Available online at www.medicinescience.org

ORIGINAL ARTICLE

Medicine Science International Medical Journal

Medicine Science 2026;15(3):1334-40

Association between systemic immune-inflammation index dynamics and time to pressure injury onset in intensive care units: A retrospective cohort study

Kivanc Oncu[1], Ismail Sarbay[2], Faruk Yildiz[1], Sezgin Uzun[3], Anil Celebi[3], Ozhan Ozcan[4]

[1]Sinop Atatürk State Hospital, Department of Anesthesiology and Reanimation, Sinop, Türkiye
[2]Sinop Atatürk State Hospital, Department of Thoracic Surgery, Sinop, Türkiye
[3]Sinop Atatürk State Hospital, Registered Nurse, Wound Care Unit, Sinop, Türkiye
[4]Sinop Atatürk State Hospital, Department of Intensive Care, Sinop, Türkiye

Received 03 March 2026; Accepted 27 April 2026
Available online 23 August 2026 with doi: 10.5455/medscience.2026.02.045

Abstract

Pressure injuries constitute a significant burden in intensive care units due to increased morbidity and healthcare costs. Since systemic inflammation is a key driver of tissue breakdown, this study evaluated the association of dynamic changes in the neutrophil-to-lymphocyte ratio, systemic immune-inflammation index, and platelet-to-lymphocyte ratio with the onset of pressure injuries in individuals with severe acute conditions. This retrospective observational analysis was carried out across the critical care departments of a regional referral center between January 2023 and June 2024. The analytical cohort was restricted solely to 51 adult critical care patients who acquired pressure injuries during their hospitalization. Baseline clinical profiles, including comorbid conditions and hematological data, were documented. The inflammatory indices were computed both upon critical care entry and concurrently with injury identification. Furthermore, the magnitude of biomarker reduction (admission-to-onset fold-decrease) was analyzed for potential correlations with the precise timeline of pressure injury formation. The analytical cohort comprised 52.9% females with an average age of 77.6 ± 9.1 years. Pressure injuries emerged, on average, at 19.4 ± 13.0 days after intensive care admission. Longitudinal tracking revealed a pronounced reduction in median inflammatory index values (all $p < 0.001$). Markedly, the proportional declines in the neutrophil-to-lymphocyte ratio and systemic immune-inflammation index demonstrated a strong positive correlation with the precise onset interval of pressure injuries ($p = 0.011$ and $p = 0.047$, respectively); however, this statistical link was absent for the platelet-to-lymphocyte ratio ($p = 0.549$). Dynamic decreases in neutrophil-to-lymphocyte ratio and systemic immune-inflammation index were associated with a longer time to pressure injury onset, suggesting that sustained systemic inflammation may accelerate tissue breakdown. Given the absence of a non-injury comparator group, these findings specifically reflect associations with the timing of onset rather than the absolute risk of occurrence. These markers may serve as useful indicators for monitoring the inflammatory trajectory in patients prone to pressure injuries.

Keywords: Pressure injury, intensive care units, biomarkers, risk assessment, inflammation

Introduction

Pressure injuries (PIs) constitute a serious complication in intensive care units (ICUs), significantly impacting patient outcomes and healthcare costs. A recent large-scale study reported a 26.6% prevalence within the adult critical care population, highlighting the magnitude of this problem globally [1]. Consequently, preventive strategies, ranging from mechanical pressure redistribution to specialized enteral nutrition support, remain a cornerstone of critical care management [2]. However, traditional clinical assessment tools often fail to capture the rapid physiological changes in critically ill patients, leading to a search for more objective biological markers. Within this framework, the Systemic Immune-Inflammation Index (SII) serves as a significant prognostic indicator for ICU settings, as it accurately captures the dynamic inflammatory state of the host [3].

The underlying pathophysiology of pressure injuries involves a complex interplay of ischemia, reperfusion injury, and cellular

CITATION
Oncu K, Sarbay I, Yildiz F, et al. Association between systemic immune-inflammation index dynamics and time to pressure injury onset in intensive care units: A retrospective cohort study. Med Science. 2026;15(3):1334-40.

