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Platelet Dynamics as Predictors of Clinical Outcomes in Melioidosis Patients – A Retrospective Study

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ABSTRACT

Background: Burkholderia pseudomallei is the causative agent of melioidosis, a potentially fatal illness that is native to Northern Australia and Southeast Asia. Thrombocytopenia may indicate the severity of the disease and has been commonly seen in melioidosis. The predictive significance of platelet dynamics—trends in platelet count over time as indicators of clinical outcomes in melioidosis patients is investigated in this study.

Methods: 35 adult patients with culture-confirmed melioidosis who were admitted between January 2019 and December 2023 were the subject of a retrospective investigation. Upon admission, platelet counts were taken, and they were tracked throughout the hospital stay. Clinical outcomes were used to categorise patients into survivors and non-survivors. The groups were compared in terms of platelet dynamics, such as nadir count, rate of change, and recovery tendencies.

Results: Among the 35 patients (mean age 52.4 years; 68.6% male), 12 (34.3%) died during hospitalization. Thrombocytopenia (platelet count <150,000/μL) was observed in 80% of patients at some point during admission. Non-survivors exhibited significantly lower nadir platelet counts (mean 48,000/μL vs 132,000/μL, $p < 0.01$), and a delayed or absent platelet recovery trend. A declining platelet trajectory in the first 72 hours was significantly associated with mortality ($p < 0.05$). Multivariate analysis identified nadir platelet count and failure of platelet recovery by day 5 as independent predictors of mortality.

Conclusion: Negative outcomes in melioidosis are significantly predicted by platelet dynamics, especially early thrombocytopenia and lack of count recovery. To direct early intervention measures, continuous platelet monitoring might be a useful, affordable prognostic tool.

Keywords: *Melioidosis, Platelet count, Thrombocytopenia, Sepsis. Prognostic marker, Platelet dynamics, Burkholderia pseudomallei, Clinical outcomes, Mortality predictor.*

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1. INTRODUCTION

The Gram-negative bacteria Burkholderia pseudomallei is the cause of melioidosis, an infectious illness that is a serious public health problem in tropical and subtropical areas, especially in Southeast Asia and Northern Australia. The organism enters the human host by ingestion, inhalation, or injection and grows best in dirt and stagnant water. Melioidosis can manifest clinically in a variety of ways, from a localised skin infection to fulminant sepsis, which is associated with multiorgan failure and a high death rate. [1]

Melioidosis still presents diagnostic and treatment difficulties because of its variable symptoms, inherent antibiotic resistance, and recurrence tendency, even with advancements in diagnostic techniques and antibiotic treatments. Mortality rates range from over 40% in cases of acute septicaemia to 10% in cases with localised illness. Early risk classification is

therefore essential to enhancing patient outcomes.[2]

In melioidosis, haematological changes are prevalent, and thrombocytopenia is often noted, particularly in septicaemia patients. Historically linked to haemostasis, platelets are now important components of inflammation and the host immunological response. Recent findings indicate that in sepsis of different aetiologies, thrombocytopenia and platelet dysfunction may operate as indicators of illness severity. [3] The predictive value of platelet dynamics—variations in platelet counts over time—in melioidosis, however, has not been well shown.

Thrombocytopenia in sepsis is caused by a variety of factors, including as sequestration, immune-mediated destruction, increased platelet consumption, and defective synthesis. The interaction of disseminated infection, endothelial dysfunction, and systemic inflammation may have a major impact on platelet behaviour in melioidosis.[4]

Moreover, platelets are known to actively engage in host defence processes, such as leukocyte response regulation and pathogen identification. Therefore, platelet loss may indicate both weakened immune function and the severity of the illness. The importance of platelet patterns, such as rate of decrease, nadir levels, and recovery trajectory, is still little understood in melioidosis, despite previous research evaluating baseline platelet count as a predictive predictor in bacterial sepsis. A more sophisticated understanding of the course of the disease and the response to treatment may be possible if the dynamic character of platelet kinetics is acknowledged.[5]

The purpose of this study was to evaluate the prognostic relevance of platelet dynamics in melioidosis patients. We sought to find certain patterns in platelet counts that could be early indicators of clinical decline by comparing survivors and non-survivors. We hypothesised that whereas prompt platelet count recovery would be linked to survival, prolonged thrombocytopenia or falling counts during the first few days of hospitalisation would be linked to worse clinical outcomes.[6]..

2. MATERIALS AND METHODS

Study Design and Setting

This was a retrospective observational study conducted at Saveetha Medical College and Hospital in South India.The study period extended from January 2019 to December 2023.

