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RESEARCH ARTICLE

Alterations in Biochemical Profiles of Patients with Severe COVID-19 Pneumonia: Analysis of Repeated Laboratory Tests

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ABSTRACT

Objective: This study was initiated to show the changes in the biochemical profile and identify the mortality risk factors of patients with severe coronavirus disease-19 (COVID-19) pneumonia.

Materials and Methods: This study was designed as non-interventional and cohort research. Demographic and clinical data were retrospectively obtained from paper-based documents and electronic health records. Complete blood counts, inflammatory markers, liver, and kidney function tests, and coagulation profiles were recorded 3 times. Two-way ANOVA for repeated measures was used to analyze for continuous dependent variables. Binary logistic regression analysis was performed to determine in-hospital mortality risk factors.

Results: Two hundred and fifty-two adult patients with severe COVID-19 pneumonia enrolled in our study – 15.8% of patients died during hospitalization. The mortality rate was 57.5% for those over 65 years of age. 61.9% of patients had at least one coexisting disease. We revealed hemoglobin, leukocyte, lymphocyte, platelet, C-reactive protein, procalcitonin, d-dimer, aspartate aminotransferase, and alanine aminotransferase, lactate dehydrogenase, creatinine, and ferritin were significantly changing within the time and also between survivors and non-survivors.

Conclusion: The study showed that blood cell counts, coagulation profiles, liver and kidney function tests, and inflammatory markers deteriorated in non-survivor COVID-19 patients. Patients with shortness of breath, history of congestive heart failure, coronary artery disease, dementia, chronic renal disease, higher Charlson comorbidity index score, the need for invasive mechanic ventilation, presence of acute respiratory distress syndrome, and intensive care unit admission are more vulnerable to death.

Keywords: Biochemical profile, COVID-19, mortality, severe pneumonia

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INTRODUCTION

In December 2019, the novel type of Severe Acute Respiratory Syndrome virus (SARS-CoV-2) was isolated as the cause of a cluster of unidentified pneumonia cases in Wuhan City, Hubei, China. Approximately 600 million people have been directly affected, and more than 6.4 million have died since then.[1] The World Health Organization has approved six different vaccines and listed them on Emergency Use List since December 2020. Despite the worldwide coronavirus disease-19 (COVID-19) vaccination initiative, more than 10.000 deaths have been recorded each week.[2]

The clinical manifestation of COVID-19 disease varies broadly, from an asymptomatic or mild upper respiratory tract infection to severe acute respiratory syndrome and even death. No symptoms have been reported in 33% of individuals infected with the SARS-CoV-2. Critical conditions such as

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shock, respiratory failure, and multiorgan dysfunction occur in 5% of patients, and the fatality rate is 2.3%.[3,4]

The risk of severe disease and death for COVID-19 remarkably increases with age. More than 80% of deaths occur in adults older than 65 years. In particular, existing comorbidities such as cardiac conditions, metabolic disorders, and chronic pulmonary diseases worsen the clinical course.[5] Charlson Comorbidity Index (CCI) is a widely used tool to determine the multimorbidity and frailty of patients. It has proved validation and reliability for predicting 1-year survival.[6] The biochemical profile of patients plays a key role in predicting the disease's severity and prognosis. Several studies have focused on various biochemical markers such as urea, alanine aminotransferases (ALT), aspartate aminotransferases (AST), and sodium levels in COVID-19 infections.[7] This study was initiated to demonstrate the changes in the biochemical profile and identify mortality risk factors of COVID-19 patients.

MATERIALS and METHODS

Population, Data Collection, and Definitions

This study was designed as a cohort non-interventional and prospective research. Ethics approval was obtained from the Turkish Ministry of Health and Istanbul Medipol University Ethics Committee (Research Protocol and IRB# 10840098-604.01.01-E.14180). The study was conducted in the Emergency Department of IUC-Cerrahpaşa Medical Faculty. Two researchers scanned electronic health records from November 2020 to April 2021. Patients with severe COVID-19 were included in the study. The coronavirus outbreak guidelines of the Turkish Ministry of Health were considered for the severity criteria. Patients with respiratory distress (dyspnea, using of accessory respiratory muscles), >30 breaths per minute, and <90% oxygen saturation were enrolled in our study as severe pneumonia.[8]

Demographic and clinical data were obtained from paper-based documents and electronic health records. Standardized data-collecting forms have been used for each patient. Obtained parameters such as age, gender, main complaints, comorbid conditions, vital signs, CCI scores, laboratory results, need for oxygen supply or mechanic ventilation, presence of acute respiratory distress syndrome (ARDS), or the emergence of macrophage activation syndrome (MAS) and length of stay were recorded and analyzed for non-survivor and survivor individuals.

