Original Article

Hematological Markers and Coagulation Profiles in COVID-19 Patients: A Retrospective Cohort in Jeddah, Saudi Arabia

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Abstract

SARS-CoV-2 infection usually results in multi-organ affection, and severe inflammatory response which associated with significant affection of blood cell count and coagulation profile. We aimed to study the changes in haematological parameters among patients infected with SARS-CoV2 and its relation to the severity of infection. A retrospective cohort investigated data from patients hospitalized at the Armed Forces Hospital in Jeddah, Saudi Arabia, between July 2020 and July 2022. Patient who tested positive for SARS-CoV 2 infection were compared to negative patients.

A total of 586 patients were included, 387 tested positive for SARS-CoV-2. COVID-19 patients had significant lower leukocyte, and eosinophil counts along with lower red blood cell indices, suggesting a potential impact on the hematopoietic system. Deviation in leukocyte count was correlated with disease severity. the mortality was higher in the non-COVID-19 group (14.1 vs. 7.5%). COVID-19 patients had significantly higher fibrinogen levels and lower prothrombin time than non-COVID-19 patients. No difference was found regarding liver enzymes, renal function tests or acute-phase reactants. Haematological parameters could be used as markers for early detection, assessment of disease severity, and prognosis of SARS-CoV-2 infection. Further studies are necessary to understand the underlying mechanisms and their clinical significance.

Keywords: COVID-19, SARS-CoV-2, Hematological parameters, Hypercoagulable state, Lymphocytopenia, Eosinopenia

Introduction

China experienced an epidemic of pneumonia in December 2019 that was brought on by the SARS-CoV-2 virus [1]. The World Health Organization (WHO) officially designated the illness as coronavirus disease 2019 (COVID-19) in February [2]. In a short amount of time, the overall number of cases increased dramatically, reaching thousands. The latest coronavirus epidemic was deemed a worldwide pandemic by the WHO on March 11, 2020 [2]. The number of cases in Saudi Arabia reached 101,914 on June 7, 2020, an average of 1,039 new cases every day, after the first case was discovered on March 2, 2020 (trend test, $p < 0.000$) [3]. The COVID-19 daily infection rate peaked on June 17, 2020, the day when the nation had the highest infection rate of 4,919, at 14 infections per 100,000 people. By November 22, 2021, when the overall number of infected cases reached 549,518 (1,556 per 100,000 individuals), this proportion had drastically decreased and was practically nonexistent. However, nearly all of the recovered cases, 538,640 (1,526 per 100,000 persons), were still present [4].

SARS-CoV-2 infection results in multi-organ affection through many mechanisms. First, there is direct cellular invasion and toxicity [5]. Second, endothelial cell injury by direct infection results in release of thrombotic factors, inhibition of fibrinolysis, activation of complement factors and coagulation pathways. The net result is imbalance between pro and anti-coagulation and microthrombi deposition and microvascular dysfunction. Also, hypoxia induced hyper-viscosity and upregulation of hypoxia induced factor add to hypercoagulable state [6]. The third is resulting from the cytokine and chemokine release. The inflammatory cytokines, notably interleukin (IL)-1β, IL-6, IL-12, IL-18, IL-33, and TNFα, which are released in an uncontrollably high

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amount during SARS-CoV-2-induced pneumonia, are described as a "cytokine storm" [7]. Elevations of C-reactive protein, erythrocyte sedimentation rate, ferritin, D-dimer, fibrinogen, and lactate dehydrogenase have all been linked to severe COVID-19 disease [7].

People of all ages are vulnerable to contracting SARS-CoV-2. Clinical manifestations might range from moderate non-specific symptoms, such as fever, dry cough, and exhaustion, to invasive mechanical ventilation needed for acute respiratory distress syndrome (ARDS) [1, 8]. Acute heart damage, thrombotic episodes, diarrhea, and neurological disorders are examples of non-respiratory affection that have been documented [1, 9]. Mild to moderate symptoms account for 80% of cases [10]. Patients who come with severe and critical conditions are often elderly and have concomitant conditions such as diabetes, hypertension, cardiovascular disease (CVD), chronic lung illnesses, and chronic kidney disease (CKD) [1, 10]. Male gender, associated diabetes, hypertension, COPD increase risk of death two times [11]. Also, obesity, low socio-economic state, immune dysfunction are predictors of severe lethal disease [8].

