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Factors in Relation with in-Hospital Mortality in Geriatric Patients with COVID-19

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Abstract

BACKGROUND/AIMS: The World Health Organization has designated a severe acute respiratory syndrome as coronavirus-2 (SARS-CoV-2) infection. It is caused by a new coronavirus novel coronavirus disease-2019 (COVID-19), which was initially diagnosed in December, 2019 in Wuhan, China. SARS-CoV-2 infection particularly affects the geriatric age group, with a more severe progress rate of the disease. This study aimed to define the clinical characteristics of geriatric individuals with COVID-19 and investigate the in-hospital mortality rate and its predictors.

MATERIALS AND METHODS: This was a descriptive and retrospective research. This research covered all individuals over the age of 60 who were admitted to hospital with a COVID-19 diagnosis. The demographics, physical examination findings, vital signs, hospital outcomes, comorbidities, and laboratory results of these patients were assessed. The main results were the discovery of the hospital mortality rate and its determinants.

RESULTS: A total of 168 elderly individuals with positive polymerase chain reaction (PCR) test findings were included in this research. 51.8 percent of the patients were female, with a median age of 67 years. The patients in our research had an in-hospital death rate of 10.7%. We found that high troponin and fibrinogen and low oxygen saturation at the time of admission had a negative effect on survival.

CONCLUSION: High troponin and fibrinogen levels, as well as low oxygen saturation levels at the time of admission, were found to be the most important predictors of in-hospital death in elderly COVID-19 patients according to our study results. We believe that it would be beneficial to monitor other parameters not only at the time of admission, but also during the hospitalization period of the patients.

Keywords: COVID-19, geriatric, mortality

INTRODUCTION

The World Health Organization (WHO) dubbed novel coronavirus disease-2019 (COVID-19), which causes a severe acute respiratory syndrome, as coronavirus 2 (SARS-CoV-2) infection. It was first diagnosed in Wuhan, China, in December 2019. Shortness of breath, myalgia, tiredness, dry cough, and fever are the primary clinical signs of this disease, which is extremely infectious. COVID-19 spread swiftly throughout China and other nations around the world since the first recorded case in Wuhan, China, in late 2019 [1,2]. The WHO labeled it as a pandemic on March 11, 2020, after a number of people died as a result of it.[1,2]

SARS-CoV-2 infection particularly touches the geriatric age group, who experience a more serious progress of the disease.[3,4] Early statistical data from China regarding the case-mortality rates of patients aged >60 years indicated that the rate was 3.6% in patients aged 60–69 years, 8% in patients aged 70–79 years, and 14.8% in individuals aged >80 years, which shows that the elderly are more vulnerable to COVID-19.[5] To the best of our knowledge, there have been few studies in the literature that have focused on the clinical features of senior COVID-19 patients, and their risk factors for death are still being investigated. Studies conducted by Chinese researchers on geriatric patients have shown that dyspnea

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at presentation, complications, including acute respiratory distress syndrome (ARDS), and comorbidities, including chronic obstructive pulmonary disease and cardiovascular disease, are some of the factors that result in poor outcomes.[6,7]

There are limited studies on this subject in Turkey. This study intended to identify the clinical features of geriatric patients who have COVID-19 and explore the in-hospital mortality rate and its predictors.

MATERIALS AND METHODS

This descriptive retrospective research was carried out in a tertiary-care training and research hospital. The study protocol was approved by the local ethics committee before implementation (no: 2012-KAEK-15/2149, date: 22/07/2020).

Study Population

Included in the study were hospitalized patients aged ≥60 years who had positive results from a polymerase chain reaction (PCR) test between March 21, 2020 and July 1, 2020. Since the first case of COVID-19 in Turkey, our hospital has conducted the hospitalization, examination, and treatment of patients in accordance with the Ministry of Health's national COVID-19 standards.[8] Patients with incomplete data and those with negative PCR test results who were followed up with a pre-diagnosis of COVID-19 were excluded from this study. The required laboratory tests [ferritin, fibrinogen, D-dimer, partial thromboplastin time (PTT), prothrombin time (PT), erythrocyte sedimentation rate, C-reactive protein (CRP), routine biochemical tests, and complete blood count) and imaging tests (posterior-anterior chest radiography and/or low-dose chest computed tomography without contrast] were performed based on the results of physical examinations.

