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IMPACT OF COVID-19 ON BLOOD COAGULATION AND HEMATOLOGICAL PARAMETERS: A REVIEW
A REVIEW OF HEMATOLOGICAL AND COAGULATION ABNORMALITIES ACROSS DISEASE SEVERITY IN SARS-COV-2 INFECTION

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ABSTRACT

Beyond its primary respiratory manifestations, SARS-CoV-2 infection produces well-documented disturbances across the hematopoietic and coagulation systems, and these disturbances track closely with disease severity and outcome. This review synthesizes findings from a retrospective cohort analysis of 500 hospitalized COVID-19 patient records alongside the wider published literature on COVID-19-associated hematological and coagulation abnormalities. The reviewed cohort showed a graded relationship between severity and several laboratory markers: lymphopenia and neutrophilia were confined to moderate and severe disease, thrombocytopenia and elevated D-dimer became progressively more common with severity, and prolongation of prothrombin time (PT) and activated partial thromboplastin time (APTT), together with rising fibrinogen, accompanied more severe illness. These patterns are consistent with a distinct COVID-19-associated coagulopathy driven by systemic inflammation, endothelial injury, and microthrombosis, rather than classical sepsis-induced disseminated intravascular coagulation alone. The review discusses the proposed mechanisms linking inflammation to coagulation activation, the prognostic value of hematological and coagulation markers, and their implications for monitoring and anticoagulant management, while noting the methodological limitations inherent to retrospective, single-center designs.

KEYWORDS: COVID-19; SARS-CoV-2; Hematological parameters; Coagulopathy; D-dimer; Thrombocytopenia; Lymphopenia; Prothrombin time; APTT.

1. INTRODUCTION

COVID-19, caused by SARS-CoV-2, was initially characterized as a respiratory illness, but evidence accumulated over the pandemic established it as a systemic disease affecting the neurological, gastrointestinal, cardiovascular, and hematopoietic systems in addition to the lungs. A substantial majority of patients experience mild disease, while a meaningful minority progress to severe or critical illness marked by exaggerated cytokine release and, in many cases, disseminated intravascular coagulation, thrombocytopenia, lymphopenia, and deranged coagulation profiles. Because these hematological and coagulation changes are closely tied to severity, complications, and mortality, laboratory markers such as complete blood count indices, D-dimer, fibrinogen, and clotting times have practical value for early risk stratification and for guiding timely supportive care.

Coagulation itself is the physiological process by which blood is converted from a liquid to a stable clot through platelet activation, adhesion and aggregation, and fibrin deposition, restoring hemostasis after vascular injury. Two convergent pathways, the tissue factor (extrinsic) pathway and the contact activation (intrinsic) pathway, feed into a common cascade that culminates in cross-linked fibrin ormation. Because SARS-CoV-2 infection appears to perturb this cascade at multiple points, understanding how hematological and coagulation parameters shift across disease severity is central to recognizing and managing COVID-19-associated coagulopathy.

2. Hematological Abnormalities in COVID-19

2.1 White Blood Cell Changes

Lymphopenia is among the most consistently reported hematological findings in COVID-19 and tends to be more pronounced in severe disease. Proposed explanations include direct infection and destruction of lymphocytes by SARS-CoV-2 and suppression of lymphocyte proliferation by an exaggerated cytokine response, both of which may weaken host immune defense and accelerate disease progression. Neutrophilia, and the resulting rise in the neutrophil-to-lymphocyte ratio (NLR), is similarly common and has been proposed as a marker of systemic inflammation with prognostic value, reflecting the dual role of neutrophils in pathogen clearance and, when excessively activated, in tissue injury.

2.2 Red Blood Cell and Platelet Changes

Changes in red blood cell counts and hemoglobin are less consistent across studies, though reduced hemoglobin has been reported in some severe cases, alongside elevated ferritin suggestive of inflammation and disrupted iron handling. Thrombocytopenia, seen in a subset of patients, has been linked to more severe disease and poorer outcomes; proposed mechanisms include increased platelet destruction, bone-marrow suppression of platelet production, and increased platelet consumption during widespread microthrombus formation. Dynamic changes in platelet count over the course of illness may themselves carry prognostic information.

