Predictive Value of the Systemic Immune-Inflammation Index Combined With the Prognostic Nutritional Index for Flap-Related Complications Following Flap Repair in Patients With Stage III and IV Pressure Injuries

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AIM: To investigate the combined predictive value of systemic immune-inflammation index (SII) and prognostic nutritional index (PNI) for postoperative flap-related complications in patients with stage III and IV pressure injuries undergoing flap repair.

METHODS: A single-center retrospective cohort study was conducted. A total of 242 patients with stage III and IV pressure injuries who underwent flap repair between January 2020 and December 2024 were enrolled. Based on postoperative flap-related complications, patients were divided into a non-complication group (n = 181) and a complication group (n = 61). Preoperative clinical data and laboratory parameters were collected, and SII and PNI were calculated. Independent risk factors were identified using univariate and multivariate logistic regression analyses, and a nomogram prediction model was subsequently developed. Receiver operating characteristic (ROC) and calibration curves were used to evaluate the predictive performance of the model.

RESULTS: Multivariate analysis demonstrated that PNI (odds ratio (OR) = 0.851, 95% CI: 0.793–0.913), SII (OR = 1.307, 95% CI: 1.143–1.494), and intraoperative blood loss (OR = 2.150, 95% CI: 1.141–4.052) were independent predictors of postoperative flap-related complications. The area under the curve (AUC) of the prediction model based on these three variables was 0.88 (95% CI: 0.83–0.93). The calibration curve demonstrated good agreement between predicted and observed outcomes (Hosmer-Lemeshow test, $p = 0.335$).

CONCLUSIONS: Elevated preoperative SII, reduced preoperative PNI, and increased intraoperative blood loss are independent risk factors for flap-related complications following flap repair in patients with stage III and IV pressure injuries. The prediction model based on these three factors demonstrates good discrimination and calibration, which may facilitate perioperative risk assessment and provide a basis for individualized clinical interventions.

Keywords: pressure injury; flap repair; systemic immune-inflammation index; prognostic nutritional index; intraoperative blood loss

Introduction

Pressure injury, also known as pressure ulcers, is a localized tissue injury characterized by ulceration and necrosis resulting from prolonged pressure that impairs blood circulation and disrupts local tissue nutrition. Pressure injuries are prone to occur at bony prominences that are easily compressed and have a thin muscle layer, such as the sacrococcygeal region and the greater trochanter of the femur, or at sites subjected to pressure with little or no muscle coverage or adipose tissue protection, such as the limbs and trunk [1]. The occurrence of pressure injuries not only causes physical and psychological suffering to patients but also imposes a heavy burden on their families.

Pressure injuries can be classified into four stages according to severity. Among these, stage I and II pressure injuries can generally be managed conservatively. In contrast, stage III and IV pressure injuries may be accompanied by sinus tracts, dead space formation, and even bone exposure, making them difficult to heal and prone to severe infection and potential life-threatening consequences. Stage III and IV pressure injuries have long represented a clinical challenge that requires effective management strategies [2]. Flap repair is an effective treatment approach for stage III and IV pressure injuries [3]. However, the postoperative recovery process remains uncertain, and some patients may develop complications such as flap-edge dehiscence, hematoma, infection, or even partial or complete flap necrosis. Patients who develop complications often require prolonged secondary debridement, repeat flap repair, or even more complex reconstructive procedures, resulting in extended hospital stay and increased healthcare costs. Recurrent infections may further worsen systemic conditions [4]. Therefore, accurately identifying high-risk patients before surgery and implementing targeted interventions through optimized perioperative management are critical for improving the surgical outcomes of pressure injuries and improving patient prognosis.

Postoperative complications following flap repair are influenced by multiple factors, among which nutritional status

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and inflammatory response are considered two core pathophysiological determinants. Malnutrition can impair tissue repair and wound-healing capacity [5,6,7], whereas excessive or uncontrolled inflammatory responses may result in microcirculatory dysfunction and tissue damage [8,9]. Although traditional single nutritional or inflammatory indices have some clinical value, their predictive performance is often limited by multiple confounding factors, resulting in low sensitivity and specificity. In recent years, composite inflammation-nutrition indicators derived from routine laboratory tests have demonstrated considerable value in predicting a variety of surgical complications, among which the prognostic nutritional index (PNI) and systemic immune-inflammation index (SII) have received extensive attention [10,11].

