AJBMS AFGHANISTAN
ISSN: 3005-6632

Afghanistan Journal of Basic Medical Sciences

2026 Jan; 3(1): 1-9.

ajbms.knu.edu.af

Hematological and Coagulation Parameters as Associated Factors in Iranian COVID-19 Patients

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ARTICLE INFO

Type: Original Article

Received: 25 April, 2025

Accepted: 10 Oct, 2025

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To cite this article:

Saharkhiz S, Ghorbani F, Rafi M, Mazloum Shahri SB, Momeni-Moghaddam MA, Moradabadi A, Zamani M. Hematological and Coagulation Parameters as Associated Factors in Iranian COVID-19 Patients. Afghanistan Journal of Basic Medical Sciences. 2026 Jan; 3(1): 1-9.

DOI

https://doi.org/10.62134/khatamuni.84

ABSTRACT

Background: The novel coronavirus SARS-CoV-2, identified as the causative agent of COVID-19, first emerged in Dec 2019 and subsequently triggered a rapid global pandemic. Despite extensive research, no definitive treatment has been established, and the disease is anticipated to re-emerge in the future, underscoring the need for continued investigation into its clinical and laboratory manifestations. We aimed to evaluate changes in hematological and coagulation parameters before and after COVID-19 infection.

Methods: We conducted a retrospective review of medical records from 94 patients admitted to Allameh Bohlool Gonabadi Hospital in Gonabad, Iran between Apr 21, 2022 and Sep 22, 2022. Complete blood count (CBC) and coagulation tests, including D-dimer, partial thromboplastin time (PTT), and prothrombin time (PT) levels, were analyzed before and after COVID-19 infection.

Results: Significant hematological and coagulation abnormalities were observed in severe COVID-19 cases. These included lymphopenia (P<0.0001), thrombocytopenia (P<0.0001), prolonged prothrombin time (PT) (P<0.0001), prolonged partial thromboplastin time (PTT) (P=0.0003), and elevated D-dimer levels (P<0.0001). The results of this study indicate demonstrate significant COVID-19-associated alterations in hematological parameters and coagulation cascades.

Conclusion: The observed dysregulation of hemostatic pathways and blood cell indices underscores the need for careful hematological monitoring in clinical management, particularly for surgical candidates. Monitoring these parameters can aid in early diagnosis, risk stratification, and treatment optimization.

Keywords: COVID-19, SARS-CoV-2, Coagulation, Thrombocytopenia, Neutrophilia, Lymphopenia

Introduction

In Dec 2019, a novel betacoronavirus, subsequently designated SARS-CoV-2, was first identified in Wuhan, Hubei Province, China. The rapid global dissemination of this pathogen prompted the WHO to declare a Public Health Emergency of International Concern (PHEIC) on January 30, 2020, marking the beginning of the COVID-19 pandemic (1). Despite ongoing research into clinical symptoms, laboratory findings, diagnosis, prevention, and treatment, no specific therapy has been established (2).

COVID-19 manifests with a broad spectrum of symptoms, ranging from mild, flu-like illness to severe, life-threatening complications. The clinical course typically progresses through four distinct stages. It begins with an upper respiratory tract infection, characterized by muscle fatigue, pain, and fever. This initial phase is followed by the development of pneumonia and dyspnea. The third stage involves a cytokine storm, leading to a dangerous hyper-inflammatory state, disseminated intravascular coagulation (DIC), vasculopathy, thrombosis, and acute respiratory distress syndrome (ARDS). In the final stage, patients will either begin to recover or ultimately succumb to the disease (2).

The emergence of SARS-CoV-2 as a novel pathogen with incompletely characterized pathophysiological properties necessitates the urgent identification of reliable prognostic biomarkers. Such predictive indicators are critical for risk stratification, therapeutic decision-making, and optimal resource allocation in clinical management. Laboratory diagnostics include real-time PCR for viral genome detection and antibody testing. Additionally, several laboratory findings have been associated with COVID-19, including prolonged partial thromboplastin time (PTT) and prothrombin time (PT), elevated inflammatory markers such as C-reactive protein (CRP), and increased lactate dehydrogenase (LDH) levels (3). Elevated erythrocyte sedimentation rate (ESR), decreased albumin, and high D-dimer levels (a fragment of fibrin degradation protein that is broken down by fibrinolysis after blood clotting) are also frequently observed.

