

Differential Risk Factors for Intestinal Ischemia and Intestinal Necrosis in Acute Irreducible Groin Hernia: Development and Internal Validation of a Two-Stage Risk Stratification Model

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Abstract

Background

In acute irreducible groin hernia, progression from intestinal ischemia to necrosis are distinct pathophysiological stages, but studies often treat these outcomes as homogeneous. We investigated stage-specific risk factors and develop two sequential risk stratification models.

Methods

We retrospectively analyzed 224 consecutive adults undergoing emergency surgery for acute irreducible groin hernia (2016–2025), comprising ischemia (n = 100) and non-ischemia (n = 124) groups. Multivariable logistic regression was employed to develop an ischemia model in the full cohort and a necrosis model in the ischemia subgroup. Models were evaluated by area under the curve (AUC), calibration, and decision curve analysis, with internal validation by bootstrapping (1000 resamples).

Results

For the full cohort, independent predictors for intestinal ischemia included symptom duration (OR 1.04, 95% CI 1.01–1.07), cardiovascular disease (OR 5.18, 1.42–18.83), bowel obstruction (OR 9.48, 3.90–23.04), a smaller hernia sac neck diameter (OR 0.85, 0.79–0.92), and an elevated neutrophil-to-lymphocyte ratio (NLR; OR 1.11, 1.04–1.18). The ischemia model demonstrated strong performance with an AUC of 0.906 (0.866–0.946), a Hosmer-Lemeshow P-value of 0.813, and a bootstrap-corrected C-statistic of 0.895. Hypertension (OR 5.06, 1.22–20.91), hernia sac fluid (OR 17.23, 4.67–63.56), and NLR (OR 1.13, 1.01–1.25) can independently predict necrosis in ischemia subgroup. Decision curve analysis indicated net benefit, though single-center optimism bias must be considered.

Conclusions

Risk factors for intestinal ischemia and its subsequent progression to necrosis in acute irreducible groin hernia are stage-specific. Ischemia is driven by symptom duration, cardiovascular disease, bowel obstruction, hernia sac neck diameter, and NLR. Conversely, the progression to necrosis was independently associated with hypertension, the presence of hernia sac fluid, and NLR. These two-stage models offer an exploratory framework for a more nuanced, stage-tailored risk assessment, but require multicenter external validation.

Background

Groin hernia is a common condition in general surgery, affecting 27–43% of men and 3–6% of women over their lifetime [1]. The 2023 updated HerniaSurge International Guidelines recommend replacing

"incarcerated hernia" with "acute irreducible hernia" and advocate initial manual reduction for acute irreducible groin hernias lacking evidence of intestinal ischemia or necrosis [2]. If vascular compromise persists, the bowel wall can progress from reversible ischemia to irreversible necrosis. Studies indicate that approximately 15% of patients with incarcerated groin hernias ultimately develop intestinal necrosis, necessitating emergency bowel resection [3]. Emergency surgical outcomes are reported to vary substantially across healthcare models. Furthermore, when bowel resection is required, mortality increases markedly. For example, a single-center retrospective study conducted in China by Dai et al. reported an overall postoperative complication rate of 40.6% [4], Sæter et al. reported a 25.3% crude mortality within 30 days of bowel resection, based on Danish national registry data [5], and a multicenter study by Brown et al. demonstrated a 16.1% post-resection mortality rate [6]. These figures illustrate that intestinal necrosis is the critical event driving adverse outcomes. However, current preoperative clinical judgment of intestinal ischemia remains heavily reliant on history, physical examination, and non-specific laboratory markers. Early clinical manifestations of intestinal ischemia are often non-specific, particularly in elderly patients where symptoms may be masked by other conditions, frequently leading to diagnostic delay—a key contributor to high mortality [7]. While leukocyte and neutrophil counts are significant risk factors for bowel resection in patients with acute irreducible hernia, their clinical utility is limited by the lack of defined clinical thresholds [8]. Furthermore, intraoperative visual assessment of ischemia is often inaccurate, with the degree frequently overestimated or underestimated, potentially leading to unnecessary bowel resection or unplanned reoperation [3]. Therefore, there is an urgent need to explore preoperative risk stratification strategies based on objective and readily available clinical variables.

To address this need, recent research has focused on identifying factors associated with intestinal ischemia and necrosis, broadly categorized into three dimensions: inflammatory markers, imaging features, and clinical factors. Among these, the neutrophil-to-lymphocyte ratio (NLR) stands out as an extensively studied inflammatory marker, owing to its ease of calculation and its ability to simultaneously reflect inflammatory activation and immunoregulatory imbalance [9]. Xie et al. [10] reported a significant association between NLR levels and the severity of incarcerated hernia (using bowel resection as the endpoint), and Köksal et al. [11] observed higher NLR levels in patients who underwent bowel resection for incarcerated hernia compared to those who did not, though the independent predictive value of NLR needs further validation. Regarding modeling methodology, Zhou et al. [12] applied LASSO regression combined with multivariable logistic regression to select five key features from 24 candidate variables for developing a bowel resection prediction model, retaining neutrophil count as a core variable whose predictive contribution was subsequently confirmed and visualized post hoc using the SHAP interpretability algorithm. Novel indices, such as the systemic immune-inflammation index, have also demonstrated promising diagnostic capabilities [13], and Yildirim et al. [14] proposed the lymphocyte-to-C-reactive protein ratio as a biomarker for predicting intestinal ischemia, imaging findings also offer critical predictive value. Specifically, narrowing of the hernia sac neck and lack of bowel wall enhancement on contrast-enhanced CT have been confirmed as independent risk factors for bowel resection in multiple retrospective studies [15, 16]. Sahin et al. [16]

further validated the independent predictive value of bowel obstruction signs for bowel resection. In a single-center preliminary study, Sakamoto et al. [17] suggested that the CT attenuation value of incarcerated bowel content could objectively and quantitatively distinguish necrosis from non-necrosis. Additionally, studies by Ceresoli et al. [18] and Dwertmann et al. [19] reported the independent discriminatory value of comorbidity burden—measured by the Charlson Comorbidity Index or ASA score—for postoperative complications and bowel resection, respectively.

While the preceding studies have identified valuable risk factors from multiple perspectives, two critical gaps persist in the existing literature. First, previous studies have often combined "intestinal ischemia" and "intestinal necrosis" in their analyses, treating them as a single, undifferentiated outcome, with bowel resection frequently serving as the endpoint [3, 8], while overlooking the possibility that the drivers may differ fundamentally during the pathophysiological transition from reversible vascular compromise to irreversible necrosis. Second, despite recent attempts to integrate clinical and imaging indicators into a single multivariable framework [16] and others have built predictive models for bowel resection based on combined clinical-laboratory variables [12] or focused solely on single CT features [15], the synergistic effects and differential weighting of inflammatory markers, imaging features, and clinical variables across distinct disease stages—mechanisms of ischemia and progression to necrosis—have not been adequately explored or quantified.

This study aimed to test the hypothesis that distinct factors drive the two stages of disease progression—from incarceration to ischemia and from ischemia to necrosis—and to develop corresponding sequential risk stratification models. The specific aims were: to identify independent risk factors for intestinal ischemia and for progression to necrosis; to determine their stage-specific distribution patterns; to develop and internally validate two sequential risk stratification models; and to explore a staged assessment strategy, ranging from initial screening for ischemia risk to identifying patients at high risk of necrosis.

Methods

Study design and ethics

This was a single-center, retrospective cohort study. The study protocol was reviewed and approved by the Ethics Committee of Jiangsu Province (Suqian) Hospital (approval number: 2026-SR-0116). Given the retrospective nature of the data analysis, the ethics committee waived the requirement for informed consent. The study adhered strictly to the principles of the Declaration of Helsinki, and all data were anonymized prior to analysis.

Study population

Inclusion criteria

(1) Patients aged ≥ 18 years who were consecutively admitted between January 2016 and December 2025 and underwent emergency surgery. (2) Acute irreducible groin hernia was clinically diagnosed if a groin mass was manually irreducible on physical examination, accompanied by acute-onset pain and/or swelling, and had a history of being reducible prior to the acute episode. The specific anatomical type (indirect, direct, or femoral) was determined intraoperative. (3) Additional criteria for inclusion were: (3) Preoperative ultrasound and intraoperative exploration confirming the presence of bowel within the hernia contents. (4) Complete preoperative key clinical data, with no missing values for any enrolled case. Key variables were defined as all demographic, clinical, laboratory, and imaging variables listed in Tables 1 and 5.