MAS is defined as persistent fever, progressive increase in C-reactive protein (CRP) without bacterial infection, elevated D-dimer and fibrinogen levels, impairment of liver function tests, and the presence of any lymphopenia neutropenia or thrombocytopenia.[8] The diagnosis of ARDS was based on the Berlin Definition.[8] CCI score of each patient was calculated through the IOS® application of MDCalc Medical Calculator Clinical Decision Support®.

The RT-PCR results of nasopharynx and throat samples were analyzed by the Cerrahpaşa COVID-19 diagnosis laboratory authorized by the ministry of health. The exclusive and distinctive part of this study is investigating the biochemical profile of the patients with severe COVID-19 pneumonia. Complete blood counts, inflammatory markers, liver, and kidney function tests, and coagulation profiles were recorded 3 times. The first biochemical parameters were taken at hospital admission. The second biochemical parameters were recorded on the median day of the hospitalization or when MAS emerged in patients. Moreover, the third biochemical parameters were recorded on the day of discharge or death of patients. IUC-Cerrahpasa Fikret Biyal Biochemistry Laboratory specifies laboratory values' upper and lower reference limits.

Statistical Analysis

Student t-test was used to ascertain the significance of differences between mean values of two continuous variables, and the Mann–Whitney U test was used for non-parametric distribution. The Chi-square test was performed to analyze differences in proportions of categorical variables between two or more groups. The "ANOVA for repeated measures" test was used if the measurements were three or more. *Post hoc* analyzes of repeated measures ANOVA were evaluated using the Bonferroni test. Wilcoxon signed-rank and Friedman tests were used for comparing not normally distributed groups, depending on group numbers. Cutoff values were identified through the ROC curve. Binary logistic regression analysis was performed to determine mortality risk factors. Odds ratio (OR) and 95% confidence interval (95% CI) were reported. The level $p<0.05$ was considered the cutoff value for significance.

RESULTS

Table 1 presents the clinical characteristics of COVID-19 patients studied. A total of 252 adult patients with severe COVID-19 pneumonia enrolled in our study. The female/male distribution of the population was 43.9% and 56.1%, respectively. The mean length of hospitalization in all patients was 13.15±9.45 days. Non-survivors (17.92±9.97 days) stayed significantly more extended than survivors (12.18±9.07 days) in the hospital ($p<0.001$). In our study, 15.8% of patients deceased during hospitalization, 84.1% patients survived. The mortality rate was 57.5% for those over 65 years of age. The

Table 1. Comparisons of clinical characteristics of patients with severe COVID-19 pneumonia

