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Research Article

Coagulation Profile in COVID-19 patients

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Abstract
Background: COVID-19 caused by the SARS-CoV-2 virus, has been associated with a range of systemic complications, including significant coagulation abnormalities. Emerging evidence suggests that COVID-19-induced coagulopathy contributes to poor clinical outcomes and is often linked to inflammatory responses. **Objective:** To assess D-dimer, prothrombin time (PT) and activated partial thromboplastin time (aPTT) in COVID-19 patients compared to healthy individuals, and to explore their association with clinical features and inflammatory markers. **Methods:** This case-control study included 38 COVID-19 patients in addition to 16 age and sex-matched as a healthy control. Laboratory investigations were done and compared between groups. Clinical data, including comorbidities and symptoms, were recorded. **Results:** COVID-19 patients showed significantly elevated D-dimer levels ($p < 0.001$) and prolonged PT and aPTT values ($p < 0.05$) compared to controls, a significant positive correlation was observed between D-dimer levels and both LDH ($r = 0.660$, $p < 0.001$) and CRP ($r = 0.434$, $p = 0.021$), indicating that higher D-dimer levels were associated with increased inflammatory markers. **Conclusion:** Our study emphasizes the significance of evaluating parameters of coagulation in COVID-19 infection. This study highlights the significant elevation of coagulation markers—particularly D-dimer—and their correlation with inflammatory parameters such as CRP and LDH in COVID-19 patients. These findings support the hypothesis that COVID-19-induced coagulopathy is closely linked to systemic inflammation. Early identification of these abnormalities may serve as prognostic indicators and guide clinical decisions regarding anticoagulation and supportive therapies.

Keywords: aPTT, INR, D-dimer, coagulation, ferritin, LDH, COVID-19.

Introduction
Since its emergence in late 2019, coronavirus disease 2019 (COVID-19), caused by the novel coronavirus SARS-CoV-2, has severely affected the global health systems, economies, and societies. Initially recognized as a primarily respiratory illness, COVID-19 is now established as a multisystem disease with significant extrapulmonary manifestations [1]. Manifestations ranged from mild respiratory issues to severe complications such as acute respiratory distress syndrome (ARDS), coagulopathy, multiorgan dysfunction, and even death [2].

One of the most frequent hematological complications of COVID-19 is coagulopathy, which has been associated with multiple organ dysfunction, venous thrombosis, and poor prognosis [3]. Studies have reported venous thrombosis in 40% of hospitalized COVID-19 patients and in 22.5% of those receiving prophylactic anticoagulation[4]. Although the exact pathogenesis of COVID-19-associated coagulopathy is not completely understood, numerous mechanisms have been proposed, including cytokine storm, neutrophil activation, endothelial dysfunction, platelet activation, tissue factor expression, and activation of the coagulation cascade [5].

COVID-19 is characterized by an atypical form of DIC, which differs from conventional DIC. It shows inconsistent abnormalities in coagulation markers across studies, particularly regarding their levels and association with disease severity and mortality [6,7]. In patients

with COVID-19-related pneumonia and ARDS, coagulation abnormalities have been frequently reported [8]. Notably, elevated D-dimer and fibrinogen levels are common findings among these patients [9,10]. Numerous studies have revealed that coagulopathy and inflammatory markers are significantly associted with COVID-19 severity [11]

However, few numbers of researches have assessed coagulation profiles, especially D-dimer and its correlation with inflammatory markers. Therefore, this study was conducted to assess the coagulation profile—including D-dimer, PT and aPTT levels in hospitalized COVID-19 cases compared with healthy individuals. Additionally, we aimed to evaluate the association of these coagulation markers with clinical parameters particularly oxygen saturation and inflammatory indicators (CRP, LDH, and ferritin) to better understand their clinical relevance and potential utility in guiding patient management and therapeutic decisions.

Subjects and methods

Study design

This prospective case–control study was done on a total of 54 participants over a six-month from January 2022 to June 2022. The study group comprised 38 patients with confirmed COVID-19 infection, all of whom tested positive for SARS-CoV-2 RNA by (RT-PCR) from nasopharyngeal swabs. These patients were hospitalized to the Chest Intensive Care Unit (ICU) of Minia University Hospital, Minia, Egypt with varying degrees of disease severity. We excluded patients with previous coagulopathy events and on oral anticoagulant and antiplatelets treatment and patients undergoing routine blood transfusion. The control group consisted of 16 apparently healthy individuals, matched to the patient group by age and sex, and confirmed to be free from acute or chronic diseases, including respiratory infections or coagulation abnormalities. The study was accepted by the Ethical Committee of the Faculty of Medicine, Minia University (Approval No.: 206:12/2021; Date of approval: 27 December 2021). All individuals (or their legal proxies) gave prior written informed consent in accordance with the Declaration of Helsinki.

