

ORIGINAL ARTICLE

Association of platelet count during the course of admission with mortality in critically ill children.

Humnah Mandvia¹, Bushra Shabbir², Ibrahim Shakoor³

ABSTRACT... Objective: To determine the pattern of platelet count during the course of admission in critically ill children and its association with in-hospital mortality. **Study Design:** Prospective Cohort study. **Setting:** Department of Pediatric, Abbasi Shaheed Hospital, Karachi, Pakistan. **Period:** March 2025 to January 2026. **Methods:** A total of 201 children admitted to PICU for more than 24 hours were included using non-probability consecutive sampling. Demographics, clinical and diagnostic evaluations were performed along with platelets count, and their association with final outcome was determined. IBM-SPSS Statistics, version 26.0 was used for data analysis. P-value < 0.05 was taken as significant. **Results:** Among 201 critically ill children, 116 (57.7%) were male, and the median age was 18.0 (6.0–48.0) months. In-hospital mortality occurred in 39 (19.4%), and 162 (80.6%) were discharged. Median PRISM-III score was higher among children with mortality ($p < 0.001$). Low platelets increased from 57 (28.4%) on day-1 to 70 (34.8%) on day-3, with no overall time effect ($p = 0.704$), but distributions differed by outcome on each day ($p < 0.001$). Mortality was associated with inotropes, respiratory support, mechanical ventilation, blood transfusion, sepsis, catheter-related bloodstream infection, aspiration pneumonia, dysglycaemia, acute kidney injury, and longer PICU stay. **Conclusion:** Serial platelet monitoring during early PICU admission provides clinically useful prognostic information and aligns with infection, organ dysfunction, and escalation of organ support.

Key words: Children, Inotropes, Mortality, Pediatric Intensive Care Unit, Platelets.

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INTRODUCTION

Critically ill children require significant medical, surgical, or trauma care as acute, life-threatening illnesses may lead to fatal outcomes which are often unanticipated.¹ Although pediatric acute critical disease is regarded as a significant worldwide issue, its actual impact is unknown due to a number of obstacles.² Routine blood tests that measure the absolute platelet count serve as a crucial, objective indicator for diagnosing and monitoring a wide spectrum of disorders across multiple organ systems.^{3,4} Hemostasis, thrombosis, inflammation, and vascular regeneration are among the many roles that platelets play.⁵ Critically ill patients often have a declining platelet count.^{6,7} Thrombocytopenia has been attributed to a number of factors, including immunological processes, disseminated intravascular coagulation, decreased platelet synthesis, increased platelet consumption, or aberrant platelet sequestration, or a combination of these.^{8,9} The most widely accepted theory is that

platelets are activated to aid in hemostasis, which causes “consumption” to outstrip “production”.¹⁰

Platelets have gained heightened scrutiny recently owing to their essential role in both the innate and adaptive immune systems. It is commonly recognized that hematological system problems can be diagnosed using platelet indices. In recent years, these markers have been connected to both the severity of patients’ illnesses and their prognosis. A decline in platelet count is regarded as an independent risk factor for critically ill patients in an intensive care unit.¹¹ In contrast to many mortality scores that use just the worst indicators within the first 24 hours after admission or at admission, the dynamic nature of daily platelet counts that account for the course of the disease is an advantage of utilizing platelets as a predictor of intensive care unit (ICU) outcome.^{12,13}

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Although studies have shown that decreased platelet counts are associated with prolonged hospitalization, increased costs, and mortality in adult populations, not enough studies have investigated the incidence of thrombocytopenia and the relationship between thrombocytopenia and the prognoses of pediatric patients who require treatment in the ICU. Local data on platelet counts and in-hospital mortality is growing but currently insufficient for comprehensive, nationwide, or long-term prognostic modeling. The current study was planned with the objective of determining the pattern of platelet count during the course of admission in critically ill children and its association with in-hospital mortality. The findings would not only be a useful addition to the existing stats but would also help clinicians to have an early diagnosis, allowing for timely intervention for better patient outcomes in the resource-constrained settings in Pakistan.