Clinical characteristics	Overall=252		Non-survivor=40		Survivor=212		p
	n	%	n	%	n	%	
Age	58.9±15.88		68.02±15.17		57.15±15.48		<0.001
>65 age	86	34.1	23	57	63	29.8	0.001
Gender (male)	141	56.1	25	62.5	116	54.7	0.462
Signs and symptoms							
Cough	164	65.1	26	65.0	138	65.1	1.000
Shortness of breath	137	54.4	35	87.5	102	48.1	<0.001
Myalgia	110	43.7	25	62.5	85	40.1	0.014
Fever (>37.8 °C)	68	27.0	16	40.0	50	23.5	0.052
Headache	31	12.3	6	15.0	25	11.8	0.600
Diarrhea	22	8.7	4	10.0	18	8.5	0.761
Loss of taste or smell	21	8.3	8	20.0	13	6.1	0.009
Asymptomatic	16	6.3	0	0.0	16	7.5	
Body temperature (°C)	37.35 ±1.58		37.37±0.97		37.13±0.87		0.146
Oxygen saturation	93.21±4.69		89.42±8.04		93.90±3.31		<0.001
Chronic diseases							
Coexisted diseases	156	61.9	35	87.5	121	57.0	<0.001
Hypertension	115	47.7	22	55.0	93	43.9	0.261
Diabetes mellitus	65	26.9	13	32.5	52	24.5	0.390
Congestive heart failure	29	12.0	9	22.5	20	9.4	0.028
Cancer	28	11.6	13	32.5	15	7.1	<0.001
Coronary artery disease	26	10.7	11	27.5	15	7.0	<0.001
Metastasis	17	6.7	12	30.0	5	2.4	<0.001
COPD	16	6.3	6	15.0	10	4.7	0.026
Immunosuppression	13	5.1	3	7.5	10	4.7	0.440
Dementia	12	4.7	8	20.0	4	1.9	<0.001
Chronic renal disease	9	3.5	4	10.0	5	2.4	0.038
Clinical severity							
CCI score	2.86±2.55		5.67±3.33		2.32±1.96		<0.001
CCI >4.5	54	21.4	23	57.5	31	14.6	<0.001
Oxygen support							<0.001
No	60	23.8	0	0.0	60	28.3	
Nasal cannula	100	39.6	6	15.0	94	44.3	
Non-rebreather mask	33	13.0	3	7.5	30	14.1	
HFNO/NIMV	21	8.3	3	7.5	18	8.4	
IMV	38	15.0	28	70.0	10	4.7	
ARDS (on the first admission)	30	11.9	16	40.0	14	6.6	<0.001
MAS (in the disease course)	81	32.1	20	50.0	61	28.7	0.014
Secondary bacterial infection	139	55.1	39	97.5	100	47.1	<0.001
Clinical course and outcomes							
ICU admission	64	25.3	39	97.5	25	11.7	<0.001
Length of stay (day)	13.15±9.45		17.92±9.97		12.18±9.07		<0.001
Mortality (in hospital)	252		40	15.8	212	84.1	

COPD: chronic obstructive pulmonary disease; CCI: Charlson comorbidity index; HFNO: high flow nasal oxygen; NIMV: non-invasive mechanic ventilation; IMV: invasive mechanic ventilation; ARDS: acute respiratory distress syndrome; MAS: macrophage activation syndrome; ICU: intensive care unit

mean age of the study population was 58.9±15.88. Non-survivor patients were significantly older than survivors (p<0.001).

Cough was the most common symptom among patients, 65.1%. 156 (61.9%) patients had at least one coexisting disease. The most detected chronic disease was hypertension, 47.7%. In comparison to survivors, congestive heart failure (p=0.038), coronary artery diseases (p<0.001), cancer (p<0.001), metastasis (p<0.001), dementia (p<0.001), chronic obstructive pulmonary disease (p=0.026), and chronic kidney disease (p=0.038) were significantly higher in non-survivors.

The mean CCI score was calculated as 5.67±3.33 for non-survivors and 2.32±1.96 for survivors (p<0.001). We determined the cutoff value of CCI as 4.5 with 70% sensitivity and 76% specificity (AUC=0.804, 95% 0.736–0.882, p<0.001). The number of patients whose CCI score was higher than 4.5 was 54 (21.4%). Moreover, the number of patients with >4.5 CCI scores was significantly higher in the non-survivors group (p<0.001). 60 (23.8%) patients who did not need oxygen supply on the first admission all survived. Other patients needed oxygen support partially. 38 (15%) patients were supported with mechanical ventilation in the emergency department, and 28 of those died in the hospital. In addition, 30 (11.9%) patients met the ARDS criteria on the first admission. MAS occurred in 81(32.1%) patients in the disease course. Both ARDS and MAS frequencies were statistically higher in the non-survivor group. Secondary bacterial infections occurred in 139 (55.1%) patients. The bacterial infection rate was significantly higher in the non-survivor group (p<0.001).

We set our study to compare the three different measurements for biochemical tests. We revealed significant differences between both two groups and within three repeated measures for those results of hemoglobin, leukocyte, lymphocyte, platelet, CRP, procalcitonin, D-dimer, AST, ALT, lactate dehydrogenase (LDH), creatinine, and ferritin. Only fibrinogen did not significantly change between non-survivors and survivors within time. While leukocyte, AST and ALT, LDH, and ferritin values increased, hemoglobin levels constantly decreased in the non-survivors group. On the other hand, despite the significant changes in mean platelet counts in non-survivors, it remained within reference values for three measurements (Table 2 and Fig. 1).

