

Evaluation of Hematological Changes in COVID-19 Patients and Their Prognostic Value in Clinical Management

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Abstract

COVID-19, caused by SARS-CoV-2, induces profound and dynamic hematological alterations that serve as reliable biomarkers for disease severity, progression, and prognosis. Key changes include lymphopenia (particularly severe ALC $<0.5 \times 10^9/L$), neutrophilia, thrombocytopenia, normocytic normochromic anemia, and elevated Red Cell Distribution Width (RDW). Integrated inflammatory indices such as the Neutrophil-to-Lymphocyte Ratio (NLR), Platelet-to-Lymphocyte Ratio (PLR), and Systemic Immune-Inflammation Index (SII) have demonstrated strong predictive power for severe disease, ICU admission, mechanical ventilation, and mortality. Biochemical markers including hyperferritinemia ($>255 \mu g/L$), elevated LDH ($>319 U/L$), CRP ($>117 mg/L$), IL-6, and D-dimer further enhance risk stratification. These abnormalities reflect underlying mechanisms such as cytokine storm, direct viral effects on hematopoietic stem cells, immune dysregulation, NETosis, and consumptive coagulopathy. In clinical practice, these hematological parameters guide timely triage, anticoagulation intensity (per ASH 2025 guidelines), and immunomodulatory therapy decisions. Longitudinal monitoring also aids in identifying persistent

changes associated with Long COVID. Overall, routine hematological assessment provides accessible, cost-effective, and actionable prognostic insights that significantly improve risk stratification and clinical management of COVID-19 patients.

Author Details

Keywords: COVID-19 Hematological Changes, Lymphopenia, NLR (Neutrophil-to-Lymphocyte Ratio), Thrombocytopenia, RDW, Hyperferritinemia, D-dimer, Cytokine Storm, Prognostic Biomarkers, Long COVID

Received on 10 Mar 2026

Accepted on 05 Apr 2026

Published on 18 Apr 2026

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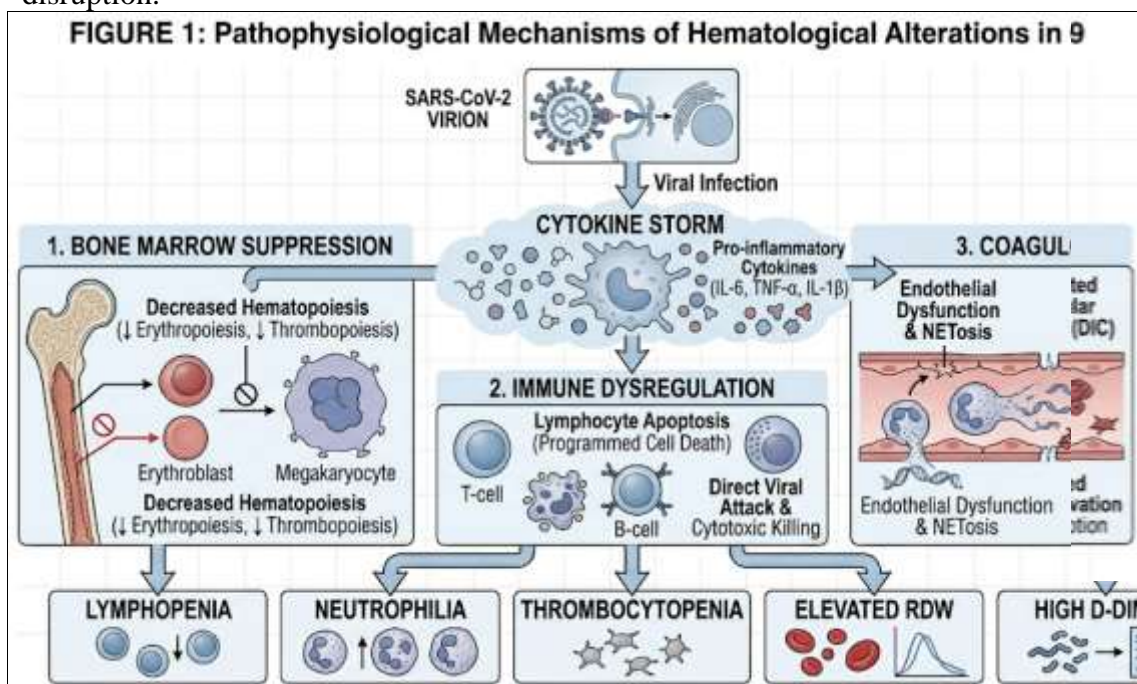
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Introduction

