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Coagulation parameters and prognosis in COVID-19 patients

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ABSTRACT

Aims: There are many factors that affect morbidity and mortality in COVID-19. Coagulopathy is thought to be one of the important mechanisms in COVID-19 mortality. In this study, it was aimed to investigate coagulation factors and their relationship with prognosis in COVID-19.

Methods: Patients diagnosed with COVID-19 were retrospectively reviewed in our hospital. The patients' demographic data, laboratory data on admission to the hospital, intensive care admissions, and surveillance were recorded. Patients were divided into two groups; Group 1 is non-critical patients followed up in the clinic, Group 2 is critical patients who need treatment in the intensive care unit.

Results: 403 patients followed up for COVID-19 were analyzed. It was determined that the average age of the patients in Group 1 was statistically significantly lower than those in Group 2. (1: 45.28±15.31 vs. 2: 60.15±15.72, respectively, $p<0.001$). It was observed that the rates of HT, DM, CHD, and COPD in Group 2 were statistically significantly higher than Group 1 ($p <0.05$). While APTT values were similar in both groups, D-Dimer values were significantly higher in Group 2. As QSOFA, SIC, DIC, and coagulation marker scores increased, the percentage of patients with death increased significantly ($p<0.05$). Age, HT, COPD, PT, and high fibrinogen levels were found to increase the mortality risk rates statistically ($p<0.05$).

Conclusion: It was determined that the most important factors determining mortality in COVID-19 are COPD and HT. APTT and D-dimer values were not found to be a prognostic factor in terms of mortality. However, PT, fibrinogen, and age are poor prognostic factors and can be used to predict mortality requiring intensive care.

Keywords: COVID-19, coagulopathy, prognosis, mortality

INTRODUCTION

Human coronavirus is a pathogen of the respiratory system that causes mostly mild upper respiratory tract infection. The viral epidemic called COVID-19 caused by SARS-CoV-2, the new coronavirus of zoonotic origin that started in China, in 2019, spread rapidly worldwide with its wide range of clinical symptoms, from asymptomatic to death. has resulted in millions of cases worldwide.[1]

In COVID-19 infection, leukopenia, lymphopenia, neutrophilia, hypoalbuminemia, hyponatremia can often be seen, while increase in levels of biochemical parameters such as D-dimer, ferritin, creatine kinase, troponin, lactic dehydrogenase (LDH), C-reactive protein (CRP), myoglobin.[2-5] The erythrocyte and platelet count usually maintains normal levels until the later stages of the disease. The procalcitonin level is typically normal in most patients.[3-6,7] It has been reported that the overall mortality rate varies in age groups between 2.3% and 12.8%, and the development of coagulopathy increases mortality.[7-9]

There is considerable variability in the extent of the effects of COVID-19 on coagulation parameters and their correlation with disease severity and mortality. Coagulopathy induced by COVID-19 (CIC) causes morbidity and mortality different from sepsis-induced coagulopathy (SIC).[2,5] While intravascular coagulation (DIC) is more common with bleeding in SIC, micro and macro vascular thrombosis and latent DIC frequently occur with CIC, and anticoagulation plays a major role in treatment.[4,5,10] In this study, we tried to reveal the effect of SARS-CoV-2 infection on coagulation parameters, the severity of COVID-19, disease progression, and mortality in patients we followed up.

METHODS

The study was approved by the Keçiören Training and Research Hospital Clinical Researches Ethics Committee (Date: 08/07/2020 Decision No: 2135). All procedures were carried

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out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

Following the approval of the local ethics committee, patients who received inpatient treatment between March 2020 and June 2020 at Keçiören Training and Research Hospital, which has been serving as a pandemic hospital since the outbreak began, and the diagnosis of COVID-19 was confirmed by RT-PCR test, were retrospectively screened. Patients with negative RT-PCR tests and diagnosed with COVID-19 due to clinical findings and computed tomography findings were excluded from the study. Demographic data of the patients, laboratory values at hospital admission, intensive care admissions, and surveillance were recorded. Patients were divided into two groups and analyzed; Group 1 is non-critical patients followed up in the clinic (no need for intensive care). Group 2 is critical patients who need treatment in the intensive care unit.

