Cumhuriyet Medical Journal, 44(1): 117-124, 2022
DOI: http://dx.doi.org/10.7197/cmj.1053522

Cumhuriyet Medical Journal

| cmj.cumhuriyet.edu.tr | Founded: 2004 Available online, ISSN:1305-0028 Publisher: Sivas Cumhuriyet Üniversitesi

Comparison of amylase, lipase and d-dimer levels in the etiopathogenesis of mortality in Covid 19 pneumonia

Fadime Güllü Haydar[1], Yavuz Otal[2]*, Gamze Avcıoğlu[3]

[1]*Department of General Surgery, Ankara City Hospital, Ankara, Turkey*

[2]*Department of Emergency Medicine, Ankara City Hospital, Ankara, Turkey*

[3]*Department of Medical Biochemistry, Karadeniz Ereğli State Hospital, Zonguldak, Turkey*

**Corresponding author*

Research Article

History

Received: 04/01/2022
Accepted: 29/03/2022

ABSTRACT

Objective: Coronavirus Disease 2019 (Covid-19) is a pandemic cause affecting the whole world with high mortality. The effects of lipase, amylase and d-dimer levels on mortality were investigated in Covid-19 pneumonia in the present study.

Method: A total of 131 patients diagnosed with pneumonia due to Covid-19 and 60 healthy control groups were included in the study prospectively. The cases were divided into 3 groups as the healthy control group, those who recovered, and those who died. Those who had pneumonia in the lung tomography among the cases diagnosed with Covid-19 were examined. The RT-PCR (Real-Time Polymerized Chain Reaction) test results were recorded from the system. The serum lipase, amylase, albumin levels, WBC (White Blood Cell), N/L (Neutrophil/Lymphocyte Ratio), and CRP (C-Reactive Protein) levels of the patients were compared with those of the Control Group.

Results: When all the data were reviewed, the Lipase, Amylase, and d-dimer levels were found to be statistically significantly higher in the exitus group when compared to the control group, and T. protein and albumin levels were lower ($p<0.01$). Other acute phase reactants, WBC, N/L Ratio, and CRP levels were significantly higher ($p<0.01$). As a result of the comparison of the recovered and exitus group, no statistically significant changes were detected in all parameters ($p>0.05$).

Conclusions: The elevation in serum amylase, lipase, and d-dimer levels may be significant in prognosis in pneumonia developing due to Covid-19.

Keywords: Covid-19; mortality; hyperlipasemia; amylase; d-dimer

Covid 19 pnömonisinde mortalite etyopatogenezinde amilaz, lipaz ve d-dimer düzeylerinin karşılaştırılması

ÖZ

Amaç: Koronavirüs Hastalığı 2019 (Covid-19), tüm dünyayı yüksek mortalite ile etkileyen pandemi nedenidir. Bu çalışmada Covid-19 pnömonisinde lipaz, amilaz ve d-dimer düzeylerinin mortalite üzerine etkisi araştırılmıştır.

Yöntem: Covid-19 nedeniyle pnömoni tanısı alan toplam 131 hasta ve 60 sağlıklı kontrol grubu prospektif olarak çalışmaya dahil edildi. Olgular sağlıklı kontrol grubu, iyileşenler ve ölenler olarak 3 gruba ayrıldı. Covid-19 tanısı konulan vakalardan akciğer tomografisinde pnömoni olanlar çalışmaya dahil edildi. RT-PCR (Real-Time Polymerized Chain Reaction) test sonuçları sistemden kaydedildi. Hastaların serum lipaz, amilaz, albümin düzeyleri, WBC (Beyaz Kan Hücresi), N/L (Nötrofil/Lenfosit Oranı) ve CRP (C-Reaktif Protein) düzeyleri Kontrol Grubu ile karşılaştırıldı.

Bulgular: Tüm veriler gözden geçirildiğinde, exitus grubunda kontrol grubuna göre Lipaz, Amilaz ve d-dimer düzeyleri istatistiksel olarak anlamlı düzeyde yüksek, Total protein ve albümin düzeyleri ise daha düşük bulundu ($p<0.01$).). Diğer akut faz reaktanları, WBC, N/L Ratio ve CRP seviyeleri anlamlı olarak yüksekti ($p<0.01$). İyileşen ve exitus grubunun karşılaştırılması sonucunda diğer tüm parametrelerde istatistiksel olarak anlamlı bir değişiklik saptanmadı ($p>0.05$).