Reverse transcription polymerase chain reaction (RT-PCR) is the gold standard method for detecting viral RNA to diagnose SARS-CoV2 infection; however, a large degree of variation in RNA viruses, improper sampling, and insufficient viral material in the specimen can result in reduced detection and false negative results [12]. As a result, SARS-Cov2 may be detected by RT-PCR, which has a high specificity but a sensitivity of 59%–71%) [13].

A number of blood indicators, including ferritin, D-dimer, IL-6, C-reactive protein (CRP), and lactate dehydrogenase (LDH), have increased after COVID-19 infection. Changes in laboratory values and hematologic abnormalities can be utilized for early diagnosis, and they were connected with the prognosis and severity of the disease, even if none were specifically for COVID-19 diagnosis [14, 15] and can help in risk stratification and early intervention [16].

LDH present in liver, muscles, lung, heart, and brain tissue. It is a non-specific marker of tissue damage and cellular death in COVID-19 infected patients [17]. Erythrocyte sedimentation rate (ESR) is also a non-specific inflammatory biomarker that correlate with the change in the size, shape of RBCs and concentration of plasma. CRP levels increased due to overproduction of inflammatory cytokines and by tissue destruction. CRP levels correlated with severity of infection and high immune response [18]. Increased liver enzyme levels in COVID-19 patients could result from direct hepatocellular injury, drug induced hepatotoxicity, or hepatic ischemia [19]. Hypoalbuminemia and high bilirubin was reported in 55% and 18% of hospitalized patients with COVID-19 infection [20, 21]. Acute kidney injury (AKI) was noted as one of the important complications of COVID-19 as a result from direct infection, indirect injury secondary to immune response or drug induced tubular dysfunction [22].

Complete blood count (CBC) is an easy, inexpensive routinely performed tool which can help in early diagnosis of the infection. While lymphopenia is the most common laboratory finding in COVID-19, it is also common to see other hematological abnormalities such as anemia, leucocytosis, neutrophilia, low eosinophil count or eosinophilia, thrombocytopenia, and infrequently thrombocytosis. Lymphopenia is found in 25–80% of patients at the time of presentation [23]. Anemia can result direct viral injury resulting in hemolysis, dysregulated iron metabolism, blood loss during different critical care interventions and excessive use of anticoagulation [24]. Lymphopeia can be caused by direct SARS-CoV-2 infection of lymphocytes because of the expression of ACE2 receptors on their surface. This can lead to cell lysis, lymphocyte apoptosis as a result of the cytokine storm, or decreased proliferation because of concurrent lactic acidosis [25]. Several studies have employed high neutrophil-to-lymphocyte ratios (NLR), neutrophil to CD4+ lymphocyte ratios (NCD4LR), and neutrophil count to albumin ratios (NAR) to predict COVID-19 infection [24]. Zhang *et al*. showed that almost half of the patients hospitalized with COVID-19 had eosinopenia (absolute counts <0.02 × 109 cells/L) [26]. Between 5% and 50% of individuals in various groups experienced thrombocytopenia. Theories include decreased bone marrow output, decreased megakaryocyte fragmentation and platelet generation as a result of COVID-19-induced lung and pulmonary capillary bed damage, or platelet consumption as a result of endothelial cell damage [14]. Coagulopathy associated with COVID-19 infection carries high risk of morbidity and mortality [27]. Disseminated intravascular coagulation (DIC) can be caused by fibrin deposition, systemic coagulation cascade activation, microvascular thrombi in different organs, and the consumption of clotting components and platelets, which can end in potentially fatal bleeding. Thirty-five percent of patients with COVID-19 infection had DIC, and ninety-five percent had thrombotic and bleeding problems, respectively. Prothrombin time (PT) activated partial thromboplastin time (APTT), D-dimer, and fibrin degradation products (FDP) are all extended in COVID-19-infected patients [26, 27]. The purpose of this study was to look at how the SARS-CoV2 infection affected several haematological markers and how that relationship related to the severity and pace of progression in hospitalized patients in the Saudi Arabian city of Jeddah.

Materials and Methods

Study Design and Participants

This study was a retrospective cohort that comprised all patients hospitalized between July 2020 and July 2022 at the Armed Forces Hospital in Jeddah, Saudi Arabia (all ages and both genders participating). Based on a positive PCR test result, the COVID-19 infection diagnosis.

