (3.67, 4.40)	4.22 (3.42, 4.57)	0.904
HGB (g/L)	123.00 (111.25, 135.00)	126.00 (110.75,136.00)	0.806	126.00 (113.00, 135.00)	124.50 (107.25, 134.00)	0.554
RDW (%)	13.20 (12.20, 14.40)	14.60 (13.28, 16.50)	< 0.001	12.95 (12.20, 14.20)	15.00 (14.22, 15.60)	< 0.001
PLT (10^9/L)	234.00 (189.00, 290.50)	220.00 (180.00, 294.5)	0.498	238.00 (183.00, 293.25)	229.50 (200.75, 290.75)	0.533
NLR	2.57 (1.84, 4.08)	2.76 (2.16, 4.54)	0.121	2.72 (1.84, 4.02)	2.76 (2.26, 4.12)	0.460
MLR	0.24 (0.17, 0.35)	0.33 (0.25, 0.44)	< 0.001	0.24 (0.17, 0.35)	0.31 (0.26, 0.41)	0.007
PLR	165.76 (120.72, 227.59)	158.61 (120.82, 221.83)	0.871	162.53 (118.46, 223.61)	197.71 (134.92, 249.39)	0.144
SII	617.05 (392.53,1051.48)	718.30 (447.49, 950.78)	0.319	631.32 (387.42,1009.54)	645.02 (433.04,1170.00)	0.456
SIRI	0.96 (0.54, 1.65)	1.41 (0.79, 2.24)	< 0.001	0.93 (0.56, 1.56)	1.23 (0.84, 1.80)	0.042

(*Continued*)

Table 2 (Continued).

Variables	Training Cohort			Validation Cohorts		
	Non-AKI Group (n = 522)	AKI Group (n = 68)	*P* value	Non-AKI Group (n = 224)	AKI Group (n = 30)	*P* value
ALT (U/L)	106.10 (32.75, 224.75)	120.85 (40.22, 234.52)	0.732	112.45 (29.90, 232.82)	93.25 (28.50, 172.12)	0.460
AST (U/L)	80.15 (29.20, 153.30)	76.80 (31.47, 167.25)	0.589	76.30 (28.77, 160.70)	64.30 (26.50, 152.52)	0.502
ALB (g/L)	37.95 (34.40, 41.00)	37.35 (33.88, 41.42)	0.613	37.40 (33.80, 40.50)	36.55 (33.98, 40.08)	0.973
TBIL (μmol/L)	48.30 (12.12, 158.55)	121.80 (21.10, 192.75)	0.010	69.30 (13.00, 182.55)	85.90 (17.38, 216.10)	0.294
ALP (U/L)	173.00 (86.25, 279.75)	298.50 (199.38, 529.75)	< 0.001	171.50 (84.75, 292.75)	225.00 (152.75, 407.75)	0.010
TBA (μmol/L)	48.05 (4.43, 160.6)	102.95 (10.23, 170.28)	0.173	64.15 (3.65, 151.90)	74.45 (5.10, 148.75)	0.763
BUN (mmol/L)	4.60 (3.69, 5.50)	4.59 (3.68, 5.92)	0.374	4.58 (3.59, 5.54)	4.83 (4.04, 5.70)	0.405
SCr (μmol/L)	53.00 (45.00, 62.00)	53.00 (44.00, 67.50)	0.647	55.00 (46.00, 62.00)	57.00 (45.00, 64.00)	0.362
eGFR [$mL \bullet min^{-1} \bullet (1.73m^2)^{-1}$]	105.92 (97.31, 114.80)	103.24 (95.37, 110.77)	0.066	104.61 (96.25, 112.66)	102.83 (95.04, 114.22)	0.629
UA (μmol/L)	214.00 (163.00, 271.00)	215.50 (165.75, 262)	0.982	217.00 (166.50, 278.25)	208.00 (167.50, 269.75)	0.688
RBP (mg/L)	29.00 (23.00, 36.00)	30.45 (20.67, 37.00)	0.774	28.62 ± 9.35	29.38 ± 7.44	0.615
CysC (mg/L)	0.87 (0.79, 0.98)	0.93 (0.84, 1.07)	0.006	0.87 (0.78, 1.00)	0.94 (0.82, 1.05)	0.132
PT (s)	11.90 (11.10, 12.70)	11.70 (11.10, 12.70)	0.579	12.00 (11.30, 12.80)	11.85 (11.35, 13.05)	0.980
PTA (%)	117.00 (100.00, 141.00)	122.00 (101.50, 141.50)	0.386	116.00 (99.00, 138.25)	108.50 (97.25, 138.00)	0.810
INR (INR)	0.92 (0.84, 1.00)	0.90 (0.84, 0.99)	0.327	0.93 (0.86, 1.01)	0.96 (0.85, 1.02)	0.851
APTT (s)	33.10 (30.13, 36.20)	32.75 (31.08, 35.83)	0.660	33.38 (30.17, 36.52)	32.70 (29.05, 36.10)	0.482
FBG (g/L)	3.96 (3.21, 4.76)	4.11 (3.24, 4.98)	0.672	3.92 (3.14, 4.87)	3.82 (3.36, 4.54)	0.737
TT (s)	17.00 (16.10, 18.20)	17.40 (16.55, 18.35)	0.167	17.11 ± 1.66	16.45 ± 1.37	0.019
Preoperative interventions						
PTCD, n (%)	26 (4.98)	5 (7.35)	0.387	13 (5.8)	2 (6.67)	0.693
Intraoperative variables						
Vasopressor use, n (%)	36 (6.90)	7 (10.29)	0.319	21 (9.38)	3 (10.00)	1.000
Blood transfusion, n (%)	247 (47.32)	29 (42.65)	0.551	102 (45.54)	15 (50.00)	0.791
Blood loss (mL)	300 (200, 500)	400 (200, 600)	0.254	300 (200, 500)	300 (200, 600)	0.697
Urine output (mL)	700 (500, 1000)	700 (500, 1000)	0.641	800 (500, 1000)	750 (400, 1000)	0.154
Operative duration (min)	370.00 (320.00, 450.00)	360.00 (318.00, 485.00)	0.895	367.50 (300.00, 450.00)	467.50 (309.75, 538.75)	0.038
Intraoperative hypotension, n (%)	62 (11.88)	9 (13.24)	0.900	25 (11.16)	6 (20.00)	0.228
Pathology, n (%)			0.526			1.000
Malignant	500 (95.79)	64 (94.12)		208 (92.86)	28 (93.33)	
Benign	22 (4.21)	4 (5.88)		16 (7.14)	2 (6.67)	

