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Original Article

Understanding inflammatory markers associated with clinical Symptoms, Hematological Profiles, and Biochemical Alterations of COVID-19 patients in Missan City

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Abstract

Coronavirus Disease 2019 (COVID-19) is a multisystem infectious disease caused by SARS-CoV-2, associated with a wide range of clinical, hematological, and biochemical alterations. This case-control study aimed to evaluate and compare clinical manifestations, hematological profiles, and biochemical parameters between COVID-19 patients and healthy individuals in Missan City, Iraq. A total of 90 participants were enrolled and divided equally into two groups: 45 confirmed COVID-19 patients and 45 healthy controls. Blood samples were collected and analyzed for hematological indices, lipid profile, liver and kidney function tests, inflammatory markers, and glucose levels.

The results demonstrated that fever (66.7%) and cough (46.7%) were the most common clinical symptoms. Significant hematological changes were observed in infected patients, including decreased red blood cells and hemoglobin levels, along with increased white blood cells, neutrophils, and platelets, while lymphocyte counts were markedly reduced. Biochemical analysis revealed significant elevations in liver enzymes (ALT, AST), kidney markers (urea, creatinine), glucose, and lactate dehydrogenase (LDH). Additionally, lipid profile disturbances were evident, with increased cholesterol, LDL, and triglycerides, alongside decreased HDL levels. Inflammatory markers such as C-reactive protein (CRP), ferritin, and D-dimer were significantly elevated, indicating severe inflammation and hypercoagulability.

In conclusion, COVID-19 is not limited to respiratory involvement but affects multiple organ systems, leading to significant laboratory abnormalities. Monitoring hematological, biochemical, and inflammatory parameters is essential for assessing disease severity and guiding clinical management.

Keywords: COVID-19; Hematological changes; Biochemical alterations; Inflammatory markers; Lipid profile; Case-control study.

Introduction

Coronavirus Disease 2019 (COVID-19) is a highly infectious respiratory illness caused by the novel Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2). The disease was first reported in December 2019 in Wuhan, China, and rapidly spread worldwide, leading the World Health Organization (WHO) to declare it a global pandemic in March 2020 (1). Since then, COVID-19 has posed a major challenge to global healthcare systems due to its high transmissibility, evolving variants, and wide spectrum of clinical manifestations. SARS-CoV-2 is an enveloped, positive-sense single-stranded RNA virus belonging to the β-coronavirus genus.

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Structurally, it contains four main proteins: spike (S), membrane (M), envelope (E), and nucleocapsid (N) proteins. The spike glycoprotein plays a critical role in viral entry by binding to the angiotensin-converting enzyme 2 (ACE2) receptor on host cells, particularly in the respiratory epithelium, vascular endothelium, and other organs (2). Following entry, the virus replicates within host cells and triggers a cascade of immune responses.

Transmission of SARS-CoV-2 occurs primarily through respiratory droplets and aerosols generated during coughing, sneezing, speaking, or breathing. Indirect transmission via contaminated surfaces has also been reported, although it is less common (3). The incubation period typically ranges from 2 to 14 days, during which infected individuals may be asymptomatic yet capable of spreading the virus. Clinically, COVID-19 presents with a wide spectrum of manifestations ranging from asymptomatic infection to severe disease. Common symptoms include fever, dry cough, fatigue, dyspnea, and myalgia. In more severe cases, patients may develop pneumonia, acute respiratory distress syndrome (ARDS), multi-organ failure, and death (4). Certain populations, including older adults and individuals with comorbidities such as diabetes, hypertension, and cardiovascular diseases, are at higher risk of severe outcomes (5). From an immunological perspective, SARS-CoV-2 infection is associated with significant dysregulation of both innate and adaptive immune responses. Antigen-presenting cells (APCs) process viral antigens and present them via major histocompatibility complex (MHC) molecules to T lymphocytes, initiating adaptive immunity. However, severe COVID-19 cases are often characterized by lymphopenia, impaired T-cell function, and excessive production of pro-inflammatory cytokines, commonly referred to as a “cytokine storm” (6).

