ORIGINAL ARTICLE

A different perspective in trauma patients: can pan-immune-inflammation value (PIV) predict mortality?

Gürkan Güneri,[1] Kevser Dilek Andıç,[2] Funda Çatan İnan,[3] Kadir Çorbacı[1]

[1]Department of General Surgery, Bilecik Şeyh Edebali University Faculty of Medicine, Bilecik-*Türkiye*

[2]Department of Anesthesia and Reanimation Bilecik Şeyh Edebali University, Faculty of Medicine, Bilecik-*Türkiye*

[3]Department of Biostatistics and Medical Informatics, Bilecik Şeyh Edebali University Faculty of Medicine, Bilecik-*Türkiye*

ABSTRACT

BACKGROUND: Trauma is a leading cause of mortality worldwide. Accurate prognostic assessment in emergency departments and intensive care units is essential for effective triage and management. Consequently, various prognostic markers have been explored in trauma populations. The pan-immune-inflammation (PIV) is a biomarker derived from a complete blood count (CBC) and can be rapidly obtained in clinical settings. This study aimed to evaluate the role of PIV in predicting the prognosis of trauma patients.

METHODS: This study examined patients admitted to a tertiary-level intensive care unit due to trauma at a training and research hospital. Established prognostic parameters, including the Revised Trauma Score (RTS), Glasgow Coma Scale (GCS), and Acute Physiology and Chronic Health Evaluation II (APACHE II) scores, were evaluated. PIV values were calculated from laboratory data. Mortality, morbidity, and length of hospital stay were retrospectively analyzed. The predictive value of PIV for mortality was assessed using statistical methods.

RESULTS: A total of 74 patients were included. The survivor group comprised seven females (11.5%) and 54 males (88.5%), while the non-survivor group included one female (7.7%) and 12 males (92.3%). PIV, RTS, GCS, and APACHE II scores were effective in predicting mortality ($p<0.001$). The cut-off value for PIV was 6367.5; patients with PIV values below this threshold had a higher risk of mortality compared to those with higher values.

CONCLUSION: Rapid and reliable prognostication is essential in emergency settings. PIV demonstrates predictive performance comparable to established prognostic scoring systems. Early assessment of PIV in trauma patients may support more effective triage and treatment planning.

Keywords: Mortality; pan-immune-inflammation value; prognosis; trauma.

INTRODUCTION

Trauma represents a major global health challenge due to its potential to cause long-term complications, permanent disability, and substantial healthcare costs. According to the World Health Organization (WHO), trauma accounts for 5.8 million deaths annually, representing 10% of all deaths and 16% of disabilities worldwide.[1] It is also one of the leading causes of mortality among young individuals, with nearly 80% of deaths in those aged 15–24 years attributed to trauma.[2] Although trauma-related mortality has declined in recent years due to advances in hemorrhage control and the management of coagulopathy, secondary immunological complications, such as hospital-acquired infections, sepsis, and multiple organ failure, remain a significant concern in this patient population. With the rising incidence of traffic accidents and interpersonal violence, trauma patients constitute a substantial proportion of intensive care unit (ICU) admissions,

Cite this article as: Güneri G, Andıç KD, İnan FÇ, Çorbacı K. A different perspective in trauma patients: can pan-immune-inflammation value (PIV) predict mortality? Ulus Travma Acil Cerrahi Derg 2026;32:558-566.

Address for correspondence: Gürkan Güneri

Department of General Surgery, Bilecik Şeyh Edebali University Faculty of Medicine, Bilecik, Türkiye

E-mail: gurkan.guneri@bilecik.edu.tr

Ulus Travma Acil Cerrahi Derg 2026;32(5):558-566 DOI: 10.14744/tjtes.2026.33746

Submitted: 23.12.2025 Revised: 13.03.2026 Accepted: 23.03.2026 Published: 05.05.2026

accounting for an estimated 10–15% of all ICU cases.[3] In low- and middle-income countries, emergency departments and ICUs are often overburdened, and the availability of advanced trauma centers is limited.[4] Therefore, reliable scoring systems are essential to ensure the efficient use of ICU resources, which are characterized by limited bed capacity and high treatment costs.

In high-acuity settings such as emergency departments, rapid assessment of large patient volumes is critical to identify life-threatening conditions and ensure timely transfer to the ICU or operating room when necessary. The ICU period represents a critical phase in trauma care, during which much of the early management is undertaken. Because the clinical status of patients with multiple trauma can change rapidly, continuous assessment of organ dysfunction, which predicts morbidity, and the use of prognostic scoring systems, which assess disease severity and predict mortality, are essential in this patient population. Early warning scores play an important role in guiding clinical monitoring and treatment.

Risk stratification at the time of admission is crucial in trauma patients, as it directly influences prognosis by supporting accurate clinical decision-making, enabling early intervention, and facilitating appropriate supportive care. Scoring systems based on physiological and anatomical parameters, such as the Acute Physiology and Chronic Health Evaluation II (APACHE II), Revised Trauma Score (RTS), Injury Severity Score (ISS), and Trauma and Injury Severity Score (TRISS), are widely used in trauma care.[5] Among these, RTS, ISS, and TRISS have been in use for more than three decades.[5] Anatomical scores (e.g., ISS and New Injury Severity Score [NISS]) are more effective in predicting ICU admission, whereas physiological scores (RTS, Glasgow Coma Scale [GCS], and APACHE II) are more closely associated with mortality prediction. The TRISS, a combined anatomo-physiological scoring system, has been shown to better predict ICU length of stay and duration of mechanical ventilation.[6] Various quantitative trauma scoring systems have been developed to support clinical decision-making in trauma management, and numerous studies have evaluated their performance. Jiang et al.[7] emphasize that the ideal scoring tool for trauma patients should be easy to use, accurately identify critically ill patients, and provide rapid and reliable mortality prediction to improve outcomes.

