

The Role of NLR and MPV in Predicting Pressure Injury Development in ICU Patients: A Retrospective Cohort Study

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Abstract

Background: Pressure injuries are a common and serious complication in critically ill patients, yet reliable and accessible biomarkers for early risk stratification remain lacking. This study investigated the predictive value of the neutrophil-to-lymphocyte ratio (NLR) and mean platelet volume (MPV) for pressure injury development in intensive care unit (ICU) patients.

Methods: This retrospective cohort study included 941 adult patients admitted to the ICU of a tertiary referral centre between January 2022 and January 2025. NLR and MPV were calculated from admission complete blood counts. Pressure injuries were identified and staged by a wound care nurse according to the NPUAP/EPUAP 2019 classification. Univariate and multivariate logistic regression, propensity score matching, competing risk analysis, ROC curve analysis, and Kaplan–Meier survival analysis were performed.

Results: Pressure injuries developed in 191 patients (20.3%), with a median onset of 9 days after ICU admission. NLR was significantly higher in patients who developed pressure injuries compared to those who did not (median 8.83 vs. 6.50; $p=0.005$), whereas MPV did not differ between groups ($p=0.355$). Propensity score matching confirmed that NLR elevation was explained by confounding from illness severity rather than a direct association with pressure injury development. Neither NLR nor MPV demonstrated clinically meaningful discriminative ability in ROC analysis (AUC 0.566 and 0.522, respectively), and neither emerged as an independent predictor in multivariate analysis. Independent predictors of pressure injury development were ICU length of stay (aOR 1.074; $p<0.001$), Braden score (aOR 0.751; $p<0.001$), and hemoglobin level (aOR 0.873; $p=0.003$). ICU mortality was markedly higher in patients who developed pressure injuries (83.8% vs. 30.7%; OR 11.67, 95% CI 7.71–17.67; $p<0.001$).

Conclusions: A single admission measurement of NLR or MPV is insufficient to reliably predict pressure injury development in critically ill patients. ICU length of stay, Braden score, and hemoglobin level are more clinically informative predictors. The strong association between pressure injury and ICU mortality underscores the need for early and sustained preventive strategies in this patient population.

BACKGROUND

Pressure injuries (PIs) are localized lesions of the skin and underlying soft tissue resulting from prolonged pressure, shear, or friction, typically occurring over bony prominences. They represent a significant global public health challenge, with considerable impact on patient morbidity, mortality, and healthcare costs (1). Among hospitalized patients, prevalence has been estimated at approximately 12%, with substantially higher rates reported in intensive care units (2).

Critically ill patients bear a disproportionate burden of this complication. An international point-prevalence study conducted across 1,117 ICUs in 90 countries found an overall PI prevalence of 26.6%, with 59.2% of all identified injuries acquired during the ICU stay itself (2). Several factors inherent to critical illness, including hemodynamic instability, impaired tissue perfusion, sedation, and prolonged

immobility, render these patients especially vulnerable. The financial consequences are equally significant, with national expenditures for PI prevention and treatment estimated to reach billions of euros annually (3).

Timely identification of at-risk patients is central to any effective prevention strategy. The Braden scale remains the most widely adopted risk assessment tool in clinical practice, yet its performance in the ICU setting has been questioned. A recent systematic review concluded that the reliability and validity of the Braden scale in intensive care are variable, and that it should be applied with caution as a standalone predictive instrument (4). These limitations have driven interest in objective, laboratory-derived parameters that could strengthen or complement clinical judgment. In this regard, serum biomarkers obtained at the time of ICU admission, when incorporated into predictive models alongside key clinical variables, have demonstrated strong discriminative ability for identifying patients at high risk of developing pressure injuries (5).

Systemic inflammation is increasingly recognized as a key contributor to PI pathophysiology. A range of inflammatory mediators, including tumor necrosis factor-alpha, interleukin-15, and C-reactive protein, have been associated with pressure injury occurrence, and their measurement has been proposed as a basis for earlier detection (6). In this context, the neutrophil-to-lymphocyte ratio (NLR) and mean platelet volume (MPV) are particularly attractive candidates. Both are derived from the complete blood count, a test universally obtained at admission, and thus carry no additional cost or procedural burden.

MPV reflects platelet size and, by extension, platelet activation; it has been established as a useful prognostic biomarker across a range of thrombotic and inflammatory conditions (7). In critically ill patients specifically, higher MPV values have been associated with worse clinical trajectories across a broad spectrum of diagnoses (8). NLR, calculated by dividing the absolute neutrophil count by the absolute lymphocyte count, captures the balance between innate immune activation and adaptive immune suppression, a pattern characteristic of systemic inflammatory states. It can be obtained from standard blood tests routinely performed at ICU admission and has shown significant correlation with disease severity and clinical outcomes in sepsis (9).

The relevance of these markers to PI development was first examined by Adiyeké and Adiyeké, who evaluated 104 ICU patients in a retrospective cohort design. Both NLR and MPV were significantly higher in patients who subsequently developed pressure ulcers, and logistic regression confirmed that, alongside age and length of ICU stay, they were independent predictors of ulcer development, suggesting that an elevated inflammatory baseline at admission carries prognostic weight (10). Building on this, a later study found that MPV was significantly higher in more advanced pressure ulcer stages, lending further support to its role as a marker of wound severity (11).

Despite these preliminary findings, evidence on the predictive utility of NLR and MPV in PI development remains sparse, and their performance across larger and more heterogeneous ICU populations has yet to be systematically evaluated. The present study therefore aimed to investigate the value of admission

NLR and MPV as inflammatory biomarkers for predicting pressure injury development in ICU patients, and to assess their potential contribution to early risk stratification in clinical practice.

METHODS

Study Design and Setting

This retrospective cohort study was conducted in the intensive care unit (ICU) of Zonguldak Atatürk State Hospital, between January 1, 2022 and January 1, 2025. The study was approved by the Zonguldak Bülent Ecevit University Non-Interventional Clinic Research Ethics Committee (Protocol No: 2026/02-09) and was conducted in accordance with the ethical principles of the Declaration of Helsinki. Given the retrospective nature of the study, the requirement for individual informed consent was waived by the ethics committee. The sample size was determined by the available patient population during the study period.

Participants

All adult patients aged 18 years or older admitted to the ICU during the study period were screened for eligibility. To be included, patients had to have undergone regular clinical assessment for pressure injury development throughout their ICU stay and to have had a complete blood count performed at or near the time of ICU admission, with available neutrophil, lymphocyte, and MPV values.

Patients were excluded if they had a pressure injury present at the time of ICU admission, a diagnosis of hematological malignancy, active hematological disease, or bone marrow failure, a history of active chemotherapy or radiotherapy likely to affect complete blood count parameters, severe burns, major trauma, or end-stage malignancy with the potential to directly influence pressure injury development, or incomplete laboratory data. Following the application of these criteria, 941 patients were included in the final analysis.

ICU Care Protocol and Pressure Injury Assessment

All ICU beds were equipped with dynamic air mattresses throughout the study period. Patients were repositioned every 120 minutes. Barrier cream was applied routinely to all patients as part of moisture management, particularly in the presence of incontinence, and prophylactic silicone foam dressings were placed over high-risk anatomical areas, including the sacrum and heels, in accordance with the same guidelines. Skin and tissue assessment was performed twice daily during routine nursing care, meeting the minimum frequency recommended by the European Pressure Ulcer Advisory Panel (EPUAP)/ National Pressure Injury Advisory Panel (NPIAP) 2019 recommendations. Nutritional support was provided individually via enteral or oral feeding based on each patient's caloric and protein requirements.

Pressure injury risk was evaluated daily using the Braden Scale; the score obtained at the time of ICU admission was used for the purposes of this study. Pressure injuries were identified and staged by a dedicated wound care nurse according to the EPUAP/ NPUAP 2019 classification system. This system defines four stages of injury severity: Stage 1 refers to non-blanchable erythema overlying intact skin, Stage 2 to partial-thickness skin loss with exposed dermis, Stage 3 to full-thickness skin loss, and Stage 4 to full-thickness skin and tissue loss.

Data Collection

Demographic characteristics, comorbidities, clinical parameters, and laboratory values were retrieved from the hospital's electronic medical records. The neutrophil-to-lymphocyte ratio (NLR) was calculated by dividing the absolute neutrophil count by the absolute lymphocyte count, both obtained from the complete blood count performed at ICU admission. Mean platelet volume (MPV) was derived from the same admission blood sample. For patients who developed pressure injuries, the date of onset, anatomical localisation, and stage at identification were recorded. ICU outcome was defined as survival to ICU discharge or in-ICU death.