Reverse transcriptase PCR (RT-PCR) testing on nasopharyngeal swabs using techniques approved by Turkey's reference network of SARS-CoV-2 confirmed the diagnosis of COVID-19. The hospital outcomes, the patients' physical examination findings, vital signs, length of hospitalization, complaints at admission, demographic data, comorbidities, and their laboratory results were obtained from the hospital management system and patient files using retrospective scanning. The patients were also divided into two groups based on their in-hospital mortality: those who survived (survivors) and those who did not survive (non-survivors). The impact of the factors that were statistically significant in univariate analysis on mortality were investigated using multivariate logistic regression analysis.

Statistical Analysis

The data analysis was performed using IBM SPSS 20.0 (IBM Corp., Chicago, IL, USA) statistical software. The normal distribution of discrete and continuous numerical variables was investigated using the Kolmogorov–Smirnov test. Descriptive statistics are presented as medians and interquartile ranges (IQR: 25–75) for discrete and continuous numerical variables and as case numbers and percentages (%) for categorical variables. The categorical and continuous variables were evaluated using the Chi-square and Mann–Whitney U tests, respectively. Univariate tests were used to assess mortality predictors, and variables that were statistically significant ($p \leq 0.2$) were included in the regression model of multivariate logistic. The correlation of those parameters that were significant in univariate tests was evaluated using the Spearman correlation test. Multivariate regression pattern fit was evaluated using the Hosmer–Lemeshow test. In addition, $p<0.05$ was considered statistically significant.

RESULTS

This research included 168 geriatric individuals who had positive PCR test results. The median age of these patients was 67 years, and 51.8 percent of them were female. The most common comorbidity was hypertension (60.1%). The most common complaint was cough and/or sputum (49.4%), and 85.7% of patients had pneumonia. The average stay in the hospital was ten days. Tables 1 and 2 show the patients' demographics and laboratory results, respectively.

When the patients' characteristics were compared according to in-hospital mortality (non-survivor/survivor), there were statistically significant differences regarding age, hypertension, heart rate, oxygen saturation, and dyspnea ($p<0.05$ for all) (Table 3). Furthermore, a comparison of the laboratory results of the survivors and non-survivors showed that the values for CRP, ferritin, D-dimer, fibrinogen, PT, urea, aspartate transaminase (AST), and troponin were higher in the non-survivors ($p<0.05$ for all values) (Table 4).

The effects of the factors in Tables 3 and 4 on mortality were investigated using multivariate logistic regression analysis, in addition to the other variables. As there was a correlation of the values for urea, creatinine, D-dimer, fibrinogen, and heart rate with body temperature, the model did not contain the PTT and PT values of all patients. The multivariate model included hypertension, body temperature, oxygen saturation, troponin, AST, urea, fibrinogen, and CRP data, as well as age and gender, with $p \leq 0.2$ from Table 3. After the Hosmer–Lemeshow test was used to determine if the model was a good fit, it was discovered that high troponin and fibrinogen levels, as well as low oxygen saturation at the time of admission, had unfavorable effects on survival (Table 5).

DISCUSSION

In this research, which investigated the factors affecting geriatric COVID-19 patients and their in-hospital mortality, two important results were found. First, the in-hospital mortality rate of the study's patients was 10.7%, and those who did not survive were older, more frequently referred to the hospital due to dyspnea, and had low oxygen saturation and higher CRP, ferritin, D-dimer, fibrinogen, PT, urea, AST, and troponin values. Second, the most prominent predictors of in-hospital mortality in geriatric COVID-19 patients were high troponin and fibrinogen and low oxygen saturation at admission.

