

PLATELET-TO-LYMPHOCYTE RATIO IN PAEDIATRIC INTENSIVE CARE: LOWER PLR AND ITS INVERSE ASSOCIATION WITH HAEMATOLOGICAL ORGAN DYSFUNCTION

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ABSTRACT

Background: The platelet-to-lymphocyte ratio (PLR) is an inexpensive biomarker derived from the complete blood count and has been widely evaluated as a prognostic indicator in adult infectious diseases. Prognostic direction and clinical utility of PLR in paediatric intensive care, particularly in settings with a high burden of viral illness and thrombocytopenia, remain insufficiently defined. This study evaluated the prognostic utility of admission PLR in children admitted to a tertiary care paediatric intensive care unit (PICU) with acute infectious diseases and examined its relationship with haematological organ dysfunction.

Methods: A prospective observational study was conducted over 18 months (May 2023 to November 2024) in a tertiary care PICU in North India. One hundred children aged 1 month to 18 years admitted with acute infectious diseases were enrolled after application of predefined inclusion and exclusion criteria. PLR was calculated from the admission complete blood count and analysed in relation to in-hospital mortality, organ dysfunction assessed by the PELOD-2 score, and duration of PICU and hospital stay. Non-parametric statistical tests were used where appropriate.

Results: Contrary to the expected adult-derived pattern, median PLR was significantly lower among non-survivors [28.05 (IQR: 5.35-63.88)] than among survivors [102.76 (IQR: 51.79-176.14)] ($p < 0.01$). Receiver operating characteristic analysis showed an AUC of 0.168 for PLR as a mortality predictor, below the chance line, with sensitivity of 16.67% and specificity of 58.51%. PLR demonstrated a moderate, statistically significant negative correlation with the haematological PELOD-2 sub-score ($r = -0.473$, $p < 0.001$). No significant association was observed with PICU or total hospital length of stay.

Conclusion: PLR showed an inverse prognostic direction in paediatric PICU patients which comprised of substantial burden of viral illnesses. The negative association with haematological dysfunction is likely to reflect consumptive thrombocytopenia in severe viral illness, particularly dengue. Potential clinical value of PLR may lie in identifying haematological severity in selected viral and thrombocytopenic contexts.

KEYWORDS: Platelet-to-lymphocyte ratio; paediatric intensive care; viral infections; thrombocytopenia; haematological organ dysfunction, prognostic marker,

INTRODUCTION

The platelet-to-lymphocyte ratio (PLR) is calculated from two parameters routinely available on the complete blood count and requires no additional sampling or cost. This practical advantage has led to increasing interest in PLR as a bedside prognostic marker. In adult populations, higher PLR has been associated with adverse outcomes across several infectious and inflammatory conditions. Elevated admission PLR has been linked to severity and mortality in COVID-19 [1,2], community-acquired pneumonia [3], sepsis [4,5] and sepsis complicating coronary artery disease [6]. Meta-analyses involving large adult cohorts have generally reported a consistent direction of effect, with higher PLR corresponding to poorer prognosis.

Paediatric infectious disease, however, has distinct immunological and epidemiological features. In conditions like severe dengue, platelet counts may fall critically while lymphopenia is relatively less pronounced, producing a paradoxically low PLR at the point of greatest clinical risk. Application of adult-derived PLR assumptions in such circumstances may therefore be misleading [7,8].

Despite these differences, paediatric PLR studies have often used thresholds and directional assumptions derived from adult data without first establishing whether these assumptions apply to children [9-12]. Indian evidence is limited, particularly from North Indian tertiary centres where dengue and viral respiratory infections constitute a substantial proportion of PICU admissions. This gap is clinically relevant because the mechanisms influencing PLR may vary substantially across infectious aetiologies.

The present prospective study evaluated PLR as a prognostic marker in children admitted to a tertiary PICU. The analysis focused on whether higher admission PLR predicted mortality in this cohort and how PLR related to organ dysfunction quantified using the PELOD-2 score[13].

MATERIALS AND METHODS

This prospective observational study was conducted in the PICU of a tertiary care hospital, of North India from May 2023 to November 2024. Ethics Committee approval was obtained before commencement of the study (Ref. No. SU/SMS&R/76-A/2023/77). Written informed consent was obtained from parents or legal guardians; children aged 13-18 years also provided written assent.