Exclusion criteria

(1) Easily reducible hernias successfully reduced manually and scheduled for elective surgery, or chronic irreducible hernias without acute symptom exacerbation. (2) Patients with missing key preoperative data or in whom preoperative ultrasound could not clearly determine the nature of the hernia contents. (3) Intraoperative exploration confirming a hernia that was not in the groin region, or hernia contents other than bowel (e.g., omentum, bladder, or adipose tissue). (4) Ambiguous intraoperative description of bowel viability precluding accurate classification. (5) History of previous ipsilateral groin hernia repair. (6) Acute abdomen and a concurrent condition, both requiring emergency surgery. (7) Pregnant or breastfeeding women. (8) Underlying diseases or medications that may affect complete blood count or inflammatory markers include: active malignancy within the past 5 years; glucocorticoid or immunosuppressant use within the past month; acute infection; active chronic inflammatory disease; or hematological disorders.

Grouping criteria and endpoint definitions

Using intraoperative findings as the gold standard, patients were assigned to the following two groups:

(1) Non-ischemia group: Intraoperative exploration confirmed the hernia contained bowel, which was pink and healthy-appearing, exhibited normal peristalsis, and showed good mesenteric vascular pulsation, with no signs of vascular compromise.

(2) Ischemia group: Intraoperative exploration confirmed bowel wall vascular compromise within the hernia contents; given its potential for reversibility, this group was further divided into two subgroups:

- - Simple ischemia subgroup (reversible): The bowel appeared dark red or cyanotic, with diminished peristalsis. However, after the incarceration was released and warm saline-soaked packs were applied, its color normalized within 10–15 minutes, peristalsis recovered, and bowel resection was ultimately unnecessary.
- - Intestinal necrosis subgroup (irreversible): During surgery, the bowel appeared black or dark purple and exhibited absent peristalsis, which did not improve after the maneuvers. The intraoperative

identification of frank perforation or necrosis necessitated segmental bowel resection. Postoperative pathological examination confirmed full-thickness ischemic necrosis of the bowel wall.

Endpoint event definitions

Primary analysis (full cohort): The dichotomous endpoint was "presence of intestinal ischemia (i.e., belonging to the ischemia group)."

Subgroup analysis (within the ischemia group): The simple ischemia subgroup was compared with the intestinal necrosis subgroup using the dichotomous endpoint of progression to intestinal necrosis (defined as requiring bowel resection).

These criteria were established to enhance the objectivity of group assignment; however, intraoperative assessment of bowel viability is acknowledged to rely, to some extent, on the operating surgeon's clinical experience. To mitigate subjective bias, the intestinal necrosis subgroup was subsequently validated through postoperative pathology reports.

Data collection

The following variables were obtained from the hospital information system: (1) Demographics: sex, age, body mass index (BMI). (2) Clinical parameters: symptom duration (time from patient- or family-reported appearance of the irreducible mass to emergency triage, recorded in hours), hernia sac fluid (presence or absence, based on preoperative ultrasound), bowel obstruction (presence or absence, defined as cessation of flatus and defecation with nausea and vomiting, confirmed by imaging or intraoperative findings), hernia type (indirect, direct, or femoral, according to intraoperative diagnosis), hernia sac neck diameter (mm, measured by preoperative ultrasound). (3) Laboratory parameters: complete blood count within 24 hours preoperatively, including white blood cell count, neutrophil count, lymphocyte count, monocyte count, hemoglobin, and platelet count. The NLR was calculated as neutrophil count/lymphocyte count. (4) Comorbidities: hypertension, diabetes mellitus, cardiovascular disease (coronary heart disease, myocardial infarction, heart failure, atrial fibrillation), and chronic obstructive pulmonary disease (COPD).

According to our center's emergency workflow, all patients underwent ultrasound examination; CT examinations were performed on a subset of patients at the discretion of the attending surgeon, based on clinical judgment. CT diagnostic conclusions did not serve as inclusion or exclusion criteria, nor were CT findings incorporated as variables in the study analysis. This study aimed to develop a risk assessment strategy utilizing exclusively bedside ultrasound and routine laboratory tests, thereby enhancing the model's applicability in emergency settings.

Statistical analysis

All statistical analyses were performed using R software (version 4.3.1, R Foundation for Statistical Computing, Vienna, Austria). Continuous variables were tested for normality. Normally distributed data are presented as mean $\pm$ standard deviation and compared using the independent-samples t-test, while non-normally distributed data are presented as median (interquartile range) and compared using the Mann–Whitney U test. Categorical data are presented as frequencies and percentages, with between-group differences assessed using the chi-squared test or Fisher's exact test. For continuous variables significant in univariate analysis, receiver operating characteristic (ROC) curves were constructed, and the area under the curve (AUC) was calculated. The optimal cutoff value was determined by maximizing Youden's index (sensitivity + specificity – 1), and the corresponding sensitivity, specificity, and Youden's index were calculated to assess their discriminatory ability for intestinal ischemia or necrosis.

Multivariable analysis was performed using logistic regression. Variables significant in univariate analysis and clinically important variables were initially included. Variance inflation factor (VIF) was computed to assess multicollinearity, and highly collinear variables were excluded. Stepwise selection was then used to identify independent risk factors. Regression coefficients (β), standard errors, odds ratios (OR), and 95% confidence intervals (CI) were calculated. Based on the independent risk factors identified in multivariable analysis, risk stratification models for intestinal ischemia and intestinal necrosis were constructed. Model performance was then evaluated: discrimination was assessed by the ROC curve and AUC (AUC confidence intervals were calculated using DeLong's method); calibration by calibration plots and the Hosmer–Lemeshow goodness-of-fit test; and internal validation by the bootstrap method (B = 1000) to calculate the corrected C-statistic. Clinical utility was evaluated based on decision curve analysis (DCA)[20], which indicated that DCA results based on single-center data carried a risk of optimism bias. All tests were two-sided, and $P < 0.05$ was considered statistically significant.

Results

Univariate analysis for intestinal ischemia

Table 1 compares baseline characteristics between the intestinal ischemia and non-ischemia groups. No statistically significant differences were identified between the groups concerning sex, hypertension, diabetes mellitus, or hernia type (all $P > 0.05$). The ischemia group was older (76.00 [66.00, 82.50] vs. 68.00 [55.50, 77.00] years, $P < 0.001$), had longer symptom duration (14.00 [7.50, 24.00] h vs. 6.50 [4.00, 14.00] h, $P < 0.001$), and had lower BMI ($P < 0.001$). The ischemia group exhibited significantly higher rates of cardiovascular disease and COPD compared to the non-ischemia group (25% vs. 5.6%, $P < 0.001$; 15% vs. 4%, $P = 0.009$, respectively).

Regarding hernia-related clinical features, the ischemia group exhibited a higher proportion of patients with hernia sac fluid and bowel obstruction (both $P < 0.001$), and a significantly smaller hernia sac neck diameter ($P < 0.001$). Laboratory tests revealed significantly elevated white blood cell count, neutrophil percentage, and absolute neutrophil count in the ischemia group (all $P < 0.001$). Conversely, lymphocyte

percentage and absolute lymphocyte count were significantly decreased (all $P < 0.001$). NLR was also significantly higher in the ischemia group (9.42 [4.91, 13.85] vs. 3.46 [2.38, 6.00], $P < 0.001$). No significant differences were observed between the two groups for hemoglobin and platelet levels (both $P > 0.05$) (Table 1).

Table 1
Baseline characteristics and univariate analysis of the intestinal ischemia group versus the non-ischemia group.

Variable	non-Ischemia(N = 124)	Ischemia(N = 100)	$\chi^2/t/z$	p
Sex			0.024	0.878
Female	37(29.8%)	28(28%)		
Male	87(70.2%)	72(72%)		
Age (years)	68.00(55.50, 77.00)	76.00(66.00, 82.50)	4.123	< 0.001
Duration (hours)	6.50(4.00, 14.00)	14.00(7.50, 24.00)	5.023	< 0.001
BMI (kg/m ²)	23.27(21.83, 25.51)	21.48(19.87, 24.22)	-4.095	< 0.001
Hypertension			0.565	0.452
No	97(78.2%)	73(73%)		
Yes	27(21.8%)	27(27%)		
Diabetes			0.77	0.38
No	116(93.5%)	97(97%)		
Yes	8(6.5%)	3(3%)		
CVD			15.392	< 0.001
No	117(94.4%)	75(75%)		
Yes	7(5.6%)	25(25%)		
COPD			6.896	0.009
No	119(96%)	85(85%)		
Yes	5(4%)	15(15%)		
Sac fluid			46.251	< 0.001
No	102(82.3%)	37(37%)		
Yes	22(17.7%)	63(63%)		
Obstruction			69.162	< 0.001
No	103(83.1%)	27(27%)		
Yes	21(16.9%)	73(73%)		
Neck diameter (mm)	12.00(10.00, 20.00)	10.00(9.50, 13.00)	-4.204	< 0.001
WBC ($\times 10^9/L$)	6.67(5.17, 8.87)	8.50(6.50, 10.82)	4.048	< 0.001