Abbreviations: NSAIDs non-steroidal anti-inflammatory drugs; ACEI/ARB, angiotensin-converting enzyme inhibitors and angiotensin receptor blockers; WBC, white blood cells; RBC, red blood cells; HGB, hemoglobin; RDW, red blood cell distribution width; PLT, platelets; NLR, neutrophil-to-lymphocyte ratio; MLR, monocyte-to-lymphocyte ratio; PLR, platelet-to-lymphocyte ratio; SII, systemic immune inflammation index; SIRI, systemic inflammation response index; ALT, alanine aminotransferase; AST, aspartate aminotransferase; ALB, albumin; TBIL, total bilirubin; ALP, alkaline phosphatase; TBA, total bile acids; BUN, blood urea nitrogen; SCr, serum creatinine; eGFR, estimated glomerular filtration rate; UA, uric acid; RBP, retinol-binding proteins; CysC, cystatin C; PT, prothrombin time; PTA, prothrombin time activity; INR, international normalized ratio; APTT, partial thromboplastin time; FBG, fibrinogen; TT, thrombin time; PTCD, percutaneous transhepatic cholangial drainage.

Multicollinearity Assessment

The results showed that the VIF values of eGFR, SCr, Age, SIRI, SII, PLR, Sex, Hypertension, ACEI/ARB, and NLR exceeded 10, indicating severe multicollinearity among these variables (Figure S2). Because these variables were clinically important for AKI prediction, LASSO regression was used to handle multicollinearity and perform variable selection simultaneously.