A complex physiological cascade is initiated within seconds of trauma, involving hemodynamic, metabolic, hemostatic, neuroendocrine, and immunological systems.[8] A systemic inflammatory response is typically triggered within approximately 30 minutes following severe injury or multiple trauma.[9] Although this response is essential for tissue repair, dysregulation of the balance between pro- and anti-inflammatory processes may lead to immune dysfunction in severe trauma, resulting in complications such as sepsis and multiple organ failure. The systemic response to trauma has long been a major focus of research; however, the underlying mechanisms are not yet fully understood. Although various markers have been investigated to assess the systemic response to trauma, none has been established as part of trauma scoring systems.[10]

Proinflammatory mediators released from damaged tissues following trauma and elevated in the circulation further exacerbate endothelial injury and inflammation.[11,12] At this stage, understanding the role of blood cell components used in calculating the pan-immune-inflammation value (PIV) is essential for elucidating trauma pathophysiology.[13,14] Neutrophils, the most abundant leukocytes in humans and key regulators of immune homeostasis, are the first cells to arrive at the site of injury.[15] They are activated by endogenous signals released from damaged and/or necrotic cells following tissue damage. These mediators stimulate neutrophil production and activation, resulting in the release of substances such as proteases and oxygen radicals that damage healthy tissues. Following major trauma, neutrophils may accumulate and injure not only damaged tissues but also healthy organs, such as the lungs and liver, potentially leading to severe complications such as acute respiratory distress syndrome (ARDS).

The pan-immune-inflammation value is a novel biomarker used to estimate the inflammatory status of patients, based on neutrophil, platelet, monocyte, and lymphocyte counts (calculated as neutrophils × platelets × monocytes / lymphocytes).[16] Derived from routine whole-blood parameters that can be rapidly and easily obtained in most clinical settings, PIV has been increasingly investigated in recent years for its association with inflammation and patient outcomes, including mortality and survival. Its advantages include low cost, wide availability, and ease of calculation, which contribute to its growing popularity. PIV was first identified as a prognostic factor in patients with metastatic colorectal cancer receiving chemotherapy by Fucà et al.[15] in 2020. Subsequently, its prognostic role has been explored in various malignancies, including esophageal, gastric, pancreatic, hepatocellular, breast, lung, melanoma, glioblastoma (GBM), prostate, and renal cancers, as well as in a range of non-malignant conditions such as multiple sclerosis, deep vein thrombosis, pulmonary embolism, cerebrovascular disease, chronic obstructive pulmonary disease (COPD), coronavirus disease 2019 (COVID-19), psoriasis, geriatric frailty, and burns. In recent years, studies have examined PIV in various immune and inflammatory diseases and specific patient populations, including hypertension, ST-elevation myocardial infarction, dyslipidemia, anti-neutrophil cytoplasmic antibody-associated (ANCA-associated) vasculitis, sepsis, and septic shock, demonstrating its association with prognosis.[17-23]

A review of the literature in scientific databases reveals a lack of studies evaluating the relationship between PIV and trauma severity, organ failure, or mortality. This gap underscores the need for simple, cost-effective biomarkers that enable rapid assessment of the systemic inflammatory response following trauma. Investigating the potential predictive role of PIV, particularly in relation to post-traumatic organ failure, sepsis, and mortality, may support clinical decision-making and contribute to improved management strategies. Therefore, the evaluation of PIV in trauma patients represents an important and clinically relevant gap in the literature.

MATERIALS AND METHODS

This retrospective study was conducted at a tertiary care center in accordance with the Declaration of Helsinki and was approved by the Bilecik Şeyh Edebali University Non-Interventional Clinical Research Ethics Committee (Date: 29.11.2023, Decision no: E-10333602-050.04.01-218622). Patients who presented to the emergency department due to trauma and subsequently required intensive care unit admission within the past two years were included.

Inclusion Criteria:

1. Age ≥18 years

2. Admission to the ICU following trauma.

Exclusion Criteria:

1. Age <18 years.

Demographic characteristics (age, sex), comorbidities, mechanisms of trauma, length of hospital stay, laboratory parameters, surgical interventions during hospitalization, and mortality outcomes were recorded. Laboratory data were obtained from routine tests performed during the patients' stay in the intensive care unit. The mean transfer time from the emergency department to the ICU was 3 hours 30 minutes (209.8 minutes), with a median of 2 hours 55 minutes (174.5 minutes). Based on laboratory data, the glucose-to-potassium ratio and the PIV were calculated. Additionally, the RTS, GCS, and APACHE II scores were retrospectively assessed. Clinical outcomes, including mortality, length of hospital stay, and need for endotracheal intubation, were obtained from hospital records, and patients were analyzed according to outcome groups. Supplemantery Appendix: https://jag.journalagent.com/travma/abs_files/UTD-47646/UTD-47646_(2)_Screen_Shot_2026-04-16_at_16.42.57.png

Using appropriate statistical methods, the predictive performance of PIV for mortality in trauma patients was evaluated and compared with established prognostic parameters.