Variable	non-Ischemia(N = 124)	Ischemia(N = 100)	$\chi^2/t/z$	p
NE% (%)	71.90 ± 11.71	82.65 ± 8.13	6.928	< 0.001
LY% (%)	20.50(13.55, 27.10)	9.10(6.10, 14.90)	-7.321	< 0.001
NE (×10 ⁹ /L)	4.55(3.30, 7.07)	7.25(5.12, 9.80)	5.15	< 0.001
LY (×10 ⁹ /L)	1.25(0.90, 1.62)	0.81(0.59, 1.08)	-5.737	< 0.001
NLR	3.46(2.38, 6.00)	9.42(4.91, 13.85)	7.342	< 0.001
Hb (g/L)	140.00(128.00, 155.00)	139.00(126.50, 152.00)	0.753	0.535
PLT (×10 ⁹ /L)	206.50(166.00, 254.00)	215.50(171.50, 247.00)	0.544	0.587
Hernia type			0.346	0.557
Inguinal	94 (75.8%)	80 (80%)		
Femoral	30 (24.2%)	20 (20%)		

Discriminatory performance of continuous variables for intestinal ischemia

Table 2 details the discriminatory performance of continuous variables for intestinal ischemia, selected based on significant differences in univariate analysis and the exclusion of highly collinear indicators. NLR demonstrated the best discriminatory performance, with an AUC of 0.785 (95% CI 0.725–0.846), an optimal cutoff (Youden's index) of 6.496, sensitivity of 0.71, and specificity of 0.806. The AUC for symptom duration was 0.695 (95% CI 0.625–0.764), while the AUCs for age, BMI, and hernia sac neck diameter ranged from 0.659 to 0.661. These reported cutoff values were used solely for univariate performance assessment and were not incorporated into the multivariable model. Similarly, complete blood count-derived variables were excluded from the multivariable model due to their high collinearity with NLR (Table 2).

Table 2

Discriminatory performance of continuous variables for intestinal ischemia (univariate analysis).

Variable	AUC (95%CI)	Optimal Cutoff	Sensitivity	Specificity	Youden Index
Age (years)	0.66 (0.589 ~ 0.732)	73.5	0.58	0.661	0.241
Duration (hours)	0.695 (0.625 ~ 0.764)	10.5	0.63	0.694	0.324
BMI (kg/m ²)	0.659 (0.586 ~ 0.732)	21.847	0.57	0.75	0.32
Neck diameter(mm)	0.661 (0.591 ~ 0.731)	15.5	0.89	0.411	0.301
NLR	0.785 (0.725 ~ 0.846)	6.496	0.71	0.806	0.516

Multivariable analysis and risk stratification model for intestinal ischemia

Variables identified as significant in univariate analysis (excluding complete blood count parameters collinear with NLR) were subsequently entered into multivariable logistic regression. This analysis revealed that symptom duration, cardiovascular disease, bowel obstruction, hernia sac neck diameter, and NLR were independent risk factors for intestinal ischemia.

Table 3

Multivariable logistic regression analysis of independent risk factors for intestinal ischemia.

(Intercept)	β	Std. Error	z value	OR (95% CI)	p
	1.897	2.025	0.937	-	0.349
Age (years)	-0.008	0.016	-0.523	0.99 (0.96, 1.02)	0.601
Duration (hours)	0.043	0.015	2.953	1.04 (1.01, 1.07)	0.003
BMI (kg/m ²)	-0.105	0.068	-1.547	0.90 (0.79, 1.03)	0.122
CVD	1.645	0.658	2.498	5.18 (1.42, 18.83)	0.012
COPD	-0.018	0.74	-0.024	0.98 (0.23, 4.19)	0.981
Sac fluid	0.776	0.433	1.791	2.17 (0.93, 5.08)	0.073
Obstruction	2.249	0.453	4.961	9.48 (3.90, 23.04)	< 0.001
Neck diameter (mm)	-0.157	0.04	-3.972	0.85 (0.79, 0.92)	< 0.001
NLR	0.104	0.033	3.115	1.11 (1.04, 1.18)	0.002

Several factors were significantly associated with ischemia risk. An approximately 4% increase in risk was observed for each additional hour of symptom duration (OR = 1.04, 95% CI 1.01–1.07, P = 0.003). The presence of cardiovascular disease significantly increased the risk by over five-fold (OR = 5.18, 95% CI 1.42–18.83, P = 0.012), while bowel obstruction markedly elevated it by nearly ten-fold (OR = 9.48, 95% CI 3.90–23.04, P < 0.001). Furthermore, each unit increase in NLR was associated with an approximately 11% higher ischemia risk (OR = 1.11, 95% CI 1.04–1.18, P = 0.002). In contrast, each 1 mm increase in hernia sac neck diameter was associated with an approximately 15% reduction in ischemia risk (OR = 0.85, 95% CI 0.79–0.92, P < 0.001). After multivariable adjustment, age, BMI, COPD, and hernia sac fluid did not reach statistical significance (all P > 0.05) (Table 3). Finally, all included variables had VIF values < 5, indicating no serious multicollinearity (Table 4).

Table 4
Collinearity diagnostics for the multivariable logistic regression in the intestinal ischemia model.

Variable	VIF
Age (years)	1.280
Duration (hours)	1.116
BMI (kg/m ²)	1.086
CVD	1.222
COPD	1.067
Sac fluid	1.115
Obstruction	1.263
Neck diameter (mm)	1.265
NLR	1.061

The risk stratification model, built upon five independent risk factors, exhibited high discrimination with an AUC of 0.906 (95% CI 0.866–0.946, DeLong's method). At an optimal cutoff value of 0.407, the model achieved a sensitivity of 0.860, a specificity of 0.839, and an overall accuracy of 0.848. Calibration was also strong, evidenced by high agreement between predicted probabilities and observed risk on the calibration curve and a non-significant Hosmer–Lemeshow test ($\chi^2 = 4.462$, df = 8, P = 0.813). The C-statistic, corrected by 1000 bootstrap resamples, was 0.895, further confirming the model's good discrimination, calibration, and stability. Decision curve analysis indicated a positive net benefit in the internal data when the risk threshold exceeded 0.1, although external validation is required to confirm this finding (Figs. 1–3).

Univariate analysis for intestinal necrosis

Within the ischemia subgroup, Table 5 compares baseline characteristics between the intestinal necrosis and non-necrosis groups. No statistically significant differences were found in sex, age, symptom duration, BMI, diabetes mellitus, cardiovascular disease, or hernia type (all $P > 0.05$). However, the prevalence of hypertension was significantly higher in the necrosis group (38.2% vs. 13.3%, $P = 0.011$), and COPD showed a marginal difference ($P = 0.067$). Regarding hernia-related features, the necrosis group exhibited a significantly higher incidence of hernia sac fluid (89.1% vs. 31.1%, $P < 0.001$), a greater rate of bowel obstruction (85.5% vs. 57.8%, $P = 0.004$), and a significantly smaller hernia sac neck diameter (10.00 mm [9.00, 10.00] vs. 12.00 mm [10.00, 14.00], $P = 0.003$).

In terms of laboratory parameters, the necrosis group demonstrated an elevated neutrophil percentage ($P = 0.002$), decreased lymphocyte percentage ($P < 0.001$), and decreased absolute lymphocyte count ($P = 0.004$). The NLR was also significantly higher (10.81 [8.24, 15.40] vs. 6.66 [3.84, 11.39], $P < 0.001$). Additionally, hemoglobin levels were significantly lower in the necrosis group (133.89 ± 20.19 g/L vs. 143.96 ± 16.85 g/L, $P = 0.009$). Conversely, white blood cell count, absolute neutrophil count, and platelet count did not differ significantly between groups (all $P > 0.05$) (Table 5).

Table 5

Baseline characteristics and univariate analysis of the intestinal necrosis group versus the non-necrosis group (within the ischemia subgroup).