Statistical Analysis

Continuous variables were expressed as mean ± standard deviation and categorical data as numbers and percentages. In the intergroup analysis of continuous variables, normality analyzes were performed using the Kolmogorov-Smirnov Goodness of Fit Test. T-Test was used for the intergroup analysis of data that conformed to the normal distribution, and Mann Whitney U test was used for the analysis of non-compliant data. Comparisons of categorical data were made with the Chi-Square Test (Fisher's Exact Test when necessary). Risk factors and odds ratio values for intensive care need due to COVID-19 were determined by Binary Logistic Regression Analysis. Analyzes were done with IBM SPSS (Statistics Package Program for Social Sciences) version 24.0 (IBM Corporation, Armonk, NY, USA). Statistical significance level was taken as $p<0.05$.

RESULTS

While the average age of 403 patients who were followed up at the pandemic hospital due to COVID-19 was 46.50±15.86 years, a statistically significant difference was found between the groups (60.15±15.72 for Group 2, 45.28±15.31 for Group 1; $p<0.001$). The body mass index (BMI) values of the patients in Group 2 (29.98±5.55 vs 27.60±5.20) and the length of hospital stay (18.88±16.47 vs. 8.99±3.47, $p=0.02$) were statistically significantly higher compared to the patients in Group 1. It was determined that 55.9% of the patients in group 1 were female, whereas 69.7% of the patients in group 2 in need of intensive care were male ($p=0.005$). No significant difference was found in terms of smoking and pregnancy ($p=0.614$, $p=1.000$, respectively) (Table 1).

The rates of hypertension (HT), diabetes mellitus (DM), chronic heart disease (CHD), and chronic obstructive pulmonary disease (COPD) (45.5%, 27.3%, 21.2%, and 27.3%, respectively) in patients in Group 2 were compared to patients in Group 1 (23.4%, 13.0%, 6.4%, and 5.7% respectively) were found to be statistically significantly higher ($p<0.05$). There was no significant difference in terms of chronic kidney disease (CKD) rates ($p=0.216$). The rates of 'severe' and 'critical' pneumonia in group 2 patients (42.4% and 45.5%, respectively) were found statistically significantly higher than Group 1 patients (4.2% and 0.6%, respectively) ($p<0.001$ respectively) at hospital admission. While all of Group 1 patients (100.0%) were discharged with recovery, 27.3% of Group 2 patients died($p<0.001$), while the COVID-19 fatality rate was found to be 27.3% in intensive care patients (Table 2).

Table 1. Comparison of groups in terms of some socio-demographic and clinical characteristics

	Group 1 (Clinic Patients) (n=370)	Group 2 (ICU Patients) (n=33)	Total (n=403)	p
Age (year) (Avg±Ss)	45.28±15.31	**60.15±15.72**	46.50±15.86	**0.013***
BMI (kg/m²) (Avg±Ss)	27.60±5.20	**29.98±5.55**	27.79±5.26	**0.020****
Length of stay in the hospital (day)	8.99±3.47	**18.88±16.47**	9.90±6.59	**0.002****
Smoking (n, %)				
No	307 (85.0%)	27 (81.8%)	373 (84.8%)	0.614***[a]
Yes	54 (15.0%)	6 (18.2%)	4 (15.2%)	
Pregnancy (n, %)				
No	340 (98.8%)	33 (100.0%)	334 (98.9%)	1.000***[a]
Yes	4 (1.2%)	0 (0.0%)	60 (1.1%)	
Gender (n, %)				
Female	**207 (55.9%)**	10 (30.3%)	217 (53.8%)	**0.005*****
Male	163 (44.1%)	**23 (69.7%)**	186 (46.2%)	
Total	370 (100.0)	33 (100.0)	403 (100.0)	

* T Test ** Mann whitney U test *** Chi-square test (aFisher's exact test)

In intensive care patients in Group 2, the median values of CRP, alanine aminotransferase (ALT), aspartate aminotransferase (AST), prothrombin time (PT), lactic dehydrogenase (LDH), fibrinogen, D-dimer, ferritin, creatinine, troponin, sedimentation, and urea were statistically significantly higher than in Group 1 patients ($p<0.05$), platelet values were found to be significantly lower ($p<0.05$) (Table 3).

In our study, while 3 or more coagulation markers were not observed in any "living" patient, they were found in 25% of the patients with "ex". Three or more SIC criteria were found to be 1.5% in survivors, 62.5% in those who were dead, and DIC criteria were found in 1.5% in surviving patients and 25% in patients who are ex. It was found that as the QSOFA, SIC, DIC, and Coagulation marker scores increased, the percentage of patients who died significantly increased (Table 4).