The emergence of the Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) in late 2019 precipitated a global health crisis, primarily characterized by acute respiratory distress but increasingly recognized as a complex, multisystemic inflammatory disorder (Bhaskar et al., 2020). While the respiratory system is the initial site of viral entry and replication, the ensuing clinical course is often dictated by the host's systemic immune response, which can lead to a state of hyperinflammation often termed a "cytokine storm" (Soy et al., 2020). Central to this pathophysiology are the profound and dynamic alterations observed within the hematological system, including significant impacts on erythropoiesis and iron metabolism (Parham et al., 2025). These changes, which manifest in routine peripheral blood parameters, serve as critical barometers of disease severity, organ dysfunction, and the likelihood of adverse clinical outcomes (Merenstein et al., 2021). As the pandemic progressed through multiple waves characterized by the emergence of variants such as Omicron and as management strategies evolved, the diagnostic and prognostic utility of hematological markers became increasingly refined (Frontiers, 2026). By 2025, clinical guidelines from organizations such as the American Society of Hematology (ASH) and the Infectious Diseases Society of America (IDSA) have integrated these markers into sophisticated triage and treatment escalation protocols, particularly for identifying patients who may benefit from immunomodulators like IL-6 or JAK inhibitors (IDSA, 2025). This report provides an exhaustive evaluation of these hematological changes, exploring their underlying mechanisms, their statistical validity as prognostic tools, and their application in contemporary clinical management (Obeagu, 2025).

Leukocyte Architecture and the Host Immune Response

The white blood cell (WBC) profile in COVID-19 represents a shift in the balance between innate and adaptive immunity. Severe infection is typically marked by a dysregulated host response, characterized by significant alterations in both the absolute counts and the relative percentages of various leukocyte subsets (Sindhushree et al., 2025). The underlying biological mechanisms driving hematological alterations in COVID-19 are summarized in Figure 1, highlighting the interconnected roles of immune dysregulation, cytokine storm, and hematopoietic disruption.



Lymphopenia and Adaptive Immune Evasion

Lymphopenia, defined as a reduction in the absolute lymphocyte count (ALC), has been consistently identified as one of the most reliable hematological markers of severe COVID-19 (Khartabil et al., 2020). Studies have reported lymphopenia in up to 83.2% of patients with critical illness. The severity of lymphopenia is particularly important; severe lymphopenia, defined as an ALC of less than $0.5 \times 10^9/L$, is strongly associated with an increased need for intensive care and mechanical ventilation (Shama et al., 2023).

The prognostic significance of lymphocyte levels is further supported by meta-analytical data indicating that a normal lymphocyte count is associated with a significantly lower risk of progression to severe disease (odds ratio, OR = 0.25) and mortality (OR = 0.21) (Erdinc et al., 2021).

Mechanistically, the depletion of lymphocytes in peripheral blood may occur through multiple pathways. These include apoptosis induced by pro-inflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF- α), as well as the potential direct infection of hematopoietic stem and progenitor cells (HSPCs) (Delshad et al., 2021).

Neutrophilia and Innate Hyper-activation

While lymphocytes are depleted, neutrophils the primary effectors of the innate immune response often undergo a significant expansion (Mahmood et al., 2025). Neutrophilia is a hallmark of severe disease, reflecting the host's attempt to counter the infection through mechanisms that often cause collateral tissue damage, such as the formation of neutrophil extracellular traps (NETs) (Beyer, 2025).

Statistical models have identified neutrophil count as a high-performance indicator for risk stratification. A cut-off value of more than $3.74 \times 10^9/L$ has demonstrated a sensitivity of 100% and a specificity of 81% for identifying patients at high risk of severe COVID-19 (Araya et al., 2021). Furthermore, neutrophilia is a robust predictor of mortality, with an odds ratio of 6.25 among patients who expired compared to survivors (Adekunle, 2022).

Table 1. Leukocyte Parameters and Statistical Risk

Leukocyte Parameter	Association	Odds Ratio (OR)	Statistical Cut-off
Elevated WBC	Severity	1.75	-
Neutrophil Count	Severity	2.62	$> 3.74 \times 10^9/L$
Neutrophil Count	Mortality	6.25	-
Normal Lymphocyte	Protective (Severity)	0.25	-
Normal Lymphocyte	Protective (Mortality)	0.21	-

Integrated Inflammatory Ratios as Prognostic Drivers

Clinical research has shifted toward integrated ratios that capture the balance between systemic inflammation and immune competence (Cicchese et al., 2018).