Variable Screening and Construction of Nomogram

The LASSO regression was first applied to the training set for preliminary predictor selection, which helps prevent model overfitting (Figure 2A and B). Consequently, MLR, RDW, and ALP were identified as the three candidate predictors. These three variables were further rescreened using multivariable logistic regression. Finally, MLR (OR = 3.813, 95% CI: 1.066–12.782, $p = 0.033$), RDW (OR = 1.463, 95% CI: 1.261–1.711, $p < 0.001$), and ALP (OR= 1.005, 95% CI: 1.003–1.006, $p < 0.001$) were identified as independent influencing factors (Table 3). A nomogram was developed using

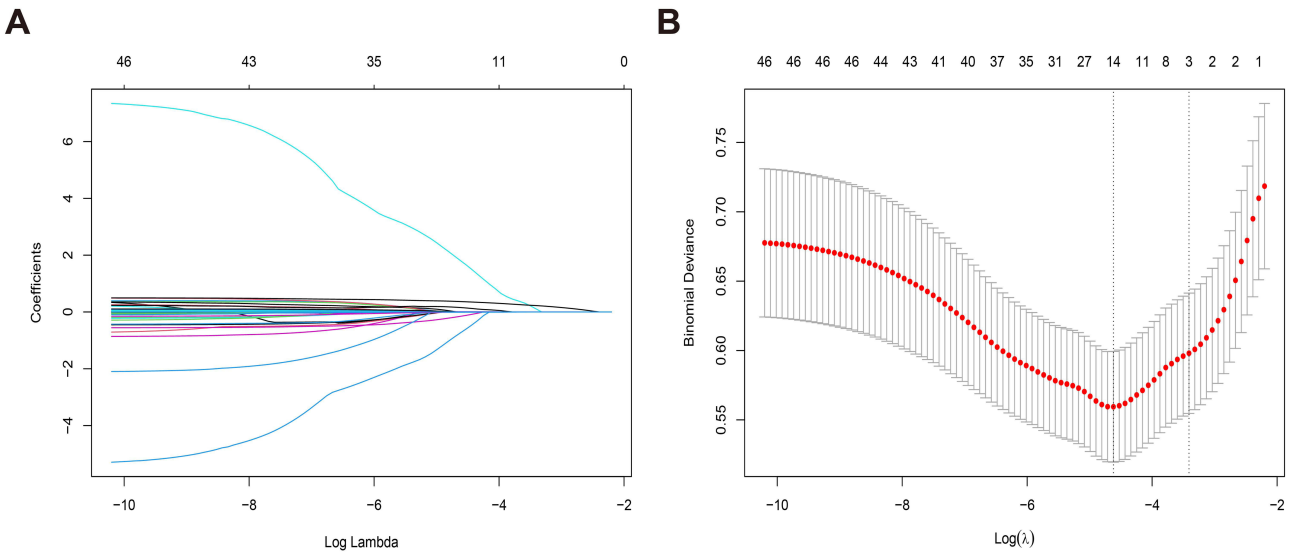

Figure 2 Predictors were chosen using a LASSO regression. (**A**) Distribution of LASSO coefficients for each clinical variable. (**B**) Optimal penalty parameter λ of the LASSO model selected using 10-fold cross-validation with the 1-SE criterion.

these three characteristics to evaluate the risk of postoperative AKI in patients undergoing pancreaticoduodenectomy (Figure 3).

Evaluation of the Nomogram Model

The discrimination of the nomogram was assessed by plotting the ROC curve in the training cohort, which demonstrated an AUC of 0.799 (95% CI: 0.742–0.856) (Figure 4A). Calibration plots revealed excellent agreement between the nomogram's predictions and actual observations (Figure 5A), with the Hosmer-Lemeshow test confirming good fit (p = 0.402). Clinical usefulness, evaluated via decision curve analysis (Figure 6A), was established by a net benefit greater than zero for threshold probabilities between 0.03 and 1.

Nomogram Model Validation

Analysis of the validation cohort yielded an AUC of 0.799 (95% CI: 0.719–0.878) for the nomogram, indicating moderate discriminative ability (Figure 4B). This finding was further supported by internal validation via bootstrap resampling, which produced a mean AUC of 0.789 (95% CI: 0.777–0.795) (Figure S3). The calibration curve demonstrates consistency between actual and predicted AKI probabilities in the validation group (Figure 5B), with a Hosmer-Lemeshow test p-value of 0.704. Furthermore, decision curve analysis demonstrated the model's clinical utility, with a positive net benefit observed across threshold probabilities ranging from 0.03 to 0.97 (Figure 6B).

