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ORIGINAL RESEARCH

Development of a Prognostic Model for Prolonged Hospital Stay After Gastrointestinal Perforation Surgery

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Objective: This study aimed to develop and validate a prognostic nomogram integrating clinical and laboratory variables to predict prolonged hospital stay in patients undergoing surgery for gastrointestinal (GI) perforation, facilitating early risk stratification and informed clinical decision-making.

Patients and Methods: A retrospective retrospective single-center study included 164 surgical patients with GI perforation from 2022–2024. Variables encompassed demographics, perforation characteristics, and preoperative/postoperative laboratory markers. The least absolute shrinkage and selection operator (LASSO) regression identified key predictors, followed by multivariate logistic regression to construct a nomogram. Model performance was evaluated using the receiver operating characteristic (ROC) curves, calibration plots, and decision curve analysis (DCA).

Results: Upper GI perforation (OR=2.93, 95% CI:1.23–6.98), smaller perforation diameter (OR=0.48, 95% CI:0.28–0.82), and lower preoperative albumin (OR=1.10 per unit increase, 95% CI:1.03–1.17) independently predicted prolonged hospitalization. The nomogram demonstrated good discrimination (training AUC=0.75; validation AUC=0.79) and calibration. DCA confirmed clinical utility, with net benefit surpassing "treat all" or "treat none" strategies across risk thresholds.

Conclusion: In summary, we developed and validated a nomogram that effectively identifies patients at high risk for prolonged hospitalization after GI perforation surgery by integrating three routinely available clinical parameters. This tool aids in optimizing resource allocation and personalized perioperative management. Further multicenter validation is warranted to enhance generalizability and incorporate dynamic biomarkers.

Keywords: gastrointestinal perforation, prognostic model, postoperative outcomes, nomogram

Introduction

Gastrointestinal perforation is a common but life-threatening surgical emergency characterized by acute onset, rapid progression, and a high mortality rate.[1] If not promptly managed, it can lead to diffuse peritonitis, septic shock, and even death.[2] Peptic ulcer disease remains the most frequent cause of GI perforation,[3] while other etiologies include the use of nonsteroidal anti-inflammatory drug, diverticular disease, connective tissue disorders, malignancies, iatrogenic injuries, and trauma.[4–8] Following perforation, patients typically present with sudden abdominal pain, which may initially be localized but can quickly evolve into signs of generalized peritonitis due to chemical and bacterial contamination of the peritoneal cavity. This can be accompanied by fever, vomiting, paralytic ileus, and, in severe cases, toxic or septic shock.[2]

Emergency surgical intervention remains the primary treatment approach for most patients with gastrointestinal perforation, aiming to control the source of infection and repair the defect.[9] However, in certain cases, particularly in patients under the age of 70 who have well-contained perforations or are considered unfit for surgery due to comorbid conditions, non-operative management may be an alternative.[10] This approach typically includes gastrointestinal decompression, antibiotic therapy, and the administration of proton pump inhibitors.[3,11] Nevertheless, conservative treatment is

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associated with risks such as intra-abdominal abscess, persistent leakage,[11] delayed surgical intervention, prolonged hospitalization, and worsening of infection, all of which can substantially increase morbidity and mortality.[12,13] Therefore, it is essential for surgeons to make timely and accurate treatment decisions and to carefully determine the appropriate timing for surgical intervention.

Recent studies have increasingly focused on identifying prognostic factors that influence recovery in patients with gastrointestinal perforation. Inflammatory markers such as the neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) have been shown to be associated with mortality in patients with perforated peptic ulcers.[14] Meta-analyses have confirmed that hypoalbuminemia is positively correlated with an increased risk of mortality.[15] Other investigations have evaluated the prognostic value of routinely available laboratory parameters and composite indices, including the systemic immune-inflammation index (SII), prognostic nutritional index (PNI), and the hemoglobin, albumin, lymphocyte, and platelet (HALP) score. Factors such as perforation site, serum total protein, albumin, hemoglobin, NLR, lymphocyte-to-monocyte ratio (LMR), PLR, and PNI have all been identified as predictors of poor postoperative outcomes.[16] However, a validated prognostic model that integrates routinely available inflammatory and nutritional parameters to specifically predict prolonged hospital stay is still lacking, even though prolonged hospitalization is a key determinant of resource utilization and recovery trajectory.

No prior studies have integrated multiple routine inflammatory and nutritional parameters into a validated prognostic nomogram for postoperative recovery in GI perforation. Unlike previous studies that focused on isolated biomarkers or clinical factors, this study integrates routinely available clinical and laboratory variables into a user-friendly nomogram for early risk stratification in GI perforation patients, which is particularly valuable for primary care and resource-limited settings.

Materials and Methods

Study Design and Population

This retrospective observational study enrolled patients diagnosed with GI perforation who underwent surgical treatment at Pingjiang First People's Hospital between January 2022 and December 2024.