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics version 26 (IBM Corp., Armonk, NY, USA). The normality of continuous variables was assessed using the Kolmogorov-Smirnov test. As all variables deviated significantly from a normal distribution, continuous variables are expressed as median with interquartile range (IQR) and were compared between groups using the Mann-Whitney U test. Categorical variables are expressed as frequencies and percentages and were compared using the Pearson Chi-square test or Fisher's exact test, as appropriate. For comparisons involving more than two independent groups, the Kruskal-Wallis test was applied, followed by pairwise Mann-Whitney U tests with Bonferroni correction for post-hoc analysis.

Univariate binary logistic regression was used to examine the association between each candidate variable and pressure injury development. Variables with $p < 0.10$ in univariate analysis were entered into a multivariate logistic regression model. Multicollinearity was assessed using the variance inflation factor (VIF); all VIF values were below 1.7, indicating no meaningful multicollinearity. Model fit was evaluated using the Hosmer-Lemeshow goodness-of-fit test and Nagelkerke R^2 , and overall discriminative ability was summarised by the c-statistic.

Receiver operating characteristic (ROC) curve analysis was performed to assess the ability of NLR, MPV, and Braden score to discriminate between patients who did and did not develop pressure injuries. Optimal cut-off values were determined using the Youden index (sensitivity + specificity - 1), and 95% confidence intervals for the area under the curve (AUC) were estimated using the bootstrap method with 2000 iterations. Differences between AUC values were evaluated using the bootstrap permutation

approach. Correlations between continuous variables were assessed using Spearman's rank correlation coefficient (r_s). A two-tailed p value of <0.05 was considered statistically significant throughout.

To address potential confounding by illness severity, a 1:1 propensity score matching (PSM) analysis was performed. Propensity scores were estimated using a logistic regression model incorporating age, APACHE II score, Braden score, sepsis, mechanical ventilation, vasopressor use, sex, and cerebrovascular disease as covariates. Matching was performed using nearest-neighbour matching with a caliper of 0.1 standard deviations of the logit of the propensity score, without replacement. Balance between matched groups was assessed using the standardised mean difference (SMD), with values below 0.10 indicating adequate balance. The association of NLR and MPV with pressure injury development was re-examined in the matched cohort using the Mann-Whitney U test and binary logistic regression.

Given the high in-ICU mortality observed in this cohort, a competing risk analysis was performed to account for death as a competing event for pressure injury development, as patients who die in the ICU cannot subsequently develop a pressure injury. Cumulative incidence functions (CIFs) for pressure injury development were estimated using the Aalen-Johansen estimator. Cause-specific Cox proportional hazards regression was used to examine the association of NLR and MPV with time to pressure injury development, treating in-ICU death as a competing event.

Finally, a pre-specified subgroup analysis was conducted stratifying patients by sepsis status, given that sepsis is a strong independent driver of NLR elevation and accounted for 51.8% of the pressure injury group. A multiplicative interaction term (NLR $\times$ sepsis and MPV $\times$ sepsis) was included in logistic regression models to formally test for effect modification. This study was reported in accordance with the STROBE (Strengthening the Reporting of Observational Studies in Epidemiology) guidelines.

RESULTS

A total of 941 patients were included in the final analysis (Fig. 1). Of these, 191 (20.3%) developed a pressure injury during their ICU stay, while 750 (79.7%) did not. The demographic, clinical, and laboratory characteristics of both groups are presented in Tables 1A, 1B, and 1C.

Patients who developed pressure injuries were significantly older than those who did not (median 81 years, IQR 74–88 vs. 74 years, IQR 64–83; $p < 0.001$) and had a higher proportion of female patients (53.4% vs. 44.3%; $p = 0.024$). Among comorbidities, cerebrovascular disease was more prevalent in the pressure injury group (30.9% vs. 12.1%; $p < 0.001$), as was psychiatric diagnosis (13.6% vs. 8.7%; $p = 0.039$). Coronary artery disease and smoking history were less frequent among patients who developed pressure injuries (26.7% vs. 35.2%, $p = 0.026$; and 14.7% vs. 22.1%, $p = 0.023$, respectively). No significant differences were observed between groups with respect to diabetes mellitus, chronic kidney disease, COPD/asthma, or malignancy (Table 1A).

Table 1
A. Demographic characteristics of patients

Age (years)	All patients (n = 941)	PI Present (n = 191)	PI Absent (n = 750)	p
	76 (65–84)	81 (74–88)	74 (64–83)	< 0.001
Sex – Female, n (%)	434 (46.1%)	102 (53.4%)	332 (44.3%)	0.024
Hypertension, n (%)	585 (62.2%)	129 (67.5%)	456 (60.8%)	0.086
Diabetes mellitus, n (%)	334 (35.5%)	72 (37.7%)	262 (34.9%)	0.476
Coronary artery disease, n (%)	315 (33.5%)	51 (26.7%)	264 (35.2%)	0.026
Chronic kidney disease, n (%)	115 (12.2%)	19 (9.9%)	96 (12.8%)	0.283
COPD/Asthma, n (%)	252 (26.8%)	58 (30.4%)	194 (25.9%)	0.210
Cerebrovascular disease, n (%)	150 (15.9%)	59 (30.9%)	91 (12.1%)	< 0.001
Smoking history, n (%)	194 (20.6%)	28 (14.7%)	166 (22.1%)	0.023
Malignancy, n (%)	146 (15.5%)	35 (18.3%)	111 (14.8%)	0.230
Psychiatric diagnosis, n (%)	91 (9.7%)	26 (13.6%)	65 (8.7%)	0.039

Values are expressed as median (IQR) for continuous variables and n (%) for categorical variables. Mann-Whitney U test was used for continuous variables; Chi-square test was used for categorical variables. PI: pressure injury; IQR: interquartile range; COPD: chronic obstructive pulmonary disease. Bold p values indicate statistical significance ($p < 0.05$).

Patients in the pressure injury group had significantly longer ICU stays (median 30 days, IQR 10–68 vs. 4 days, IQR 2–9; $p < 0.001$), higher APACHE II scores (18, IQR 13–22 vs. 12, IQR 7–19; $p < 0.001$), and lower Braden scores at admission (12, IQR 11–13 vs. 15, IQR 13–17; $p < 0.001$). Sepsis (51.8% vs. 27.2%; $p < 0.001$), mechanical ventilation (42.4% vs. 17.7%; $p < 0.001$), and vasopressor use (70.2% vs. 32.0%; $p < 0.001$) were all significantly more common in the pressure injury group (Table 1B). The most frequent admission diagnoses in the pressure injury group were pneumonia (23.0%), cerebrovascular accident (19.9%), and urosepsis (7.3%), whereas postoperative patients, including those admitted following lumbar stabilisation (0.5%) or femur fracture repair (0.0%), rarely developed pressure injuries ($p < 0.001$ for both)

Table 1
B. Clinical characteristics of patients

ICU length of stay (days)	All patients (n = 941)	PI Present (n = 191)	PI Absent (n = 750)	p
	5 (3–15)	30 (10–68)	4 (2–9)	< 0.001
APACHE II score	13 (8–20)	18 (13–22)	12 (7–19)	< 0.001
Braden score	15 (12–17)	12 (11–13)	15 (13–17)	< 0.001
MV duration (days)	0 (0–5)	20 (4–55)	0 (0–2)	< 0.001
Sepsis, n (%)	303 (32.2%)	99 (51.8%)	204 (27.2%)	< 0.001
Mechanical ventilation, n (%)	214 (22.7%)	81 (42.4%)	133 (17.7%)	< 0.001
Vasopressor use, n (%)	374 (39.7%)	134 (70.2%)	240 (32.0%)	< 0.001
Weaning, n (%)	34 (3.6%)	12 (6.3%)	22 (2.9%)	0.027
ICU outcome – Exitus, n (%)	390 (41.4%)	160 (83.8%)	230 (30.7%)	< 0.001

Values are expressed as median (IQR) for continuous variables and n (%) for categorical variables. Mann-Whitney U test was used for continuous variables; Chi-square test was used for categorical variables. PI: pressure injury; ICU: intensive care unit; APACHE II: Acute Physiology and Chronic Health Evaluation II; MV: mechanical ventilation. Bold p values indicate statistical significance ($p < 0.05$).

Regarding laboratory parameters, NLR was significantly higher in patients who developed pressure injuries compared to those who did not (median 8.83, IQR 3.94–16.16 vs. 6.50, IQR 3.07–12.90; $p = 0.005$). Neutrophil count and hemoglobin also differed significantly between groups ($p = 0.016$ and $p = 0.005$, respectively). In contrast, MPV did not differ between groups (median 10.40 fL in both; $p = 0.355$), nor did lymphocyte count or platelet count. Both NLR and Braden score showed weak but statistically significant correlations with APACHE II score ($r = 0.090$ and $r = -0.126$, respectively; $p < 0.05$ for both), though the magnitude of these associations was too small to be clinically informative (Table 1C).