Table 3 Multivariable Logistic Regression Analysis in the Training Cohort

Variable	OR	95% CI	*P* value
MLR	3.813	1.066–12.782	0.033
RDW	1.463	1.261–1.711	< 0.001
ALP	1.005	1.003–1.006	< 0.001

Abbreviations: MLR, monocyte-to-lymphocyte ratio; RDW, red blood cell distribution width; ALP, alkaline phosphatase.

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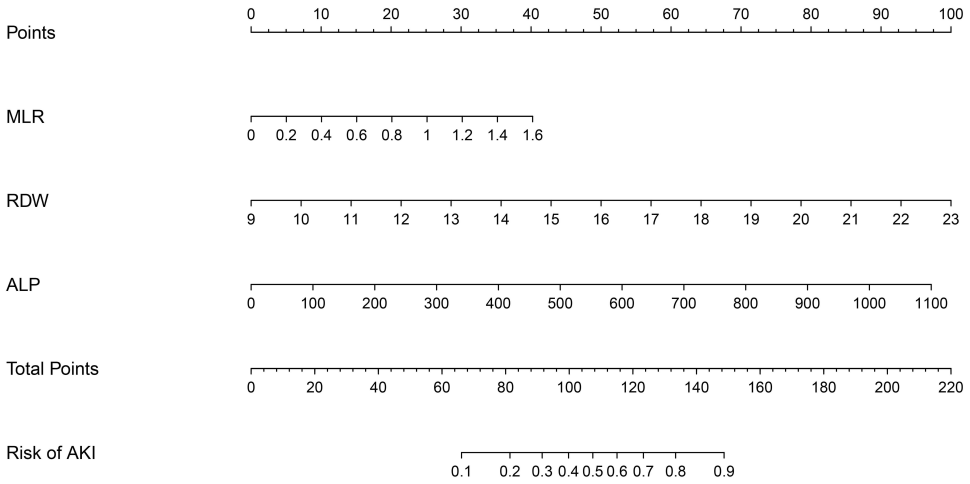

Figure 3 The nomogram for predicting postoperative AKI in patients undergoing pancreaticoduodenectomy.

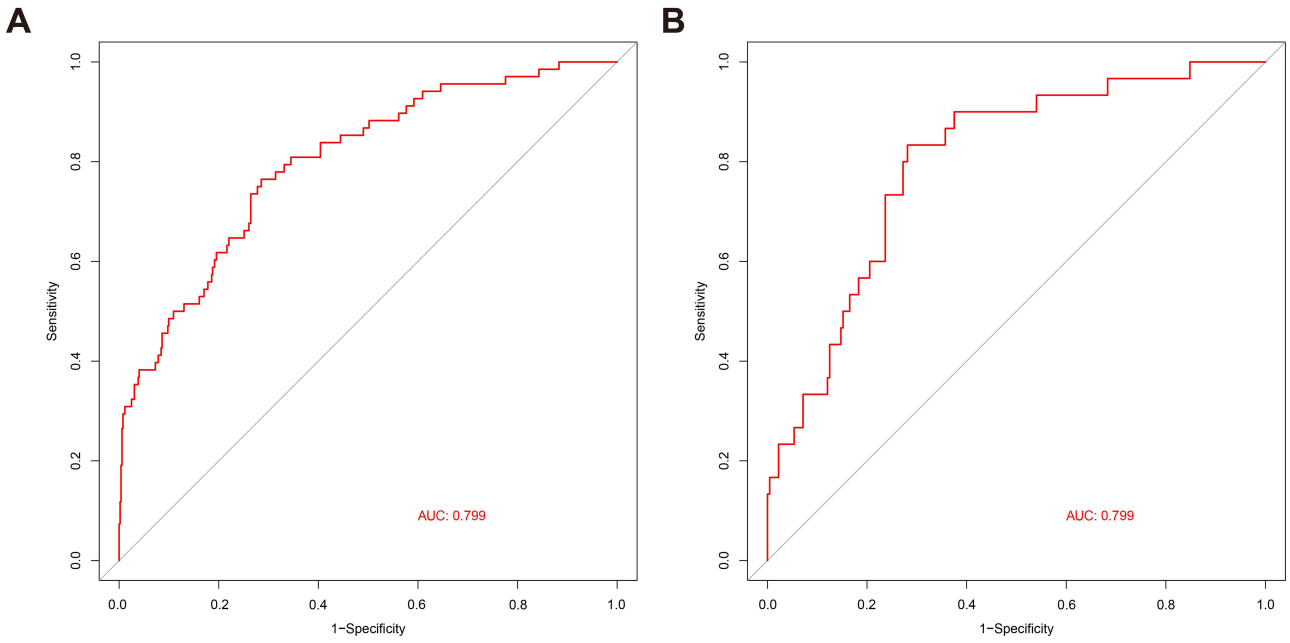

Figure 4 ROC curves of the predictive model in the training group (**A**) and validation group (**B**).