Sonuç: Covid-19'a bağlı gelişen pnömonide prognozda serum amilaz, lipaz ve d-dimer düzeylerindeki yükselme anlamlı olabilir.

Süreç

Geliş: 04/01/2022
Kabul: 29/03/2022

Anahtar sözcükler: Covid-19; ölüm; hiperlipazemi; amilaz; d-dimer

[a] *gulluercanhaydar@gmail.com* *https://orcid.org/0000-0001-9134-5706*
[b] *dryotal@gmail.com* *https://orcid.org/0000-0001-8529-4957*
[c] *gamzeeavcioglu@gmail.com* *https://orcid.org/0000-0002-4333-2641*

How to Cite: Haydar FG, Otal Y, Avcıoğlu G (2022) Comparison of amylase, lipase and d-dimer levels in the etiopathogenesis of mortality in Covid 19 pneumonia, Cumhuriyet Medical Journal, March 2022, 44 (1): 117-124

Introduction

The Coronavirus Disease 2019 (COVID-19) outbreak was caused by a novel coronavirus infection called Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) [1]. The infection spread rapidly in the entire world, and was declared a pandemic by the World Health Organization. There were 150.110.310 documented cases reported worldwide, and 3.158.792 patients were reported to die on April 30, 2021 [2]. It is very important to identify risk factors for mortality because there is currently no specific treatment or medication for this novel virus.

In severe infection, pulmonary involvement is a serious complication requiring admission to the Intensive Care Unit. Lungs are usually affected bilaterally. Mechanical ventilation may be required as a result of severe respiratory failure. There is no specific medication in the treatment of Covid-19 patients, and supportive treatments are used. The blood test results of these patients may be variable. Examining the blood test results in severe pneumonia cases due to Covid-19, which is accompanied by comorbid diseases, is a guide for the prognosis and treatment.

Lipase and amylase tests, which indicate pancreatic functions in cases treated for Covid-19 pneumonia, may vary during treatment. Also, acute phase reactants WBC (White Blood Cell), N/L Ratio (Neutrophil/Lymphocyte Ratio), and CRP (C-Reactive Protein) levels show the severity of the disease.

In COVID-19 patients, the mechanism of pancreatic injury still remains unclear. It can result from a variety of mechanisms, which include direct virus infection, immune damage, drug-induced liver injury, systemic inflammatory response, ischemia and hypoxia, and relapse or exacerbation of any underlying liver disease. In the present study, the relations between lipase, amylase, and d-dimer levels, which indicate pancreatic functions in Covid-19 pneumonia, with other acute phase reactants and their effects on mortality were investigated.

Material and Methods

The blood samples taken from 131 patients diagnosed with Covid-19 and 60 healthy volunteers were studied in the laboratory, and the data were analyzed after the approval of Ankara City Hospital Ethics Committee. Those with positive RT-PCR (Real-Time Polymerized Chain Reaction) Test in the diagnosis of Covid-19 and pulmonary pneumonia on Thoracic Computed Tomography were included in the study.

The cases were divided into 3 groups as the healthy control group, those who recovered, and those who were exitus. The demographic data of the 3 groups that were included in the study, lipase, amylase, total protein, albumin, AST, ALT, GGT, LDH, PT, PTT, T. Bilirubin, D. Bilirubin test results, WBC, N/L Ratios, and CRP values were entered into the statistical program, and detailed analyses were done.

Those who were under the age of 18, trauma patients, pregnant women, and those who were vaccinated against Covid-19 were excluded from the study. Those who had Covid-19 for the first time were included in the study.

Statistical Analysis

The Statistical Package for Social Sciences for Windows, Version 22 (IBM, Armonk, NY, USA) was used for the statistical analyses. The Kolmogorov–Smirnov test was used for the normality of variables. Mean ± standard deviation (SD) was used for the parameters with normal distribution, and median (interquartile range) (IQR) was used for the parameters not consistent with the normal distribution. The One-way ANOVA test was used for parameters that were normally distributed. Those with not normal distribution were evaluated with the Kruskal-Wallis test. Comparisons for categorical variables were performed using the Chi-square test or the Fisher's exact test. The Receiver Operation Characteristic (ROC) curve was performed to analyze the efficiency of the COVID-19 severity. The optimal cut-off values of the Amylase, lipase, CRP, procalcitonin, WBC, D-dimer, and NLR were calculated by applying the ROC analysis; and a p-value <0.05 was considered to be statistically significant.