Table 1
C. Laboratory characteristics of patients

NLR	All patients (n = 941)	PI Present (n = 191)	PI Absent (n = 750)	p
	7.00 (3.20–13.56)	8.83 (3.94–16.16)	6.50 (3.07–12.90)	0.005
MPV (fL)	10.40 (9.70–11.20)	10.40 (9.75–11.30)	10.40 (9.70–11.10)	0.355
Neutrophil ($\times 10^3/\mu\text{L}$)	8.16 (5.45–12.51)	8.89 (5.93–14.23)	7.95 (5.34–12.27)	0.016
Lymphocyte ($\times 10^3/\mu\text{L}$)	1.21 (0.74–1.95)	1.03 (0.72–1.78)	1.25 (0.75–2.00)	0.119
Hemoglobin (g/dL)	11.50 (9.80–13.30)	10.80 (9.30–12.90)	11.70 (10.00–13.30)	0.005
Platelet ($\times 10^3/\mu\text{L}$)	231.0 (172.0–295.0)	235.0 (166.5–319.0)	231.0 (174.0–292.0)	0.560

Values are expressed as median (IQR). Mann-Whitney U test was used for all comparisons. PI: pressure injury; NLR: neutrophil-to-lymphocyte ratio; MPV: mean platelet volume. Bold p values indicate statistical significance ($p < 0.05$).

Among the 191 patients who developed pressure injuries, the median time to onset was 9 days (IQR 3–23). The most common localisation was the sacrum, accounting for 144 patients (75.4%), followed by the heel in 17 patients (8.9%) and the combined sacrum and heel region in 11 patients (5.8%). With respect to injury stage, 73 patients (38.2%) had Stage 1, 93 (48.7%) had Stage 2, 21 (11.0%) had Stage 3, and 1 (0.5%) had Stage 4 injury.

ROC curve analysis was performed to evaluate the ability of NLR, MPV, and Braden score to discriminate between patients who did and did not develop pressure injuries (Fig. 2). The Braden score demonstrated the strongest discriminative performance, with an AUC of 0.785 (95% CI 0.751–0.816), an optimal cut-off of ≤ 13 , a sensitivity of 76.4%, and a specificity of 70.5%. NLR yielded a modest AUC of 0.566 (95% CI 0.519–0.612), with an optimal cut-off of ≥ 6.99 , a sensitivity of 60.7%, and a specificity of 52.4%. MPV showed no meaningful discriminative ability, with an AUC of 0.522 (95% CI 0.474–0.569). The difference in AUC between NLR and MPV was not statistically significant ($\Delta\text{AUC} = 0.045$, 95% CI -0.014 – 0.104 ; $p = 0.503$). The distribution of NLR and MPV values across both groups is illustrated in Fig. 3, and the corresponding numerical results are detailed in Table 2.

Receiver operating characteristic (ROC) curves for the neutrophil-to-lymphocyte ratio (NLR), mean platelet volume (MPV), and Braden score in predicting pressure injury development. AUC values with 95% confidence intervals (bootstrap, 2000 iterations): NLR = 0.566 (0.519–0.612); MPV = 0.522 (0.474–0.569); Braden score = 0.785 (0.751–0.816). The diagonal dashed line represents chance-level discrimination (AUC = 0.50). AUC: area under the curve; NLR: neutrophil-to-lymphocyte ratio; MPV: mean platelet volume.

Box plots showing the distribution of the neutrophil-to-lymphocyte ratio (NLR) and mean platelet volume (MPV) in patients with and without pressure injury. Boxes represent the interquartile range (IQR; 25th–75th percentiles); horizontal lines within boxes indicate medians; whiskers extend to 1.5×IQR; circles represent outliers. NLR was significantly higher in patients who developed pressure injuries (median 8.83 vs. 6.50; $p = 0.005$, Mann–Whitney U test). MPV did not differ significantly between groups (median 10.40 vs. 10.40 fL; $p = 0.355$). NLR: neutrophil-to-lymphocyte ratio; MPV: mean platelet volume; PI: pressure injury; n.s.: not significant.

Table 2

ROC curve analysis for NLR, MPV, and Braden score in predicting pressure injury development

NLR	AUC (95% CI)	Optimal cutoff	Sensitivity	Specificity	PPV	NPV	Accuracy
	0.566 (0.519–0.612)	≥ 6.99	60.7%	52.4%	24.5%	84.0%	54.1%
MPV	0.522 (0.474–0.569)	≥ 10.70 fL	44.5%	61.1%	22.5%	81.2%	57.7%
Braden score	0.785 (0.751–0.816)	≤ 13	76.4%	70.5%	39.8%	92.2%	71.7%

Optimal cut-off points were determined using the Youden index. 95% CI was calculated by the bootstrap method (2000 iterations). The difference in AUC between NLR and MPV was not statistically significant ($\Delta\text{AUC} = 0.045$, 95% CI – 0.014–0.104, $p = 0.503$). AUC: area under the curve; PPV: positive predictive value; NPV: negative predictive value; CI: confidence interval; NLR: neutrophil-to-lymphocyte ratio; MPV: mean platelet volume.

In univariate logistic regression analysis, several variables were significantly associated with pressure injury development, including age, ICU length of stay, APACHE II score, Braden score, hemoglobin, female sex, cerebrovascular disease, sepsis, mechanical ventilation, vasopressor use, coronary artery disease, smoking history, psychiatric diagnosis, and weaning. NLR (OR 1.006, 95% CI 0.998–1.013; $p = 0.146$) and MPV (OR 0.991, 95% CI 0.946–1.038; $p = 0.689$) were not significantly associated with pressure injury development and were therefore not entered into the multivariate model. In the multivariate analysis, three variables emerged as independent predictors of pressure injury development: longer ICU length of stay (aOR 1.074, 95% CI 1.058–1.089; $p < 0.001$), lower Braden score (aOR 0.751, 95% CI 0.681–0.828; $p < 0.001$), and lower hemoglobin level (aOR 0.873, 95% CI 0.798–0.956; $p = 0.003$). Coronary artery disease was independently associated with a lower likelihood of pressure injury development (aOR 0.457, 95% CI 0.275–0.758; $p = 0.002$). The model demonstrated good overall fit (Hosmer–Lemeshow $p = 0.187$) and strong discriminative ability (c-statistic = 0.917), with a Nagelkerke R^2 of 0.565 (Table 3).

Table 3
Factors predicting pressure injury development — logistic regression analysis (n = 941)

Age	Univariate OR (95% CI)	p	Multivariate aOR (95% CI)	p
	1.039 (1.026–1.052)	< 0.001	1.013 (0.995–1.032)	0.165
ICU length of stay	1.079 (1.065–1.094)	< 0.001	1.074 (1.058–1.089)	< 0.001
APACHE II	1.057 (1.038–1.076)	< 0.001	1.017 (0.990–1.046)	0.219
Braden score	0.690 (0.645–0.739)	< 0.001	0.751 (0.681–0.828)	< 0.001
NLR	1.006 (0.998–1.013)	0.146	—	—
MPV	0.991 (0.946–1.038)	0.689	—	—
Hemoglobin	0.911 (0.854–0.972)	0.005	0.873 (0.798–0.956)	0.003
Sex (Female)	1.443 (1.049–1.985)	0.024	1.587 (0.989–2.545)	0.055
Cerebrovascular disease	3.237 (2.220–4.720)	< 0.001	1.379 (0.805–2.363)	0.242
Sepsis	2.880 (2.078–3.991)	< 0.001	1.430 (0.889–2.299)	0.141
Mechanical ventilation	3.069 (2.184–4.311)	< 0.001	0.610 (0.356–1.047)	0.073
Vasopressor	4.996 (3.535–7.060)	< 0.001	1.561 (0.928–2.625)	0.093
Coronary artery disease	0.671 (0.471–0.955)	0.027	0.457 (0.275–0.758)	0.002
Smoking	0.604 (0.391–0.935)	0.024	0.879 (0.471–1.642)	0.686
Psychiatric diagnosis	1.661 (1.022–2.699)	0.041	0.972 (0.476–1.986)	0.938
Weaning	2.218 (1.078–4.567)	0.031	1.632 (0.533–5.001)	0.391

Variables with $p < 0.10$ in univariate analysis were entered into the multivariate model. NLR and MPV were not included in the multivariate model (univariate $p > 0.10$). Variance inflation factor (VIF) analysis excluded multicollinearity (all VIF < 1.7). Nagelkerke $R^2=0.565$; C-statistic = 0.917; Hosmer–Lemeshow $p = 0.187$. The reversal of mechanical ventilation from univariate OR = 3.069 to multivariate aOR = 0.610 reflects confounding by ICU length of stay ($rs = 0.222$), Braden score ($rs = -0.449$), and vasopressor use ($rs = 0.494$), and does not represent a data error. Bold values indicate statistical significance ($p < 0.05$). OR: odds ratio; aOR: adjusted odds ratio; CI: confidence interval.

Kruskal–Wallis analysis revealed no significant differences in NLR, MPV, or Braden score across pressure injury Stages 1 through 3 ($p = 0.527$, $p = 0.469$, and $p = 0.233$, respectively), and post-hoc

pairwise comparisons with Bonferroni correction confirmed that no statistically significant differences existed between any two stages for any of these parameters (Table 4).