COVID-19 has a particularly negative impact on the aged population in terms of illness prevalence, severity, and death rates. In studies of hospitalized COVID-19 patients in all age groups, the median age ranged from 49 to 56 years.[9-11] A study conducted in China reported that the rates of hospitalization due to COVID-19 diagnosis increased with age.[12] Old age is also linked to a higher risk of death.[5,13], and studies in China and Italy have shown that mortality increases as age increases.[5,14] In our study, the median age of patients who did not survive was higher, similar to results in the literature, but there was no gender difference. This higher median age may have been a result of the curfew restriction applied to the elderly in Turkey for the majority of the study period.

One of the mechanisms explaining the high incidence seen in elderly COVID-19 patients may be increased viral shedding. As older patients have higher peak viral loads, they are more likely to spread the virus.

Table 1. Socio-demographic data of patients (n=168)	
Age, years, median (IQR: 25–75)	**67 (63–73)**
Sex, n (%)	
Female	87 (51.8%)
Male	81 (48.5%)
Current smoker, n (%)	20 (11.9%)
Comorbidity, n (%)	
Hypertension	101 (60.1%)
Diabetes mellitus	79 (47%)
Congestive heart failure/coronary artery disease	30 (17.9%)
Chronic lung disease	23 (13.7%)
Malignancy	7 (4.2%)
Chronic renal disease	11 (6.5%)
Other diseases	41 (24.14%)
Vital signs, median (IQR: 25–75)	
Respiratory rate	15 (14–18)
Saturation	92 (90–95)
Pulse	80 (73.2–88)
Fever (temperature)	37.2 (36.5–38.0)
***Complaints of patients, n (%)**	
Without complaint	23 (13.7%)
Cough/sputum	83 (49.4%)
Fever	73 (43.5%)
Dyspnea	48 (28.6%)
Myalgia/fatigue	73 (43.5)
Sore throat	18 (10.7%)
Diarrhea	9 (5.4%)
Nausea or vomiting	15 (8.9%)
Headache	13 (7.7%)
Anosmia	8 (4.8%)
Pneumonia severity, n (%)	
Without pneumonia	24 (14.3%)
Mild	96 (57.1%)
Moderate/severe	48 (28.6%)
Bilateral infiltration in thorax CT, n (%)	72 (42.9%)
Day from disease onset to hospital admission, median (IQR: 25–75)	2 (1–4)
Treatment, n (%)	
Nasal oxygen	50 (29.8%)
High flow oxygen	20 (11.9%)
Non-invasive ventilation	17 (10.1%)
Invasive ventilation	20 (11.9%)
Medical treatments, n (%)	
Chloroquine	168 (100%)
Azithromycin	72 (42.9%)
Favipravir	65 (38.7%)
Oseltamivir	27 (16.1%)
Immune plasma	14 (8.3%)
Enoxaparin treatment dose	53(31.5%)
Enoxaparin prophylaxis	102 (60.7%)
Antibiotics	53 (31.5%)
Plasma	14 (8.3%)
Tocilizumab	8 (4.8%)
Prognosis	
Healed and discharged	150 (89.3%)
Death	18 (10.7%)
Intensive care unit, n (%)	32 (19%)
Length of stay of ICU, median (IQR: 25–75)	7.5 (4–26.7)
Length of stay of hospital, median (IQR: 25–75)	10 (7–13)

*Some patients may have had more than one complaint.
IQR: interquartile range, CT: computed tomography, ICU: intensive care unit, n: number.