METHODS

This prospective cohort study was carried out at the Pediatric Department of Abbasi Shaheed Hospital, Karachi, Pakistan, from March 2025 to January 2026, following approval from the institution's ethics committee (IRB/KMDC/KMU/100/2025, dated: 27/2/2025). A sample size of 201 was calculated using the OpenEpi sample size calculator, taking the anticipated frequency of thrombocytopenia (at least one episode) as 25%¹⁴ in patients admitted to PICU. The confidence level was set at 95% and the margin of error at 6%. The inclusion criteria were patients of either gender aged 1 month to 12 years who were critically ill and admitted to PICU for more than 24 hours with an illness history of less than a week. The exclusion criteria were children with any underlying congenital deformity or who had a cardiopulmonary resuscitation event before PICU admission. Those with HIV infection, malignancies, and hematological disorders were also excluded. The World Health Organization (WHO) defines pediatric critical illness as any severe problem involving the airway, breathing, circulation, acute deterioration of consciousness, or life-threatening conditions that require intensive medical care and monitoring. Sample selection was done using the non-probability consecutive sampling technique. The study purpose, associated benefits, and safety were explained to the parents/caregivers to take

their informed written consent from them.

The eligible subjects went through documentation of their demographics, like gender (male/female), age (months), height (cm), and weight (kg). A detailed physical examination for each patient was performed. Clinical data regarding the main reason for PICU admission (digestive, respiratory, cardiovascular, neurological, hematological, infection, or metabolic), final diagnosis, suspected infections (yes/no), and culture positive (yes/no) were documented. A blood sample of 3 mL was drawn from each patient using the aseptic technique and sent to the institutional laboratory for laboratory investigations, including platelet count. Laboratory investigations were repeated after every 24 hours, 48 hours, and 72 hours of their stay in the PICU to follow the platelet pattern. A platelet count $<150 \times 10^9/L$ was deemed as low (thrombocytopenia), $150-450 \times 10^9/L$ as normal, and $>450 \times 10^9/L$ as high. A standardized method was followed for all laboratory investigations based on hospital protocols and guidelines. The Pediatric Risk of Mortality (PRISM-III) score was calculated for each patient. This tool comprised 17 physiological variables containing both clinical and laboratory parameters, evaluating systolic BP, heart rate, temperature, mental status, pupillary response, acidosis, pH, pCO_2 , total CO_2 , PaO_2 , glucose, potassium, creatinine, BUN, WBC count, platelet count, prothrombin time, and partial thromboplastin time. The score ranged from 0 to 63, and a score higher than 8 was deemed a predictor of mortality. Patients were treated until their final outcome (discharge/in-hospital mortality). The use of inotropes (yes/no), respiratory support (yes/no), oxygen therapy (yes/no), and blood transfusion (yes/no) was noted. Complications, which included sepsis (yes/no), nosocomial infection (yes/no), aspiration pneumonia (yes/no), catheter-related BSI (yes/no), hyperglycemia (yes/no), and hypoglycemia (yes/no), were also documented. The data were collected and documented on a pre-designed proforma.

The collected data were analyzed using the IBM-SPSS Statistics version 26.0. Frequencies and percentages were computed for categorical variables. Numerical variables were expressed as mean with standard deviation (SD) or median with

interquartile range (IQR), as appropriate. Assumption of normality was tested using the Shapiro-Wilk test. Final outcome as discharged or mortality was compared using chi-square/Fisher's exact test for categorical data, or Independent sample t-test, or Mann-Whitney U test for numeric data. A p-value <0.05 was taken as statistically significant.

RESULTS

In a total of 201 critically ill children admitted to the PICU, 116 (57.7%) were males, and 85 (42.3%) females). The median age was 18.0 (6.0-48.0) months. At the time of admission, 128 (63.7%) were having suspected infection, while 58 (28.9%) turned out to be culture positive. In-hospital mortality occurred in 39 (19.4%) children, and 162 (80.6%) were discharged.