According to univariate logistic regression analysis for risk factors for mortality due to COVID-19; Age, HT, COPD, PT, and high fibrinogen levels were found to increase the mortality risk rates statistically ($p<0.05$). It was found that a 1 unit increase in age increased the need for intensive care by 1.13 times (OR=1.130, 95% CI=1.068-1.197). It was determined that the most important factors increasing the mortality risk were the presence of COPD (OR=18.385, 95% CI=4.633-72.953) and the presence of HT (OR=3.832, 95% CI=1.008-14.576). Age, PT, and fibrinogen levels were found to increase mortality 1-1.1 times (Table 5). D-dimer levels were not detected as a risk factor for mortality (OR=1.001, 95% CI=1.000-1.001).

Table 2. Comparison of groups according to comorbid disease rates

	Group 1 (Clinic Patients) (n=353)	Group 2 (ICU Patients) (n=33)	Total (n=403)	p
Hypertension (n, %)				
No	265 (76.6%)	18 (54.5%)	283 (74.7%)	**0.010*[a]**
Yes	81 (23.4%)	**15 (45.5%)**	96 (25.3%)	
DM (n, %)				
No	301 (87.0%)	24 (72.7%)	325 (85.8%)	**0.035*[a]**
Yes	45 (13.0%)	**9 (27.3%)**	54 (14.2%)	
CHD (n, %)				
No	322 (93.6%)	26 (78.8%)	348 (92.3%)	**0.008*[a]**
Yes	22 (6.4%)	**7 (21.2%)**	29 (7.7%)	
CKD (n, %)				
No	335 (97.7%)	31 (93.9%)	366 (97.3%)	0.216*[a]
Yes	8 (2.3%)	2 (6.1%)	10 (2.7%)	
COPD (n, %)				
No	333 (94.3%)	24 (72.7%)	357 (92.5%)	**<0.001*[a]**
Yes	20 (5.7%)	**9 (27.3%)**	29 (7.5%)	
Total	353 (100.0)	33 (100.0)	386 (100.0)	
Presence of pneumonia (n, %)				
No	**114 (34.23%)**	2 (6.1%)	116 (31.7%)	**<0.001***
Mild	**203 (61.0%)**	2 (6.1%)	205 (56.0%)	
Severe	14 (4.2%)	**14 (42.4%)**	28 (7.7%)	
Critical	2 (0.6%)	**15 (45.5%)**	17 (4.6%)	
Treatment Result (n, %)				
Discharged with healing	**370 (100.0%)**	19 (57.6%)	389 (96.5%)	**<0.001***
Exitus	0 (0.0%)	**9 (27.3%)**	9 (2.2%)	
Referral to another hospital (Unknown outcome	0 (0.0%)	**5 (15.2%)**	5 (1.2%)	
Total	370 (100.0)	33 (100.0)	403 (100.0)	

* Chi-Square Test (aFisher's Exact Test)

Table 3. Comparison between and within groups of COVID-19 cases followed in intensive care and ward in terms of cytokine levels

	Group 1 (Clinic Patients) (n=370)	Group 2 (ICU Patients) (n=33)	p
	Median (min-max)	**Median (min-max)**	
HGB	13.8 (5.2-18.1)	14.1 (10.1-16.9)	0.433*
WBC	5400 (2300-20800)	5600 (3700-26700)	0.211*
Lymphocyte	1490 (117-28400)	1120 (370-22800)	0.314*
Platelet	208000 (15000-616000)	**166500 (25000-391000)**	**0.011***
CRP	6.3 (0.2-218.8)	**51.02 (5.98-508)**	**<0.001***
ALT	21 (6-400)	24 (3-118)	0.139*
AST	23 (1-1020)	**33 (15-101)**	**<0.001***
PT	10.5 (0.94-28.7)	**11.6 (1.05-16.9)**	**0.001***
aPTT	24.8 (0.8-38.7)	25 (1.2-36.7)	0.780*
LDH	199 (17-520)	**328.5 (122-1523)**	**<0.001***
Fibrinogen	318 (54-683)	**439 (85-696)**	**0.001***
D-dimer	370 (0-4800)	**520 (40-3890)**	**0.004***
Ferritin	66.8 (1-1486)	**160.5 (10-975)**	**0.010***
Creatinine	0.85 (0.53-13.7)	**1 (0.6-11)**	**0.009***
Troponin	2.72 (0.7-198.9)	**8.11 (2.5-72.1)**	**<0.001***
Sedimentation	24 (1-107)	**47 (9-80)**	**<0.001***
Urea	27.8 (3-74)	**34.2 (7-188)**	**0.002***