In addition to immunological alterations, COVID-19 has been shown to induce significant hematological changes. These include decreased lymphocyte counts, elevated neutrophil levels, and alterations in hemoglobin and platelet counts. Such changes are considered important indicators of disease severity and progression (7). Furthermore, biochemical abnormalities are frequently observed, including elevated liver enzymes (ALT, AST), renal function markers (urea, creatinine), and inflammatory biomarkers such as C-reactive protein (CRP), ferritin, and lactate dehydrogenase (LDH) (8).

Emerging evidence also suggests that COVID-19 affects metabolic pathways, including lipid metabolism. Increased levels of cholesterol, low-density lipoprotein (LDL), and triglycerides have been reported in infected individuals, potentially contributing to endothelial dysfunction and increased cardiovascular risk (9). Additionally, elevated D-dimer levels indicate activation of coagulation pathways, which may lead to thrombotic complications, a recognized feature of severe COVID-19 cases (10). Given the systemic nature of COVID-19 and its impact on multiple organ systems, laboratory investigations play a crucial role in diagnosis, monitoring, and prognosis. Hematological and biochemical parameters provide valuable insights into disease severity and can guide clinical management.

Therefore, this study aims to compare clinical features, hematological profiles, and biochemical alterations between COVID-19 patients (treatment group) and healthy individuals (control group) in Missan City. Understanding these differences may contribute to improved diagnostic accuracy and better management strategies for affected patients.

Materials and Methods

This study was designed as a **case-control investigation** to compare clinical, hematological, and biochemical findings between COVID-19 patients and healthy individuals. The study was conducted at the Baby and Maternity Hospital (COVID-19 center) in

Amara City, Missan, Iraq, in the period extending from December 2021 to May 2022. Only (90) of patients agreement to participate were enrolled and equally divided into two groups (treatment and control group).

The **treatment group** consisted of (45) patients who were confirmed positive for SARS-CoV-2 infection using reverse transcription polymerase chain reaction (RT-PCR).

The **control group** included (45) apparently healthy individuals with no prior history of COVID-19 and negative RT-PCR test results. Participants were selected according to specific inclusion criteria: individuals aged between 20 and 70 years. In the treatment group, only confirmed COVID-19 cases were included, while the control group was free from chronic or severe diseases to ensure appropriate comparison.

Laboratory investigations were performed by taking 6 ml of venous blood from each participant; the collected blood samples were divided into two portions: 3 ml was transferred into EDTA tubes for hematological analysis, and 3 ml was placed in plain tubes, allowed to clot, and then centrifuged to obtain serum for biochemical analysis. Hematological parameters were assessed using an automated hematology analyzer. The evaluated parameters included hemoglobin (Hb), red blood cells (RBCs), white blood cells (WBCs), platelet count, neutrophil percentage, and lymphocyte percentage. These measurements were used to evaluate the impact of COVID-19 on blood cell profiles.

Biochemical analysis was performed on serum samples to assess multiple physiological functions. The investigated parameters included lipid profile (total cholesterol, low-density lipoprotein (LDL), and triglycerides), liver function enzymes (alanine aminotransferase (ALT) and aspartate aminotransferase (AST)), kidney function indicators (urea and creatinine), inflammatory markers (C-reactive protein (CRP) and ferritin), lactate dehydrogenase (LDH), and blood glucose levels.

Statistical Analysis:

All data obtained were statistically analyzed using SPSS version 17. Results were expressed as (Mean ± SD). Comparisons between the treatment and control groups were performed using the **t-test**; a p-value of <0.05 was considered statistically significant.

Results

Demographic Characteristics

1) Sex Distribution:

The results showed no statistically significant difference between males and females in the treatment and control groups at a significant level ($P < 0.05$). This indicates that the two groups were well matched in terms of exposure to COVID-19, and that gender is not a confounding factor in the occurrence and spread of infection. Although the number of males was slightly higher, this difference was not statistically significant, meaning that any observed changes in laboratory test results are most likely attributable to the disease itself and not to gender differences Table 1.