Corresponding Author: Kivanc Oncu, Sinop Atatürk State Hospital, Department of Anesthesiology and Reanimation, Sinop, Türkiye
Email: dr.kivanc.oncu@gmail.com

DOI: 10.5455/medscience.2026.02.045

stress. A persistent and uncontrolled inflammatory response is the defining feature of chronic wounds, severely delaying the normal physiological transition to the proliferative phase of wound closure [4]. This prolonged inflammation, exacerbated by oxidative stress and enzymatic tissue degradation, results in delayed wound healing and accelerated tissue breakdown [5]. The clinical consequences of this inflammatory burden are profound; pressure injury complications are significantly associated with increased mortality risks, particularly in vulnerable elderly populations [6].

Given this background, monitoring inflammatory trajectories becomes crucial for risk profiling. Hemogram-based inflammatory indices —such as the neutrophil-to-lymphocyte ratio (NLR), SII, and platelet-to-lymphocyte ratio (PLR)—have demonstrated high predictive value for disease severity and outcomes in various other acute clinical conditions, including pneumonia and intracerebral hemorrhage [7].

However, limited data exist regarding the temporal relationship between these markers and the specific timeline of tissue breakdown in the ICU. Distinct from previous studies that primarily focused on static admission values, the present research evaluates the association linking dynamic changes in systemic inflammatory metrics to the period to PI onset. Therefore, the aim of this study was to evaluate the association between dynamic changes in systemic inflammatory metrics and the time to PI onset in a targeted cohort of intensive care patients.

Material and Methods

The research protocol adhered strictly to the ethical tenets of the Declaration of Helsinki, receiving formal authorization from the Sinop University Human Research Ethics Committee (Date: 27.09.2024; Decision: 2024/267). Because of the retrospective design of this research, the necessity for informed consent was officially waived. Participant privacy was protected by ensuring thorough data anonymization. Furthermore, our methodology and reporting align with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines.

Study Design and Population

This retrospective cohort analysis was carried out at a regional referral center from January 2023 through June 2024. This cohort included 51 adult ICU patients (> 18 years) who developed new-onset PIs and had complete blood count data at both admission and PI onset. To ensure injuries were hospital-acquired, patients with pre-existing wounds on admission or an ICU stay < 48 hours were excluded. Patients presenting with prior chronic wounds, hematological neoplasms, or undergoing immunocompromising regimens were omitted from the analysis. Throughout the critical care follow-up, patients received protocol-based management guided by institutional policies.

Epidermal surveillance was carried out daily by at least two independent evaluators (certified wound care nurses and ICU intensivists) to detect early signs of tissue damage. The staging and classification of PIs followed the international framework established by the European Pressure Ulcer Advisory Panel and the National Pressure Injury Advisory Panel in 2019 [8]. Herein, 'PI onset' was defined as the first clinical observation of a Stage 1 (non-blanchable erythema) or higher-grade PI. The Braden Scale served as the primary tool for risk evaluation at admission.

Data Collection

Data extraction, encompassing clinical and demographic variables, was performed through the institutional electronic healthcare record platform. The collected variables encompassed patient age, gender, baseline comorbidities (including cardiovascular disease, diabetes, and hypertension), primary reasons for ICU admission, total duration of stay, and final disposition (survival, transfer, or in-hospital mortality). Detailed PI data, including anatomical location and severity level, were formally recorded for each participant. The 'time to PI onset' represented the duration in days between a patient's admission to the ICU and the initial medical charting of a Stage 1 or higher PI. Routine hemogram data were extracted at dual clinical time points: upon initial critical care entry and concurrently with the manifestation of the pressure injury.

The following metrics were computed from standard cell counts: the NLR and PLR represented the simple ratios of neutrophils and platelets to lymphocytes. The SII, integrating all three cell lines, was quantified as the neutrophil-platelet product divided by the lymphocyte denominator.

Statistical Analysis

Statistical processing was facilitated by IBM SPSS Statistics 27.0. Baseline demographics were summarized using frequencies for categorical data, while continuous variables were described via means (± described via means with standard deviation (SD) or medians with interquartile range (IQR) based on their distribution. The Kolmogorov-Smirnov procedure was employed for distribution fitting; consequently, variables lacking parametric assumptions were evaluated through non-parametric testing.