Table 2. Laboratory results of all geriatric patients	
Parameters, median (IQR: 25–75)	
Hemoglobin	13.5 (12.6–14.4)
White blood cell	6000 (4700–7350)
Neutrophil	3795(268–5252)
Lymphocyte	1315 (980–1857.5)
Platelet	192 (160.2–239.7)
Neutrophil lymphocyte ratio	2.94 (1.89–4.89)
Platelet lymphocyte ratio	147.5 (103.1–209.9)
C-reactive protein	17.6 (7.6–49.50)
Sedimentation	37 (23–55)
Ferritin	165.8 (81.3–299.2)
D-dimer	550 (390–1020)
Fibrinogen	165.8 (81.3–301.9)
Prothrombin time	11.3 (10.5–11.9)
Partial thromboplastin time	24 (21.7–27.1)
Urea	36.4 (27.8–44.3)
Creatinine	0.97 (0.83–1.13)
Aspartate aminotransferase	27.5 (19–36)
Alanine aminotransferase	21 (15–31.7)
Lactate dehydrogenase	233 (198–279)
Troponin	6.05 (3.82–15.24)

IQR: interquartile range.

The cause of increased viral loads in the elderly is not entirely clear, but it may be the result of the effects of aging on both airway patency and the immune system.[15]

Cough, fever, and dyspnea were the most prevalent symptoms in the research, in that order. The novel coronavirus is mostly responsible for lung infections. Reduced airway clearance, lung reserve, and defensive barriers, as well as muscle atrophy, produce changes in the physiological activities of the respiratory system in the aged.[3] Lung infections were more prevalent in COVID-19-infected senior individuals than in young patients, and the disease had a more severe course in geriatric patients. In our study, 85.7% of the patients showed relevant findings on thorax imaging, and 28.6% had moderate-to-severe pneumonia. In addition, those patients who did not survive presented with lower oxygen saturation levels, which can be a predictor of mortality.

Biochemical monitoring of COVID-19 patients using *in vitro* diagnostic tests is imperative for assessing the diagnosis, progression, and severity of the disease and for monitoring its treatment. The human angiotensin-

Table 3. A comparison of the patient characteristics regarding in hospital mortality (survivor/non-survivor)			
	Survivor (n=150)	**Dead (n=18)**	**p-value**
Age, years, median (IQR: 25–75)	67 (62–72)	72 (68–81.5)	0.005
Sex, n (%)			
Male	71 (47.3%)	10 (55.6%)	0.509
Female	79 (52.7%)	8 (44.4%)	
Comorbidity, n (%)			
Hypertension	86 (57.3%)	15 (83.3%)	0.033
Diabetes mellitus	69 (46%)	10 (55.6%)	0.443
Congestive heart failure/coronary artery disease	26 (17.3%)	4 (22.2%)	0.533*
Chronic lung disease	20 (13.3%)	3 (16.7%)	0.717*
Chronic renal disease	8 (5.3%)	3 (16.7%)	0.099
Vital signs, median (IQR: 25–75)			
Pulse	84.5 (72–95.2)	95 (79.5–98)	0.012
Saturation	93 (90–95)	88.5 (85–92)	<0.001
Fever (temperature)	37.1 (36.5–38)	37.9 (37.1-38.2)	0.071
Complaint of patients', n (%)			
Fever	63 (42%)	10 (55.6%)	0.273
Cough/sputum	72 (48%)	11 (61.1%)	0.293
Dyspnea	38 (25.3%)	10 (55.6%)	0.007
Fatigue /myalgia	65 (43.3%)	8 (44.4%)	0.928
Diarrhea	8 (5.3%)	1 (5.6%)	0.968*
Presence of pneumonia, n (%)	127 (84.7%)	17 (94.4%)	0.475
Bilateral infiltration in thorax CT, n (%)	65 (43.3%)	7 (38.9%)	0.719
Day from disease onset to hospital admission, median (IQR: 25–75)	2 (1–3)	3 (1–5)	0.376

*Fisher exact test.
IQR: interquartile range, CT: computed tomography, n: number.