The Neutrophil-to-Lymphocyte Ratio (NLR)

The NLR has emerged as a superior biomarker for differentiating between moderate and severe/critical COVID-19 cases. By combining innate activation (neutrophils) with adaptive depletion (lymphocytes), the NLR provides a sensitive indicator of immune dysregulation (Manças et al., 2024).

In recent cohorts, admission NLR demonstrated a strong association with severity, with an optimal cut-off of 4.3 identified in specific populations. Other studies have

confirmed that higher NLR values correlate with increased odds of mortality (OR = 11.0 for NLR > 10) and the need for invasive mechanical ventilation (IMV) (Ullah et al., 2020).

Platelet-to-Lymphocyte Ratio (PLR) and Systemic Triage

The PLR reflects the relationship between megakaryocyte activity in the bone marrow and lymphoid turnover. While less frequently prioritized than the NLR, the PLR remains a valuable indicator of prolonged hospital stays and disease progression (Tudurachi et al., 2023). Elevated PLR levels have been significantly correlated with the severity of COVID-19 pneumonia as quantified by Computed Tomography Scan Severity Scores (CTSS) (Obeagu, 2025).

Table 2. Integrated Ratios and Severity Prediction

Ratio	Outcome Predicted	Cut-off Value	Sensitivity (%)	Specificity (%)
NLR	Severity	4.3	-	-
NLR	Mortality	8.9	75.2	74.9
NLR	Severe/Critical	3.8	95.0	74.0
PLR	Severity	106	70.0	50.0
PLR	Severity	217	-	-

Platelet Dynamics and the Systemic Immune-Inflammation Index (SII)

Platelets play a dual role in COVID-19, acting both as immune modulators and as primary drivers of coagulation. Thrombocytopenia is common in severe cases and serves as a poor prognostic sign (Beyer, 2025).

Pathophysiology of Thrombocytopenia

Thrombocytopenia in COVID-19 involves decreased production, increased destruction, and massive consumption. Autopsy studies have identified platelet-rich thrombi in various organs, indicating that consumption in microvascular beds is a primary driver (Rapkiewicz et al., 2020). The risk associated with a low platelet count is significant; a normal platelet count is protective against mortality (OR = 0.43) (Rasizadeh et al., 2024).

Systemic Immune-Inflammation Index (SII)

The SII, calculated as (Platelet count x Neutrophil count) / Lymphocyte count, integrates information from three distinct hematopoietic lineages. This composite index has been evaluated for its ability to predict mortality and viral shedding duration. For mortality, the SII has shown an optimal cut-off value often identified around 835 (Zeidan et al., 2025).

Erythroid Parameters and Red Cell Distribution Width

The erythroid lineage provides unique insights into metabolic and nutritional status. Anemia is prevalent, particularly among older adults (Wacka et al., 2024).

Prevalence and Morphology of Anemia

In specific study populations, anemia was detected in approximately 50.4% to 60.4% of patients. The morphology was predominantly normocytic normochromic, suggesting anemia of inflammation driven by cytokine-mediated disruption of iron metabolism (Sindhushree et al., 2025).

Red Cell Distribution Width (RDW) and Physiological Stress

RDW has emerged as a potent marker of physiological stress and inflammatory burden in critically ill patients. Higher RDW values at admission are strongly

associated with increased mortality; each 1% increase in RDW correlates with a hazard ratio (HR) of 1.04 for 30-day all-cause mortality (Alqhtani et al., 2025).

Table 3. Erythroid Markers and Patient Outcomes

Erythroid Marker	Significance	Outcome Association
Hemoglobin (Low)	Anemia of inflammation	ICU Admission (OR 0.14 for Normal)
RDW (High)	Physiological stress	30-day Mortality (HR 1.04)
RAR (High)	Nutri-inflammatory index	Early Diagnosis/Severity

The Lymphocyte-to-C-Reactive Protein Ratio (LCR)

The LCR integrates immune resilience (lymphocytes) with inflammatory burden (C-reactive protein) (Manaças et al., 2024).

Prognostic Utility of LCR

Low LCR values at presentation are highly predictive of clinical deterioration and in-hospital mortality. For patients with a mortal course, the LCR demonstrated a "good" ability to distinguish risk, with an optimal cut-off for mortality identified as less than or equal to 8.87 (Ullah et al., 2020).