The Wilcoxon signed-rank test served to compare the levels of inflammatory markers (SII, NLR, PLR) at the time of ICU admission versus the day of pressure injury diagnosis. For group comparisons involving two or multiple independent cohorts, the Mann-Whitney U and Kruskal-Wallis H tests were respectively executed. Lastly, associations linking the inflammatory ratios to the onset timing of pressure injuries were determined through Spearman's rank correlation analysis. To quantify the magnitude of inflammatory reduction, change ratios were computed for each index by dividing the admission value by the onset value (admission/onset). Additionally, patients were stratified into four fold-change tiers according to these calculated ratios to evaluate

DOI: 10.5455/medscience.2026.02.045

subgroup variances. For all comparative analyses, a two-tailed p-value below 0.05 denoted statistical significance.

Given the lack of established consensus on dynamic inflammatory cut-offs for pressure injury onset, these specific fold-change tiers were determined empirically based on the data distribution and preliminary exploratory analyses. This approach allowed us to effectively capture distinct clinical trajectories of the inflammatory response, ranging from no or minimal reduction (0 – 0.99) to profound decreases (≥ 5, ≥ 6, or ≥ 7, depending on the specific biomarker).

Results

Study Population

At the outset, the preliminary cohort comprised 64 intensive care patients who acquired PIs during their inpatient stay, all of whom underwent eligibility screening. Subsequent to the application of exclusion criteria (removing six patients presenting with solid organ or hematological malignancies, five patients on immunosuppressive therapies, and two cases with incomplete clinical records), 51 patients were retained for the ultimate analysis (Figure 1).

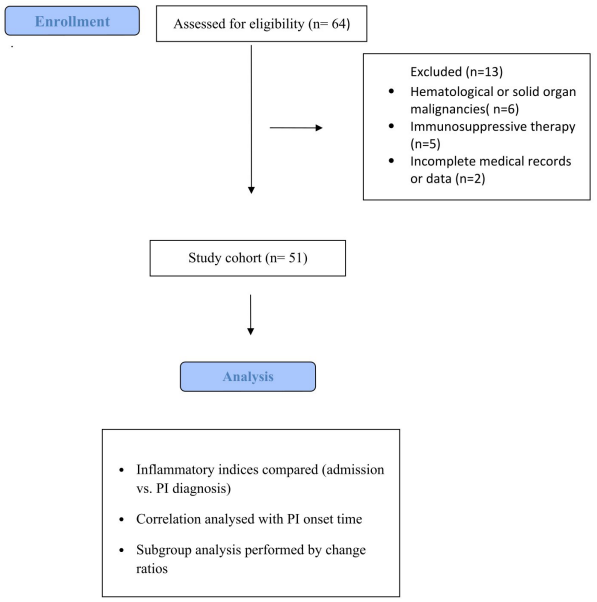

Figure 1. Flowchart of patient inclusion and analysis

Demographic and Clinical Characteristics

The demographic profile of the cohort revealed an average age of 77.63 ± 9.12 years (extremes: 52 – 92 years), with a slight female predominance (52.9% vs. 47.1% male). The baseline comorbidity burden was primarily driven by hypertension (58.8%), alongside high prevalences of neurological disorders or stroke (54.9%) and heart failure (52.9%). Other notable underlying pathologies comprised chronic obstructive pulmonary disease (COPD)/asthma, diabetes mellitus, and chronic kidney disease, affecting 25.5%, 23.5%, and 23.5% of the patients, respectively (Table 1).

Table 1. Demographic and clinical characteristics of the study population

Variable	n	%
Gender		
Female	27	52.9
Male	24	47.1
Age		
Mean ± SD / Min.-Max.	77.63 ± 9.12	52-92
Comorbidities		
Hypertension	30	58.8
Neurological disorders	28	54.9
Heart failure	27	52.9
COPD/Asthma	13	25.5
Diabetes Mellitus	12	23.5
Chronic kidney disease	12	23.5
Thyroid disorders	4	7.8
Time to PI development		
1-7 days (Week 1)	10	19.6
8-14 days (Week 2)	11	21.6
15-21 days (Week 3)	13	25.5
22-28 days (Week 4)	8	15.7
≥ 29 days (Week 5 and beyond)	9	17.6
Mean ± SD / Min.-Max.	19.35 ± 13.00	1-61
Anatomical region of PI		
Sacrum	39	76.5
Heels	3	5.9
Spine	2	3.9
Scapula	2	3.9
Trochanter	2	3.9
Leg	1	2.0
Gluteal region	1	2.0
Shoulder	1	2.0
Length of ICU stay		
11-30 days(1 month)	18	35.3
31-60 days (2 months)	20	39.2
≥ 61 days (3+ months)	13	25.5
Mean ± SD / Min.-Max.	50.14 ± 33.47	11-139
Outcome		
Discharged	11	21.6
Transferred to ward	3	5.9
Deceased	37	72.5