Table 4. Comparisons of laboratory findings			
	Survivor (n=150)	**Dead (n=18)**	**p-value**
Hemoglobin	13.5 (12.6–14.4)	13.7 (12.3–14.4)	0.994
White blood cell	6000 (4700–7200)	6300 (4625–10,350)	0.547
Neutrophil	3765 (2715–5132)	4000 (2370–8077.5)	0.462
Lymphocyte	1335 (980–1812.5)	1245 (932.5–2027)	0.843
Platelet	192.5 (163–239.2)	173(132–254.7)	0.433
Neutrophil lymphocyte ratio	2.81 (1.81–4.58)	3.5 (2.01–7.06)	0.204
Platelet lymphocyte ratio	150.2 (103.8–210)	134 (82.6–200)	0.214
C-reactive protein	16.1 (7.06–35.4)	74.9 (29.6–173.1)	<0.01
Sedimentation	37 (23–54)	45 (24–63.2)	0.531
Ferritin	160 (77.6–272.3)	295 (100.3-576.2)	0.020
D-dimer	520 (390–942.5)	1045 (505–2350)	0.005
Fibrinogen	160 (77.6–272.3)	295.7 (100.3–576.2)	0.030
Prothrombin time	11.2 (10.5–11.9)	11.8 (11.3–12.9)	0.005
Partial thromboplastin time	23.8 (21.8–26.3)	26.3 (21.3–34.2)	0.099
Urea	36.4 (27.8–42.8)	41.7 (33.6–83.8)	0.013
Creatinine	0.97 (0.82–1.12)	1.05 (0.89–1.55)	0.133
Aspartate aminotransferase	26 (134.2)	38 (24.2–69.2)	0,012
Alanine aminotransferase	20 (15–31)	23.5 (14.7–41.7)	0.322
Lactate dehydrogenase	228 (198–277.5)	243 (201.7–394.2)	0.312
Troponin	5.99 (3.51–11.9)	24.6 (5.2–76)	0.001

n: number.

converting enzyme 2 (ACE2) cell surface receptor is bound by this new coronavirus. Although the disease mainly affects the lower respiratory tract, the virus can also bind to other organs. High ACE2 expression has been detected in bladder urothelial cells, the ileum and esophageal epithelium, kidney proximal tubule cells, myocardial cells, and type II alveolar cells, indicating that these organs and tissues are targets of SARS-CoV-2.[16,17] Our study found that patients who did not survive had higher levels of urea, and those with chronic kidney disease also had a higher mortality rate. Studies have shown that the blood urea nitrogen and creatinine levels in patients who were followed up due to COVID-19 infection were generally high, and their glomerular filtration rates were <60 mL/min/1.73 m^2. During the patients' hospitalizations, proteinuria and hematuria were also observed, as was increased creatinine.[18] Hence, the kidney functions of COVID-19 patients may be affected, primarily by the presence of an existing disease or multi-organ dysfunction.

It is believed that SARS-CoV-2 can infect the liver bile duct endothelial cells and result in inflammatory damage to the liver. Studies have shown a general increase in liver enzymes. However, it was found that there is no direct relationship between the increase in enzymes and the disease's severity. Although the cause of liver damage in COVID-19 is not fully understood, it is suggested that the elevation in liver enzymes is caused by cytokine storm syndrome and drug-induced liver damage.[19] CRP is an acute phase reactant which is synthesized in the liver and increases during inflammation due to infection or tissue damage. In the vast majority of COVID-19 patients, it was found to be high, and it is associated with the disease's severity.[17,20] It is also believed to be an early marker for sepsis and mortality. A retrospective study by Luo et al.[20] suggested that CRP levels, particularly at the time of admission, may be important in grading disease severity. Although those patients in our study who did not survive had elevated AST and CRP levels, no significant results were achieved in the model we established. We believe that the changes in these acute phase reactants during follow-up, rather than their levels at presentation, may be significant for mortality.

Ferritin, a protein involved in iron storage, increases in COVID-19 as a result of the activation of macrophages and hepatocytes. The stimulation of macrophages and hepatocytes causes ferritin, a protein implicated in iron storage, to rise in COVID-19. High blood ferritin levels and a life-threatening hyper-inflammation maintained by a cytokine storm define COVID-19 hyperferritinemic syndromes and systemic inflammatory response, which finally results in multi-organ failure.[21] In a study of patients hospitalized due to COVID-19, the level of ferritin in the blood was shown to be associated with the severity of the condition.[22] Our study also found higher ferritin levels in those patients who did not survive. Also, the troponin levels of the patients who did not survive were elevated at the time of hospital admission; this was another mortality predictor, as the regression model demonstrates.