PNI is calculated based on serum albumin concentration and peripheral blood lymphocyte count. Previous studies have shown that PNI is a useful tool for assessing the risk of complications in patients with malignant tumors. In head and neck flap repair, low preoperative PNI has also been identified as an independent risk factor for postoperative flap-related complications [5,6,12]. SII is calculated from three key peripheral blood cell components, including neutrophils, platelets, and lymphocytes. An elevated SII indicates a state of "high inflammation and low immunity", characterized by neutrophil and platelet activation together with relative depletion of lymphocytes. This state has been closely associated with tumor progression, the severity of infectious diseases, and various postoperative flap-related complications [13,14].

To date, no study has investigated the combined predictive value of SII and PNI for flap-related complications following flap repair in patients with pressure injuries. Therefore, this study aimed to systematically evaluate the predictive efficacy of SII and PNI for postoperative flap-related complications in patients with pressure injuries through a retrospective cohort analysis. This study adopted a retrospective cohort design and focused on patients with pressure injuries who underwent flap repair. Univariate and multivariate logistic regression analyses were performed to identify independent factors associated with postoperative complications. Based on the identified independent predictors, a nomogram prediction model was developed to provide a visual tool for individualized risk assessment. The findings of this study are expected to provide an objective basis for the implementation of individualized perioperative interventions, thereby improving surgical outcomes.

Methods

Study Design and Subjects

This was a single-center retrospective study. Clinical data of patients with stage III and IV pressure injuries who underwent flap surgery at Hangzhou First People's Hospital Chengbei Campus between January 2020 and December 2024, were collected consecutively from the electronic medical record system. The study protocol strictly adhered to the principles of the Declaration of Helsinki and was approved by the Ethics Committee of Hangzhou First People's Hospital (approval number: 2026ZN096-1). All clinical data were obtained from the electronic medical record system, and data collection and analysis were performed after anonymization to ensure patient confidentiality. Therefore, the requirement for informed consent was waived.

The inclusion criteria were as follows: (1) stage III or IV pressure injury; and (2) flap surgery performed for pressure injury repair. The exclusion criteria were as follows: (1) requirement for intensive care after surgery; (2) repeat flap repair at the same anatomical site; and (3) substantial missing clinical data. Ultimately, 242 patients met the eligibility criteria and were included in the analysis. According to postoperative outcomes, patients were divided into a non-complication group (n = 181) and a complication group (n = 61).

Outcome Definition and Data Collection

The primary outcome was defined as major flap-related complications requiring reoperation, including any of the following: significant wound dehiscence, hematoma, flap infection, or flap necrosis that could not be resolved through conservative wound management and therefore required surgical intervention. Patient data were collected through a review of medical records and included the following: (1) demographic characteristics: age and gender; (2) pressure injury characteristics: location, stage, and presence of bone exposure; (3) comorbidities: diabetes mellitus and osteomyelitis (confirmed by preoperative imaging or intraoperative bone biopsy); (4) surgical variables: intraoperative blood loss (mL) and flap type (local flap or myocutaneous flap); and (5) laboratory indicators obtained within 24 hours before surgery: serum albumin (g/L), C-reactive protein (CRP, mg/L), white blood cell count (WBC, $\times 10^9$/L), neutrophil count, lymphocyte count, and platelet count ($\times 10^9$/L).

The indices were calculated using the following formulas:

PNI = serum albumin (g/L) + 5 × lymphocyte count ($\times 10^9$/L).

SII = (neutrophil count × platelet count) / lymphocyte count.

Statistical Analysis

All statistical analyses were performed using SPSS (version 26.0, IBM Corp., Armonk, NY, USA), and selected analyses and visualizations were completed using R (version 4.2.2, R Foundation for Statistical Computing, Vienna, Austria). The Shapiro-Wilk test was used to assess the normality of continuous variables. Variables with a normal distribution were expressed as mean ± standard deviation (SD), and comparisons between groups were performed using the independent-samples *t*-test. Variables with a non-normal distribution were expressed as median (interquartile range) [M (Q_1, Q_3)] and compared using the Mann-Whitney

U test. Categorical variables were expressed as the number of cases (percentages) [n (%)] and analyzed using the chi-square test or Fisher's exact test, as appropriate.

Variable selection for univariate analysis was guided by clinical relevance and prior evidence rather than by automatic inclusion of all available variables. Variables with $p < 0.05$ in the univariate analysis were subsequently included in the multivariate logistic regression model using backward stepwise selection to identify independent risk factors for postoperative flap-related complications. The results were expressed as odds ratios (ORs) with 95% confidence intervals (CIs).