SD: Standard Deviation, Min. – Max.: Minimum – Maximum, COPD: Chronic obstructive pulmonary disease, PI: Pressure injury, ICU: Intensive Care Unit; "Deceased" refers to in-hospital mortality during ICU stay; Time to pressure injury onset is reported both categorically (by week) and as continuous data (mean ± SD); Anatomical regions reflect the location of the pressure injury. Length of ICU stay is categorized in clinically relevant intervals; Comorbidities were recorded based on diagnoses at ICU admission

DOI: 10.5455/medscience.2026.02.045

Regarding spatial distribution, sacral lesions were the most prevalent (76.5%), while heel (5.9%), spinal (3.9%), and other topographical injuries (gluteal, trochanteric, scapular) were less frequent. The precise timeline to PI formation averaged 19.35 ± 13.00 days (range: 1 – 61). Weekly incidence rates were distributed as follows: week one (19.6%), week two (21.6%), week three (25.5%), week four (15.7%), and beyond week four (17.6%). Overall critical care admission duration stood at 50.14 ± 33.47 days (spanning up to 139 days). By the conclusion of the study period, the mortality burden was high (72.5%), with only 21.6% of patients reaching discharge status and 5.9% undergoing intra-hospital transfer (Table 1).

Inflammatory Indices at ICU Admission and PI Development

The Wilcoxon signed-rank test was applied to determine shifts in the patients' immune-inflammatory status by contrasting the index values recorded on the day of ICU entry with those obtained at the time of PI development. Across all three evaluated biomarkers, statistically significant reductions were observed between these two clinical milestones (Table 2).

- **SII:** Baseline admission values demonstrated a median of 2240.37 (IQR: 1354.31 – 3605.58), which markedly decreased to 1193.24 (IQR: 630.74 – 1956.07) at the time of PI development ($p < 0.001$).
- **NLR:** A similarly pronounced decline occurred in the NLR, dropping from an admission median of 10.65 (IQR: 6.52 – 18.29) down to 6.25 (IQR: 3.94 – 9.77) at the time of PI development ($p < 0.001$).
- **PLR:** The initial median was recorded at 268.83 (IQR: 185.71 – 428.57), which subsequently decreased to 165.38 (IQR: 100.00 – 285.37) at the time of PI development ($p < 0.001$).

Table 2. Comparison of inflammatory index values between ICU admission and day of pressure injury diagnosis

Parameter	Mean	SD	Quartiles			Z	p
			Q1	Q2 (Median)	Q3		
SII at ICU admission	2895.83	2478.17	1354.31	2240.37	3605.58	**-3.41**	**< 0.001**
SII at PI Diagnosis	1525.20	1310.90	630.74	1193.24	1956.07		
NLR at ICU admission	13.53	9.94	6.52	10.65	18.29	**-3.94**	**< 0.001**
NLR at PI Diagnosis	7.72	5.41	3.94	6.25	9.77		
PLR at ICU admission	302.12	157.18	185.71	268.83	428.57	**-3.63**	**< 0.001**
PLR at PI Diagnosis	203.45	132.45	100.00	165.38	285.37		

SII: Systemic Immune-Inflammation Index, NLR: Neutrophil-to-Lymphocyte Ratio, PLR: Platelet-to-Lymphocyte Ratio, ICU: Intensive Care Unit, PI: Pressure Injury, SD: Standard Deviation; Z-values represent the test statistics obtained from the Wilcoxon signed-rank test; Q1, Q2, and Q3 refer to the first, second (median), and third quartiles, respectively; Comparisons were made between inflammatory index values at ICU admission and on the day of pressure injury diagnosis

Correlation Between Inflammatory Indices and Time to PI Development

Spearman's rank correlation was executed to determine how the calculated inflammatory ratios aligned with the time to PI development (Table 3).