Both patient and control groups underwent: Comprehensive history taking, with emphasis on comorbidities (e.g., diabetes, hypertension), symptoms (e.g., fever, cough, dyspnea, loss of smell/taste), and medication use. Full clinical examination, including vital signs and systemic evaluation. Radiological assessment, including chest X-ray or CT scan as clinically indicated. Pulse oximetry, to record oxygen saturation (SpO_2) on room air at time of blood sampling.

Laboratory investigations

Blood samples were collected from all participants under sterile conditions. Approximately 8 mL of venous blood was obtained from each participant using a disposable plastic syringe and was splitted as follows: 1.8 mL of blood was placed in a 0.2 mL trisodium citrate containing tube for the measurement of (PC), (APTT), and D-dimer. 4 mL of blood was collected into a plain tube. The blood was allowed to clot for 30 minutes in an incubator and then centrifuged at 3000 rpm for 15 minutes. The separated serum was used for the determination of renal function tests, liver enzymes, C-reactive protein (CRP), lactate dehydrogenase (LDH), and serum ferritin and 2 mL of blood was evacuated into Ethylenediaminetetraacetic acid (EDTA) containing tube for complete blood count (CBC).

CBC was performed using an automated hematology analyzer (Celltac G, Nihon Kohden Corporation, Japan). The differential leukocytic count was confirmed by microscopic examination of a Leishman-stained blood film. Prothrombin concentration (PC) and international normalized ratio (INR) were measured using the Stago STA Compact Max (France). APTT was determined by a turbodensitometric method using the Labitec CoaData 4004 (Biochemical Technology GmbH, Germany) with the Labitec APTT Reagent Kit. C-reactive protein (CRP) was measured by nephelometry using the Genuri Biotech Inc. kinetic assay (China). Renal function tests (blood urea and serum creatinine) and liver enzymes (ALT and AST) were measured using an automated analyzer (Selectra PRO XL, Elitech Group, Netherlands). D-dimer and serum ferritin levels were determined by kinetic fluorescence immunoassay using the TOSOH AIA 360 Automated Immunoassay Analyzer (Japan). LDH was measured by the kinetic method using a kit supplied by Spectrum Diagnostic

Company (Germany) and analyzed with the Microlab 300 (Elitech, Netherlands).

Statistical analysis:

SPSS software, version 25 was used for Data analysis. The Shapiro-Wilk test was used to evaluate the normality of the data. Parametric quantitative variables were mentioned as mean ± standard deviation (SD), together with their minimum and maximum values. Non-parametric quantitative data were reported as median and interquartile range (IQR). Qualitative data were showed using frequencies and percentages. Independent Samples t-test was used for Group comparisons for parametric data and the Mann-Whitney U test for non-parametric data. For comparison of categorical variables, the Chi-square test was used.

Pearson's correlation coefficient was used to evaluate the relationship between continuous variables. A p-value less than 0.05 was considered statistically significant.

Results

Demographic data and clinical manifestations of the COVID-19 patients:

The COVID-19 patient group (n = 38) had a mean age of 58.2 ± 16.1 years (range: 23–89), with an equal distribution of males and females (50% each). Among the patients, 60.5% had diabetes mellitus and 63.2% had hypertension. The most frequent presenting manifestations were fever (76.3%), dyspnea (71.1%), and cough (68.4%). Sore throat was present in 28.9% of patients, while loss of taste and smell and gastrointestinal (GIT) manifestations were reported in 13.2% and 10.5% of cases, respectively **(Table 1).**

Table (1): Demographic data and clinical manifestations of the studied groups:

Variables	**Patients N=38**
Age (years)	
Range	(23-89)
Mean ± SD	58.2±16.1
Sex n (%)	
Male	19(50%)
Female	19(50%)
Diabetes mellitus n (%)	
No	15(39.5%)
Yes	23(60.5%)
Hypertension n (%)	
No	14(36.8%)
Yes	24(63.2%)
Fever n (%)	
No	9(23.7%)
Yes	29(76.3%)
Cough n (%)	
No	12(31.6%)
Yes	26(68.4%)
Dyspnea n (%)	
No	11(28.9%)
Yes	27(71.1%)
Sore throat n (%)	
No	27(71.1%)
Yes	11(28.9%)
Loss of Taste & Smell n (%)	
No	33(86.8%)
Yes	5(13.2%)
GIT manifestations n (%)	
No	34(89.5%)
Yes	4(10.5%)

: Significant level at p value< 0.05

Comparison between studied groups as regard oxygen saturation and biochemical investigations: Patients showed high statistically significant decrease in oxygen saturation (90.5% ± 5.9) in comparison with the control group (98.8% ± 0.9) **(P value = <0.001)**. There was high statistically significant increase in serum urea, creatinine, ALT enzyme, AST enzyme and LDH enzyme in patients group when compared to control group **(P value = <0.001)**.

Moreover, there was high statistically significant increase in CRP and serum ferritin in patients group when compared to control group **(P value = <0.001) (Table 2).**

Table (2): Comparison between the studied groups regarding oxygen saturation, renal function tests, liver enzymes and LDH:

Variables	Patients N=38	Control N=16	P value
Oxygen saturation (%) *Range* *Mean ± SD*	 (60-96) 90.5±5.9	 (97-100) 98.8±0.9	***<0.001****
Urea (mg/dl) *Median* *IQR*	48 (35.8-64.8)	23 (20.3-28.5)	***<0.001****
Creatinine (mg/dl) *Median* *IQR*	1.2 (0.9-1.4)	0.7 (0.7-0.8)	***<0.001****
ALT (U/L) *Median* *IQR*	36.5 (24-53.8)	18 (12.5-21.5)	***<0.001****
AST (U/L) *Median* *IQR*	40.5 (27-62.8)	18 (16-21.8)	***<0.001****
LDH (U/L) *Median* *IQR*	360 (213.5-501.8)	131.5 (107-204.8)	***<0.001****
CRP (mg/l) *Median* *IQR*	24 (12-48)	1.5 (0.6-2.8)	***<0.001****
S. ferritin (ng/ml) *Median* *IQR*	583 (362.5-854.3)	81 (64-107.8)	***<0.001****

****: Significant level at p value< 0.05***

Comparison between the studied groups regarding different haematological parameters:
There was statistically significant decrease in Hb level in patients group when compared to control group (p value=0.010*). Also, there was statistically significant increase in TLC count in patients group when compared to control group (p value=0.009*). Moreover, there was high statistically significant decrease in relative lymphocytic count and absolute lymphocytic count in patients group when compared to control group (p value <0.001*). Also, there was statistically significant increase in relative neutrophilic count and absolute neutrophilic count in patients group when compared to control group (p value <0.001*, 0.001) respectively. There were no statistical significances regarding platelets, monocytes, eosinophils and basophils counts when compared between patients and control groups (p=0.086, p=0.587, p=0.488, p=0.804) respectively **(Table 3).**

Table (3): Comparison between the studied groups regarding different haematological parameters:

Variables	Patients N=38	Control N=16	P value
Hb (g/dl) *Range* *Mean ± SD*	 (6.1-17) 12±2.7	 (12.1-16.4) 13.9±1.4	***0.010******
TLC (x10^3 /µl) *Median* *IQR*	 11.5 (6.8-16.4)	 6.6 (5.7-9.5)	***0.009******
PLT (x10^3 /µl) *Median* *IQR*	 220 (159-291.8)	 285.5 (220.3-323.3)	*0.086*
Lymphocytes (relative%) *Median* *IQR*	 8 (6-17.3)	 31.5 (25.8-37.5)	***<0.001****
Absolute lymphocytes (count/ µl) *Median* *IQR*	 1002 (697-1536.8)	 2159 (1638.8-3115)	***<0.001****
Monocyte (relative%) *Median* *IQR*	 4.5 (3-5.3)	 5 (4-5)	*0.587*
Neutrophil (relative%) *Median* *IQR*	 79 (70-84)	 59.5 (56.3-65.5)	***<0.001****
Absolute neutrophils (count/ µl) *Median* *IQR*	 9317 (5376-13831.5)	 4132 (3789.8-5916.3)	***0.001****
Eosinophil (relative%) *Median* *IQR*	 1 (0-1)	 1 (1-1.8)	*0.488*
Basophil (%) *Median* *IQR*	 0 (0-1)	 0 (0-1)	*0.804*
Band (%) *Median* *IQR*	 3 (3-5.3)	 1 (1-2)	***<0.001****