Variable	non-Necrosis(N = 45)	Necrosis(N = 55)	$\chi^2/t/z$	<i>p</i>
Sex			0.884	0.347
Female	10(22.2%)	18(32.7%)		
Male	35(77.8%)	37(67.3%)		
Age (years)	75.00(64.00, 83.00)	76.00(67.50, 82.00)	0.309	0.760
Duration (hours)	12.00(6.00, 24.00)	17.00(8.50, 42.00)	1.789	0.074
BMI (kg/m ²)	21.48(19.65, 24.22)	21.48(20.00, 24.13)	-0.014	0.992
Hypertension			6.544	0.011
No	39(86.7%)	34(61.8%)		
Yes	6(13.3%)	21(38.2%)		
Diabetes			0.000	1.000
No	44(97.8%)	53(96.4%)		
Yes	1(2.2%)	2(3.6%)		
CVD			0.121	0.728
No	35(77.8%)	40(72.7%)		
Yes	10(22.2%)	15(27.3%)		
COPD			3.347	0.067
No	42(93.3%)	43(78.2%)		
Yes	3(6.7%)	12(21.8%)		
Sac fluid			33.249	< 0.001
No	31(68.9%)	6(10.9%)		
Yes	14(31.1%)	49(89.1%)		
Obstruction			8.266	0.004
No	19(42.2%)	8(14.5%)		
Yes	26(57.8%)	47(85.5%)		
Neck diameter (mm)	12.00(10.00, 14.00)	10.00(9.00, 10.00)	-2.990	0.003
WBC ($\times 10^9/L$)	8.39(6.26, 10.53)	8.51(6.89, 12.02)	0.641	0.524

Variable	non-Necrosis(N = 45)	Necrosis(N = 55)	$\chi^2/t/z$	p
NE% (%)	79.80 ± 9.67	84.97 ± 5.71	2.519	0.002
LY% (%)	11.90(7.90, 19.50)	7.90(5.65, 10.15)	-3.697	< 0.001
NE (×10 ⁹ /L)	6.99(4.57, 8.92)	7.32(5.82, 10.62)	1.143	0.254
LY (×10 ⁹ /L)	0.93(0.69, 1.35)	0.80(0.43, 0.98)	-2.865	0.004
NLR	6.66(3.84, 11.39)	10.81(8.24, 15.40)	3.530	< 0.001
Hb (g/L)	143.96 ± 16.85	133.89 ± 20.19	2.718	0.009
PLT (×10 ⁹ /L)	215.00(161.00, 239.00)	218.00(178.00, 253.50)	0.863	0.39
Hernia type			0.063	0.802
Inguinal	37 (82.2%)	43 (78.2%)		
Femoral	8 (17.8%)	12 (21.8%)		

Discriminatory performance of continuous variables for intestinal necrosis

Within the ischemia subgroup, NLR demonstrated an AUC of 0.706 (95% CI 0.601–0.811) for discriminating necrosis. Its optimal cutoff (Youden's index) was 7.626, providing a sensitivity of 0.80 and a specificity of 0.622. For comparison, the AUCs were 0.668 (95% CI 0.561–0.776) for hernia sac neck diameter and 0.639 (95% CI 0.531–0.748) for hemoglobin. These cutoff values were used exclusively for univariate performance assessment and were not incorporated into the multivariable model. Additionally, complete blood count variables collinear with NLR were excluded from the multivariable model (Table 6).

Table 6

Discriminatory performance of continuous variables for intestinal necrosis (univariate analysis, ischemia subgroup).

Variable	AUC (95%CI)	Optimal Cutoff	Sensitivity	Specificity	Youden Index
Neck diameter (mm)	0.668 (0.561 ~ 0.776)	10.5	0.818	0.556	0.374
NLR	0.706 (0.601 ~ 0.811)	7.626	0.800	0.622	0.422
Hb (g/L)	0.639 (0.531 ~ 0.748)	138.5	0.582	0.644	0.226

Multivariable analysis and risk stratification model for intestinal necrosis

Variables significant in univariate analysis (hypertension, hernia sac fluid, bowel obstruction, hernia sac neck diameter, NLR, hemoglobin) were entered into multivariable logistic regression. This analysis identified hypertension, hernia sac fluid, and NLR as independent risk factors for intestinal necrosis.

Patients with hypertension showed a significantly increased risk of necrosis (OR = 5.06, 95% CI 1.22–20.91, $P = 0.025$). The presence of hernia sac fluid was associated with a substantially elevated risk (OR = 17.23, 95% CI 4.67–63.56, $P < 0.001$). Furthermore, each unit increase in NLR was linked to an approximately 13% rise in necrosis risk (OR = 1.13, 95% CI 1.01–1.25, $P = 0.027$). In contrast, bowel obstruction, hernia sac neck diameter, and hemoglobin did not reach statistical significance after multivariable adjustment (all $P > 0.05$) (Table 7). All variables included in the model had VIF values < 5 , indicating no serious multicollinearity (Table 8).

Table 7
Multivariable logistic regression analysis of independent risk factors for intestinal necrosis (ischemia subgroup).

(Intercept)	β	Std. Error	z value	OR (95% CI)	<i>p</i>
	1.997	2.501	0.799	-	0.425
Hypertension	1.621	0.724	2.239	5.06 (1.22, 20.91)	0.025
Sac fluid	2.847	0.666	4.274	17.23 (4.67, 63.56)	< 0.001
Obstruction	0.469	0.673	0.697	1.60 (0.43, 5.98)	0.486
Neck diameter (mm)	-0.104	0.07	-1.481	0.90 (0.78, 1.03)	0.138
NLR	0.118	0.054	2.205	1.13 (1.01, 1.25)	0.027
Hb (g/L)	-0.031	0.018	-1.774	0.97 (0.94, 1.00)	0.076

Table 8
Collinearity diagnostics for the multivariable logistic regression in the intestinal necrosis model.

Variable	VIF
Hypertension	1.130
Sac fluid	1.197
Obstruction	1.047
Neck diameter (mm)	1.054
NLR	1.091
Hb (g/L)	1.089

The necrosis risk stratification model, developed using three independent risk factors (hypertension, hernia sac fluid, and NLR), demonstrated an AUC of 0.883 (95% CI 0.815–0.951, DeLong's method). At the optimal cutoff of 0.643, the model achieved a sensitivity of 0.818, specificity of 0.867, and an accuracy of 0.84. The C-statistic, corrected by 1000 bootstrap resamples, was 0.873. For calibration, the curve demonstrated close agreement between predicted and observed risk, and the Hosmer–Lemeshow test yielded $\chi^2 = 10.19$, $df = 8$, $P = 0.252$. Decision curve analysis further indicated that the model's net benefit exceeded that of both "treat all" and "treat none" strategies when the risk threshold was > 0.1 . However, these findings require validation in external data (Figs. 4–6).

Discussion

This study analyzed a cohort of acute irreducible groin hernias, identifying distinct risk factor distributions for two sequential stages: progression from incarceration to intestinal ischemia, and subsequent progression from ischemia to intestinal necrosis. Based on these findings, two risk stratification models were developed. This supports our central hypothesis that the occurrence of intestinal ischemia and its progression to necrosis are driven by different sets of variables, rather than following a homogeneous, single continuum.

It is crucial to note that these two models are currently exploratory. Their significance lies in their use of multivariable analysis to reveal a differential pattern: mechanical factors predominantly drive the occurrence of ischemia, while patient-related factors and inflammatory status primarily drive the progression to necrosis. Consequently, these developed risk stratification models should be considered exploratory auxiliary reference tools and must not be directly applied to clinical decision-making until adequate external validation is obtained.

In the ischemia development stage, the duration of incarceration emerged as an independent risk factor for intestinal ischemia. Specifically, the study showed that each additional hour of incarceration increased the risk of intestinal ischemia by approximately 4% (OR = 1.04). This prolonged incarceration time suggests persistent vascular compromise of the bowel, leading to progressively worsening venous outflow obstruction and ultimately impaired arterial supply. A notable finding requiring further exploration is that incarceration time was statistically significant in the ischemia model ($P = 0.003$) but not in the necrosis model (univariate $P = 0.074$). The existing literature, furthermore, remains controversial regarding whether incarceration time is an independent risk factor for bowel resection. The EAST guidelines [21] based on a systematic review, conditionally recommend early intervention within 6 hours of incarceration to reduce the risk of bowel resection. A prospective observational investigation by Atila et al. [22] also confirmed that early intervention (< 6 hours) reduced the bowel resection rate (7.5% vs. 23.8%). Kurt et al. [23] reported a significant association between incarceration time exceeding 6 hours and the risk of bowel resection, and a larger-sample study by Zhou et al. [3] found that prolonged incarceration (especially > 6 hours) was an independent risk factor for bowel resection ($P = 0.034$). However, other studies found that while incarceration time was significant in univariate analysis, it was not statistically significant in multivariable analysis and was not an independent risk factor for bowel

resection [15, 24]. This discrepancy, which may be attributable to differences in study populations, sample sizes, and variable control, helps explain why some patients develop intestinal necrosis after only a short period of incarceration, while others with prolonged incarceration exhibit only simple ischemia.