Patients aged ≥18 years who were admitted to the ICU due

Table 1. Distribution of demographic characteristics and vital signs in survivor and non-survivor patients

	Survivor n (%)	Non-survivor n (%)	Test value	p-value
Sex				
Female	7 (11.5)	1 (7.7)	0.159	0.690
Male	54 (88.5)	12 (92.3)		
Age*	36.26	43.31	321.0	0.283
Type of trauma				
Blunt	57 (93.4)	11 (84.6)	1.121	0.290
Sharp force	4 (6.6)	2 (15.4)		
Total hospital stay*	41.91	16.81	127.5	<0.001
Diagnosis at admission				
Thoracic trauma	7 (11.5)	1 (7.7)		
Head trauma	36 (59.0)	8 (61.5)		
Extremity trauma	9 (14.8)	0 (0)	3.636	0.304
Abdominal trauma	9 (14.8)	4 (30.8)		
Concomitant injury				
Yes	41 (67.2)	9 (69.2)		
No	20 (32.8)	4 (30.8)	0.020	0.888
Intubation				
Yes	19 (31.1)	13 (100)		
No	42 (68.9)	0 (0)	20.699	<0.001
Surgical intervention				
Yes	28 (45.9)	8 (61.5)		
No	33 (54.1)	5 (38.5)	1.049	0.306

Chi-square test; *Mann–Whitney U test (mean rank).

to trauma were included in the study. Exclusion criteria were age <18 years and transfer to another hospital for any reason. A total of 81 trauma patients were screened, and seven were excluded based on these criteria.

Statistical Analysis

All data were analyzed using SPSS version 26.00 (SPSS Inc., Chicago, USA). The normality of continuous variables was assessed using the Kolmogorov–Smirnov test. Categorical variables were compared using the Pearson chi-square test. Differences between two groups with non-normally distributed continuous variables were analyzed using the Mann–Whitney U test. Continuous variables were expressed as median (first and third quartiles). Optimal cut-off values for PIV, RTS, GCS, glucose-to-potassium ratio, and APACHE scores were determined using receiver operating characteristic (ROC) curve analysis. A p value <0.05 was considered statistically significant.

RESULTS

Sample size estimation was performed using G*Power based on α=0.05, power=0.8, and an effect size of 0.7. The minimum sample size required to achieve statistical power was calculated as 70 patients. A total of 74 patients were included in the study, of whom 13 (17.6%) were non-survivors and 67 (82.4%) were survivors. In the survivor group, there were seven females (11.5%) and 54 males (88.5%), while the non-survivor group included one female (7.7%) and 12 males (92.3%). The mean age was 50.23±22.03 years in non-survivors and 44.67±20.57 years in survivors. The mean length of hospital stay was 7.85±14.37 days in non-survivors and 12.44±7.78 days in survivors. Table 1 presents the distribution of demographic characteristics and vital signs among survivor and non-survivor groups. There were statistically significant differences between the groups in terms of intubation status and length of hospital stay (p<0.001).

Table 2 shows the comparison of laboratory parameters and trauma scores between the groups. Comparative analysis of laboratory parameters and trauma scores revealed significantly higher values of PIV, RTS, GCS, hematocrit, hemoglobin, platelet count, and diastolic and systolic blood pressure in survivors. In contrast, APACHE II scores, lactate, glucose, potassium, glucose-to-potassium ratio, and lymphocyte count were higher in non-survivors. There was no statistically significant difference in PIV values between females and males (p=0.095). Similarly, no significant difference was observed between blunt and sharp-force injuries (p=0.984).

ROC analysis showed that the area under the curve (AUC) for PIV, RTS, GCS, and APACHE II was statistically significant (p<0.001) (Fig. 1). The AUC values for PIV, RTS, GCS, and APACHE II were 0.702, 0.871, 0.805, and 0.115, respectively (Table 3), demonstrating that these parameters have predictive value for mortality.[24] The optimal cut-off values for PIV, RTS, GCS, and APACHE II were 6367.5, 3.5, 14.5, and

Table 2. Comparison of laboratory parameters and trauma scores between survivor and non-survivor patients

	Survivor Median (first and third quartiles)	Non-survivor Median (first and third quartiles)	Test value	p-value
PIV 1875.0 (1078.5-4367.5)	484.2 (73.68-4145.5)	236.0	0.023	
RTS8.0 (6.5-8.0)	4.0 (3.0-5.0)	102.5	<0.001	
APACHE II	13.0 (8.0-17.0)	28.0 (22.5-39.5)	91.0	<0.001
GCS	14.0 (9.0-15.0)	3.0 (1.0-6.0)	155.0	<0.001
Lactate	1.8 (1.20-2.55)	4.6 (2.71-10.95)	130.5	<0.001
Glucose	135.0 (119.0-163.0)	210.0 (122.0-240.0)	249.0	0.036
Potassium	4.10 (3.81-4.39)	4.64 (3.68-5.21)	271.0	0.075
G/P	33.0 (29.1-39.5)	43.7 (25.9-54.4)	311.5	0.227
Hematocrit (Hct)	36.8 (33.3-40.7)	31.2 (16.8-35.75)	195.0	0.004
Hemoglobin (Hb)	12.5 (11.3-13.8)	10.7 (5.45-12.1)	184.5	0.002
Neutrophils	11.07 (8.72-15.86)	7.6 (3.63-20.4)	327.0	0.324
Monocytes	0.73 (0.55-1.02)	0.7 (0.18-1.28)	325.0	0.310
Platelets	190.0 (161.5-232.0)	155.0 (110.5-204.0)	232.5	0.020
Lymphocytes	0.87 (0.5-1.14)	1.3 (0.85-2.08)	204.5	0.006
Diastolic blood pressure	75.0 (70.0-81.0)	52.0 (34.5-62.5)	83.5	<0.001
Systolic blood pressure	124.0 (113.0-137.0)	86.0 (66.0-112.0)	123.5	<0.001

PIV: Pan-immune-inflammation value; RTS: Revised trauma score; GCS: Glasgow coma scale; APACHE II: Acute Physiology and Chronic Health Evaluation I.

