

Study of Platelet Indices for Assessment of Severe Sepsis: A Hospital-Based Prospective Observational Study.

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Abstract

Background: Sepsis is a leading cause of intensive care unit (ICU) morbidity. Platelet indices—mean platelet volume (MPV), platelet distribution width (PDW), and immature platelet fraction (IPF)—are inexpensive, rapidly available parameters that may reflect the inflammatory and thrombopoietic alterations of sepsis. This study evaluated the utility of these indices in assessing sepsis severity. **Methods:** A hospital-based prospective observational study was conducted in a tertiary care centre North India, from April 2024 to September 2025. Eighty-four patients aged 18–65 years admitted to the ICU with sepsis or severe sepsis/septic shock were enrolled after informed consent and institutional ethics approval. Patients with trauma, pulmonary embolism, myocardial infarction, immune thrombocytopenic purpura, platelet dysfunction, coagulopathy, or non-infectious causes of sepsis were excluded. Complete blood count with platelet indices, C-reactive protein (CRP), and a standard panel of investigations were performed at enrolment. Data were analysed using SPSS 29.0; Student's t-test and chi-square/Fisher's exact test were applied, with $p < 0.05$ considered significant. **Results:** Of 84 patients, 51 (60.71%) had severe sepsis/septic shock and 33 (39.29%) had sepsis. Pneumonia (33.3%) was the commonest source. Compared with sepsis, severe sepsis was associated with significantly higher CRP (202.08 ± 41.32 vs 153.07 ± 41.41 mg/L; $p < 0.001$), lower platelet count (185 ± 86 vs $263 \pm 68 \times 10^3/\mu\text{L}$; $p < 0.001$), higher MPV (8.08 ± 0.87 vs 7.61 ± 0.85 fL; $p = 0.03$), higher PDW (18.02 ± 0.87 vs $17.61 \pm 0.69\%$; $p = 0.018$), and higher IPF (6.17 ± 5.64 vs $4.08 \pm 3.69\%$; $p = 0.04$). **Conclusion:** MPV, PDW, and IPF are simple, low-cost haematological markers that correlate significantly with sepsis severity. Platelet indices may complement clinical severity scoring in early risk stratification of septic patients, though larger prospective studies are warranted to confirm their role in outcome prediction.

Keywords: Sepsis; severe sepsis; mean platelet volume; platelet distribution width; immature platelet fraction; C-reactive protein; ICU.



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INTRODUCTION

Sepsis is a life-threatening organ dysfunction caused by a dysregulated host response to infection and remains a leading cause of morbidity among critically ill patients worldwide. Despite advances in antimicrobial therapy and organ support, severe sepsis and septic shock. ¹⁻³ Rudd et al. estimated approximately 48.9 million sepsis cases and 11 million sepsis-related deaths globally in 2017, with a disproportionately greater burden in low- and middle-income countries.⁴

Sepsis is characterised by immune dysregulation, endothelial injury, coagulation activation, microvascular dysfunction, and multi-organ impairment. Cytokine-driven inflammation activates the coagulation cascade, producing platelet consumption, microthrombi, and tissue ischaemia.^{5,6} Consequently, haematological derangements—particularly thrombocytopenia—are common and correlate with disease severity and outcome.⁷ However, platelet count alone may not

adequately reflect platelet function or the bone marrow's thrombopoietic response. Platelet indices generated routinely by automated haematology analysers—mean platelet volume (MPV), platelet distribution width (PDW), plateletcrit (PCT), and immature platelet fraction (IPF)—offer additional insight into platelet biology at no incremental cost. Larger, younger platelets released in response to peripheral consumption produce a rise in MPV and IPF, while heterogeneity in platelet size manifests as elevated PDW. Multiple studies have linked these parameters to sepsis severity.^{8–12} Compared with composite scores such as SOFA or APACHE II, platelet indices are objective, rapidly available, and accessible even in resource-limited settings.¹³

Against this background, the present study was designed to evaluate platelet indices (MPV, PDW, and IPF) as markers of sepsis severity in patients admitted to a tertiary care ICU in Jaipur, India.