Biochemical Markers: Ferritin, LDH, and CRP

Hematological changes are accompanied by elevations in biochemical markers that reflect tissue damage (Henry et al., 2020).

Ferritin and Hyperferritinemia

Elevated ferritin (cut-off > 255 ug/L) has been identified as a top discriminator for severity. In multivariate analysis, hyperferritinemia remains a robust independent factor for severe disease (aOR = 1.44) (Beyer, 2025).

Lactate Dehydrogenase (LDH) and C - reactive protein (CRP)

High LDH levels (cut-off > 319 U/L) and CRP (> 117 mg/L) are strong predictors of ICU admission and mortality. High CRP is associated with a seven-fold increase in the likelihood of mortality (OR = 7.09) (Avan, 2018).

Table 4. Biochemical Markers and Severity Association

Biochemical Marker	Threshold	AUROC	Association
Ferritin	> 255 ug/L	0.74	Severity (aOR 1.44)
LDH	> 319 U/L	0.76	Severity
CRP	> 117 mg/L	0.75	Mortality (OR 7.09)
IL-6	High	-	Mortality (OR 13.87)

Coagulopathy and D-dimer Dynamics

One of the most dangerous manifestations is a hypercoagulable state monitored through D-dimer levels (ASH, 2025).

D-dimer as a Prognostic Sentinel

D-dimer levels are almost universally elevated in severe COVID-19, and their predictive power for mortality is profound. A meta-analysis found D-dimer to be a top-tier predictor for mortality (OR = 6.36) (Katz, 2024).

Coagulation Profiles in Fatal Cases

Fatal cases often exhibit patterns consistent with Disseminated Intravascular Coagulation (DIC), including prolonged prothrombin time (PT). Prolonged PT has an odds ratio of 3.19 for mortality (Alqhtani et al., 2025).

Central Hematopoietic Suppression and Bone Marrow Pathophysiology

SARS-CoV-2 impacts the bone marrow and hematopoietic stem and progenitor cells (HSPCs) (Elahi, 2022).

Bone Marrow Autopsy Findings

Autopsy studies have revealed a hypercellular bone marrow where erythrophagocytosis macrophages engulfing red blood cells is observed in 54% of specimens. This directly correlates with lower hemoglobin levels. Bone marrow specimens also show left-shifted myelopoiesis in 64% of cases, reflecting systemic demand for neutrophils (Beyer, 2025).

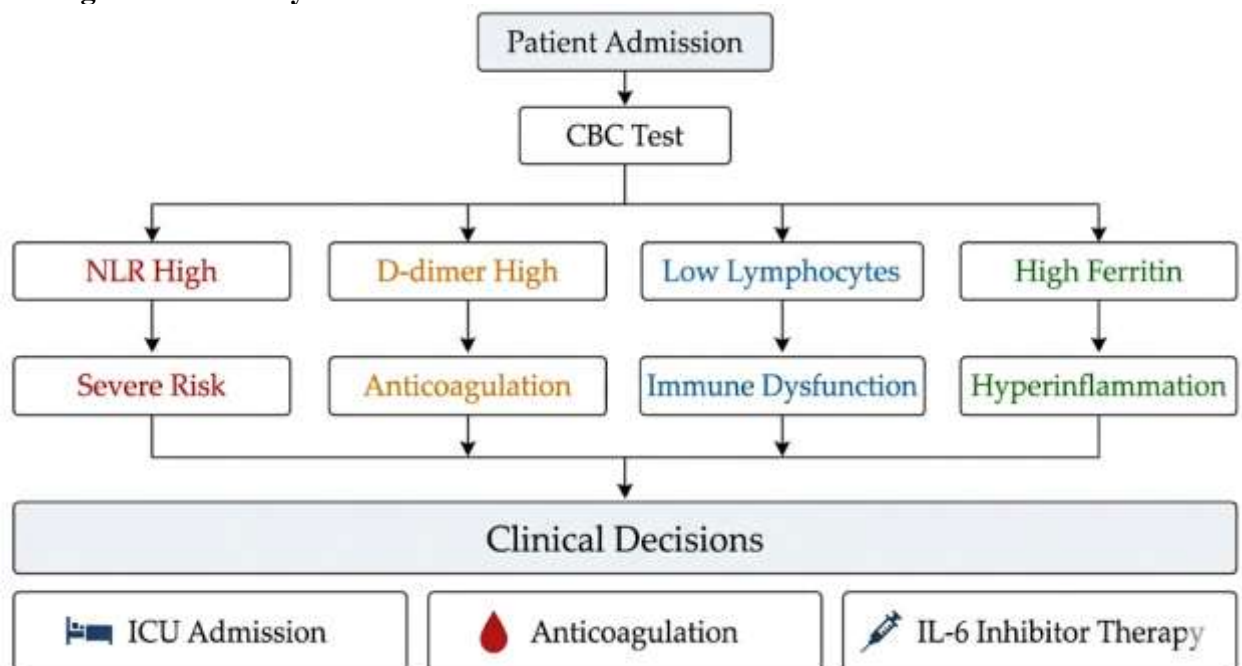
Direct Infection and Inflammaging of HSPCs