Interleukin-6 (IL-6) serves as a pivotal proinflammatory cytokine that upregulates hepatic synthesis of acute-phase proteins, including C-reactive protein and fibrinogen. This cascade promotes a prothrombotic state manifesting as thrombotic and embolic complications, while simultaneously serving as a potential prognostic indicator for disease severity.

Hematologic clinical tests play an important role in providing useful prognostic markers to the medical team. COVID-19 has significant effects on the hematopoietic system and is often associated with thrombocytopenia, lymphopenia, and increased neutrophils (4). A rapid and significant depletion of both $CD8^+$ and $CD4^+$ T lymphocyte populations is observed during SARS-CoV-2 infection, with the degree of depletion correlating with disease severity and poor clinical outcomes (5). Moreover, a decrease in hemoglobin is observed in these patients. This virus also affects red blood cells. Tumor necrosis factor (TNF-α) and IL-1 may account for the large variation in red blood cells (RBCs) size (6). Lymphocytes, especially lymphocyteT subsets, serve as central regulators of immune homeostasis, modulating both innate and adaptive inflammatory responses through cytokine signaling and direct cellular interactions. They play an inflammatory response in the whole body. It is believed that realizing the mechanism of reducing blood lymphocytes is an effective strategy for providing treatment for COVID-19 patients (7). At the early stages of the disease, days 1-14, the number of blood leukocytes and lymphocytes is normal or slightly reduced (8). After 7-14 d since the primary symptoms begin, the increase of inflammatory mediators and "cytokine storm" occurs (9).

Currently, significant lymphopenia (including both B and T cell lines) occurs so inflammatory factors increase in peripheral blood. Unlike lymphocytes neutrophils show an increase during the disease. Neutrophils are the most plentiful immune cells in human blood. They are the first responders to many infections (10). The neutrophil-to-lymphocyte ratio (NLR) is calculated by dividing the neutrophil count by the absolute lymphocyte count, and it serves as an important indicator of overall inflammation and the severity of SARS-CoV-2 infection.

Another factor that affects the severity of the disease is the count of platelets which is an uncomplicated and accessible biomarker and independently related to SARS-COV-2 severity and mortality risk in intensive care (ICU) (11).

Coagulation disorders are relatively common among patients with COVID-19 (12), especially those with a severe form of the disease. COVID-19 can activate the coagulation cascade through different mechanisms and lead to severe hypercoagulability. A significant augment in the level of coagulation factors has been observed in COVID-19 (13). Elevated levels of von Willebrand factor (vWF) and coagulation factor VIII are relatively common in COVID-19, directly related to the occurrence of thrombosis. A marked elevation in coagulation factor V levels has also been noted in COVID-19 patients, which further promotes the occurrence of thrombotic events (14). Endothelial cell damage and the release of procoagulant factors can stimulate the coagulation cascade and activate platelets, promoting thrombus formation. Additionally, thrombosis in COVID-19 may arise from vessel wall injury, which triggers tissue factor (TF) release. This triggers the extrinsic coagulation pathway and plays a crucial role in the formation of thrombosis (15). Given that elevated factor VIII levels are frequently linked to thrombosis in COVID-19, patients with severe factor VIII deficiency (hemophilia A) have a lower thrombosis risk (16). Early anticoagulant treatment can stop clot formation and reduce microthrombosis, thus reducing the risk of major damage to organs (17).

A notable aspect of the COVID-19 pandemic has been the variability in clinical symptoms and outcomes, particularly among different racial groups (18). Therefore, investigating the hematological profile of patients would obtain valuable insight for prognosis and treatment. As hematological parameters show heterogeneity, we aimed to examine the blood parameters and coagulation factors of patients before and after contracting the virus, in case of change, it can be introduced as a valuable parameter to doctors.

Material and Methods

In this retrospective study, 94 patients (42 males and 52 females) aged 21 to 80 yr with confirmed COVID-19 infection (via Real-time PCR or CT scan) were selected. According to medical records, these patients had undergone routine examinations and blood cell counts and coagoulation tests in the Allameh Bohlool Gonabadi Hospital, Gonabad, Iran System during six months between Apr 21, 2022 and Sep 22, 2022. All participants provided informed consent.

Exclusion criteria included patients with malignancy, underlying autoimmune diseases, readmission due to COVID-19, or incomplete medical records. We reviewed the medical records of 94 patients and analyzed the results of complete blood counts (CBC) performed using cell counter (Sysmex KX-21). The evaluated parameters included RBC count, hematocrit (HCT), hemoglobin (Hb), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), platelet count, mean platelet volume (MPV), and white blood cell (WBC) count, including neutrophils and lymphocytes.