MATERIALS AND METHODS

Study design and setting

This was a hospital-based, prospective observational study conducted in the Department of General Medicine, Mahatma Gandhi Medical College & Hospital, Jaipur, Rajasthan, from April 2024 to September 2025 (1.5 years).

Ethics and consent

The study was approved by the Institutional Ethics Committee. Written informed consent was obtained from each participant; in patients unable to consent due to their clinical condition, consent was obtained from a legally authorised attendant or relative.

Study population

Adults aged 18–65 years of either sex admitted to the ICU with sepsis fulfilling the inclusion criteria were enrolled consecutively. Exclusion criteria were trauma, pulmonary embolism, myocardial infarction, known immune thrombocytopenic purpura, platelet dysfunction disorders, coagulopathy, and non-infectious causes of a systemic inflammatory response.

Definitions

Sepsis was defined as Systemic Inflammatory Response Syndrome (SIRS) with a proven or suspected source of infection. SIRS required ≥ 2 of: temperature $>38^{\circ}\text{C}$ or $<36^{\circ}\text{C}$; heart rate $>90/\text{min}$; respiratory rate $>20/\text{min}$ or $\text{PaCO}_2 <32 \text{ mmHg}$; total leukocyte count $>12,000/\text{mm}^3$ or $<4,000/\text{mm}^3$ or $>10\%$ immature forms. Severe sepsis was defined as sepsis with organ dysfunction and/or hypoperfusion abnormality.

Sample size

Sample size was estimated using MedCalc software (comparison of means) with $\alpha = 0.01$, $\beta = 0.10$, mean difference = 2.6, standard deviation = 3 in each group, and 1:1 allocation, yielding 84 patients.

Investigations and data collection

All patients underwent a complete blood count with platelet indices (MPV, PDW, IPF), arterial blood gas analysis, urine routine, renal and liver function tests with total protein, prothrombin time/INR, activated partial thromboplastin time, CRP, random blood sugar, serum electrolytes, chest radiography, erythrocyte sedimentation rate, and ultrasonography of the abdomen. Treatment followed standard hospital protocol; no intervention was performed for the study. Clinical, laboratory, and outcome data were captured on a pre-designed proforma.

Statistical analysis

Data were analysed using SPSS version 29.0. Continuous variables are expressed as mean $\pm$ standard deviation and compared using Student's t-test; categorical variables are expressed as frequencies (percentages) and compared using the chi-square test or Fisher's exact test, as appropriate. A two-sided p-value <0.05 was considered statistically significant.

RESULTS

Eighty-four patients fulfilling the inclusion criteria were analysed. The majority ($n = 51$; 60.71%) had severe sepsis/septic shock and 33 (39.29%) had sepsis (Table 1). Pneumonia was the most frequent source of infection (33.3%), followed by urinary tract infection (21.4%) and intra-abdominal infection (14.2%) (Table 4).

Table 1. Distribution of patients according to severity of sepsis ($n = 84$)

Severity	No. of patients (n)	Percentage (%)
Sepsis	33	39.29
Severe sepsis / septic shock	51	60.71
Total	84	100.00

Table 2. Age distribution among patients with sepsis and severe sepsis

Age group (years)	Sepsis n (%)	Severe sepsis n (%)
25–34	1 (3.03)	0
35–44	1 (3.03)	0
45–54	2 (6.06)	0
55–64	21 (63.64)	42 (82.35)
>64	8 (24.24)	9 (17.65)
Total	33	51
Mean $\pm$ SD (years)	59.52 $\pm$ 8.12	62.33 $\pm$ 2.40
Overall mean $\pm$ SD	61.23 $\pm$ 5.59	p = 0.057

The mean age was 62.33 $\pm$ 2.40 years in severe sepsis versus 59.52 $\pm$ 8.12 years in sepsis (p = 0.057). Most patients in both groups were aged 55–64 years (Table 2). Males predominated in both groups (63.64% in sepsis; 56.86% in severe sepsis), but gender distribution did not differ significantly (p = 0.48) (Table 3).

