DOI: 10.37990/medr.1729590 Med Records 2025;7(3):683-90

MEDICAL RECORDS-International Medical Journal

Research Article

Evaluation of Pancreatic Injury in COVID-19 Patients: A Single Center Retrospective Study

Aycan Acet[1], Cagla Ozdemir[2], Cumali Yalcin[3], Hasan Col[2], Turkan Pasali Kilit[1], Sertas Erarslan[1], Can Ozlu[3], Borahan Bora[1]

[1]Kütahya Health Sciences University Faculty of Medicine, Departmant of Internal Medicine, Kütahya, Türkiye
[2]Kütahya Health Sciences University Faculty of Medicine, Departmant of Family Medicine, Kütahya, Türkiye
[3]Kütahya Health Sciences University Faculty of Medicine, Departmant of Internal Medicine, Division of Hematology, Kütahya, Türkiye

Abstract

Aim: Pancreatic injury has become a significant complication linked to COVID-19 infection. Our study's aim was to compare the clinical and laboratory findings, intensive care requirements, and mortality rates between patients with and without pancreatic enzyme elevation among those diagnosed with COVID-19.

Material and Method: The retrospective investigation was carried out at a tertiary hospital and involved PCR-confirmed COVID-19 patients. Data from 457 participants was collected, and 376 eligible participants were analyzed using statistical methods. Patients with amylase or lipase levels up to three times normal were classified as having pancreatic injury, while those with normal levels were the control group. Mann-Whitney U test was utilized for group comparisons. Factors predicting intensive care unit (ICU) admission were identified using logistic regression analysis; $p<0.05$ is considered statistically significant.

Results: The study involved 376 participants (50 with pancreatic injury and 326 without). Patients with pancreatic injury experienced longer durations of antibiotic therapy ($p=0.009$), extended hospital stays ($p=0.018$), and higher rates of ICU admissions within the first week ($p=0.045$). Biochemically, levels of creatinine ($p=0.001$), white blood cell ($p<0.001$), and neutrophils ($p<0.001$) were significantly elevated, whereas the estimated glomerular filtration rate ($p=0.003$) was decreased in the pancreatic injury group. Survival rates did not differ significantly between the two groups.

Conclusion: Elevated pancreatic enzyme levels up to three times the upper limit of normal did not significantly impact all-cause mortality, but were linked to higher rates of inflammatory markers and ICU admission in the first week of hospitalization.

Keywords: COVID-19, pancreatic enzyme elevations, amylase, lipase

INTRODUCTION

The COVID-19 pandemic has impacted people's health, the economy, and societal structure. Understanding the complex nature of this virus, its transmission, and its impacts on different populations is crucial for effective management and prevention strategies. COVID-19 usually presents with respiratory symptoms and, in some cases, can progress to severe pneumonia, leading to acute respiratory distress syndrome (ARDS) (1). The clinical spectrum of coronavirus is broad, encompassing both asymptomatic cases and severe illnesses that require intensive care. Besides respiratory symptoms, gastrointestinal symptoms like nausea, with or without vomiting, have been noted in some patients, although they are less common (2). The disease has also been connected to various orofacial manifestations, highlighting the diverse clinical presentations that can occur (3). Acute pancreatitis has emerged as a notable complication associated with COVID-19 infection, with various studies emphasizing its potential mechanisms and prevalence among affected patients. COVID-19-related acute pancreatitis may result from direct and indirect effects of the virus on the pancreas. The existence of angiotensin-converting enzyme 2 (ACE2) receptors in pancreatic cells suggests that SARS-CoV-2 can directly infect these cells, leading to inflammation and damage (4-7). The inflammatory response caused by COVID-19, marked by a cytokine storm, can aggravate pancreatic damage through mechanisms like vasculitis

CITATION
Acet A, Ozdemir C, Yalcin C, et al. Evaluation of Pancreatic Injury in COVID-19 Patients: A Single Center Retrospective Study. Med Records. 2025;7(3):683-90. DOI:1037990/medr.1729590

Received: 28.06.2025 **Accepted:** 12.08.2025 **Published:** 09.09.2025
Corresponding Author: Aycan Acet, Kütahya Health Sciences University Faculty of Medicine, Departmant of Internal Medicine, Kütahya, Türkiye
E-mail: aycanacet80@icloud.com

and microthrombosis (8,9). It is still uncertain whether the rise in pancreatic enzymes is directly related to disease severity or if it indicates a greater risk of death. Our research aims to investigate how elevated pancreatic enzymes relate to the outcomes of COVID-19 patients.

MATERIAL AND METHOD

Ethical approval for this study was granted by the Kütahya Health Sciences University Faculty of Medicine Local Ethics Committee (Decision No. 2023/13-06; Date: 28 November 2023).