Results

The demographic data of the cases with Covid-19 pneumonia and the control group are shown in Table 1. No significant differences were detected between age and gender. The RT-PCR test results of 71 patients who died and 60 patients who recovered were positive. The Thoracic Computed Tomography results of all patients were examined, and the findings were entered into the system. The Computed Tomography images of 2 different patients with Covid-19 pneumonia, who recovered on the left and who died on the right are given in Figure 1. The co-morbidities of the patients are shown in Table 1.

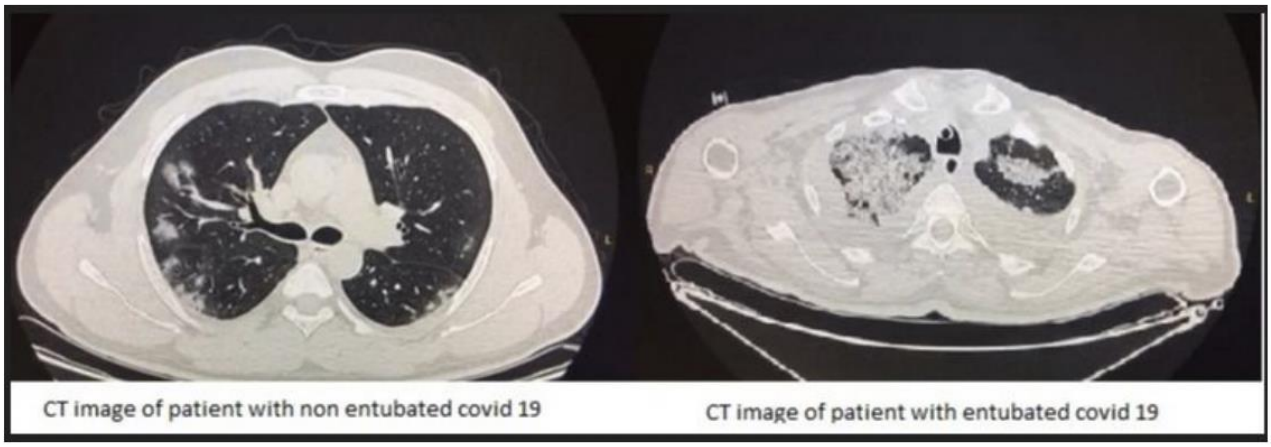

Figure 1. Computed tomography (CT) samples of patients with COVID-19.

In the statistical analysis made, the p value was calculated for each parameter by comparing the groups. The cases were divided into 3 groups as the healthy control group, the exitus (severe), and the recovered (mild) group. As can be seen in detail in table 1, when the data of these three groups were reviewed, the serum total protein and albumin levels were lower at a statistically significant level ($p<0.01$). As seen in Figure 2 and table 2, the Lipase, Amylase, LDH, GGT, AST, aPTT, and T. bilirubin levels were higher at a statistically significant level in the severe and mild patient groups when compared to the Control Group ($p<0.01$). Other acute phase reactants, namely, the WBC, N/L Ratios, and CRP levels were significantly higher ($p<0.01$). As a result of the comparisons of the recovered and exitus group, no statistically significant changes were detected in all parameters ($p>0.05$).