Table 3 gives univariable and multivariable regression analyses of mortality among patients with severe COVID-19 pneumonia. In the univariable study, scoring above 4.5 from CCI has significant odds (OR: 7.56, 95% CI, p<0.001). After adjusting to other factors, getting a score above 4.5 from CCI is a significant predictor of in-hospital mortality (OR: 90.89, 95% CI, p=0.035). Furthermore, in the multivariable analysis, the presence of ARDS (OR: 9.22, 95% CI, p=0.019), the need for invasive mechanic ventilation (IMV) (OR: 34.90, 95% CI, 0.022), and intensive care unit (ICU) admission (OR: 138.38, 95% CI, p<0.001) were significant risk factors for in-hospital mortality.

DISCUSSION

We retrospectively evaluated the demographic and clinical characteristics of the laboratory-confirmed COVID-19 patients. The male/female ratio was approximately 1:1. The mean age of the study population was almost the same as in the literature.[9] In the non-survivor group, most patients were over 65 years of age. The minimum age was 24 among the non-survivor group in this study. The mean age of the non-survivors was slightly lower than the previous similar studies.[2,9] Older age is one of the most critical determinant factors to predict mortality in several studies.[9]

According to the Center for Disease Control COVID-19 surveillance report, cough, fever, and myalgia are the most common symptoms.[10] Cough is the most reported symptom among COVID-19 patients also in this presented study. We only reported 27% febrile patients on the first admission, and this result was approximately similar to Varol et al.'s[11] findings, which was another study within the Turkish population. Dyspnea is the most significant symptom for determining the severity of the disease. Because shortness of breath is associated with admission to ICU, use of IMV, and death,[12] Wang et al.'s[9] Cox regression models suggested that expectoration and dyspnea can be used to predict the prognosis of the disease. Wei et al.[13] also showed that patients with dyspnea, cough, and fever have a more severe clinical course. We also revealed that shortness of breath could be used to predict in-hospital mortality. Interestingly, symptoms of myalgia and loss of smell and taste were significantly more common in non-survivors.

The previous observational studies have revealed that SARS-CoV-2 infection affects patients with comorbid diseases more severely.[14] Kuswardhani et al.'s[15] systematic review and meta-analysis showed that higher CCI was associated with increased mortality and disease severity in patients with COVID-19. In addition, Varol et al.[11] determined the cut-off value of CCI as 2.5 with 78% sensitivity and 74% specificity to predict mortality in COVID-19 patients. Likewise, we also found significantly higher CCI scores among non-survivors. We indicated 4.5 points as the cutoff value of CCI to predict the mortality of COVID-19 patients.

If we focus on comorbid diseases separately, hypertension, diabetes mellitus, congestive heart failure, cancer, and coronary artery disease draw attention. Those were the most

Table 2. Comparisons of laboratory results of patients with severe COVID-19 pneumonia