Study Design

From January 1 to December 31, 2021, we retrospectively evaluated our study at a tertiary education and research hospital. In our study, we analyzed patient data, such as demographics, medical history, imaging results, laboratory results, treatment plans, and outcomes.

Patient Selection

Data from 457 patients in internal medicine and intensive care unit (ICU) were reviewed. Patients with insufficient data and those without a confirmed COVID-19 diagnosis by PCR testing weren't included in the research. In fulfillment of the inclusion requirements outlined below, the study involved 376 patients (Figure 1).

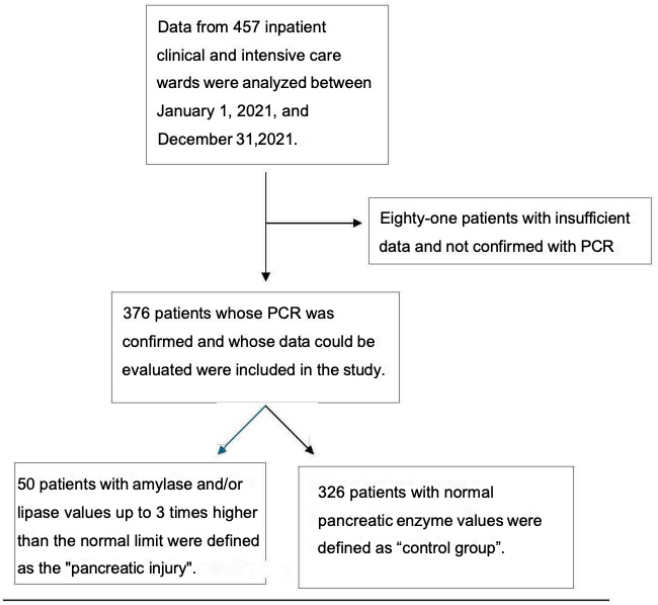

Figure 1. The flowchart illustrating the study design provides an overview of the patient selection process

It was planned to include patients: 1. Whose records were accessed through the automated system, 2. Who were older than 18, 3. Who were not diagnosed with acute pancreatitis for any other reason (e.g., alcohol, gallstones, hyperlipidemia) during their stay in the hospital, 4. Who were monitored in the inpatient intensive care unit and internal medicine wards with a diagnosis of COVID-19 according to the Turkish COVID-19 Adult Patient Treatment Guide of the Ministry of Health (10). A positive PCR result from the patient's nasopharyngeal swab samples was used to confirm a COVID-19 diagnosis. The patient was admitted to the hospital.

ICU Admission Criteria

Patients meeting intensive care criteria upon admission, such as those with severe respiratory failure, hemodynamic irregularity, change in mental status, multiorgan failure, rapidly progressing radiological findings, elevated inflammatory markers, and persistent clinical deterioration despite optimal medical treatment, were transferred to the ICU for supportive treatment and follow-up. Those not requiring ICU criteria were hospitalized in the clinics; however, patients developing intensive care indications during treatment were transferred to the ICU. The criteria were evaluated according to each patient's condition and determined by the physician.

ICU admission criteria for COVID-19 pneumonia patients included:

- Severe respiratory failure (oxygen saturation [SpO_2] <90% in patients receiving high-flow oxygen therapy or with a rate of respiration greater than 30 breaths per minute),
- Hemodynamic abnormality (need for vasopressors or systolic blood pressure below 90 mmHg),
- Changes in mental state (Glasgow Coma Scale score <13),
- Multiorgan failure (elevated creatinine, aspartate aminotransferase/alanine aminotransferase (AST/ALT), or lactate levels >2 mmol/L),
- Rapidly progressing radiological findings (e.g., bilateral pulmonary infiltrates),
- Elevated inflammatory markers (e.g., White blood cell, D-dimer, C-reactive protein) and persistent clinical deterioration despite optimal medical treatment.

Definition of COVID-19 Severity

COVID-19 severity was categorized based on NIH Guidelines as follows: mild=1, moderate=2, severe=3, and critical=4 (National Institutes of Health [NIH], 2021). The probability of pneumonia was evaluated based on the CO-RADS criteria (11,12).

Data Collection

The medical history, demographic information, clinical findings, laboratory data, and observation results for the patients were gathered using the electronic system. Follow-up and outcome information were also recorded in this manner.

Pancreatic Injury and Pancreatitis Diagnosis

The diagnosis of pancreatitis was made using the 2012 Modified Atlanta criteria, which define The diagnosis of pancreatitis was made using the 2012 Modified Atlanta criteria, which define what constitutes 'pancreatic injury' as amylase and/or lipase levels elevated to three times greater than the normal limit (13). The range of laboratory

levels of serum amylase measured in the local laboratory was accepted as (22-80 U/L), and the lipase value was accepted as (0-67 U/L). Patients with normal pancreatic enzymes were planned to be separated as the control group. Of the 376 patients evaluated, 50 were classified as the 'pancreatic injury group' and 326 as the control group. Clinical, demographic, and laboratory characteristics of the two groups were used to compare them.