****: Significant level at p value< 0.05***

Comparison between the studied groups regarding coagulation parameters:

Compared to the control group, COVID-19 patients showed statistically significant alterations in coagulation parameters. The mean prothrombin concentration (PC) was markedly reduced in patients (78.9 ± 15.2%) compared to controls (99.6 ± 1.5%) ($p < 0.001$). The international normalized ratio (INR) was significantly elevated in patients (1.2 ± 0.21) versus controls (1.004 ± 0.01) ($p = 0.001$), as was the activated partial thromboplastin time (APTT), which was prolonged in patients (39.4 ± 9.2 seconds) compared to controls (31.2 ± 3.1 seconds) ($p = 0.001$). Additionally, D-dimer levels were significantly higher in the patient group, with a median of 0.6 µg/ml compared to 0.2 µg/ml in controls ($p < 0.001$) **(Table 4).**

Table (4): Comparison of the studied groups regarding coagulation parameters:

Variables	Patients N=38	Control N=16	P value
PC (%)			
Range	(36-100)	(94-100)	***<0.001****
Mean ± SD	78.9±15.2	99.6±1.5	
INR			
Range	(1-2.2)	(1-1.05)	***0.001****
Mean ± SD	1.2±0.21	1.004±0.01	
APTT(seconds)			
Range	(27-71)	(27-36)	***0.001****
Mean ± SD	39.4±9.2	31.2±3.1	
D.D(µg/ml)			
Median	0.6	0.2	***<0.001****
IQR	(0.4-1.4)	(0.2-0.3)	

: Significant level at p value< 0.05

Correlations between oxygen saturation and coagulation profile in COVID-19 patients:
There was significant positive correlation between oxygen saturation and PC in COVID 19 patients (r = 0.366*, p value = 0.024*) (Table 5), Figure (1).

Table (5): Correlations between oxygen saturation and coagulation profile in COVID-19 patients:

Variable	Oxygen saturation	
	r	**P**
PC	**0.366**	**0.024***
APTT	-0.260	0.115
D-dimer	-0.186	0.264

*** Correlation is significant at P value < 0.05.**
**** Correlation is highly significant level at P value < 0.001.**

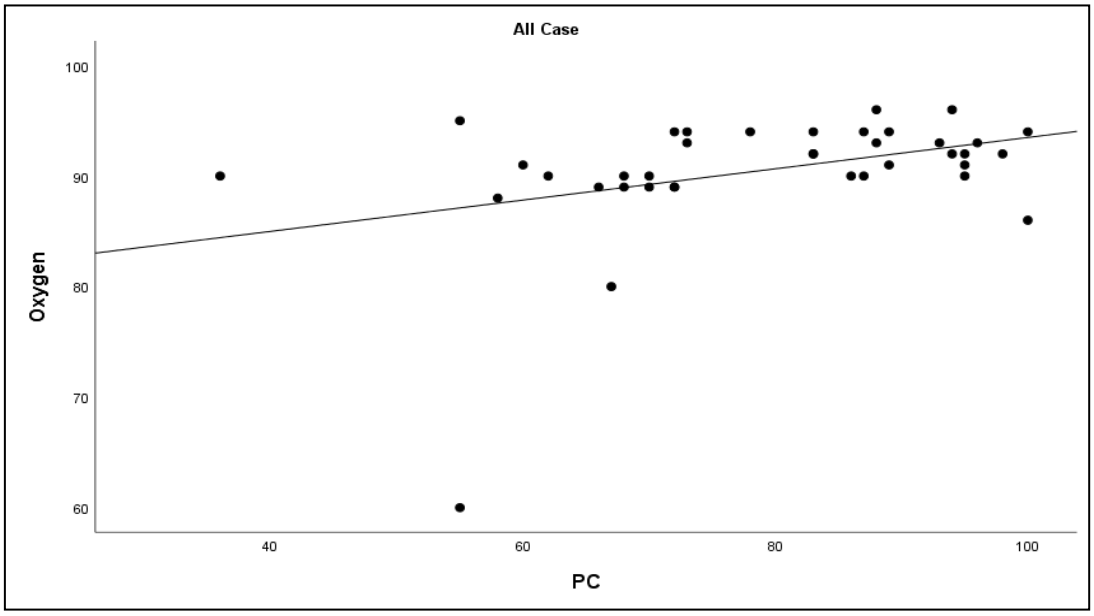

Figure (1): Positive correlation of oxygen saturation vs PC among COVID- 19 patients.

