Research Article RJ-2

Researches on Multidisciplinary Approaches 2026 (1) ISSN:2791-9099

Prognostic Value and Timing of Thrombocytopenia in the ICU: Associations with Mortality, Sepsis, and APACHE II[1]

Kenan Kart* / MD
Karabuk University, Training and Research Hospital, Department of Anesthesiology and Reanimation
kenankart@karabuk.edu.tr

Nagehan Ümit Karaca / MD
Karabuk University, Training and Research Hospital, Department of Anesthesiology and Reanimation
drnagehan33@gmail.com

*Corresponding Author

Abstract

In intensive care units, thrombocytopenia is often encountered, yet its prognostic value remains incompletely understood. This study aimed to explore how platelet count reductions relate to mortality, and whether the timing of thrombocytopenia development offers any additional prognostic insight, particularly when considered alongside sepsis and APACHE II scores. We retrospectively reviewed 232 patients treated in the Anesthesia ICU at Karabük Training and Research Hospital between 2023 and 2024. Thrombocytopenia was defined as a platelet count <150,000/μL. We recorded demographic data, laboratory results (WBC, Hb, aPTT, PLT), APACHE II scores, presence of sepsis, and mortality. Thrombocytopenia was found in 21.9% of cases. These patients had significantly higher APACHE II scores, a greater likelihood of sepsis, and sharply increased mortality ($p < 0.001$). They also showed decreased hemoglobin and platelet levels, and elevated aPTT. Interestingly, WBC did not differ between groups ($p = 0.901$). Among those who died, thrombocytopenia tended to occur around 10 days before death and showed moderate correlation with both APACHE II scores and sepsis. Thrombocytopenia, especially when occurring later during ICU admission, may be a subtle but meaningful indicator of clinical decline. When interpreted with APACHE II and sepsis data, the timing of thrombocytopenia could help refine mortality prediction. Our findings suggest that platelet monitoring deserves greater clinical attention—not just as a routine lab value, but as a potentially dynamic prognostic marker in the ICU setting.

Keywords: APACHE II, Intensive Care, Mortality, Platelet Trends, Sepsis, Thrombocytopenia.

JEL Codes: I10, I12, I18

[1] This study was previously presented at the Academic Studies Group – Uzbekistan Congress Special Issue.

Citation: Kart, K., & Ümit Karaca, N. (2026). Prognostic value and timing of thrombocytopenia in the ICU: Associations with mortality, sepsis, and APACHE II. *Researches on Multidisciplinary Approaches (ROMAYA Journal)*, 2026(1).

Submission Date : 08.07.2025
Acceptance Date : 27.10.2025

1. Introduction

Thrombocytopenia, while often overlooked as a routine laboratory abnormality, is frequently encountered in critically ill patients and may reflect far more than hematologic dysfunction. Clinically, it is defined as a platelet count below 150,000/μL, but its implications extend well beyond a simple numerical threshold. Prior studies estimate its incidence in the ICU to range between 35% and 44% (Levi & Lowenberg, 2008; Coskun et al., 2016; Greinacher & Selleng, 2010). However, those figures may not always reflect the diverse case mixes seen across institutions.

The causes of thrombocytopenia in the ICU are varied and include hemodilution, postoperative bleeding, sepsis, liver dysfunction, and drug-induced cytopenias. In some patients, a single factor is the main determinant, while in others, several mechanisms act together to reduce platelet counts. Thrombocytopenia remains strongly associated with adverse outcomes, particularly in patients with multiple organ failure (Anthon et al., 2024).

Scoring systems such as APACHE II and SOFA are frequently used to reflect clinical deterioration. Several studies have shown that patients with thrombocytopenia typically score higher on these indices, reflecting the broader systemic impact of their condition (Péju et al., 2023). These findings emphasize the relevance of incorporating platelet dynamics into comprehensive patient evaluation.

Although thrombocytopenia is routinely monitored, the timing of its onset has not been extensively studied. Clinical observations suggest that platelet counts begin to drop days before overt clinical decline, particularly in patients with sepsis. Whether this decline can serve as an early prognostic marker remains uncertain and was one of the motivations for the present study (Wang & Zhang, 2024).

This study aimed to evaluate both the occurrence and the timing of thrombocytopenia in ICU patients, and to determine whether a decline in platelet count provides additional prognostic value when assessed together with established parameters such as APACHE II scores and the presence of sepsis.