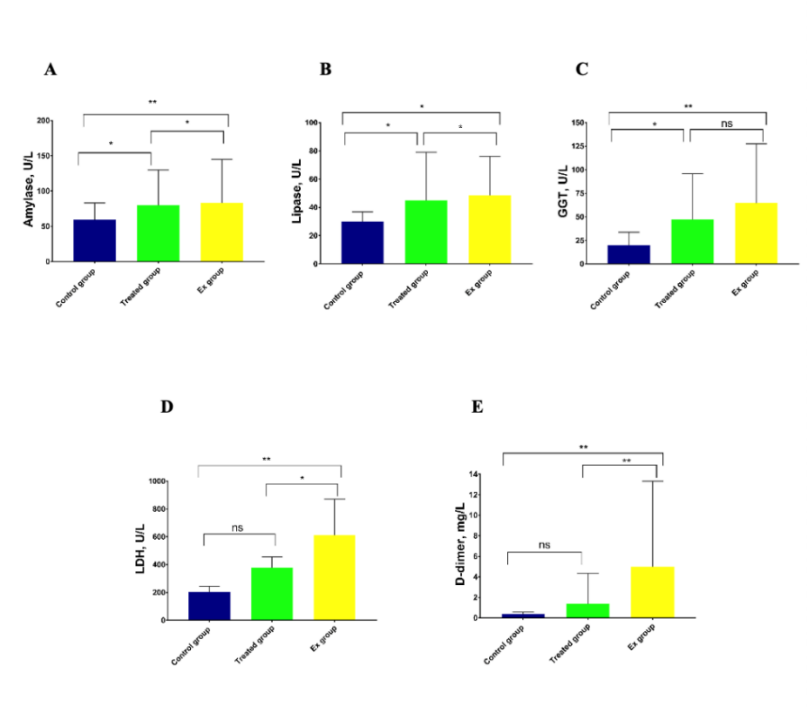

Figure 2: Comparison levels of amylase (A), lipase (B), GGT (C), LDH (D), and D-dimer (E) of control, treated, and ex groups. p values less than .05 were considered significant highlighted in asterisk. ns= not significant

The exitus group was set as the positive group, and the treated group was determined as the negative group. The ROC curve was established to analyze the efficacy of various parameters of blood routine in the diagnosis of severe COVID-19 on admission. We analyzed the optimal cut-off values calculated by the ROC analysis, and the ROC curves are given in Fig. 3. NLR, D-dimer, PCT, and CRP had the highest AUC in the ROC analysis (0.953; 0.943; 0.932; 0.930, respectively). Lipase and WBC had moderate AUC in the ROC analysis. Amylase cannot be used as a potential diagnostic biomarker for subsequent analysis. The AUC value of amylase was close to 0.50. The optimal cut-off values were given in table 3.

Among the Covid 19 cases, serum lipase levels were found to be higher than the upper limit of 60 U/L in 50 of 131 cases, as seen in the table 4. Pancreatitis was not diagnosed in our study, in which hyperlipasemia was detected in 38%.

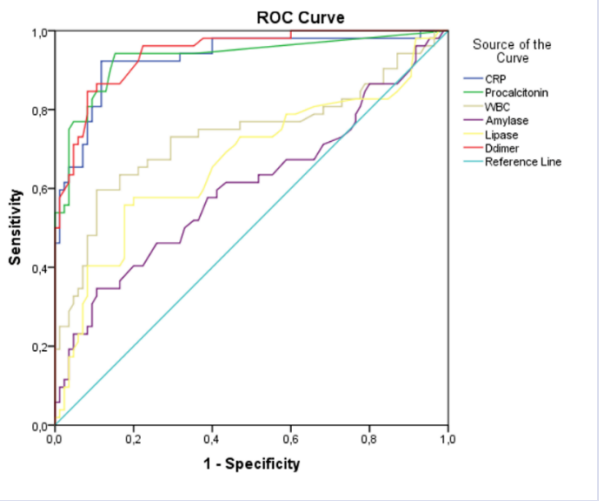

Figure 3: The ROC curves of D-dimer, NLR, amylase, lipase, procalcitonin, WBC, and CRP in predicting severe SARS-CoV-2 infection, on admission. CRP: C reactive protein, NLR: Neutrophils-to-lymphocytes ratio.

Table 1. Demographic, clinical, and radiologic characteristics of the COVID-19 patients.