Inclusion criteria: patients aged ≥18 years with confirmed GI perforation who underwent surgical repair. Exclusion criteria: patients managed conservatively, those with incomplete medical records, or those who died within 48 hours postoperatively.

The study protocol was approved by the Institutional Review Board of Pingjiang First People's Hospital, and written informed consent was obtained from all participants. The ethics approval number is KY-20250320001.Clinical trial number: not applicable. All procedures were conducted in accordance with the Declaration of Helsinki.

Data Collection

Clinical data were extracted from the hospital's electronic medical record system. Collected variables included demographic characteristics (age, sex, height, and weight), routinely obtained laboratory indicators (preoperatively and on postoperative Days 1 and 2), surgical details, etiology and size of the perforation, total length of hospital stay, and the presence of complications such as sepsis or infectious shock. Based on the median length of stay (12 days), patients were categorized into short-stay and long-stay groups. The 12-day cutoff was determined based on preliminary analysis of our cohort data, which indicated this median as a clinically relevant and practical threshold for distinguishing prolonged hospitalization in our institutional setting.

Prior to analysis, the dataset was carefully reviewed, variables with >20% missing data were excluded from analysis, while variables with ≤20% missing data were handled using multiple imputation. In addition, ICU admission was not included in the analysis because of its potential to directly influence hospital stay and introduce bias into the predictive modeling process.

Statistical Analysis

All statistical analyses were performed using R software (version 4.3.2). Descriptive statistics were used to summarize the data. Continuous variables were tested for normality using the Shapiro–Wilk test. Non-normally distributed variables were presented as medians and interquartile ranges, and compared between the two groups using the Mann–Whitney *U*-test. Categorical variables were expressed as frequencies and percentages, and group comparisons were made using the chi-square test or Fisher's exact test where appropriate.

To identify potential predictors of prolonged hospitalization, the LASSO regression was first used for variable selection. Given the cohort size and to mitigate potential overfitting in multivariable modeling, LASSO was used to reduce the number of candidate predictors, with an events-per-variable (EPV) ratio considered adequate for stable estimation in conjunction with regularization. The top five variables identified by LASSO were then subjected to univariate logistic regression, followed by multivariate logistic regression to determine independent predictors. Based on the multivariate model, a nomogram was constructed to estimate the individual probability of prolonged hospital stay. The dataset was randomly divided into a training cohort (70%) and a validation cohort (30%). The predictive performance of the model was assessed using ROC curves and the area under the curve (AUC) to evaluate discrimination. Calibration of the model was verified using calibration plots based on 1000 bootstrap samples. Clinical applicability was further evaluated through DCA.

Result

Participants Characteristics

A total of 164 patients with gastrointestinal perforation who underwent surgery were included in the study. These patients were divided into two groups based on whether their length of hospital stay was greater than or equal to 12 days: the long-stay group and the short-stay group. Tables 1 and 2 provides a detailed overview of all predictive variables for the dataset.

LASSO Regression Analysis Was Employed to Screen for Important Variables

Lasso regression was employed to select important variables by constraining regression coefficients, thereby shrinking the coefficients of less important variables to zero. Figure 1A illustrates the Lasso path, depicting how coefficients of different variables are reduced as the regularization parameter (alpha) increases. The optimal alpha, corresponding to the

Table 1 Characteristics of Included Patients Stratified by Length of Hospital Stay

Variables	Total (n = 164)	<12d Hospital Stay (n = 72)	≥12d Hospital Stay (n = 92)	Statistic	*P*
Age*	68.00 (56.75, 75.00)	71.00 (61.00, 76.00)	65.50 (51.00, 73.00)	Z=−2.68	0.007
Gender, n (%)				χ^2=1.13	0.287
Female	39 (23.78)	20 (27.78)	19 (20.65)		
Male	125 (76.22)	52 (72.22)	73 (79.35)		
BMI*	20.94 (19.49, 21.48)	20.94 (18.65, 22.03)	20.94 (20.23, 21.36)	Z=−1.23	0.219
Hypertension, n (%)				χ^2=0.03	0.867
No	115 (70.12)	50 (69.44)	65 (70.65)		
Yes	49 (29.88)	22 (30.56)	27 (29.35)		
Gastrointestinal tract, n (%)				χ^2=6.15	0.013
Lower	58 (35.37)	33 (45.83)	25 (27.17)		
Upper	106 (64.63)	39 (54.17)	67 (72.83)		
Reason, n (%)				χ^2=3.27	0.353
Gastroduodenal ulcer	32 (19.51)	11 (15.28)	21 (22.83)		
Long-term oral medication	26 (15.85)	11 (15.28)	15 (16.30)		
Other	40 (24.39)	22 (30.56)	18 (19.57)		
Unknown	66 (40.24)	28 (38.89)	38 (41.30)		
Diameter*	0.80 (0.50, 1.50)	1.00 (0.60, 2.12)	0.60 (0.50, 1.00)	Z=−3.95	<0.001