Statistical Analysis

All data analyses were performed using SPSS version 27 (IBM®, Chicago, USA). The normality of continuous variables was assessed with the Shapiro-Wilk test and visual inspection of histograms. For continuous variables, data with a normal distribution were summarized using the mean±standard deviation (SD), while data without a normal distribution were summarized using the median (minimum–maximum). The pancreatic injury and control groups were compared using the Chi-square test for categorical variables and the Mann-Whitney U test for nonparametric continuous variables. For the comparison of proportions, the Pearson chi-square test was applied when all expected cell counts exceeded 5, and Fisher's exact test was used when any expected count was less than 5. To identify independent predictors of ICU admission among patients with pancreatic injury, univariate and multivariate logistic regression analyses were performed. The multivariate model included variables that were significant in univariate analysis, such as age, creatinine, eGFR (estimated glomerular filtration rate), CRP (C-reactive protein), AST, uric acid, amylase, lipase, white blood cell (WBC) count, D-dimer, and neutrophil count. The results of the logistic regression are presented as odds ratios (OR) with their 95% confidence intervals (CIs). Significance was set at $p<0.05$ for all analyses.

RESULTS

The study involved 376 participants (50/326). Table 1 presents the demographic data distribution and the clinical parameters of the participants. The mean age was comparable between those with pancreatic injury (M=68.40, SD=15.49) and those without (M=64.94, SD=15.60). Male gender was significantly more prevalent among those with pancreatic injury (X2(1)=4.902, p=0.027). The duration of antibiotherapy (p=0.009), length of hospital stay (p=0.018), and requirement for intensive care at week 1 (p=0.045) were significantly higher in the pancreatic injury group (Figure 2). In terms of disease severity, the control group and the pancreatic injury group were not significantly different; however, the pancreatic injury group had a higher rates of severe disease. Figures 3 and 4 show the correlation between the levels of amylase and lipase and the severity of illness in patients admitted to the intensive care unit within the first fourteen days of illness. Furthermore, there were no variations in all-cause mortality or comorbidity between the groups.

Table 1. Comparison of sociodemographic and clinical characteristics between groups (n=376)

	All patients (n=376)	Pankreatic injury (+) (n=50)	Pankreatic injury (-) (n=326)	p value
Age, year[1]	65.40±15.64	68.40±15.49	64.94±15.60	0.110
Gender, male[2]	201 (53.5)	34 (68)	167 (51.2)	0.027*
Comorbidities (+)[2]				
Hypertension	212 (56.5)	32 (64)	180 (55.2)	0.253
Diabetes mellitus	144 (38.4)	16 (32)	128 (39.2)	0.318
COPD	66 (17.6)	8 (16)	58 (17.8)	0.750
Coronary artery disease	62 (16.5)	6 (12)	56 (17.1)	0.354
Congestive heart failure	29 (7.7)	6 (12)	23 (7.1)	0.225
Chronic kidney disease	28 (10.3)	7(14)	21 (6.4)	0.130
Malignancy	46 (12.3)	8(16)	38 (11.6)	0.387
Severity of disease[2]				0.157
Mild	112 (29.9)	18 (36)	94 (28.9)	
Moderate	139 (37.1)	12 (24)	127 (39.1)	
Severe	118(31.3)	18 (36)	100 (30.6)	
Critical	7 (1.9)	2 (4)	5 (1.5)	
Duration of antibiotherapy[1]	12.39±7.31	15.20±8.56	11.95±7	0.009*
Length of hospitalization, days[1]	14.72±8.01	17.40±9	14.29±7.78	0.018*
Need for intensive care (first seven days)[2]	176 (46.8)	30 (60)	146 (44.8)	0.045*
Mortality[2]	109 (30.2)	16 (32)	93 (29.9)	0.764

[1]Mean±sd, Mann Whitney U test; [2]n (%), Pearson Chi-square test; COPD: chronic obstructive pulmonary disease; * p<0.05

