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Article

The Spectrum of Severity: Clinical and Hematological Markers in Jakarta's COVID-19 Patients

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Abstract. Jakarta, a densely populated megacity with high hybrid immunity, presents a unique epidemiological landscape for COVID-19. Understanding the clinical and hematological markers in this context is vital for improving clinical management. This study aims to analyze the clinical and hematological profiles of COVID-19 patients in Jakarta to identify markers associated with disease severity. This cross-sectional study analyzed secondary data from 100 confirmed COVID-19 patients (26 mild, 54 moderate, 20 severe) at two Jakarta hospitals. ANOVA and Kruskal-Wallis tests were employed to compare variables across severity groups, while categorical variables were analyzed using Chi-square tests. Significant associations with increasing severity were found for higher HR and RR. Among hematological parameters, basophil levels decreased significantly with higher severity. Although not statistically significant, trends of decreasing lymphocytes and platelets, alongside increasing blood glucose and neutrophils, were observed. Diabetes was the most prevalent comorbidity in severe cases. In conclusion, HR, RR, and basophils roles as significant markers of COVID-19 severity in our population. Trends in lymphocyte, thrombocyte, blood glucose, and diabetes prevalence, align with known patterns of severe disease with insignificant statistically, due to sample size limitations.

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1. Introduction

The year 2020 marked a period of global shock as the COVID-19 pandemic emerged. During those early days, the treatment protocols were often generalized and built on rapidly evolving global data. Today, the virus has transitioned to an endemic presence, continuing to circulate with evolving variants [1-2]. While the acute emergency has subsided, understanding the disease's manifestation in specific populations remains crucial for optimizing clinical management. We understand that COVID-19 is not a monolithic disease but a complex illness with a spectrum of severity, influenced by many factors, including variants, vaccination status, and crucially, the patient's unique physiological response.

COVID-19 presents a heterogeneous clinical spectrum, from asymptomatic infection to severe pneumonia and multi-organ failure [3-4]. This variability underscores the need for reliable prognostic markers to guide clinical decisions, especially in dense urban settings like Jakarta. Hematological parameters, easily obtained from a complete blood count (CBC), have emerged as pivotal indicators of disease severity [5-6]. Markers such as lymphopenia, neutrophilia, elevated neutrophil-to-lymphocyte ratio (NLR), and thrombocytopenia are consistently associated with hyperinflammation and worse clinical outcomes [7-9]. Similarly, vital signs like tachypnea and tachycardia, along with comorbidities such as diabetes, are well-established risk factors for severe disease [10-13].

Jakarta has experienced multiple infection waves and achieved high levels of hybrid immunity through vaccination and prior infection [14]. Several studies have examined COVID-19 in Jakarta., such as Ariawan et al. study [14] estimated seroprevalence in Jakarta was 44.5%. Asysyifa et al. study [15] found a positive correlation between admission blood glucose level and severity in 340 patients. However, these studies often focused on single markers or specific hospital cohorts. What remains less explored is a simultaneous, multivariable analysis of clinical, hematological, and comorbidity profiles in a single Jakarta-based cohort, to examine how these factors collectively correlate with the spectrum of COVID-19 severity, particularly in the period following the initial pandemic waves.

Our study differentiates itself by providing an integrated analysis of demographic, physical, and hematological markers in patients from two hospitals, offering a more holistic snapshot of severity determinants in this specific population. The clinical urgency is underscored by local data: even in the post-emergency phase, COVID-19 continues to cause significant morbidity. For context, during earlier peaks, Jakarta's case fatality rate (CFR) reached approximately 3-4%, and hospitalization rates for severe cases strained healthcare resources [16-17]. Understanding current risk profiles is therefore essential for early identification of high-risk patients and effective resource allocation.

2. Experimental Section

2.1 Material

This study employed a cross-sectional design using secondary data from medical records of COVID-19 patients. Data were collected from two hospitals in Jakarta: Jakarta Islamic Hospital, Pondok Kopi, and Goenawan Partowidigdo Pulmonary Hospital. Data were collected from medical records of patients admitted between June 2021 and December 2021, corresponding to the Delta variant wave in Indonesia. This period was chosen as it represented a phase with high hospitalization rates, providing a sufficient sample for severity analysis. Ethical approval was granted by the Research Ethics Committee of the University of Muhammadiyah Prof. DR. HAMKA, Jakarta (protocol number

KEPKK/FK/002/07/2021). Demographic, clinical, and laboratory data were extracted from medical records.

2.2 Tips

The inclusion criteria were: (1) confirmed SARS-CoV-2 infection by RT-PCR, (2) age ⩾ 18 years, and (3) complete medical records for the variables under study (demographics, severity classification, vital signs, and CBC). Exclusion criteria included: (1) pregnant patients, and (2) patients with known hematological disorders or on immunosuppressive therapy that could confound blood cell counts. A total sampling method was used, including all patients from the specified period who met the inclusion criteria, resulting in a final sample of 100 patients.