Variance inflation factors (VIFs) were calculated to assess multicollinearity among continuous variables included in the final multivariate model. A nomogram was constructed based on the final multivariate model to provide a visual tool for individualized risk prediction. Model discrimination was evaluated by constructing a receiver operating characteristic (ROC) curve and calculating the area under the curve (AUC). Internal validation was performed using the bootstrap method with 1000 resamples, and a calibration curve was generated to evaluate the agreement between predicted probabilities and observed outcomes. The Hosmer-Lemeshow goodness-of-fit test was used to evaluate model calibration. A two-sided p-value < 0.05 was considered statistically significant.

Results

Baseline Characteristics of Patients

A total of 242 patients who met the eligibility criteria were included in this study. Among them, 61 patients developed flap-related complications, whereas 181 patients experienced no complications. The demographic and clinicopathological characteristics of the two groups are summarized in Table 1. Comparative analysis between the two groups showed significant differences in intraoperative blood loss, neutrophil count, platelet count, serum albumin level, lymphocyte count, CRP level, PNI, and SII ($p < 0.05$). However, no significant differences were observed in age, WBC count, gender, history of diabetes mellitus, pressure injury location, pressure injury stage, osteomyelitis, bone exposure, or flap type between the two groups (all $p > 0.05$).

Univariate Logistic Regression Analysis of Factors Associated With Postoperative Flap-Related Complications

Univariate logistic regression analysis demonstrated (Table 2) that intraoperative blood loss (odds ratio (OR) = 2.620, 95% CI: 1.567–4.379), CRP (OR = 1.018, 95% CI: 1.005–1.031), neutrophil count (OR = 1.758, 95% CI: 1.445–2.140), platelet count (OR = 1.020, 95% CI: 1.013–1.028), lymphocyte count (OR = 0.459, 95% CI: 0.278–0.760), SII (OR = 1.527, 95% CI: 1.350–1.727), serum albumin (OR = 0.770, 95% CI: 0.714–0.830), and PNI (OR = 0.797, 95% CI: 0.747–0.851) were significantly associated with postoperative flap-related complications (all $p < 0.05$).

Multivariate Logistic Regression Analysis of Factors Associated With Postoperative Flap-Related Complications

Variables with $p < 0.05$ in the univariate analysis were included in the multivariate logistic regression model. However, because neutrophil count, platelet count, lymphocyte counts, and serum albumin level are constituent components of SII and PNI, simultaneous inclusion of these variables would result in mathematical redundancy. Based on clinical relevance and prior evidence supporting the use of composite indices, only SII, PNI, and intraoperative blood loss were entered into the final multivariate model.

Multicollinearity diagnostics were performed for these three variables. The results showed that the VIF values of PNI, SII, and intraoperative blood loss were 1.379, 1.363, and 1.034, respectively. All VIF values were well below 5, indicating no significant multicollinearity among the variables. The results of the multivariate analysis demonstrated (Table 3) that PNI (OR = 0.851, 95% CI: 0.793–0.913), SII (OR = 1.307, 95% CI: 1.143–1.494), and intraoperative blood loss (OR = 2.150, 95% CI: 1.141–4.052) were independent factors associated with postoperative flap-related complications (all $p < 0.05$). Among these variables, PNI acted as a protective factor, whereas elevated SII and increased intraoperative blood loss were identified as risk factors for postoperative flap-related complications.

Nomogram for Predicting Postoperative Flap-Related Complications

Based on the independent predictors identified above, a logistic regression model was established to predict postoperative flap-related complications, and a visual nomogram was subsequently constructed (Fig. 1). Using this nomogram, clinicians can estimate the corresponding total score according to the intraoperative blood loss, SII, and PNI of patients, and then intuitively evaluate the individualized probability of postoperative flap-related complications.

Evaluation of the Predictive Performance of the Model

A prediction model for postoperative flap-related complications was constructed based on the three independent risk factors described above, and the ROC curve was generated (Fig. 2). The area under the curve (AUC) was 0.88 (95% CI: 0.83–0.93), indicating that the model exhibited good discriminatory capacity.

Calibration of the Prediction Model

As shown in Fig. 3, the calibration curve of the model was generated using the Bootstrap method with 1000 resamples. The bias-corrected calibration curve closely approximated the ideal reference line. The excellent calibration performance of the model was further supported by the

Table 1. Baseline characteristics of patients.