Table 3. Areas under the curve, cut-off values, sensitivity, and specificity of the, Acute Physiology and Chronic Health Evaluation II, Glasgow Coma Scale, and glucose-to-potassium ratio for predicting mortality

Variable	Area	Asymptotic sig.	Asymptotic 95% CI		Cut-off	Sensitivity (%)	Specificity (%)
			Lower	Upper			
PIV	0.702	0.046	0.504	0.901	6367.5	0.846	0.148
RTS	0.871	<0.001	0.758	0.981	3.5	0.462	0.984
GCS	0.805	<0.001	0.676	0.933	14.5	0.923	0.361
G/P	0.393	0.335	0.175	0.611	25.3	0.077	0.918
APACHE II	0.115	<0.001	0.028	0.201	4.5	0	0.934

PIV: Pan-immune-inflammation value; RTS: Revised Trauma Score; GCS: Glasgow Coma Scale; APACHE II: Acute Physiology and Chronic Health Evaluation I.

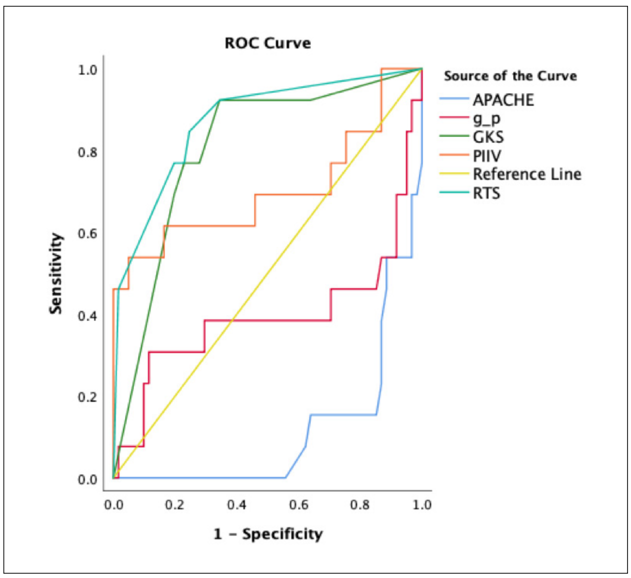

Figure 1. Receiver operating characteristic (ROC) curves of Acute Physiology and Chronic Health Evaluation II (APACHE II), glucose-to-potassium ratio, Glasgow Coma Scale (GCS), pan-immune-inflammation value (PIV), and Revised Trauma Score (RTS) for predicting mortality.

4.5, respectively, demonstrating their diagnostic value for mortality. At these thresholds, the sensitivity values for PIV, RTS, GCS, and APACHE II were 0.846, 0.462, 0.923, and 0, respectively, while the corresponding specificity values were 0.148, 0.984, 0.361, and 0.934.

DISCUSSION

When demographic and clinical characteristics were compared between survivor and non-survivor groups, no significant differences were observed in terms of sex, age, trauma mechanism, primary diagnosis at admission, or the presence of additional injuries. The length of hospital stay was significantly longer among survivors, which is consistent with the expected course of recovery. A marked difference was observed in intubation status, as all patients in the non-survivor group were intubated, compared with only a minority of survivors. Surgical intervention rates did not differ significantly between the groups. Overall, these findings suggest that demographic factors did not have a significant impact on mortality in our study.

In the present study, PIV was found to be associated with mortality in trauma patients. In addition to supporting the predictive value of established prognostic markers reported in the literature, our study strengthens the evidence linking PIV to trauma outcomes and highlights the need for further research in this area.[25,26] There are no published studies in the literature evaluating the prognostic value of PIV in polytrauma patients. However, a limited number of recent studies in conditions such as burn injuries and earthquake-related injuries—both of which involve trauma-like inflammatory responses—suggest its potential relevance, although direct evidence remains limited. Agan et al.[14] evaluated 76 patients with earthquake-related crush injuries and reported that higher PIV values were associated with increased rates of ICU admission and dialysis requirement. These findings suggest that PIV may serve as an early indicator to guide triage and treatment planning. Similarly, in a retrospective study including 140 patients with earthquake-induced crush injuries and 200 control patients, Yasar et al.[27] evaluated PIV alongside other inflammatory biomarkers, such as neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), monocyte-to-lymphocyte ratio (MLR), systemic immune-inflammation index (SII), and systemic inflammation response index (SIRI), and assessed clinical outcomes, including length of hospital stay, dialysis requirement, and blood product transfusion. PIV values were significantly higher in the earthquake group; however, no significant associations were observed with dialysis requirement, blood transfusion, or length of hospital stay. Nevertheless, elevated PIV levels were suggested to be associated with poorer overall outcomes. Furthermore, two recent studies have evaluated PIV values in burn patients.[25,26] Dincer et al.[25] reported significantly higher PIV values in a cohort of 100 burn patients who died, suggesting that PIV may serve as a reliable biomarker for predicting in-hospital mortality. Similarly, Xi et al.,[26] in a study of 367 patients with facial burns, found that higher PIV values were associated with increased scar formation. In contrast to these studies, our study examined the relationship between PIV and key clinical outcomes in polytrauma patients, including injury severity, development of organ failure, need for and duration of intubation, length of intensive care and hospital stay, and mortality. These aspects underscore the novelty and clinical relevance of our study.