In this study, hernia sac neck diameter emerged as another key structural parameter, with Multivariable analysis revealing that each 1 mm increase in its diameter was associated with an approximately 15% reduction in the risk of intestinal ischemia (OR = 0.85). Sahin et al. [16] demonstrated that a small hernia ring diameter was an independent predictor of bowel resection. A narrow hernia ring exerts sustained mechanical compression on its contents, first impeding venous return by compressing veins and then, as intrasac pressure rises, ultimately compromising arterial blood supply. Notably, among patients with established ischemia, this factor did not significantly predict progression to necrosis ($P = 0.138$). This difference implies that hernia sac neck diameter primarily exerts its effect during the development of ischemia. Once ischemia is established, however, secondary changes such as inflammation and fluid accumulation within the hernia sac may become dominant, masking the influence of the initial anatomical structure.

Preliminary findings from this study suggest that once the bowel wall reaches the ischemic threshold, the focus of assessment for conditions such as bowel obstruction should transition from merely mechanical obstruction to parameters that better reflect host responsiveness and tissue tolerance. Univariate analysis in this study revealed a significant association between bowel obstruction and the risk of intestinal necrosis ($P < 0.001$), consistent with existing literature. Furthermore, a meta-analysis by Chen et al. identified bowel obstruction as a significant risk factor for bowel resection (OR = 3.67) [8]. Xie et al. reported that 86.1% of patients in the bowel resection group exhibited signs of bowel obstruction [10], and Using multivariable analysis, Yilmaz et al. confirmed that bowel obstruction is an independent risk factor for bowel resection in patients with incarcerated hernia (OR = 54.92, $P < 0.001$) [25]. In our necrosis multivariable model, bowel obstruction did not retain independent significance. This discrepancy may be attributed to several factors, including heterogeneity among study populations. Yilmaz et al. [25] studied a population that included ventral hernias such as incisional and umbilical hernias, whereas our study and Xie et al. [10] focused on incarcerated groin hernias. The independent effect of bowel obstruction within the model may be influenced by variations in anatomical location and the composition of hernia sac contents. Although Yilmaz et al. [25] adjusted their multivariable model to account for multiple markers, including NLR, CRP, lactate, and LDH, the effect size of bowel obstruction remained substantial (OR > 50). The inclusion of these inflammatory markers did not attenuate its independence, suggesting that, in that population, bowel obstruction itself might represent a more severe incarceration status or a longer disease course.

From a pathophysiological perspective, acute irreducible hernia causing bowel obstruction leads to increased intraluminal pressure and progressively aggravates venous outflow obstruction, thereby creating a strong association between bowel obstruction and the occurrence of ischemia. Therefore, bowel obstruction may serve as a more pronounced warning sign in the early stage of the disease.

However, once the bowel wall reaches irreversible ischemia, the evaluation focus must shift from mechanical factors to the tissue's intrinsic reactivity and tolerance. At this critical juncture, a comprehensive assessment incorporating indicators such as hernia sac fluid characteristics, NLR, and lactate – which reflect tissue injury and systemic status – becomes necessary, rather than relying solely on the single sign of bowel obstruction.

Hernia sac fluid demonstrated significant discriminatory performance in the necrosis model (OR = 17.23). Zhou and Yuan [26] identified hernia sac fluid as an independent predictor of intestinal necrosis. Loftus et al. [27] noted hernia sac fluid as an independent predictor of surgical site infection after incarcerated hernia repair. The present study extends this finding, demonstrating its utility for preoperative necrosis risk stratification. These findings suggest that hernia sac fluid is not merely a passive marker of ischemia but may play an active role in the necrotic cascade, serving as a microenvironment for bacterial translocation, endotoxin enrichment, and local pro-inflammatory cytokine accumulation. The loss of independent significance of bowel obstruction in the necrosis multivariable model further supports the hypothesis that the severity signal associated with bowel obstruction might be largely accounted for by hernia sac fluid and NLR. Collectively, these findings confirm that in assessing necrosis progression, the focus should shift from purely mechanical signs toward composite markers that better capture inflammation-immune imbalance.

NLR was the only independent risk factor present in both stages and warrants in-depth discussion. Zahorec [9] first elucidated its pathophysiological basis as an indicator of systemic inflammatory stress in critical illness. Subsequently, Xie et al. [10] and Köksal et al. [11] validated the role of NLR in predicting the severity of incarcerated hernia and the need for bowel resection in their respective single-center cohorts. This study not only corroborates these findings but also, employing a two-stage stratified design, reveals the consistent nature of this effect, characterized by stable and directionally consistent effect sizes across both stages: OR = 1.11 (95% CI 1.04–1.18) in the ischemia stage and OR = 1.13 (95% CI 1.01–1.25) in the necrosis stage. Yilmaz et al. [25] compared the predictive performance of various blood markers for bowel resection in incarcerated hernia. They observed significantly elevated NLR levels in the bowel resection group ($P < 0.001$), confirming its value in risk stratification. While a single preoperative NLR measurement demonstrated independent predictive value, the incremental benefit of serial perioperative NLR monitoring requires further evaluation in prospective studies.

Regarding patient-related factors, this study identified cardiovascular disease as a strong predictor of intestinal ischemia (OR = 5.18). This association likely arises because patients with cardiovascular disease have reduced baseline cardiac output reserve. This vulnerability makes them more susceptible to intestinal hypoperfusion under the stress of incarceration, thus accelerating the development of ischemia. Bala et al. [7], in their guidelines for acute mesenteric ischemia, corroborate this mechanism by noting that decreased cardiac output and hypovolemia are core drivers of intestinal hypoperfusion. Although cardiovascular disease did not retain independent significance in the necrosis model, this finding clinically suggests that patients with cardiovascular disease who develop an acute irreducible

hernia warrant early consideration for intestinal ischemia, even in the absence of pronounced systemic symptoms.

A notable, stage-specific effect of hypertension was also observed: it was not significant in the ischemia model but became an independent risk factor in the necrosis model. This finding is consistent with Dwertmann's et al. [19] observations of increased ASA scores and comorbidity burden and the comorbidity–mortality association pathway identified by Ceresoli et al. [18] observed an association between comorbidities and adverse outcomes in elderly patients undergoing emergency hernia repair. The present study further refines this association by identifying hypertension as a measurable single indicator. While the underlying mechanism requires further elucidation, it is plausible that, given the known microcirculatory pathophysiological effects of hypertension, this comorbidity facilitates progression to necrosis by lowering the tissue's ischemic tolerance threshold.

Based on these findings, this study delineates a "two-stage" risk evolution pattern. In the first stage, from incarceration to ischemia, risk is influenced by symptom duration, mechanical factors (such as hernia sac neck diameter), cardiovascular disease, bowel obstruction, and NLR; bowel obstruction exhibits the strongest effect among these. In the second stage, from ischemia to necrosis, biological variables like inflammatory status (NLR, hernia sac fluid) and patient-related factors (hypertension) become increasingly significant. Our findings complement the multivariate prediction model established by Chen et al. [13], which utilized the systemic immune-inflammation index as a novel indicator to predict the overall probability of necrosis. In contrast, our study specifies the differential distribution of core drivers across distinct stages, potentially offering insights for stage-specific interventions.

This study demonstrates potential for clinical translation. Our findings can be summarized as a tiered auxiliary tool, comprising low-cost and readily accessible indicators such as NLR, history of hypertension, bowel obstruction signs, and hernia sac fluid. This tool is particularly well-suited for dissemination within tiered healthcare delivery models. Based on these insights, we propose three exploratory recommendations: First, during emergency triage, patients presenting with prolonged symptom duration and imaging evidence of a narrow hernia sac neck might be prioritized for surgical exploration, even before inflammatory markers significantly rise. Second, if intestinal ischemia is identified intraoperatively, the presence of hernia sac fluid and a history of hypertension, combined with the NLR level, could serve as reference information for assessing necrosis risk. Third, looking ahead, should these models undergo adequate external validation within regional medical consortia, these variables could be integrated into emergency information systems. This would establish a standardized sequential assessment module, thereby reducing inter-hospital decision-making variability. However, it is crucial to emphasize that until these models are adequately externally validated, supported by prospective study evidence, and demonstrate clinical net benefit through decision curve analysis, they must be strictly regarded as exploratory auxiliary reference tools.

As a single-center retrospective investigation, this study has several limitations. First, due to its retrospective design, we could only identify an independent association between hypertension and

necrosis progression, not establish causality. Second, C-reactive protein (CRP), a key inflammatory marker, was not routinely tested in the emergency setting, resulting in a high rate of missing data. Consequently, CRP could not be included in our model, precluding direct comparison with indicators like the lymphocyte-to-C-reactive protein ratio proposed by Yildirim et al. [14]. Third, selection bias may have been introduced because only a subset of patients underwent CT examination, and the relevant imaging variables were not incorporated into the model analysis. Fourth, as a single-center study, the qualitative assessment of hernia sac fluid via preoperative ultrasound, lacking standardized quantitative criteria, may introduce measurement bias and limit model generalizability. Fifth, with only a limited number of events, the necrosis subgroup barely met the minimum requirements for multivariable analysis, thereby restricting the exploration of non-linear relationships and variable interactions. Sixth, focusing on preoperative risk stratification, the study did not incorporate outcome information such as the type of hernia repair material or postoperative complications. As a result, the longitudinal association between stratification results and postoperative outcomes could not be investigated.

