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# PLATELET COUNT AS A PROGNOSTIC MARKER FOR ACUTE RESPIRATORY DISTRESS SYNDROME

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## ABSTRACT

**Background:** Acute respiratory distress syndrome (ARDS) is a severe inflammatory lung condition associated with substantial morbidity and mortality.

**Objective:** To assess whether platelet count at baseline can be used to predict in-hospital mortality at 28 days in ARDS.

**Methods:** This was a prospective observational cohort study of 93 patients aged 18–60 years, who met Berlin criteria for ARDS at Jinnah Postgraduate Medical Centre, Karachi. Platelet count was obtained at the time of intensive care unit admission as a baseline value. Patients were followed for 28 days or until they were discharged from hospital or died. Demographic, clinical, laboratory, ARDS severity, mechanical ventilation and outcome data were documented. Binary logistic regression and receiver operating characteristic (ROC) curve analyses were conducted.

**Results:** The mean age of participants was 44.8±11.3 years, and 58 (62.4%) were male. Overall, 37 (39.8%) patients died within 28 days. The median platelet count was significantly lower among non-survivors than survivors (76 [42–119] versus 154 [108–211] $\times 10^9$/L; $p<0.001$). The mortality rate was higher in patients with platelet counts $\leq 100\times 10^9$/L (58.7%) versus those with higher platelet counts (21.3%). The platelet count $\leq 100\times 10^9$/L was still an independent predictive factor for mortality after adjustment for age, sex, chronic obstructive pulmonary disease, the severity of ARDS and mechanical ventilation (adjusted OR 3.84, 95% CI 1.35-10.93, p=0.012). The area under the curve was 0.79.

**Conclusion:** Baseline platelet count was independently associated with 28-day mortality in ARDS and seemed to be a simple, readily available prognostic tool.

## INTRODUCTION

The acute respiratory distress syndrome (ARDS) was a serious inflammatory lung disease that included diffuse alveolar injury, pulmonary vascular permeability, hypoxia that was resistant to therapy, and decreased lung compliance. Although significant improvements have been made in mechanical ventilation and critical care, ARDS continues to be a significant cause of morbidity and mortality. It was therefore important to identify patients who are at risk of deterioration early, to provide timely treatment and better clinical decision making. Platelets were known to be involved in the processes of hemostasis, inflammation, endothelial damage and immune regulation. [1].

The platelet count changes had correlated with the severity of disease and system inflammatory responses in the critically ill patients. A recent study found that platelet count had previously shown to have prognostic value in patients with ARDS, and was correlated with the poor clinical outcome. Likewise, an abnormal platelet count had been linked to higher mortality, for all causes, among ARDS patients in a cohort study [2]. A correlation had also been found between the onset of ARDS and thrombocytopenia, and platelet depletion might have been a reflection of inflammatory and microvascular injury [3].

This relationship between platelet count and death had not been linear all the time. A study of patients with ARF in multiple centres found a non-linear association between platelet count and short-term in-hospital mortality [4]. Prognostic information had also been available using platelet-based inflammatory indices. Neutrophil-to-lymphocyte ratio, and platelet ratio have been predictive of 28-day mortality in ARDS patients [5] and the red blood cell distribution width-to-platelet ratio had been associated with mortality and disease severity [6].

Other platelet related ratios were able to distinguish severe ARDS from mild or moderate ARDS [7]. Further hematological markers have aided in the assessment of ARDS severity in patients suffering burn injuries [8]. Systemic immune-inflammation index that included platelet count, had also been proven to be of prognostic value in ARDS [9]. Furthermore, the platelet-albumin-bilirubin grade had been linked to 30-day mortality suggesting that platelet related parameters might have been a reflection of inflammatory burden and organ dysfunction [10]. Platelet-derived indices had been studied in multiple studies, but there was limited evidence to explain the independent prognostic significance of routine platelet count in ARDS. Based on this, the present study was undertaken to see if platelet count had been a readily available, inexpensive and clinically useful predictor of ARDS severity and outcome.

**METHODS**

It was a prospective observational cohort study conducted in Chest Medicine Ward 12 and intensive care unit of Jinnah Postgraduate Medical Centre, Karachi for six months period. The study was carried out following the guidelines of the Declaration of Helsinki. All participants (or legally authorised representative, if participant was critically ill) provided written informed consent.

