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PLATELET COUNT AND PLATELET INDICES (MEAN PLATELET VOLUME, PLATELET DISTRIBUTION WIDTH) AND RED CELL DISTRIBUTION WIDTH AND THEIR SIGNIFICANCE IN SEVERE SEPSIS: A HOSPITAL-BASED OBSERVATIONAL STUDY

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ABSTRACT

Background: Sepsis remains a leading cause of morbidity and mortality in intensive care settings worldwide. Early risk stratification is central to improving outcomes, but conventional biomarkers such as C-reactive protein (CRP) are limited by cost and delayed availability in resource-constrained settings. Platelet indices and red cell distribution width (RDW), both derived from a routine complete blood count, may serve as inexpensive surrogates of inflammation, coagulopathy and bone-marrow stress.

Aim: To evaluate the association of platelet count, platelet indices (mean platelet volume [MPV], platelet distribution width [PDW], plateletcrit [PCT]) and RDW with disease severity (SOFA, qSOFA, APACHE II scores) and clinical outcome in patients with severe sepsis.

Materials and Methods: This hospital-based observational study enrolled 90 adults with severe sepsis at a tertiary-care teaching hospital over two years. Platelet count, MPV, PDW, PCT, RDW and CRP were recorded at admission and serially at 24 and 72 hours, and correlated with SOFA, qSOFA and APACHE II scores and with clinical outcome (improvement, worsening or death). A p-value <0.05 was considered statistically significant.

Results: Thrombocytopenia was present in 60.0% of patients at admission and was associated with poorer outcome. MPV and PDW correlated positively with SOFA, qSOFA and APACHE II scores ($r = 0.34$-0.51, $p < 0.01$) and with CRP ($p < 0.05$), while platelet count correlated negatively with CRP ($p < 0.05$). Patients with elevated MPV/PDW had a markedly lower rate of clinical improvement (68.8%) and a higher rate of death (14.5%) than those with normal indices (95.2% and 2.4%, respectively). RDW was elevated (>14.5%) in 57.8% of patients and was associated with a longer ICU stay and greater ventilator requirement, with a significant decline over 72 hours in patients who improved ($p < 0.05$).

Conclusion: Platelet count, platelet indices and RDW - all obtainable from a single, inexpensive complete blood count - correlate closely with validated severity scores and clinical outcome in severe sepsis. Their combined interpretation, alongside CRP, offers a practical, low-cost tool for early risk stratification, particularly in resource-limited settings.

Keywords: *Sepsis; Platelet indices; Mean platelet volume; Platelet distribution width; Red cell distribution width; C-reactive protein; SOFA score; APACHE II.*

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INTRODUCTION

Sepsis is defined as life-threatening organ dysfunction caused by a dysregulated host response to infection, and despite advances in critical care it continues to carry high morbidity and mortality worldwide [1]. The Sepsis-3 consensus

operationalises this definition as an acute increase of two or more points in the Sequential Organ Failure Assessment (SOFA) score in the setting of infection, with progression to septic shock markedly increasing the risk of death [1,2]. Because clinical presentation is heterogeneous and the underlying pathophysiology complex, early identification of high-risk patients remains one of the most important unmet needs in sepsis care [3]. C-reactive protein (CRP), an acute-phase reactant synthesised by hepatocytes under the stimulus of interleukin-6, is the inflammatory marker most widely used at the bedside because of its availability and low cost.[4,5] However, CRP lacks specificity, peaks with a delay of several hours, and provides no direct information about coagulation or microvascular dysfunction - limitations that have prompted interest in complementary, more mechanistically informative markers [4,5]. Platelets are increasingly recognised as active participants in the septic process rather than passive elements of haemostasis. Activated platelets interact with leucocytes and endothelium to promote microvascular thrombosis and capillary leak, and their increased consumption and destruction commonly produce the thrombocytopenia frequently observed in sepsis.[6-8] Beyond platelet count, the indices mean platelet volume (MPV) and platelet distribution width (PDW) capture the compensatory release of larger, less mature and more reactive platelets from the bone marrow, while plateletcrit (PCT), the product of platelet count and MPV, reflects total circulating platelet mass [9,10]. Elevated MPV and PDW, and reduced PCT, have repeatedly been linked to greater disease severity and mortality in sepsis [10,11]. Red cell distribution width (RDW), a routinely reported measure of variation in erythrocyte size, has similarly emerged as a marker of systemic inflammation, oxidative stress and impaired erythropoiesis. Elevated RDW predicts mortality in critically ill patients independent of established severity scores [12,13]. Because platelet indices and RDW arise from distinct but interlinked pathophysiological processes - platelet activation and turnover on one hand, marrow and erythrocyte stress on the other - their combined assessment, together with CRP, may offer a more complete picture of sepsis severity than any single parameter. Since advanced biomarkers and formal severity-score calculation are not uniformly available, particularly in resource-limited settings, there is a clear rationale for evaluating whether routinely available haematological indices can meaningfully stratify risk in severe sepsis. This study was undertaken to determine the association of platelet count, platelet indices and RDW with validated severity scores (SOFA, qSOFA, APACHE II) and with clinical outcome in patients with severe sepsis, and specifically to examine the correlation of MPV, PDW and RDW with disease severity, with CRP, and with each other.