Table 3. Gender distribution among patients with sepsis and severe sepsis

Gender	Sepsis n (%)	Severe sepsis n (%)
Female	12 (36.36)	22 (43.14)
Male	21 (63.64)	29 (56.86)
Total	33	51
p-value	0.48	

Table 4. Distribution of causes of sepsis in the study population

Cause of sepsis	No. of patients	Percentage (%)
Pneumonia (respiratory infection)	28	33.3
Urinary tract infection	18	21.4
Intra-abdominal infections	12	14.2
Skin and soft-tissue infections	9	10.7
Bloodstream infection (primary bacteraemia)	7	8.3
Central nervous system infections	4	4.7
Post-operative infections	3	3.5
Miscellaneous	3	3.5
Total	84	100.0

Inflammatory and haematological parameters by severity

Mean CRP was significantly higher in severe sepsis than in sepsis (202.08 $\pm$ 41.32 vs 153.07 $\pm$ 41.41 mg/L; p < 0.001), while mean platelet count was significantly lower (185 $\pm$ 86 vs 263 $\pm$ 68 $\times 10^3/\mu\text{L}$; p < 0.001). Leukocyte and neutrophil counts did not differ significantly between groups (Table 5).

Table 5. Inflammatory and haematological parameters in sepsis vs severe sepsis

Parameter	Sepsis (n = 33)	Severe sepsis (n = 51)	p-value
CRP (mg/L)	153.07 $\pm$ 41.41	202.08 $\pm$ 41.32	<0.001
Leukocytes (/ μL)	12,623 $\pm$ 5,262	14,808 $\pm$ 5,041	0.06
Neutrophils (/ μL)	12,489 $\pm$ 4,411	12,112 $\pm$ 4,023	0.68
Platelets ($\times 10^3/\mu\text{L}$)	262.77 $\pm$ 68.30	185.12 $\pm$ 86.25	<0.001

Platelet indices by severity

All three platelet indices were significantly higher in severe sepsis compared with sepsis: MPV (8.08 $\pm$ 0.87 vs 7.61 $\pm$ 0.85 fL; p = 0.03), PDW (18.02 $\pm$ 0.87 vs 17.61 $\pm$ 0.69%; p = 0.018), and IPF (6.17 $\pm$ 5.64 vs 4.08 $\pm$ 3.69%; p = 0.04) (Tables 6).

Table 6. Comparison of Platelet Indices According to Severity of Sepsis

Platelet Index	Sepsis (n = 33) Mean $\pm$ SD	Severe Sepsis (n = 51) Mean $\pm$ SD	p-value
Mean Platelet Volume (MPV, fL)	7.61 $\pm$ 0.85	8.08 $\pm$ 0.87	0.03
Platelet Distribution Width (PDW, %)	17.61 $\pm$ 0.69	18.02 $\pm$ 0.87	0.018
Immature Platelet Fraction (IPF, %)	4.08 $\pm$ 3.69	6.17 $\pm$ 5.64	0.04

DISCUSSION

In this prospective observational study of 84 ICU patients, platelet indices—MPV, PDW, and IPF—were significantly elevated in severe sepsis compared with sepsis. The findings support the role of these inexpensive, routinely reported parameters as adjuncts to clinical assessment in early risk stratification.

Prevalence and aetiology

Severe sepsis/septic shock predominated in our cohort (60.71%), likely reflecting the tertiary referral nature of the study centre, delayed presentation, and prior treatment at peripheral facilities. Comparable variability has been reported across settings: the multicentric INDICAP study from India documented severe sepsis in 28.3% of ICU admissions, whereas European studies have reported figures around 27%.¹⁴⁻¹⁷ Differences likely reflect heterogeneity in setting, study design, sepsis definitions (SIRS-based vs Sepsis-3), and population characteristics.

Pneumonia was the leading source of infection (33.3%), followed by urinary tract and intra-abdominal infections (21.4% and 14.2%, respectively). This distribution is consistent with the INDICAP and ANZICS cohorts and with Western series, in which respiratory infection consistently ranks first.^{3,14} Contributing factors include the rapid systemic dissemination from extensive alveolar involvement, frequent catheterisation predisposing to urinary infection, and the prevalence of post-procedural and skin/soft-tissue infections in older comorbid patients.