Table 4
NLR, MPV, and Braden score according to pressure injury stage

NLR	Stage 1 (n = 73)	Stage 2 (n = 93)	Stage 3 (n = 21)	Stage 4 (n = 1)*	p
	9.66 (5.38–15.36)	9.20 (3.88–16.17)	6.99 (3.23–17.70)	9.16	0.527
MPV (fL)	10.50 (9.90–11.40)	10.40 (9.50–11.30)	10.60 (10.20–11.20)	9.50	0.469
Braden score	12.0 (11.0–13.0)	12.0 (10.0–13.0)	11.0 (11.0–13.0)	8.0	0.233

Values are expressed as median (IQR). Kruskal-Wallis test was applied for Stages 1–3. Post-hoc pairwise Mann-Whitney U with Bonferroni correction: all comparisons non-significant. *Stage 4 (n = 1) was excluded from statistical analysis and presented descriptively only. NLR: neutrophil-to-lymphocyte ratio; MPV: mean platelet volume; IQR: interquartile range.

ICU mortality was markedly higher among patients who developed pressure injuries compared to those who did not (83.8% vs. 30.7%; $p < 0.001$), with an odds ratio of 11.67 (95% CI 7.71–17.67). The Kaplan–Meier curves illustrated a significantly lower survival probability in the pressure injury group throughout the ICU stay (log-rank $p < 0.001$), with a median ICU length of stay of 39 days in the pressure injury group versus 16 days in those without pressure injury (Fig. 4). Within the pressure injury group, NLR and MPV did not differ significantly between survivors and non-survivors ($p = 0.151$ and $p = 0.140$, respectively). Across the entire cohort, however, both markers were significantly elevated in patients who died during ICU admission compared to survivors: NLR 9.16 (IQR 4.01–17.16) vs. 5.91 (IQR 2.82–11.41), $p < 0.001$; and MPV 10.60 (IQR 9.90–11.30) vs. 10.30 (IQR 9.70–11.05), $p < 0.001$.

Kaplan–Meier curves showing ICU survival according to pressure injury status. Patients who developed pressure injuries had significantly lower survival compared to those who did not (log-rank $p < 0.001$). Median ICU length of stay: 39 days in the PI present group vs. 16 days in the PI absent group. Shaded areas represent 95% confidence intervals. PI: pressure injury; ICU: intensive care unit.

To examine whether the association between NLR and pressure injury development was confounded by baseline differences in illness severity, a PSM analysis was conducted. Following 1:1 nearest-neighbour matching on age, APACHE II score, Braden score, sepsis, mechanical ventilation, vasopressor use, sex, and cerebrovascular disease, 170 matched pairs were identified (n = 340). Adequate covariate balance was achieved across all matching variables, with SMD values below 0.10 for each (range 0.013–0.075). In the matched cohort, NLR did not differ significantly between patients who developed pressure injuries and those who did not (median 8.74, IQR 3.89–16.21 vs. 8.29, IQR 3.65–16.54; $p = 0.759$), nor did MPV (median 10.40, IQR 9.60–11.30 vs. 10.50, IQR 9.90–11.20; $p = 0.620$). Logistic regression in the matched

cohort confirmed that neither marker was associated with pressure injury development: NLR OR = 1.000 (95% CI 0.989–1.010; $p = 0.943$), MPV OR = 0.936 (95% CI 0.782–1.121; $p = 0.473$) (Table 5).

Table 5

Propensity score matching – covariate balance and NLR/MPV comparison in matched cohort (n = 340)

	PI Present (n = 170)	PI Absent (n = 170)	SMD	p
Covariate balance				
Age (years)	—	—	0.075	0.766
APACHE II score	—	—	0.040	0.490
Braden score	—	—	0.013	0.872
Sepsis, n (%)	—	—	0.012	1.000
Mechanical ventilation, n (%)	—	—	0.022	0.596
Vasopressor use, n (%)	—	—	0.064	0.637
Sex (Female), n (%)	—	—	0.070	0.588
Cerebrovascular disease, n (%)	—	—	0.054	0.710
Outcome markers – matched cohort				
NLR	8.74 (3.89–16.21)	8.29 (3.65–16.54)	—	0.759
MPV (fL)	10.40 (9.60–11.30)	10.50 (9.90–11.20)	—	0.620

SMD: standardised mean difference; values < 0.10 indicate adequate covariate balance. Continuous variables expressed as median (IQR); Mann-Whitney U test. Categorical variables: Chi-square test. Logistic regression in matched cohort: NLR OR = 1.000 (95% CI 0.989–1.010; $p = 0.943$); MPV OR = 0.936 (95% CI 0.782–1.121; $p = 0.473$). PI: pressure injury; SMD: standardised mean difference; NLR: neutrophil-to-lymphocyte ratio; MPV: mean platelet volume; IQR: interquartile range.

In the overall cohort, 230 patients died during ICU admission without having developed a pressure injury, representing a substantial competing risk for the outcome of interest. Using the Aalen-Johansen estimator, the cumulative incidence of pressure injury development, accounting for death as a competing event, was 21.2% at day 30 and 33.6% at day 60. In cause-specific Cox regression treating in-ICU death as a competing event, NLR showed a borderline association with time to pressure injury development (HR = 1.154, 95% CI 1.016–1.310; $p = 0.028$), while MPV did not reach statistical significance (HR = 0.630, 95% CI 0.347–1.145; $p = 0.130$) (Table 6).

Table 6
Cause-specific Cox regression – NLR and MPV
with in-ICU death as competing event

NLR (per SD)	HR (95% CI)	p
	1.154 (1.016–1.310)	0.028
MPV (per SD)	0.630 (0.347–1.145)	0.130

Adjusted for age, APACHE II score, Braden score, sepsis, mechanical ventilation, and vasopressor use. Variables are standardised (per 1 SD increase). Cumulative incidence of pressure injury development (Aalen–Johansen estimator): 21.2% at day 30 and 33.6% at day 60. HR: hazard ratio; CI: confidence interval; NLR: neutrophil-to-lymphocyte ratio; MPV: mean platelet volume; SD: standard deviation.

In the sepsis subgroup (n = 303, PI incidence 32.7%), NLR did not differ between patients who did and did not develop pressure injuries (median 10.07, IQR 5.47–18.14 vs. 10.20, IQR 5.05–19.88; p = 0.934), and its discriminative ability was negligible (AUC = 0.503, 95% CI 0.438–0.571). MPV was similarly non-significant in this subgroup (p = 0.115, AUC = 0.444). In the non-sepsis subgroup (n = 638, PI incidence 14.4%), NLR remained non-significant (p = 0.127, AUC = 0.550), whereas MPV showed a borderline positive association with pressure injury development (OR = 1.264, 95% CI 1.025–1.557; p = 0.028, AUC = 0.560). Formal interaction testing confirmed a statistically significant MPV × sepsis interaction (p = 0.008), indicating that the relationship between MPV and pressure injury risk differs according to sepsis status. No significant NLR × sepsis interaction was identified (p = 0.242) (Fig. 5).

Grouped bar charts showing median NLR and MPV values in patients with and without pressure injuries (PI), presented for the overall cohort and stratified by sepsis status. Bars represent median values; error bars represent the interquartile range (IQR). Median values are shown within each bar. Significance brackets indicate p values from the Mann–Whitney U test; n.s.: not significant. A statistically significant MPV × Sepsis interaction was identified (interaction p = 0.008), indicating that the association between MPV and pressure injury risk differs according to sepsis status. No significant NLR × Sepsis interaction was observed (interaction p = 0.242). NLR: neutrophil-to-lymphocyte ratio; MPV: mean platelet volume; PI: pressure injury; IQR: interquartile range.

DISCUSSION

This study investigated the predictive value of admission NLR and MPV for pressure injury development in a cohort of 941 critically ill patients, representing one of the larger single-centre datasets examined for this purpose in the literature. The overall PI incidence was 20.3%, which is consistent with the range reported across international studies. The landmark DecubICUs study, the largest global epidemiological survey of PI in the ICU setting, identified an ICU-acquired prevalence of 16.2% across 1,117 units in 90 countries (2). The somewhat higher rate observed in our cohort likely reflects the clinical complexity of our patient population, characterised by high rates of sepsis (51.8%), vasopressor dependence (70.2%), and prolonged mechanical ventilation.