MATERIALS AND METHODS

Study design and setting

This hospital-based observational study was conducted over a period of two years in the Department of General Medicine, S.S. Institute of Medical Sciences and Research Centre, Davangere, Karnataka, India.

Study Population

Patients aged more than 18 years attending the medicine outpatient department or admitted to the inpatient wards, who fulfilled clinical criteria for sepsis - fever (oral temperature >38°C) or hypothermia (<36°C), tachypnoea (>24 breaths/min), tachycardia (>90 beats/min), and leucocytosis (>12,000/µL), leucopenia (<4,000/µL) or >10% band forms, with proven or suspected microbial infection - were enrolled after written informed consent.

Exclusion criteria

Age <18 years; active haemorrhage; known haematological disorders (anaemia, hypersplenism, lymphoma, leukaemia, marrow suppression); recent blood transfusion; anticoagulant therapy; pregnancy; and inability to provide informed consent.

Sample size

Based on an anticipated sensitivity of 93.9% and an acceptable margin of error of 5% at a 95% confidence level ($Z\alpha = 1.96$), the calculated minimum sample size was 88 patients; this was rounded up to 90 for the study.

Investigations

All patients underwent a complete blood count from which platelet count, MPV, PDW and RDW were derived, together with serum CRP, at admission and at 24 and 72 hours. Reference ranges applied were: platelet count 150-450 $\times 10^9$/L (thrombocytopenia defined as <150 $\times 10^9$/L); MPV 7.5-11.5 fL; PDW 9-14 fL; PCT 0.22-0.24%; RDW 11.5-14.5%; and CRP <5 mg/L as normal.

Severity and outcome assessment

Disease severity was classified according to the Sepsis-3 framework using the SOFA, quick SOFA (qSOFA) and APACHE II scoring systems, based on clinical and laboratory parameters recorded at admission.[1,2,14] Septic shock was defined as sepsis with persistent hypotension requiring vasopressor support to maintain a mean arterial pressure ≥65 mmHg together with a serum lactate >2 mmol/L despite adequate fluid resuscitation. Patients were followed through their hospital stay and outcome was classified as clinical improvement, clinical worsening, or death.

Statistical analysis

Data were entered in Microsoft Excel and analysed using SPSS statistical software. Quantitative variables were expressed as mean ± standard deviation and qualitative variables as frequencies and percentages. Student's t-test and correlation analysis (Pearson/Spearman, as appropriate) were used to compare groups and to assess relationships between haematological parameters, severity scores and CRP. A p-value <0.05 was taken as statistically significant.

Ethical considerations

The study was approved by the Institutional Ethics Committee of S.S. Institute of Medical Sciences and Research Centre, Davangere (approval number to be inserted), and written informed consent was obtained from all participants before enrolment.