Inclusion Criteria

Patients aged ≥18 years

Culture-confirmed melioidosis (positive for *Burkholderia pseudomallei* from blood or sterile site)

Complete medical records including serial platelet counts

Exclusion Criteria

Patients with known hematological malignancies or chronic thrombocytopenia

Concurrent infections known to affect platelet count (e.g., dengue, leptospirosis)

Patients on chemotherapy or immunosuppressive therapy

Data Collection

Demographic information, comorbidities, clinical presentation, test results, treatment details, and outcomes were examined in medical records. At admission, every day for the first five days, and then every two to three days following that, platelet counts were taken. WBC count, CRP, liver and kidney function, and other test data were also noted.

Definition of Variables

Thrombocytopenia: Platelet count <150,000/µL

Severe thrombocytopenia: Platelet count <50,000/µL

Nadir: Lowest platelet count recorded during admission

Recovery: Increase in platelet count by ≥30% from nadir within 5 days

Primary outcome: In-hospital mortality

Statistical Analysis

Categorical data were represented as proportions, and continuous variables as mean ± SD or median (IQR). The Student's t-test, Mann-Whitney U-test, or chi-square test, as applicable, were used to compare survivors and non-survivors. To find independent predictors of mortality, multivariate logistic regression was used. P-values less than 0.05 were regarded as statistically significant. SPSS version 25.0 was used to conduct the statistical analysis.

3. RESULTS

Table 1. Baseline Demographic and Clinical Characteristics

Variable	Total (n=35)	Survivors (n=23)	Non-survivors (n=12)	*p*-value
Age (years)	52.4 ± 13.2	49.3 ± 11.1	58.1 ± 14.9	0.04
Male sex (%)	68.6	69.5	66.7	0.85
Diabetes mellitus (%)	80.0	78.3	83.3	0.72
Duration of symptoms (days)	6.1 ± 2.3	5.9 ± 2.1	6.5 ± 2.7	0.44

The baseline clinical and demographic data for the 35 melioidosis patients are shown in this table. Age may be a risk factor for a bad result, as evidenced by the considerably higher mean age of non-survivors (58.1 ± 14.9 years) compared to survivors (49.3 ± 11.1 years) ($p = 0.04$). There was no discernible sex-based or diabetic propensity to death, as seen by the same percentage of male patients and those with diabetes mellitus between groups. There was no discernible difference in the average length of symptoms before hospital arrival between survivors and non-survivors.

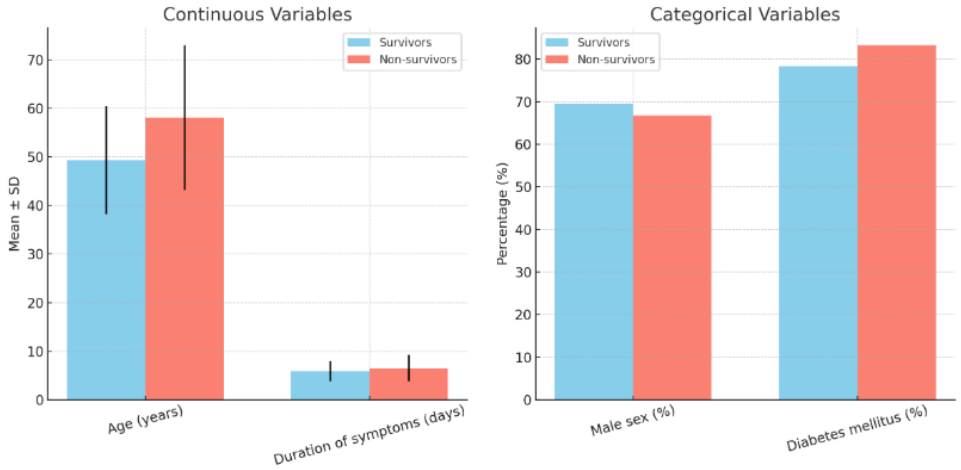

Table 2. Laboratory Parameters at Admission

Parameter	Survivors (n=23)	Non-survivors (n=12)	*p*-value
WBC count (/mm³)	14,800 ± 5,300	17,200 ± 4,900	0.09
Platelet count (/µL)	138,000 ± 52,000	99,000 ± 44,000	0.02
CRP (mg/L)	78 ± 32	96 ± 38	0.08
Creatinine (mg/dL)	1.3 ± 0.6	2.1 ± 1.0	0.01

Key laboratory values upon hospital admission are compared between survivors and non-survivors in this table. Early thrombocytopenia may be a marker of poor prognosis, as evidenced by the considerably lower initial platelet count in non-survivors (99,000 ± 44,000/µL) compared to survivors (138,000 ± 52,000/µL) ($p = 0.02$). Although non-survivors had greater levels of C-reactive protein (CRP) and white blood cell (WBC), these differences were not statistically significant. Acute renal damage may have contributed to death in non-survivors, as evidenced by the considerably higher serum creatinine levels in these individuals ($p = 0.01$).