Table 2 Blood Test Indicators of Included Patients Stratified by Length of Hospital Stay

Variables	Total (n = 164)	<12d Hospital Stay (n = 72)	≥12d Hospital Stay (n = 92)	Statistic	*P*
Albumin					
0	42.10 (36.15, 45.02)	39.45 (33.68, 42.45)	43.60 (39.25, 46.00)	Z=−4.55	<0.001
Hb					
0	128.50 (109.75, 143.00)	119.00 (98.50, 135.00)	131.00 (116.75, 145.25)	Z=−3.08	0.002
1	−9.95 (−17.51, −0.78)	−8.33 (−15.38, 0.00)	−11.88 (−19.08, −3.57)	Z=−1.86	0.063
2	−0.83 (−6.81, 8.07)	−2.42 (−8.41, 6.61)	0.00 (−5.06, 8.12)	Z=−1.65	0.099
WBC					
0	10.88 (7.22, 15.33)	10.53 (5.69, 15.57)	10.91 (7.92, 14.47)	Z=−0.37	0.714
Lymph					
0	0.56 (0.36, 0.92)	0.55 (0.34, 0.79)	0.56 (0.37, 0.96)	Z=−0.86	0.390
1	4.16 (−28.90, 57.90)	−4.24 (−37.02, 40.27)	11.24 (−10.81, 62.88)	Z=−1.80	0.072
2	24.82 (−9.88, 68.95)	39.37 (−7.26, 85.14)	13.99 (−12.12, 56.24)	Z=−1.42	0.155
Neut					
0	9.62 (6.15, 13.04)	9.79 (5.38, 13.62)	9.54 (6.76, 12.63)	Z=−0.09	0.931
1	−3.73 (−33.85, 31.81)	−1.09 (−33.18, 58.66)	−8.50 (−34.13, 30.88)	Z=−0.58	0.562
2	−24.62 (−46.29, 5.55)	−14.01 (−40.25, 26.17)	−32.49 (−51.57, −15.06)	Z=−3.44	<0.001
Monocyte					
0	0.41 (0.24, 0.58)	0.40 (0.20, 0.53)	0.43 (0.30, 0.59)	Z=−1.50	0.132
1	0.00 (−31.86, 35.41)	1.03 (−32.33, 52.50)	0.00 (−31.82, 32.33)	Z=−0.67	0.504
2	11.15 (−16.87, 61.54)	19.82 (−6.28, 74.31)	7.15 (−20.71, 51.38)	Z=−1.89	0.058
Platelet					
0	216.50 (182.25, 275.00)	211.00 (178.00, 291.25)	218.50 (184.50, 270.75)	Z=−0.03	0.976
1	−15.36 (−25.56, 0.00)	−14.22 (−27.59, 0.00)	−15.61 (−24.32, 0.00)	Z=−0.60	0.547
2	4.74 (−10.78, 22.17)	−0.78 (−15.10, 19.19)	7.32 (−7.32, 23.35)	Z=−1.81	0.070
SII					
0	3239.72 (1912.09, 4206.61)	3595.73 (1617.29, 6312.95)	3182.70 (2037.49, 5681.82)	Z=−0.07	0.941
1	−10.73 (−41.89, 33.31)	−3.12 (−41.82, 65.07)	−16.33 (−42.19, 18.35)	Z=−1.01	0.315
2	−33.66 (−55.52, 4.11)	−9.42 (−50.84, 30.52)	−35.41 (−57.85, −1.85)	Z=−1.40	0.163
PNI					
0	45.20 (39.11, 48.55)	41.83 (36.69, 46.07)	46.62 (42.89, 50.25)	Z=−4.30	<0.001
HALP					
0	12.76 (7.27, 21.12)	10.32 (5.73, 18.05)	15.03 (8.66, 23.16)	Z=−2.83	0.005
PLR					
0	389.48 (234.42, 631.53)	397.60 (249.01, 695.68)	382.72 (226.55,607.50)	Z=−1.07	0.286
1	−19.67 (−45.52, 15.92)	−3.70 (−42.35, 25.70)	−23.29 (−46.73, 1.58)	Z=−1.29	0.197
2	−17.74 (−38.85, 12.22)	−27.83 (−43.82, 1.07)	−9.63 (−29.74, 15.82)	Z=−2.51	0.012
NLR					
0	15.40 (9.58, 26.28)	14.11 (9.56, 9.60)	15.78 (9.58, 25.98)	Z=−0.06	0.948
1	−8.51 (−47.67, 47.94)	0.00 (−47.94, 75.31)	−21.38 (−47.16, 25.45)	Z=−1.56	0.118
2	−38.17 (−65.46, 8.38)	−31.89 (−56.64, 19.68)	−44.65 (−67.90, −11.92)	Z=−2.03	0.042
LMR					
0	1.59 (0.88, 2.52)	1.73 (0.91, 3.25)	1.51 (0.87, 2.21)	Z=−0.91	0.363
1	−8.51 (−47.67, 47.94)	0.00 (−47.94, 75.31)	−21.38 (−47.16, 25.45)	Z=−1.56	0.118
2	−38.17 (−65.46, 8.38)	−31.89 (−56.64, 19.68)	−44.65 (−67.90, −11.92)	Z=−2.03	0.042