All consecutive children aged 1 month to 18 years admitted with a provisional diagnosis of acute infectious disease were enrolled. Post-operative and surgical admissions, chronic infections (HIV and tuberculosis), haematological malignancies, known platelet disorders including immune thrombocytopenia, immunocompromised states (transplant recipients and children receiving steroids or immunosuppressive therapy), multiple congenital anomalies, and PICU stay below two hours were excluded. Among 114 screened patients, 14 patients were excluded as per exclusion criteria and 100 were included in the final analysis.

Blood samples were collected at admission and processed on a five-part automated haematology analyser (Sysmex XN-1000). PLR was calculated as absolute platelet count divided by absolute lymphocyte count; values above 120 were classified as elevated according to Harriet Lane reference ranges [14]. The primary endpoint was in-hospital mortality. Additional analyses assessed the relationship of PLR with each of the five PELOD-2 organ system sub-scores [13] and with total PICU and hospital duration.

Statistical Analysis

Data were entered in Microsoft Excel 2017 and analysed using SPSS Version 22. Normality was assessed with the Kolmogorov-Smirnov test. As most continuous variables were not normally distributed, results are presented as medians with interquartile ranges (IQRs), and between-group comparisons were performed using the Mann-Whitney U test. Categorical variables were compared using Fisher's exact test. Spearman's rank correlation was used for correlation analyses. Receiver operating characteristic curves were constructed to assess discriminatory performance. A p value of 0.05 or less was considered statistically significant.

RESULTS

The study included 100 patients, comprising 37 boys (37%) and 63 girls (63%). Infants younger than one year formed the largest age group (38%), followed by children aged 1-5 years (30%), 6-10 years (17%), and older than 10 years (15%). Lower respiratory tract infection was the most frequent diagnosis (40%), followed by acute gastroenteritis (12%), dengue (10%), sepsis (8%), and pleural effusion (6%). Meningitis, meningoencephalitis, dengue encephalitis, multi-organ dysfunction syndrome, septic shock, and acute respiratory distress syndrome accounted for the remaining cases. Six patients (6%) died during admission. Baseline characteristics stratified by outcome are summarised in **Table 1**.

Table 1: Baseline characteristics of the study cohort stratified by outcome

Characteristic	Total (n = 100)	Survivors (n = 94)	Non-survivors (n = 6)
Age group, n (%)			
< 1 year	38 (38.0%)	37 (97.4%)	1 (2.6%)
1–5 years	30 (30.0%)	30 (100.0%)	0 (0.0%)
6–10 years	17 (17.0%)	15 (88.2%)	2 (11.8%)
> 10 years	15 (15.0%)	12 (80.0%)	3 (20.0%)
p value (age vs outcome)	0.03* (Chi-square test)		
Sex, n (%)			
Male	37 (37.0%)	31 (83.8%)	6 (16.2%)
Female	63 (63.0%)	63 (100.0%)	0 (0.0%)
p value (sex vs outcome)	0.08 (Fisher's exact test)		

Diagnosis category, n (%)			
LRTI	40 (40.0%)	40 (100.0%)	0 (0.0%)
Acute gastroenteritis	12 (12.0%)	12 (100.0%)	0 (0.0%)
Dengue / dengue encephalitis	12 (12.0%)	10 (83.3%)	2 (16.7%)
Sepsis / septic shock	9 (9.0%)	8 (88.9%)	1 (11.1%)
Pleural effusion / empyema	6 (6.0%)	6 (100.0%)	0 (0.0%)
Meningitis / meningoencephalitis	6 (6.0%)	5 (83.3%)	1 (16.7%)
MODS / ARDS	3 (3.0%)	1 (33.3%)	2 (66.7%)
Miscellaneous	12 (12.0%)	12 (100.0%)	0 (0.0%)
Severity and key biomarker at admission, median (IQR)			
PELOD-2 score	4 (2–6)	3 (2–5)	19.5 (18.5–24.75)*
p value (PELOD-2)	<0.001* (Mann-Whitney U)		
Admission PLR, median (IQR)	97.38 (45.89–176.14)	102.76 (51.79–176.14)	28.05 (5.35–63.88)*
p value (PLR)	<0.01* (Mann-Whitney U)		

*Statistically significant ($p < 0.05$). Bold/highlighted cells indicate non-survivor medians that differed significantly from survivor medians. LRTI: lower respiratory tract infection; MODS: multi-organ dysfunction syndrome; ARDS: acute respiratory distress syndrome; PELOD-2: Paediatric Logistic Organ Dysfunction-2; PLR: platelet-to-lymphocyte ratio; IQR: interquartile range.