Discussion

This study developed a nomogram model incorporating MLR, RDW, and ALP to predict early-stage AKI risk following pancreaticoduodenectomy. The nomogram model demonstrates moderate discrimination, as evidenced by AUC values exceeding 0.7 in both the training and validation groups. In clinical practice, this means that the model can effectively distinguish patients at high and low risk of AKI following pancreaticoduodenectomy, which may help clinicians with early risk stratification, timely monitoring, and targeted intervention to improve renal prognosis.The calibration curve shows that the nomogram model's predicted and actual probabilities are consistent, indicating high calibration ability. The decision curve showed a good net benefit rate for diagnosing AKI after pancreaticoduodenectomy using this model.

Inflammation is widely recognized as a key pathological mechanism in AKI,[11] supporting the rationality of establishing postoperative AKI prediction models using baseline inflammatory markers. The predictive value of inflammatory markers (MLR, NLR, PLR, SIRI) for postoperative AKI is surgery-specific. Significant differences exist in the key inflammatory

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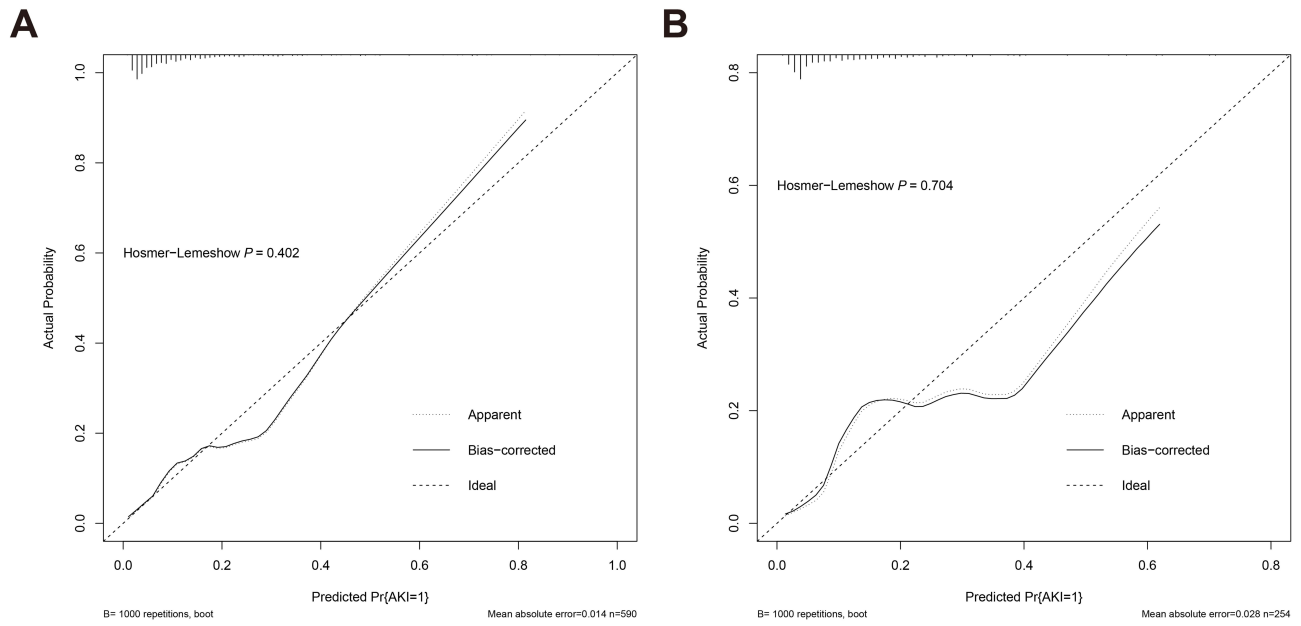

Figure 5 Calibration curves of the predictive model in the training group (**A**) and validation group (**B**).