Conclusions

This single-center study investigated the pathophysiological progression from intestinal ischemia to necrosis in acute irreducible hernia, identifying distinct risk factors for each stage and developing two sequential risk stratification models. Our findings reveal that risk factors for progression from intestinal ischemia to necrosis in acute irreducible groin hernia are stage-specific. Specifically, symptom duration, cardiovascular disease, bowel obstruction, hernia sac neck diameter, and NLR were independently associated with the initial occurrence of intestinal ischemia. For progression from ischemia to necrosis, independent risk factors included hypertension, hernia sac fluid, and NLR. Notably, NLR emerged as a pan-stage independent risk factor, its effect evident across both stages. Hernia sac fluid was the single variable with the greatest impact in the necrosis stage, while hypertension displayed a stage-specific, lagged effect. These three factors—NLR, hernia sac fluid, and hypertension—are priority targets for subsequent validation and mechanistic studies. However, due to the single-center, retrospective design and the limited sample size within the ischemia subgroup, these models are exploratory auxiliary reference tools. They require adequate external validation before direct application in clinical decision-making.

Abbreviations

AUC

Area under the receiver operating characteristic curve

BMI

Body mass index

CI

Confidence interval

CVD

Cardiovascular Disease

COPD
Chronic obstructive pulmonary disease
CT
Computed tomography
DCA
Decision curve analysis
HB
Hemoglobin
LY
Lymphocyte
NE
Neutrophil
NLR
Neutrophil-to-lymphocyte ratio
OR
Odds ratio
PLT
Platelet
ROC
Receiver operating characteristic
VIF
Variance inflation factor
WBC
White Blood Cell

Declarations

Ethics approval and consent to participate

This study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of Jiangsu Province (Suqian) Hospital (approval number: 2026-SR-0116). The requirement for informed consent was waived by the Ethics Committee of Jiangsu Province (Suqian) Hospital due to the retrospective and non-interventional nature of the study.

Consent for publication

Not applicable.

Availability of data and materials

The datasets used and/or analyzed 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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Authors' contributions

Weihuan Luo and Shiping Wang conceived and designed the study. Weihuan Luo and Xiaolan Zhang collected and organized the data. Cheng Zhang performed data verification and statistical analysis. Weihuan Luo drafted the manuscript. Yu Chen and Shiping Wang critically reviewed and revised the manuscript for important intellectual content. All authors read and approved the final version.

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Figures

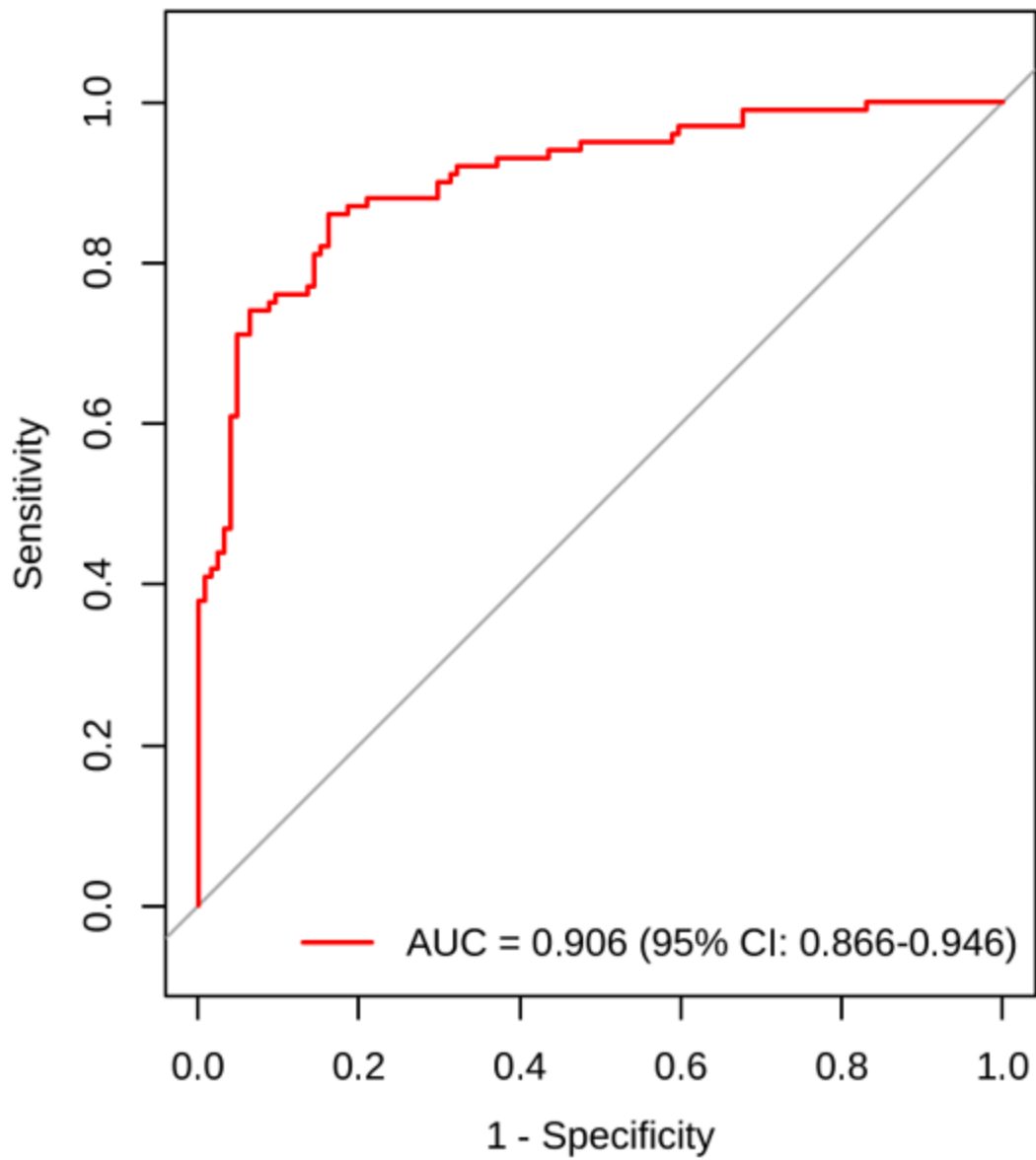


Figure 1

Receiver operating characteristic (ROC) curve of the intestinal ischemia risk stratification model.