PLR and Mortality: An Inverse Relationship

The most notable finding was the direction of association between PLR and mortality. Median PLR was lower, rather than higher, among non-survivors. Non-survivors had a median PLR of 28.05 (IQR: 5.35–63.88), compared with 102.76 (IQR: 51.79–176.14) among survivors ($p < 0.01$). When the conventional PLR >120 threshold was applied, only one of six non-survivors had an elevated PLR (16.67%), and Fisher's exact test showed no significant association in the expected direction ($p = 0.39$) (**Table 2**).

Receiver operating characteristic analysis reinforced this inverse pattern. PLR yielded an AUC of 0.168 (95% CI: 0.000–0.348, $p = 0.007$), below the threshold expected by chance. This indicates that the marker performed in the opposite direction when interpreted using the adult-derived assumption that higher PLR predicts worse outcome. At the optimal cut-off, sensitivity was 16.67%, specificity was 58.51%, positive predictive value was 2.50%, and negative predictive value was 91.67% (**Table 3**).

Table 2: PLR at admission: comparison between survivors and non-survivors

Parameter	Survivors (n = 94)	Non-survivors (n = 6)
Median PLR (IQR)	102.76 (51.79–176.14)	28.05 (5.35–63.88)
PLR elevated >120 , n (%)	39 (41.5%)	1 (16.7%)
p value	<0.01 (Mann-Whitney U); $p = 0.39$ for dichotomised PLR >120 (Fisher's exact)	

IQR: Interquartile range. PLR >120 considered elevated per Harriet Lane reference values.

Table 3: Diagnostic performance of PLR for in-hospital mortality prediction

Statistic	Value (95% CI)
AUC (ROC)	0.168 (0.000–0.348)
p value (AUC vs 0.5)	0.007
Optimal cut-off (PLR)	<120
Sensitivity	16.67% (0.42–64.12%)

Specificity	58.51% (47.88–68.59%)
Positive predictive value	2.50% (0.42–13.49%)
Negative predictive value	91.67% (88.10–94.24%)
Positive likelihood ratio	0.40 (0.07–2.44)
Overall accuracy	56.00% (45.72–65.92%)

An AUC below 0.50 indicates that higher PLR was associated with survival rather than mortality in this cohort, reflecting performance opposite to adult-derived prognostic expectations. ROC: receiver operating characteristic; AUC: area under the curve; CI: confidence interval.