2. Materials and Methods

This study was designed as a retrospective observational analysis and was approved by the Ethics Committee of Karabük University (Approval No: E-77192459-050.99-426155, Date: 16.04.2025, Decision No: 2025/2211). Data were collected from patient records between January 2023 and January 2024 at the Anesthesia Intensive Care Unit (ICU) of Karabük Training and Research Hospital.

A total of 369 patient records were screened during the study period. After applying the predefined criteria, 232 patient records were eligible and included in the final analysis. The remaining 137 records were excluded for the following reasons: pregnancy (n = 5), age below 18 years (n = 7), terminal-stage malignancy (n = 24), active major bleeding (n = 18), hematologic disorders affecting platelet count (n = 15), history of drug-induced thrombocytopenia (n = 12), and ICU stay shorter than 24 hours (n = 56). The inclusion criteria were defined as age ≥18 years, ICU stay ≥24 hours, and at least one platelet count measurement during admission. These criteria were chosen to reduce variability and focus on medical ICU patients rather than surgical or oncology cases.

Thrombocytopenia was defined as a platelet count below 150,000/μL, either at the time of ICU admission or developing later during the hospital stay. For each patient, we extracted demographic data information, APACHE II scores, presence of comorbidities, laboratory values (including white blood cell count, hemoglobin, platelet count, and activated partial thromboplastin time [aPTT]), and clinical outcome (discharged or deceased). If available, both initial and final values for laboratory parameters were recorded.

Sepsis was defined in accordance with the Sepsis-3 criteria, which describe it as life-threatening organ dysfunction due to a dysregulated host response to infection. A SOFA score of ≥ 2 was used as the threshold for organ dysfunction (Singer et al., 2016).

3. Statistical Analysis

All statistical analyses were performed using IBM SPSS version 25.0. Data distribution was tested with the Kolmogorov–Smirnov test. Depending on normality, continuous variables were analyzed using either the Student's t-test or the Mann–Whitney U test. Categorical variables were evaluated using the chi-square test or Fisher's exact test. Spearman's Rho was applied for correlation analysis. A p-value below 0.05 was considered statistically significant.

4. Results

A total of 232 patients were included in this analysis. The average patient age was relatively advanced, and overall APACHE II scores reflected a critically ill population. Thrombocytopenia at the time of ICU admission was identified in 21.9% of patients. Among all causes of admission, respiratory failure was the most frequent, followed by cardiac and neurological etiologies (Table 1).

Table 1. Demographic Characteristics and Reasons for ICU Admission

Variable	Value	Statistical Method
Age (years)	72.0 ± 16.9	*
Sex		†
• Female	113 (48.5%)	
• Male	119 (51.1%)	
APACHE II Score	20.4 ± 7.6	*
Thrombocytopenia on Admission		†
• Present	51 (21.9%)	
• Absent	181 (77.7%)	
Reason for ICU Admission		†
• Respiratory	96 (41.2%)	
• Cardiac	33 (14.2%)	
• Neurological	32 (13.7%)	
• Post-CPR	15 (6.4%)	
• Sepsis	6 (2.6%)	
• Metabolic	4 (1.7%)	
• Other	47 (20.1%)	

*Values are presented as mean ± standard deviation or percentage (%). APACHE: Acute Physiology and Chronic Health Evaluation. ICU: Intensive Care Unit. CPR: Cardiopulmonary Resuscitation. * Mann–Whitney U test. † Chi-square or Fisher's exact test.

When comparing laboratory data between ICU admission and discharge, a clear pattern emerged: there was a statistically significant drop in platelet counts and hemoglobin levels, while aPTT values increased. Interestingly, white blood cell counts remained largely unchanged, suggesting that thrombocytopenia and coagulopathy evolved independently of general inflammatory markers (Table 2).

Table 2. Comparison of Laboratory Values at ICU Admission and Discharge

Variable	Admission Mean ± SD	Discharge Mean ± SD	p-value	Statistical Method
Platelet (×10³/µL)	239.6 ± 107.5	205.2 ± 123.3	<0.001	*
White Blood Cell (×10³/µL)	12.3 ± 6.9	11.7 ± 8.1	0.309	*
Hemoglobin (g/dL)	10.5 ± 2.5	9.2 ± 2.1	<0.001	*
aPTT (sec)	28.2 ± 15.3	35.1 ± 22.1	<0.001	*

Data are presented as mean ± standard deviation. A p-value of <0.05 was considered statistically significant. aPTT: Activated Partial Thromboplastin Time. * Wilcoxon signed-rank test was used for paired samples.