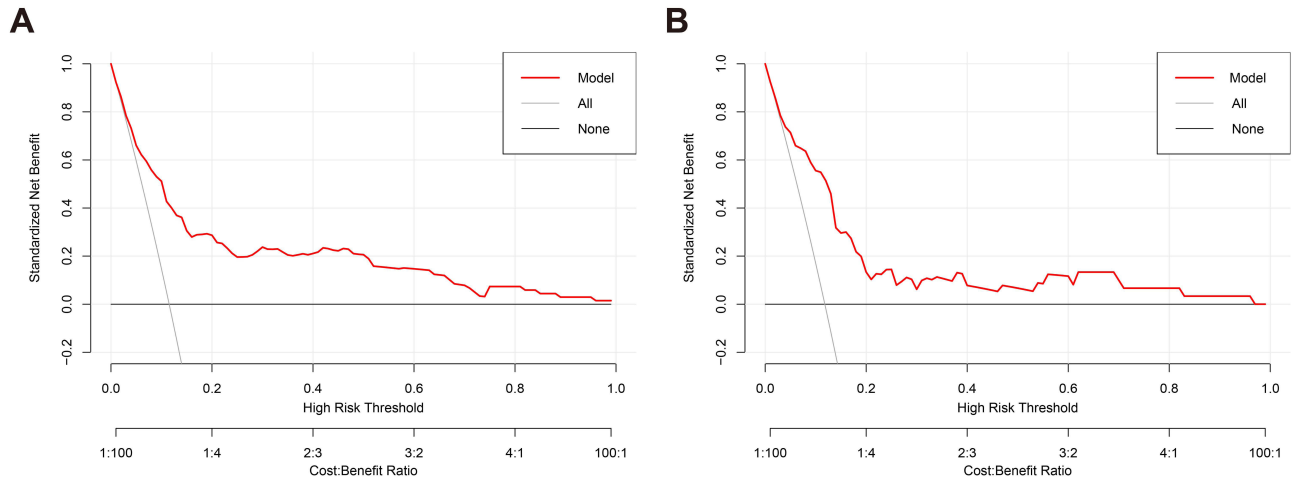

Figure 6 DCA curves of the prediction model in the training group (**A**) and validation group (**B**).

markers that exert critical predictive effects across different surgical procedures, and the detection time window of these markers also exhibits a surgery-dependent pattern. Previous studies have clearly demonstrated that in patients undergoing joint arthroplasty, SIRI and MLR can serve as potential predictors of postoperative AKI.[12] In elderly patients undergoing laparoscopic abdominal surgery, preoperative NLR was positively correlated with AKI severity, and its combination with nutritional markers further improved predictive performance.[22] Among patients undergoing cardiopulmonary bypass cardiac surgery, PLR at 6–12 hours postoperatively was independently associated with AKI.[23] In our study, we evaluated five inflammatory markers (MLR, NLR, PLR, SII, and SIRI) and found that only MLR showed predictive value for AKI after this surgical procedure. This difference suggests that the predictive efficacy of inflammatory markers is not universal. In clinical practice and research, it is necessary to select appropriate inflammatory markers and testing time windows based on the specific type of surgery in order to improve the accuracy of AKI prediction.

The RDW serves to indicate the degree of variation in red blood cell size, thereby assisting in the assessment and diagnosis of diverse hematological conditions. Accumulating evidence indicates that elevated RDW levels are strongly correlated with

a higher risk of AKI and worse subsequent outcomes across various patient populations. For instance, a study of patients undergoing coronary angiography revealed that elevated preoperative RDW levels were associated with a higher incidence of contrast-induced AKI.[24] Similarly, elevated RDW was related to an increased incidence of AKI and 28-day mortality in patients with acute respiratory distress syndrome.[25] Another investigation of critically ill diabetic patients with concurrent AKI showed that elevated RDW was linked to a significant reduction in 28-day survival and served as a predictor of all-cause mortality.[26] Furthermore, a predictive model incorporating RDW exhibited favorable prognostic value for 28-day mortality in AKI patients undergoing continuous renal replacement therapy.[27] Our findings also confirm that RDW levels are higher in AKI patients and that RDW contributes to predictive modeling, which is consistent with the existing body of research. The association between elevated RDW and the development of AKI remains unclear. Previous studies have reported significant correlations between RDW and inflammatory or oxidative stress markers, such as high-sensitivity C-reactive protein, SOD, MDA, TLR4, and IL-18.[28,29] Elevated RDW indirectly reflects a state of systemic inflammation and oxidative stress, which are the key pathogenic mechanisms in AKI. Therefore, an increase in RDW is more likely to serve as an indicator or suggestive marker for AKI. Further investigation is required to clarify the direct causal relationship between RDW elevation and AKI.