In addition, a PTT and PT results obtained using a Nihon Kohden instrument were reviewed from the patient records. Additionally, D-dimer test results were reviewed for 37 patients who underwent testing using the Succeeder kit.

Statistical analysis

Data are showed as mean ± standard deviation (SD). Statistical analyses were conducted using paired *t*-tests in GraphPad Prism version 9 (GraphPad Software, USA), with a *P*-value of less than 0.05 considered statistically significant.

Ethics approval and consent to participate

Ethics approval was granted by the Ethics Committee of Gonabad University of Medical Sciences (Ethics No. IR.GMU.REC.1401.022).

Results

CBC test was used to measure changes in blood parameters (RBCs, MCV, HCT, hemoglobin, MCH, platelets, WBCs, lymphocytes, and neutrophils) before and after infection with COVID-19, and D-dimer, PT, and PTT tests were used to examine coagulation changes. The results and their changes are presented in Table 1.

Table 1: Changes in blood and coagulation parameters in patients with COVID-19 infection. All samples were from 94 patients except D-dimer which was from 37 patients

Test	*Mean±SD Before*	*Mean±SD After*	*P-value*
Prothrombin Time (PT)	13.04±1.02	14.27±2.41	<0.0001*
Partial Thromboplastin Time (PTT)	31.94±4.35	36.02±10.97	0.0003*
D-Dimer	472.4±302.1	1210±992.1	<0.0001*
Platelet	242032±74840	202500±85229	<0.0001*
Red blood cells (RBCs)	4.62±0.51	4.37±0.73	0.0004*
Hematocrit (HCT)	38.43±6.59	40.63±7.36	0.0045*
Hemoglobin (Hb)	13.46±1.60	12.81±2.20	0.001*
Mean corpuscular hemoglobin (MCH)	29.42± 2.236	29.32± 1.800	0.5190
Mean corpuscular volume (MCV)	87.06±5.32	85.91±5.69	0.015*
White blood cells (WBCs)	8296±2635	9933±4020	0.0003*
Neutrophil (Neu)	5781±2632	7808±3659	<0.0001*
Lymphocyte (Lymph)	1976±1048	1429±963.8	<0.0001*

* Indicates statistical significance

The results indicated a significant decrease after COVID-19 infection in platelet count (*P*<0.0001), lymphocyte count (*P*<0.0001), RBCs count (*P*=0.0004), hemoglobin level (*P*=0.001), and MCV (*P*=0.015). A decrease in MCH was also observed, but it was not statistically significant (*P*=0.519). In contrast, WBCs count (*P*=0.0003), neutrophil count (*P*<0.0001), and HCT (*P*=0.0045) increased significantly after infection.

Coagulation profiles were also significantly altered. The results demonstrated a significant increase in PT (*P*<0.0001) and PTT (*P*=0.0003) after COVID-19 infection. D-dimer levels were measured in a subset of 37 patients and were also found to be significantly elevated (*P*<0.0001). Fig. 1 illustrates the changes in these parameters.