* Mann whitney U test

Table 4. Comparison of Qsofa and coagulation markers in deceased and surviving patients

	Living Patients (n=133)	Ex Patients (n=8)	Total (n=141)	p
Qsofa (n, %)				
0	104 (78.2%)	2 (25.0%)	106 (75.2%)	**<0.001*[a]**
1	27 (20.3%)	2 (25.0%)	29 (20.6%)	
2	2 (1.5%)	3 (37.5%)	5 (3.5%)	
3	0 (0.0%)	1 (12.5%)	1 (0.7%)	
SIC (n, %)				
0-2			134 (98%)	
3-5	131 (98.5%)	3 (37.5%)	12 (8.5%)	
	2 (1.5%)	5 (62.5%)		**<0.001*[a]**
DIC (n, %)				
0-2	131 (98.4%)	6 (75.0%)	110 (78%)	**0.001*[a]**
3-4	2 (1.5%)	2 (25%)	4 (2.8%)	
Coagulation markers (n, %)				
0-2	133 (100%)	6 (75%)	139 (98.6%)	**<0.001*[a]**
3-5	0 (0%)	2 (25%)	2 (1.4%)	
Total	133 (100.0)	8 (100.0)	144 (100.0)	

* Chi-square test (aLinear-by-Linear Association)

Table 5. Univariate logistic regression analysis for risk factors for the need for intensive care due to COVID-19

Risk Factors	B	SE	OR (Exp β)	95% CI	p
Age	0.123	0.029	**1.130**	**1.068-1.197**	**<0.001***
HT					
No	1.343	0.682	1.0		**0.049***
Yes			**3.832**	**1.008-14.576**	
COPD					
No	2.912	0.703	1.0		**<0.001***
Yes			**18.385**	**4.633-72.953**	
PT	0.152	0.064	**1.164**	**1.027-1.320**	**0.018***
Fibrinogen	0.07	0.03	**1.007**	**1.001-1.013**	**0.023***
D-dimer	0.001	0.00	**1.001**	**1.000-1.001**	**0.020***

*Binary logistic regression test (Enter method)
**OR=Odds ratio, CI=Confidence interval, SE=Standard error

DISCUSSION

As a result of our study, it was observed that the majority of critically ill patients in need of intensive care were older, had higher BMIs, and had additional systemic diseases such as HT, DM, and COPD. When laboratory tests were examined, it was found that platelet values were lower, D-Dimer, fibrinogen, PT, LDH, and CRP values were higher in patients requiring intensive care. It was seen that the most important factors determining mortality were the presence of COPD and HT. PT, fibrinogen, and increased age were found to be poor prognostic factors.

Coagulation parameters can be affected at different levels in the COVID-19 clinic, depending on the severity of the disease. Among these parameters, D-dimer, fibrinogen, and fibrin split products (FSP) increase significantly, while PT, aPTT may be normal or slightly prolonged. Although thrombocytopenia is uncommon, it appears in the later days of the disease in parallel with the severity of the disease.[11]

In their study in which Tang et al.[10] associated coagulopathy with poor prognosis in the first months of the pandemic, they found that D-dimer and fibrinogen, as well as prolonged PT, aPTT values, and thrombocytopenia were observed in COVID-19 patients at the first admission. In subsequent studies, it was emphasized that the platelet counts were higher than other coronavirus infections.[12] During the follow-up in a series of 50 patients in an ICU in Ireland, high D-dimer and fibrinogen values, platelet counts, and PT were found to be normal.[13] Since the disease is not recognized, the implications of Tang are thought to be due to the patients being admitted to the hospital with a more serious clinic and late.

D-dimer, one of the coagulation parameters, is the degradation product of cross-linked fibrin showing increased thrombin formation and fibrin melting by plasmin.[14] However, increased D-dimer levels are also common in acutely ill individuals with a range of infectious and inflammatory diseases. COVID-19 related D-dimer has been reported to increase at the level of 0.9 mg /L in 36% of cases.[6,7] In critically ill patients, D-dimer levels are frequently higher compared to cases with a mild clinical course and are inversely proportional to survival.[4,6,14] Zhou et al.[6,7] showed that 24% of living patients in their study had D-dimer above 1 mg/L, whereas 81% of mortal cases had D-dimer higher than 1 mg/L and increased gradually. Similarly, Tang et al.[10] showed that the D-dimer was higher than 3 mg/L in more than 85% of COVID-19 cases with death. In addition, although the threshold value of D-Dimer measurements in showing coagulation cannot be determined exactly, studies have recognized that D-Dimer measurements are also valuable in identifying individuals who could potentially benefit from anticoagulation treatment.[5,10,14] In our study, as a result of univariate logistic regression analysis, the D-Dimer value was not evaluated as a prognostic factor predicting intensive care admission. However, in the first admission tests, D-dimer values were found to be statistically significantly higher in critical patients who required ICU admission compared to patients followed in the clinic.