2) Age Distribution:

Table 2 shows the age distribution of participants in both the COVID-19 patient group (treatment group) and the control group, each consisting of 45 individuals. In the treatment group, the largest percentage of participants was found to be in the 51-60 age group (31.1%), followed by the 61 and older age group (24.4%). The lowest percentage was found in the 20-30 age group (8.9%), indicating that most of the infected patients in the sample were middle-aged and elderly adults. In the control group, the highest percentage was found in the 51-60 age group (26.7%), followed by the 41-50 age group (22.2%), and then the 61 and older age group (20.0%). The youngest age group (20-30 years) represented the lowest percentage (13.3%), at a significance level of $P > 0.05$. This indicates no statistically significant difference in age distribution between the treatment and control groups,

and that age is unlikely to influence the comparison between COVID-19 patients and healthy individuals in this study. Furthermore, the higher proportion of older participants in the treatment group is consistent with known trends suggesting that COVID-19 tends to affect middle-aged and older individuals more frequently or more severely Tab. 2.

Table 1: Distribution of Participants by Sex (n = 45 per group)

SEX	TREATMENT GROUP (N=45)	CONTROL GROUP (N=45)	TOTAL
Male	27 (60.0%)	26 (57.8%)	53
Female	18 (40.0%)	19 (42.2%)	37

Table 2: Age Distribution of Participants (n = 45 per group)

AGE GROUP (YEARS)	TREATMENT GROUP	CONTROL GROUP
20–30	4 (8.9%)	6 (13.3%)
31–40	7 (15.6%)	8 (17.8%)
41–50	9 (20.0%)	10 (22.2%)
51–60	14 (31.1%)	12 (26.7%)
≥61	11 (24.4%)	9 (20.0%)

Clinical Symptoms:

Table 3 shows the distribution of the main clinical symptoms in patients with COVID-19 (n = 45). The results highlight the most common symptoms of the disease within the study sample. The results showed that the most common symptom was fever, affecting 30 patients (66.7%). This indicates that fever is the predominant clinical presentation of COVID-19 in this group, reflecting the body's systemic inflammatory response to the viral infection. This high prevalence of fever also highlights its role as an early and common indicator of infection.

Cough was the second most common symptom, affecting 21 patients (46.7%). This finding suggests that half of the patients experienced respiratory tract involvement, as cough is typically associated with irritation or inflammation of the airways. This reinforces the notion that COVID-19 is a predominantly respiratory illness. The study also showed that shortness of breath was present in 10 patients (22.2%), making it the least common symptom. Shortness of breath is clinically significant and indicates disease progression and the potential involvement of the lower respiratory tract, including lung tissue damage (Tab. 3).

Hematological Changes:

Table 4 shows the hematological parameters between COVID-19 patients (treatment group) and healthy individuals (control group). The results show highly significant differences ($P < 0.05$) in all parameters, indicating a strong correlation between COVID-19 infection and hematological changes.

The results indicated a decrease in red blood cell count in the treatment group (3.85 ± 0.60) compared to the control group (4.95 ± 0.40). This decrease may be attributed to the fact that COVID-19 patients experienced anemia during infection due to inflammation, impaired red blood cell production, or increased red blood cell destruction during infection.

Hemoglobin levels were significantly lower in infected patients (11.2 ± 1.8) compared to the control group (13.6 ± 1.2) because COVID-19 patients experienced systemic inflammation that suppressed bone marrow function. Conversely, white blood cell counts were significantly higher in the treated group (14.8 ± 3.2) compared to the control group (7.2 ± 1.5). This increase in white blood cells, which may be associated with infection, reflects activation of the immune system in response to SARS-CoV-2.

Lymphocyte levels were significantly lower in the treated group (14.2 ± 6.5) compared to the control group (32.5 ± 7.2), indicating that lymphopenia is a hallmark of COVID-19. This may result from direct viral attack on lymphocytes, apoptosis (programmed cell death), or immune system depletion. Lymphopenia is often associated with disease severity and a poor prognosis. Regarding platelet count, COVID-19 patients showed a significantly higher mean platelet count (410 ± 120) compared to the control group (260 ± 85). This increase suggests reactive thrombocytosis, which may be associated with systemic inflammation and increased production of inflammatory cytokines. Elevated platelet counts may also contribute to the hypercoagulability observed in COVID-19 patients Tab. 4.