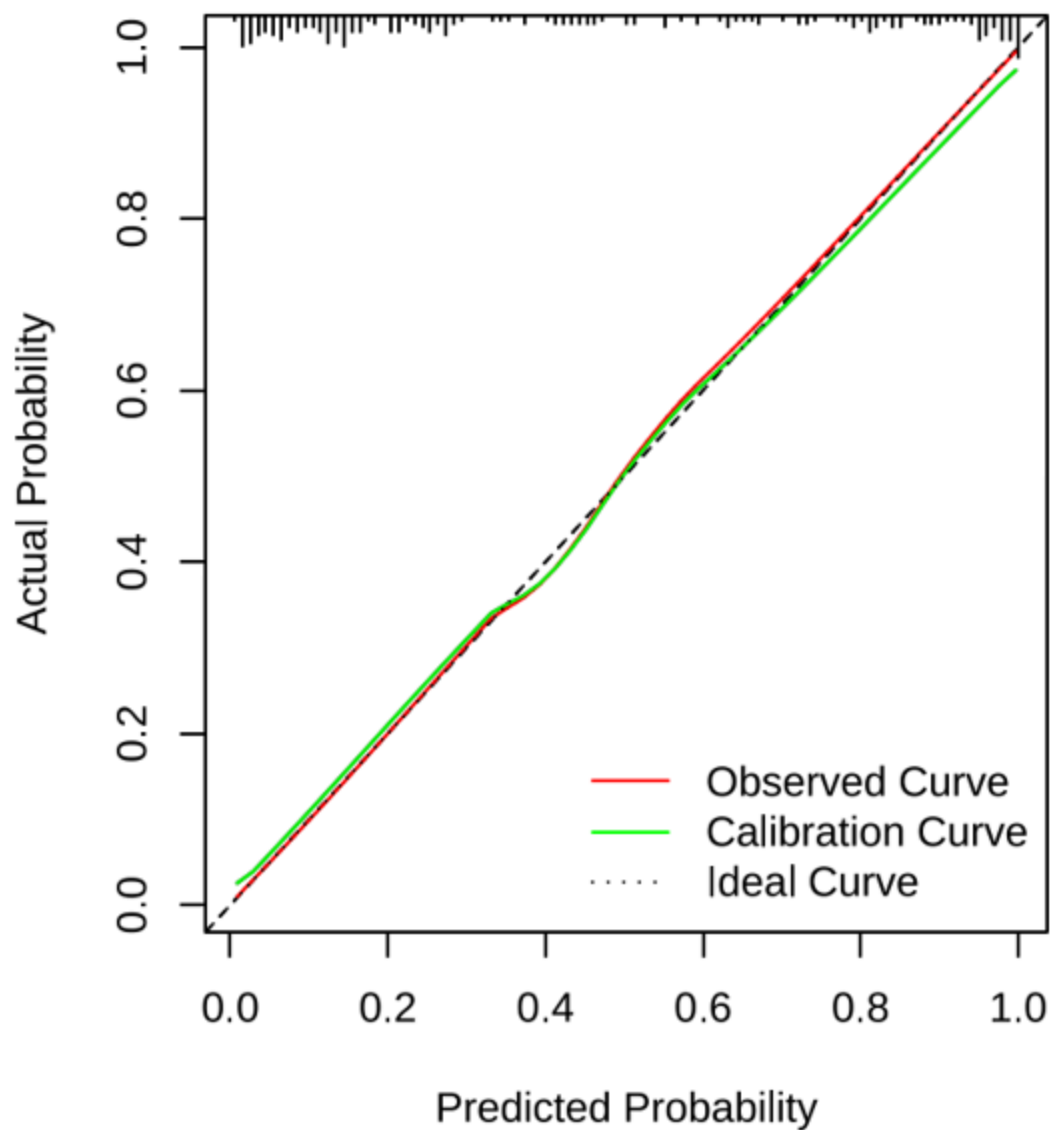


Figure 2

Calibration curve and bootstrap internal validation results of the intestinal ischemia risk stratification model.

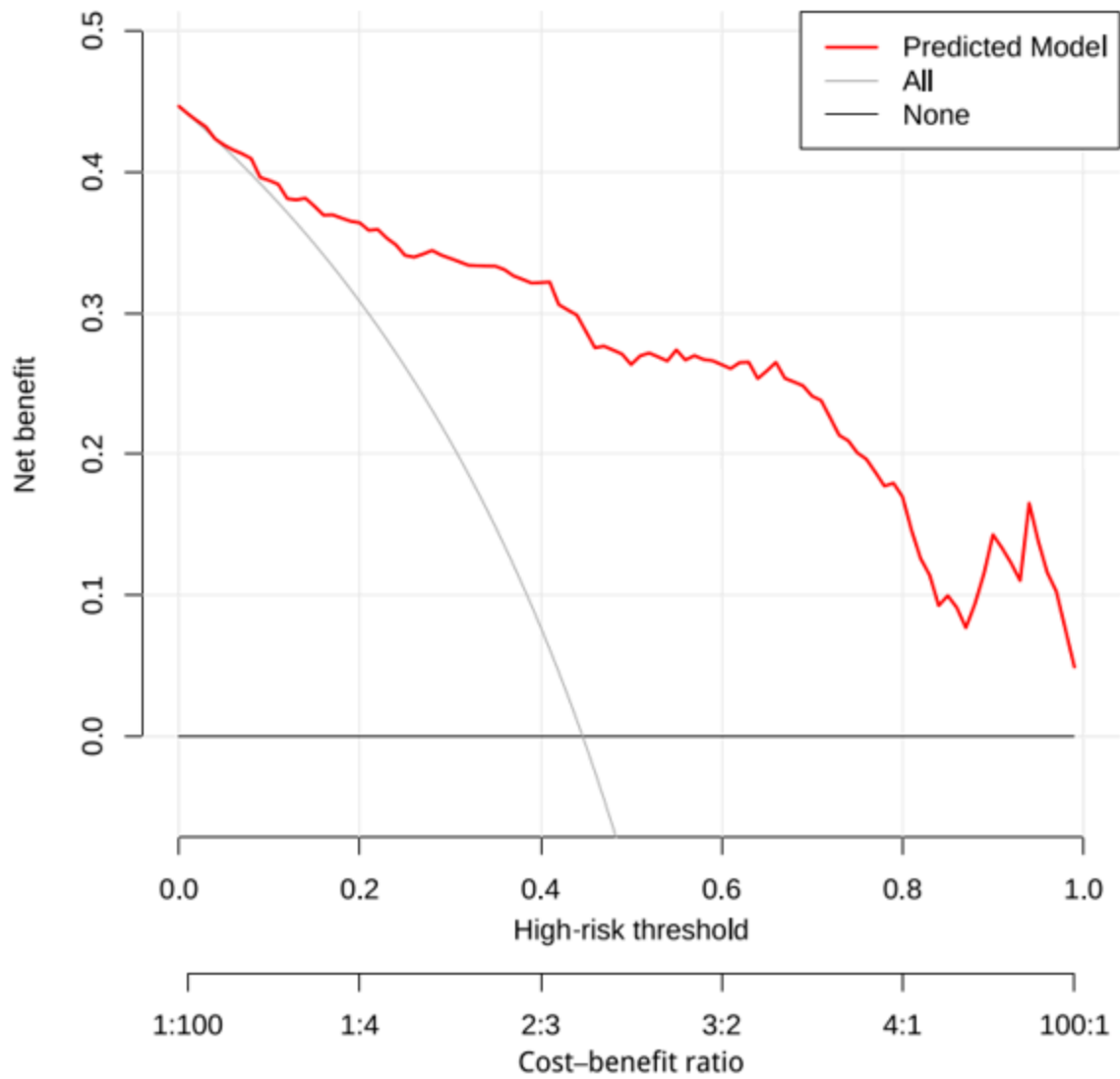


Figure 3

Decision curve analysis of the intestinal ischemia risk stratification model.

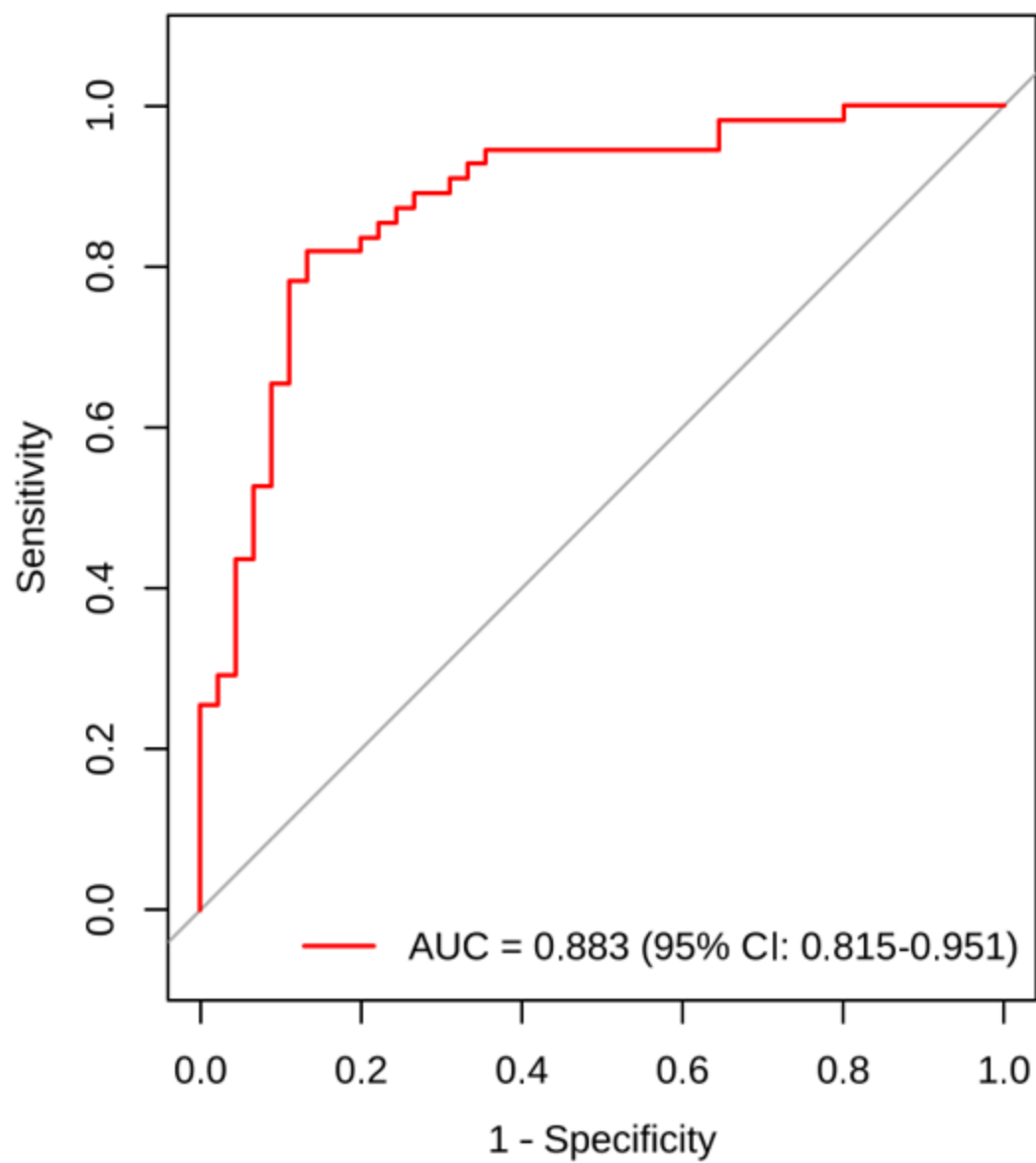


Figure 4

Receiver operating characteristic (ROC) curve of the intestinal necrosis risk stratification model.