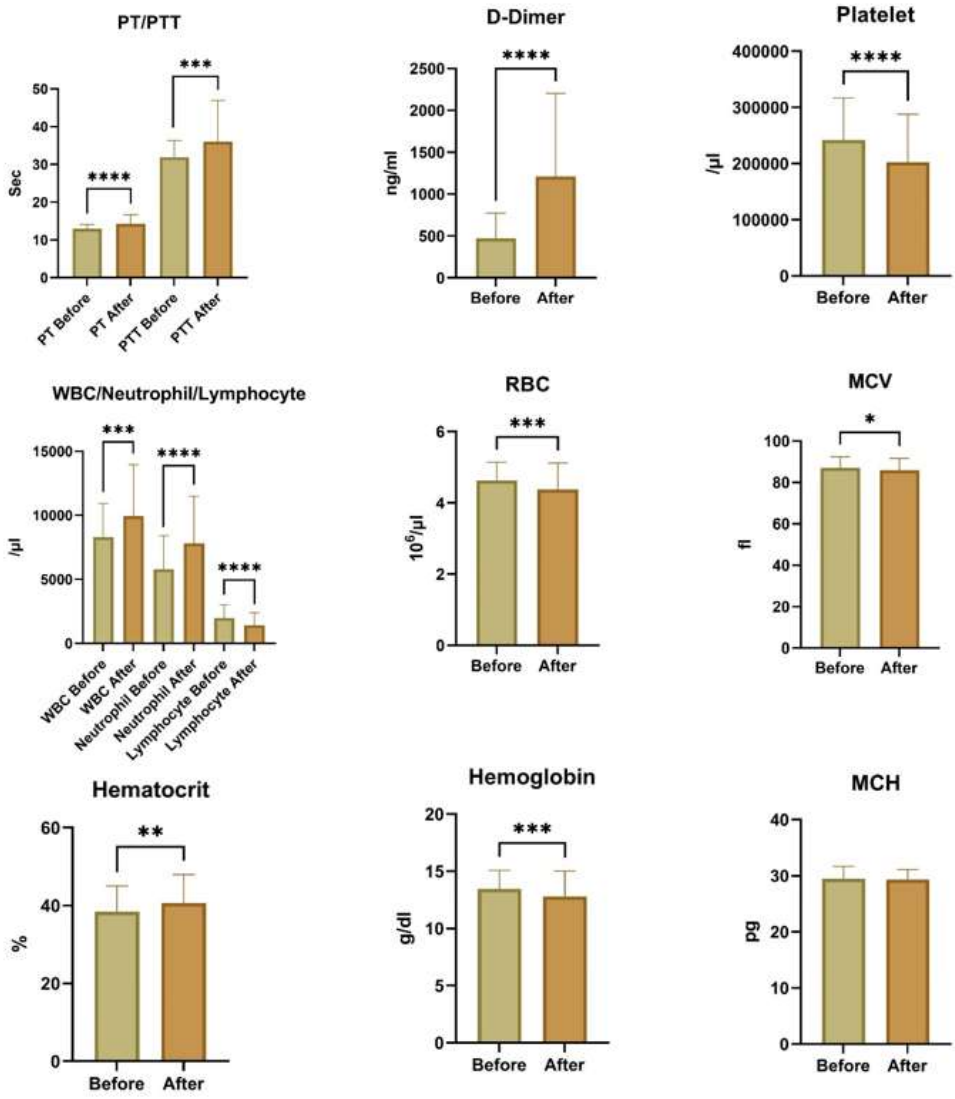

Fig. 1: Changes in hematological parameters before and after COVID-19 infection were analyzed. Data were collected from 94 patients, except for D-dimer levels, which were available for 37 patients. Following infection with COVID-19, significant increases were observed in prothrombin Time (PT), partial thromboplastin time (PTT), D-dimer, white blood cells (WBCs), hematocrit and neutrophils. Conversely, there were significant decreases in lymphocytes, red blood cells (RBCs), mean corpuscular volume (MCV), platelet count and hemoglobin levels. Although mean corpuscular hemoglobin (MCH) also decreased, the change was not statistically significant. These hematological shifts reflect the systemic impact of COVID-19 and underscore their clinical relevance in disease monitoring and management

Discussion

A noticeable feature of the COVID-19 pandemic has been the heterogeneity in clinical presentations and outcomes (18). To better understand this variability, we investigated the hematological profiles of patients, aiming to provide insights for prognosis and treatment. In this study, records of 97 patients were reviewed, and parameters including RBC count, MCV, HCT, Hb, MCH, platelets, WBCs, lymphocytes, neutrophils, PT, aPTT, and D-dimer were

compared before and after contracting COVID-19. The severity of coagulation disorders varies across different racial groups, and as the virus spreads rapidly across borders, reevaluating hematological factors in diverse populations is crucial (18). During SARS-CoV-2 infection, the first line of defense is the immune response; however, excessive and dysfunctional activity of immune cells, particularly neutrophils and lymphocytes, may lead to tissue damage (19). Hematological findings from patients at Allameh Bohlool Gonabadi Hospital revealed leukocytosis, characterized by a slight decrease in lymphocytes and significant increase in neutrophils. Elevated leukocyte and neutrophil counts are associated with more severe COVID-19 and poorer patient prognosis (20). Neutrophils increase in the severe form of the disease (10), but neutrophil counts decline following treatment with anti-inflammatory and antiviral medications and subsequent recovery (21). Lymphopenia happens as the result of lysis and cytokine storm. Lymphocytes in the oral mucosa, digestive system, and lungs express the angiotensin-converting enzyme 2 (ACE2) receptor on their surfaces, making them direct targets for SARS-CoV-2 infection and subsequent lysis. The expression of the ACE2 receptor is upregulated in individuals with hypertension, cardiovascular diseases, and diabetes, rendering these patients more susceptible to severe COVID-19. Additionally, the cytokine secresion, marked by increased levels of IL-7, IL-6, IL-2, granulocyte colony-stimulating factor (G-CSF), and monocyte chemoattractant protein-1 (MCP1), accelerates lymphocyte apoptosis, exacerbating lymphopenia (19, 22).