Relevant variables were collected form medical records including:

1. Demographic and Clinical data: age, sex, nationality, weight, height, body mass index, symptoms and signs of the disease and duration of time from admission to discharge either living or dead.
2. Medical history of hypertension or diabetes.
3. Laboratory parameters: blood type; complete blood count (CBC), which counts red blood cells and their indices as mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), red cell distribution width (RDW), count of red blood cells (RBC), count of reticulocytes; count of white blood cells with differential count; count of platelets; and count of other platelets as mean platelet volume (MPV).
4. Hemostatic parameters, which are prothrombin time (PT), activated partial thromboplastin time (APTT), fibrinogen, and D -dimers.
5. Biochemical and inflammatory markers which are serum creatinine, LDH, liver enzymes, C-reactive protein and ferritin.

Outcomes

1. Compare the difference in clinical characteristics and the mean of hematological and biochemical parameters among patients infected with COVID-19 and COVID-19 negative patients including:
   - Blood cells count and morphology.
   - Coagulation profile
   - Iron stores
2. Correlate the hematological parameters with markers of disease severity in patients infected with COVID-19.

Statistical Analysis

Version 20.0 of the IBM SPSS software program was used to feed data into the computer and analyze it (Armonk, NY: IBM Corp.). When describing quantitative data that was not regularly distributed, the terms median and range were used, whereas mean ± SD was used for properly distributed data. The Kolmogorov-Smirnov test was employed to confirm that the distribution was normal. Descriptive statistics were used to analyze baseline attributes. Depending on how the data were distributed, the Student t-test, Mann-Whitney U test, or Kruskal-Wallis test were used to compare the groups' quantitative data. The relative risk ratio and the chi-square test were applied to the qualitative data. Correlation with Pearson was used to test for any association between variables.

Results and Discussion

Data of all patients admitted to Armed Forces Hospital in Jedda city, Saudi Arabia during the period from July 2020 to July 2022 (n=586) is represented in **Table 1**. Mean age was 48.12 ± 23 years. Males constituted 57.3% of the cohort. 513 patients (96.8.5%) were Saudi citizens and the remaining 19 (3.2%) were from other nationalities. Mean duration of admission was 10.5± 25.8 days. Mean Hb was 11.98 ± 2.41 gm/dl. Of all patients, 387 patients (66.1%) tested PCR-positive for SARS-CoV2 infection and 199 patients tested negative (33.9%).

Table 1. Baseline clinical characteristics of all patients

	Mean	Standard Deviation	Median	Minimum	Maximum
HGB	11.98	2.41	12.30	3.92	16.50
MCV	84.6	9.3	86.0	46.3	123.0
MCH	27.7	3.1	28.0	15.1	42.4
RDW	14.51	2.72	13.90	2.45	34.30
RBC	4.41	.89	4.47	1.42	7.22
WBC	8.17	6.19	7.08	1.57	109.00
Neut %	59.68	17.53	60.90	2.55	97.50
Lymph %	26.67	14.18	25.00	1.62	89.40
Mono %	9.914	4.424	9.240	.000	36.600
Eos %	1.926	2.316	1.180	.008	14.900
Baso %	.312	.446	.138	.000	5.000
PLT	263.7	126.6	240.0	11.0	879.0
MPV	8.40	1.39	8.34	2.27	14.50
PT	15.2	3.4	14.3	10.0	41.3
APTT	39.00	8.90	37.10	25.10	90.50
Fibrinogen	414.0	218.7	372.1	112.5	1200.0
D-Dimer	2.18	3.62	1.08	.10	21.00
Creatinine	97.11	75.89	71.00	21.00	586.00
LDH	287.3	171.1	233.0	49.0	1856.0
SGOT/ALT	47.5	379.3	19.0	5.0	8516.0

Ferritin	276.0	304.1	174.5	2.5	1642.4
Duration of admitted	10	18	4	1	202

Table 2 shows the variations in clinical features between COVD-19 individuals and non-COVID patients. Males made up 113 (56.8%) of the patients who were not infected with COVID-19, compared to 223 (56.86%) who were. 96.6% of non-COVID-19 patients and 97.0% of COVID-19 patients were Saudi nationals. When comparing COVID-19 patients (10.1%) to non-COVID-19 patients (31.7%), hypertension was more common. Compared to 35.4% of non-COVID-19 patients, 33.3% of COVID-19 patients had diabetic millets. The non-COVID-19 group had a greater mortality rate (14.1 vs. 7.5%, respectively) than the COVID-19 group.