Notes: Variables in the table are expressed as medians with interquartile ranges, denoted as M (Q_1, Q_3). Variables with suffixes "0" "1" and "2" are indicates preoperative tests, the first and second postoperative tests, respectively. *P* <0.001 means that when the statistical analysis yields a very small p-value, shown as 0.000, it is expressed in the table as *P* <0.001. The term "change n" denotes the rate of change between consecutive test results.

minimum log loss on the cross-validation set, was determined (Figure 1B). Ultimately, Albumin0, Gastrointestinal tract, Diameter, and Neut_change2 were identified as risk factors (Figure 1C).

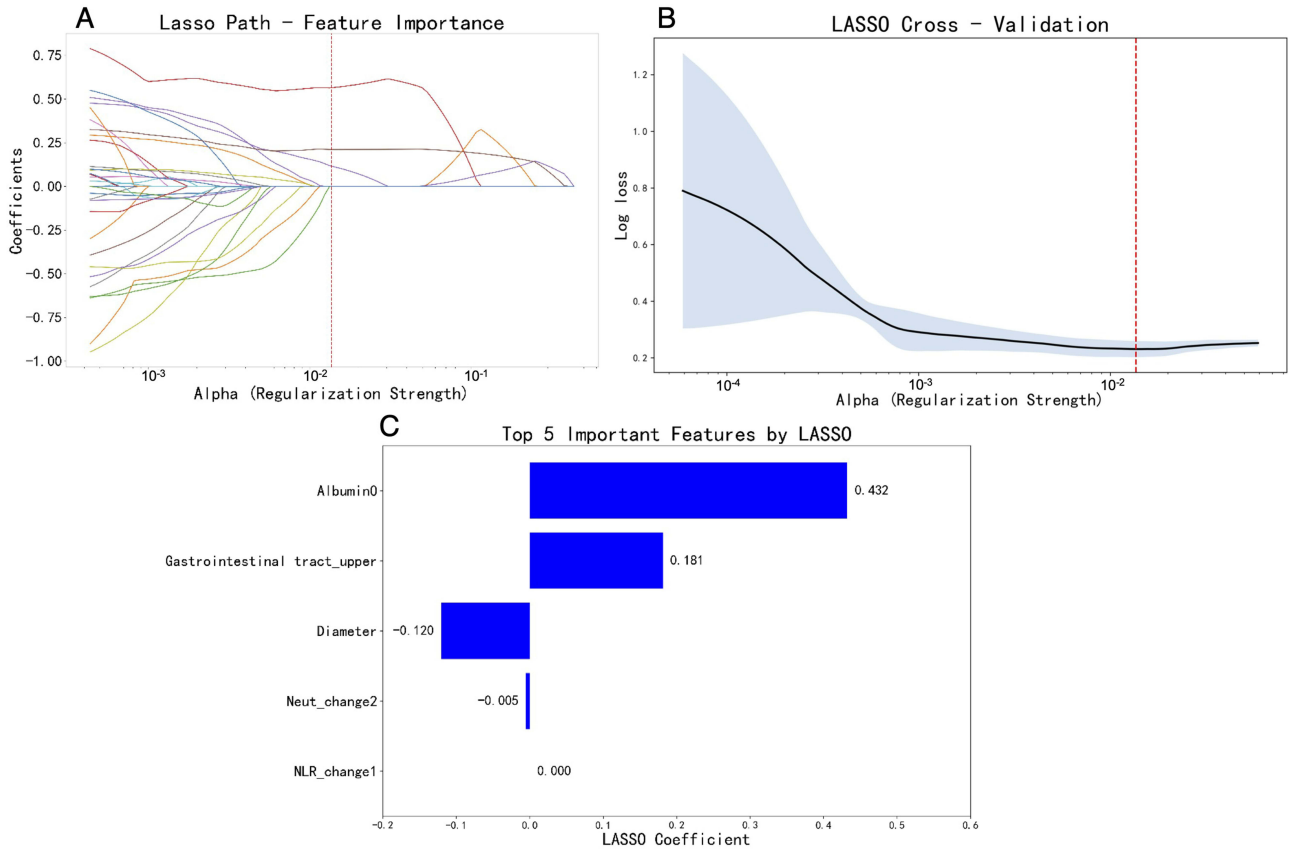

Figure 1 LASSO variable selection process. (**A**) The Lasso path. The red vertical line indicates the optimal alpha value, corresponding to the minimum model error. When coefficients shrink to zero below this line, it signifies that the variables are relatively unimportant in the Lasso regression model. (**B**) The cross-validation performance of LASSO, illustrating the Log loss and its fluctuation range under different regularization strengths. (**C**) The important variables selected by Lasso regression. After Lasso regression screening, Albumin0, Gastrointestinal tract, Diameter, and Neut_change2 were identified as important variables.