Correlations between coagulation parameters and different laboratory parameters in COVID-19 Patients:

In the present study, a significant positive correlation was observed between D-dimer levels and both LDH ($r = 0.660$, $p < 0.001$) and CRP ($r = 0.434$, $p = 0.021$), indicating that higher D-dimer levels were associated with increased inflammatory markers. Additionally, INR demonstrated a significant negative correlation with serum ferritin ($r = -0.416$, $p = 0.028$), **(figure 2), (Table 6).**

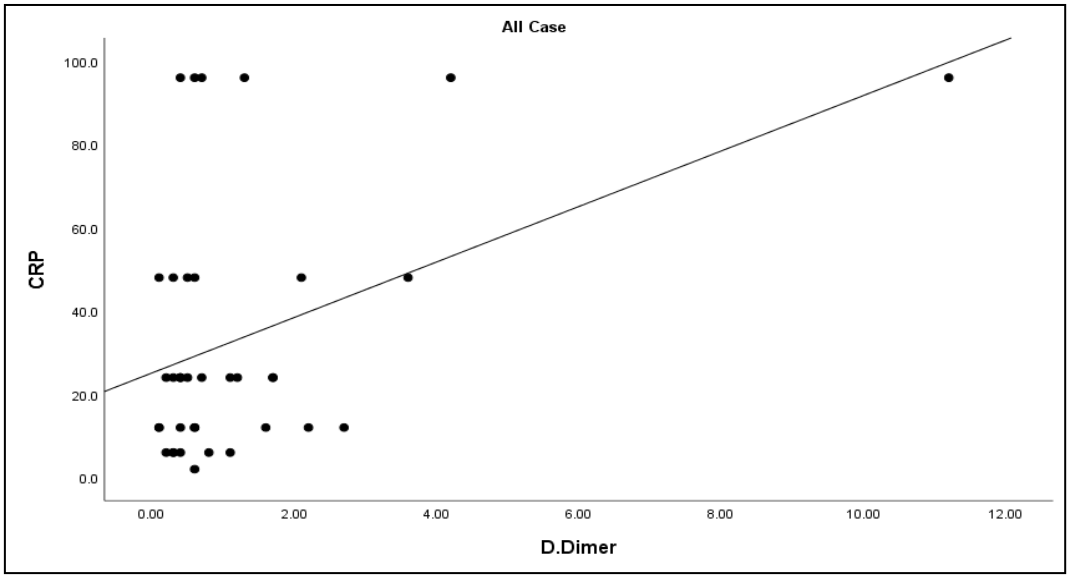

Figure (2): Positive correlation of CRP vs D.dimer among COVID 19 patients

Table (6): Correlations between coagulation parameters and different laboratory parameters in COVID-19 Patients:

Variable	PC		INR		APTT		D-dimer	
	r	P	R	P	r	P	R	P
LDH	0.146	0.458	-0.106	0.592	-0.047	0.812	**0.660**	**<0.001***
CRP	0.039	0.845	-0.040	0.839	-0.151	0.442	**0.434**	**0.021***
S.Ferritin	0.280	0.148	-0.270	0.165	**-0.416**	**0.028***	-0.003	0.990
Absolute lymphocytes	0.263	0.177	-0.223	0.253	-0.100	0.613	0.289	0.136
Absolute neutrophils	-0.155	0.431	0.247	0.206	0.037	0.852	0.142	0.469

Discussion

COVID-19 is a complex disease with diverse clinical symptoms, starting from mild symptoms to severe respiratory complications and multi-organ dysfunction. Understanding the clinical, inflammatory, and coagulation profiles of affected patients is critical for early risk stratification and effective management [12]. This study investigated a cohort of COVID-19 patients, comparing them with healthy controls, to explore differences in clinical features, inflammatory markers, and coagulation parameters, and to assess their inter-relationships.