Variables	Overall cohort (n = 242)	No complication (n = 181)	Complication (n = 61)	Test statistic	*p*-value
Intraoperative blood loss (mL), mean ± SD	224.769 ± 61.990	216.027 ± 62.607	250.711 ± 52.547	$t = -3.888$	<0.001
Neutrophil count ($\times 10^9$/L), mean ± SD	6.743 ± 1.910	6.302 ± 1.623	8.052 ± 2.103	$t = -5.933$	<0.001
Platelet count ($\times 10^9$/L), mean ± SD	220.178 ± 52.448	208.669 ± 50.970	254.328 ± 40.866	$t = -6.340$	<0.001
Serum albumin (g/L), mean ± SD	35.427 ± 6.409	37.341 ± 5.582	29.747 ± 5.261	$t = 9.320$	<0.001
Age (years), mean ± SD	65.136 ± 7.234	64.785 ± 7.247	66.180 ± 7.150	$t = -1.305$	0.193
CRP (mg/L), M (Q_1, Q_3)	21.200 (16.700, 30.275)	20.400 (15.800, 28.600)	25.500 (18.800, 34.100)	$Z = -3.355$	<0.001
WBC count ($\times 10^9$/L), M (Q_1, Q_3)	9.840 (8.450, 13.695)	9.860 (8.390, 13.330)	9.780 (8.650, 15.160)	$Z = -1.260$	0.208
Lymphocyte count ($\times 10^9$/L), M (Q_1, Q_3)	2.525 (2.100, 3.128)	2.560 (2.180, 3.190)	2.340 (1.950, 2.790)	$Z = -2.916$	0.004
SII, M (Q_1, Q_3)	561.022 (365.680, 788.651)	471.510 (333.223, 688.378)	858.029 (624.640, 1167.208)	$Z = -7.336$	<0.001
PNI, mean ± SD	48.524 ± 7.576	50.809 ± 6.604	41.746 ± 6.105	$t = 9.443$	<0.001
Gender, n (%)				$\chi^2 = 0.044$	0.834
Male	160 (66.116)	119 (65.746)	41 (67.213)		
Female	82 (33.884)	62 (34.254)	20 (32.787)		
Diabetes mellitus, n (%)				$\chi^2 = 0.033$	0.856
No	233 (96.281)	175 (96.685)	58 (95.082)		
Yes	9 (3.719)	6 (3.315)	3 (4.918)		
Pressure injury location, n (%)				$\chi^2 = 0.084$	0.959
Sacrococcygeal	151 (62.397)	112 (61.878)	39 (63.934)		
Ischial tuberosity	70 (28.926)	53 (29.282)	17 (27.869)		
Greater trochanter	21 (8.678)	16 (8.840)	5 (8.197)		
Pressure injury stage, n (%)				$\chi^2 = 0.062$	0.803
III	5 (2.066)	3 (1.657)	2 (3.279)		
IV	237 (97.934)	178 (98.343)	59 (96.721)		
Bone exposure, n (%)				$\chi^2 = 1.399$	0.237
No	173 (71.488)	133 (73.481)	40 (65.574)		
Yes	69 (28.512)	48 (26.519)	21 (34.426)		
Flap type, n (%)				$\chi^2 = 0.003$	0.955
Local flap	88 (36.364)	66 (36.464)	22 (36.066)		
Musculocutaneous flap	154 (63.636)	115 (63.536)	39 (63.934)		
Concomitant osteomyelitis, n (%)				$\chi^2 = 0.928$	0.335
No	233 (96.281)	176 (97.238)	57 (93.443)		
Yes	9 (3.719)	5 (2.762)	4 (6.557)		

Abbreviations: CRP, C-reactive protein; WBC, white blood cell; SII, systemic immune-inflammation index; PNI, prognostic nutritional index.

Hosmer-Lemeshow goodness-of-fit test ($p = 0.335$), which was above the predefined significance threshold of 0.05. These findings indicate no significant difference between the predicted and observed outcomes, demonstrating good agreement between model predictions and actual observations.

Discussion

Using a retrospective cohort design, this study systematically evaluated the predictive factors associated with flap-related complications following flap repair in patients with stage III and IV pressure injuries. The results showed that, among patients with stage III and IV pressure injuries who underwent flap repair, elevated preoperative SII, reduced preoperative PNI, and increased intraoperative blood loss were independently associated with postoperative flap-related complications. After adjustment for other potential confounding factors, the prediction model based on these variables demonstrated good discrimination and calibration.