In the present study, elevated lactate levels and reduced systolic and diastolic blood pressure were identified as significant predictors of mortality. These parameters reflect tissue hypoxia, inadequate perfusion, and shock—well-established biochemical and clinical markers strongly associated with early mortality.[27] Additionally, the glucose-to-potassium (Glu/K) ratio was significantly higher in non-survivors. Consistent with our findings, Katipoglu et al.,[28] in a study of 99 patients with blunt abdominal trauma, also reported higher Glu/K ratios among non-survivors. Following trauma, sympathetic activation and catecholamine release increase serum glucose levels while reducing potassium levels. Moreover, post-traumatic hyperglycemia and insulin resistance are recognized contributors to increased mortality.

Numerous studies have compared the prognostic performance of established trauma scoring systems, such as RTS, GCS, and ISS.[29-31] Consistent with the literature, RTS and GCS were strong predictors of mortality in our study. The lack of an association between APACHE II scores and mortality may be attributable to the relatively small sample size. Furthermore, APACHE II requires assessment of the worst values within the first 24 hours, limiting its utility in emergency settings. GCS and RTS also have certain limitations, particularly the influence of endotracheal intubation and patient age, as respiratory rate is not standardized in intubated patients or younger individuals.[32] Additionally, the use of GCS alone has limited sensitivity for detecting traumatic brain injury in polytrauma patients.[33]

In recent years, inflammatory biomarkers derived from complete blood count parameters have attracted increasing interest for predicting clinical outcomes.[20,34,35] The PIV is a novel biomarker incorporating four immune cell types—neutrophils, monocytes, platelets, and lymphocytes—reflecting the systemic immune-inflammatory response. Numerous studies in oncology, cardiology, and critical care have demonstrated strong associations between PIV, disease severity, and mortality.[16] Although PIV has been linked to clinical outcomes in conditions such as malignancy, sepsis, acute burns, stroke, and heart failure, its prognostic utility in trauma patients—particularly those with polytrauma—remains insufficiently explored. Given that trauma-induced physiological and inflammatory responses are closely associated with mortality, investigating PIV in this population is of particular importance.

In our study, lower PIV values were significantly associated with mortality. ROC analysis confirmed that PIV had significant predictive ability for mortality, suggesting its potential role as a prognostic biomarker in trauma patients. Across the literature, PIV values vary substantially among different disease groups, suggesting that their clinical interpretation may differ between acute inflammatory states, such as trauma, and chronic conditions, such as cancer or cardiometabolic diseases.

For example, Lin et al.[36] demonstrated improved overall survival in breast cancer patients with lower PIV values in a cohort of 1,312 patients. Baş et al.[34] reported that higher PIV levels were associated with frailty in 450 geriatric patients. In patients with hypertension, Wu et al.[30] found that elevated PIV values were linked to increased mortality.[30] Furthermore, a meta-analysis by Hai-Jing et al.,[37] including 8,799 cancer patients across 30 studies, concluded that high PIV values were significantly associated with reduced overall survival.

The differences observed in trauma may be related to the distinct nature and temporal dynamics of the inflammatory response. In chronic conditions such as cancer or cardiovascular disease, inflammation is persistent, low-grade, and prolonged, leading to elevation of immune cell counts and, consequently, higher PIV values. In contrast, trauma induces a sudden hyperinflammatory response that may rapidly transition into a state of immune suppression. Previous studies have demonstrated that cellular reserves and functions decline rapidly following severe trauma. Therefore, PIV should not be interpreted in isolation but rather in the context of the underlying disease process and its temporal course.

Given the variables influencing PIV, the potential effects of blood transfusions and therapeutic interventions on hematological parameters should be considered. To minimize confounding, PIV in our study was calculated using laboratory values obtained at initial presentation. In a study involving 82 patients with septic shock, PIV did not reliably predict mortality, likely due to treatment-related fluctuations in immune cell counts.[35] Nevertheless, even in that study, patients with lower PIV values demonstrated better survival compared with those with higher values.

In our cohort, in contrast to studies involving burns or earthquake-related crush injuries—where elevated PIV has been associated with poorer outcomes—we found that lower PIV values were associated with mortality. This discrepancy may be explained by the heterogeneity of trauma mechanisms, compared with the more homogeneous populations observed in burn or crush injury studies.

Several mechanisms may account for this inverse relationship:

1. Early post-traumatic immune exhaustion: Severe trauma may lead to rapid depletion or functional impairment of neutrophils, monocytes, and platelets, resulting in reduced cell counts and lower PIV values.[38]

2. Failure of lymphocyte recovery: Persistent lymphopenia has been associated with increased mortality in trauma, independent of leukocytosis patterns.[39]

3. Functional, not just numerical, immune deficits: Trauma impairs neutrophil chemotaxis, phagocytosis, receptor expression, and T-cell function, leading to inadequate antigen presentation and dysregulated cytokine response.[40]

Taken together, the association between high PIV and poor outcomes described in chronic inflammatory or more homogeneous acute conditions may not apply to severe trauma. In this context, low PIV may reflect immune exhaustion, dysfunction, or impending immunosuppression, factors closely associated with increased mortality. Therefore, the clinical interpretation of PIV should consider trauma type, timing, and the phase of the immune response (early hyperinflammation vs. late immunosuppression). Future studies should evaluate serial PIV measurements (e.g., at 0–24 hours, 48 hours, and day 7) to better characterize the dynamic relationship between PIV, immune dysfunction, and mortality.