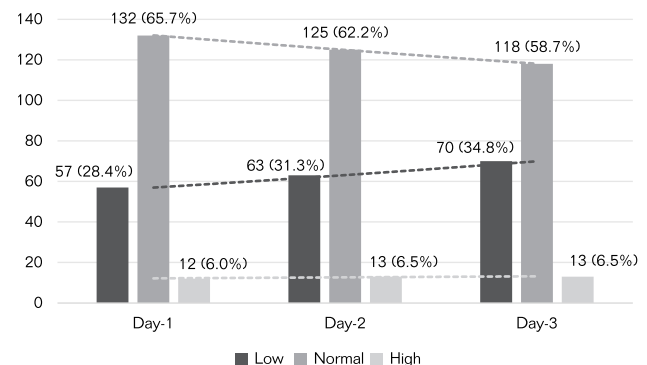
The gender ($p=0.586$) distribution, and median age ($p=0.020$) did not differ significantly with respect to final outcome. The median PRISM-III score was significantly higher among children who died ($p<0.001$). At admission, suspected infection ($p=0.008$), and culture positivity ($p=0.001$) were significantly associated with mortality. Table-I is showing details about the association of demographic and clinical characteristics of children with respect to final outcome.

Overall, low platelet count was present in 57 (28.4%) children on day-1, 63 (31.3%) on day-2, and 70 (34.8%) on day-3. Normal platelet count was recorded in 132 (65.7%) on day-1, 125 (62.2%) on day-2, and 118 (58.7%) on day-3. High platelet count was recorded in 12 (6.0%) on day-1, 13 (6.5%) on day-2, and 13 (6.5%) on day-3, with no statistically significant overall change across time points during

1st days of PICU admission ($p=0.704$) (Figure-1).

FIGURE-1

Distribution of platelet count categories over the first 72 hours of PICU admission among critically ill children (N=201)



On day-1, low platelet count was present in 39 (24.1%) discharged children, and 18 (46.2%) deaths, normal platelet count in 119 (73.5%) discharged, and 13 (33.3%) deaths, and high platelet count in 4 (2.5%) discharged, and 8 (20.5%) deaths ($p<0.001$). Relatively similar patterns were observed on day-2 ($p<0.001$), and day-3 ($p<0.001$), with low platelet count showing significant association with mortality (Table-II).

Frequency of inotropes utilization ($p<0.001$), respiratory support ($p=0.002$), mechanical ventilation ($p<0.001$), and blood transfusion ($p<0.001$) was significantly more among children with mortality. Sepsis ($p<0.001$), catheter-related bloodstream infection ($p=0.004$), aspiration pneumonia ($p=0.028$), hyperglycaemia ($p=0.008$), hypoglycaemia ($p=0.043$), and acute kidney injury ($p<0.001$) were significantly more frequent among children with mortality.

TABLE-I

Association of final outcome with demographic and clinical characteristics of critically ill children admitted to PICU (N=201)

Characteristics		Total (N=201)	Discharged (n=162)	Mortality (n=39)	P-Value
Gender	Male	116 (57.7%)	95 (58.6%)	21 (53.8%)	0.586
	Female	85 (42.3%)	67 (41.4%)	18 (46.2%)	
Age (months), median (IQR)		18.0 (6.0-48.0)	16.0 (6.0-42.0)	30.0 (90.0-60.0)	0.020
PRISM-III score, median (IQR)		9.0 (6.0-14.0)	8.0 (5.0-12.0)	16.0 (12.0-22.0)	<0.001
PRISM-III score > 8		111 (55.2%)	78 (48.1%)	33 (84.6%)	<0.001
Suspected infection		128 (63.7%)	96 (59.3%)	32 (82.1%)	0.008
Culture positive		58 (28.9%)	38 (23.5%)	20 (51.3%)	0.001

TABLE-II

Platelets evaluation categories at Day 1, Day 2, and Day 3 stratified by final outcome among critically ill children admitted to the PICU (N=201)

Time-point	Platelet category	Total (N=201)	Discharged (n=162)	Mortality (n=39)	P-Value
Day-1	Low	57 (28.4%)	39 (24.1%)	18 (46.2%)	<0.001
	Normal	132 (65.7%)	119 (73.5%)	13 (33.3%)	
	High	12 (6.0%)	4 (2.5%)	8 (20.5%)	
Day-2	Low	63 (31.3%)	39 (24.1%)	18 (46.2%)	<0.001
	Normal	125 (62.2%)	119 (73.5%)	13 (33.3%)	
	High	13 (6.5%)	4 (2.5%)	8 (20.5%)	
Day-3	Low	70 (34.8%)	39 (24.1%)	18 (46.2%)	<0.001
	Normal	118 (58.7%)	119 (73.5%)	13 (33.3%)	
	High	13 (6.5%)	4 (2.5%)	8 (20.5%)	