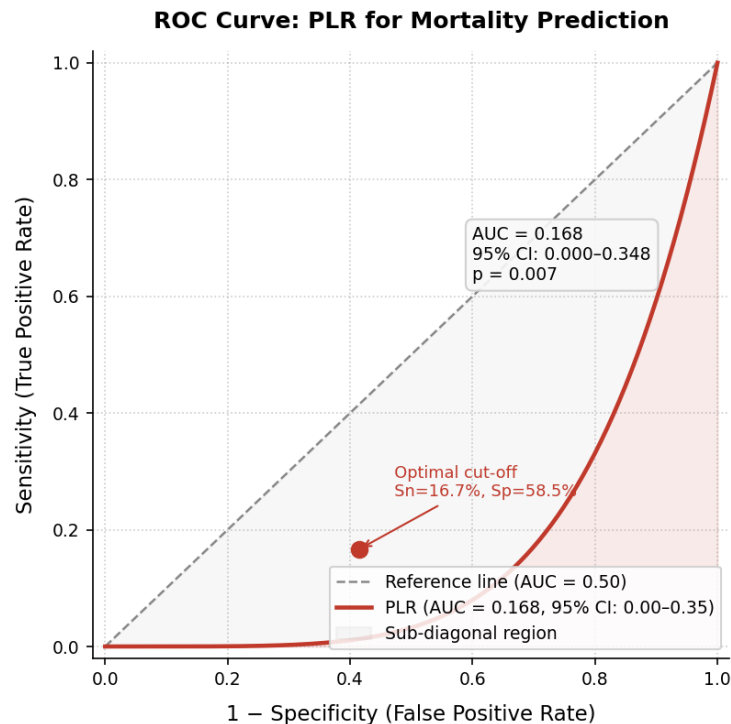


Figure 1: ROC curve for PLR as a predictor of in-hospital mortality

The ROC curve for PLR lies below the reference diagonal, indicating that higher PLR was associated with survival rather than mortality in this cohort (AUC = 0.168, 95% CI: 0.000-0.348, $p = 0.007$) (Figure 1). The filled circle denotes the optimal operating point (sensitivity 16.67%, specificity 58.51%). This sub-diagonal performance supports an inverse relationship between PLR and disease severity in this predominantly viral paediatric PICU population.

Across the five PELOD-2 organ sub-scores, PLR showed no significant correlation with neurological, cardiovascular, renal, or respiratory dysfunction. The exception was the haematological sub-score, which demonstrated a moderate negative correlation with PLR ($r = -0.473$, $p < 0.001$), the strongest correlation observed in the dataset (Table 4). Children with more severe haematological organ dysfunction therefore tended to have lower PLR values.

Table 4: Spearman correlation of PLR with PELOD-2 organ system sub-scores

Organ System (PELOD-2 Subscore)	r value	p value
Neurological	-0.122	0.225
Cardiovascular	0.035	0.726
Renal	-0.06	0.50
Respiratory	-0.043	0.672
Haematological	-0.473*	<0.001*

*Statistically significant ($p < 0.05$). Highlighted row denotes significant finding.

Admission PLR did not correlate significantly with duration of PICU stay ($r = 0.087$, $p = 0.392$) or total hospital stay ($r = 0.174$, $p = 0.084$). In this cohort, length of stay appeared to be influenced by factors other than admission PLR.

DISCUSSION

The findings differed from the expected adult-derived pattern. On the basis of adult literature, elevated admission PLR would be expected to correlate with worse outcomes, including mortality and greater organ dysfunction. In the present cohort, however, children who died had lower PLR values than survivors, and the ROC AUC of 0.168 indicated performance in the opposite direction. Interpretation of this finding requires attention to the underlying case mix.

Dengue, including dengue with warning signs and dengue encephalitis, accounted for 12% of admissions. The immunological profile of dengue is characterised by thrombocytopenia driven by immune-mediated destruction through cross-reactive antibodies [7], direct viral suppression of megakaryopoiesis in the bone marrow [8] and consumption at sites of endothelial injury during the plasma leakage phase. In severe dengue, platelet counts may fall below $10,000/\mu\text{L}$ while lymphopenia remains comparatively mild. The platelet numerator of PLR therefore declines sharply while the lymphocyte denominator is relatively preserved, producing a very low PLR in children who may be critically ill. This mechanism explains why the directional assumption commonly used in adult PLR literature may not apply in this context.

Lower respiratory tract infection was the single largest diagnostic category (40%), and most cases were viral. Viral respiratory infections may produce a weaker platelet response than bacterial infections, with less endothelial activation, less consumptive coagulopathy, and relative preservation of lymphocyte counts in some patients [15]. This differs from bacterial sepsis, where platelet counts may rise during early inflammation before consumption develops, while lymphopenia may be marked. Combining bacterial and viral illnesses under a single PLR framework can therefore obscure clinically important differences in prognostic behaviour.

The negative correlation between PLR and the haematological PELOD-2 subscore ($r = -0.473$, $p < 0.001$) requires cautious interpretation. The haematological component of PELOD-2 assigns higher scores for abnormal leucocyte counts and thrombocytopenia [13]. Because platelet count is also the numerator of PLR, severe thrombocytopenia will simultaneously increase the haematological PELOD-2 score and decrease PLR. This shared mathematical component may partly inflate the correlation coefficient. The association remains clinically relevant, but its strength should not be interpreted as entirely independent biological signal.