Patients who developed thrombocytopenia during their ICU stay had significantly worse clinical profiles. Their APACHE II scores were higher, they were more likely to have sepsis, and their mortality rate was substantially elevated compared to non-thrombocytopenic patients. The white blood cell counts and aPTT values did not significantly differ between the two groups, though mean platelet counts were understandably lower in the thrombocytopenic group (Table 3).

Table 3. Comparison of Clinical Characteristics According to Thrombocytopenia Status (Laboratory Values at ICU Admission)

Parameter	Thrombocytopenia (+)	Thrombocytopenia (-)	p-value	Statistical Method
APACHE II Score	23.3 ± 7.9	19.1 ± 7.1	0.000	*
Sepsis Presence	20 (27.0%)	18 (11.3%)	0.005	†
Mortality (Exitus)	69 (93.2%)	59 (37.3%)	0.000	†
White Blood Cell (WBC)	12.4 ± 7.9	12.2 ± 6.4	0.901	*

Platelet (PLT)	216.7 ± 94.3	253.4 ± 114.5	0.017	*
Hemoglobin (Hb)	10.1 ± 2.4	10.6 ± 2.5	0.174	*
aPTT (sec)	27.8 ± 10.4	28.3 ± 17.1	0.819	*

Data are presented as mean ± standard deviation or percentage (%). APACHE: Acute Physiology and Chronic Health Evaluation. WBC: White Blood Cell. PLT: Platelet Count. Hb: Hemoglobin. aPTT: Activated Partial Thromboplastin Time. * Student's t-test. † Chi-square test.

A separate analysis comparing survivors and non-survivors revealed notable trends. Patients who died were significantly older and had higher APACHE II scores, elevated WBC and aPTT values, and lower hemoglobin. Surprisingly, mean platelet counts did not differ significantly at first glance. However, as discussed later, this was influenced by a small number of extreme outliers (Table 4).

Table 4. Clinical and Laboratory Parameters Associated with Mortality

Parameter	Exitus (+)	Exitus (-)	p-value	Statistical Method
Age	76.9 ± 12.8	65.9 ± 19.3	<0.001	*
APACHE II Score	23.0 ± 7.9	17.3 ± 6.0	<0.001	*
White Blood Cell (WBC)	13.3 ± 8.1	11.0 ± 4.8	0.009	*
Hemoglobin (Hb)	10.1 ± 2.4	10.9 ± 2.5	0.010	*
Platelet (PLT)	247.3 ± 108.8	234.8 ± 110.7	0.387	*
aPTT (sec)	30.1 ± 17.8	25.6 ± 10.7	0.020	*
Sepsis Presence	29 (22.7%)	9 (8.6%)	0.007	†

Data are presented as mean ± standard deviation or percentage (%). APACHE: Acute Physiology and Chronic Health Evaluation. WBC: White Blood Cell. PLT: Platelet Count. Hb: Hemoglobin. aPTT: Activated Partial Thromboplastin Time. * Student's t-test. † Chi-square test.

Among patients who died, platelet count decline was observed an average of 10.2 days prior to death. This timing showed a moderate but meaningful correlation with both APACHE II scores and the presence of sepsis. In contrast, no significant relationship was found between the timing of thrombocytopenia and patient age. These findings suggest that platelet decline may occur earlier in patients with greater illness severity and higher inflammatory burden (Table 5).

Table 5. Timing of Platelet Decline and Its Relationship with Clinical Parameters

Parameter	Value
Mean (±SD)	10.2 ± 8.2
Median (Min–Max)	7.0 (1.0–40.0)
Valid Observations	64
Correlation with Age (Spearman)	0.06
Correlation with APACHE II (Spearman)	0.32
Correlation with Sepsis Presence (Spearman)	0.29

Values are presented as mean ± standard deviation or median (minimum–maximum). ρ: Spearman's correlation coefficient. APACHE: Acute Physiology and Chronic Health Evaluation.

5. Discussion

Thrombocytopenia is one of the most frequently encountered laboratory abnormalities in ICU patients, and its clinical implications have been well documented in previous studies. Beyond serving as a hematologic parameter, thrombocytopenia may indicate broader systemic disturbances. In this study, it was identified in 21.9% of patient records, a rate lower than the 35% to 45% incidence reported in previous studies (Akbaş, 2025; Coskun et al., 2016; Greinacher & Selleng, 2010; Levi & Lowenberg, 2008; Péju et al., 2023). This difference may be partly explained by the characteristics of the ICU population. The unit primarily admits non-surgical medical patients, and individuals with active bleeding or hematologic malignancies were excluded according to predefined criteria. In addition, patients who died within the first 24 hours were not included, which may have contri-

buted to the lower incidence observed. These contextual factors should be considered when interpreting prevalence and related outcomes.