Sample size was determined by OpenEpi software. Wang et al. showed that patients with ARDS who had a platelet count of $\leq 100 \times 10^9$/L had a 28-day mortality of 40.13% [12]. With an absolute margin of error of 10% and a 95% confidence level, a sample of 93 patients was calculated. Non-probability consecutive sampling was used in recruiting the participants.

The patients studied were those 18–60 years of age of either sex admitted during the study period who met the Berlin definition of acute respiratory distress syndrome. Those with a known haematological disorder affecting platelet count (e.g. leukaemia, myelodysplastic syndrome), who had a platelet transfusion prior to baseline platelet measurement, and who refused consent were excluded.

The diagnosis of ARDS was made using the Berlin definition [11]. To make a diagnosis, new or worsening respiratory symptoms had to occur within 1 week after a known clinical insult, there had to be bilateral pulmonary opacities on chest radiography or computed tomography that could not be completely attributed to pulmonary masses, lobar or pulmonary collapse or pleural effusion, and respiratory failure had to be the primary cause of the opacities, excluding cardiac failure or fluid overload. Cardiac failure was excluded from the list based on clinical presentation and echocardiographic findings as appropriate. Hypoxaemia was considered a $PaO_2/FiO_2$ ratio $\leq$300 mmHg with positive end-expiratory pressure (PEEP) and/or continuous positive airway pressure (CPAP) $\geq$5 cm $H_2O$. ARDS was classified as mild, moderate and severe according to the following $PaO_2/FiO_2$ ratios: >200–300 mmHg, >100–200 mmHg and $\leq$100 mmHg, respectively.

The main exposure variable was the platelet count at baseline (within 24 hours of admission to the ICU, prior to platelet transfusion). Venous blood sample was taken aseptically in a ethylene diaminetetra acetic acid tube and analysed by the automated haematology analyser at the institutional laboratory. The cut-off point for the platelet count was set at $100 \times 10^9$/L, reflecting the cut-off point in the reference study [12]. Platelet count was grouped into severe thrombocytopenia (<50 × 109/L), moderate thrombocytopenia (50-99 × 109/L), mild thrombocytopenia (100-149 × 109/L), normal platelet count (150-450 × 109/L) and thrombocytosis (>450 × 109/L) for descriptive and secondary analyses, and was also analysed as a continuous variable.

Main outcome was 28-day in-hospital mortality, which was defined as death within the hospital, for any cause, within 28 days of the date of arrival in the ICU. For the 28-day in-hospital mortality analysis, patients that left the hospital alive prior to 28 days were considered survivors. ICU length of stay was a secondary outcome, defined as the number of days from time of admission to the ICU to the day of discharge or death.

Patients were included in the study in a consecutive order. Demographic and clinical data such as age, sex, duration of the symptoms, and other associated comorbidities were obtained in a structured proforma. Chronic obstructive pulmonary disease, hypertension, diabetes mellitus, coronary heart disease, chronic kidney disease and malignancy constituted other co-morbid conditions. These conditions were established based on the available treatment records and investigations and confirmed from the medical history. Pulse rate, respiratory rate, body temperature, systolic and diastolic blood pressure and peripheral oxygen saturation were taken as baseline. Symptoms such as dyspnoea, tachypnoea, tachycardia, productive cough, fever, hypotension, chest pain and pulmonary crackles were observed. The intensity of the chest pain was assessed on a numerical rating scale (NRS) of 0-10.

Chest radiography and computed tomography of the chest (when clinically available) were performed. Arterial blood gas was collected and the $PaO_2/FiO_2$ ratio calculated to confirm ARDS and to grade it. The need for mechanical ventilation (invasive or non-invasive) was documented. All participants were treated as per institution standard clinical protocols. All patients were followed from enrollment to hospital discharge or death or the end of 28 days, whichever came first. The research work was done with direct clinical assessment, laboratory reports and hospital records.