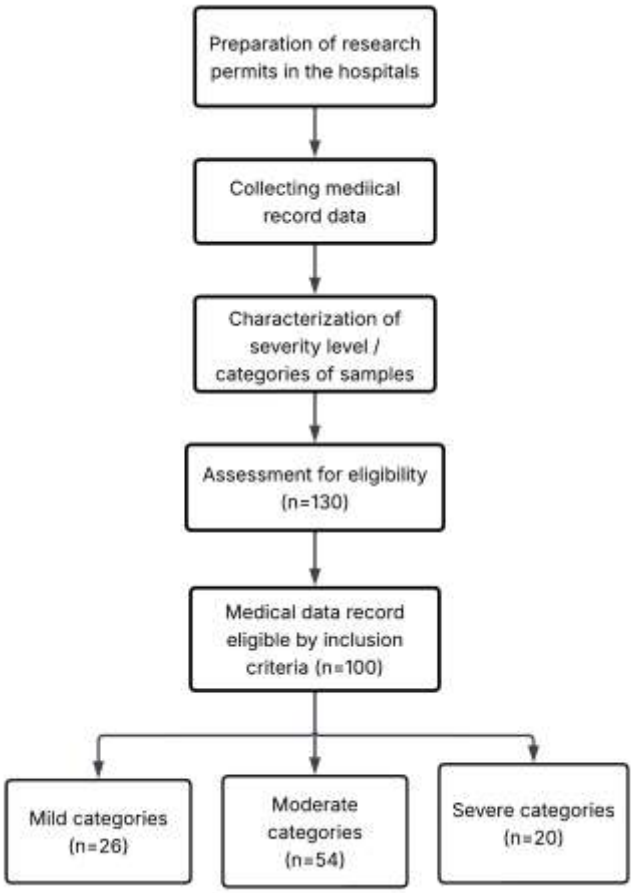

Figure 1. Flowchart of research

Disease severity was classified based on the patient's worst clinical status during hospitalization, according to WHO and US-NIH criteria [4]: Mild: Symptomatic patients (fever, cough, fatigue, etc.) without evidence of pneumonia or hypoxia (SpO2 ⩾ 94% on room air). Moderate: Clinical signs of pneumonia (fever, cough, dyspnea) but no signs of severe pneumonia, with SpO2 ⩾ 90% on room air. Severe: Clinical signs of pneumonia plus one of the following: respiratory rate > 30 breaths/min, severe respiratory distress, or SpO2 < 90% on room air. This group also included patients with critical illness (ARDS, sepsis, etc.).

The data were analysed using SPSS version 31. Descriptive statistics (means, standard deviations, frequencies and percentages) were calculated for all variables. The normality of the continuous data was assessed using the Shapiro–Wilk test. One-way analysis of variance (ANOVA) was used for normally distributed continuous data to compare across the three severity groups (mild, moderate, and severe), and the Kruskal – Wallis test was used for non-normally distributed data. Categorical variables (e.g. gender and comorbidities) were compared using the chi-squared test or Fisher's exact test, as appropriate. A p-value of <0.05 was considered statistically significant. Due to the sample size and exploratory nature of the study, multivariate analysis was not performed. The primary goal was to identify univariate associations that could inform future, larger-scale studies.

3. Results and Discussion

We analyzed data from 100 COVID-19 patients, with 26 classified as mild, 54 as moderate, and 20 as severe. As shown in Table 1, while the proportion of male patients was higher in the severe group (70%), the difference in sex distribution across groups was not statistically significant (p=0.257). There was a trend of increasing mean age with severity, but this also did not reach statistical significance (p=0.203). Similarly, mean BMI was slightly lower in the severe group, but this difference was not significant (p=0.623). The lack of statistical significance for age, a well-known risk factor, is likely attributable to the limited sample size of this study. The observed trend, however, aligns with the established understanding of immunosenescence and the accumulation of comorbidities in older adults, which increase vulnerability to severe outcomes [10], [18].

The analysis of demographic parameters—including age, sex, BMI, and comorbidities—is crucial for understanding viral behavior, identifying vulnerable populations, and developing equitable and effective public health responses. The vulnerability of older adults can be attributed to two primary, interrelated factors: immunosenescence and the accumulation of comorbidities [10]. Age serves as a proxy for the increased prevalence of conditions such as hypertension, diabetes, cardiovascular disease, and chronic kidney disease [11-13]. These conditions contribute to a pro-inflammatory baseline state and reduce physiological reserve, thereby impairing the body's ability to withstand the acute insult of severe infection. Regarding immunosenescence, older adults exhibit a diminished naïve T-cell and B-cell repertoire, which reduces their capacity to mount an effective primary immune response against novel pathogens. Concurrently, they often experience a state of chronic, low-grade inflammation—termed "inflammaging"—which can predispose them to an exaggerated inflammatory response following SARS-CoV-2 infection, potentially leading to cytokine storm and acute respiratory distress syndrome (ARDS) [18-19].