RESULTS

Of the 90 patients with severe sepsis enrolled, the majority (51.1%) were aged 51-60 years, and 73.3% were older than 40 years. There was a mild male predominance (52.2% vs 47.8%). At admission, fever or hypothermia was the most frequent

finding (86.7%), followed by tachycardia (81.1%), tachypnoea (74.4%) and leucocyte count abnormality (69.9%); all patients had proven or suspected infection. The respiratory tract (33.3%) and urinary tract (27.8%) were the commonest identifiable sources of sepsis, and microbiological cultures were positive in 34.4% of patients, most often from urine (20.0%). These findings are summarised in Table 01. Thrombocytopenia was present in 60.0% of patients at admission, with normal counts in 32.2% and thrombocytosis in 7.8%. The proportion with thrombocytopenia fell progressively over the course of treatment, from 60.0% at admission to 52.2% at 24 hours and 45.6% at 72 hours, while the proportion with normal counts rose correspondingly. A parallel decline was seen in platelet activation: the proportion with elevated MPV fell from 48.9% at admission to 33.3% at 72 hours, and elevated PDW fell from 46.7% to 31.1% over the same interval (Table 02).

Both MPV and PDW showed a statistically significant, graded relationship with severity scores. Mean MPV and PDW were significantly higher in patients with SOFA ≥6 (12.1±1.3 fL and 15.4±2.2 fL) than SOFA <6 (10.6±1.1 fL and 13.8±1.9 fL; $p = 0.002$ and $p = 0.004$ respectively), a pattern mirrored for qSOFA ≥2 versus <2 ($p = 0.006$ and $p = 0.009$) and, most strongly, for APACHE II ≥20 versus <20 ($p < 0.001$ for both) (Table 3). On correlation analysis, APACHE II showed the strongest association with MPV ($r = 0.51$, $p < 0.001$) and PDW ($r = 0.47$, $p < 0.001$), followed by SOFA ($r = 0.42$ and 0.39) and qSOFA ($r = 0.36$ and 0.34). CRP correlated positively with MPV and PDW ($p < 0.05$ for both) and negatively with platelet count ($p < 0.05$), and mean CRP rose progressively across increasing SOFA, qSOFA and APACHE II severity categories (Table 04). RDW was elevated (>14.5%) in 57.8% of patients at admission. Elevated RDW was associated with a longer ICU stay (9.2±3.6 vs 5.8±2.1 days) and a greater requirement for ventilatory support (53.8% vs 26.3%) compared with normal RDW ($p < 0.05$ for both), although mortality did not differ significantly between the two groups (7.7% vs 7.9%). Mean RDW declined progressively from 15.6±1.8% at admission to 15.1±1.6% at 24 hours and 14.6±1.4% at 72 hours ($p < 0.05$) (Table 05).

Overall, 84.4% of patients showed clinical improvement, 7.8% worsened, and 7.8% died. Outcome varied by platelet count category: improvement was seen in 89.7% of patients with a normal platelet count compared with 81.5% of those with thrombocytopenia and 71.4% of those with thrombocytosis, while death occurred more often in the thrombocytopenia (9.3%) and thrombocytosis (14.3%) groups than in those with normal counts (3.4%). The prognostic impact of platelet indices was more pronounced still: patients with elevated MPV/PDW had a markedly lower improvement rate (68.8%) and higher rates of worsening (16.7%) and death (14.5%) than those with normal indices (95.2%, 2.4% and 2.4% respectively) (Table 06).

Table 01: Baseline demographic and clinical characteristics of the study population (N = 90)

Characteristic	n	%
Age group: ≤30 years	13	14.5
Age group: 31-40 years	11	12.2
Age group: 41-50 years	20	22.2
Age group: 51-60 years	46	51.1
Male sex	47	52.2
Female sex	43	47.8
Fever / hypothermia	78	86.7
Tachycardia (>90/min)	73	81.1
Tachypnoea (>24/min)	67	74.4
Leucocyte count abnormality	63	69.9
Source: respiratory tract	30	33.3
Source: urinary tract	25	27.8
Source: gastrointestinal tract	17	18.9
Source: skin / soft tissue	10	11.1
Source: no identifiable focus	8	8.9
Microbiological culture positive	31	34.4
Clinical outcome: improved	76	84.4
Clinical outcome: worsened	7	7.8
Clinical outcome: death	7	7.8

Table 02: Temporal trend of platelet count and platelet indices (N = 90)