3. Coagulation Abnormalities and Coagulopathy

Severe COVID-19 is associated with a coagulation disorder that resembles, but is mechanistically distinct from, classical sepsis-induced disseminated intravascular coagulation. Elevated D-dimer is one of the most consistent laboratory findings and correlates strongly with severity and mortality. Prolongation of PT and APTT indicates disruption of the coagulation pathways, while fibrinogen is often elevated early in the disease course, contributing to a hypercoagulable state; in later or more severe stages, consumptive coagulopathy can instead drive fibrinogen and platelet levels down, particularly in non-survivors. Mechanistically, a systemic inflammatory response (the so-called cytokine storm) activates the coagulation cascade, while SARS-CoV-2-mediated endothelial injury promotes platelet activation and thrombin generation; the resulting microthrombosis has been observed across multiple organs and contributes to complications such as pulmonary embolism and multi-organ dysfunction.

4. Evidence from a Retrospective Cohort of 500 Patients

The cohort spanned working-age and older adults, with the largest representation in the 21–30 and 46–55 year bands and comparatively fewer records in the youngest (16–20 years) group, and a modest female majority (261 female versus 231 male) among the 500 records reviewed. This age and sex distribution is broadly consistent with hospitalized COVID-19 cohorts reported elsewhere and provides useful context for interpreting the severity-stratified laboratory findings that follow, since age in particular is a well-established determinant of COVID-19 severity and outcome.

A retrospective cohort analysis of 500 hospitalized COVID-19 patient records, drawn from admissions between March 2020 and May 2021 and confirmed by CT imaging and RT-PCR,

illustrates how these abnormalities distribute across mild, moderate, and severe disease. Lymphopenia was entirely absent among mild cases but present in roughly two-thirds of moderate cases and all severe cases, while hemoglobin values below the normal range were confined to moderate and severe disease. White blood cell counts followed a similar severity gradient, with the highest range (10.1–15 $\times 10^9$/L) observed exclusively in severe illness. Neutrophil percentage and the neutrophil-to-lymphocyte ratio rose in step with severity, while the proportion of lymphocytes fell correspondingly, mirroring the inflammatory pattern described in the broader literature.

Parameter	Mild	Moderate	Severe
Lymphopenia present	0 / 200	121 / 180	120 / 120
Elevated D-dimer (>1000 ng/mL)	48 / 200	84 / 180	77 / 120
Thrombocytopenia (<150 $\times 10^9$/L)	10 / 200	102 / 180	120 / 120
Prolonged PT (>14 s)	0 / 140	40 / 180	180 / 180
Prolonged APTT (>36 s)	0 / 140	0 / 180	200 / 200

Coagulation findings in this cohort followed the same gradient. D-dimer values rose progressively with severity, and classification of coagulation status showed thrombocytopenia predominating in mild disease, elevated D-dimer in moderate disease, and disseminated intravascular coagulation confined to severe cases. PT was prolonged only in moderate and severe disease, while APTT prolongation was essentially restricted to severe illness, indicating that intrinsic-pathway disruption became apparent later in the disease course than the extrinsic-pathway changes reflected by PT. Fibrinogen levels also rose with severity, consistent with an early hypercoagulable, inflammation-driven state rather than an immediate consumptive coagulopathy.

Classifying each record by its predominant coagulation abnormality further clarifies how the clinical picture shifts with severity. Thrombocytopenia was the dominant coagulation finding in mild disease, elevated D-dimer predominated in moderate disease, and disseminated intravascular coagulation was recorded exclusively among severe cases, with a residual normal-profile group also confined to the severe category. This progression — from an isolated platelet-count reduction, through a D-dimer-driven picture, to a full disseminated intravascular coagulation phenotype — maps onto the staged model of COVID-19 coagulopathy described in the wider literature, in which an initially localized hemostatic disturbance evolves into systemic consumptive coagulopathy as illness advances.