As a composite inflammatory index, SII integrates three types of blood cells: neutrophils (pro-inflammatory), platelets (involved in inflammation and microthrombosis), and lymphocytes (responsible for immune surveillance and

Table 2. Univariate logistic regression analysis of factors associated with postoperative flap-related complications.

Variables	β	SE	Z	*p*-value	OR (95% CI)
Gender					
Male					1.000
Female	–0.066	0.315	–0.209	0.834	0.936 (0.505–1.734)
Diabetes mellitus					
No					1.000
Yes	0.411	0.723	0.569	0.570	1.509 (0.366–6.225)
Pressure injury location					
Sacrococcygeal					1.000
Ischial tuberosity	–0.082	0.335	–0.245	0.806	0.921 (0.478–1.776)
Greater trochanter	–0.108	0.545	–0.199	0.843	0.897 (0.308–2.612)
Pressure injury stage					
Stage III					1.000
Stage IV	–0.699	0.925	–0.755	0.450	0.497 (0.081–3.048)
Bone exposure					
No					1.000
Yes	0.375	0.318	1.179	0.238	1.455 (0.780–2.712)
Flap type					
Local flap					1.000
Musculocutaneous flap	0.017	0.308	0.056	0.955	1.017 (0.556–1.861)
Concomitant osteomyelitis					
No					1.000
Yes	0.904	0.688	1.315	0.189	2.470 (0.641–9.512)
Age (per 1 year increase)	0.027	0.021	1.302	0.193	1.027 (0.987–1.069)
Intraoperative blood loss (per 100 mL increase)	0.963	0.262	3.674	<0.001	2.620 (1.567–4.379)
CRP (per 1 mg/L increase)	0.018	0.007	2.715	0.007	1.018 (1.005– ~1.031)
WBC count (per 1 × 10^9/L increase)	0.056	0.029	1.917	0.055	1.058 (0.999–1.120)
Neutrophil count (per 1 × 10^9/L increase)	0.564	0.100	5.632	<0.001	1.758 (1.445–2.140)
Platelet count (per 1 × 10^9/L increase)	0.020	0.004	5.417	<0.001	1.020 (1.013–1.028)
Lymphocyte count (per 1 × 10^9/L increase)	–0.778	0.257	–3.027	0.002	0.459 (0.278–0.760)
SII (per 100 unit increase)	0.423	0.063	6.739	<0.001	1.527 (1.350–1.727)
Serum albumin (per 1 g/L increase)	–0.262	0.039	–6.772	<0.001	0.770 (0.714–0.830)
PNI (per 1 point increase)	–0.227	0.033	–6.787	<0.001	0.797 (0.747–0.851)

OR, odds ratio.

Table 3. Multivariate logistic regression analysis of independent predictors of postoperative flap-related complications.

Variables	β	SE	Z	*p*-value	OR (95% CI)
PNI (per 1-point increase)	–0.162	0.036	–4.502	<0.001	0.851 (0.793–0.913)
SII (per 100-unit increase)	0.267	0.068	3.920	<0.001	1.307 (1.143–1.494)
Intraoperative blood loss (per 100-mL increase)	0.766	0.323	2.368	0.018	2.150 (1.141–4.052)

tissue repair). A high SII reflects excessive activation of the neutrophil and platelet systems, along with a relative depletion of lymphocytes [15]. This state may adversely affect the healing of pressure injury wounds. Overactivated neutrophils can release large amounts of reactive oxygen species and proteolytic enzymes, resulting in tissue injury and potentially contributing to flap necrosis [16]. In a dysregulated inflammatory environment, the reparative functions of platelets may also become impaired, leading to abnormal tissue remodeling and excessive fibrosis [17]. This finding is consistent with the study by Liu et al. [18], which demonstrated that elevated SII was associated with an increased risk of delayed healing of esophagojejunal anastomotic fistula. The present study further extends this association to flap repair in patients with pressure injuries, suggesting that the preoperative systemic inflammatory burden is a key factor influencing flap survival.

PNI is derived from serum albumin concentration and lymphocyte count, and a low PNI reflects protein-energy malnutrition and impaired immune function [19]. A study on head and neck free-flap reconstruction has shown that preoperative nutritional status is an important determinant of flap survival [5]. This observation is consistent with the findings of the present study.