Furthermore, impaired nutrient circulation hampers hemoglobin synthesis and destabilizes RBC membranes, resulting in increased RDW and a reduction in RBC counts (23). A reduction in hemoglobin levels was observed in patients with COVID-19. Inflammatory responses triggered by SARS-CoV-2 infection can disrupt erythropoiesis, leading to decreased hemoglobin concentrations. In patients with severe COVID-19, hemoglobin levels are notably lower compared to those in the non-severe group, suggesting that declining hemoglobin levels may serve as an indicator of disease progression (4). Although DeMartino et al. (24) reported that neither hemolytic anemia nor alterations in the hemoglobin–oxygen dissociation curve were observed in COVID-19 patients; more studies could help clarify the effects of the disease on RBC counts and hemoglobin concentration.

Platelet count is a rapid, accessible, and cost-effective laboratory parameter that can differentiate between severe and mild infections (25). In this investigation, the number of platelets was favorable with previous studies (26). If the patient's hospitalization time increases, thrombocytopenia is observed (27), however, the patient's platelet count may fluctuate during the hospitalization (28). Several distinct mechanisms contribute to thrombocytopenia in COVID-19 patients.

Firstly, SARS-CoV-2 can directly infect bone marrow stromal and hematopoietic cells, leading to apoptosis and disrupted cell proliferation. Secondly, immune complexes and autoantibodies can cause immune-mediated damage to blood cells. Another key mechanism is a mild form of DIC, which arises from impaired coagulation time and an increase in D-dimer, ultimately consuming and decreasing the number of platelets. Finally, the virus may also reduce platelet production or increase their consumption within damaged lung tissue (27). Studies have reported varying results regarding PT and PTT changes in COVID-19 patients. An increase has been shown in PT in patients (13, 29, 30), whereas Shukla et al. (31) reported no such increase. Elevated PT and aPTT have also been noted in association with reduced fibrinogen levels (13). In contrast, Shukla et al. (31) found no increase in aPTT, although, did report elevated D-dimer levels correlated with disease severity. Overall, increases in D-dimer and PT

are characteristic of severe COVID-19 and may serve as predictive markers for mortality (13, 29).

This study identified hematological alterations in patients with COVID-19; however, several limitations must be acknowledged. The cohort size, particularly for D-dimer analysis, was limited, which may affect the generalizability of the findings and warrants validation in a larger population. Furthermore, the analysis was restricted to quantitative changes in cell counts and standard laboratory parameters. Functional assays and molecular analyses were not performed, which would be necessary to elucidate the underlying mechanisms of the observed cellular changes. Finally, since this study used hospital patient records, participants, especially those with a history of coagulation tests, may have had underlying conditions. This could affect the results and limit their generalizability to healthy individuals.

Conclusion

COVID-19, caused by the novel coronavirus SARS-CoV-2, has created substantial challenges for healthcare systems worldwide due to its rapid transmission and variable clinical outcomes. Effective disease management requires the identification of reliable biomarkers to predict disease severity and prognosis, enabling tailored clinical interventions. This study focused on evaluating hematological and coagulation parameters before and after SARS-CoV-2 infection. Key findings revealed neutrophilia, lymphopenia, thrombocytopenia, elevated D-dimer levels, and prolonged PT and PTT. These alterations are especially critical in managing COVID-19 patients, particularly those requiring surgical care. The observed changes in blood and coagulation profiles underscore the importance of continuous monitoring to assess disease progression and guide treatment strategies. Early detection of these abnormalities can help anticipate complications such as thrombosis, cytokine storms, and multi-organ failure, thereby enhancing patient outcomes.

Acknowledgements

This study was supported by Gonabad University of Medical Sciences research committee [Grant No.969]. Authors would like to acknowledge the Department of Hematology at Gonabad University of Medical Sciences and Allameh Bohlool Hospital (Iran) for their great help.

Conflict of interest

The authors declare that there is no conflict of interests.

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