In this ICU cohort, mortality was significantly higher among patients with thrombocytopenia. This finding was not unexpected, as similar trends have been consistently reported in previous studies (Moreau et al., 2007; Vanderschueren et al., 2000). For example, Greinacher and Selleng (2010) discussed a clear link between low platelet counts and higher death rates in critically ill patients. Strauss et al. (2002) found something similar. They reported an overall ICU mortality of 31%, but when thrombocytopenia was present, it jumped to 44%. Without it, mortality dropped to just 16%. The findings in this study were consistent with previous reports, suggesting that thrombocytopenia may represent more than a marker of disease severity and could contribute to anticipating the clinical course.

Although an initial comparison found no statistically significant difference in average platelet counts between deceased and surviving patients ($p = 0.387$), further analysis revealed that two patients in the mortality group had markedly elevated platelet counts (571 and 572 $\times 10^3/\mu L$), which skewed the overall mean. When these outliers were excluded, the intergroup difference in platelet count reached statistical significance ($p < 0.001$). This finding reinforces the prognostic value of thrombocytopenia but also highlights the importance of examining data distributions and handling outliers appropriately in statistical evaluation. Clinical interpretation of thrombocytopenia should therefore consider timing of onset, sepsis status, and APACHE II scores collectively.

In a multicenter prospective study, Akca et al. (2002) looked at when thrombocytopenia begins and how that timing might relate to patient survival. They noticed that platelet counts usually hit their lowest around day 4. Patients who began to recover after that had better outcomes. But if there was no clear recovery by the 14th day, mortality tended to be much higher. Another group of researchers reported something similar: patients who already had low platelets when they entered the ICU did better than those who developed thrombocytopenia later on (Ece, 2021). We saw a comparable pattern in our own study. Every patient who developed thrombocytopenia after day 14 did not survive. This might mean that late-onset thrombocytopenia is a sign of worsening condition and poor prognosis.

The APACHE II scoring system is widely used to estimate disease severity and mortality risk in ICU patients (Knaus et al., 1985). Numerous studies have shown a robust relationship between higher APACHE II scores and increased mortality (Li et al., 2025; Sevim et al., 2011; Warren et al., 2001). In our study, this association was confirmed, and APACHE II scores were significantly higher in patients with thrombocytopenia ($p = 0.000$). This reinforces the notion that thrombocytopenia may serve as a marker of critical illness severity and is associated with established prognostic scoring systems.

Low platelet counts are frequently observed in patients with sepsis and seem to arise from multiple overlapping biological processes. These include immune-related destruction of platelets, shifts in how the immune system responds overall, and changes to how platelets interact with their environment at the receptor level (Cheng et al., 2023). Ghimire et al. (2021) reported that more than half of septic patients—up to 55%—developed thrombocytopenia. Their findings also pointed to a strong link between this condition and worse clinical outcomes.

Schupp et al. (2022) explored how platelet levels could help predict both diagnosis and prognosis in patients with sepsis and septic shock. They found that thrombocytopenia was strongly associated with an increased risk of 30-day mortality from any cause. Interestingly, platelet counts tended to drop to their lowest levels around day 5. Although recovery in the following days (between day 5 and 10) didn't appear to correlate clearly with survival, a decrease of more than 25% in platelet levels was particularly linked with poor outcomes. Supporting this, Claushuis et al. (2016), in a multicenter study involving 931 patients, also noted a strong relationship between low platelet counts and mortality. Similarly, Semeraro et al. (2018) reported that as platelet levels dropped further, the risk of death increased proportionally.

In this cohort, 38 patients were diagnosed with sepsis, and nearly a quarter of them (23.7%) were also thrombocytopenic. In this subgroup, the mortality rate reached 77.8%. Although the sample size is modest, the observed association between low platelet counts and poor outcomes in septic patients is striking and deserves attention. These observations are in line with patterns noted in prior research and highlight the need for careful platelet monitoring in septic cases treated in intensive care.