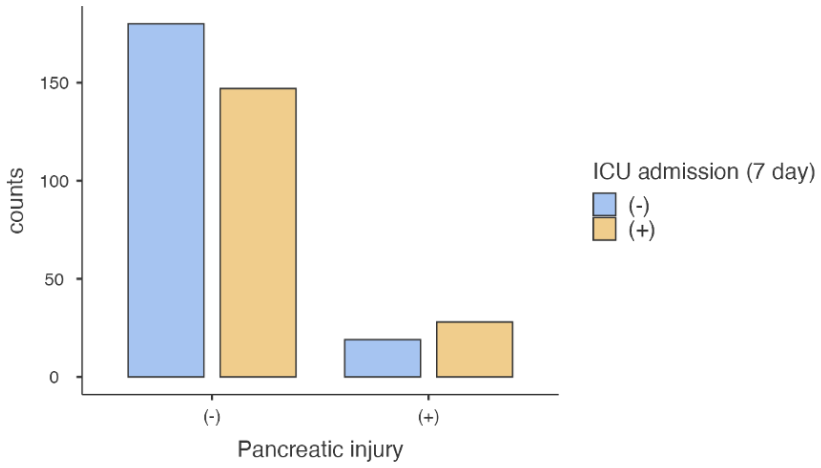

Figure 2. Distribution of ICU admission in the first 7 days between groups with and without pancreatic injury; ICU: Intensive care unit

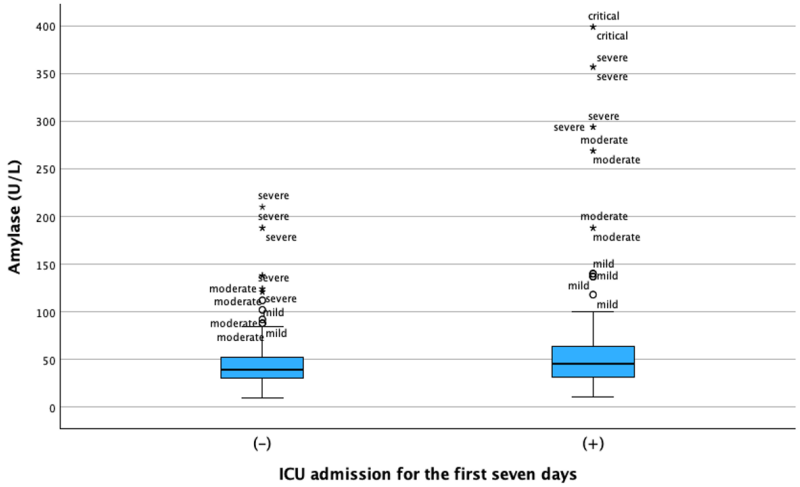

Figure 3. Association between severity of illness and amylase levels in patients admitted to intensive care unit within the first week of hospitalization

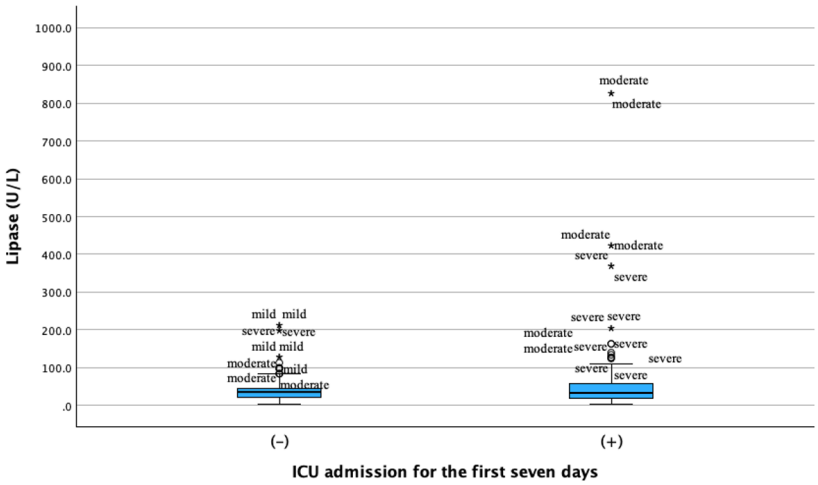

Figure 4. Association between severity of illness and lipase levels in patients admitted to intensive care unit within the first week of hospitalization

The Mann-Whitney U test was employed to evaluate the biochemical values of the groups categorized by pancreatic damage in Table 2. The group with pancreatic injury had considerably greater levels of creatinine (p=0.001), white blood cells (WBC) (p<0.001), and neutrophils (p<0.001), while the group without pancreatic injury had significantly lower levels of estimated glomerular filtration rate (eGFR) (p=0.003). There were not significant variations between the groups in uric acid, CRP, D-dimer, or lymphocyte count. Patients without pancreatic damage had significantly higher AST values (p=0.025).