Fibrinogen is the most specific test for DIC diagnosis (100%), but its sensitivity is low (22%).[5,15] In the International Society of Thrombosis and Hemostasis (ISTH) guidelines, recommended as one of the overt DIC diagnostic parameters.[5,16,17] Fibrinogen is often elevated in patients with sepsis but may be low in severe cases of DIC. Fibrinogen levels are monitored at 4.55 g/L and above in most COVID-19 patients. The degree of fibrinogen elevation, which is strongly associated with interleukin (IL)-6 level, has not been shown to be consistently associated with mortality.[5,10] However, progressive reduction in fibrinogen level has been associated with poor prognosis but has been shown that it occurs in later stages of the disease, and it is measured less than 1g/L in approximately 29% of patients who died. With these detections, fibrinogen does not appear to be useful in early detection of poor progression in COVID-19. In our study, in critically ill patients who need to be followed up in the intensive care unit, fibrinogen levels were found to be statistically significantly higher than those followed in the clinic. According to regression analysis, the OR value of 1.007 makes fibrinogen a weak predictor of poor prognosis.

PT elevation is among the DIC diagnostic criteria determined by ISTH and PT is used for the diagnosis of overt DIC.[5,16,17] Unlike traditional sepsis, the PT is normal or slightly prolonged in most patients in COVID-19, prolonged PT is detected in 5% of COVID-19 cases.[3,5] However, significant PT prolongation has been shown in critical and dying cases of COVID-19.[4,5,7] In the study of Tang et al.[10], mean PT values were found to be 1.9 seconds longer in mortal COVID-19 cases. In addition, they reported that approximately 48% of mortal cases showed prolonged PT prolongation in the course of the disease for more than 6 seconds. Therefore, the level of PT helps clinical follow-up and evaluation of the course of the disease, especially in severe cases. Progressive prolongation of PT is considered a predictor of poor prognosis and mortality.[5] In accordance with the literature, in our study, it was found that patients with long PT measurements at first admission required more intensive care (OR 1.164) and were found to be a poor prognosis indicator.

APTT is often elevated in the severe form of DIC yet, it is not included in criteria for diagnosing overt DIC by ISTH.[15,16] Unlike traditional sepsis, aPTT is usually normal in COVID-19 patients, and aPTT prolongation occurs only in 6% of patients.[3,5] APTT were not different in critically ill patients and no significant correlation with disease progress was shown.[4,5,10] In our evaluations, no significant difference was found between critical and non-critical patients in terms of aPTT values. Therefore, aPTT has not been evaluated as an indicator of poor prognosis in COVID-19.

In patients who progress to DIC, thrombocytopenia is common and often means clinical decompensation and organ dysfunction. However, in the COVID-19 clinic, the platelet count is usually normal or slightly decreased and thrombocytopenia is occurred in 12-36% of patients with a platelet count below 100x10^9/L in 5% of cases.[3-6,10] In a meta-analysis of 1779 COVID-19 patients, male patients with severe COVID-19 infection found more thrombocytopenia than women with mild clinical course.[11] Severe thrombocytopenia has been reported with the progression of the disease in COVID-19 patients resulted in death. In our study, although the course of platelets was investigated during the treatment process, the first platelet values on admission to the hospital were found to be significantly lower in critical patients who needed follow-up at ICU compared to clinical patients, and it was found to be consistent with the literature. In our study, platelet values at the first admission to the hospital were found to be significantly lower in critically ill patients requiring follow-up in the ICU compared to clinical patients. In our study, the course of platelet amount during treatment was not included in discussion. Consistent with literature, PLT levels at hospitalization were found to be significantly lower in patients who needed ICU.