Data was later input and analysed with the Statistical Package for the Social Sciences (SPSS) version 25.0. To evaluate the data accuracy, the completed proforma was reviewed and the electronic dataset was tested for missing, duplicate and implausible data. We evaluated the distribution of quantitative variables by the Shapiro–Wilk test, histograms and Q–Q plots. Quantitative variables were normally distributed and presented as mean and standard

deviation; otherwise, they were normally presented by the median and interquartile range. Frequencies and percentiles were used for categorical variables.

Normal and non-normal quantitative variables were compared between survivors and non-survivors using independent-samples t-test and Mann–Whitney U test respectively. One-way analysis of variance or the Kruskal–Walli's test was used to make comparisons among more than 2 platelet or ARDS severity groups, as appropriate. The chi-square test was used to compare categorical variables, with Fisher's exact test used when any cell had an expected frequency of less than five.

The relationship between the platelet count at baseline and the 28-day in-hospital mortality was first investigated using univariable binary logistic regression. Next, multivariable binary logistic regression was done to see if platelet count was an independent predictor of mortality after controlling for clinically relevant factors such as ARDS severity, mechanical ventilation, comorbidity status and sex. The number of variables were limited on the basis of the number of mortality events to prevent overfitting of the model. Adjusted and unadjusted odds ratios with 95% confidence intervals were reported. The variance inflation factors were used to determine multicollinearity and model calibration was performed with the Hosmer–Lemeshow goodness-of-fit test.

Receiver operating characteristic curve analysis was used to determine if baseline platelet count discriminated the occurrence of death at 28 days. The area under the curve was reported along with their 95% confidence interval. If available, the best platelet cut-off point was determined by Youden index calculation and sensitivity, specificity, positive predictive value and negative predictive value were determined. Stratified analyses were conducted for sex, age, ARDS severity, mechanical ventilation and major comorbidities as potential effect modifiers. All statistical tests were two-sided, and a p-value of ≤0.05 was used as statistically significant.

**RESULTS**

The study included 93 patients with ARDS. The average age of the participants was 44.8±11.3 years. Of the total participants, 58 (62.4%) were male and 35 (37.6%) were female. The most common co-morbidities were hypertension (31.2%), diabetes mellitus (26.9%), chronic obstructive pulmonary disease (chronic obstructive pulmonary disease, 25.8%), coronary heart disease (14.0%), chronic kidney disease (11.8%) and malignancy (7.5%). 37 (39.8%) patients died within 28 days of the admission to the ICU, while 56 (60.2%) survived. The mean age was significantly higher among the non-survivors than survivors (47.8±10.7 versus 42.8±11.2 years; p=0.035). The demographic characteristics and comorbidity profile according to 28-day outcome are presented in **Table 1.**

Non-survivors had significantly higher mean pulse and RR than survivors. There were also significantly lower systolic blood pressure and $PaO_2/FiO_2$ ratio values in the non-survivors. The mean $PaO_2/FiO_2$ was 119.4±52.8 mmHg in the non-survivors, and 182.6±61.4 mmHg in survivors (p<0.001). Non-survivors had significantly lower numbers of platelets at baseline than survivors: 76 (42–119) $\times 10^9$/L versus 154 (108–211) $\times 10^9$/L, respectively (p<0.001). Thirty-one (83.8%) non-survivors required mechanical ventilation, while 21 (37.5%) survivors did (p<0.001). The median length of stay in the intensive care unit (ICU) was also significantly longer for non-survivors. Mortality status and clinical and laboratory findings are presented in **Table 2**.

Severe, moderate, mild and normal platelet count was identified in 14 (15.1 %), 32 (34.4 %), 21 (22.6 %) and 26 (28.0 %) patients respectively. The number of deaths rose in a stepwise fashion as platelet counts fell. Ten (71.4%) severe thrombocytopenic, 17 (53.1%) moderate thrombocytopenic, 6 (28.6%) mild thrombocytopenic and 4 (15.4%) normal platelet counts died (p=0.001). Mechanical ventilation and severe ARDS and longer length of stay in the ICU were also significantly associated with lower platelet-count categories, as shown in **Table 3.**

Platelet count ≤100×$10^9$/L was independently associated with mortality after adjustment for age, sex, chronic obstructive pulmonary disease and ARDS severity and mechanical ventilation (adjusted OR 3.84; 95% CI 1.35–10.93; p=0.012). Severe ARDS and mechanical ventilation were also independent risk factors for death. **Table 4** shows the results of univariable and multivariable regression analysis.