Table 2. Comparison of biochemical parameters between groups (n=376)				
	All patients (n=376)	Pankreatic injury (+) (n=50)	Pankreatic injury (-) (n=326)	p value
Creatinine (mg/dL)	1.42±1.27	2.43±2.15	1.27±1.01	0.001*
eGFR (mL/min/1.73m^2)	64.07±31.17	53.84±47.25	65.64±27.68	0.003*
ALT (U/L)	34.44±33.23	35.12±31.29	33.57±33.48	0.061
AST (U/L)	47.82±157.97	45.36±31.19	48.19±169.28	0.025*
Uric acid (mg/dL)	6.27±3.19	6.55±2.51	6.21±3.32	0.185
WBC (x10^3/μL)	8914±16497	10730±4051	8631±17651	<0.001*
Neutrophil count (x10^3/μL)	6499±4570	9151±3901	6090±4533	<0.001*
Lymphocyte count (x10^3/μL)	1900±15164	1118±762	2021±16296	0.845
C- Reactive Protein (mg/L)	92.15±77.69	82.28±64.36	93.68±79.53	0.597
D-dimer (ng/mL)	1487±1319	1654±1118	1461±1348	0.076

Mean±sd, Mann Whitney U test; eGFR: estimated glomerular filtration rate, ALT: alanine aminotransferase, AST: aspartat aminotransferase, WBC: white blood cell; *p<0.05

A comparison of the biochemical and clinical features of pancreatic injury patients who were admitted to the ICU and those who were not was conducted using the Mann-Whitney U test along with the Chi-Square Test, as shown in Table 3. Intensive care unit patients had significantly higher WBC (p=0.014), D-dimer (p=0.015), and neutrophil count (p<0.001).

Table 3. Comparison of biochemical and clinical characteristics in patients with pancreatic injury, grouped by the need for intensive care in the first week (n=50)			
	ICU (+) (n=30)	ICU (-) (n=20)	p value
Age, year[1]	71.87±13.88	63.20±16.65	0.095
Gender, male[2]	22 (73.3)	12 (60)	0.322
Disease severity[2]	21(70)	10 (65)	0.859
Creatinine (mg/dL)[1]	2.97±2.65	1.79±1.09	0.777
eGFR (mL/min/1.73m^2)[1]	53.46±56.17	54.40±30.67	0.234
ALT (U/L)[1]	33.07±21.60	43.20±41.81	0.721
AST (U/L)[1]	43.47±29.21	48.20±34.54	0.552
Uric acid (mg/dL)[1]	6.72±2.32	6.37±2.76	0.189
Amilase (U/L)[1]	173.27±104.18	126.30±40.89	0.191
Lipase (U/L)[1]	175.66±214.15	101.40±60.51	0.634
WBC (x10^3/μL)[1]	11982±4002	8852±3419	0.014*
Neutrophil count (x10^3/μL)[1]	10659±3917	6889±2619	<0.001*
Lymphocyte count (x10^3/μL)[1]	921±575	1413±916	0.113
C-reactive protein (mg/L)[1]	81.40±60.39	83.60±71.50	0.663
D-dimer (ng/mL)[1]	1792±1302	1235±1281	0.015*

[1]Mean±sd, Mann Whitney U test; [2]n (%), Chi-square; eGFR: estimated glomerular filtration rate, ALT: alanine aminotransferase, AST: aspartat aminotransferase, WBC: white blood cell; * p<0.05

Table 4 represents the results of univariate and multivariate logistic regression analyses evaluating factors associated with the need for intensive care during the first week of hospitalization in patients with pancreatic injury. In the univariate analysis, older age (OR=1.035, 95% CI: 1.021–1.050, p<0.01), elevated creatinine (OR=1.281, 95% CI: 1.056–1.555, p=0.012), lower eGFR (OR=0.993, 95% CI: 0.987–1.000, p=0.046), higher AST (OR=1.009, 95% CI: 1.002–1.016, p=0.015), uric acid (OR=1.098, 95% CI: 1.001–1.204, p=0.048), amylase (OR=1.006, 95% CI: 1.002–1.011, p=0.008), lipase (OR=1.005, 95% CI: 1.001–1.010, p=0.022), neutrophil count (p<0.001), C-reactive protein (OR=1.005, 95% CI: 1.002–1.007, p=0.001), and D-dimer (p<0.001) were significantly associated with the requirement for intensive care during the first week of hospitalization in patients with pancreatic injury. Age (OR=1.044, 95% CI: 1.016–1.074, p=0.002), amylase (OR=0.992, 95% CI: 0.984–0.999, p=0.033), lipase (OR=1.008, 95% CI: 1.001–1.015, p=0.033), and neutrophil count (p=0.018) were independent predictors in the multivariate analysis.