- **NLR Fold-Change:** Similarly, the fold-decrease in NLR exhibited a significant positive relationship with the time interval to PI manifestation ($r = 0.353$, $p = 0.011$).
- **SII Fold-Change:** The magnitude of SII reduction (fold-decrease) demonstrated a statistically significant, positive correlation with the time interval to PI manifestation ($r = 0.280$, $p = 0.047$).
- **PLR Fold-Change:** Conversely, the alteration in PLR levels did not yield any significant correlation with the time interval to PI manifestation ($r = 0.086$, $p = 0.549$).

Table 3. Correlation between inflammatory index change ratios and time to pressure injury onset

Spearman's correlation		SII	NLR	PLR
Pressure injury onset (days)	**r**	**0.280**	**0.353**	0.086
	p	**0.047**	**0.011**	0.549

SII: Systemic Immune-Inflammation Index, NLR: Neutrophil-to-Lymphocyte Ratio, PLR: Platelet-to-Lymphocyte Ratio; Correlations were assessed using Spearman's rank correlation analysis; r represents the correlation coefficient, and p indicates the significance level; A positive r value indicates a direct relationship between the index change ratio and time to pressure injury onset

Subgroup Analysis Based on Change Ratios

To evaluate variations in the timing of PI development, the study population was stratified into four distinct cohorts utilizing the fold-change ratios of NLR, SII, and PLR (Table 4).

DOI: 10.5455/medscience.2026.02.045

Table 4. Comparison of time to pressure injury onset across different categories of inflammatory index change ratios

Index	Change Ratio (Fold-Change)*	n	Median (IQR)	*H*	p	Post-hoc (Significant Difference)
SII	0 – 0.99 (a)	17	14 (8 – 21)	8.38	0.039	d > a, b
	1 – 2.99 (b)	14	12.5 (6 – 21)			
	3 – 4.99 (c)	9	20 (11 – 28)			
	≥ 5 (d)	11	22 (18 – 34)			
NLR	0 – 0.99 (a)	11	14 (8.5 – 22)	8.77	0.033	c, d > b
	1 – 2.99 (b)	26	12.5 (6 – 21)			
	3 – 4.99 (c)	10	20.5 (17 – 27)			
	≥ 6 (d)	4	32.5 (19.5 – 44.5)			
PLR	0 – 0.99 (a)	16	18.5 (11 – 22.5)	2.47	0.480	-
	1 – 2.99 (b)	25	17 (8 – 24)			
	3 – 4.99 (c)	5	27 (17 – 27)			
	≥ 7 (d)	5	15 (10 – 19)			

SII: Systemic Immune-Inflammation Index, NLR: Neutrophil-to-Lymphocyte Ratio, PLR: Platelet-to-Lymphocyte Ratio, IQR: Interquartile Range. *H*: Test statistic from the Kruskal – Wallis H test; *Change ratio was calculated as the value at ICU admission divided by the value on the day of pressure injury diagnosis (Admission/Onset fold-decrease); Significant differences are based on post-hoc pairwise comparisons, where expressions such as "d > a, b" indicate that group d had a significantly longer time to pressure injury onset compared to groups a and b

Subsequent evaluation via the Kruskal-Wallis procedure demonstrated pronounced differences in the timeline to PI manifestation among the stratified categories for both the NLR and SII.

- **NLR Change Ratio:** Marked variances were also identified across the NLR brackets ($p = 0.033$). Specifically, patients exhibiting higher fold-decreases (3 – 4.99 and ≥ 6) showed a pronounced tendency toward delayed PI development relative to the 1 – 2.99 cohort.
- **SII Change Ratio:** Individuals situated in the highest SII reduction category (≥ 5) experienced a significantly prolonged time to PI development when contrasted with cohorts in the lower ratio brackets (0 – 0.99 and 1 – 2.99) ($p = 0.039$).
- **PLR Change Ratio:** Conversely, categorizing participants by their PLR change ratios yielded no statistically significant variations regarding the time to PI development ($p = 0.480$).