COVID-19 can cause serious inflammatory events which can increase thrombosis and myocardial infarction. In elderly individuals with COVID-19 pneumonia, cardiac problems such as myocardial infarction, arrhythmia, and/or worsening/novel heart failure are prevalent.[22,23]

COVID-19 individuals have been shown to have a coagulation problem predisposition. Hence, coagulation tests are important in diagnosing and evaluating disseminated intravascular coagulation (DIC), which is a serious complication in COVID-19 patients. The levels of PT, fibrinogen, and D-dimer are the prominent markers linked to disease severity. It was observed that levels of fibrinogen and D-dimer are significantly higher in geriatric intensive care patients and patients with severe pneumonia.[17,23] In our study, those patients who did not survive had higher PT, D-dimer, and fibrinogen levels at the time of admission than those who survived. A study of 225 patients reported that 6.4% of those who did not survive had elevated DIC and fibrinogen levels.[24] As a statistically significant positive correlation was observed between fibrinogen and D-dimer in our study model, when only fibrinogen was included in the mortality model, a high level of fibrinogen at the time of admission was shown to be another mortality predictor. The literature emphasizes the importance in hospitalized patients of sequential follow-up of D-dimer levels, rather than the D-dimer value at the time of admission. In a review published by Mucha et al.[25] D-dimer, fibrinogen, PT, and PTT follow-up were recommended every 48 hours for venous thromboembolism (VTE) prophylaxis in patients hospitalized due to COVID-19. Furthermore, they recommended a VTE ultrasound in individuals with level of D-dimer ≥3.0 µg/mL.

Table 5. Multivariate regression model to predict 28-day mortality

	Wald	p-value	Odds ratio	(95% CI)
Age	3.448	0.063	1.106	0.99–1.23
Gender	1.419	0.234	0.362	0.07–1.92
Hypertension	0.015	0.903	1.117	0.18–6.62
Temperature	0.396	0.529	0.972	0.89–1.06
Saturation	9.756	0.002	0.612	0.45–0.83
Troponin	5.044	0.025	1.012	1.01–1.02
Aspartate aminotransferase	2.326	0.127	1.021	0.99–1.04
Urea	0.728	0.393	1.01	0.98–1.03
Fibrinogen	6.251	0.012	1.008	1.02–1.03
C-reactive protein	0.015	0.901	0.999	0.98–1.01

CI: confidence interval.

Chronic diseases in the elderly are a common problem in the field of global public health. Dai et al.[26] showed that the older patients with COVID-19 exhibited a relatively higher proportion of comorbidities than non-elderly patients. Compared with geriatric patients with a single disease, the hospitalization rates and fatality rates of patients with comorbidities are higher, and the clinical prognosis is significantly poorer.[27] D-dimer and fibrinogen can be influenced by other clinical conditions associated with additional fibrin formation, including old age, malignancies and infections. In our study, D-dimer and fibrinogen levels may have increased due to both COVID-19 disease and other accompanying comorbid diseases.

Although COVID-19 does not yet have a particular medication, studies are ongoing. Various therapies are being used to treat the disease. Since the first case in Turkey, guidelines and treatment regimens have been recommended by the Ministry of Health.[8] Patients in our hospital receive standard treatment in accordance with the ministry's guidelines. We believe that the application of these standard treatments had no effect on mortality. In our research, elevated troponin levels reflected myocardial injury in the COVID-19 patients. Due to fibrinogen's negative effects on atherogenesis, inflammation, platelet activity, coagulation, and plasma viscosity, high fibrinogen levels may increase cardiovascular risk.[28] Although the regulation of anticoagulant cure in patients with COVID-19 was made according to the D-dimer levels of the patients at the time we conducted our study in our country, these treatments can be started earlier and closely monitored, especially in elderly patients

with high fibrinogen and troponin levels. It was determined that seriously sick older individuals with COVID-19 who received early HFNC had a better prognosis than those who received HFNC later.[29] Similarly, we suggest that non-invasive or invasive oxygen treatment could be considered early in this disease process.