Table 4. Univariate and multivariate logistic regression analysis of patients with pancreatic injury requiring intensive care during the first week of hospitalization (n=50)

	Univariate			Multivariate		
	OR	CI 95%	p	OR	CI 95%	p
Age, year	1.035	1.021-1.050	<0.01*	1.044	1.016-1.074	0.002*
Gender, male	0.743	0.494-1.116	0.152			
Disease severity	0.986	0.593-1.642	0.958			
Creatinine (mg/dL)	1.281	1.056-1.555	0.012*	1.317	0.934-1.858	0.116
eGFR (mL/min/1.73m²)	0.993	0.987-1.000	0.046*	1.019	1.001-1.037	0.059
ALT (U/L)	1.002	0.996-1.009	0.452			
AST (U/L)	1.009	1.002-1.016	0.015*	1.001	0.996-1.006	0.674
Uric acid (mg/dL)	1.098	1.001-1.204	0.048*	1.067	0.946-1.204	0.293
Amylase (U/L)	1.006	1.002-1.011	0.008*	0.992	0.984-0.999	0.033*
Lipase (U/L)	1.005	1.001-1.010	0.022*	1.008	1.001-1.015	0.033*
WBC (x10³/μL)	1.000	1.000-1.000	<0.001*	1.000	1.000-1.000	0.659
Neutrophil count (x10³/μL)	1.000	1.000-1.000	<0.001*	1.000	1.000-1.000	0.018*
Lymphocyte count (x10³/μL)	1.000	1.000-1.000	0.505			
C-reactive protein (mg/L)	1.005	1.002-1.007	0.001*	1.003	0.998-1.007	0.234
D-dimer (ng/mL)	1.000	1.000-1.001	<0.001*	1.000	1.000-1.000	0.869
-2 Log likelihood: 247.175, Hosmer and Lemeshow Test: p=0.902						

*p<0.05; eGFR: estimated glomerular filtration rate, ALT: alanine aminotransferase, AST: aspartate aminotransferase, WBC: white blood count

DISCUSSION

Aside from respiratory problems, the COVID-19 pandemic has resulted in significant increases in pancreatic enzymes like lipase and amylase. In our study, the term 'pancreatic injury' was deliberately defined more broadly than acute pancreatitis, encompassing cases with elevated amylase and/or lipase levels up to three times the upper limit of normal, regardless of the presence of clinical or radiological features of pancreatitis. This approach recognizes that such enzyme elevations can occur without meeting the diagnostic criteria for acute pancreatitis and may instead reflect systemic inflammation or other extra-pancreatic processes. These rises are linked to various levels of pancreatic damage, which may manifest as acute pancreatitis or hyperenzymemia without obvious clinical symptoms. The pathophysiology behind these enzyme elevations may have multiple causes, including direct viral effects, systemic inflammatory responses, potential microvascular injury complications, and drug-induced effects (14-16).

ACE-2 receptors in pancreatic tissue are key to understanding COVID-19-associated pancreatic injury. These receptors enable SARS-CoV-2 to enter host cells, and their presence in pancreatic cells can cause direct damage, leading to increased amylase and lipase levels in the blood (14). As evidenced by elevated C-reactive protein and other inflammatory markers like interleukin-6, systemic inflammation exacerbates pancreatic damage (15,16).

According to our findings, patients with elevated pancreatic enzymes had a significantly longer hospital stay, required more antibiotics, and required intensive care during the first week. Secondary infections in this patient group may be caused by mechanisms such as an intense inflammatory response and immunosuppression. Additionally, prolonged antibiotic use may lead to the colonization of resistant bacteria and decreased antibiotic efficacy. This situation might also lead to an increase the risk of infection exposure in COVID-19 patients, raising treatment costs and creating an added burden on their morbidity.

In epidemiological studies, increased levels of pancreatic enzymes were found to be highly associated with COVID-19 severity. A study with 1,249 patients found that 2.96% showed elevated amylase and lipase levels, with a higher occurrence in severe cases compared to mild infections. (17). Similarly, in our study, 4.3% of patients developed hyperlipasemia, 12.2% developed hyperamylasemia, and 3.2% developed both hyperamylasemia and hyperlipasemia. Research indicates that up to 17% of patients with severe COVID-19 may have abnormal enzyme levels, which are indicative of pancreatic injury. (18,19). We reported 17.5% of pancreatic injuries in moderate, severe, and critical cases. This indicates that monitoring pancreatic enzyme levels could be an important prognostic tool in managing COVID-19 patients.

The elevation of lipase and amylase is not limited to pancreatitis and, in many cases, does not indicate direct pancreatic injury at all. Our study therefore emphasizes that enzyme elevation, as defined in our broader 'pancreatic injury' category, can also serve as a surrogate marker of systemic inflammation in COVID-19 patients. Other conditions, such as renal failure, liver disease, and gastrointestinal disorders, can also cause increased levels of these enzymes (20). It is essential to decide

if the hyperlipasemia and/or hyperamylasemia seen in COVID-19 patients indicates significant pancreatic damage or merely reflects systemic illnesses. Nearly fifty percent of those with elevated levels of lipase did not fit the criteria for acute pancreatitis, according to a study, which suggests that many cases of hyperlipasemia may have non-pancreatic causes (21,22). The severity of the illness and tissue and organ ischemia may both have an important role in the increase in pancreatic enzyme levels. Due to its complexity, diagnosing pancreatitis in COVID-19 requires careful clinical evaluation and imaging tests for confirmation.