Time point	Thrombocytopenia n (%)	Normal platelets n (%)	Thrombocytosis n (%)	Elevated MPV n (%)	Elevated PDW n (%)
Admission	54 (60.0)	29 (32.2)	7 (7.8)	44 (48.9)	42 (46.7)
24 hours	47 (52.2)	34 (37.8)	9 (10.0)	38 (42.2)	36 (40.0)
72 hours	41 (45.6)	37 (41.1)	12 (13.3)	30 (33.3)	28 (31.1)

Table 03: Association between severity scores and platelet indices (MPV, PDW)

Severity score	Category (n)	Mean MPV ± SD (fL)	P value	Mean PDW ± SD (fL)	P value
SOFA	<6 (n=62)	10.6 ± 1.1	-	13.8 ± 1.9	-
SOFA	≥6 (n=28)	12.1 ± 1.3	0.002	15.4 ± 2.2	0.004
qSOFA	<2 (n=52)	10.7 ± 1.0	-	13.9 ± 1.8	-
qSOFA	≥2 (n=38)	11.9 ± 1.4	0.006	15.1 ± 2.1	0.009
APACHE II	<20 (n=57)	10.5 ± 1.1	-	13.7 ± 1.7	-
APACHE II	≥20 (n=33)	12.3 ± 1.5	<0.001	15.6 ± 2.3	<0.001

Table 04: Correlation of severity scores and CRP with platelet indices and platelet count

Parameter 1	Parameter 2	r / direction	P value
SOFA score	MPV	0.42	0.001
SOFA score	PDW	0.39	0.003
qSOFA score	MPV	0.36	0.006
qSOFA score	PDW	0.34	0.009
APACHE II score	MPV	0.51	<0.001
APACHE II score	PDW	0.47	<0.001
CRP	MPV	Positive	<0.05
CRP	PDW	Positive	<0.05
CRP	Platelet count	Negative	<0.05

Table 05: Red cell distribution width (RDW): association with severity indicators, clinical outcome, and trend over time

Parameter	Normal RDW (n=38)	Elevated RDW (n=52)	P value
Clinical improvement, n (%)	30 (78.9)	46 (88.5)	-
Worsened, n (%)	5 (13.2)	2 (3.8)	-
Death, n (%)	3 (7.9)	4 (7.7)	NS
Duration of ICU stay, days (mean ± SD)	5.8 ± 2.1	9.2 ± 3.6	<0.05
Ventilator requirement, %	26.3	53.8	<0.05

Trend of mean RDW over time: Admission 15.6 ± 1.8%; 24 hours 15.1 ± 1.6%; 72 hours 14.6 ± 1.4% ($p < 0.05$ for decline over time).

Table 06: Clinical outcome according to platelet count category and platelet index status

Category	Improved n (%)	Worsened n (%)	Death n (%)
Thrombocytopenia (n=54)	44 (81.5)	5 (9.3)	5 (9.3)
Normal platelet count (n=29)	26 (89.7)	2 (6.9)	1 (3.4)

Category	Improved n (%)	Worsened n (%)	Death n (%)
Thrombocytosis (n=7)	5 (71.4)	1 (14.3)	1 (14.3)
Elevated MPV / PDW (n=48)	33 (68.8)	8 (16.7)	7 (14.5)
Normal MPV / PDW (n=42)	40 (95.2)	1 (2.4)	1 (2.4)

DISCUSSION

In this cohort of 90 patients with severe sepsis, more than 70% were older than 40 years and there was a mild male predominance, a pattern consistent with several previous reports [15, 16]. Advancing age is associated with immunosenescence, greater comorbidity burden and reduced haematopoietic reserve, all of which are likely to exaggerate platelet consumption and activation in sepsis; comparator studies have similarly reported more pronounced abnormalities in platelet indices and higher mortality in older patients [10, 16]. The high prevalence of classical systemic inflammatory response features at admission - fever or hypothermia, tachycardia, tachypnoea and leucocyte abnormalities - reflects a robust early inflammatory response that, in this and other cohorts, runs in parallel with the haematological derangements central to this study.[10,15] The respiratory and urinary tracts were the leading identifiable sources of infection, in keeping with previously reported distributions of the focus of sepsis in similar hospital-based cohorts. Thrombocytopenia was the most common haematological abnormality in this study and, consistent with prior work, was associated with poorer clinical outcome [10,15,17]. Sepsis-associated thrombocytopenia arises from a combination of increased peripheral consumption at sites of endothelial injury, immune-mediated destruction, sequestration and, in more severe cases, bone-marrow suppression by circulating cytokines [6,8,10]. The concurrent rise in MPV observed in patients with lower platelet counts is consistent with a compensatory release of larger, younger platelets from the marrow, a pattern also described by Guclu et al. and Gupta et al [10,15].