Demographic features

The mean age was higher in severe sepsis than in sepsis (62.33 ± 2.40 vs 59.52 ± 8.12 years; $p = 0.057$) and most patients in both groups were 55–64 years old, but age was not a significant determinant of severity or outcome in this cohort. A male predominance was observed without statistical significance for severity ($p = 0.48$) or outcome ($p = 0.37$).

Mean Platelet Volume

MPV reflects platelet size, activation status, and bone marrow response. Larger platelets are metabolically and enzymatically more active and have greater prothrombotic potential. Accelerated peripheral platelet destruction in sepsis prompts release of younger, larger platelets, raising MPV. In our cohort, MPV was significantly higher in severe sepsis (8.08 ± 0.87 vs 7.61 ± 0.85 fL; $p = 0.03$), in keeping with Becchi et al. and Vardon-Bouines et al., who reported higher MPV values in critically ill septic patients and association with adverse outcomes.^{8,9}

Platelet Distribution Width

Platelet Distribution Width (PDW) reflects the variability in platelet size and tends to increase in conditions associated with accelerated platelet turnover, where both newly released larger platelets and older senescent platelets coexist. In the present study, PDW was significantly higher among patients with severe sepsis compared to those with sepsis ($18.02 \pm 0.87\%$ vs $17.61 \pm 0.69\%$; $p = 0.018$). These findings are consistent with the observations of Yadav et al., who reported significantly elevated PDW values in patients with sepsis compared to healthy controls. Similarly, studies conducted by Zhang et al. and Gao et al. demonstrated that increased PDW was associated with greater severity of sepsis and could serve as a predictor of severe sepsis.^{15–17}

Immature Platelet Fraction

IPF measures circulating platelets that still contain residual RNA, providing a direct estimate of bone marrow thrombopoietic activity. It is particularly useful for distinguishing decreased production from increased peripheral destruction. Inflammatory mediators including interleukin-1 can promote megakaryocyte rupture, releasing larger, immature platelets and raising IPF in sepsis.¹²

In our cohort, IPF was significantly higher in severe sepsis than in sepsis (6.17 ± 5.64 vs $4.08 \pm 3.69\%$; $p = 0.04$), consistent with reports by Di Mario et al., Enz Hubert et al., De Blasi et al., and Buoro et al., who described elevated IPF in confirmed sepsis—often rising approximately 2 days before clinical onset.¹⁶⁻¹⁹

Inflammatory and standard haematological parameters

CRP was significantly higher in severe sepsis (202.08 ± 41.32 vs 153.07 ± 41.41 mg/L; $p < 0.001$), and platelet count significantly lower (185 ± 86 vs $263 \pm 68 \times 10^3/\mu\text{L}$; $p < 0.001$), reinforcing their role as severity markers. Leukocyte and neutrophil counts did not discriminate severity ($p = 0.06$ and 0.68 , respectively), consistent with the known limited specificity of total leukocyte indices in critically ill patients. Lobo et al. and Póvoa et al. have shown that absolute CRP values, and particularly their trends, correlate with severity and treatment response.^{20,21}

Strengths and limitations

Strengths include the prospective design, exclusion of confounders that affect platelet indices, and use of routinely available analyser-derived parameters that are translatable to most clinical laboratories. Limitations include the single-centre setting, a relatively small sample size particularly for outcome analysis, measurement at a single time point rather than serially,

the use of SIRS-based definitions of sepsis severity (predating Sepsis-3 in concept), and the absence of receiver-operating-characteristic analysis to define optimal cut-offs. Multivariable adjustment for comorbidities, organ dysfunction scores, and source of infection was not performed.

CONCLUSION

Platelet indices derived from routine complete blood count analysis—MPV, PDW, and IPF—were significantly higher in patients with severe sepsis than in those with sepsis, reflecting heightened platelet activation, consumption, and bone marrow turnover with increasing systemic infection.

DECLARATIONS

Ethics approval and consent to participate: Approved by the Institutional Ethics Committee. Written informed consent was obtained from all participants or their legally authorised representatives.