Laboratory	Time interval	Overall 252	Non-Survivor 40	Survivor 212	p* without time effect	p** time X group effect
Hemoglobin	t1	12.50±1.81	11.70±1.80	12.65±1.78		
(13–17 g/dl)	t2	11.78±1.75	10.49±1.61	12.02±1.67	<0.001	<0.001
	t3	11.62±1.96	9.82±1.98	11.97±1.76		
Leukocyte	t1	7.05±3.75	9.14±5.69	6.66±3.12		
(3.8–10×10^3/µl)	t2	7.29±5.40	13.59±9.55	6.10±2.96	<0.001	<0.001
	t3	8.99±7.52	18.81±10.38	7.13±5.06		
Lymphocyte	t1	1.26±0.68	0.83±0.51	1.35±0.68		
(1.3–3.5×10^3/µl)	t2	1.09±0.59	0.58±0.36	1.18±0.58	<0.001	0.017
	t3	1.63±0.81	0.96±0.83	1.75±0.74		
Platelet	t1	208.87±85.32	213.96±96.29	207.90±83.29		
(150–400×10^3/µl)	t2	221.28±100.59	220.05±125.40	221.52±95.53	<0.001	<0.001
	t3	299.82±137.71	175.34±125.04	323.42±127.12		
CRP	t1	69.91±75.57	111.85±84.10	58.39±70.91		
(<5 mg/dl)	t2	103.47±94.17	185.28±105.23	87.97±83.57	<0.001	<0.001
	t3	38.23±73.69	146.05±126.55	17.78±29.26		
Procalcitonin	t1	0.31±0.81	0.78±1.14	0.22±0.69		
(0–0.5 ng/l)	t2	0.68±1.92	2.85±3.79	0.27±0.80	<0.001	<0.001
	t3	0.48±1.61	2.56±3.34	0.09±0.26		
D-Dimer	t1	2.39±7.75	6.96±17.34	1.53±3.33		
(0-0.5mg/l)	t2	5.77±12.65	15.54±18.88	3.92±10.14	<0.001	<0.001
	t3	3.82±7.76	13.93±12.95	1.90±4.18		
Fibrinogen	t1	475.59±170.95	520.72±183.79	467.04±167.50		
(170–350 mg/dl)	t2	543.69±206.87	564.72±272.30	539.70±192.55	0.763	0.683
	t3	403.36±191.19	434.52±327.03	397.45±153.08		
AST	t1	40.97±41.32	68.45±81.09	35.77±25.31		
(<41 mg/dl)	t2	63.72±72.27	110.57±118.00	54.84±55.99	<0.001	<0.001
	t3	69.60±217.59	258.57±506.92	33.78±23.46		
ALT	t1	32.53±31.05	41.35±48.60	30.86±26.29		
(<40 mg/dl)	t2	78.38±246.64	209.44±585.03	53.53±70.53	<0.001	<0.001
	t3	87.67±210.61	225.07±485.61	56.32±66.74		
LDH	t1	321.66±171.32	483.75±263.93	290.93±128.31		
(<250 mg/dl)	t2	433.52±261.02	697.02±371.09	383.56±199.50	<0.001	<0.001
	t3	382.60±459.60	994.17±914.43	266.66±106.90		
Creatinine	t1	1.03±0.72	1.63±1.59	0.92±0.27		
(0.7–1.2 mg/dl)	t2	1.15±0.94	2.02±1.94	0.98±0.42	<0.001	<0.001
	t3	1.12±1.01	2.42±1.99	0.87±0.29		
Ferritin	t1	474.36±623.12	996.52±1203.83	375.37±362.39		
(30–400 ng/l)	t2	486.61±847.00	1651.80±1613.27	589.89±564.28	<0.001	<0.001
	t3	563.96±850.77	1824.35±1585.07	312.74±331.97		

*: P-value obtained from two-way ANOVA for repeated measures without time effect. Considered Greenhouse-Geisser correction; **: P-value obtained from two-way ANOVA for repeated measures time X group effect. Considered Bonferroni adjustment. g: Gram; dl: Deciliter; µl: Microliter; mg: Milligram; ng: Nanogram; l: Liter; t1: On first admission; t2: The median day of hospitalization or when MAS emerged; t3: The time of discharge or death; CRP: C-reactive protein; AST: aspartate aminotransferase; ALT: alanine aminotransferase; LDH: lactate dehydrogenase