Discussion

In this retrospective cohort analysis, we demonstrated that dynamic reductions in SII and NLR following intensive care admission are significantly associated with a delayed onset of pressure injuries, whereas no such temporal relationship exists for the PLR.

PIs in individuals with severe acute conditions result from complex interactions between immobility, hemodynamic instability, and sustained systemic inflammation. Chronic inflammation, in particular, impairs wound healing through pro-inflammatory mediator release and enzymatic tissue degradation [4,5]. Recently, hematological indices like the SII, NLR, and PLR have emerged as simple, accessible markers of the systemic inflammatory burden [7-11].

This study evaluated the relationship linking temporal fluctuations in systemic inflammatory indices to the precise timeline of pressure injury manifestation in patients with severe acute conditions. Our findings indicate that a steeper downward trajectory (greater fold-decrease) in the SII and NLR metrics—measured from baseline critical care entry to the point of ulcer detection—was strongly linked to an extended latency prior to PI manifestation. Conversely, patients who maintained high inflammatory burdens (lower change ratios) experienced an accelerated timeline of tissue breakdown. In contrast, no statistically significant association was observed regarding the PLR. Collectively, these findings suggest that the trajectory of the inflammatory burden during the ICU stay is closely linked to the timeline of tissue breakdown.

The clinical relevance of these hematological ratios is well-documented within critical care literature. Specifically, a high SII has been identified as a predictor of unfavorable outcomes, such as increased sepsis-related mortality, reflecting its role as an indicator of protracted inflammatory stress [12]. Similarly, a high NLR is frequently linked to a poor prognosis in various critical conditions, including multi-organ dysfunction and impaired wound repair [8,9]. In concordance with existing paradigms,

DOI: 10.5455/medscience.2026.02.045

our data posit that an ongoing systemic inflammatory burden—reflected by persistently high hematological profiles—compromises local tissue defense mechanisms, thereby accelerating ulcerogenesis.

Significantly, the present research expands upon earlier findings by illustrating that longitudinal shifts in these biomarkers—as opposed to solitary, static values—provide deeper insights into the inflammatory trajectory and its correlation with tissue injury. This longitudinal perspective accounts for the dynamic fluctuations in host immunity that cross-sectional assessments often overlook. Moreover, the stratified analyses indicated that individuals with more substantial reductions in SII and NLR exhibited significantly delayed PI manifestation. These results reinforce the premise that the attenuation of early-stage systemic immune activation prolongs the integrity of the tissue, whereas sustained systemic inflammation precipitates the timeline of pressure injury development.

While the PLR has been suggested as a potential inflammatory marker, the present study found no significant association with the onset of PIs. This discrepancy likely arises from the thrombosis-centric profile of the PLR, which primarily captures platelet reactivity rather than the leukocytic shifts (neutrophilic or lymphocytic) observed in other indices. Previous research highlights that the PLR is more reflective of systemic coagulation status and thrombotic risk, parameters that may not be directly involved in the localized tissue ischemia and inflammatory breakdown that define the pathogenesis of PI formation [13].

These observations are in agreement with contemporary research identifying robust correlations between the SII and traditional inflammatory parameters like C-reactive protein (CRP), especially in scenarios involving chronic, low-grade inflammation [14]. Such a relationship reinforces the potential of the SII to function as a proxy for the total systemic inflammatory load in critical care settings. Being both objective and readily accessible, this biomarker offers a quantifiable dimension that could enhance standard bedside clinical monitoring.

Crucially, although clinical scoring systems like the Braden Scale remain the cornerstone of PI risk stratification, their efficacy is inherently tied to subjective clinical assessments and functional monitoring. The incorporation of objective inflammatory indicators, specifically the SII and NLR, into current intensive care protocols could significantly augment clinical monitoring and facilitate more timely prophylactic measures. Earlier research has underscored the synergistic benefit of merging quantifiable laboratory data with traditional clinical scales to refine patient management in high-acuity environments [15].