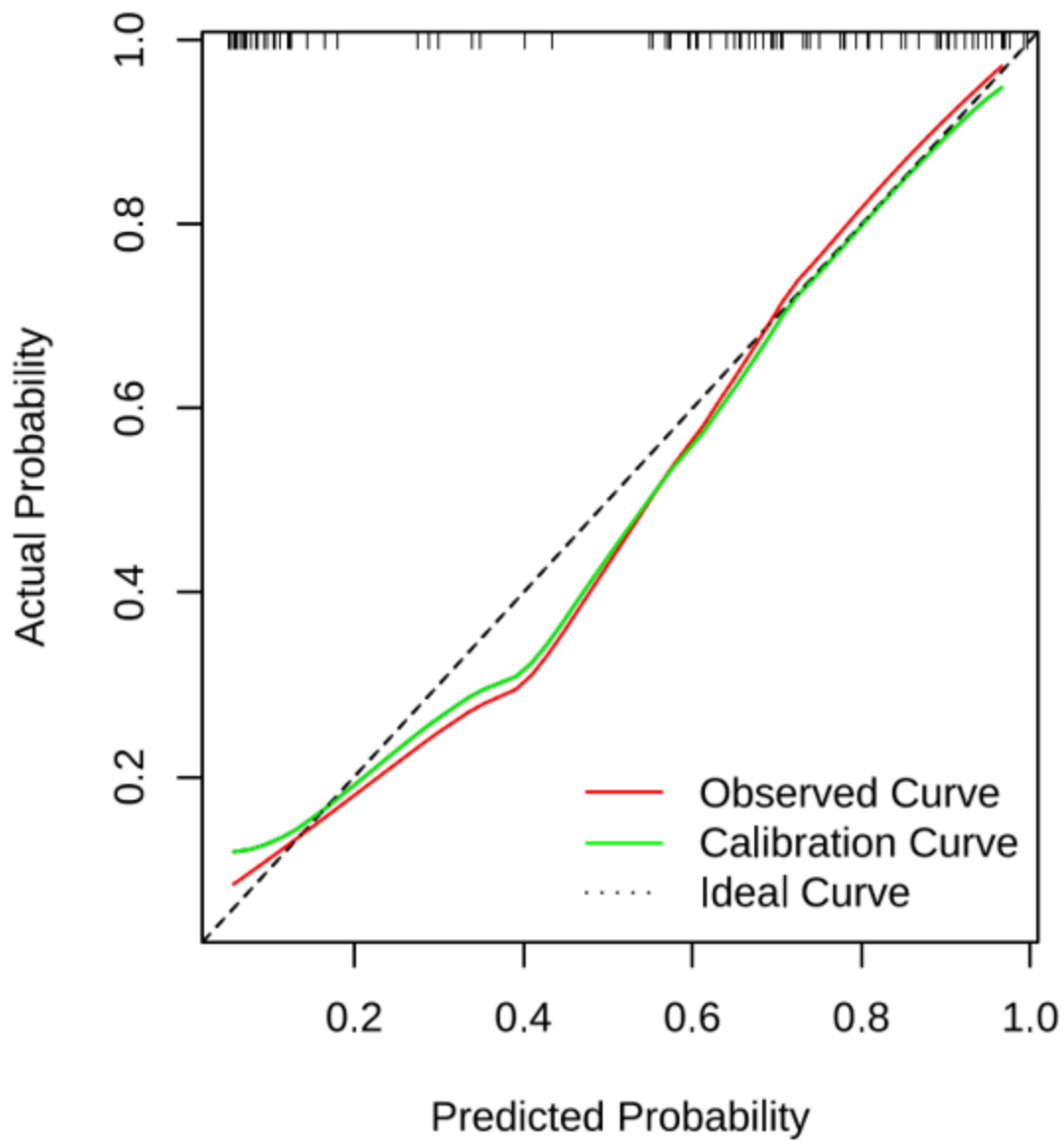


Figure 5

Calibration curve and bootstrap internal validation results of the intestinal necrosis risk stratification model.

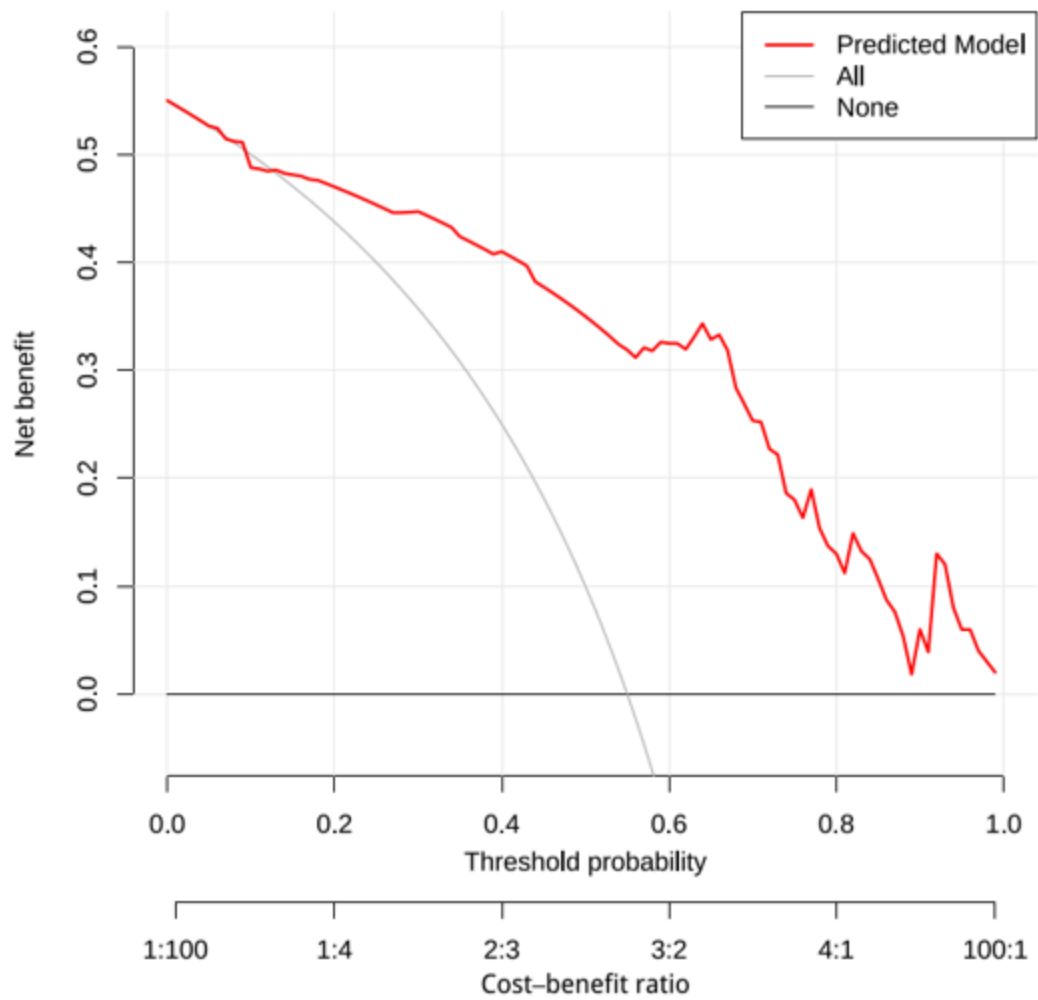


Figure 6

Decision curve analysis of the intestinal necrosis risk stratification model.