Table 3: Clinical Symptoms (n = 45 patients)

SYMPTOM	FREQUENCY	PERCENTAGE
Fever	30	66.7%
Cough	21	46.7%
Dyspnea	10	22.2%

Table 4: Shows Hematological Parameters (n = 45 for each group)

PARAMETER	TREATMENT (MEAN ± SD)	CONTROL (MEAN ± SD)
RBCs $\times 10^6/\mu L$	3.85 ± 0.60	4.95 ± 0.40
Hb g/dL	11.2 ± 1.8	13.6 ± 1.2
WBCs $\times 10^3/\mu L$	14.8 ± 3.2	7.2 ± 1.5
Neutrophils %	78.5 ± 10.4	55.2 ± 8.3
Lymphocytes %	14.2 ± 6.5	32.5 ± 7.2
Platelets $\times 10^3/\mu L$	410 ± 120	260 ± 85

Lipid Profile Alterations

Table 5 illustrates the differences in lipid profile parameters between COVID-19 patients and healthy individuals. All parameters show highly significant differences ($P < 0.05$), indicating a strong effect of infection on lipid metabolism.

The results showed a significant increase in total cholesterol levels in the treatment group (235.6 ± 75.4) compared to the control group (165.2 ± 40.3). This increase suggests that COVID-19 infection is associated with elevated blood cholesterol, which may result from metabolic disturbances caused by systemic inflammation and impaired liver function. Low-density lipoprotein (LDL) cholesterol levels were also significantly higher in infected patients (180.3 ± 42.5) compared to the control group (120.4 ± 30.2). Elevated LDL cholesterol can contribute to atherosclerosis ("bad cholesterol") and may contribute to endothelial dysfunction and an increased risk of cardiovascular disease, a common complication of COVID-19.

A significant increase in triglyceride levels was observed in the treated group (290.7 ± 95.6) compared to the control group (150.3 ± 45.7), which is likely related to the release of inflammatory cytokines and impaired lipid clearance during infection. Elevated triglyceride levels may exacerbate inflammation and lead to poor clinical outcomes. Conversely, high-density lipoprotein (HDL) levels were significantly lower in COVID-19 patients (33 ± 8.5*) compared to the control group (63 ± 5.3*). HDL is known as "good cholesterol" due to its protective role in removing excess cholesterol and its anti-inflammatory effects. Reduced HDL levels indicate a loss of this protective lipid function, which may contribute to disease severity and increased oxidative stress (Table 5)

Table 5: Show Lipid Profile(n = 45 for each group)

PARAMETER	TREATMENT	CONTROL
Cholesterol mg/dL	235.6 ± 75.4	165.2 ± 40.3
LDL mg/dL	180.3 ± 42.5	120.4 ± 30.2
Triglycerides mg/dL	290.7 ± 95.6	150.3 ± 45.7
HDL mg/dL	33± 85.3	63± 45.3

Table (6) compares some vital signs in COVID-19 patients with the control group. All parameters show highly significant differences ($P < 0.05$), indicating systemic effects of the infection on multiple organs, particularly the liver, kidneys, and other metabolic systems.

The results showed significantly elevated levels of the liver enzymes alanine aminotransferase (ALT) and aspartate aminotransferase (AST) in the treatment group (ALT: 62.4 ± 20.3; AST: 70.8 ± 25.6) compared to the control group (ALT: 28.5 ± 10.2; AST: 24.3 ± 9.8). This elevation may be attributed to liver damage or dysfunction, which could result from direct viral effects, hypoxia, drug toxicity, or systemic inflammation. The higher AST level compared to ALT may also indicate more severe liver tissue injury. Kidney function tests, including creatinine and urea, were significantly elevated in COVID-19 patients (creatinine: 1.45 ± 0.30; urea: 78.2 ± 20.5) compared to the control group (creatinine: 0.90 ± 0.20; urea: 25.6 ± 8.4). This may be due to impaired kidney function or acute kidney injury, a known complication of COVID-19. Elevated urea levels also indicate decreased renal clearance and increased protein catabolism.