The primary finding of this study is that while NLR was significantly higher in patients who developed pressure injuries, it did not emerge as an independent predictor in multivariate analysis and demonstrated only modest discriminative ability (AUC = 0.566). MPV showed no significant association with PI development at any analytical level. These results partially diverge from the findings of Adiyeké and Adiyeké, who reported that both NLR and MPV were independent predictors of PI development in a cohort of 104 ICU patients (10). Similarly, Çelik et al., in a study of 158 patients who had already developed pressure injuries, observed that MPV was significantly higher in more advanced PI stages (11). The discrepancy between these earlier findings and ours is most plausibly explained by sample size. With 941 patients, nearly nine times the size of the Adiyeké cohort and six times that of the Çelik cohort, our study had substantially greater statistical power to detect the true, and apparently limited, predictive value of these markers. In smaller studies, elevated NLR and MPV may appear to track PI risk simply because they mirror the same underlying severity of illness that predisposes patients to PI, rather than representing independent biological risk factors. The propensity score matching analysis in our study provides the most direct evidence for this interpretation. After matching patients on age, APACHE II score, Braden score, sepsis, mechanical ventilation, vasopressor use, sex, and cerebrovascular disease, the apparent difference in NLR between groups disappeared entirely ($p = 0.759$), and logistic regression in the matched cohort confirmed the absence of any meaningful association (OR = 1.000; $p = 0.943$). This finding strongly supports the conclusion that NLR elevation in PI patients reflects confounding by illness severity rather than an independent pathophysiological link to pressure injury development.

The biological rationale for investigating NLR in the context of PI is well established. NLR reflects the balance between neutrophilia, driven by innate immune activation, and lymphopenia, driven by adaptive immune suppression, a combination that is closely associated with overall disease burden in severe critical illness (9). Elevated NLR has been associated with adverse outcomes across a wide range of critical care conditions, including sepsis, acute respiratory failure, and trauma (9). However, its utility as a clinical tool depends fundamentally on whether it provides additive predictive information beyond what is already captured by existing risk stratification instruments, and in the context of PI, our data suggest it does not. The NLR appears to function as a general marker of critical illness burden rather than a specific indicator of PI susceptibility.

The biological rationale for MPV is similarly coherent in principle. Platelets are not merely effectors of hemostasis; they actively participate in wound healing through the release of growth factors, cytokines, and pro-inflammatory mediators (7). Larger platelets, as reflected by a higher MPV, are metabolically more active and have been proposed to drive greater inflammatory and prothrombotic signalling (7). An increase in MPV during ICU admission has been associated with higher in-hospital mortality, consistent with its role as a marker of platelet stress and systemic inflammation (8). In the context of PI, one might hypothesise that heightened platelet activation perpetuates the local inflammatory cascade that impairs tissue tolerance to pressure. Our data do not support this hypothesis, however. MPV at admission showed no significant difference between patients who did and did not develop PI ($p = 0.355$), nor did it differ across injury stages ($p = 0.469$). A plausible explanation is that MPV is a relatively non-specific and

temporally unstable marker; a single measurement at admission may not capture the dynamic platelet changes that occur throughout the course of critical illness (8). Serial MPV assessments over the first several days of ICU admission may be necessary to reveal any clinically meaningful signal, and this remains an area warranting prospective investigation.

This interpretation is further supported by the ROC analysis. An AUC of 0.566 for NLR is only marginally above chance-level discrimination and falls well below the threshold conventionally considered clinically useful ($AUC > 0.70$). For MPV, an AUC of 0.522 renders this marker effectively non-discriminative. The difference in AUC between the two markers was itself non-significant ($\Delta AUC = 0.045$, $p = 0.503$), confirming that neither offers a meaningful advantage over the other. Taken together, these findings suggest that hematological inflammation indices, as measured at a single time point at ICU admission, cannot reliably identify patients destined to develop pressure injuries.

The high in-ICU mortality observed in this cohort raises an important methodological consideration. Patients who die during their ICU stay cannot subsequently develop a pressure injury, and standard Kaplan-Meier analysis does not account for this competing risk. Using the Aalen-Johansen estimator, the cumulative incidence of pressure injury development was 21.2% at day 30 and 33.6% at day 60, values somewhat higher than the crude incidence of 20.3%, reflecting the fact that mortality removes a proportion of patients from the risk set before they can develop PI. In cause-specific Cox regression incorporating death as a competing event, NLR showed a borderline positive association with time to pressure injury development ($HR = 1.154$; $p = 0.028$), while MPV did not reach significance ($p = 0.130$). This borderline finding for NLR warrants cautious interpretation: given that NLR is independently associated with mortality in critically ill patients, it may partially reflect the inflammatory milieu that accelerates both clinical deterioration and skin breakdown, rather than exerting a direct effect on PI susceptibility. This result should not be interpreted as evidence for clinical utility, particularly in light of its absence of significance in the propensity-matched analysis and the overall weak discriminative ability demonstrated in ROC analysis (12, 13).

The subgroup analysis by sepsis status yielded a finding of particular interest. In septic patients, NLR showed no relationship whatsoever with pressure injury development ($AUC = 0.503$; $p = 0.934$), consistent with the well-established observation that sepsis uniformly drives NLR elevation regardless of other clinical outcomes, effectively eliminating any discriminative signal it might carry for PI risk (14). In non-septic patients, NLR remained non-significant, but MPV showed a borderline positive association with PI development ($OR = 1.264$; $p = 0.028$), and the $MPV \times$ sepsis interaction term was statistically significant ($p = 0.008$). This interaction suggests that the relationship between MPV and PI risk may be modified by sepsis status: in the absence of the dominant platelet-activating stimulus of sepsis, MPV may more faithfully reflect the localised inflammatory processes involved in PI pathogenesis. While this finding is exploratory and should be interpreted with caution given the multiplicity of comparisons and the modest effect size, it may point towards a more selective role for MPV in non-septic ICU populations and warrants prospective investigation.

In contrast, the Braden score demonstrated substantially stronger discriminative ability (AUC = 0.785), a negative predictive value of 92.2% at the optimal cut-off of ≤ 13 , and remained an independent predictor in the multivariate model. This confirms the continued clinical value of the Braden scale as the primary bedside risk stratification tool in the ICU. A meta-analysis of the Braden scale's predictive validity in ICU populations reported a pooled AUC of approximately 0.78, with high sensitivity but limited specificity, a profile consistent with our findings (15). A recent systematic review cautioned that the psychometric properties of the Braden scale in intensive care are variable, and that it should be applied as part of a broader clinical assessment rather than as a standalone instrument (4). The weak but statistically significant negative correlations between both NLR and Braden score ($r_s = -0.126$) and MPV and Braden score ($r_s = -0.098$) are consistent with the idea that systemic inflammation contributes to lower functional reserve and greater vulnerability to skin injury, yet these correlations are too small to be clinically actionable (6). NLR and MPV therefore appear to capture the same dimension of illness severity as the Braden score, rather than complementing it.

Among the independent predictors identified in the multivariate model, ICU length of stay emerged as the strongest (aOR 1.074 per day, $p < 0.001$), consistent with extensive prior literature. An updated systematic review of PI risk factors in critical care identified longer ICU stay as one of the most consistently reported independent predictors across high-quality studies (3). The DecubICUs data showed that patients with ICU stays exceeding 12 days faced a 7.5-fold increase in PI risk compared to those staying three days or fewer (2). The median time to PI onset of 9 days (IQR 3–23) observed in our cohort defines a meaningful preventive window and suggests that the first two weeks of ICU admission represent the period during which intensification of skin care, repositioning vigilance, and risk reassessment may yield the greatest benefit.

The independent association of lower hemoglobin with PI development (aOR 0.873, $p = 0.003$) deserves clinical attention. Low hemoglobin impairs peripheral oxygen delivery, diminishes the metabolic capacity of skin and soft tissue to withstand mechanical loading, and has been increasingly recognised as a modifiable PI risk factor (16). An overview of reviews on hospital-acquired PI risk factors highlighted hemoglobin level as one of the consistently identified contributors to PI development, alongside vasopressor use and ICU length of stay (16). A multicentre observational study similarly demonstrated that higher hemoglobin levels were inversely associated with both PI development and PI stage severity (17). Taken together, these findings suggest that anemia management, including appropriate recognition of transfusion thresholds and optimisation of erythropoietic support, should be considered an integral component of PI prevention in critically ill patients.

The independent predictive value of serum biomarkers in PI risk assessment has been an active area of inquiry. It has been shown that a combination of serum biomarkers measured at ICU admission, including white blood cell count and electrolyte levels, can achieve an AUC of 0.910 in a predictive model for PI among patients with Braden scores ≤ 9 (5). These findings suggest that laboratory-based predictors, when carefully selected and combined with clinical risk scores, may augment bedside PI risk

stratification. Our data indicate, however, that NLR and MPV in isolation do not reach the discriminative threshold required for clinical utility in an unselected ICU population.

The apparent protective association of coronary artery disease with PI development (aOR 0.457, $p = 0.002$) warrants careful interpretation. This counterintuitive finding is unlikely to reflect a true biological mechanism. A more plausible explanation involves survivorship bias: patients with established coronary artery disease who survive long enough to be hospitalised in an ICU may represent a selected subgroup receiving closer cardiovascular monitoring, more structured nursing care, and more active management overall. Alternatively, patients with severe coronary disease may experience earlier in-ICU mortality before a PI can develop, paradoxically reducing the observed PI rate in this subgroup. This association should not be interpreted as clinically protective and merits further investigation in prospective cohort studies.