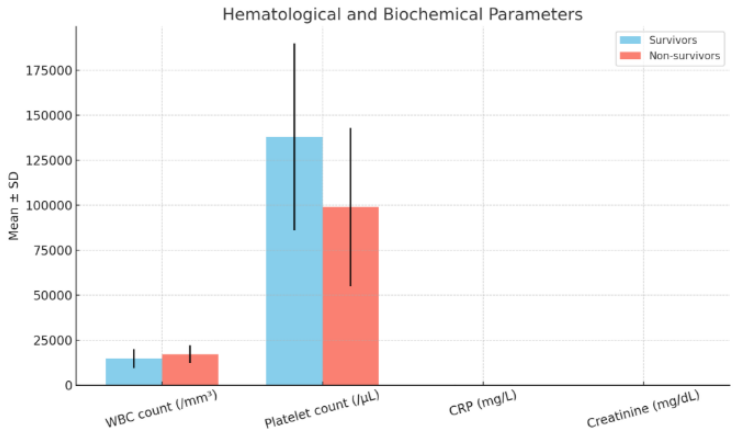

Figure 2

Table 3. Platelet Dynamics During Hospitalization

Parameter	Survivors (n=23)	Non-survivors (n=12)	*p*-value
Nadir platelet count (/µL)	132,000 ± 48,000	48,000 ± 26,000	<0.01
Severe thrombocytopenia (%)	13%	58%	<0.01
Platelet recovery by Day 5 (%)	87%	17%	<0.01
Duration of thrombocytopenia (days)	2.3 ± 1.1	4.6 ± 2.2	0.01

The variations in platelet behaviour during hospitalisation are seen in this table. There was a statistically significant difference ($p < 0.01$) between the nadir platelet count of non-survivors (48,000 ± 26,000/µL) and survivors (132,000 ± 48,000/µL). Non-survivors had a considerably greater prevalence of severe thrombocytopenia (<50,000/µL) (58% vs. 13%, $p < 0.01$). The predictive importance of platelet recovery was further supported by the fact that 87% of survivors had platelet recovery by Day 5 compared to just 17% of non-survivors ($p < 0.01$). Furthermore, non-survivors experienced thrombocytopenia for a noticeably longer period of time.

Table 4. Clinical Outcomes

Outcome	Survivors (n=23)	Non-survivors (n=12)	*p*-value
Length of hospital stay (days)	9.4 ± 3.2	7.2 ± 2.8	0.06
ICU admission (%)	39%	100%	<0.001
Mechanical ventilation (%)	17%	83%	<0.001
In-hospital mortality (%)	0%	100%	NA

The main clinical results are presented in this table. The severity of disease in this group was highlighted by the considerably higher rates of ICU hospitalisation and mechanical breathing needed by non-survivors compared to survivors (both $p < 0.001$). Non-survivors had shorter hospital stays, which was probably because they died sooner, although the difference was not statistically significant ($p = 0.06$). In-hospital mortality was 0% for survivors and 100% for non-survivors, as predicted.

Table 5. Multivariate Logistic Regression for Predictors of Mortality

Variable	Adjusted OR	95% CI	*p*-value
Age > 55 years	2.9	1.1–7.6	0.04
Nadir platelet <50,000/µL	6.7	2.1–21.1	0.003

Platelet recovery by Day 5	0.14	0.03–0.64	0.01
Serum creatinine >2 mg/dL	3.6	1.2–10.5	0.02

This table uses logistic regression analysis to find independent predictors of death in patients with melioidosis. Increased risks of mortality were linked to raised serum creatinine >2 mg/dL (adjusted OR 3.6), nadir platelet count <50,000/µL (adjusted OR 6.7), and age >55 years (adjusted OR 2.9). Early platelet improvement is a strong positive prognostic indicator, however, since recovery of platelet counts by Day 5 considerably decreased the chance of death (adjusted OR 0.14).