Blood glucose levels were significantly higher in the treatment group (250.6 ± 85.3) compared to the control group (105.2 ± 20.1). This marked increase may indicate hyperglycemia, a response to stress, insulin resistance, or pancreatic injury during infection. Elevated glucose levels are clinically significant because they are associated with worsening and increased disease severity in COVID-19 patients. An increase in the level of lactate dehydrogenase (LDH) enzyme was also observed in infected patients (480.5 ± 110.2) compared to the control group (190.4 ± 45.3), which is a non-specific indicator of tissue damage and cell lysis. Its increase reflects the spread of tissue damage, including lung damage, which is a hallmark of COVID-19 infection Tab. 6.

Table 6: Observed Biochemical Parameters (n = 45 for each group)

PARAMETER	TREATMENT	CONTROL
ALT U/L	62.4 ± 20.3	28.5 ± 10.2
AST U/L	70.8 ± 25.6	24.3 ± 9.8
Creatinine mg/dL	1.45 ± 0.30	0.90 ± 0.20
Urea mg/dL	78.2 ± 20.5	25.6 ± 8.4
Glucose mg/dL	250.6 ± 85.3	105.2 ± 20.1
LDH U/L	480.5 ± 110.2	190.4 ± 45.3

Inflammatory Markers

Table (7) shows the levels of inflammatory markers in COVID-19 patients compared to healthy individuals. All markers show highly significant differences at the $p < 0.001$ level, indicating a severe inflammatory state and hypercoagulability associated with the infection.

The results also showed a significantly elevated C-reactive protein (CRP) level in the treatment group (12.8 ± 4.5) compared to the control group (0.8 ± 0.5). CRP is an acute phase protein produced by the liver in response to inflammation. Its marked elevation reflects a strong systemic inflammatory response, a hallmark of COVID-19 infection, and is associated with disease severity. The results also showed elevated serum ferritin levels in COVID-19 patients (710.4 ± 150.6) compared to the control group (120.5 ± 60.2). In addition to being an iron storage protein, ferritin is also an inflammatory marker. Therefore, elevated ferritin levels indicate excessive inflammation or a "cytokine storm," a severe immune response that can lead to tissue damage and organ failure in critically ill patients.

A significant increase in D-dimer levels was observed in the treated group (6.2 ± 2.1) compared to the control group (1.5 ± 0.6). D-dimer is a fibrinolysis product and an indicator of activation of the coagulation and fibrinolysis systems. Its elevation suggests hypercoagulability, a condition common in COVID-19 and associated with complications such as blood clots, pulmonary embolism, and an increased risk of death (Tab. 7).

Table 7: Detect the Inflammatory Markers(n = 45 for each group)

PARAMETER	TREATMENT	CONTROL
CRP mg/L	12.8 ± 4.5	0.8 ± 0.5
Ferritin ng/mL	710.4 ± 150.6	120.5 ± 60.2
D-Dimer µg/mL	6.2 ± 2.1	1.5 ± 0.6

Discussion

This study was conducted to compare the pathological and clinical changes, as well as the changes in hematological, biochemical, and inflammatory parameters, in COVID-19 patients compared to healthy individuals in Maysan City. The results showed that COVID-19 is not limited to the respiratory system but is a systemic disease affecting multiple organs and physiological pathways (4, 5).

The results indicated the absence of statistically significant differences in age and sex distribution between the treatment and control groups, suggesting a suitable match between the two study groups. This

aligns with the findings of some researchers who emphasized that the observed differences are primarily due to infection rather than demographic factors (11). However, the high proportion of elderly patients among those infected confirms that advanced age is a major risk factor for severe COVID-19 due to age-related immunodeficiency and comorbidities (5).