TABLE-III

Distribution of treatment exposures and in-hospital complications by final outcome (N=201)

Treatment and Complications	Total	Discharged (n=162)	Died (n=39)	P-Value
Inotropes	79 (39.3%)	53 (32.7%)	26 (66.7%)	<0.001
Respiratory support	121 (60.2%)	89 (54.9%)	32 (82.1%)	0.002
Mechanical ventilation	68 (33.8%)	44 (27.2%)	24 (61.5%)	<0.001
Non-invasive ventilation	41 (20.4%)	36 (22.2%)	5 (12.8%)	0.186
Oxygen therapy	156 (77.6%)	123 (75.9%)	33 (84.6%)	0.236
Blood transfusion	46 (22.9%)	28 (17.3%)	18 (46.2%)	<0.001
Sepsis	83 (41.3%)	56 (34.6%)	27 (69.2%)	<0.001
Catheter-related blood stream infection	18 (9.0%)	10 (6.2%)	8 (20.5%)	0.004
Aspiration pneumonia	22 (10.9%)	14 (8.6%)	8 (20.5%)	0.028
Hyperglycemia	44 (21.9%)	29 (17.9%)	15 (38.5%)	0.008
Hypoglycemia	27 (13.4%)	18 (11.1%)	9 (23.1%)	0.043
Acute kidney injury	31 (15.4%)	18 (11.1%)	13 (33.3%)	<0.001
PICU length of stay (days), median (IQR)	5.0 (3.0-8.0)	4.0 (3.0-6.0)	7.0 (5.0-11.0)	<0.001

The median PICU length of stay was 7.0 (5.0–11.0) days among children who died, compared with 4.0 (3.0–6.0) days among children discharged successfully ($p<0.001$). Distribution of treatment exposures and in-hospital complications by final outcome are shown in Table-III.

DISCUSSION

Platelet evaluations during early PICU admission carried prognostic signal in this study. In an analysis using a large pediatric intensive care database, early thrombocytopenia was associated with increased in hospital mortality, and short term recovery of platelet count to at least $150 \times 10^9/L$ was linked with better prognosis.¹⁵

Similar associations have been reported in more recent observational work, where thrombocytopenia at PICU admission or early in the course remained associated with higher mortality risk after adjustment for illness severity.¹⁶ A focused review of thrombocytopenia in critically ill children may also support the interpretation of thrombocytopenia as a marker of critical illness burden and organ dysfunction, particularly in septic phenotypes, which fits the case mix in many general PICUs.¹⁷

The temporal pattern observed across the first 72 hours is clinically relevant because platelet kinetics in critical illness often reflect evolving inflammatory and endothelial injury, consumption in microvascular

beds, marrow suppression, and iatrogenic contributors such as frequent sampling or transfusion practices. Contemporary sepsis literature in ICU populations describes distinct platelet patterns over time that map to outcomes, with persistently low or falling platelets associated with worse survival than stable or recovering trajectories.¹⁸ Within pediatric cohorts, a platelet course that does not recover early has been associated with adverse outcomes independent of baseline platelet value, suggesting that serial monitoring adds risk discrimination beyond a single admission measure.¹⁵ This supports the operational choice of repeated platelet assessment at 24, 48, and 72 hours, which mirrors the window when resuscitation, infection control, and early organ support decisions are expected to shift physiology.

The finding that the overall platelet distribution categories did not show a statistically significant change across the first three days, while outcome stratified comparisons remained strongly different, deserves emphasis because it indicates that group level stability can mask clinically meaningful divergence. This phenomenon has been described in critical care biomarker work where average trajectories flatten at the population level while high risk subgroups follow a different path.¹⁹ Platelet count interpretation could be most informative when anchored to outcome relevant strata such as survival or high risk severity categories, rather than being interpreted only as a unit level descriptive trend.