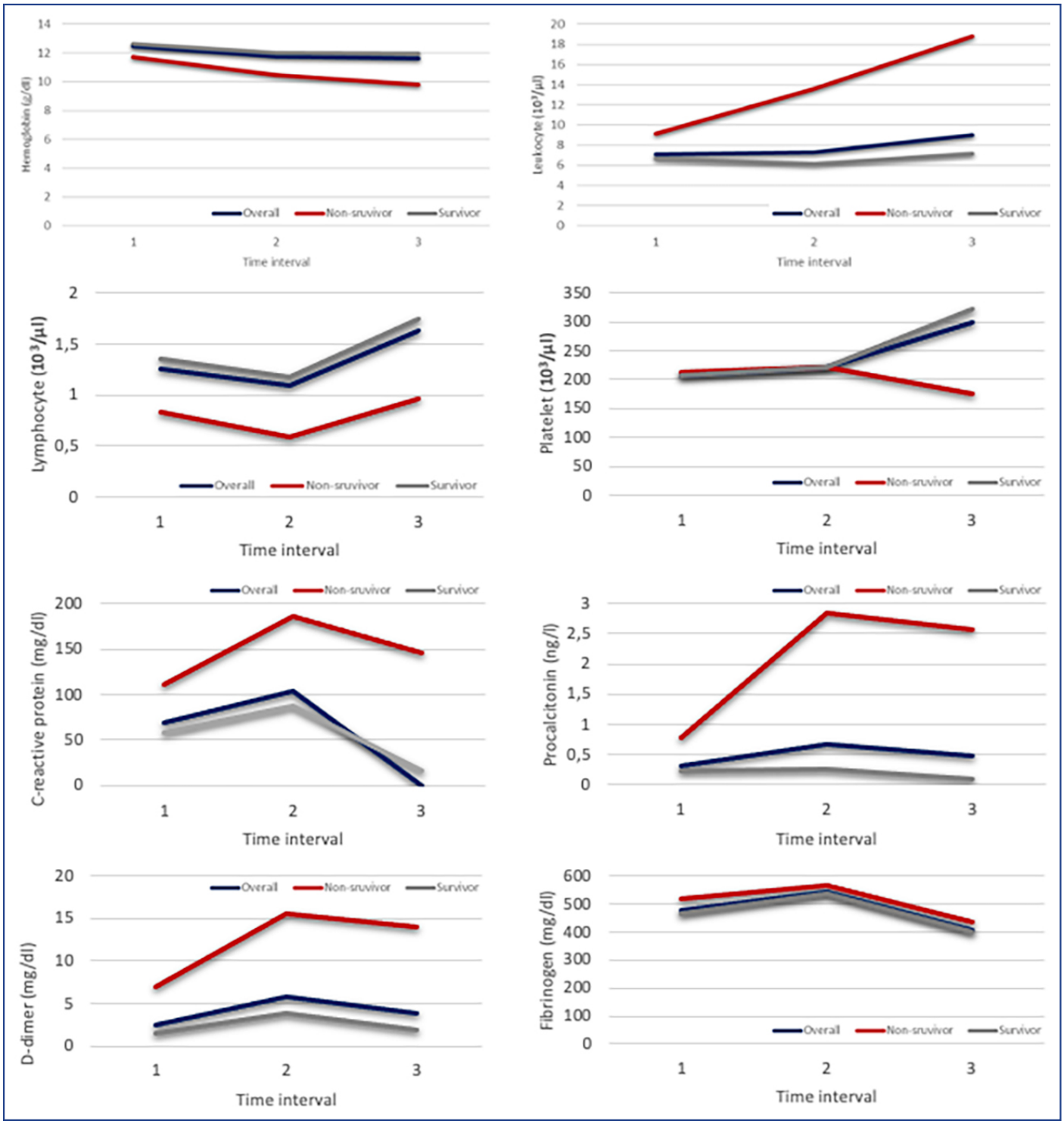

Figure 1. Changes in the laboratory results of patients with severe COVID-19 pneumonia. The line charts above showed the changing of the biochemical profile between groups and within 3-time points apart

reported co-existing diseases in our study population. According to Grasselli et al.[16] Cox regression model, COPD, diabetes, and hypercholesterolemia were significantly associated with mortality.[16] Kim et al.[17] demonstrated the adjusted risk for in-hospital mortality for cardiovascular, neurologic, and renal diseases. Our multivariable regres-

Table 3. Univariate and multivariate regression analysis for in-hospital mortality

Parameters	Univariate			Multivariate		
	B	OR 95% CI	p	B	OR 95% CI	p
Age >65	1.18	2.273 (1.63–6.54)	0.001			0.304
Shortness of breath	2.02	7.54 (2.84–20.01)	<0.001	3.87	12.39 (1.88–115.58)	0.019
Myalgia	0.91	2.49 (1.24–4.99)	0.01			0.983
Loss of taste or smell	1.34	3.82 (1.47–9.96)	<0.001			0.474
Oxygen Saturation	–0.17	0.83 (0.77–0.90)	<0.001			0.459
Congestive heart failure	1.02	2.78 (1.16–6.67)	0.021	5.68	0.03 (0.00–057)	0.030
Cancer	1.84	6.32 (2.71–14.71)	<0.001			0.317
Coronary artery disease	1.75	5.80 (2.37–14.17)	<0.001	3.31	27.43 (1.08–692.03)	0.044
Metastasis	2.87	17.74 (5.81–54.13)	<0.001			0.356
COPD	1.27	3.56 (1.21–10.44)	0.021			0.555
Dementia	2.56	0.07 (0.02–0.27)	<0.001	2.82	16.81 (1.32–53.52)	0.029
Chronic renal disease	1.52	4.60 (1.17–17.95)	0.028	3.70	40.45 (1.72–149.52)	0.022
Getting a score above 4.5 from CCI	2.02	7.56 (3.58–15.95)	<0.001	4.51	90.89 (1.37–6014,95)	0.035
IMV	3.85	47.13 (18-64–119.17)	<0.001	3.55	34.90 (6.86–177.97)	<0.001
ARDS (on the first admission)	2.24	9.42 (4.09–21.68)	<0.001	2.22	9.22 (1.44–58.97)	0.019
MAS (in the course of the disease)	0.90	2.47 (1.24–4.92)	0.01			0.348
Bacterial infection	3.77	43.68 (5.89–323.77)	<0.001			0.348
ICU admission	5.67	291.72 (38.37–2217.49)	<0.001	4.93	138.38 (7.73–2475.51)	<0.001