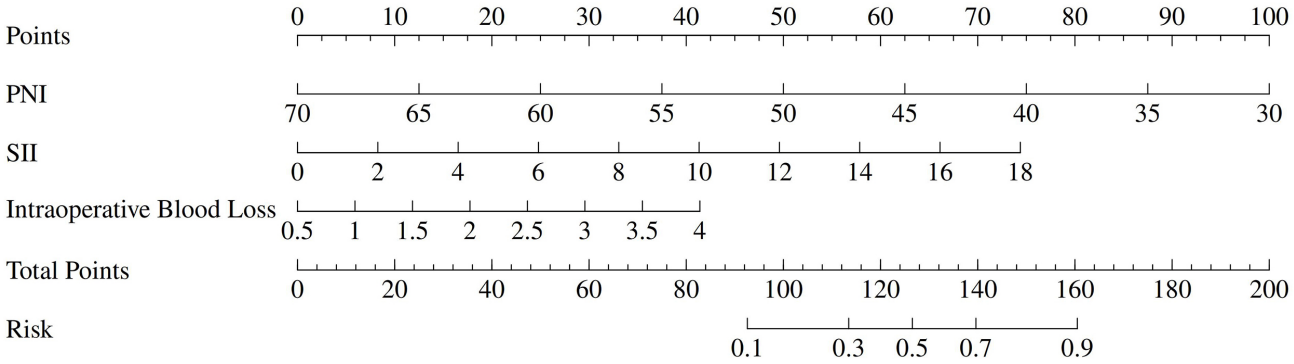

Fig. 1. Nomogram for predicting postoperative flap-related complications in patients with stage III and IV pressure injuries. Note: Intraoperative blood loss was measured in mL and modeled per 100-mL increase in logistic regression; SII was modeled per 100-unit increase; and PNI was modeled per 1-point increase. SII, systemic immune-inflammation index; PNI, prognostic nutritional index.

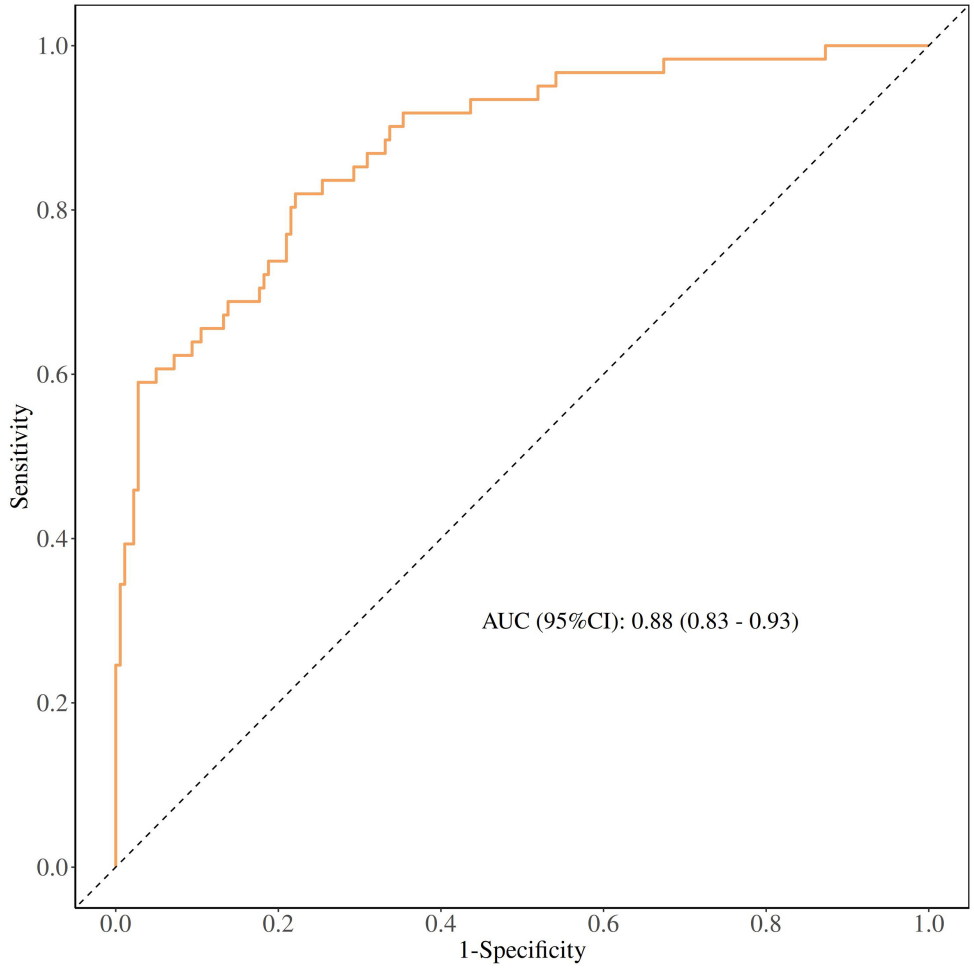

Fig. 2. Receiver operating characteristic (ROC) curve of the prediction model for postoperative flap-related complications. AUC, area under the curve.