Variable Selection for the Nomogram

The results of univariate and multivariate logistic regression analyses (Table 3) showed that patients with upper gastrointestinal tract perforations had a significantly higher risk of poor outcomes compared to those with lower gastrointestinal perforations, with adjusted odds ratio (OR) of 2.93 (95% CI: 1.23–6.98, P=0.016). Smaller perforation diameter was associated with increased risk, with an adjusted OR of 0.48 (95% CI: 0.28–0.82, P=0.008). Higher preoperative albumin levels were linked to better prognosis (adjusted OR 1.10, 95% CI: 1.03–1.17, P=0.004). Changes in neutrophil count on postoperative day 2 showed a slight association with outcomes in univariate analysis (OR 0.99, 95% CI: 0.99–0.99, P=0.036) but were not significant in multivariate analysis. Overall, upper GI perforation location, smaller perforation size, and lower preoperative albumin were identified as independent predictors of poorer prognosis.

Table 3 Results of Univariate and Multivariate Logistic Regression

Variables	Univariate		Multivariate	
	OR (95% CI)	*P*	OR (95% CI)	*P*
Gastrointestinal tract				
Lower	1.00 (Reference)		1.00 (Reference)	
Upper	2.37 (1.10–5.12)	0.028	2.93 (1.23–6.98)	0.016
Diameter	0.51 (0.32–0.81)	0.004	0.48 (0.28–0.82)	0.008
Albumin0	1.11 (1.04–1.18)	0.001	1.10 (1.03–1.17)	0.004
Neut change2	0.99 (0.99–0.99)	0.036		

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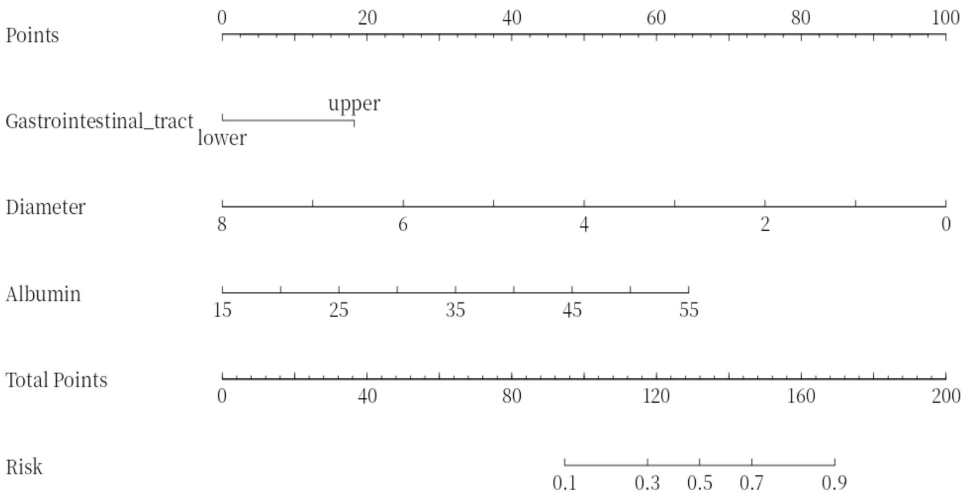

Figure 2 Nomogram for risk prediction of the length of hospital stay for surgical patients with gastrointestinal perforation/rupture. GI tract upper, Diameter, and Albumin0 were identified as independent risk factors. Different values of each variable correspond to different scores. Points: Represents the score points corresponding to the vertical line of each variable. Total Points: Obtained by summing up the score points of all selected factors. Risk: Based on the total score, the risk probability of the patient's length of hospital stay can be read via the probability conversion axis on the nomogram.

Nomogram Construction and Validation

In this study, we constructed a nomogram model (Figure 2) to predict the length of hospital stay for surgical patients with gastrointestinal rupture based on the significant factors from the multivariate Logistic regression.