The current study showed that both males and females are equally affected with COVID-19 infection, and this was in agreement with Sahu et al., [13] who showed that men and women showed similar probability of being infected by SARS-CoV-2, However Chen et al., [14] found men prevelance among COVID-19 infected subjects. In the current study the mean age of patient group was 58.2 years and this was in

agreement with Shahid et al., [15] who reported that patients aged over sixty and those with comorbidities were more vulnerable to severe disease and death.

This study also showed that the majority of our patients were diabetics (60.5%) and hypertensive (63.2%) and this was in agreement with Muniyappa and Gubbi [16] who showed that diabetic, hypertensive, and severe obesed (BMI $\geq$ 40 kg/m^2) patients are highly susceptible to COVID-19.

Moreover, fever, cough and dyspnea were present in 76.3 %, 68.4% and 71.1% of patients group respectively, Cascella et al., [17] stated that fever, cough, and shortness of breath are the most frequent symptoms found in their research. In this study, sore throat was found in 28.9% of patients group. This was supported by the study done by Weng et al., [18].

Regarding oxygen saturation, this study revealed that there was statistically significant decrease in saturation in COVID-19 patients when compared to control group and this was in agreement with Al-Hadrawi et al., [19] who demonstrated that lung injuries as detected by chest CT scans are linked to decreased oxygen saturation, which can intensify inflammation and may persist post-recovery. This study revealed marked elevation in serum urea and creatinine levels in the patient group compared to the control group (P < 0.001 for both), consistent with the findings of Temiz MZ et al.,[20] Several pathophysiological mechanisms may explain this renal impairment in COVID-19 patients., direct viral injury plays a role, as SARS-CoV-2 binds to ACE2 receptors, which are abundantly expressed in proximal tubular cells and podocytes, leading to acute tubular injury and glomerular damage. Secondly, the cytokine storm associated with COVID-19 involves an excessive release of inflammatory cytokines, such as IL-6 and TNF-α, which can contribute to multi-organ dysfunction, including acute kidney injury (AKI). Moreover COVID-19 patients in the current study showed highly significant increase in AST and ALT enzymes than the control group. This was in agreement with Saini et al., [21] study who had found that more than half of patients admitted to the hospital with SARS-CoV-2 infection had an abnormal liver function tests which was found to be associated with raised levels of inflammatory markers. In contrary with Dong et al., [22] who found that only a total of 3% of COVID-19 patients had liver disease.

Regarding LDH, this study found that its serum levels were significantly increased in the patient group compared to the control group. And this was in line with Several studies [23,24] mechanisms may explain this elevation in COVID-19 patients. Firstly, tissue damage due to viral cytopathic effects plays a key role, as SARS-CoV-2 can infect and damage various cell types, including lung epithelial and endothelial cells, leading to cell lysis and subsequent release of LDH. Secondly, pulmonary injury and hypoxia, commonly seen in COVID-19 pneumonia and acute respiratory distress syndrome (ARDS), result in alveolar damage and leakage of LDH into the circulation. Additionally, the cytokine storm and systemic inflammatory response characteristic of severe COVID-19 contributes to multi-organ damage, further increasing LDH levels due to release from injured tissues such as the liver, heart, kidneys, and skeletal muscles.

Moreover, CRP levels were significantly elevated in patients group than control group (P=< 0.001*). This was in line with Wang et al, [25] who identified a positive association between CRP concentrations and both the radiologic extent of lung involvement and clinical disease severity. This implies that early COVID-19-related lung damage and disease severity may be reflected by CRP levels [25]. In the present study, there was significant increase in serum level of ferritin when comparing patients group with control group, this finding was in aline with analysis that found elevated levels of serum ferritin in COVID-19 patients (Gómez-Pastor and Weigand, et al., [26], Severe COVID-19 can trigger a cytokine storm, characterized by marked production of pro-inflammatory cytokines (e.g., IL-6, IL-1β, TNF-α). IL-6 stimulates hepatocytes and macrophages to produce ferritin as part of the acute-phase response.

Regarding hemoglobin level, this study showed that there was statistically significant difference between the patients group and control group (p=0.010*), as Hb value was lower in patient group compared to control and this is in

agreement with Anai et al., [27] who reported decrease in Hb level during the short period in COVID-19 patients.