Variables	Total (n=131)	Treated group (*n* = 61)	Ex group (*n* = 70)	*p* Value
Age, years	65 (62-76)	58.2 (56-72)	60.6 (55-76)	**0.047**
Gender (male/female)	73/58	31/30	42/28	0.159
PCR positive, (%)	101 (77.1)	37 (60.6)	64 (91.4)	**<0.001**
Abnormalities on chest CT	71 (54.2)	32 (52.5)	39 (55.7)	**0.042**
Ground-glass opacity	55 (77.5)	25 (78.1)	30 (76.9)	0.828
Viral pneumonia	16 (22.5)	7 (21.9)	9 (23.1)	0.810
Length of hospital stay, median (IQR)	7 (4-13)	6 (2-11)	8 (5-16)	0.087
Comorbidities	119 (90.8)	53 (86.9)	66 (94.3)	
Systemic hypertension, n, (%)	59 (45.0)	25 (47.2)	34 (51.5)	0.224
Diabetes mellitus, n, (%)	46 (35.1)	20 (37.7)	26 (39.4)	0.230
Congestive hearth disease, n, (%)	13 (9.9)	5 (9.4)	8 (12.1)	0.857
Ischemic hearth disease, n, (%)	4 (3.1)	3 (5.7)	1 (1.5)	0.102
Chronic renal disease, n, (%)	6 (4.6)	2 (3.8)	4 (6.1)	0.884
Cancer, n, (%)	9 (6.9)	3 (5.7)	6 (9.1)	0.856
Cerebrovascular events, n, (%)	9 (6.9)	1 (1.9)	8 (12.1)	0.108
Chronic obstructive pulmonary disease, n, (%)	10 (7.6)	3 (5.7)	7 (10.6)	0.676
Signs and Symptoms				
Fever	49 (37.4)	27 (44.3)	22 (31.4)	**<0.001**
Dry cough	32 (24.4)	18 (29.5)	14 (20.0)	**0.009**
Diarrhea	3 (2.3)	1 (1.6)	2 (2.9)	0.918
Dyspnea	34 (26.0)	13 (21.3)	21 (30.0)	0.779
Weakness	11 (8.4)	7 (11.5)	4 (5.7)	**0.050**

Table 2. Blood routine parameters characteristics of patients group according to treated, exitus and control

Variables	Control group (n=60)	Treated group (*n* = 61)	Exitus group (*n* = 70)	*p* Value	*Pairwise comparison*
Age, years	59 (30-62)	58.2 (56-72)	60.6 (55-76)	0.106[a]	-
Gender, male/female	32/28	30/31	37/33	0.075[b]	-
Albumin, g/L	46 (44-48)	34 (31-38)	30 (27-35)	**<0.001**[c]	GT vs. C, ***p*<0.001** GT vs. GE ***p*<0.001** GE vs. C, ***p*<0.001**
Total protein, g/L	71 (69-74)	58 (54-64)	55 (51-60)	**<0.001**[c]	GT vs. C, *p*<**0.001** GT vs. GE *p* =0.057 GE vs. C, ***p*<0.001**
Amylase, U/L	59 (51-83)	80 (35-130)	83 (50-145)	**<0.001**[c]	GT vs. C, *p* = 0.217 GT vs. GE ***p*=0.049** GE vs. C, ***p*<0.001**
Lipase, U/L	30 (25-37)	45 (27-79)	48 (28-71)	**0.001**[c]	GT vs. C, ***p* = 0.003** GT vs. GE *p*=0.992 GE vs. C, ***p*=0.001**
WBC, x10^9/L	7.7 (6.8-10.4)	6.6 (5.1-8.5)	12.8 (7.5-17.6)	**<0.001**[c]	GT vs. C, *p* = **0.332** GT vs. GE *p*<**0.001** GE vs. C, *p*<**0.001**
N/L	1.9 (1.4-4.7)	3.9 (2.1-7.5)	17.1 (9.5-30.7)	**<0.001**[c]	GT vs. C, *p* = 0.707 GT vs. GE ***p*<0.001** GE vs. C, ***p*<0.001**
Na, mEq/L	140 (138-142)	137 (135-140)	142 (138-148)	**<0.001**[c]	GT vs. C, ***p* = 0.033** GT vs. GE ***p*<0.001** GE vs. C, ***p*=0.002**
K, mEq/L	4.2 (4.0-4.5)	4.0 (3.8-4.4)	4.6 (4.0-5.0)	**<0.001**[c]	GT vs. C, *p* = 0.899 GT vs. GE ***p*=0.003** GE vs. C, ***p*=0.003**
Ca, mEq/L	9.3 (9.0-9.6)	8.6 (8.4-9.2)	7.7 (7.2-8.2)	**<0.001**[c]	GT vs. C, ***p* = 0.033** GT vs. GE ***p*<0.001** GE vs. C, ***p*<0.001**
Hemoglobin, g/dL	13.8 (12.7-14.5)	11.4 (9.9-13.1)	11.0 (9.1-12.6)	**<0.001**[c]	GT vs. C, ***p* <0.001** GT vs. GE *p*=0.422 GE vs. C, ***p*<0.001**
Platelet, x10^9/L	252 (203-308)	279 (190-360)	199 (115-270)	**<0.001**[c]	GT vs. C, *p*=0.330 GT vs. GE ***p*<0.001** GE vs. C, ***p*=0.003**
D-dimer, mg/L	0.4 (0.2-0.6)	1.4 (0.8-4.4)	5 (2-13.1)	**<0.001**[c]	GT vs. C, *p*=0.083 GT vs. GE ***p*<0.001** GE vs. C, ***p*<0.001**
Glucose, mg/dL	100 (85-110)	109 (93-163)	136 (107-195)	**<0.001**[c]	GT vs. C, ***p*=0.019** GT vs. GE *p*=0.195 GE vs. C, ***p*<0.001**
Urea, mg/dL	29 (22-39)	43 (30-58)	100 (68-148)	**<0.001**[c]	GT vs. C, ***p* =0.024** GT vs. GE ***p*=0.001** GE vs. C, ***p*<0.001**
Creatinine, mg/dL	0.77 (0.70-0.92)	0.75 (0.54-1.05)	1.51 (0.97-3.44)	**<0.001**[c]	GT vs. C, ***p* =0.049** GT vs. GE ***p*<0.001** GE vs. C, ***p*<0.001**
CRP, g/L	0.003 (0.001-0.006)	0.003 (0.002-0.04)	0.138 (0.05-0.192)	**<0.001**[c]	GT vs. C, *p* =0.661 GT vs. GE ***p*<0.001** GE vs. C, ***p*<0.001**
PCT, µg/L	0.02 (0.01-0.03)	0.06 (0.01-0.24)	0.98 (0.26-8.89)	**<0.001**[c]	GT vs. C, *p* =0.975 GT vs. GE ***p*=0.028** GE vs. C, ***p*=0.001**
aPTT, sec	23.5 (21.3-24.8)	24.8 (23.2-27.8)	27.2 (23.7-33.3)	**<0.001**[c]	GT vs. C, *p*=0.342 GT vs. GE ***p* =0.032** GE vs. C, *p*=0.716
Prothrombin Time, sec	11.4 (10.7-12.8)	12.7 (11.7-13.5)	14.4 (12.9-16.2)	0.378[c]	GT vs. C, *p*=0.859 GT vs. GE *p*=0.357 GE vs. C, *p*=0.716
AST, U/L	17 (14-22.5)	42 (30-63)	69 (42-143)	**0.014**	GT vs. C, *p*=0.983 GT vs. GE ***p*=0.050** GE vs. C, ***p*=0.035**
ALT, U/L	23 (18-325)	44 (21-74)	45 (22-120)	**0.004**[c]	GT vs. C, *p*=0.897 GT vs. GE ***p*=0.030** GE vs. C, ***p*=0.008**
ALP, U/L	55 (44-59)	81 (63-112)	95 (69-160)	0.108[c]	GT vs. C, *p*=0.368 GT vs. GE *p* =0.409 GE vs. C, *p* = 0.153