There are certain limitations to our research. Firstly, because of the study's retrospective format, all laboratory tests, including those for interleukin 6 (IL-6), IL-2, IL-10, and interferon levels, had not been performed in all of the patients. Therefore, the roles of those tests in predicting in-hospital mortality could not be investigated. Secondly, the patients' laboratory test results during their hospitalizations were not examined. Thirdly, because of the Ministry of Health guidelines for COVID-19, there were slight differences in the processes regarding the hospitalization, examination, and treatment of the patients over the course of the pandemic. During the period just after the first case was detected, patients were hospitalized and followed up even if they were asymptomatic; however, the indications for hospitalization were changed during the following periods, which may have caused a difference in our results. We did not investigate the background characteristics of the patients or the features of the disease. The baseline characteristics and co-morbidities of the patients when they were admitted to the hospital may have influenced the course of the disease. Also, since our study is a descriptive study, our results may include some bias due to the lack of statistical testing.

This study investigated the factors affecting geriatric COVID-19 patients and their in-hospital mortality. The rate of mortality for geriatric COVID-19 patients was found to be 10.7%. In addition, laboratory tests were especially important for monitoring the recovery rates, severity, mortality, and treatment of COVID-19 patients. Those patients who did not survive tended to be older, have numerous pneumonia symptoms, and show higher levels of CRP, ferritin, D-dimer, fibrinogen, PT, urea, AST, and troponin. The most prominent predictors for in-hospital mortality in geriatric COVID-19 individuals were high troponin and fibrinogen and low oxygen saturation levels at the beginning of hospitalization. We believe that it would be beneficial to monitor other parameters during the patients' hospitalization periods rather than only at the beginning of hospitalization.

CONCLUSION

This study investigated the factors affecting geriatric COVID-19 patients and their in-hospital mortality. The rate of mortality for geriatric COVID-19 patients was found to be 10.7%. In addition, laboratory tests were especially important for monitoring the recovery rates, severity, mortality, and treatment of COVID-19 patients. Those patients who did not survive tended to be older, have numerous pneumonia symptoms, and show higher levels of CRP, ferritin, D-dimer, fibrinogen, PT, urea, AST, and troponin. The most prominent predictors for in-hospital mortality in geriatric COVID-19 individuals were high troponin and fibrinogen and low oxygen saturation levels at the beginning of hospitalization. We believe that it would be beneficial to monitor other parameters during the patients' hospitalization periods rather than only at the beginning of hospitalization.

MAIN POINTS

- COVID-19 particularly affects the geriatric age group who experience a more severe course of the disease.
- The in-hospital mortality rate of the study's patients was 10.7% for COVID-19 geriatric patients.
- High troponin and fibrinogen levels, as well as poor oxygen saturation upon admission, were the most important predictors of in-hospital death in elderly COVID-19 patients.

ETHICS

Ethics Committee Approval: Ethics committee approval for this study was obtained from Ankara Keçiören Training and Research Hospital Ethics Committee (decision no: 2012-KAEK-15/2149, date: 22/07/2020).

Informed Consent: Retrospective study.

Peer-review: Externally peer-reviewed.

Authorship Contributions

Concept: E.E., Design: E.E., E.F.G., Data Collection and/or Processing: E.F.G., Supervision: F.K., Analysis and/or Interpretation: E.F.G., S.D., Literature Search: S.D., H.U., Writing: E.E., S.D., H.U., Critical Review: E.E., H.U.

DISCLOSURES

Conflict of Interest: No conflict of interest was declared by the authors.

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