Regarding PI localisation, the sacrum accounted for 75.4% of cases, followed by the heel (8.9%), a distribution consistent with global data, in which the sacrum and heels are similarly the most commonly affected sites (2). The numerically higher NLR observed in patients with combined sacral and heel involvement (17.8 vs. 8.8 in sacrum-only cases) may suggest a more pronounced systemic inflammatory burden in those with multi-site injury; the small number of patients in this subgroup, however, precludes any definitive statistical inference.

The mortality findings of this study are clinically striking. PI was associated with an 11.67-fold increase in the odds of in-ICU death (95% CI 7.71–17.67), with a mortality rate of 83.8% in the PI group versus 30.7% in those who did not develop PI. A previous large-scale study reported a more modest but statistically significant association between PI and hospital mortality, with odds ratios ranging from 1.5 for Stage I to 2.8 for Stage III or worse (2). The substantially higher odds ratio in our cohort most likely reflects the overall severity of illness in our population, in whom PI may function more as a marker of irreversible physiological deterioration than as an independent driver of death. This interpretation is reinforced by our finding that, within the PI group itself, NLR and MPV did not differ significantly between survivors and non-survivors ($p = 0.151$ and $p = 0.140$, respectively). Across the entire cohort, however, both markers were significantly elevated in patients who died, underscoring their broader association with critical illness severity rather than PI-specific pathophysiology.

Limitations

Several limitations of this study should be acknowledged. Its retrospective, single-centre design limits both causal inference and the generalisability of findings to other ICU populations and care contexts. NLR and MPV were measured only at the time of admission, and the dynamic trajectory of these markers throughout the ICU stay, which may be more informative than a single admission value, was not captured. Evidence from other critical care contexts suggests that the predictive utility of NLR may be substantially greater when assessed as a dynamic change over the first 24 to 48 hours of ICU admission rather than as a static single measurement (18). Nutritional status was not formally quantified using

validated indices or markers such as albumin or prealbumin, despite nutrition being a well-established determinant of both PI risk and tissue repair capacity. Adherence to the preventive care protocol, including repositioning frequency, skin assessment, and barrier cream application, was not systematically documented, introducing the possibility of unmeasured variation in care delivery. Finally, the high degree of diagnostic heterogeneity in our cohort, with over 60 distinct admission diagnoses, may limit the applicability of our findings to more homogeneous patient populations. Future prospective, multicentre studies incorporating serial hematological measurements, validated nutritional assessment, and standardised documentation of prevention practices are needed to more definitively characterise the role of inflammatory biomarkers in PI risk stratification.

CONCLUSION

This study demonstrated that while NLR was significantly elevated in ICU patients who developed pressure injuries, propensity score matching confirmed that this association was entirely explained by confounding from underlying illness severity. Neither NLR nor MPV emerged as independent predictors in multivariate analysis, and both showed insufficient discriminative ability in ROC analysis. Competing risk analysis yielded no clinically actionable signal for MPV, and only a borderline association for NLR that warrants cautious interpretation. A significant MPV $\times$ sepsis interaction was identified, suggesting that the relationship between MPV and PI risk may differ according to sepsis status and merits prospective investigation in non-septic populations.

The Braden score retained substantially stronger predictive performance and remains the most clinically relevant bedside tool for PI risk stratification. ICU length of stay, admission Braden score, and hemoglobin level were the most clinically actionable independent predictors, highlighting the importance of prolonged immobility, baseline risk assessment, and anemia management in PI prevention. The markedly elevated mortality associated with PI development reinforces the need for early and sustained preventive strategies in critically ill patients. Future prospective studies incorporating serial hematological measurements and stratification by sepsis status are warranted to more definitively characterise the role of inflammatory markers in this context.

Abbreviations

APACHE II: Acute Physiology and Chronic Health Evaluation II; AUC: area under the curve; CI: confidence interval; CIF: cumulative incidence function; COPD: chronic obstructive pulmonary disease; EPUAP: European Pressure Ulcer Advisory Panel; HR: hazard ratio; ICU: intensive care unit; IQR: interquartile range; MPV: mean platelet volume; MV: mechanical ventilation; NLR: neutrophil-to-lymphocyte ratio; NPIAP: National Pressure Injury Advisory Panel; NPV: negative predictive value; OR: odds ratio; aOR: adjusted odds ratio; PI: pressure injury; PPV: positive predictive value; PSM: propensity score matching; ROC: receiver operating characteristic; SMD: standardised mean difference; VIF: variance inflation factor.

Declarations

Ethics approval and consent to participate

This study was approved by the Zonguldak Bülent Ecevit University Non-Interventional Clinic Research Ethics Committee (Protocol No: 2026/02-09) and was conducted in accordance with the ethical principles of the Declaration of Helsinki. Given the retrospective nature of the study, the requirement for individual informed consent was waived by the ethics committee.

Consent for publication

Not applicable. This study does not contain any individual person's data in any form (including individual details, images, or videos).

Availability of data and materials

The datasets used and/or analysed 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

DFS: conceptualization, data collection, statistical analysis, writing – original draft, writing – review and editing. MGB: conceptualization, data collection, clinical interpretation, writing – review and editing. All authors read and approved the final manuscript.