**Table 1: Demographic characteristics and comorbidities according to 28-day outcome (n=93)**

| Variable | Total (n=93) | Survivors (n=56) | Non-survivors (n=37) | p-value |
|---|---|---|---|---|
| Age, years | 44.8±11.3 | 42.8±11.2 | 47.8±10.7 | 0.035 |
| Male gender | 58 (62.4) | 34 (60.7) | 24 (64.9) | 0.683 |
| Female gender | 35 (37.6) | 22 (39.3) | 13 (35.1) | |
| | | | | |
| Chronic obstructive pulmonary disease | 24 (25.8) | 10 (17.9) | 14 (37.8) | 0.031 |
| Hypertension | 29 (31.2) | 14 (25.0) | 15 (40.5) | 0.114 |
| Diabetes mellitus | 25 (26.9) | 12 (21.4) | 13 (35.1) | 0.144 |
| Coronary heart disease | 13 (14.0) | 5 (8.9) | 8 (21.6) | 0.084 |
| Chronic kidney disease | 11 (11.8) | 4 (7.1) | 7 (18.9) | 0.086 |

| | | | | |
|---|---|---|---|---|
| Malignancy | 7 (7.5) | 2 (3.6) | 5 (13.5) | 0.076 |

**Table 2: Clinical and laboratory findings according to 28-day outcome (n=93)**

| Variable | Survivors (n=56) | Non-survivors (n=37) | p-value |
|---|---|---|---|
| Pulse rate, beats/minute | 102.3±15.7 | 111.2±17.6 | 0.014 |
| Respiratory rate, breaths/minute | 27.1±5.2 | 31.3±6.1 | 0.001 |
| Temperature, °F | 100.1±1.2 | 100.4±1.3 | 0.262 |
| Systolic blood pressure, mmHg | 118.2±17.4 | 104.3±19.7 | 0.001 |
| $PaO_2/FiO_2$ ratio, mmHg | 182.6±61.4 | 119.4±52.8 | <0.001 |
| Platelet count, $\times 10^9$/L | 154 (108–211) | 76 (42–119) | <0.001 |
| Mechanical ventilation required | 21 (37.5) | 31 (83.8) | <0.001 |
| ICU stay, days | 8 (5–12) | 12 (8–17) | 0.002 |

$PaO_2/FiO_2$: ratio of arterial oxygen partial pressure to fractional inspired oxygen concentration; ICU: intensive care unit.

**Table 3: Outcomes according to baseline platelet-count categories (n=93)**

| Platelet-count category | Patients, n (%) | Mortality, n (%) | Mechanical ventilation, n (%) | Severe ARDS, n (%) | ICU stay, days |
|---|---|---|---|---|---|
| Severe thrombocytopenia, <50×10⁹/L | 14 (15.1) | 10 (71.4) | 12 (85.7) | 9 (64.3) | 13 (9–18) |
| Moderate thrombocytopenia, 50–99×10⁹/L | 32 (34.4) | 17 (53.1) | 20 (62.5) | 14 (43.8) | 11 (7–15) |
| Mild thrombocytopenia, 100–149×10⁹/L | 21 (22.6) | 6 (28.6) | 11 (52.4) | 6 (28.6) | 8 (6–12) |
| Normal platelet count, 150–450×10⁹/L | 26 (28.0) | 4 (15.4) | 9 (34.6) | 5 (19.2) | 7 (5–10) |
| p-value | — | 0.001 | 0.015 | 0.026 | <0.001 |

ARDS: acute respiratory distress syndrome; ICU: intensive care unit. ICU stay is presented as median (interquartile range).