Table 2. Comparison between COVID-19 patients and non-COVID-19 patients in baseline characteristics

		Non COVID-19 patients		COVID-19 patients	
		Count	Column N %	Count	Column N %
Sex	Female	86	43.2%	164	42.4%
	Male	113	56.8%	223	57.6%
Nationality	Saudi	193	97.0%	374	96.6%
	Non-Saudi	6	3.0%	13	3.4%
Blood Group and RH	O+	94	52.2%	139	50.7%
	A+	49	27.2%	70	25.5%
	B+	19	10.6%	37	13.5%
	AB+	8	4.4%	11	4.0%
	O-	2	1.1%	5	1.8%
	A-	6	3.3%	9	3.3%
	B-	2	1.1%	2	0.7%
	AB-	0	0.0%	1	0.4%
History of Hypertension	No	136	68.3%	348	89.9%
	Yes	63	31.7%	39	10.1%
History of Diabetes	No	128	64.6%	258	66.7%
	Yes	70	35.4%	129	33.3%

Laboratory parameters were examined between COVID-19 patients and non-COVID-19 patients. The mean RBC count was substantially greater in COVID-19-positive patients compared to the negative group, although the mean indices of MCV, MCH, and RDW were significantly lower. The mean Hb level, on the other hand, did not alter much. The overall leucocytic count was much lower in the COVID-19 group. The average indices showed a substantial decrease in eosinophil and basophil counts and an increase in monocyte counts; however, there was no significant difference in the mean counts of neutrophils and lymphocytes. the mean platelet count showed a non-significant difference. Regarding coagulation profile, COVID-19 patients had significantly higher fibrinogen levels and lower prothrombin time than non-COVID-19 patients. Tests for liver enzymes and renal function did not significantly differ between the two groups. Patients with COVID-19 have much reduced levels of the enzyme lactate dehydrogenase (LDH). The levels of acute-phase reactants, such as ferritin and C-reactive protein (CRP), did not change significantly. The length of hospital stay was not significantly different for either group **(Table 3)**.

Table 3. Comparison between COVID-19 patients and non-COVID-19 patients regarding laboratory parameters

	Non COVID-19 patients		COVID-19 patients	
	Mean	Standard Deviation	Mean	Standard Deviation
HGB	11.45a	2.51	12.25b	2.31
MCV	85.7a	9.5	84.1b	9.1
MCH	28.3a	3.0	27.4b	3.1
RDW	15.00a	2.75	14.26b	2.67
RBC	4.13a	.97	4.55b	.81

WBC	9.53a	4.43	7.47b	6.82
Neut %	60.76a	18.08	59.14a	17.26
Lymph %	25.99a	14.94	27.00a	13.80
Mono %	9.350a	4.102	10.194b	4.554
Eos %	2.412a	2.561	1.685b	2.147
Baso %	.418a	.451	.259b	.434
PLT	261.7a	117.5	264.7a	131.2
MPV	8.47a	1.45	8.36a	1.35
PT	15.9a	3.9	14.8b	3.1
APTT	39.38a	9.15	38.80a	8.79
Fibrinogen	354.0a	164.0	441.1a	236.8
D-Dimer	1.78a	.90	2.24a	3.87
Creatinine	94.61a	64.76	98.37a	81.01
LDH	332.2a	244.5	272.4b	135.7
SGOT/ALT	95.1a	700.9	28.0a	33.1
C reactive protein	73.7a	86.5	51.7b	81.0
Ferritin	90.2a	61.5	307.7b	317.4
Duration of admitted	13a	22	9b	15

Note: The two-sided test of equality for column means shows a significant difference at p<.05 for values in the same row and subtable that do not share the same subscript. Subscript-free cells are excluded from the test. Equivalent variances are assumed in tests.

D-dimer level had a positive correlation with red cell distribution width (RDW) and prothrombin time (PT) and was negatively correlated with red blood cell count (RBC). C-reactive protein (CRP) level showed a positive correlation with RDW, WBC and PT and was negatively correlated with RBC **(Table 4)**.