The clinical presentation of COVID-19-related pancreatic injury can vary significantly. Some patients may display classic signs of acute pancreatitis, such as abdominal pain and nausea, while others might only show mild increases in pancreatic enzymes without notable clinical symptoms (23,24). In a cohort study, only 7.5% of patients with elevated lipase levels had imaging-confirmed pancreatitis, highlighting the need for a comprehensive diagnostic approach (25,26). The lack of a distinct connection between enzyme levels and clinical symptoms makes managing these patients more difficult because elevated enzyme levels do not always signify acute pancreatitis.

An unexpected finding in our study was that AST levels were significantly higher in the group without pancreatic injury compared to the group with pancreatic injury (p=0.025, Table 2). This observation appears to contrast with the general trend in our results, where the pancreatic injury group demonstrated more pronounced systemic inflammation and organ dysfunction markers. One possible explanation is that in the non-pancreatic injury group, AST elevation may have been driven by alternative mechanisms, such as concomitant hepatic injury due to viral hepatitis, drug-induced liver injury, ischemic hepatitis secondary to hypoxia, or muscle injury (given AST's non-specific tissue distribution). Additionally, pre-existing chronic liver disease or other comorbid conditions not evenly distributed between groups could have contributed to this difference.

Despite the fact that the two groups' differences in D-dimer levels were not statistically significant, we found that the group with pancreatic injury tended to have higher D-dimer levels. Our analysis also found that the main reasons for the pancreatic injury group being admitted to the intensive care unit in the first week were higher leukocyte counts and D-dimer levels. This elevation in D-dimer levels among patients with pancreatic injury suggests a potential link to systemic coagulation alterations, which may complicate their clinical course. Since elevated D-dimer levels are indicative of the underlying inflammatory and coagulopathic processes that can occur during critical illness, they have been linked to poor outcomes in acute pancreatitis (27). Furthermore, comorbidities like diabetes mellitus may worsen these complications, increasing the risk of severe pancreatic damage and subsequent ICU admissions. (28). It's crucial for clinicians to grasp these connections, as tracking D-dimer levels can help predict pancreatic injury severity and guide treatments to improve patient outcomes.

Our study has some weaknesses. The retrospective design complicates causal clarification and limits result generalizability. Also, we can't say for sure that the data are due to SARS-CoV-2 effects because there wasn't a control group to compare the results to people who didn't have COVID-19. However, because the patients' full clinical findings and imaging results were not completely available, verifying the acute pancreatitis diagnosis was difficult. Finally, the insufficient number of patients for subgroup analyses concerning various comorbidities prevents a comprehensive understanding of the clinical course of enzyme elevation in these patients.

CONCLUSION

The complex nature of elevated amylase and lipase in COVID-19 patients necessitates additional research to fully understand its implications. Understanding the connection between direct viral effects, systemic inflammation, and comorbidities makes it challenging to interpret these enzyme levels. Clinicians should monitor pancreatic enzyme levels in COVID-19 patients closely, as they may signal underlying pancreatic damage or systemic illness. Future research should address the underlying processes that cause these enzyme elevations and how they affect patient care outcomes.

Financial disclosures: *The authors declared that this study has received no financial support.*

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

Ethical approval: *The study received approval from the local ethics committee at Kütahya Health Sciences University (Approval No. 2023/13-06; Date: 28 November 2023).*

REFERENCES

1. Mofijur M, Fattah IMR, Alam MA, et al. Impact of COVID-19 on the social, economic, environmental and energy domains: Lessons learnt from a global pandemic. Sustain Prod Consum. 2020;26:343-59.
2. Pinzon RT, Angela, Pradana AW. Vitamin D deficiency among patients with COVID-19: case series and recent literature review. Trop Med Health. 2020;48:102.
3. Halboub E, Al-Maweri SA, Alanazi RH, et al. Orofacial manifestations of COVID-19: a brief review of the published literature. Braz Oral Res. 2020;34:e124.
4. Fiore V, Beretta R, De Vito A, et al. Emerging clinical features of COVID-19 related pancreatitis: case reports and review of the literature. Front Med (Lausanne). 2022;8:779118.
5. Simou EM, Louardi M, Khaoury I, et al. Coronavirus disease-19 (COVID-19) associated with acute pancreatitis: case report. Pan Afr Med J. 2020;37:150.
6. Kataria S, Sharif A, Ur Rehman A, et al. COVID-19 induced acute pancreatitis: a case report and literature review. Cureus. 2020;12:e9169.