To evaluate the performance of this model, we used the Bootstrap method for internal validation, specifically performing 1000 Bootstrap resampling procedures. Additionally, we employed the Hosmer–Lemeshow test to assess the goodness - of - fit of the model. The P-values obtained for the training set and the test set were 0.172 and 0.144, respectively. Given that these P-values exceed 0.05, they do not indicate poor model fit. Furthermore, by plotting the ROC curve (Figure 3), we evaluated the discriminatory ability of the model. The AUC value of the training set was 0.75 (95% confidence interval: 0.65–0.84), and that of the validation set was 0.79 (95% confidence interval: 0.67–0.92). This indicates that the predictive model showed good discrimination in both the training and validation sets. Moreover, we used the Calibration curves (Figure 4A and B) to compare the agreement between the actual probability and the predicted probability of the length of hospital stay. The results showed good consistency between the actual and predicted probabilities in both the training and validation sets. Finally, we evaluated the clinical utility of the prediction model for the length of hospital stay at different thresholds through decision curve analysis (DCA). The results indicated that the net benefit of the overall model in both the training and validation sets was significantly higher than the "treat all" (ALL) and "treat none" (NONE) strategies between the risk thresholds of 0.05–0.8 (as shown in Figure 4C and D). This suggests that the model has practical application value in clinical decision - making.

Discussion

The primary aim of this study was to identify independent predictors of prolonged hospital stay in patients undergoing surgery for GI perforation and to develop a simple, clinically applicable nomogram for early risk stratification. By integrating key clinical characteristics and routinely available laboratory parameters, we sought to provide a practical tool to support perioperative decision-making and postoperative management. The results demonstrated that perforation location, perforation size, and preoperative serum albumin level were the most influential factors associated with prolonged hospitalization.

The finding that upper GI tract perforations are associated with a significantly higher risk of adverse outcomes and longer hospital stay is in line with prior research. Anatomically and physiologically, the upper GI tract, including the stomach and duodenum, contains a higher concentration of digestive enzymes and bacterial flora compared to the lower tract.[17] This leads to more severe peritoneal contamination and systemic inflammatory responses following perforation,

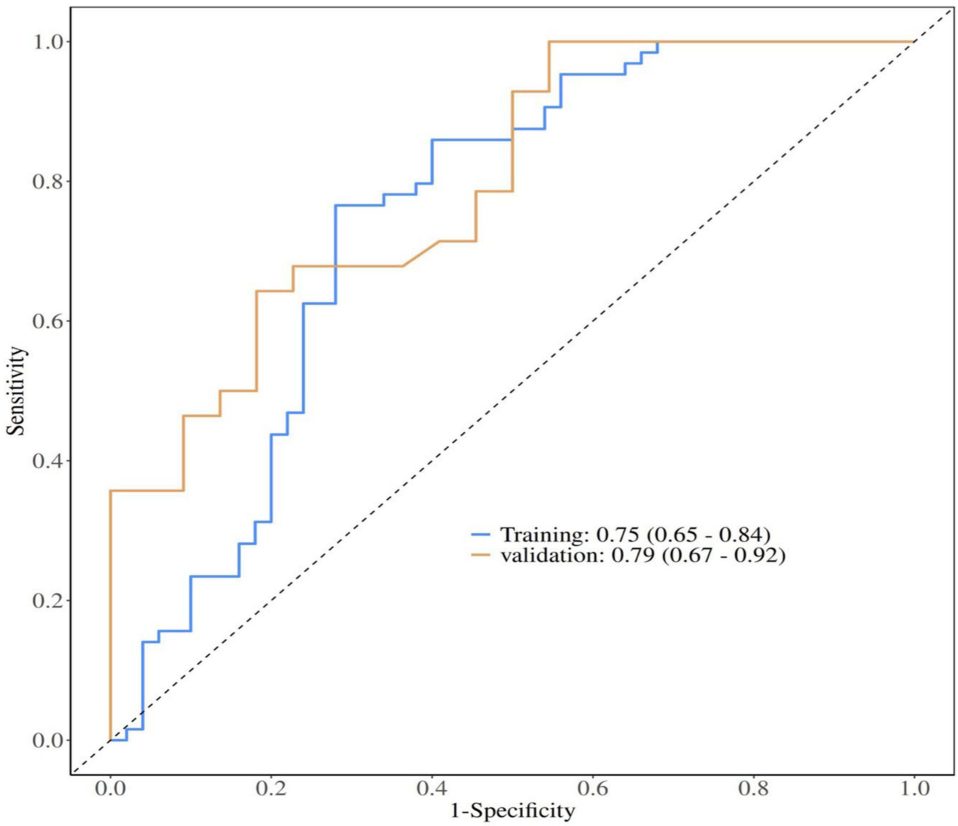

Figure 3 ROC curves for the model training and validation sets. The ROC curves were used to assess the model's discriminative ability. The model achieved high AUC values of 0.75 in the training set and 0.79 in the test set.

resulting in more complicated clinical courses. Previous studies have reported increased morbidity and mortality rates in patients with upper GI perforations compared to those with lower tract perforations, which is attributable to the more aggressive local and systemic sequelae.[18] Our findings corroborate this clinical reality, underscoring the need for heightened vigilance and possibly more aggressive perioperative management in this subgroup.