Illness severity measured by PRISM score showed strong separation between outcomes and a PRISM score above the selected cut off was associated with mortality. External validation work has shown that PRISM III retains discriminative ability for PICU and in hospital mortality prediction across diverse settings, though calibration can vary by case mix and resource context.²⁰ Local data have also continued to use cut off based approaches for risk stratification in PICU settings, including thresholds around 8 for higher risk groups in some local scoring comparisons.²¹ The linkage with platelet abnormalities is clinically coherent because platelet count is embedded within PRISM physiology and can also mirror the same underlying pathobiology

that drives worsening organ dysfunction. The association between higher severity scores and worse platelet categories in deaths is consistent with work linking thrombocytopenia to multi organ failure phenotypes in children.¹⁷

Suspected infection and culture positivity were associated with mortality in this cohort, a pattern that fits the broader pediatric sepsis literature where infection related admissions account for a high proportion of PICU burden and a sizeable share of deaths.²² Interpretation of culture positivity requires nuance because culture negative sepsis remains common and can carry differing trajectories depending on pathogen, antibiotic timing, and sampling quality. In a septic shock cohort, culture negative cases had more ICU free days than culture positive cases, while mortality differences were not consistent across studies, underscoring that culture positivity can behave as a marker of pathogen burden or delayed effective therapy in some settings rather than a direct determinant of outcome.²³ In resource constrained environments, culture positivity may also correlate with severity of presentation and higher likelihood of invasive devices, which can contribute to secondary infections and longer stays, linking to the catheter related bloodstream infection signal seen here.

The association of mortality with inotrope use and invasive mechanical ventilation parallels contemporary evidence that cardiovascular and respiratory failure requiring escalation to vasoactive support and intubation identifies a high risk subgroup. In pediatric critical illness, inotrope requirement often reflects shock and impaired perfusion, which coexists with endothelial injury and consumptive coagulopathy, pathways also linked to platelet consumption and thrombocytopenia.²⁴ Non invasive ventilation did not show a significant association with mortality, which may reflect patient selection, earlier stage respiratory failure, or timely transition to invasive ventilation when indicated. Published work evaluating non invasive ventilation exposure and timing indicates that outcomes depend on context, including whether non invasive support delays intubation in failing patients. A multicentre respiratory care analysis reported higher mortality among those receiving non invasive ventilation prior

to intubation compared with those not treated with non invasive ventilation before intubation, consistent with confounding by indication and potential harm from delayed escalation in selected cases.²⁵ These observations support a practice implication that non invasive ventilation should be paired with objective early failure recognition and predefined escalation thresholds, particularly in septic or multi organ dysfunction phenotypes where work of breathing is only one driver of decline.

Platelet count measured serially within the first 72 hours can support early risk stratification alongside PRISM scoring, especially when low platelet categories persist or worsen. Integration of platelet course into sepsis pathways may support earlier recognition of consumptive coagulopathy phenotypes and prompt review of infection source control, antimicrobial adequacy, device necessity, and transfusion strategy. The strong association of mortality with inotropes, mechanical ventilation, sepsis, and acute kidney injury supports prioritising timely escalation of haemodynamic and respiratory support with parallel surveillance for secondary infections and kidney injury prevention bundles.

Limitations should be considered when considering findings of this study. The single centre design limits generalizability across PICUs with different admission profiles and resource availability. Platelet evaluation was based on categorical cut offs, which supports bedside interpretability but may reduce granularity. The analysis window was restricted to the first 72 hours, which captures early physiology but may miss late thrombocytopenia linked to nosocomial infection or prolonged organ support. Extending platelet monitoring to day 5 or day 7 and linking trajectories to time varying complications such as catheter infections could clarify temporal ordering.

CONCLUSION

Serial platelet monitoring during early PICU admission provides clinically useful prognostic information and aligns with infection, organ dysfunction, and escalation of organ support. Integration of platelet trajectories with severity scoring and structured sepsis and organ support pathways can support earlier identification of high risk children and guide

targeted supportive care, while larger multicentre cohorts are needed to refine trajectory based risk models and standardize management pathways across settings.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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AUTHORSHIP AND CONTRIBUTION DECLARATION

1	Humnah Mandvia: Data collection, data analysis, drafting.
2	Bushra Shabbir: Methodology, drafting, proof reading.
3	Ibrahim Shakoor: Conception, critical revision.