ALP, an endogenous enzyme abundant in bone, intestine and renal tubules, participates in host defense and innate immunity.[30] Basic research has found that ALP exerts potential protective effects against AKI by inhibiting inflammatory responses, modulating purinergic signaling, and improving barrier function.[30] Likewise, the renal protective role of ALP has been confirmed in clinical trials involving human subjects. Recombinant human ALP has shown potential to reduce adverse renal events in sepsis-associated AKI patients.[31] A recent study assessing the perioperative administration of ALP in living donor kidney transplantation found it to be safe and feasible.[32] Although previous studies have consistently reported the renal benefits of ALP supplementation, our study and previous clinical investigations[33-35] both found that higher concentrations of ALP are a risk factor for AKI. This finding may suggests that the role of ALP is context-dependent. The elevated circulating ALP may reflect a compensatory response to tubular injury or may simply be a marker of cellular damage release, rather than an effective protective mechanism. Consequently, endogenous ALP concentration may serve primarily as a risk marker reflecting disease severity, whereas its therapeutic benefits are likely achieved only at supra-physiological, pharmacological doses.

The study's limitations include the lack of routine postoperative urine output measurement, restricting AKI diagnosis to serum creatinine levels and potentially underestimating postoperative AKI incidence due to the inability to identify cases based on reduced urine volume. Additionally, all cases in this study came from a single institution. Although internal validation was performed, both the training and validation cohorts were drawn from the same hospital, which may introduce selection bias. Therefore, larger, multi-center studies are needed to validate these clinical outcomes. Finally, our study only focused on preoperative inflammatory marker levels and did not collect postoperative inflammatory data. Pancreaticoduodenectomy is the most extensive abdominal surgery, and the inflammatory injury it induces cannot be overlooked. Changes in postoperative inflammatory levels are equally important for the development and progression of AKI. Therefore, attention should be given to the predictive value of dynamic changes in inflammatory markers for postoperative AKI.

Conclusion

In conclusion, the study identified three key predictors: monocyte-to-lymphocyte ratio (MLR), red blood cell distribution width (RDW), and alkaline phosphatase (ALP) to develop nomograms with moderate discriminative power in both training and validation cohorts, confirming the model's validity and applicability. The incidence of AKI after pancreaticoduodenectomy could be predicted clinically based on the sum of scores for each risk factor.

Data Sharing Statement

The data supporting this study's findings can be obtained from the corresponding author (Fengmin Shao, E-mail: fengminshao@126.com) upon request.

Ethics Approval and Informed Consent

Approval for the study was obtained from the Ethics Committees of Henan Provincial People's Hospital (Ethics approval number: 2021-Lunshen-77), and informed consent was waived because it was a retrospective study. This study was

conducted according to the Declaration of Helsinki. In addition, this study followed the RECORD (REporting of studies Conducted using Observational Routinely-collected health Data) guideline.

Author Contributions

Wenwen Zhang: Conceptualization, Formal analysis, Investigation, Writing–Original Draft.
Zengyuan Qin: Formal analysis, Investigation, Writing–review & editing.
JunTao Wang: Formal analysis, Investigation, Data Curation.
Chunling Huang: Methodology, Data Curation, Software.
Xiaoru Zhao: Methodology, Investigation, Software.
Ziyang Liu: Investigation, Formal analysis, Writing–review & editing.
Limeng Wang: Methodology, Investigation, Funding Acquisition.
Lei Yan: Validation, Data Curation, Software, Visualization.
Yue Gu: Supervision, Resources, Conceptualization, Funding Acquisition.
Fengmin Shao: Supervision, Resources, Conceptualization, Project administration, Funding Acquisition.

All authors contributed significantly to this work in various aspects, including but not limited to the conception, design, execution, data acquisition, analysis, or interpretation. They also participated in writing or critically reviewing the article, approved the final version for publication, agreed on the submission journal, and take responsibility for all parts of the study.

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Disclosure

The authors state they have no conflicts of interest.

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