The routine surveillance of SII and NLR trajectories holds profound relevance for critical care. In contrast to conventional, cross-sectional scoring systems, the continuous appraisal of these hematological ratios equips nursing staff with the foresight to recognize preemptive inflammatory surges well before macroscopic tissue degradation becomes evident [15]. This proactive foresight is especially crucial for physiologically vulnerable patients, allowing for timely adjustments in preventive strategies such as repositioning schedules, nutritional support, and intensified skin surveillance [3,6]. Incorporating the longitudinal trajectories of these biomarkers into standard nursing protocols cultivates a highly tailored and proactive strategy for PI prophylaxis. Such an integration resonates strongly with modern paradigms of patient-centric management and targeted metabolic modulation [16,17].

The observed association between dynamic inflammatory changes and PI onset emphasizes the need for a holistic approach in critical care. Our findings should be interpreted alongside recent evidence advocating for patient-centered care, particularly regarding individualized nutritional strategies. As highlighted in critically ill neurological and oncological populations, tailoring nutritional support is crucial not only for meeting metabolic demands but also for modulating inflammatory responses and improving patient-centered outcomes [16]. Furthermore, the utility of systemic inflammatory indices extends beyond PI surveillance. Recent studies suggest that early biomarkers, such as NLR and platelet-derived indices, are pivotal for risk stratification in sepsis and for predicting infections caused by multidrug-resistant organisms [17]. Therefore, monitoring these dynamic indices provides a comprehensive window into the patient's immune-inflammatory status, alerting clinicians to a heightened risk of both infectious complications and tissue vulnerability.

Furthermore, the complex interplay between systemic inflammation and local tissue perfusion is inevitably influenced by critical clinical confounders. For instance, high-dose vasopressor administration not only exacerbates peripheral ischemia through severe vasoconstriction but can also synergistically amplify local inflammatory cascades, thereby accelerating tissue necrosis [1,4]. Similarly, profound sedation restricts spontaneous micro-movements, resulting in sustained mechanical stress that impairs capillary perfusion and prevents the clearance of pro-inflammatory cytokines from vulnerable tissue beds [5,8]. Although the sample size of the present cohort precluded multivariate adjustments for these variables, their profound potential to interact with systemic inflammatory indices highlights the necessity of incorporating these factors into future comprehensive risk assessment models.

In conclusion, the present research underscores the clinical significance of monitoring the dynamic evolution of systemic inflammatory indices—specifically the SII and NLR—to gain essential insights into the risk and exact timeline of PI development in ICU populations. Translating these quantifiable inflammatory markers into clinical practice actively drives the deployment of patient-specific surveillance and preventive pathways in the ICU.

DOI: 10.5455/medscience.2026.02.045

Conclusion

This study provides compelling evidence that the magnitude of reduction (fold-decrease) in systemic inflammatory indices—especially NLR and SII—is directly associated with the timing of PI onset. Profound reductions in these markers indicate a delayed onset, whereas sustained inflammatory elevation appears to accelerate tissue breakdown. Unlike static admission values, monitoring the dynamic trajectory of these indices offers a real-time window into the patient's inflammatory status and tissue vulnerability. Although these markers should be interpreted within the context of a multimodal clinical assessment, our findings suggest that they represent valuable, cost-effective, and easily accessible adjunctive tools that can refine inflammatory surveillance and support personalized care decisions in the ICU.

Limitations

Certain limitations of this study should be noted. Primarily, the retrospective methodology, single-institution framework, and relatively modest sample size constrain the generalizability of the findings. Second, the cohort consisted exclusively of patients who developed PIs. The lack of a non-PI comparator group prevents the calculation of relative risks; thus, our results reflect associations with the timing of onset rather than the risk of occurrence. Third, sample size constraints precluded multivariate adjustments for major clinical confounders. Specifically, critical variables such as sedation depth, exact mobility status, vasopressor dosages, and combined nutritional-inflammatory metrics (e.g. CRP/albumin ratio [CAR] or hemoglobin, albumin, lymphocyte and platelet [HALP] score) could not be integrated into our analysis. Future prospective studies incorporating these multidimensional parameters alongside a control group are essential to validate and expand upon our findings.

Ethical Approval
This study was approved by the Sinop University Human Research Ethics Committee (Date: 27.09.2024, Decision No: 2024/267).