Interestingly, our data indicated that smaller perforation diameter was independently associated with a longer hospital stay, a finding that contrasts with the intuitive expectation that larger perforations lead to more severe complications. While larger perforations might theoretically cause greater immediate contamination, smaller perforations could be more difficult to detect clinically and radiologically, potentially resulting in delayed diagnosis and intervention. It is plausible that such diagnostic delays, rather than the perforation size itself, might contribute to the progression of localized infection and subsequently extend the recovery period. Consequently, missed or initially underestimated small perforations may evolve into abscess formation, diffuse peritonitis, or systemic sepsis, thereby prolonging hospitalization. This observation underscores the need for thorough diagnostic evaluation, including repeated imaging and close clinical surveillance, particularly when initial findings are equivocal. Further research is warranted to investigate whether enhanced diagnostic approaches or novel biomarkers could facilitate earlier identification and management of such cases.

Serum albumin emerged as a key protective factor, consistent with a large body of literature linking nutritional status and systemic inflammation to surgical outcomes.[19] Albumin reflects both the patient's baseline nutritional reserve and the presence of systemic inflammatory states.[20] Hypoalbuminemia is associated with impaired wound healing, increased susceptibility to infections, and overall poorer resilience to surgical stress.[21] In GI perforation patients, low preoperative albumin may indicate chronic malnutrition or ongoing inflammation, both of which compromise recovery. Our findings reinforce the clinical importance of early nutritional assessment and, where possible, preoperative optimization through nutritional support to improve outcomes. This also aligns with enhanced recovery after surgery principles, which advocate for the correction of nutritional deficits before surgery.

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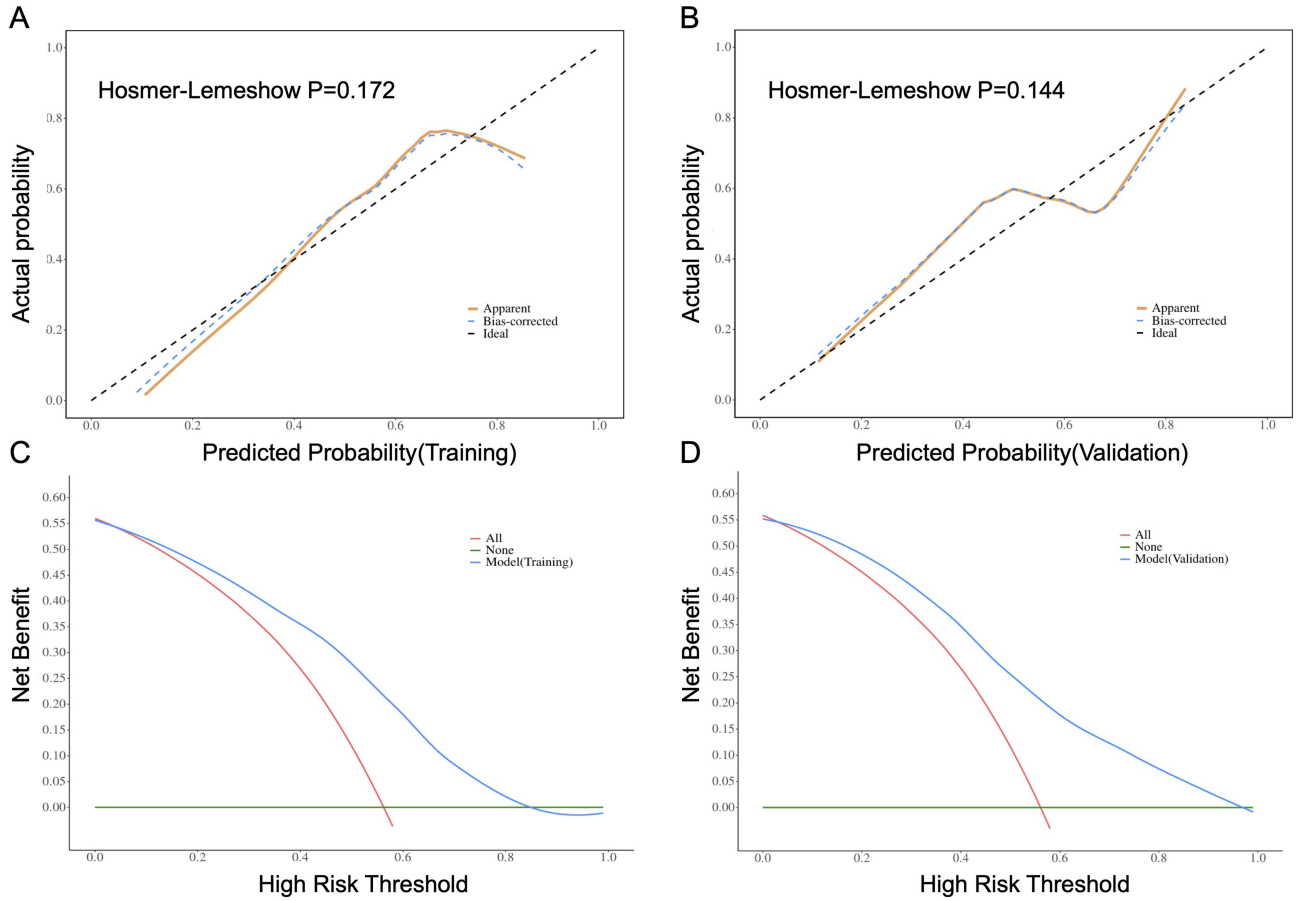