DIC is an acquired syndrome that triggers intravascular activation of coagulation causing organ dysfunction by causing microvascular damage.[15-18] DIC is seen in 30-50% of patients with severe sepsis.[19,20] Thrombosis and bleeding it causes result in organ dysfunction. DIC can be overt or latent. Latent DIC cannot be easily noticed since it is caused by an instability between activation and inhibition of the coagulation system and occurs with thrombus rather than bleeding. Overt DIC, that caused by significant irregularity in the coagulation system, results in widespread microvascular thrombosis, bleeding, and consumption coagulopathy and is easier to diagnose.[1,5,18,19] Unlike sepsis-associated coagulopathy in coagulopathy due to COVID-19, most patients do not have DIC, and it is interpreted as clinical latent DIC in detected cases. Therefore, the development of DIC in COVID-19 patients often occurs with vascular thrombosis. This is a late sign for all possible medical interventions that could reverse the underlying process in treatment.[5] In our study, two (22.2%) of the cases who died met the DIC criteria, and no evidence of bleeding was observed in the clinic in both cases.

In Tang's study, DIC was detected in 71.4% of the cases who died, and the average time from admission to DIC was reported as 4 days.[10] Although thromboembolic events are generally seen with the increase in the severity of the disease, a case admitted to the hospital with acute splenoportal mesenteric vein thrombosis with elevated CRP, d-dimer has been reported.[21] Detection of diffuse alveolar damage and microvascular thrombus in patients who died in postmortem autopsy studies reveals the effect of coagulation disorders on prognosis in covid-19.[22]

The COVID-19 epidemic in our country started in March 2020, and the health system reacted quickly to combat the epidemic, awareness of the disease was created in our society, and the follow-up process began with early application to health centers. However, scientific research on the disease process is also planned.[23-26] An important reason why we found the rate of DIC to be low in our study may be that, thanks to the strong health system in our country, patients were admitted early and the patients were observed in a light clinic.

Hadid et al.[5] noted that with a few key differences, severe CIC in its late stages can progress like sepsis-induced coagulopathy (SIC). Significant thrombocytopenia, occurring in 22-58% of patients with SIC, is either absent or very mild in most COVID-19 patients with CIC.[5,27] Again, in these patients, mostly PT and aPTT are detected as normal or slightly long, while the prevalence of hypofibrinogenemia is less than classical sepsis.[5] These differences may explain the rarity of bleeding in COVID-19 patients.[5,14] On the other hand, it is stated that the disproportionate high level of D-dimer with respect to changes in coagulation parameters frequently seen in COVID-19 reflects a significant increase in thrombin production and fibrinolysis. However, since DIC is not typically seen in COVID-19, it is thought that organ damage during this process may be limited to certain organs such as the lungs and kidneys.[5]

In our study, it is known that severe pneumonia, which is the strongest marker of poor prognosis in triage, has distinctive features compared to traditional pneumonia. COVID-19 patients have been found to develop an unregulated host response that causes excessive release of inflammatory cytokines and chemokines. The release of these molecules induces a macrophage activation syndrome-like pathway that triggers cytokine storm, which initiates or further increases pulmonary coagulopathy and microvascular thrombosis.[5,28]

In addition to the SIC (PT, Platelet, SOFA), DIC (Platelet D-dimer, PT, Fibrinogen) scoring recommended by ISTH during the hospitalization of the patients, the coagulation criteria recommended in the guideline of the ministry (platelet <100.000, PT>3 sec, APPT>5 sec, fibrinogen <150mg, D-dimer >4-6 times) was followed. As a result of our study, it was found that the percentage of patients who died increased significantly as the scores of QSOFA, SIC, DIC, and coagulation markers increased.

CONCLUSION

The fact that our study covers the beginning period of the pandemic and data from a single center can be considered as a limitation. However, analysis of laboratory parameters and risk factors is important in understanding the clinical course of the disease and will guide future studies. During this period, all patients diagnosed with COVID-19, including patients with mild clinical symptoms, were hospitalized and isolated, and the patients were taken under clinical observation. In a study conducted in this population, coagulation parameters were found to be a weak marker to show a poor prognosis. Rather than evaluating the coagulation parameters as isolated, having a coagulation marker score of 3 or above is seen as a stronger indicator in predicting a poor prognosis. In order to reveal the effectiveness of coagulation parameters in predicting intensive care need and mortality, it is necessary to conduct studies in larger series of the later stages of the pandemic.

ETHICAL DECLARATIONS

Ethics Committee Approval: The study was approved by the Keçiören Training and Research Hospital Clinical Researches Ethics Committee (Date: 08.07.2020, Decision No: 2135).

Informed Consent: Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process: Externally peer-reviewed.

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

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