Intraoperative blood loss was also identified as an independent risk factor associated with postoperative flap-related complications. Excessive blood loss can directly reduce effective circulating blood volume and impair tissue perfusion. Patients with pressure injuries often experience prolonged immobilization and may have underlying cardiovascular comorbidities, resulting in limited physiological reserve. Consequently, substantial intraoperative blood loss may increase the risk of intraoperative or early postoperative hypotension, leading to insufficient flap perfusion and

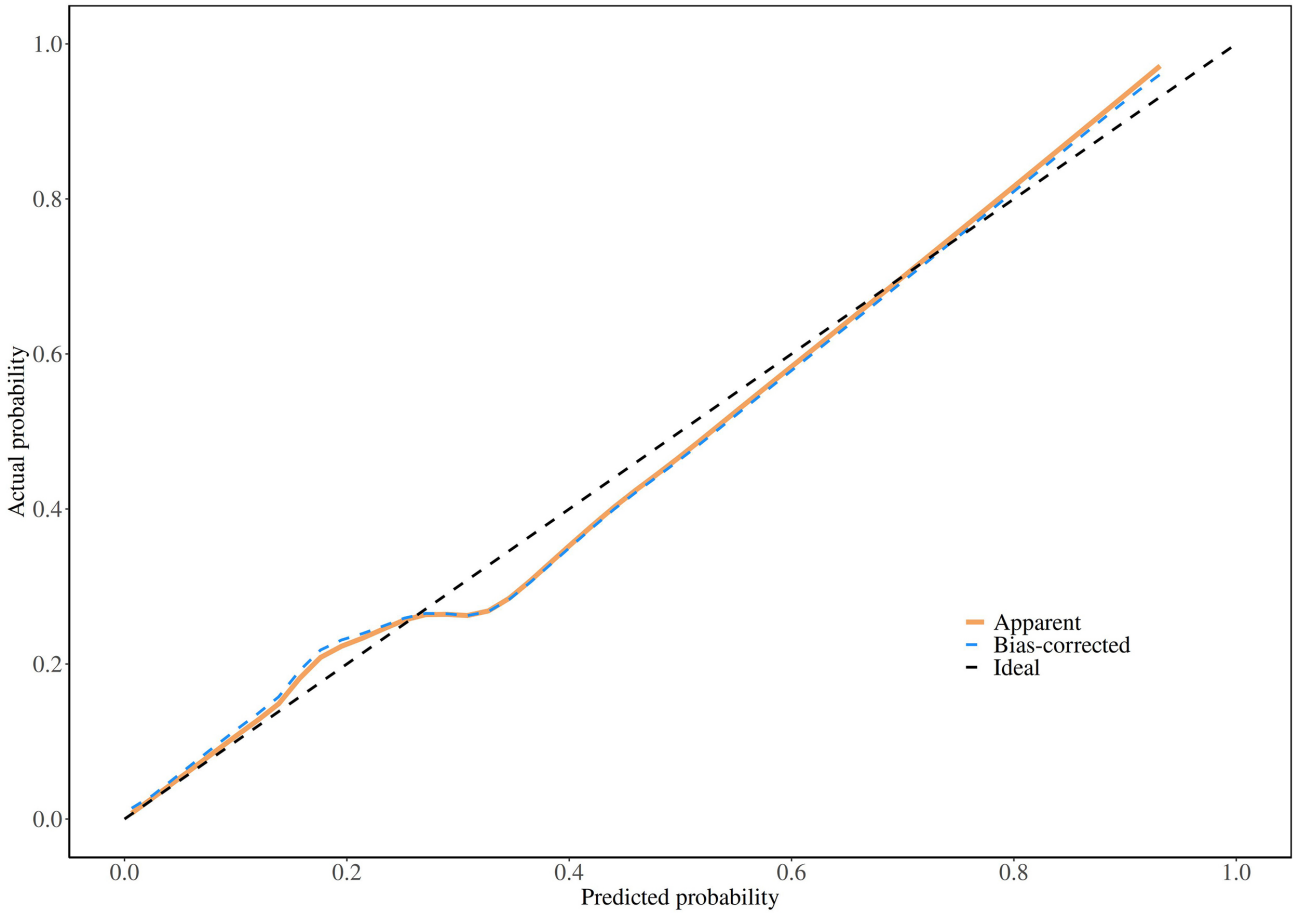

Fig. 3. Calibration curve of the prediction model for postoperative flap-related complications.

a critical state of ischemia and hypoxia. These pathophysiological changes may ultimately contribute to the development of flap-related complications.