Haksoyler et al. (2019) noted that white blood cell counts tended to decrease over time in patients with thrombocytopenia, possibly due to overlapping immune-related processes. In this study, however, no statistically significant difference in leukocyte counts was observed between patients with and without thrombocytopenia ($p = 0.901$). A possible explanation for this discrepancy may relate to variation in clinical factors, such as differences in sepsis prevalence or the use of immunosuppressive therapy. Similar findings were reported by Tang et al. (2003), who also did not identify a consistent association between leukopenia and thrombocytopenia. Taken together, these findings suggest that leukopenia does not consistently accompany thrombocytopenia and should not be assumed as a universal feature.

Analysis of the patient data indicated that the timing of platelet decline was closely associated with clinical outcomes. Specifically, among those who did not survive, platelet counts typically began to fall around 10 days prior to death. This temporal relationship may reflect the onset of clinical deterioration. Chen et al. (2022) reported a similar finding, showing that changes in platelet trends were significantly linked to 28-day ICU mortality. We also observed that higher APACHE II scores were modestly correlated with earlier platelet decline ($r = 0.32$), suggesting that patients with more severe illness might develop hematologic abnormalities sooner. This aligns with the study by Al Saleh et al. (2021), which reported a strong connection between elevated APACHE II scores and thrombocytopenia development. Additionally, we found a positive correlation between sepsis and the timing of platelet decline ($r = 0.29$). This may support the idea that heightened inflammatory activity contributes to earlier drops in platelet levels. Wang et al. (2021) have previously shown that inflammatory markers like IL-6 and TNF-α were closely associated with reductions in platelet counts among septic patients, further reinforcing this interpretation.

No significant correlation was observed between patient age and the timing of platelet decline ($r = 0.06$), suggesting that age alone may not be a major determinant of the onset of thrombocytopenia. This observation is consistent with previous reports indicating that organ failure in the ICU is more often driven by underlying physiological disturbances than by chronological age alone (Sinha et al., 2021). When considered alongside APACHE II scores and sepsis status, the timing of platelet decline provides supplementary information for the evaluation of overall prognosis.

This study is among the limited retrospective analyses with an adequate sample size that evaluated the association between thrombocytopenia and ICU mortality from multiple perspectives. We analyzed not only APACHE II scores and sepsis status but also platelet dynamics and the timing of decline, offering a comprehensive approach rarely seen in existing literature. Furthermore, both clinical and laboratory parameters were jointly assessed to provide a deeper understanding of the prognostic value of thrombocytopenia.

This study has several limitations that should be considered when interpreting the results. First, the retrospective and single-center design may restrict the broader applicability of our findings to other settings or patient populations. Second, although we aimed to isolate thrombocytopenia as a prognostic factor, the exact causes behind it were not fully explored, as some subgroups were excluded due to strict inclusion criteria. Lastly, the absence of daily platelet count measurements may have led to missed variations in platelet trends, potentially affecting the accuracy of temporal associations. Data on bone marrow suppression markers such as reticulocyte counts were not available in our cohort, which represents an additional limitation of this study.

Dialysis may also play a role in the development of thrombocytopenia in ICU patients. Previous reports have described dialysis-related platelet reductions, which can worsen the clinical course in patients with sepsis or multi-organ dysfunction (Duayer et al., 2022). In our study, dialysis use was not systematically recorded, and this should be acknowledged as a limitation. Future investigations that include dialysis status will help to clarify its potential impact on thrombocytopenia and mortality.

Future studies are warranted to validate these findings through prospective, multicenter designs that can accommodate greater variability in patient characteristics and allow for more precise tracking of platelet dynamics. Investigating thrombocytopenia with daily platelet monitoring and a more detailed examination of its etiology may help elucidate underlying causal pathways. Additionally, future research may benefit from examining how platelet trends correlate with inflammatory markers such as interleukins, coagulation parameters, and therapeutic interventions, as this may offer further insights into their prognostic value in critical care settings.

6. Conclusion

In this study, we found that the development and timing of thrombocytopenia in ICU patients were significantly associated with mortality, particularly in those with sepsis or high APACHE II scores. Late-onset thrombocytopenia was especially predictive of poor outcomes. These findings suggest that platelet trends offer meaningful prognostic information beyond routine laboratory interpretation. Regular monitoring of platelet counts, in conjunction with clinical severity scores, may aid in early detection of clinical deterioration and support timely decision-making in critical care settings.

References

Akbas T. Thrombocytopenia as an indicator of illness severity in ICU patients. VHS J. 2025;15(1):28–35.

Akca S, Haji-Michael P, de Mendonça A, et al. Time course of platelet counts in critically ill patients. Crit Care Med. 2002;30(4):753–6.

Al Saleh K, AlQahtani RM. Platelet count patterns and outcomes in sepsis: beyond APACHE. Medicine (Baltimore). 2021;100(18):e25013.