Recent research has demonstrated that SARS-CoV-2 can efficiently infect HSPCs, triggering "inflammaging" premature aging and functional decline of the hematopoietic system (Ortiz, 2022).

Clinical Management and 2025 Therapeutic Guidelines

Management is heavily informed by hematological markers, with updated protocols for anticoagulation and immunomodulation (IDSA, 2025). Hematological parameters directly guide clinical decision-making in COVID-19 management pathways, as shown in Figure 2.

Figure 2: Clinical Utility of Hematological Biomarkers in COVID-19 Management Pathway



Anticoagulation Strategies: ASH 2025 Recommendations

ASH 2025 guidelines emphasize clinical severity as the primary guide for anticoagulation intensity. For critically ill patients, ASH suggests prophylactic-intensity over therapeutic-intensity. For acutely ill (non-critical) patients, therapeutic-

intensity is suggested, particularly for those with elevated D-dimer and low bleeding risk (ASH, 2025).

Table 5. Anticoagulation Recommendations by Clinical Category

Patient Category	Recommended Intensity	Rationale
Critically Ill (ICU)	Prophylactic	Avoids bleeding risk; no mortality benefit
Acutely Ill (Ward)	Therapeutic	Beneficial for those with high D-dimer
Post-Discharge	None (Routine)	Use only for high-risk exceptions

Immunomodulatory Drugs and Laboratory Monitoring

The initiation of immunomodulators like IL-6 inhibitors (tocilizumab) or JAK inhibitors (baricitinib) requires specific laboratory assessments. Elevated liver transaminases (AST/ALT) are contraindications for IL-6 inhibitors and remdesivir. Furthermore, severe neutropenia (less than 500/uL for baricitinib) serves as a contraindication for certain therapies (Merenstein et al., 2021).

Special Patient Populations and Clinical Variations

The response to COVID-19 is not uniform across demographics (Sindhushree et al., 2025).

Pediatric Considerations

Unlike adults, pediatric patients more frequently exhibit neutropenia. Marrow suppression in children can mimic serious conditions such as leukemia, requiring a high index of suspicion (Bhaskar et al., 2020).

Long COVID and Persistent Hematological Sequelae

Long COVID is associated with sustained alterations including anemia, lymphopenia, and elevated ferritin and D-dimer. These findings suggest a lasting imprint on the hematopoietic system (Zeidan et al., 2025).

Conclusion

Hematological changes in COVID-19 patients are not merely epiphenomena but represent critical, dynamic reflections of the host's immune response, systemic inflammation, and coagulopathy, offering substantial prognostic value for clinical management. Lymphopenia, neutrophilia, elevated NLR/PLR/SII, thrombocytopenia, anemia with high RDW, and deranged biochemical markers (ferritin, LDH, CRP, D-dimer) reliably predict disease severity, progression to critical illness, need for intensive care, and mortality risk. These parameters enable early risk stratification, guide decisions on anticoagulation, immunomodulation (IL-6/JAK inhibitors), and resource allocation, while supporting monitoring of therapeutic response and Long COVID sequelae. As variants continue to emerge and management strategies evolve, integrating these accessible laboratory markers into routine protocols enhances precision medicine approaches in both acute and post-acute phases. Future research should focus on standardized cut-offs across populations, longitudinal dynamics, and integration with multi-omics and AI-driven predictive models. Ultimately, hematological profiling remains a cornerstone of effective, evidence-based clinical management for COVID-19, contributing significantly to reduced morbidity and improved patient outcomes.

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