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Figures

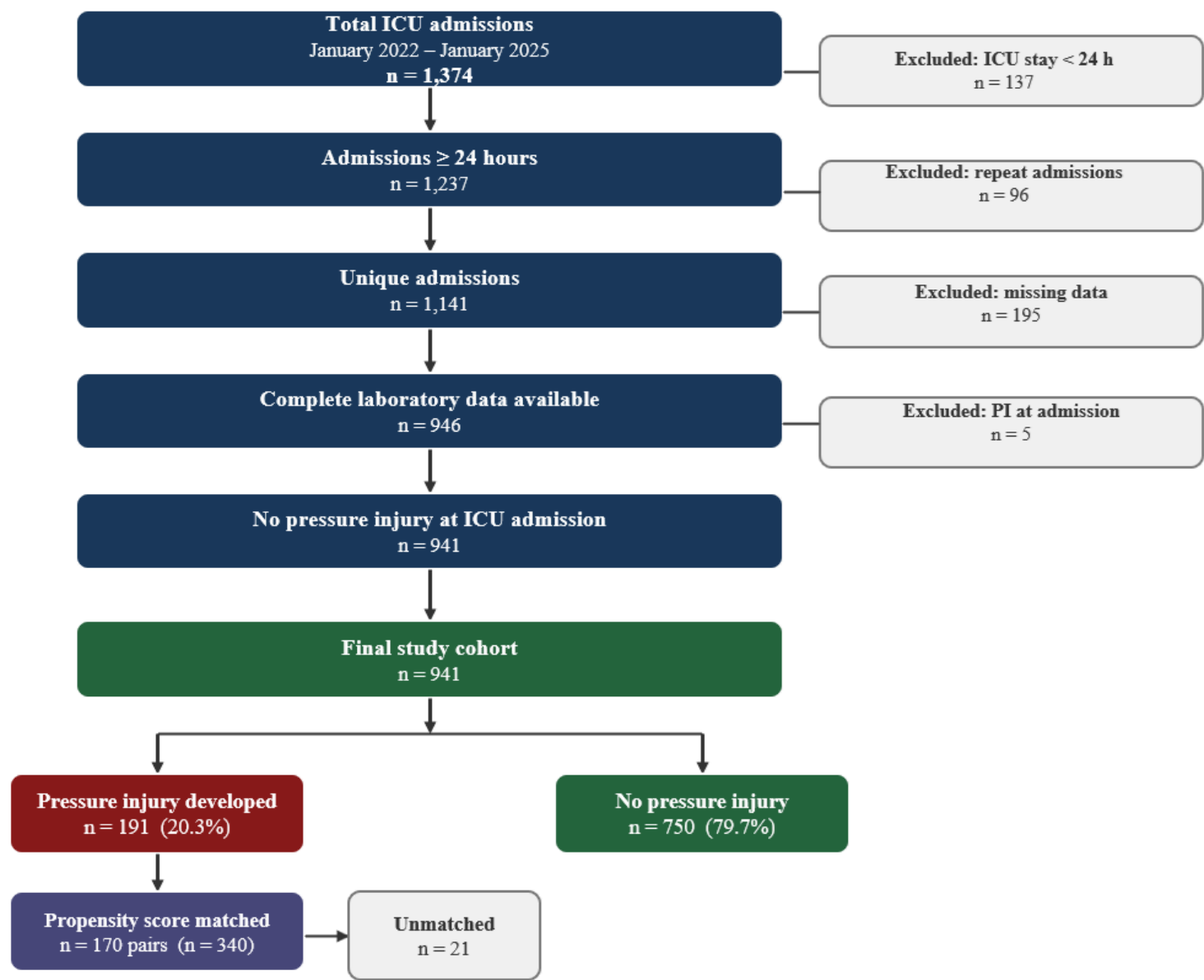


Figure 1

Flow diagram of patient selection and study enrollment

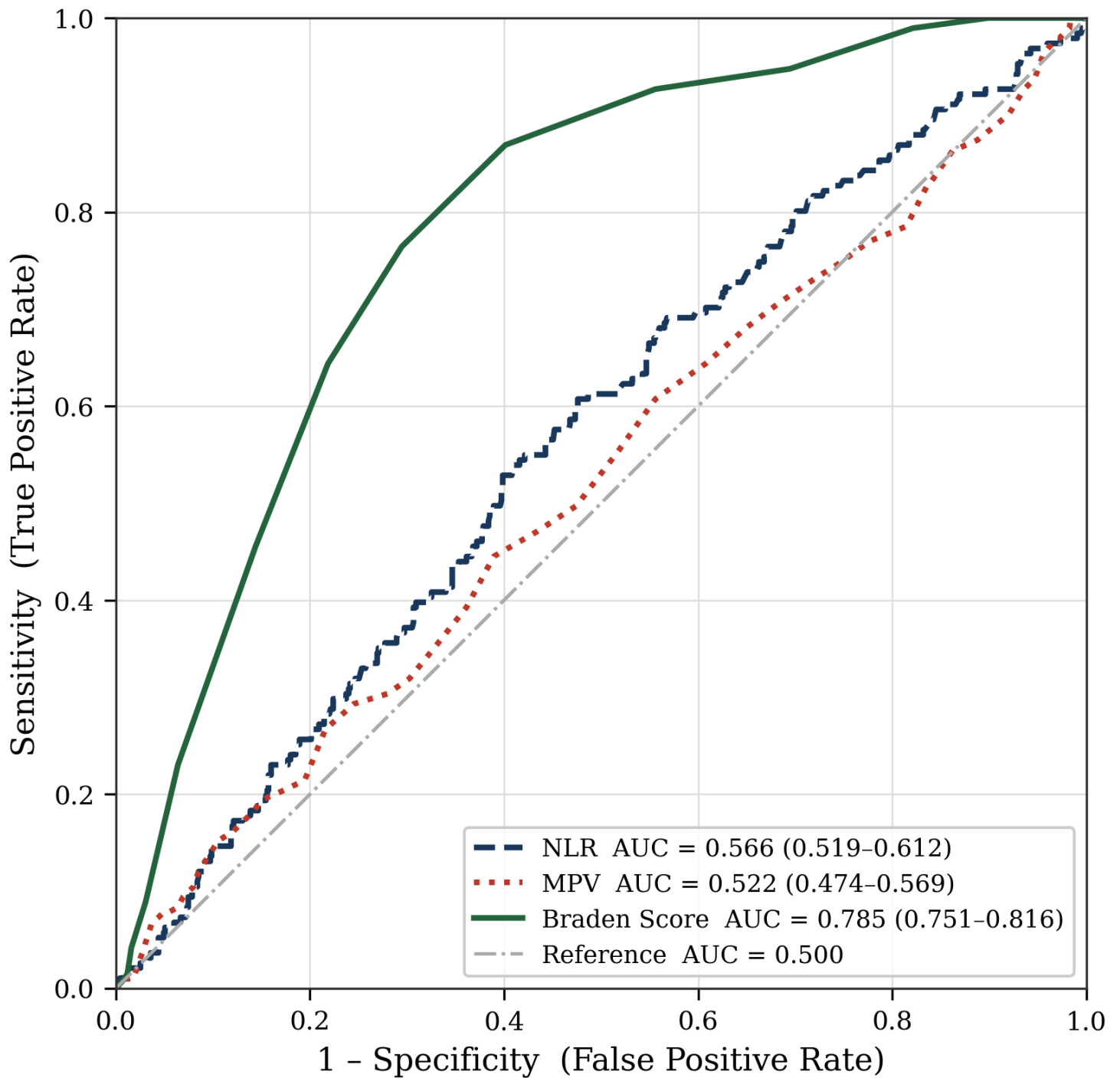


Figure 2

ROC curves for NLR, MPV, and Braden score in predicting pressure injury development

Receiver operating characteristic (ROC) curves for the neutrophil-to-lymphocyte ratio (NLR), mean platelet volume (MPV), and Braden score in predicting pressure injury development. AUC values with 95% confidence intervals (bootstrap, 2000 iterations): NLR = 0.566 (0.519–0.612); MPV = 0.522 (0.474–0.569); Braden score = 0.785 (0.751–0.816). The diagonal dashed line represents chance-level discrimination (AUC = 0.50). AUC: area under the curve; NLR: neutrophil-to-lymphocyte ratio; MPV: mean platelet volume.

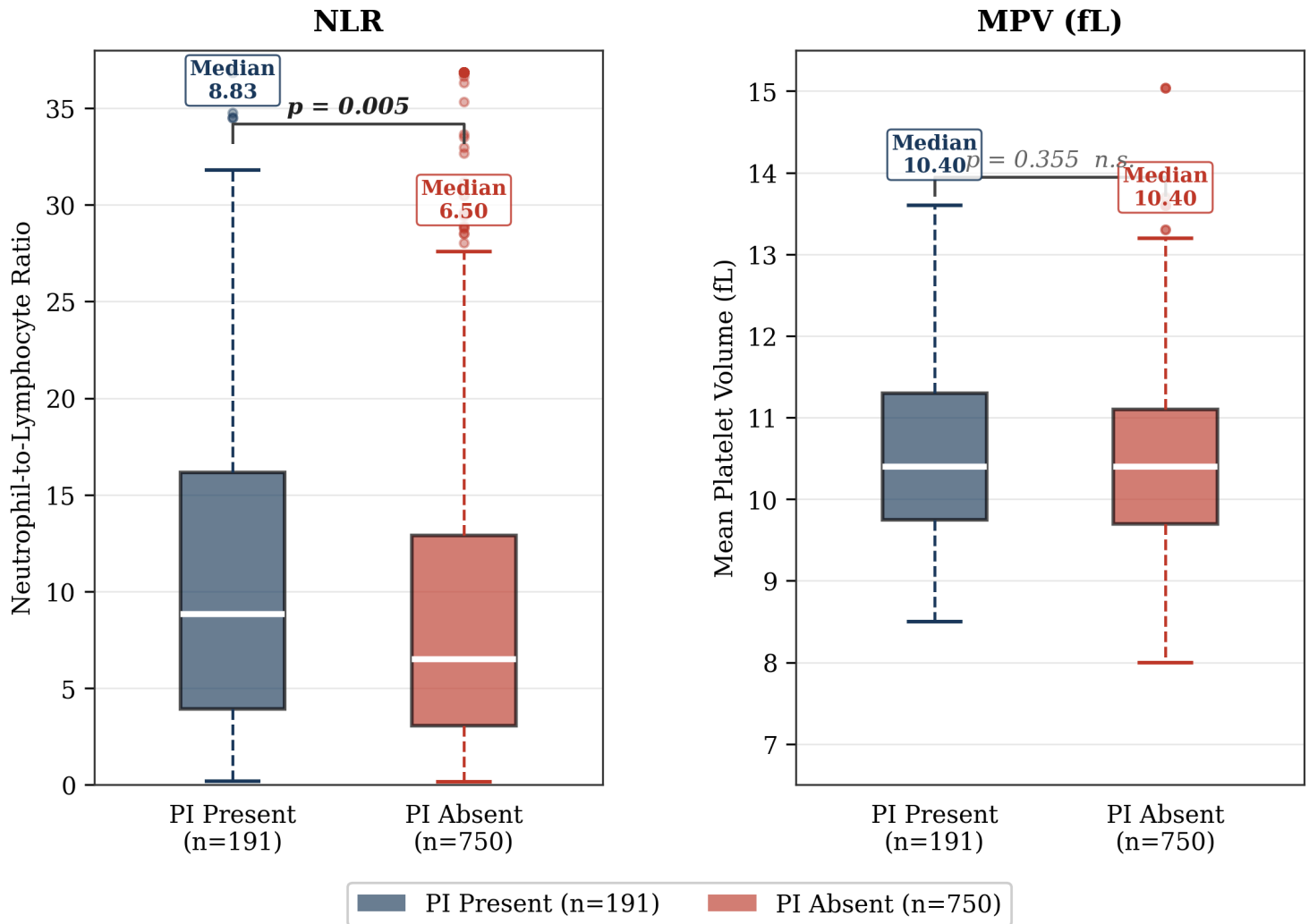


Figure 3

Distribution of NLR and MPV values according to pressure injury status

Box plots showing the distribution of the neutrophil-to-lymphocyte ratio (NLR) and mean platelet volume (MPV) in patients with and without pressure injury. Boxes represent the interquartile range (IQR; 25th–75th percentiles); horizontal lines within boxes indicate medians; whiskers extend to $1.5 \times \text{IQR}$; circles represent outliers. NLR was significantly higher in patients who developed pressure injuries (median 8.83 vs. 6.50; $p = 0.005$, Mann–Whitney U test). MPV did not differ significantly between groups (median 10.40 vs. 10.40 fL; $p = 0.355$). NLR: neutrophil-to-lymphocyte ratio; MPV: mean platelet volume; PI: pressure injury; n.s.: not significant.