7. Ayyıldız A, Çınaroğlu A, Yelken B. Acalculous acute pancreatitis in COVID-19 patient; a rare case report. Acta Med Nicomedia. 2022;5:32-5.
8. Kumaran NK, Karmakar BK, Taylor OM. Coronavirus disease-19 (COVID-19) associated with acute necrotising pancreatitis (ANP). BMJ Case Rep. 2020;13:e237903.
9. Chan WF, Toh YN. Acute necrotizing pancreatitis complicated by multiple splanchnic venous thromboses and bilateral renal infarctions in a patient with recent COVID-19 infection: a case report. Cureus. 2022;14:e28049.
10. COVID-19 Salgın Yönetimi ve Çalışma Rehberi. https://covid19.saglik.gov.tr/TR-66393/covid-19-salgin-yonetimi-ve-calisma-rehberi.html access date 16.03.2025.
11. Coronavirus Disease 2019 (COVID-19) Treatment Guidelines [Internet]. Bethesda (MD): National Institutes of Health (US); 2021 Apr 21-2024 Mar 1. https://www.ncbi.nlm.nih.gov/books/NBK570371/ access date 16.03.2025.
12. Prokop M, van Everdingen W, van Rees Vellinga T, et al. CO-RADS: a categorical CT assessment scheme for patients suspected of having COVID-19-definition and evaluation. Radiology. 2020;296:E97-104.
13. Banks PA, Bollen TL, Dervenis C, et al. Classification of acute pancreatitis--2012: revision of the Atlanta classification and definitions by international consensus. Gut. 2013;62:102-11.
14. Liu F, Long X, Zhang B, et al. ACE2 Expression in pancreas may cause pancreatic damage after SARS-CoV-2 infection. Clin Gastroenterol Hepatol. 2020;18:2128-30.e2.
15. Stevens JP, Brownell JN, Freeman AJ, Bashaw H. COVID-19-associated multisystem inflammatory syndrome in children presenting as acute pancreatitis. J Pediatr Gastroenterol Nutr. 2020;71:669-71.
16. Bacaksız F, Ebik B, Ekin N, Kılıç J. Pancreatic damage in COVID-19: Why? How?. Int J Clin Pract. 2021;75:e14692.
17. Alkan S, Gökçe NO, Çinpolat H. The frequency of pancreatic enzyme elevations and effect on disease severity in COVID-19. Sağlık Bilimlerinde Değer. 2023;13:209-15.
18. Grusova G, Bruha R, Bircakova B, et al. Pancreatic injury in patients with SARS-Cov-2 (COVID-19) infection: a retrospective analysis of CT findings. Gastroenterol Res Pract. 2021;2021:5390337.
19. Aloysius MM, Thatti A, Gupta A, et al. COVID-19 presenting as acute pancreatitis. Pancreatology. 2020;20:1026-7.
20. Benias PC, Inamdar S, Wee D, et al. Analysis of outcomes in COVID-19 patients with varying degrees of hyperlipasemia. Pancreas. 2021;50:1310-3.
21. McNabb-Baltar J, Jin DX, Grover AS, et al. Lipase elevation in patients with COVID-19. Am J Gastroenterol. 2020;115:1286-8.
22. Benias PC, Inamdar S, Wee D, et al. A comprehensive analysis of outcomes between COVID-19 patients with an elevated serum lipase compared to those with pancreatitis. medRxiv. Published online April 14, 2021. doi: 10.1101/2021.04.13.21252871
23. Hadi A, Werge M, Kristiansen KT, et al. Coronavirus Disease-19 (COVID-19) associated with severe acute pancreatitis: Case report on three family members. Pancreatology. 2020;20:665-7.
24. Ahmed AOE, Badawi M, Ahmed K, Mohamed MFH. Case report: COVID-19 masquerading as an acute surgical abdomen. Am J Trop Med Hyg. 2020;103:841-3.
25. Hatch-Vallier B, Jarodiya V, Hawa F, Daniel R. Rare presentation of acute pancreatitis in mild COVID-19. BMJ Case Rep. 2021;14:e246720.
26. Karaali R, Topal F. Evaluating the effect of SARS-Cov-2 infection on prognosis and mortality in patients with acute pancreatitis. Am J Emerg Med. 2021;49:378-84.
27. Wan J, Yang X, He W, et al. Serum D-dimer levels at admission for prediction of outcomes in acute pancreatitis. BMC Gastroenterol. 2019;19:67.
28. Abramczyk U, Nowaczyński M, Słomczyński A, et al. Consequences of COVID-19 for the pancreas. Int J Mol Sci. 2022;23:864.