Table 1. Distribution and analysis of demographical parameters

Variable			Severity level			p-value	CI / F_{value}
			Mild	Moderate	Severe		
Gender	Male	n (%)	14	28	14	0.257	0.138 – 1.582
	Female	n (%)	12	26	6		
Age		Mean	44.6	46.9	52	0.203	1.606
BMI		Mean	26.04	25.23	24.65	0.623	0.125

Globally, data indicate that infection rates between males and females are similar; however, males have a significantly higher risk of severe disease and mortality [20]. The X chromosome carries several genes critical to immune function. Females, with two X chromosomes, may possess a more robust and nuanced immune response. Furthermore, sex hormones such as estrogen have been shown to modulate ACE2 receptor expression and possess anti-inflammatory properties, whereas

testosterone may suppress specific immune responses [21]. These factors suggest an inherent biological shield for females. This theory also aligns with our finding that the number of male patients was higher than that of female patients, although the difference was not statistically significant.

Table 2. Physical parameters analysis of COVID-19 patients

Variables		Severity level			p-*value*	F_{value}
		Mild	Moderate	Severe		
Blood Pressure (mm Hg)						
Systole	Mean	126.3	131.6	126.1	0.245	1.368
Diastole	Mean	82.5	81.2	86.2	0.468	1.284
Heart Rate (HR) / minute						
	Mean	86.4	90.5	99.7	0.006	5.035
Respiration Rate (RR) / minute						
	Mean	20.4	22.1	23.9	0.010	4.190
Body Mass Index						
	Mean	26.14	25.72	25.60	0.623	0.125
Blood Glucose (mg/dL)						
	Mean	125.2	141.9	203.8	0.077	1.439

As presented in Table 2, analysis of physical parameters showed no significant differences in systolic and diastolic blood pressure across severity groups. However, statistically significant differences were observed for both heart rate (HR) and respiration rate (RR). Mean HR increased progressively from 86.4 bpm in mild cases to 99.7 bpm in severe cases (p=0.006). Similarly, mean RR increased from 20.4 to 23.9 breaths/min (p=0.010). These findings are clinically expected, as tachypnea and tachycardia are physiological responses to hypoxemia and systemic inflammation in severe pneumonia and ARDS.

Blood glucose levels showed a marked increasing trend with severity, with the severe group exhibiting a mean level of 203.8 mg/dL compared to 125.2 mg/dL in the mild group. Although this trend is clinically striking, it did not reach statistical significance (p=0.077), likely due to high within-group variability and the modest sample size. This trend is consistent with previous research, including a larger Jakarta-based study by Asysyifa et al. [15] which found a significant positive correlation between glucose levels and severity, and a meta-analysis by Chen et al. [22] confirming hyperglycemia as a marker of severe illness [23-24].

In the mild to moderate group, blood glucose may be slightly elevated, especially in individuals without diabetes. The mild to moderate condition also could triggers a stress response, leading to increased production of counter-regulatory hormones (cortisol, glucagon, catecholamines) that promote insulin resistance and hepatic glucose production [25-26]. In addition, the mild to moderate case can cause a systemic inflammatory response, with cytokines like IL-6 contributing to insulin resistance [23-24].

The hematological parameters (Table 3) present the only statistically significant finding was a decrease in basophil percentage with increasing severity, from 0.29% in mild cases to 0.04% in severe cases (p=0.002). The clinical significance of basophil in COVID-19 is less defined but may reflect bone marrow suppression or increased margination during severe inflammation. A clear decreasing trend was observed for lymphocyte percentage, from 23.07% in mild cases to 15.67% in severe cases. This trend approached but did not achieve statistical significance (p=0.053). Lymphopenia is a hallmark of severe COVID-19, resulting from direct viral effects on lymphocytes, cytokine-mediated apoptosis, and sequestration in the lungs [27]. The near-significant finding here is highly suggestive and

consistent with this well-established pattern. Similarly, platelet counts showed a decreasing trend with severity, aligning with reports of thrombocytopenia in severe disease due to consumption and immune destruction [9]. Neutrophil-segment percentages increased with severity (61.3% to 71.63%, p=0.191), a trend that, while non-significant, mirrors the neutrophilia associated with hyperinflammation [27-28].