Regarding WBC's count, this study showed increased WBCs among patient groups compared to control group (p=0.009*) and this is in agreement with Hong et al.,[28] who reported in their study that leukocytosis was found among COVID-19 patients frequently, especially among the severe cases . While, It was in contrary with Huang et al.,[29] who suggested that COVID-19 infection resulted in a lowered white cell count.

As regarding absolute neutrophilic count in our work, we found that patients group showed marked elevation in absolute neutrophilic count compared to control group, which is consistent with studies reporting an increase in absolute neutrophil counts among COVID-19 patients[30]. Immune disturbance that occurs in COVID-19 leads to neutrophil production and lymphocyte apoptosis. Thus, neutrophilia occurs together with lymphopenia. Additionally, superimposed bacterial infection that could occur among COVID-19 patients may attribute to this neutrophilia [31].

Regarding absolute lymphocytic count, it was significantly lower in diseased group compared to control group and this was in accordance with Niu et al., [32] who showed that in patients infected with SARS-CoV-2, lymphopenia is a common clinical feature, Moreover, excessive production pro-inflammatory cytokines could directly suppress the proliferation of lymphocytes and induce early apoptosis.

Also in current study we found that INR values were significantly elevated in patient group when compared with control group and this is in agreement with some studies [33] that revealed that in COVID-19 patients, there were elevated INR values, that strongly suggest the presence of systemic coagulopathy and associated with both severe illness and increasing COVID-19 patients mortality. Also, this was in agreement with Araya et al., [34] who revealed that coagulation abnormalities are most frequent found in COVID-19 patients and are associated with poor prognosis and reduced survival. In this study, we found that aPTT was significantly longer in patients group when compared with healthy control group , and this was in agreement with the study that found that aPTT value was significantly longer in severely ill and non-surviving COVID-19 patients [35].

This study demonstrated higher D-dimer levels in COVID-19 patients compared to healthy controls, supporting existing evidence that elevated D-dimer is a common finding among individuals infected with SARS-CoV-2 [36].

Furthermore, it should be noted that increased D-dimer is a highly non- specific marker of venous thromboembolism and may indicate inflammation rather than thrombosis [37]. This study is further supported by the study that revealed that D-dimer levels are frequently higher in patients infected with corona virus [38].

In this study, we aimed to investigate the relationship between coagulation markers, oxygen saturation, and inflammatory markers in COVID-19 patients. D-dimer levels showed a significant positive correlation with both lactate dehydrogenase (LDH) and C-reactive protein (CRP), highlighting the close link between coagulation activation and systemic inflammation in the course of COVID-19. Similarly, a retrospective study by Sukrisman L.[39] conducted in Indonesia found a positive correlation between D-dimer and CRP levels in COVID-19 patients, further supporting this association. However, no correlation was observed between D-dimer and ferritin levels in that study. These findings suggest that the hyperinflammatory state induced by the immune response to COVID-19 contributes to the activation of coagulation pathways. Elevated D-dimer is widely recognized as a marker of thrombotic activity and poor prognosis, and its association with inflammatory markers reinforces the concept of COVID-19 as both a hyperinflammatory and hypercoagulable condition. These data suggest that concurrent monitoring of D-dimer and inflammatory markers may provide valuable insights into disease severity and progression. This study has several limitations.

The relatively small sample size of 38 COVID-19 patients and 16 controls may limit the generalizability of the findings to broader populations. The cross-sectional nature of the study, with measurements taken at only one time point, restricts the ability to observe changes in coagulation and inflammatory

markers over the course of the disease., future research should focus on larger, multicenter studies that include more diverse populations to enhance the external validity of the results. Longitudinal follow-up of patients is also recommended to track the dynamics of coagulation and inflammatory markers over time and their relationship with disease progression and outcomes. Moreover, investigating the therapeutic effects of anticoagulant and anti-inflammatory treatments on coagulation and inflammatory markers could inform more effective management strategies.

Conclusion

In conclusion, our study emphasises the significance of evaluating parameters of coagulation in COVID-19 infection. This study highlights the significant elevation of coagulation markers—particularly D-dimer—and their correlation with inflammatory parameters such as CRP and LDH in COVID-19 patients. These findings support the hypothesis that COVID-19-induced coagulopathy is closely linked to systemic inflammation. Early identification of these abnormalities may serve as prognostic indicators and guide clinical decisions regarding anticoagulation and supportive therapies.

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