In the context of major trauma and mass-casualty scenarios, incorporating PIV into simplified scoring systems may enhance triage accuracy during the "golden hours." Moreover, real-time digital or mobile tools for calculating PIV could support rapid clinical decision-making in emergency settings.

Limitations

The primary limitations of this study include its retrospective and single-center design. Although the sample size was relatively small compared with other studies, it exceeded the minimum required based on G*Power analysis. Another limitation is the inability to precisely determine the time interval between the trauma event and laboratory evaluation. Only the time between emergency department admission and intensive care unit admission could be obtained from the hospital system. Additionally, due to the limited sample size, subgroup analyses based on trauma type could not be performed. Larger, multicenter prospective studies are needed to further clarify the relationship between PIV and trauma outcomes.

CONCLUSION

In this trauma cohort, RTS, GCS, APACHE II scores, intubation status and duration, length of hospital stay, serum lactate levels, glucose-to-potassium ratio, and systolic and diastolic blood pressures differed significantly between survivors and non-survivors. Notably, this study is the first to evaluate the early prognostic value of PIV at initial presentation in trauma patients, demonstrating a significant association between PIV and mortality.

Rapid and reliable prognostication is essential in emergency settings. Early inflammatory responses following trauma are closely linked to subsequent organ failure. Given that PIV is simple to calculate, cost-effective, and based on widely available laboratory parameters, it may serve as a valuable adjunct for triage and treatment planning in trauma care. In particular, low PIV values may warrant increased clinical vigilance.

Acknowledgments: We would like to thank our valued family members for their understanding and support during the time devoted to this work.

Ethics Committee Approval: This study was approved by the Bilecik Şeyh Edebali University Non-Interventional Clinical Research Ethics Committee (Date: 29.11.2023, Decision No: E-10333602-050.04.01-218622).

Informed Consent: Informed consent was waived by the institutional review board due to the retrospective nature of the study.

Authorship Contributions: Concept: G.G., K.Ç.; Design: K.Ç., K.D.A.; Supervision: K.Ç.; Resource: K.D.A., G.G.; Materials: K.D.A., F.Ç.I.; Data collection and/or processing: F.Ç.I., K.Ç.; Analysis and/or interpretation: F.Ç.I., K.Ç.; Literature review: G.G., K.D.A.; Writing: G.G., K.Ç., K.D.A., F.Ç.I.; Critical review: K.D.A., K.Ç.

Conflict of Interest: None declared.

Financial Disclosure: The author declared that this study has received no financial support.

REFERENCES

1. Mortaz E, Zadian SS, Shahir M, Folkerts G, Garssen J, Mumby S, et al. Does Neutrophil Phenotype Predict the Survival of Trauma Patients? Front Immunol 2019;10:2122. [CrossRef]
2. Akkoca M, Tokgöz S, Yılmaz KB, Güler S, Akıncı M, Balas Ş, et al. Mortality determiners for fall from height cases. Ulus Travma Acil Cerrahi Derg 2018;24:445-9. [CrossRef]
3. Adiyaman E, Tokur ME, Bal ZM, Gokmen AN, Koca U. Retrospective evaluation of trauma patients followed-up and treated in an anesthesia intensive care unit. Turk J Intensive Care 2019;17:146-53. [CrossRef]
4. Bartlett ES, Lim A, Kivlehan S, Losonczy LI, Murthy S, Lowsby R, et al. Critical care delivery across health care systems in low-income and low-middle-income country settings: A systematic review. J Glob Health 2023;13:04141. [CrossRef]
5. Deng Q, Tang B, Xue C, Liu Y, Liu X, Lv Y, et al. Comparison of the ability to predict mortality between the ınjury severity score and the new ınjury severity score: A meta-analysis. Int J Environ Res Public Health 2016;13:825. [CrossRef]
6. Chun M, Zhang Y, Becnel C, Brown T, Hussein M, Toraih E, et al. New injury severity score and trauma injury severity score are superior in predicting trauma mortality. J Trauma Acute Care Surg 2022;92:528–34. [CrossRef]
7. Jiang D, Chen T, Yuan X, Yang Y, Shen Y, Huang Z. Predictive value of the Trauma Rating Index in Age, Glasgow Coma Scale, Respiratory rate and Systolic blood pressure score (TRIAGES) for the short-term mortality of older patients with isolated traumatic brain injury: a retrospective cohort study. BMJ Open 2024;14:e082770. [CrossRef]
8. MacColl H, Morton S, Vasireddy A. The metabolic response to trauma. MPSUR 2024;42:373-7. [CrossRef]
9. Brøchner AC, Toft P. Pathophysiology of the systemic inflammatory response after major accidental trauma. Scand J Trauma Resusc Emerg Med 2009;17:43. [CrossRef]
10. Ilyas MF, Lado A, Budiono EA, Suryaputra GP, Ramadhana GA, Novika

RGH. Platelet-to-lymphocyte ratio as a prognostic predictive marker on adults with traumatic brain injury: Systematic review. Surg Neurol Int 2024;15:205. [CrossRef]