Table 3. Hematological parameters analysis of COVID-19 patients

Determinant	Severity level			p-*value*	F_{value}
	Mild	Moderate	Severe		
	(Mean)				
Hb (g/dL)	14.2	13.7	14.5	0.600	0.513
Hematocrit (%)	40.5	39.9	40.4	0.662	0.414
Erythrocyte (10^6/µL)	4.8	4.8	4.9	0.676	0.394
Thrombocyte (10^3/ul)	359.9	244	187	0.089	2.488
Leukocyte (10^3/ul)	8.429	7.55	7.65	0.601	0.512
Basophil (%)	0.29	0.22	0.04	0.002	6.636
Eosinophil (%)	0.85	0.79	0.57	0.630	0.645
Neutrophil-segment (%)	61.3	70.17	71.63	0.191	1.696
Lymphocyte (%)	23.07	21.71	15.67	0.053	3.309
Monocyte (%)	8.24	7.55	8.90	0.585	0.539
NLR	2.66	3.23	4.57	0.108	4.446

The neutrophil-to-lymphocyte ratio (NLR), a key composite marker of inflammation, was calculated post-hoc. The mean NLR increased progressively: 2.66 (Mild), 3.23 (Moderate), and 4.57 (Severe). While this trend is clinically meaningful, a Kruskal-Wallis test showed it was not statistically significant (p=0.108), again likely due to sample size and variability. The lack of statistical significance for many of these trends, despite their alignment with known pathophysiology, is a crucial finding. It highlights the limitations of a smaller sample size (n=100) in detecting true differences, especially when within-group variability is high. This study's results should therefore be interpreted as hypothesis-generating trends that require confirmation in larger, adequately powered studies.

Table 4. The prevalence of risk factors and comorbid disease

Risk Factor / Comorbid		Severity		
		Mild (n=21)	Moderate (n=45)	Severe (n=14)
Smoking	n (%)	4 (19.1)	13 (27.7)	3 (21.4)
Heart disease	n (%)	1 (4.5)	4 (8.5)	0 (0)
Hypertension	n (%)	4 (18.2)	14 (29.8)	4 (26.7)
Diabetes (DM2)	n (%)	5 (22.7)	12 (25.5)	6 (40)
Lung disease	n (%)	2 (9)	4 (8.5)	4 (26.7)

The assessment of risk factors and comorbidities (Table 4) revealed that smoking, heart disease, and hypertension were not significantly associated with severity in this cohort. The prevalence of diabetes mellitus was higher in the severe group (40.0%) compared to the moderate (25.5%) and mild (22.7%) groups. However, this difference was not statistically significant (p=0.428). The prevalence of

lung disease also appeared higher in the severe group (26.7%) but was not significant (p=0.128). These findings must be interpreted with caution. The lack of statistical significance for diabetes, despite the clear numerical trend, contrasts with robust global evidence identifying it as a major risk factor [29], [30]. The non-significant result here is almost certainly a consequence of the study's small sample size and the resulting low statistical power to detect differences in prevalence. The p-value of 0.428 indicates that the observed difference could easily have occurred by chance in a sample of this size. Therefore, it would be inappropriate to conclude that diabetes is not a risk factor based on this data. Instead, the finding should be reported as a non-significant trend that is consistent with the established literature but requires verification in a larger Jakarta-based study.

The primary limitation of this study is its small sample size, which severely limits statistical power. This is the most plausible explanation for why many well-established markers of severe COVID-19 (age, lymphopenia, thrombocytopenia, diabetes) did not reach statistical significance in this analysis, despite showing consistent directional trends. The lack of multivariate analysis means we cannot assess the independence of these factors or calculate odds ratios. Furthermore, the data are from 2021 and may not fully represent the clinical picture of current circulating variants. Finally, the absence of inflammatory markers (e.g., CRP, IL-6, D-dimer) and detailed immune profiling limits the depth of pathophysiological insight. Future prospective studies with larger, multi-center cohorts are essential. Such studies should incorporate serial immune profiling, multivariate regression to control for confounders, and sample size calculations to ensure adequate power to detect clinically meaningful differences.

4. Conclusion

This study provides a snapshot of clinical and hematological markers associated with COVID-19 severity in a Jakarta cohort from 2021. Significant associations with increasing severity were confirmed for elevated heart rate and respiratory rate, highlighting their continued value as bedside clinical indicators. A significant decrease in basophil levels was also observed. Consistent with global patterns, trends of lymphopenia, thrombocytopenia, neutrophilia, and hyperglycemia were observed in more severe cases, although these did not achieve statistical significance.

Similarly, a higher prevalence of diabetes was noted in the severe group, but this finding was not statistically significant. These non-significant trends are likely attributable to the study's limited sample size and resultant low statistical power, rather than an absence of true association. This study underscores that while these risk profiles can be context-specific, definitive conclusions, especially regarding the role of comorbidities like diabetes, require confirmation through larger, well-powered studies in the Jakarta population.

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