Coagulation Category	Mild	Moderate	Severe
Thrombocytopenia	140 / 140	0 / 180	0 / 180
Elevated D-dimer	0 / 140	180 / 180	0 / 180
DIC	0 / 140	0 / 180	100 / 180
Normal profile	0 / 140	0 / 180	80 / 180

5. DISCUSSION

The severity-graded pattern observed in this cohort — lymphopenia and neutrophilia in moderate-to-severe disease, progressive thrombocytopenia, rising D-dimer, and prolongation of PT and APTT concentrated in more severe illness — is consistent with the broader literature on COVID-19-associated coagulopathy. The convergence of these markers around a common inflammatory and endothelial-injury mechanism supports their combined use, rather than reliance on any single parameter, for early risk stratification. D-dimer elevation, falling platelet counts, and prolonged clotting times together have repeatedly been associated with thrombotic complications, including deep venous thrombosis and pulmonary embolism, particularly in intensive-care settings, and monitoring these parameters has been proposed to help guide decisions around prophylactic or therapeutic anticoagulation.

Several limitations temper these conclusions. The retrospective, single-center, convenience-sampled design used in the reviewed cohort is susceptible to selection bias and lacks the granularity of prospective, multicenter designs; category boundaries for severity and laboratory ranges were also fixed at data-collection time rather than derived from continuous statistical modeling, which limits comparability with studies using continuous variables or multivariate regression. In addition, because coagulation and inflammatory markers evolve dynamically over the course of illness, single time-point measurements — as used in most retrospective chart reviews, including this one — cannot fully capture the trajectory of coagulopathy, and serial measurement would likely offer greater prognostic precision.

A further consideration is that severity classification in most chart-review studies, including the cohort discussed here, is typically assigned using clinical and radiological criteria recorded at the time of care rather than a standardized, study-specific severity index applied uniformly in retrospect. This can introduce variability in how borderline mild, moderate, and severe cases are categorized. Despite this caveat, the consistency of the severity gradient across independently measured hematological, inflammatory, and coagulation parameters lends confidence to the overall pattern described, even where individual patient-level classification may carry some uncertainty.

6. Prognostic and Clinical Implications

The consistency with which these markers track severity supports their use as practical, low-cost prognostic tools in settings where advanced risk-scoring systems are unavailable. A rising D-dimer trend, progressive prolongation of PT or APTT, and a falling platelet count together signal a patient moving toward a more severe clinical trajectory, and have been associated in the wider literature with higher rates of venous and arterial thromboembolism, particularly among patients requiring intensive care. The neutrophil-to-lymphocyte ratio, easily derived from a routine complete blood count, has similarly been proposed as an accessible adjunct to more specialized inflammatory markers such as CRP and IL-6 for early triage.

From a management standpoint, these findings reinforce the rationale for routine monitoring of coagulation parameters in hospitalized COVID-19 patients to guide decisions on prophylactic or therapeutic anticoagulation, most commonly with low-molecular-weight heparin. Early recognition of a hypercoagulable pattern, characterized by elevated fibrinogen and D-dimer with relatively preserved platelet counts, may allow intervention before the later, more consumptive phase associated with falling fibrinogen and platelets seen in some severe and non-surviving patients. This staged understanding of COVID-19 coagulopathy — moving from an early hypercoagulable state toward, in the most severe cases, a consumptive pattern resembling disseminated intravascular coagulation — offers a useful framework for interpreting serial laboratory results at the bedside.

7. CONCLUSION AND FUTURE DIRECTIONS

Taken together, the evidence reviewed here confirms that COVID-19 meaningfully disturbs both hematological indices and coagulation parameters, with the degree of disturbance tracking disease severity. Lymphopenia, neutrophilia, thrombocytopenia, elevated D-dimer, prolonged PT/APTT, and altered fibrinogen levels collectively characterize a distinct COVID-19-associated coagulopathy rooted in systemic inflammation, endothelial injury, and microthrombosis. Continuous monitoring of these parameters may support earlier identification of patients at risk of severe disease or thrombotic complications and can help inform anticoagulation strategy.

Future research would benefit from prospective, multicenter cohorts with serial rather than single time-point measurement of hematological and coagulation markers, formal multivariate analysis of their independent prognostic value, and closer integration with inflammatory biomarkers such as CRP and IL-6 to build composite risk scores. Comparative

work across viral variants and vaccinated versus unvaccinated populations, as well as evaluation of standardized anticoagulation protocols guided by these markers, represent additional priorities for advancing clinical management of COVID-19-associated coagulopathy. As the pandemic transitions to an endemic phase, retaining these hematological and coagulation markers within routine admission workups for respiratory viral illness more broadly may also help clarify whether the coagulopathy pattern described here is specific to SARS-CoV-2 or reflects a more general severe-viral-pneumonia response.

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