GGT, U/L	20 (15-32)	47 (21-95)	65 (35-128)	**<0.001**[c]	GT vs. C, ***p*=0.015** GT vs. GE *p*= 0.416 GE vs. C, ***p*<0.001**
LDH, U/L	201 (174.5-244)	379 (286-456)	611 (467-870)	**<0.001**[c]	GT vs. C, *p*=0.403 GT vs. GE ***p*=0.001** GE vs. C, ***p*<0.001**
Total bilirubin, mg/dL	0.6 (0.4-0.9)	0.5 (0.3-0.8)	0.7 (0.4-1.1)	**0.014**[c]	GT vs. C, *p*= 0.748 GT vs. GE ***p*= 0.118** GE vs. C, ***p*<0.017**
Conjugated bilirubin, mg/dL	0.2 (0.1-0.3)	0.2 (0.1-0.4)	0.4 (0.2-0.7)	**<0.001**[c]	GT vs. C, *p*= 0.193 GT vs. GE ***p*=0.029** GE vs. C, ***p*<0.001**

Descriptive statistics were presented as mean ± standard deviation or median (IQR). T, Treated, E, Exitus and C, control. [a]ANOVA (analysis of variance), [b]chi square test test, [c]Kruskal–Wallis test. Bonferroni correction was used for pairwise comparisons. The significance level was set at $p < 0.05$.

Table 3. The value of blood routine parameters in diagnosis of severely ill patients with COVID-19 on admission.