Patient Consent
Written informed consent was not obtained because of the retrospective, non-interventional design of the study. All data were extracted from existing electronic medical records and were fully anonymized before analysis, and no identifying information was collected. Accordingly, the requirement for informed consent was waived by the Sinop University Human Research Ethics Committee (Date: 27.09.2024, Decision No: 2024/267).

Conflict of Interest
The authors confirm the absence of any conflicts associated with this work.

Funding
The authors declare that they have received no financial support for the study.

References

1. Labeau SO, Afonso E, Benbenishty J, et al. Prevalence, associated factors and outcomes of pressure injuries in adult intensive care unit patients: the DecubICUs study. Intensive Care Med. 2021;47:160-9. Erratum in: Intensive Care Med. 2021;47:503-20.
2. Cox J, Rasmussen L. Enteral nutrition in the prevention and treatment of pressure ulcers in adult critical care patients. Crit Care Nurse. 2014;34:15-28.
3. Yılmaz H, Kazezoğlu C, Gedikbaşı A. Predictive value of systemic immune-inflammation index in patients hospitalized in the intensive care unit. Med J Bakirkoy. 2022;18:364-9.
4. Zhao R, Liang H, Clarke E, et al. Inflammation in chronic wounds. Int J Mol Sci. 2016;17:2085.
5. Diegelmann RF, Evans MC. Wound healing: an overview of acute, fibrotic and delayed healing. Front Biosci. 2004;9:283-9.
6. Song YP, Shen HW, Cai JY, et al. The relationship between pressure injury complication and mortality risk of older patients in follow-up: a systematic review and meta-analysis. Int Wound J. 2019;16:1533-44.
7. Wang RH, Wen WX, Jiang ZP, et al. The clinical value of neutrophil-to-lymphocyte ratio (NLR), systemic immune-inflammation index (SII), platelet-to-lymphocyte ratio (PLR) and systemic inflammation response index (SIRI) for predicting the occurrence and severity of pneumonia in patients with intracerebral hemorrhage. Front Immunol. 2023;14:1115031.
8. Kottner J, Cuddigan J, Carville K, et al. Prevention and treatment of pressure ulcers/injuries: the protocol for the second update of the international Clinical Practice Guideline 2019. J Tissue Viability. 2019;28:51-8.
9. Ucar MA, Tombak A, Akdeniz A, et al. Immune and inflammation markers as a predictor of overall survival in patients with hematologic malignancies: a retrospective cohort study. Medicina (Kaunas). 2025;61:1019.
10. Pan WG, Chou YC, Wu JL, Yeh TT. Impact of hematologic inflammatory markers on the prognosis of geriatric hip fracture: a systematic review and meta-analysis. Eur J Med Res. 2024;29:609.
11. Fois AG, Paliogiannis P, Scano V, et al. The systemic inflammation index on admission predicts in-hospital mortality in COVID-19 patients. Molecules. 2020;25:5725.
12. Jiang D, Bian T, Shen Y, Huang Z. Association between admission systemic immune-inflammation index and mortality in critically ill patients with sepsis: a retrospective cohort study based on MIMIC-IV database. Clin Exp Med. 2023;23:3641-50.
13. Xu R, Chen L, Yan C, et al. Elevated platelet-to-lymphocyte ratio as a predictor of all-cause and cardiovascular mortality in hypertensive individuals. J Clin Hypertens (Greenwich). 2025;27:e14980.
14. Ustundag Y, Huysal K, Karadag Gecgel S, Unal D. Relationship between C-reactive protein, systemic immune-inflammation index, and routine hemogram-related inflammatory markers in low-grade inflammation. Int J Med Biochem. 2018;1:24-8.
15. Keegan MT, Gajic O, Afessa B. Severity of illness scoring systems in the intensive care unit. Crit Care Med. 2011;39:163-9.
16. Stoian M, Stoian A, Bănescu C, et al. Nutrition and hydration at the end of life in intensive care and general end-of-life care settings: balancing clinical evidence, patient-centered care, and ethical and legal principles—a narrative review. Nutrients. 2025;17:3705.
17. Stoian M, Azamfirei L, Stîngaciu AC, et al. Early diagnostic markers and risk stratification in sepsis: prognostic value of neutrophil-to-lymphocyte ratio, platelets, and the Carmeli score. Biomedicines. 2025;13:2658.