MPV and PDW both showed a graded, statistically significant association with disease severity in this study, rising with increasing SOFA, qSOFA and APACHE II scores and correlating positively with CRP. This is in keeping with a substantial body of literature: Zhang et al. reported that a higher MPV-to-platelet-count ratio independently predicted ICU mortality in sepsis;[16] Samuel et al., Sharma et al. and Taha et al. found that persistently elevated MPV during follow-up carried a worse prognosis than an isolated admission value;[18-20] and the systematic review and meta-analysis by Tajarernmuang et al. confirmed that a rising MPV trend was more predictive of death than a single baseline measurement across a range of critically ill populations.[21] Similar associations between elevated MPV and adverse outcome have been reported in paediatric sepsis by Sayed et al., and in broader critically ill populations by Kim et al. and Zampieri et al.[17,22,11] Mechanistically, larger, more reactive platelets carry higher concentrations of thromboxane A_2 and β-thromboglobulin, and cytokine-driven megakaryopoiesis in sepsis favours their release into the circulation, providing a biological basis for the association between MPV, PDW and severity observed here and elsewhere.[9,10] PDW, which captures the heterogeneity rather than the mean of platelet volume, behaves similarly and has been reported by Gupta et al. and Guclu et al. to rise even before overt thrombocytopenia becomes apparent, suggesting a potential role as an early marker of platelet activation.[10,15] Although plateletcrit was not analysed as an independent endpoint in every comparator study, the pattern observed in this cohort - a falling platelet count with rising MPV - implies a net reduction in total circulating platelet mass in patients with more severe disease, consistent with reports by Gupta et al. and Mangalesh et al. that reduced PCT tracks closely with disease severity and mortality.[15,23] This finding underlines that platelet count, MPV and PCT provide complementary rather than redundant information, since a normal platelet count with elevated MPV may still indicate significant platelet turnover.

RDW was elevated in the majority of patients in this study and was associated with a longer ICU stay and a greater need for ventilatory support, findings that align with those of Torres et al. and Krishna et al., who reported RDW to be an independent predictor of adverse outcome in sepsis, including after adjustment for established severity scores.[12,13] The biological basis for this relationship is multifactorial: pro-inflammatory cytokines such as interleukin-6 and tumour necrosis factor-alpha impair erythroid maturation and iron handling, while oxidative stress shortens red-cell lifespan and increases anisocytosis, together producing the rise in RDW seen with worsening systemic inflammation.[12,24] The progressive fall in RDW over 72 hours observed in this cohort, paralleling the fall in MPV and PDW, suggests that serial rather than single measurements may better capture the trajectory of illness, an observation also made for MPV by Tajarernmuang et al. and for combined red-cell and platelet indices by Dubey et al.[21,25] Notably, while elevated RDW in this study was associated with greater illness severity, it was not independently associated with mortality, in contrast to the platelet indices; this may reflect the relatively small number of deaths in this cohort and warrants confirmation in larger studies.

The correlation between CRP and platelet indices observed here - positive with MPV and PDW, negative with platelet count - supports the concept that CRP-reflected inflammatory burden and platelet activation/consumption evolve together during sepsis, a relationship also described by Gupta et al. and Maricar et al.[15,26] Because CRP indicates the magnitude of inflammation while platelet indices and RDW additionally capture platelet turnover and marrow/erythrocyte stress, their combined interpretation is likely to provide more complete prognostic information than any parameter used in isolation, a conclusion supported by Kumar et al., who found progressively

worse platelet-index profiles across sepsis, severe sepsis and septic shock [27].

Taken together, these findings support the clinical utility of a simple, complete-blood-count-based panel - platelet count, MPV, PDW, PCT and RDW - interpreted alongside CRP, for early risk stratification in severe sepsis. Because all of these parameters are generated automatically by routine automated haematology analysers at negligible additional cost, this approach is particularly attractive in resource-limited settings where advanced biomarkers, serial lactate measurement or dedicated severity-score calculation may not be readily available, a point also emphasised by Saharia et al., Yadav et al. and Gao et al. in similarly designed studies [28-30].