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REFERENCES

1. Singer M, Deutschman CS, Seymour CW, et al. The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). *JAMA*. 2016;315(8):801–810.
2. Angus DC, van der Poll T. Severe sepsis and septic shock. *N Engl J Med*. 2013;369(9):840–851.
3. ANZICS Clinical Trials Group. Mortality related to severe sepsis and septic shock among critically ill patients in Australia and New Zealand. *JAMA*. 2014;311(13):1308–1316.
4. Rudd KE, Johnson SC, Agesa KM, et al. Global, regional, and national sepsis incidence and mortality, 1990–2017: analysis for the Global Burden of Disease Study. *Lancet*. 2020;395(10219):200–211.
5. Levi M, van der Poll T. Coagulation and sepsis. *Thromb Res*. 2017;149:38–44.
6. Gawaz M, Dickfeld T, Bogner C, Fateh-Moghadam S, Neumann FJ. Platelet function in septic multiple organ dysfunction syndrome. *Intensive Care Med*. 1997;23(4):379–385.
7. 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.
8. Becchi C, Al Malyan M, Fabbri LP, Marsili M, Boddi V, Boncinelli S. Mean platelet volume trend in sepsis: is it a useful parameter? *Minerva Anesthesiol*. 2006;72(9):749–756.
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. Vagdatli E, Gounari E, Lazaridou E, Katsibourlia E, Tsikopoulou F, Labrianou I. Platelet distribution width: a simple, practical and specific marker of activation of coagulation. *Hippokratia*. 2010;14(1):28–32.
11. Akin H, Akalin H, Budak F, Ener B, Ocakoglu G, Gurcuoglu E. Prognostic value of plateletcrit in severe sepsis. *J Crit Care*. 2014;29(5):885.e1–5.
12. De Blasi RA, Cardelli P, Costante A, Sandri M, Mercieri M, Arcioni R. Immature platelet fraction in predicting sepsis in critically ill patients. *Intensive Care Med*. 2013;39(4):636–643.
13. Vincent JL, Moreno R, Takala J, et al. The SOFA score to describe organ dysfunction/failure. *Intensive Care Med*. 1996;22(7):707–710.
14. Todi S, Chatterjee S, Bhattacharyya M, et al. Epidemiology of severe sepsis in India: an update (INDICAPS). *Crit Care*. 2014;18(Suppl 1):P5.
15. Yadav DK, et al. Role of platelet indices in sepsis: a prospective observational study. *Int J Res Med Sci*. 2019;7(8):2941–2945.
16. Zhang Z, Xu X, Ni H, Deng H. Platelet indices are novel predictors of hospital mortality in intensive care unit patients. *J Crit Care*. 2014;29(5):885.e1–6.
17. Gao Y, Li Y, Yu X, et al. The impact of various platelet indices as prognostic markers of septic shock. *PLoS One*. 2014;9(8):e103761.
18. Di Mario A, et al. Immature platelet fraction in patients with neutrophilic leukocytosis. *Lab Med*. 2009;40(11):665–668.
19. Enz Hubert RM, Rodrigues MV, Andreguetto BD, et al. Association of the immature platelet fraction with sepsis diagnosis and severity. *Sci Rep*. 2015;5:8019.
20. De Blasi RA, et al. Immature platelet fraction in critically ill patients with sepsis. *Intensive Care Med*. 2013;39:636–643.
21. Buoro S, Manenti B, Seghezzi M, et al. Innovative haematological parameters for early diagnosis of sepsis in adult patients admitted to ICU. *J Clin Pathol*. 2018;71(4):330–335.
22. Lobo SM, Lobo FR, Bota DP, et al. C-reactive protein levels correlate with mortality and organ failure in critically ill patients. *Chest*. 2003;123(6):2043–2049.

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23. Póvoa P, Coelho L, Almeida E, et al. C-reactive protein as a marker of infection in critically ill patients. *Clin Microbiol Infect.* 2005;11(2):101–108.
24. Kaukonen KM, Bailey M, Pilcher D, Cooper DJ, Bellomo R. Systemic inflammatory response syndrome criteria in defining severe sepsis. *N Engl J Med.* 2015;372(17):1629–1638.