Table 4. Correlation between CRP and D-Dimer and haematological parameters in patients infected with COVID-19

		HGB	MCV	MCH	RDW	RBC	WBC	PT	APTT	Fibrinogen
D-Dimer	Pearson Correlation	-.038	.016	-.015	.303**	-.269**	.086	.258**	.125	.252
	Sig. (2-tailed)	.564	.810	.816	.000	.000	.193	.000	.088	.196
	N	229	229	229	229	229	229	223	187	28
C reactive protein	Pearson Correlation	.025	.000	-.035	.142**	-.229**	.323**	.195**	.097	-.320
	Sig. (2-tailed)	.631	.994	.501	.007	.000	.000	.000	.118	.079
	N	366	366	366	366	366	366	332	262	31

*. Correlation is significant at the 0.05 level (2-tailed).
**. Correlation is significant at the 0.01 level (2-tailed).

SARS-CoV2-caused COVID-19 pandemic struck the world in December 2019, with severe cases leading to acute respiratory failure and pneumonia. Within two years, the number of infected patients exceeded 400 million [28]. There have been reports of non-respiratory affection, including thrombotic episodes, acute heart damage, gastrointestinal (GI) signs (diarrhea, abdominal discomfort), and neurological diseases [28]. The majority of COVID-19 patients had lymphocytopenia and leucocytes that are either normal or reduced, according to preliminary blood studies [9]. Additionally, there was often an increase in inflammatory cytokines such as TNF-α, IL-10, and IL-6 [29]. Haematological changes associated with SARS-CoV2 infection can help to understand pathophysiological mechanisms, and could provide early clues for identification of infection, evaluation of severity and expect prognosis of infected patients. few studies have investigated haematological parameters associated with COVID-19 infection in Saudi Arabia. In this context, this retrospective study compared the haematological parameters including CBC, coagulation profile, acute phase reactants, liver and kidney functions between COVID-19 patients and non-COVID-19 patients who were admitted to Armed Force hospital in Jedda city, Saudi Arabia during July 2020 to July 2022 This included a total of 586 patients, of which 387 were COVID-19 infected patients.

COVID-19 infection was responsible for two-third of the Armed Force hospital admissions during the period of the study (66.1%). However, the mortality was higher in non-

COVID-19 group (14.1% vs 7.5%). During the COVID-19 pandemic, fewer patients were admitted for common emergencies like heart attacks, strokes, and asthma flare-ups [30] although this may not necessarily indicate a decrease in incidence; instead, it could be the result of fewer people visiting hospitals out of concern for coming into contact with COVID-19-infected clients. In many US areas, the number of emergency visits dropped by 40%, and during the early pandemic phase, the percentage of visits connected to infectious diseases was four times greater [31]. According to a Centers for Disease Control and Prevention (CDC) report, there were approximately 300,000 excess deaths in the United States between January 26 and October 3, 2020. Up to 60% of these deaths were directly related to COVID-19, with the remaining deaths possibly being caused by delayed medical attention or the worsening of preexisting chronic conditions [32].

In this cohort, the COVID-19 group had significantly lower total leucocytic count (TLC). The blood film analysis revealed lower eosinophil and basophil counts. SARS-CoV2 infection is associated with alteration in TLC [9, 28]. The extent of deviation in TLC correlated with disease severity in many studies. Higher WBC and neutrophilia were found in patients with severe presentations and ICU patients rather than patients with mild presentations and most patients exhibit lymphocytopenia [9, 29]. While the majority of the patients were younger and had modest clinical signs, another investigation found that 68% of infected individuals had normal WBC and 42% had lymphocytopenia. In several additional viral infections, lymphocytopenia is noted as a result of direct infection, cytokine production, and glucocorticoid therapy [33]. A group of 140 hospitalized COVID-19 patients likewise showed 52.9% eosinopenia, and following hospital admission, eosinophil levels significantly linked with lymphocyte counts in both severe (r =.486, P <.001) and non-severe (r =.469, P <.001) patients [34].

The mean indices of MCV, MCH, and RDW were all considerably lower in COVID-19 individuals, although the mean RBC count was significantly greater. In this cohort, there was no significant change in the blood type or platelet count. A low hemoglobin level and thrombocytopenia were observed by several authors in severe instances, despite the majority of publications [9, 29, 35] showing that COVID-19 infection did not alter hemoglobin level, RBC, or platelet count [36]. Individuals with COVID-19 had considerably lower levels of Hb concentration, MCH, MCHC, and MCV compared to negative individuals, according to another retrospective cohort from King Khalid Hospital in Najran, Saudi Arabia [37].