11. Li R, Ye JJ, Gan L, Zhang M, Sun D, Li Y, et al. Traumatic inflammatory response: pathophysiological role and clinical value of cytokines. Eur J Trauma Emerg Surg 2024;50:1313-30. [CrossRef]
12. Potempa M, Hart PC, Rajab IM, Potempa LA. Redefining CRP in tissue injury and repair: more than an acute pro-inflammatory mediator. Front Immunol 2025;16:1564607. [CrossRef]
13. Zhang P, Tang C, Zhou Y, Zheng Z, Chen Y, Ni H, et al. Association between inflammatory score, coagulopathy, and hemorrhagic progression in patients with traumatic intraparenchymal hemorrhage: an exploratory study with interaction and mediation models. J Neurosurg 2025;1:1-10. [CrossRef]
14. Agan FZ, Cindolu C, Abuska D, Abouelsoud A. Evaluating the utility of complete blood count-derived inflammatory indices for predicting clinical outcomes in earthquake-related crush injuries: the 2023 Turkey-Syria earthquake. Disaster Med Public Health Prep 2025;19:e289. [CrossRef]
15. Yang L, Shi F, Cao F, Wang L, She J, He B, et al. Neutrophils in tissue ınjury and repair: molecular mechanisms and therapeutic targets. MedComm (2020) 2025;6:e70184. [CrossRef]
16. Fucà G, Guarini V, Antoniotti C, Morano F, Moretto R, Corallo S, et al. The pan-ımmune-ınflammation value is a new prognostic biomarker in metastatic colorectal cancer: results from a pooled-analysis of the Valentino and TRIBE first-line trials. Br J Cancer 2020;123:403-9. [CrossRef]
17. Nomura RM, Cabar FR, Costa VN, Miyadahira S, Zugaib M. Cardiac troponin T as a biochemical marker of cardiac dysfunction and ductus venosus Doppler velocimetry. Eur J Obstet Gynecol Reprod Biol 2009;147:33-6. [CrossRef]
18. Karadağ I, Karakaya S, Yılmaz ME, Çakmak Öksüzoğlu ÖB. The potential prognostic novel markers PIV and PILE score to predict survival outcomes at hepatocellular cancer. Eur Rev Med Pharmacol Sci 2022;26:7679-86.
19. Yilmaz Y, Kelesoglu S. The ımportance of pan-ımmune ınflammation value (PIV) in predicting coronary collateral circulation in stable coronary artery patients. Angiology 2026;77:68-74. [CrossRef]
20. Kaplangoray M, Toprak K, Deveci E, Caglayan C, Sahin E. Could pan-immune-inflammation value be a marker for the diagnosis of coronary slow flow phenomenon? Cardiovasc Toxico. 2024;24:519-26. [CrossRef]
21. Ocak T, Lermi N, Bozkurt ZY, Yagiz B, Coskun B, Dalkilic E, et al. Pan-immune-inflammation value could be a new marker to differentiate between vascular Behçet's disease and non-vascular Behçet's disease. Eur Rev Med Pharmacol Sci 2024;28:7384-91.
22. Lokesh K, Shenoy GC, Hiremani MS, AH R, Rajeev LK, Saldanha SC, et al. Pan-ımmune-inflammation value: a novel marker for chemotherapy response in locally advanced breast cancer. Indian J Med Paediatr Oncol 2025;46:42-7. [CrossRef]
23. Cao G, Liu Q, Wen H, Zeng Y. The prognostic importance of the pan-immune-inflammation value (PIV) in lung cancer: a systematic review and meta-analysis. Transl Lung Cancer Res 2025;14:4357-70. [CrossRef]
24. Kushimoto S, Akaishi S, Sato T, Nomura R, Fujita M, Kudo D, et al., Lactate, a useful marker for disease mortality and severity but an unreliable marker of tissue hypoxia/hypoperfusion in critically ill patients. Acute Med Surg 3:293-7. [CrossRef]
25. Dincer HA, Koci S, Cennet O, Konan A. Pan-ımmune ınflammation value as a novel comprehensive predictor of ın-hospital mortality in patients with severe burns: a single-center retrospective analysis. Medicina (Kaunas) 2025;61:1705. [CrossRef]
26. Xi D, Yu H, Yu T. Development of a predictive model for the relationship between serum pan-immunoinflammatory index levels and scar formation in facial burn patients. Am J Transl Res 2025;17:2197. [CrossRef]
27. Yasar B, Ozbilgehan P, Sen M, Guvendik A. Influence of systemic inflammatory indices on hospital stay and dialysis post-earthquake: A clinical study. PLoS One 2024;19:e0299737. [CrossRef]
28. Katipoglu B, Demirtas E. Evaluation of serum glucose-to-potassium ratio as a predictor for morbidity and mortality in patients with blunt abdominal trauma. Ulus Travma Acil Cerrahi Derg 2022;28:1413-9.
29. Orhon R, Eren SH, Karadayı S, Korkmaz I, Coşkun A, Eren M, et al. Comparison of trauma scores for predicting mortality and morbidity on trauma patients. Ulus Travma Acil Cerrahi Derg. 2014;20:258-64. [CrossRef]
30. Wu SC, Chou SE, Liu HT, Hsieh TM, Su WT, Chien PC, et al. Performance of prognostic scoring systems in trauma patients in the intensive care unit of a trauma center. Int J Environ Res Public Health 2020;17:7226. [CrossRef]
31. Llompart-Pou J, Chico-Fernández M, Sánchez-Casado M, Salaberria-Udabe R, Carbayo-Górriz C, Guerrero-López F, et al. Scoring severity in trauma: comparison of prehospital scoring systems in trauma ICU patients. Eur J Trauma Emerg Surg 2017;43:351-7. [CrossRef]
32. Filipescu R, Powers C, Yu H, Yu J, Rothstein DH, Harmon CM, et al. Improving the performance of the Revised Trauma Score using Shock Index, Peripheral Oxygen Saturation, and Temperature–a National Trauma Database study 2011 to 2015. Surgery 2020;167:821-8. [CrossRef]
33. Schucht JE, Rakhit S, Smith MC, Han JH, Brown JB, Grigorian A, et al. Beyond Glasgow Coma Scale: Prehospital prediction of traumatic brain injury. Surgery 2025;179:108893. [CrossRef]
34. Okyar Baş A, Güner M, Ceylan S, Hafızoglu M, Sahiner Z, Balam Doğu B, et al. Pan-immune inflammation value; a novel biomarker reflecting inflammation associated with frailty. Aging Clin Exp Res 2023;35:1641–9. [CrossRef]
35. Turan YB. The prognostic importance of the pan-immune-inflammation value in patients with septic shock. BMC Infect Dis 2024;24:69. [CrossRef]
36. Lin F, Zhang LP, Xie SY, Huang HY, Chen XY, Jiang TC, et al. Pan-Immune-ınflammation value: a new prognostic ındex in operative breast cancer. Front Oncol 2022;12:830138. [CrossRef]
37. Hai-Jing Y, Shan R, Jie-Qiong X. Prognostic significance of the pretreatment pan-immune-inflammation value in cancer patients: an updated meta-analysis of 30 studies. Front Nutr 2023;10:1259929. [CrossRef]
38. Vanzant EL, Lopez CM, Ozrazgat-Baslanti T, Ungaro R, Davis R, Cuenca AG, et al. Persistent inflammation, immunosuppression, and catabolism syndrome after severe blunt trauma. J Trauma Acute Care Surg 2014;76:21-30. [CrossRef]
39. Heffernan DS, Monaghan SF, Thakkar RK, Machan JT, Cioffi WG, Ayala A. Failure to normalize lymphopenia following trauma is associated with increased mortality, independent of the leukocytosis pattern. Crit Care 2012;16:R12. [CrossRef]
40. Thompson KB, Krispinsky LT, Stark RJ. Late immune consequences of combat trauma: a review of trauma-related immune dysfunction and potential therapies. Mil Med Res 2019;6:11. [CrossRef]