Variables	Cut-off value	AUC (95% CI)	Sensitivity (%)	Specificity (%)	*p* value
Amylase	>64.5	0.600 (0.497-0.703)	61.5	55.4	**0.052**
Lipase	>31.5	0.666 (0.566-0.765)	73.1	51.8	**0.001**
Procalcitonin	>0.075	0.932 (0.883-0.981)	90.4	85.5	**<0.001**
CRP	>0.0104	0.930 (0.884-0.977)	92.3	88.0	**<0.001**
NLR	>6.825	0.953 (0.919-0.987)	92.3	86.7	**<0.001**
WBC	>8.77	0.736 (0.640-0.831)	73.1	71.1	**<0.001**
D-dimer	>1.35	0.943 (0.907-0.980)	86.5	86.7	**<0.001**

AUC: area under the curve; CRP:C-reactive protein; NLR: Neutrophils-to-lymphocytes ratio. Asymptotic significance less than 0.05 were considered significant.

Tablo 4. Serum lipase levels based on severity of hyperlipasemia (serum lipase upper limit of normal: 60 U/L) among patients hospitalized for COVID-19.

Increased serum lipase n=50/117 (38,1%)	
Serum lipase level, U/L	***n* (% of total COVID-19 group)**
60–120	29 (22,1)
120–180	7 (5,3)
>180	14 (10,6)

Discussion

The SARS-CoV-2 infection causes a wide clinical spectrum that ranges from asymptomatic to severe pneumonia. In severe respiratory failure cases, mechanical ventilation inevitably comes to the agenda in the treatment. The length of stay in the Intensive Care Units and the recovery period increase in such patients [3]. In this process, the evaluation of prognostic factors is important in giving priority to patients who require Intensive Care Unit more. There is no specific treatment for COVID-19 except for infection control and supportive treatment. Multi-organ support treatment is fundamental for the management of critically ill patients with COVID-19. For this reason, determining the prognostic severity criteria is essential to provide early intervention for patients who may require ICU support [3, 4].

The amylase and lipase enzymes are two important enzymes involved in important metabolic events in pancreatic cells. There have been publications on serum amylase and lipase levels, which are biochemical indicators of pancreatic functions since the onset of the Covid-19 pandemic [5-8]. It was argued in these studies that high serum lipase levels are associated with mortality. In our study, we found that serum amylase and lipase levels were elevated. Our findings are compatible with the literature data.

Lipase is synthesized by the pancreas and excreted by the kidneys. Renal failure and diarrhea are two factors contributing to elevated serum lipase values. Increased serum lipase levels can be observed in kidney diseases because of decreased lipase excretion [9]. The renal

functions were within normal limits in the cases included in the study. There is a study in the literature reporting moderate serum lipase elevation without causing acute pancreatitis [10]. Acute pancreatitis did not develop in any of our cases. A total of 71 cases who died from Covid-19 had hyperlipasemia in our study, which makes us think that serum lipase elevation may be a biomarker as a prognostic indicator without causing acute pancreatitis in the early period.

In the study that was conducted by Wang et al., it was hypothesized that hyperamylasemia is not always associated with pancreatic damage. They also reported that all of their 9 COVID-19 patients who had pancreatic damage (increased serum amylase level) had significantly increased creatinine levels when compared to COVID-19 patients without pancreatic damage. Also, one patient who had hyperamylasemia had diarrhea. As explained previously, renal failure and diarrhea are two factors that contribute to elevated serum lipase values.

The mechanisms of coagulopathy in Covid-19 have not yet been elucidated completely. The inflammatory cytokines, lymphocyte cell death, hypoxia, and dysregulated immune responses induced by endothelial damage are presumed to play roles in this respect. Bleeding tendency is rare even in cases with prolonged coagulation tests [11, 12]. D-dimer levels were associated with increased mortality; and low molecular weight heparin was also recommended in previous studies to avoid thromboembolic complications [13, 14]. The elevated d-dimer levels found in our study are consistent with the literature data. Elevated D-dimer affects mortality along with high amylase and lipase levels.

There are some publications in which the optimum cut-off value of some serum biochemical parameters was identified by using severe disease ROC curve as a prognosis indicator in Covid-19 cases [4]. In this respect, %LUC levels can be used as independent prognostic indicators. In our study, D-dimer, LDH, NLR, procalcitonin, and WBC had the highest AUC in the ROC analysis. The AUC values of amylase and lipase, which show pancreatic functions, had elevated values to show the severity of Covid-19 infection. According to the ROC Analysis, the lipase levels of these tests were at significant levels to indicate the prognosis. The ROC Analysis shows us that hyperlipasemia can be significant in showing the prognosis in Covid-19 cases.