With that limitation acknowledged, the finding has practical value [11,12,16]. A very low PLR in a child with dengue or another thrombocytopenic viral illness can serve as a marker of significant haematological organ dysfunction, even though it cannot predict survival with adequate precision. PLR may therefore retain bedside utility when interpreted as a severity signal within an appropriate clinical context rather than as a universal mortality predictor.

Adult studies have generally reported a consistent association between higher PLR and poorer outcomes. Sarkar et al. [1] pooled data from 32 COVID-19 studies and found that higher admission PLR correlated with mortality and severity. Wang et al. [4] reported similar findings in sepsis, and Lee et al. [3] described elevated PLR in severe pneumonia. These studies are robust within their populations and are most applicable to adult, predominantly bacterial or hyperinflammatory clinical contexts.

Paediatric evidence is more heterogeneous. Mathews et al. [9] reported PLR sensitivity of 77.8% for PICU mortality, although the diagnostic composition of that cohort may have included a larger proportion of bacterial illness. Rampengan et al. [11] and Kurniawan et al. [12] found positive correlations between PLR and PELOD-2 in paediatric sepsis, whereas the present study found the opposite direction of association. This discrepancy is plausibly explained by differences in disease biology: bacterial sepsis may be dominated by platelet activation, while viral thrombocytopenic illnesses may be dominated by platelet consumption or destruction. The present results therefore complement, rather than necessarily contradict, sepsis-focused paediatric studies.

Other studies also support the importance of context. Cui et al. [17] reported variability in PLR performance across pneumonia subgroups. Shen et al. [5] demonstrated that the relationship between PLR and sepsis mortality interacts with disease severity. Chen et al. [18] found that low PLR predicted acute kidney injury and mortality in critically ill neonates, a directionally similar observation. Mısırlıoğlu et al. [19] reported limited utility of PLR in paediatric acute respiratory distress syndrome. Celegen et al. [16] and Pociute et al. [15] highlighted clinically meaningful differences in platelet behaviour between viral and bacterial paediatric infections. In a large multicentre dataset of critically ill children, Ma et al. [20] emphasised that haematological ratios should be interpreted in relation to disease context. Collectively, the literature suggests that PLR is neither uniformly useful nor uniformly uninformative; its value depends heavily on patient population and infectious aetiology.

Three practical implications emerge from these findings. First, PLR should not be used as a standalone mortality predictor in mixed paediatric PICU populations where viral infections are common. An AUC of 0.168 is clinically unhelpful and may be misleading if a low PLR in a critically ill child is interpreted as reassuring.

Second, a very low PLR in a child with dengue [7,8] or another thrombocytopenic illness should prompt heightened clinical vigilance, including close platelet monitoring, bleeding risk assessment, and careful consideration of transfusion thresholds. In this setting, PLR functions less as a mortality predictor and more as an indicator of haematological severity. [11,13,16]

Third, future studies should stratify analyses by disease category, including bacterial versus viral infection, dengue versus non-dengue illness, and thrombocytopenic versus non-thrombocytopenic presentations. A single universal PLR threshold is unlikely to perform reliably across mechanistically distinct PICU populations.

LIMITATIONS

Several limitations should be considered. Although the sample size of 100 was adequate for the primary analysis, only six deaths occurred, limiting robust subgroup analyses by disease category. The single-centre design restricts generalisability, as the case mix reflects patterns specific to a North Indian tertiary centre. PLR was measured only at admission; therefore, the prognostic value of serial PLR trends could not be assessed. Potential confounders, including organism-specific virulence, time to presentation, and treatment received before admission, were not fully controlled. Larger multicentre studies stratified by infectious aetiology are required to validate and refine these observations.

CONCLUSION

In a pediatric intensive care population which has a substantial burden of viral illness, admission PLR did not predict mortality in the direction as suggested by adult literature. Non-survivors had lower PLR values than survivors, and ROC analysis confirmed sub-chance performance when interpreted through adult-derived assumptions. PLR retained clinically relevant significance through its association with haematological organ dysfunction, suggesting that very low PLR may identify significant haematological involvement in selected clinical contexts even when it cannot reliably predict survival.

DECLARATIONS

Ethical Approval: Approved by the Institutional Ethics Committee, SSMS&R, Sharda University (Ref. No. SU/SMS&R/76-A/2023/77, dated 28/04/2023).

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Conflicts of Interest: The authors declare no conflicts of interest.

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