ORİJİNAL ÇALIŞMA - *ÖZ*

Travma hastalarında farklı bir bakış açısı kohortu: PIV ölüm oranını öngörebilir mi?

AMAÇ: Travma, dünya genelinde ölümlerin önemli bir bölümünü oluşturmaktadır. Acil servislerde ve yoğun bakım ünitelerinde bu hastaların prognozunu tahmin etmek, etkili triyaj ve takip planlaması için çok önemlidir. Bu nedenle, travma hastalarında çok sayıda prognostik belirteç araştırılmaktadır. Panimmün-inflamasyon (PIV), tam kan sayımından (CBC) hesaplanabilen ve tıbbi ünitede hızlı bir şekilde uygulanabilen bir biyobelirteçtir. Bu çalışma, PIV değerinin travma hastalarının prognozunda rol oynayıp oynamadığını değerlendirmeyi amaçlamıştır.
GEREÇ VE YÖNTEM: Bu çalışma, üçüncü basamak yoğun bakım olanaklarına sahip bir eğitim ve araştırma hastanesinde travma nedeniyle yoğun bakım ünitesine yatırılan hastaları incelemiştir. Bilinen prognostik parametreler (RTS, GCS ve APACHE II gibi) değerlendirilmiş, PIV değerleri hesaplanmış ve mortalite, morbidite ve hastanede kalış süresi retrospektif olarak analiz edilmiştir. PIV değerinin mortaliteyi tahmin etme değeri istatistiksel olarak analiz edilmiştir.
BULGULAR: Çalışmaya toplam 74 hasta dahil edilmiştir. Hayatta kalanlar grubunda 7 kadın (%11.5) ve 54 erkek (%88.5) bulunurken, ölenler grubunda 1 kadın (%7.7) ve 12 erkek (%92.3) vardı. PIV, RTS, GKS ve APACHE skorlarının mortaliteyi tahmin etmede etkili olduğu bulundu ($p<0.001$). PIV değeri için eşik değer 6367.5 olarak bulundu ve bu değerin altında PIV değerine sahip hastaların, daha yüksek değerlere sahip olanlara göre ölme olasılığı daha yüksekti.
SONUÇ: Acil durumlarda hızlı ve güvenilir prognoz çok önemlidir. PIV değeri, diğer bilinen prognoz skorlama sistemlerine benzer bir mortalite tahmin etkisi yaratma potansiyeline sahiptir. Travma hastalarında PIV değerinin hızlı değerlendirilmesi, daha etkili triyaj ve tedavi planlamasını kolaylaştırabilir.

Anahtar sözcükler: Mortalite; panimmün-enflamasyon değeri; prognoz; travma.

Ulus Travma Acil Cerrahi Derg 2026;32(5):558-566 DOI: 10.14744/tjtes.2026.33746