Strengths and limitations

This study systematically evaluated multiple platelet indices and RDW together, rather than relying on a single parameter, and correlated them with three independent, validated severity scores as well as with clinical outcome, using standardised sepsis diagnostic criteria. It is, however, a single-centre study with a modest sample size (n = 90), which limited subgroup analysis and precluded multivariate adjustment for potential confounders such as comorbidities. Platelet indices and RDW were not measured beyond 72 hours, and CRP was the only inflammatory marker used, without comparison to procalcitonin or interleukin-6. These limitations should be addressed in larger, multicentre studies with longer serial follow-up.

CONCLUSION

Platelet count, platelet indices (MPV, PDW, PCT) and RDW - parameters obtainable from a single, inexpensive complete blood count - correlated significantly with validated severity scores (SOFA, qSOFA, APACHE II) and with clinical outcome in this cohort of patients with severe sepsis. Thrombocytopenia with elevated MPV and PDW, reduced PCT, and elevated RDW characterised patients with more severe disease and poorer outcome. When interpreted together with CRP, these routinely available haematological parameters provide a practical, cost-effective tool for early risk stratification in severe sepsis, and may be particularly valuable in resource-limited settings where advanced biomarkers are not readily accessible. Larger, multicentre studies with serial measurements are warranted to validate and refine their prognostic role.

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AUTHOR CONTRIBUTIONS

Hoysalakumar D.P.: conceptualisation, study supervision, manuscript review and editing. Dr. Satheesha B.C.: contribution details to be confirmed. Pradeep G.S.: literature review, data interpretation, manuscript review. Sharath Kumar V.T.: data collection, statistical analysis, drafting of the original manuscript. All authors approved the final version.

CONFLICT OF INTEREST

None declared.

SOURCE OF FUNDING

Nil.

ETHICAL CLEARANCE

Obtained from the Institutional Ethics Committee, S.S. Institute of Medical Sciences and Research Centre, Davangere (approval number to be inserted).

REFERENCES

1. Singer M, Deutschman CS, Seymour CW, Shankar-Hari M, Annane D, Bauer M, et al. The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). JAMA. 2016;315(8):801-810.
2. Seymour CW, Liu VX, Iwashyna TJ, Brunkhorst FM, Rea TD, Scherag A, et al. Assessment of clinical criteria for sepsis: for the Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). JAMA. 2016;315(8):762-774.
3. Ventetuolo CE, Levy MM. Sepsis: a clinical update. Clin J Am Soc Nephrol. 2008;3(2):571-577.
4. Fan SL, Miller NS, Lee J, Remick DG. Diagnosing sepsis - the role of laboratory medicine. Clin Chim Acta. 2016;460:203-210.
5. Povoa P, Coelho L, Almeida E, Fernandes A, Mealha R, Moreira P, et al. C-reactive protein as a marker of infection in critically ill patients. Clin Microbiol Infect. 2005;11:101-108.
6. Golebiewska EM, Poole AW. Platelet secretion: from haemostasis to wound healing and beyond. Blood Rev. 2015;29:153-162.
7. Margetic S. Inflammation and haemostasis. Biochem Med (Zagreb). 2012;22:49-62.
8. Venkata C, Kashyap R, Farmer JC, Afessa B. Thrombocytopenia in adult patients with sepsis: incidence, risk factors, and its association with clinical outcome. J Intensive Care. 2013;1(1):9.
9. Vardon-Bounes F, Ruiz S, Gratacap MP, Garcia C, Payrastre B, Minville V. Platelets are critical key players in sepsis. Int J Mol Sci. 2019;20(14):3494.
10. Guclu E, Durmaz Y, Karabay O. Effect of severe sepsis on platelet count and their indices. Afr Health Sci. 2013;13(2):333-338.
11. Zampieri FG, Ranzani OT, Sabatoski V, de Souza HP, Barbeiro H, da Neto LMC, et al. An increase in mean platelet volume after admission is associated with higher mortality in critically ill patients. Ann Intensive Care. 2014;4(1):20.
12. Torres VM, Royuela A, Rubio EM, et al. Red blood cell distribution width as a prognostic factor in sepsis. J Crit Care. 2022;71:154069.
13. Krishna V, et al. Red cell distribution width as a mortality predictor in patients with sepsis: a prospective observational study. Indian J Med Res. 2020;151(3):285-291.
14. Jones AE, Trzeciak S, Kline JA. The Sequential Organ Failure Assessment score for predicting outcome in