Anthon CT, Pène F, Perner A, et al. Platelet transfusions in adult thrombocytopenic ICU patients: protocol for a PLOT-ICU sub-study. Acta Anaesthesiol Scand. 2024;68(3):434–40.

Chen J, Gao X, Shen S, et al. Platelet trajectory and ICU mortality: a multi-cohort study. Front Immunol. 2022;13:936662.

Cheng J, Zeng H, Chen H, et al. Thrombocytopenia in sepsis and COVID-19. Front Immunol. 2023;14:1213510.

Claushuis TAM, van Vught LA, Scicluna BP, et al. Thrombocytopenia and dysregulated host response in sepsis. Blood. 2016;127(24):3062–72.

Coskun R, Mendil NOA, Kaya MM, et al. Incidence of thrombocytopenia in ICU patients and contributing factors. J Crit Intensive Care. 2016;7(1):3–6.

Duayer IF, Araújo MJCLN, Nihei CH, Barcelos MAF, Braga O, Britto ZML, Elias RM. Dialysis-related thrombocytopenia: a case report. J Bras Nefrol. 2022 Jan-Mar;44(1):116-120.

Ece C. Frequency and related factors of thrombocytopenia in ICU patients. J Biotech Strat Health Res. 2021;5(1):33–43.

Ghimire S, Ravi S, Budhathoki R, et al. Sepsis-induced thrombocytopenia: current insights. Eur J Haematol. 2021;106(3):301–5.

Greinacher A, Selleng K. Thrombocytopenia in ICU patients. Hematology Am Soc Hematol Educ Program. 2010;2010:135–43.

Haksoyler V, et al. Thrombocytopenia and mortality/morbidity in internal medicine ICUs. Cukurova Med J. 2019;44(2):632–41.

Knaus WA, Draper EA, Wagner DP, et al. APACHE II: a severity of disease classification system. Crit Care Med. 1985;13(10):818–29.

Levi M, Lowenberg EC. Thrombocytopenia in critically ill patients. Semin Thromb Hemost. 2008;34(5):417–24.

Li D, Wei Y, Zhang C, et al. Prognostic value of SOFA, APACHE II, and WBC count in septic patients: a retrospective study. Medicine (Baltimore). 2025;104(20):e42464.

Moreau D, Timsit JF, Vesin A, et al. Platelet count decline: an early prognostic marker in long-stay ICU patients. Chest. 2007;131(6):1735–41.

Péju E, Fouqué G, Charpentier J, et al. Clinical significance of thrombocytopenia in septic shock: a retrospective observational study. J Crit Care. 2023;76:154293.

Schupp T, Weidner K, Rusnak J, et al. Platelets in sepsis and septic shock: diagnostic and prognostic role. Platelets. 2022;34(1).

Semeraro F, Colucci M, Caironi P, et al. Platelet drop and fibrinolytic shutdown in sepsis. Crit Care Med. 2018;46(3):e221–8.

Sevim E, Çelik İ, Karlıdağ GE. Risk factors for mortality in nosocomial sepsis in ICUs. Fırat Med J. 2011;16(2):71–7.

Singer M, Deutschman CS, Seymour CW, et al. The third international consensus definitions for sepsis and septic shock (Sepsis-3). JAMA. 2016;315(8):801–10.

Sinha H, Maitra S, Anand RK, et al. Prognostic value of hematologic parameters in sepsis. Indian J Crit Care Med. 2021;25(6):660–7.

Strauss R, Wehler M, Mehler K, et al. Thrombocytopenia in medical ICU patients: bleeding, transfusion, outcome. Crit Care Med. 2002;30(8):1765–71.

Tang DM, Zhang HJ, Jing BW, et al. Platelets and systemic inflammatory response in critical illness. Chin Crit Care Med. 2003;15(1):35–7.

Vanderschueren S, De Weerdt A, Malbrain M, et al. Thrombocytopenia and prognosis in the ICU. Crit Care Med. 2000;28(6):1871–6.

Wang Q, Zhang G. Platelet count as a prognostic marker in ARDS. BMC Pulm Med. 2024;24(1):396.

Wang X, Zhang Q, Yan Y, et al. Pro-inflammatory cytokines and coagulation in septic co-infection. Shock. 2021;56(3):396–402.

Warren DK, Zack JE, Elward AM, et al. Nosocomial bloodstream infections in ICU patients. Clin Infect Dis. 2001;33(8):1329–35.