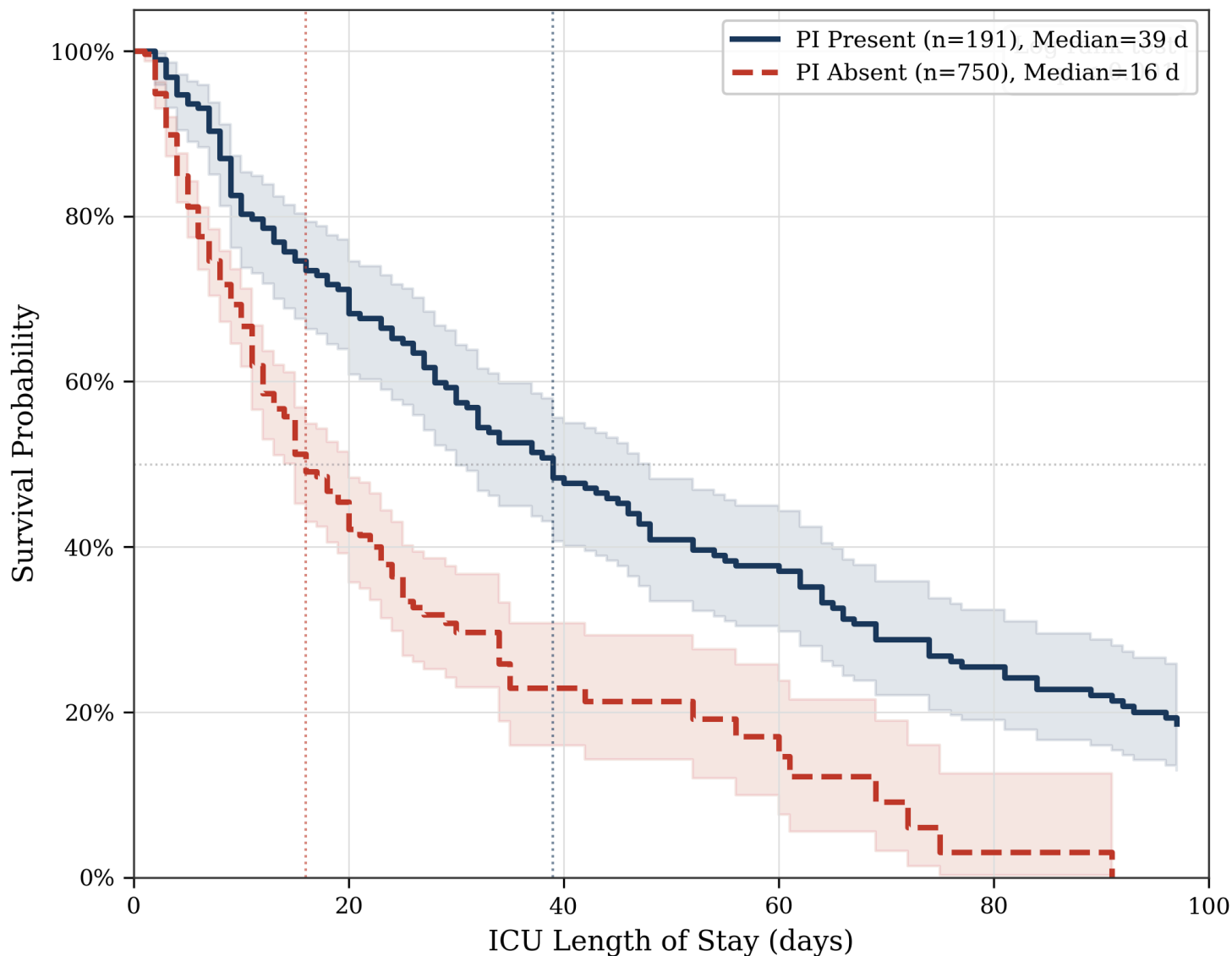
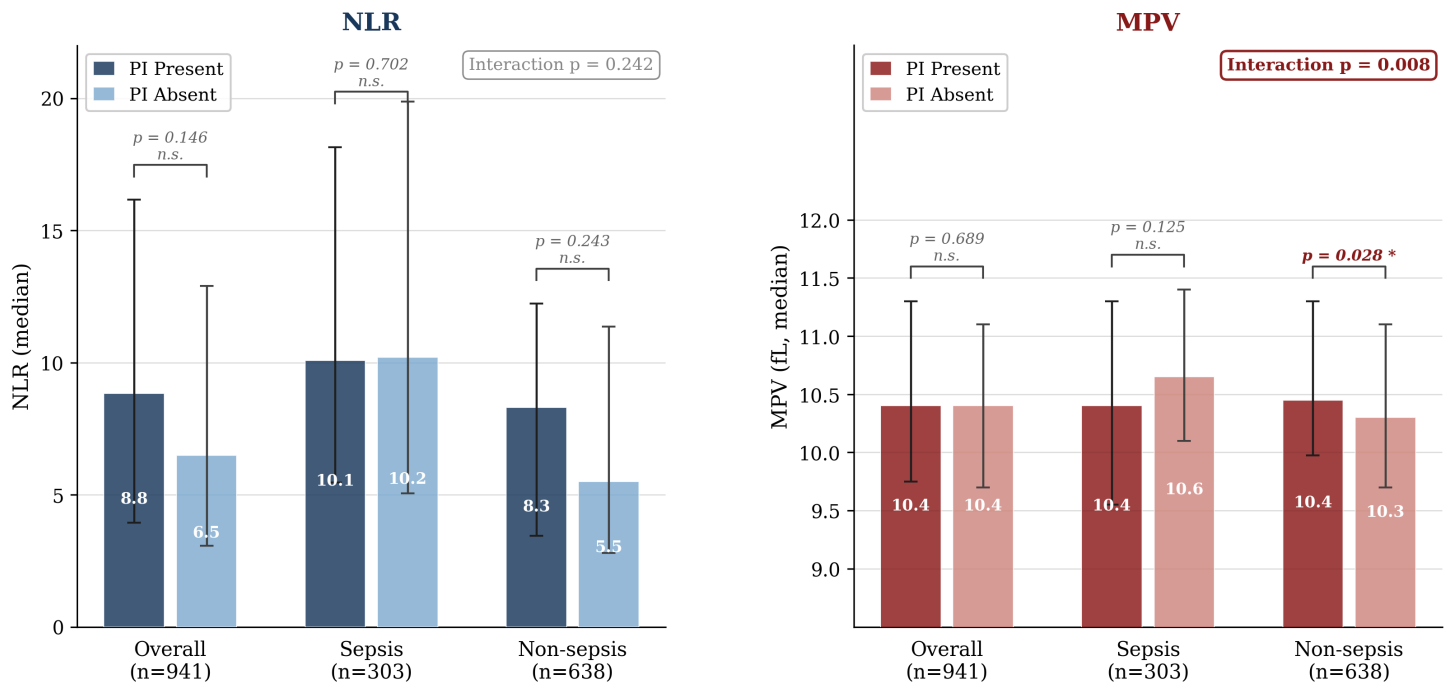


Figure 4

Kaplan–Meier survival curves according to pressure injury status

Kaplan–Meier curves showing ICU survival according to pressure injury status. Patients who developed pressure injuries had significantly lower survival compared to those who did not (log-rank $p < 0.001$). Median ICU length of stay: 39 days in the PI present group vs. 16 days in the PI absent group. Shaded areas represent 95% confidence intervals. PI: pressure injury; ICU: intensive care unit.



Bars show median values; error bars represent IQR. Significance brackets show Mann-Whitney U test results. Interaction p values from multiplicative logistic regression.

Figure 5

Median NLR and MPV values according to pressure injury status, stratified by sepsis subgroup

Grouped bar charts showing median NLR and MPV values in patients with and without pressure injuries (PI), presented for the overall cohort and stratified by sepsis status. Bars represent median values; error bars represent the interquartile range (IQR). Median values are shown within each bar. Significance brackets indicate p values from the Mann-Whitney U test; n.s.: not significant. A statistically significant MPV × Sepsis interaction was identified (interaction $p = 0.008$), indicating that the association between MPV and pressure injury risk differs according to sepsis status. No significant NLR × Sepsis interaction was observed (interaction $p = 0.242$). NLR: neutrophil-to-lymphocyte ratio; MPV: mean platelet volume; PI: pressure injury; IQR: interquartile range.