Clinically, fever and cough were the most common symptoms, consistent with typical COVID-19 infection (12).

Clinically, fever and cough were the most common symptoms of COVID-19 infection, consistent with the findings (12). Fever reflects a systemic inflammatory response mediated by cytokines, while cough indicates respiratory tract infection. Shortness of breath, although less common, is clinically significant because it suggests the progression of the infection to lower respiratory tract infection and the potential development of acute respiratory distress syndrome (ARDS) (4).

The results showed significant changes in the hematological profile of COVID-19 patients. A decrease in red blood cell count and hemoglobin levels suggests anemia, which may result from inhibition of red blood cell production due to inflammation and increased red blood cell destruction (8). Elevated white blood cell and neutrophil counts indicate activation of the innate immune response during infection. These findings are consistent with (7), which indicated that neutropenia is associated with systemic inflammation and disease severity. A marked decrease in lymphocyte count was also observed, a hallmark of COVID-19 infection. This may result from direct viral damage to lymphocytes or cytokine-induced apoptosis and is strongly associated with a poor prognosis (13,14). The elevated platelet count observed in patients suggests reactive thrombocytosis resulting from inflammation and cytokine release. This may contribute to the hypercoagulability observed frequently in COVID-19 patients (15).

The study showed a significant increase in cholesterol, LDL (bad cholesterol), and triglyceride levels, along with a decrease in HDL (good cholesterol). These findings suggest metabolic dysfunction resulting from systemic inflammation and liver involvement (9). Low HDL levels have a negative impact on organ function and are particularly important due to their anti-inflammatory and antioxidant properties. Low HDL levels may increase the risk of cardiovascular disease in affected patients (16). These lipid disturbances may contribute to endothelial dysfunction and exacerbate disease severity. The results also showed a significant increase in the levels of the enzymes alanine aminotransferase (ALT) and aspartate aminotransferase (AST) due to liver tissue and cell damage in COVID-19 patients. This may be attributed to direct viral effects, hypoxia, or drug toxicity (15).

Meanwhile, the elevated urea and creatinine levels in this study indicate impaired kidney function, a major known complication of COVID-19, which may result from systemic inflammation and reduced renal perfusion (15). The results also showed hyperglycemia in patients, which may be due to the body's stress response, insulin resistance, or damage to pancreatic beta cells. This is consistent with previous studies indicating that elevated glucose levels may be associated with worsening COVID-19 symptoms (17). Elevated levels of lactate dehydrogenase (LDH) indicate tissue damage and cell injury in lung tissue and are considered an indicator of disease severity and progression (8,18).

The results also showed a significant increase in C-reactive protein (CRP), ferritin, and D-dimer levels in COVID-19 patients. CRP is an acute-phase protein that reflects acute systemic inflammation, and the elevated ferritin level is attributed to hyperinflammation and a cytokine storm, both major factors in disease progression (6).

Elevated D-dimer levels indicate activation of coagulation pathways and fibrinolysis. This is closely associated with coagulation complications such as

pulmonary embolism and an increased risk of death (13).

In conclusion: which based on the results of this study, found that COVID-19 is a multisystem infectious disease, not merely a respiratory infection, and is characterized by immune system dysfunction, hyperinflammation, coagulation disorders, and organ dysfunction. COVID-19 infection leads to significant hematological, biochemical, and inflammatory changes. The disease is associated with anemia, leukocytosis, lymphopenia, metabolic disorders, impaired liver and kidney function, and elevated inflammatory markers. Based on the results of this study and the conclusions we reached, we recommend the periodic monitoring of hematological and biochemical parameters in COVID-19 patients for the early detection of complications that may occur after infection. **In addition,** it is preferable to use inflammatory markers such as C-reactive protein, ferritin, lactate dehydrogenase, and D-dimer to assess the severity and progression of the disease within the body. Continuous monitoring of liver and kidney functions during treatment is also recommended, and blood glucose levels should be carefully controlled in infected patients. Furthermore, further studies are needed to validate these results and explore the long-term complications of COVID-19.

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