OR: Odds ratio; 95% CI: 95% Confidence Interval; COPD: Chronic obstructive pulmonary disease; CCI: Charlson comorbidity index; IMV: Invasive mechanic ventilation; ARDS: Acute respiratory distress syndrome; MAS: Macrophage activation syndrome; ICU: intensive care unit

sion model identified those comorbidities, including congestive heart failure, coronary artery disease, dementia, and chronic renal failure, as significant risk factors for in-hospital mortality. We did not find a significant difference in hypertension and diabetes mellitus between survivors and non-survivors. Hypertension and diabetes mellitus are prevalent conditions in Turkey. Large-scale epidemiological studies reported that hypertension prevalence is 33.7%, and diabetes mellitus and pre-diabetes prevalence is 13% in Turkish adults.[18,19] These high frequencies of diabetes and hypertension in the Turkish population might affect our study population. Therefore, it can be the reason for the statistical indifference for those variables between survivors and non-survivors.

Hypoxemia is the main challenge in managing patients with severe COVID-19 pneumonia.[20] Critical care providers have preferred early IMV support during the pandemic due to concerns about nosocomial viral transmission through aerosol released from the NIMV.[21] An international systematic review and meta-analysis by Lim et al.[22] reported a 45% of fatality rate for adult COVİD-19 patients receiving IMV. Similar to the previous studies, our study showed that the need for IMV was a significant risk factor for in-hospital mortality.[9,22,23]

Zhou et al.[23] evaluated the complete blood counts obtained from COVID-19 patients on their first admission. It reported a significant difference between survivors and non-survivors in white blood cell count, lymphocyte and platelet levels. Our findings also showed significant differences in leukocyte, lymphocyte, platelet, and hemoglobin levels between survivors and non-survivors. While the leukocyte counts constantly increased in the non-survivor group, the hemoglobin levels gradually decreased conversely. Despite the statistical change of mean platelet counts for repeated measurements, they remained within reference values in non-survivors. A systematic review and meta-analysis by Tian et al.[24] noted that higher values of inflammatory markers such as CRP, interleukin-6, procalcitonin, and erythrocyte sedimentation rate are higher in the non-survivors group. It also reported impaired liver and renal function tests amongst non-surviving COVID-19 patients. Furthermore, the machine learning model of Bertsimas et al.[25]

showed that elevated blood urea nitrogen, CRP, creatinine, AST, and platelet levels increase the risk of death. In addition, increased D-dimer levels have been demonstrated as a prominent risk factor for poor prognosis in patients with severe COVID-19.[26] The findings of our study had coherence with the previous studies. CRP, procalcitonin, AST, ALT, LDH, ferritin, creatinine, and D-dimer levels were significantly higher in the non-survivor group for three repeated measurements. As a result of gradual worsening in clinical conditions and multiorgan failure of COVID-19 patients, AST, ALT, LDH, ferritin, creatinine, and D-dimer levels have continuously increased in the non-survivor group.

Our study has some limitations. Due to the retrospective design of the study, we were unable to obtain some patient characteristics and had to exclude these parameters. Second, we considered only in-hospital deaths. Therefore, we might underestimate some long-term risk factors. Third, although we had an adequate sample size for a single-center study, larger sample size and a multi-center study would be more effective in identifying the determinants of mortality.

CONCLUSION

This study showed that blood cell counts, coagulation profiles, liver, and kidney function tests, and inflammatory markers deteriorated in non-survivor COVID-19 patients. Patients with shortness of breath, history of congestive heart failure, coronary artery disease, dementia, chronic renal disease, higher CCI score, the need for IMV, presence of ARDS, and ICU admission are more vulnerable to death.

Disclosures

Ethics Committee Approval: The study was approved by the İstanbul Medipol University Non-interventional Clinical Research Ethics Committee (No: 345, Date: 18/03/2021).

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