The prediction model proposed in this study provides a practical tool for risk stratification and perioperative intervention planning. Unlike relatively static factors such as age, pressure injury location, or osteomyelitis, which are difficult to modify within a short period, the inflammatory and nutritional status reflected by SII and PNI are potentially modifiable. However, it is important to distinguish between statistical association and clinical utility. Although higher SII and lower PNI were associated with an increased risk of postoperative complications in this study, whether interventions targeting these markers (e.g., anti-inflammatory therapy or nutritional support) can effectively reduce complication rates remains uncertain.

For patients identified as high risk preoperatively (high SII and low PNI), clinicians should consider optimizing inflammatory and nutritional status as much as possible before surgery. Measures such as appropriate anti-inflammatory therapy, optimized pain management, and effective infection control may help reduce the systemic inflammatory burden. In addition, intensive and individualized nutritional support, especially supplementation with high-quality protein and immune-enhancing nutrients, may improve nutritional status and immune reserve. At the same time, the surgical team may develop a more detailed operative strategy for high-risk patients to minimize intraoperative blood loss. This individualized perioperative management strategy, based on biomarker assessment, has the potential to transform clinical practice from passive management of complications to proactive optimization of patient condition, thereby reducing the risk of postoperative complications.

This study has several limitations. First, as a single-center retrospective study, selection bias is unavoidable, and the generalizability of the findings should be interpreted with caution. Furthermore, our assessment of comorbidities was limited to diabetes mellitus and osteomyelitis. Patients with pressure injuries often have multiple additional conditions, such as prolonged immobilization and neurological impairment, which may confound the observed associations. Although PNI and SII were included as laboratory-based indicators to partially reflect nutritional and inflammatory status, residual confounding cannot be ruled out. Second, the model was validated only internally using bootstrap resampling. Internal validation alone is insufficient to support strong conclusions about clinical implementation. There-

fore, the generalizability and clinical applicability of the model require external validation in multicenter prospective cohorts before routine clinical use can be recommended. Third, only laboratory data obtained at a single preoperative time point were analyzed. Consequently, the potential prognostic information contained in dynamic perioperative changes of SII and PNI could not be evaluated. Fourth, most patients included in this study had stage IV pressure injuries. Although this reflects the clinical reality that patients with severe pressure injuries are more likely to require surgical intervention, the applicability of the conclusions to patients with other stages of pressure injuries may be limited. Future studies should include patients across a broader range of disease stages to further evaluate the generalizability of the model. Future research should focus on multicenter collaboration for external validation of the model. Moreover, prospective interventional studies are warranted to determine whether optimizing SII and PNI through targeted interventions, such as enhanced anti-inflammatory treatment and nutritional support, can reduce the incidence of postoperative flap-related complications.

Conclusions

This study demonstrated that preoperative SII and PNI are practical and convenient biomarkers for predicting flap-related complications following flap repair in patients with pressure injuries. A prediction model incorporating intraoperative blood loss, along with preoperative SII and PNI, enables individualized perioperative risk stratification for preoperative flap-related complications. However, these findings require external validation before broader clinical application. Furthermore, the proposed perioperative optimization strategy may provide a foundation for future interventional studies to improve surgical outcomes in this patient population.

Availability of Data and Materials

The data analyzed are available from the corresponding author upon reasonable request.

Author Contributions

SSZ and DQX designed the research study. SSZ and LJW performed the research. DQX and LJW analyzed the data. DQX drafted the article. All authors have been involved in revising the manuscript critically for important intellectual content. All authors gave final approval of the version to be published. All authors have participated sufficiently in the work to take public responsibility for appropriate portions of the content and agreed to be accountable for all aspects of the work in ensuring that questions related to its accuracy or integrity.

Ethics Approval and Consent to Participate

The study was approved by the Ethics Committee of Hangzhou First People's Hospital (approval number: 2026ZN096-1), and all procedures followed the principles of the Declaration of Helsinki. All clinical data were obtained from the electronic medical record system, and data collection and analysis were performed after anonymization to ensure patient confidentiality. Therefore, informed consent was waived.

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Conflict of Interest

The authors declare no conflict of interest.

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