**Table 4: Univariable and multivariable logistic regression analysis of predictors of 28-day mortality (n=93)**

| Variable | Crude OR (95% CI) | p-value | Adjusted OR (95% CI) | p-value |
|---|---|---|---|---|
| Age, per one-year increase | 1.04 (1.00–1.08) | 0.041 | 1.03 (0.99–1.08) | 0.117 |
| Male gender | 1.19 (0.50–2.82) | 0.688 | 1.11 (0.39–3.16) | 0.845 |
| Chronic obstructive pulmonary disease | 2.80 (1.08–7.28) | 0.034 | 1.62 (0.60–4.39) | 0.343 |
| Platelet count ≤100×10⁹/L | 5.26 (2.08–13.29) | <0.001 | 3.84 (1.35–10.93) | 0.012 |
| Severe ARDS | 4.34 (1.78–10.58) | 0.001 | 3.12 (1.10–8.87) | 0.033 |
| Mechanical ventilation | 8.61 (3.10–23.90) | <0.001 | 4.76 (1.47–15.39) | 0.009 |

OR: odds ratio; CI: confidence interval; ARDS: acute respiratory distress syndrome. The final model demonstrated adequate calibration on the Hosmer–Lemeshow goodness-of-fit test (p=0.710).

## DISCUSSION

The present study assessed the platelet count as a prognostic factor in acute respiratory distress syndrome, and demonstrated that patients with lower platelet counts had a more severe disease, higher rate of mechanical ventilation, longer intensive care unit stay, and higher 28-day mortality. The death rate was higher in patients with platelet counts ≤100 x 10(9)/L than in patients with more elevated platelet counts, and the association between thrombocytopenia and death was independent of other factors. The results confirmed the notion that platelet

depletion was a reflection of the systemic inflammation, endothelial injury, coagulation activation and multiorgan dysfunction present in ARDS.

The biological plausibility of the relationship between platelet count and mortality was due to coagulation abnormalities, platelet consumption, microvascular thrombosis and endothelial dysfunction that were associated with ARDS. Defective coagulations profiles have been linked to higher in-hospital mortality in severe ARDS in previous studies [11]. Similarly, the coagulopathy parameters were predictive of poor outcome in sepsis-induced ARDS, suggesting that coagulopathy parameters were associated with the progression of disease [12]. This finding of the present study was also supported by evidence of prognostic value of longitudinal platelet trajectories, with low and decreasing platelet counts associated with higher ICU mortality in critically ill patients [13].

Platelet-related biomarkers also may have been a reflection of the interaction of inflammation and vascular injury. Blood cell-derived indices such as platelet-based parameters were helpful for discriminating improving ARDS from persistent disease [14]. Similarly, inflammatory indexes with platelet count were good predictors in ARDS associated with sepsis, implying that platelet counts may help in the early risk stratification [15]. Thrombocytopenia has also been demonstrated to be a poor prognostic indicator in respiratory failure in the context of COVID-19, where a reduction in platelet count and dysfunction of platelets have been linked to more severe disease and worse outcomes [16]. Beyond a platelet count reduction, platelet dysfunction in ARDS was seen in platelet hyperreactivity and abnormal thrombin responses [17].

The present findings are also corroborated by the results of other studies that platelet indices also offered prognostic information in severe viral respiratory illness [18]. Significant coagulation abnormalities such as thrombocytopenia were linked to disease severity and patient survival in COVID-19 patients [19]. Furthermore, the manner in which platelets move in the patient's body will predict the prognosis of patients who are in critical condition with low platelets, thus demonstrating the importance of repeated platelet assessment, not only one platelet measurement [20].

**Strengths and Limitations of the Study:** Defined Berlin criteria, prospective follow-up, standardised platelet measurement and multivariable analysis were used in the study. However, it was done in one centre only, the sample size was small, and was a non-probability sampling. Baseline platelet count was the only platelet measurement evaluated, and serial changes, platelet indices, coagulation markers, sepsis severity scores and treatment-related factors were only partially evaluated. Confounding was thus possible.

**Recommendations:** Platelet count should be taken into consideration in routine assessment of risks for ARDS, as it is readily available, inexpensive, and readily accessible. The proposed threshold should be confirmed in multicentre studies involving larger patient cohorts and to assess serial platelet trajectories in comparison to other severity scores and coagulation markers.

## CONCLUSION

Independent factors associated with ARDS severity and 28-day mortality were lower baseline platelet count. Platelet count seemed to be a feasible and helpful prognostic indicator, but further large-scale prospective studies were needed before it could be used as a single predictor.

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