Severe SARS-CoV2 infection associated with thrombotic events and hypercoagulable state which results from direct endothelial cell injury and release of thrombotic factors, inhibition of fibrinolysis, activation of complement factors and coagulation pathways. Also, hypoxia induced hyper-viscosity and upregulation of hypoxia induced factor add to the hypercoagulable state [6]. In comparison to non-COVID-19 patients, COVID-19 patients exhibited considerably reduced prothrombin times and greater fibrinogen levels, according to the current study. According to a meta-analysis, the incidence of deep vein thrombosis (DVT) was 27% in 32 studies (n = 2552) and the prevalence of pulmonary embolism (PE) was 32% in 17 studies (n = 3973). The meta-analysis focused on venous thromboembolic events (VTE) in individuals infected with COVID-19. greater mean D-dimer readings and a greater incidence were seen in ICU patients [38]. When fibrin is crosslinked (by factor XIII), D-dimer is the resultant breakdown product. It represents continued fibrinolysis and coagulation. Numerous investigations found that in COVID-19-infected individuals, elevated levels of D-dimer and fibrinogen as well as extended PT were linked to severe infection and a poor outcome. Guidelines from the Italian Society on Thrombosis and Haemostasis (SISET) [39] and the International Society on Thrombosis and Haemostasis (ISTH) [40] suggested that D-dimer levels, PT, and platelet counts be the main parameters of observation for COVID-19 patients.

Lactate dehydrogenase (LDH) is an intracellular enzyme responsible for energy production [41]. The high plasma LDH level indicate extensive tissue and cell destruction associated with many diseases such as liver disease, lymphoproliferative disorders, and interstitial lung disease [41]. In SARS-CoV2 infected patients high LDH level indicated severe viral infection or lung damage [42]. A mortality prediction model developed by Yan *et al*. including more than 70 features of 485 COVID-19 infected patients, found that LDH, lymphocytes and hs-CRP together has 90% accuracy in mortality prediction [43]. Many studies used LDH and acute phase reactants such as CRP and ferritin as potential predictors of diseases severity and pulmonary function [42, 44]. In contrast, ferritin and CRP levels did not substantially differ between COVID-19 and non-COVID-19 individuals in this research, while LDH levels were considerably lower in COVID-19 patients. Given the retrospective nature of the study and the lack of clinical data for both groups, it is challenging to interpret these results; nevertheless, since the COVID-19 group had a lower death rate, we may anticipate that the majority of hospitalized COVID-19 patients had mild to moderate presentations.

A retrospective cohort form Iran included 320 confirmed COVID-19 patients observed a significant difference in D-dimer, WBCS, PMN, Lymph, monocytes, eosinophil, and RDW between the patients with poor outcome and good outcome (P<0.001), D-dimer levels showed a significant association with WBCs, PMN, and RDW (P<0.05) [45].

As described, several studies have shown that haematological parameters could be valuable and feasible markers of predicting disease severity and progression in daily clinical practice. High Neutrophil to lymphocyte ratio (NLR), low Lymphocyte to C–reactive protein ratio (LCR) [46, 47], lymphopenia [46, 48] and high D–dimer levels [49] are all

associated with severe cases and of a great prognostic value. Two studies reported that patients with blood group A were more prone to infection, however, the mechanisms responsible for this effect are unknown [50].

Strength and Limitations

The limitation of this study is the lack of data about clinical presentation, severity, and outcome of COVID-19 patients, and the nature of the clinical illness of non-COVID-19 patients which could affect the haematological findings. However, to our knowledge this is the largest cohort in Saudi Arabia describing the haematological parameters of hospital admitted COVID-19 patients over two years since the pandemic start.

Conclusion

In conclusion, this retrospective study compared the haematological parameters of COVID-19 patients and non-COVID-19 patients in Saudi Arabia. COVID-19 patients exhibited decreased leucocyte count, lymphocytopenia, eosinopenia, and altered red blood cell indices. They also had higher fibrinogen levels and shorter prothrombin time, indicating a hypercoagulable state. Haematological markers such as D-dimer, LDH, and acute phase reactants showed potential for predicting disease severity. Monitoring these parameters can aid in early identification, assessing disease severity, and predicting prognosis in COVID-19 patients. Further research is needed to understand the underlying mechanisms and clinical implications of these haematological changes.

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