patients with severe sepsis and evidence of hypoperfusion at the time of emergency department presentation. Crit Care Med. 2009;37(5):1649-1654.
15. Gupta A, Kumar S, Acharya S, Sarode R, Agrawal S, Gemnani R, et al. Utility of platelet indices as prognostic markers of sepsis: a medical intensive care unit-based cross-sectional study at a rural setup. Cureus. 2024;16(2):e54490.
16. Zhang Z, Xu X, Ni H. Mean platelet volume to platelet count ratio as a prognostic marker in patients with sepsis. PLoS One. 2022;17(2):e0262356.
17. Sayed SZ, Mahmoud MM, Moness HM, Mousa SO. Admission platelet count and indices as predictors of outcome in children with severe sepsis: a prospective hospital-based study. BMC Pediatr. 2020;20:387.
18. Samuel D, Bhat AN, Joseph J, et al. Platelet indices as predictive markers of prognosis in critically ill patients: a prospective study. Indian J Crit Care Med. 2020;24(9):817-822.
19. Sharma DJ, Ganguly S, Rakesh M, et al. Utility of platelet indices as a predictive marker in sepsis: an observational study. Cureus. 2023;15(4):e38095.
20. Taha RS, Afandy ME, Elbadawi AH, Abd El Ghafar MS. Platelet indices in critically ill septic patients as a predictor of mortality. Egypt J Anaesth. 2023;39(1):56-62.
21. Tajarernmuang P, Phrommintikul A, Limsukon A, Pothirat C, Chittawatanarat K. The role of mean platelet volume as a predictor of mortality in critically ill patients: a systematic review and meta-analysis. Crit Care Res Pract. 2016;2016:4370834.
22. Kim CH, Kim SJ, Lee MJ, Kwon YE, Kim YL, Park KS, et al. An increase in mean platelet volume from baseline is associated with mortality in patients with severe sepsis or septic shock. PLoS One. 2015;10(3):e0119437.
23. Mangalesh S, Dudani S, Malik A. Platelet indices and their kinetics predict mortality in patients of sepsis. Indian J Hematol Blood Transfus. 2021;37(4):600-608.
24. Acikgoz S, Akduman D, Eskici ZM, et al. Thrombocyte and erythrocyte indices in sepsis and disseminated intravascular coagulation. J Med Biochem. 2012;31:60-64.
25. Dubey A, Kumar S, Acharya S, Wanjari AK, Bawankule S, Agrawal S, et al. Impact of red cell and platelet distribution width in patients of medical intensive care unit. J Lab Physicians. 2021;13(4):309-316.
26. Maricar MH, Kathiravan AR, Balamurugan AR, Radhakrishnan V. Study of effect of sepsis on platelet count and their indices - mean platelet volume and platelet distribution width and their prognostic significance and correlation with C-reactive protein. Int J Acad Med Pharm. 2023;5(6):989-993.
27. Kumar S, Patel R, Shah P. Platelet indices as predictive markers for sepsis and septic shock. Glob J Med Clin Case Rep. 2022;12:294.
28. Saharia HK, Das O, Vignesh S. Correlation of platelet indices with severity of sepsis patients in ICU: a prospective observational study. Int J Curr Pharm Rev Res. 2025;17(3):133-140.
29. Yadav S, Chaitany BSB, Meena LP, Kumar S, Gupta S, Rai A. Study to evaluate the role of platelet count and platelet indices as predictors of outcome in patients with sepsis at a tertiary care hospital. J Contemp Clin Pract. 2025;11(2):637-643.
30. Gao Y, Li Y, Yu X, Guo S, Ji X, Sun T, et al. The impact of various platelet indices as prognostic markers of septic shock. PLoS One. 2014;9(8):e103761.