Conclusion

The elevation in serum amylase, lipase, and d-dimer levels may be significant in prognosis in pneumonia developing due to Covid-19.

Limitations

Our study population is small. Could it cause worsening of Covid-19 pneumonia accompanied by undiagnosed chronic pancreas disorders? Does it have a role in etiopathogenesis? Extensive studies may be required.

The authors declare that there is no conflict of interest between them.

References

1. Oran DP, Topol EJ. Prevalence of Asymptomatic SARS-CoV-2 Infection : A Narrative Review. Ann Intern Med. 2020 Sep 1;173(5):362-367. doi: 10.7326/M20-3012.
2. 2.https://www.who.int/emergencies/diseases/novel-coronavirus-2019.
3. Lauer SA, Grantz KH, Bi Q, Jones FK, Zheng Q, Meredith HR, et al. The Incubation Period of Coronavirus Disease 2019 (COVID-19) From Publicly Reported Confirmed Cases: Estimation and Application. Ann Intern Med. 2020 May 5;172(9):577-582. doi: 10.7326/M20-0504.
4. Bastug A, Bodur H, Erdogan S, Gokcinar D, Kazancioglu S, Kosovali BD, et al., Clinical and laboratory features of COVID-19: Predictors of severe prognosis. Int Immunopharmacol, 2020. 88: p. 106950.
5. Ahmed A, Fisher JC, Pochapin MB, Freedman SD, Kothari DJ, Shah PC,et al. Hyperlipasemia in absence of acute pancreatitis is associated with elevated D-dimer and adverse outcomes in COVID-19 disease. Pancreatology. 2021 Jun;21(4):698-703. doi: 10.1016/j.pan.2021.02.021.
6. McNabb-Baltar J, Jin DX, Grover AS, Redd WD, Zhou JC, Hathorn KE et al. Lipase Elevation in Patients With COVID-19. Am J Gastroenterol. 2020 Aug;115(8):1286-1288. doi: 10.14309/ajg.0000000000000732.
7. De-Madaria E, Siau K, Cárdenas-Jaén K. Increased Amylase and Lipase in Patients With COVID-19 Pneumonia: Don't Blame the Pancreas Just Yet! Gastroenterology. 2021 Apr;160(5):1871. doi: 10.1053/j.gastro.2020.04.044. ,
8. Wang F, Wang H, Fan J, Zhang Y, Wang H, Zhao Q. Pancreatic Injury Patterns in Patients With Coronavirus Disease 19 Pneumonia. Gastroenterology. 2020 Jul;159(1):367-370. doi: 10.1053/j.gastro.2020.03.055.
9. Pribadi RR, Simadibrata M. Increased serum amylase and/or lipase in coronavirus disease 2019 (COVID-19) patients: Is it really pancreatic injury? JGH Open. 2020 Dec 28;5(2):190-192. doi: 10.1002/jgh3.12436.
10. McNabb-Baltar J, Jin DX, Grover AS, Redd WD, Zhou JC,, et al. Lipase Elevation in Patients With COVID-19. Am J Gastroenterol. 2020 Aug;115(8):1286-1288. doi: 10.14309/ajg.0000000000000732.
11. Iba T, Levy JH, Levi M, Thachil J. Coagulopathy in COVID-19. J Thromb Haemost, 2020. 18 (9):p.2103-2109.
12. Giannis, D., I.A. Ziogas, P. Gianni, Coagulation disorders in coronavirus infected patients: COVID-19, SARS-CoV-1, MERS-CoV and lessons from the past. J Clin Virol, 2020. 127: p. 104362.
13. Di Micco P, Russo V, Carannante N, Imparato M, Rodolfi S, Cardillo G, et al., Clotting Factors in COVID-19: Epidemiological Association and Prognostic Values in Different Clinical Presentations in an Italian Cohort. J Clin Med, 2020. 9(5).

14. Tang N, Bai H, Chen X, Gong J, Li D, Sun Z. Anticoagulant treatment is associated with decreased mortality in severe coronavirus disease 2019 patients with coagulopathy. J Thromb Haemost, 2020. 18(5): p. 1094-1099.