Figure 4 Calibration and clinical decision curves of the model. (**A** and **B**) The calibration curves for the training and validation sets, respectively, indicating that the predicted probabilities of length of stay align well with observed outcomes in both sets. (**C** and **D**) The DCA for the training and validation sets, respectively. Model: Net benefit of treatment decisions based on the prediction model. ALL: Net benefit assuming all patients are treated, calculated based on the actual incidence of prolonged length of stay. NONE: Net benefit assuming no patients are treated, set at zero. High Risk Threshold: Probability at which patients or clinicians consider treatment worthwhile if the predicted length of stay exceeds a certain threshold. Net Benefit: The proportion of true positives minus the proportion of false positives weighted by the threshold probability.

Although neutrophil count changes on postoperative day 2 showed a statistically significant association with outcomes in univariate analysis, this did not hold in multivariate modeling, suggesting that its predictive value may be limited when considered alongside other variables. Neutrophil dynamics reflect the acute inflammatory response to surgery and infection,[22] but the complexity of immune responses likely requires more comprehensive markers or composite indices to better capture patient trajectories. Parameters such as NLR, PLR, or SII have shown promise in other studies and might provide more robust predictive information if included in future models.

The nomogram we constructed demonstrated favorable discrimination and calibration in both training and validation cohorts, with AUC values exceeding 0.75, indicating good predictive accuracy. Decision curve analysis further confirmed the clinical utility of the model by showing higher net benefit across a wide range of risk thresholds compared with universal treatment or no treatment strategies. This suggests that the model could meaningfully aid clinical decision-making by identifying patients at higher risk for prolonged hospitalization, who may benefit from intensified monitoring, early interventions, or tailored perioperative care pathways. Such risk stratification is especially valuable in resource-limited settings or primary care hospitals, where rapid and reliable prognostic tools can help prioritize care and allocate resources efficiently.

Nevertheless, several limitations should be acknowledged. This was a retrospective, single-center study, which may limit the generalizability of the findings due to institutional practice patterns and population characteristics. The exclusion of variables with significant missing data, such as postoperative day 3 laboratory markers and ICU admission status, was necessary to maintain data integrity but may have omitted potentially important predictors. Additionally, the sample size,

while adequate for initial modeling, warrants expansion and external validation in multicenter, prospective cohorts to confirm model robustness and applicability across diverse clinical settings. Furthermore, the model currently focuses on predicting length of hospital stay as a surrogate for prognosis. Integrating additional outcomes such as postoperative complications, readmission rates, or long-term functional recovery could provide a more comprehensive clinical tool.

Conclusion

In conclusion, this study identifies three key clinical factors as independent predictors of prolonged hospitalization following GI perforation surgery: upper GI perforation, smaller perforation diameter, and lower preoperative albumin. Based on these predictors, we developed and validated a practical nomogram for early risk stratification. This tool enhances individualized perioperative management and supports clinical decision-making, with potential utility in resource optimization, particularly in primary care settings. Future research should focus on multicenter validation and the integration of dynamic biomarkers to further improve predictive accuracy and clinical applicability.

Abbreviations

GI, gastrointestinal; LASSO, least absolute shrinkage and selection operator; ROC, receiver operating characteristic; DCA, decision curve analysis; NLR, neutrophil-to-lymphocyte ratio; PLR, platelet-to-lymphocyte ratio; SII, systemic immune-inflammation index; PNI, prognostic nutritional index; HALP, hemoglobin, albumin, lymphocyte, and platelet; LMR, lymphocyte-to-monocyte ratio; AUC, area under the curve; OR, odds ratio.

Data Sharing Statement

The data used to support the findings of this study are available in the article.

Ethics Approval

The study protocol was approved by the Institutional Review Board of Pingjiang First People's Hospital, and written informed consent was obtained from all participants. The ethics approval number is KY-20250320001. Clinical trial number: not applicable.

Author Contributions

W.Z. conceived the study and designed the research plan. Y.Y., W.F., and L.Z. conducted the experiments, performed data acquisition, and carried out data analysis. Y.Y. was responsible for data visualization. During manuscript preparation, all authors contributed to drafting the initial manuscript and participated in subsequent substantive revisions and critical review of the intellectual content. All authors have agreed on the choice of the submission journal, have reviewed and approved all versions of the manuscript throughout the submission, revision, and final acceptance process, and assume full responsibility for the integrity and accuracy of the work presented.

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Disclosure

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

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