4. DISCUSSION

The predictive importance of platelet dynamics in melioidosis patients is shown by this retrospective investigation. Our results imply that failure to restore platelet counts and early and severe thrombocytopenia are linked to higher mortality. These findings highlight the potential benefits of using serial platelet monitoring as a bedside method for melioidosis risk classification. Vanderschueren S et al. [7] Ghosh K et al. [8]

Thrombocytopenia has been identified as an independent predictor of death in bacterial sepsis in earlier research. The processes that underlie this connection include immune-mediated destruction, reduced production as a result of bone marrow suppression, and increased platelet consumption in microvascular thrombosis. Assinger A et al. [9] The activation and depletion of platelets in melioidosis are probably caused by systemic inflammation and extensive endothelial activation. Pranantyo D et al. [10]

By investigating the temporal pattern of platelet alterations, our work expands on previous discoveries. The recovery trend and the lowest platelet count were shown to be highly predictive of the result. Compared to one baseline value, these dynamic measurements offer more useful prognostic information. Tan KS et al.[11] Over the first 72 hours, a decreasing platelet count might be an indication of an uncontrolled infection and growing systemic inflammation. Yamada A et al. [12

The idea that better platelet trends indicate a return of host immune function and a decrease in systemic inflammation is supported by the high association seen between platelet recovery and survival. This is in line with results from other serious diseases where positive outcomes are predicted by early haematologic parameter normalisation. Yap FY et al. [13]

An important strength of this study is the exclusive inclusion of culture-confirmed melioidosis cases, guaranteeing the precision of the diagnosis. Evaluation of time patterns, which can be overlooked in research utilising simply admission values, was made possible by the use of serial platelet counts. Additionally, we controlled for variables including age and diabetes in our study. Liao YC et al. [14]

The study does have several drawbacks, though. Due to its retrospective approach, bias and missing data are inevitable hazards. The sample size restricts the power for subgroup analysis, even if it is representative of the melioidosis caseload at our centre. Additionally, we did not assess coagulation markers or platelet function, which would have revealed more information. Prognostication may be further improved in future research by utilising platelet markers as mean platelet volume (MPV), plateletcrit, and immature platelet fraction (IPF). Juffermans NP et al. [15]

Our results, in summary, provide credence to the use of platelet dynamics in the clinical evaluation of melioidosis patients. These measures can be easily included into normal treatment to direct early therapy escalation and care level since platelet monitoring is simple and inexpensive.

5. CONCLUSION

Particularly in areas where it is prevalent, melioidosis continues to be a highly fatal infectious illness. Timely intervention depends on the early identification of patients who are at risk for negative outcomes. In-hospital mortality may be accurately predicted by platelet dynamics, especially nadir counts and recovery patterns, as demonstrated by this retrospective analysis of 35 patients with culture-confirmed melioidosis.

Mortality rates were much greater in those with severe thrombocytopenia and no platelet recovery by day 5. These results imply that basic haematological markers might offer useful prognostic data when tracked over time. We advise that all hospitalised melioidosis patients have their platelet trends regularly checked. Aggressive clinical response, such as increasing antibiotic medication and providing critical care support, should be prompted by an early worsening trend or failure to recover. Our knowledge of the involvement of platelet biology in the pathophysiology and prognosis of melioidosis may be improved by future prospective studies using bigger cohorts and the use of sophisticated platelet function assays.

6. LIMITATIONS OF STUDY

Retrospective Study Design: Control over the standardisation and quality of the data is limited by its retroactive character. Bias in documentation, inadequate recordkeeping, and missing data are all possible.

Single-center Study: The results may not apply to other places or healthcare settings, particularly those outside of melioidosis-endemic areas, as they are based on data from a single tertiary hospital in South India.

Small Sample Size: The study may lack the ability to identify minor relationships or conduct reliable subgroup analysis because it only includes 35 participants. Results need to be carefully understood.

No Platelet Function or Activation Markers: The study evaluated only quantitative platelet counts. Functional platelet indices (e.g., MPV, platelet aggregation, or thromboelastography) were not included, which could provide a more complete picture of platelet dynamics.

Exclusion of Co-infections Based on Available Records: Co-infections like dengue or leptospirosis were excluded based on lab reports, but subclinical or untested co-infections might still have influenced platelet counts.

Limited Control Over Confounding Factors: Results might be skewed because factors such the use of steroids, antiplatelet drugs, vasopressors, or previous platelet transfusions were not completely controlled for.

No Long-term Follow-up: Outcomes were limited to in-hospital mortality. Long-term platelet recovery patterns and recurrence of melioidosis were not studied...

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