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RESEARCH ARTICLE

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Covid-19 Impact on Blood Sugar Levels and Renal Function Deterioration: A Comparative Study

Hussein Ali Mashkoor Shubbar[a], Ebru Halvaci[b], Nour Elhouda Tiri[c], Aysenur Aygun[d], Nihal Yigit Ertas[e], Saadet Celikozlu[f], Fatih Sen[g]*

[a]*Sen Research Group, Department of Biochemistry, Dumlupinar University, 43000 Kutahya, Türkiye, ORCID: 0000-0003-3849-7342*
[b]*Sen Research Group, Department of Biochemistry, Dumlupinar University, 43000 Kutahya, Türkiye, ORCID: 0009-0003-6062-7622*
[c]*Sen Research Group, Department of Biochemistry, Dumlupinar University, 43000 Kutahya, Türkiye, ORCID: 0000-0001-8153-3738*
[d]*Sen Research Group, Department of Biochemistry, Dumlupinar University, 43000 Kutahya, Türkiye, ORCID: 0000-0002-8547-2589*
[e]*Sen Research Group, Department of Biochemistry, Dumlupinar University, 43000 Kutahya, Türkiye, ORCID: 0000-0002-7563-2067*
[f]*Sen Research Group, Department of Biochemistry, Dumlupinar University, 43000 Kutahya, Türkiye, ORCID: 0000-0001-9825-6458*
[g]*Sen Research Group, Department of Biochemistry, Dumlupinar University, 43000 Kutahya, Türkiye, ORCID: 0000-0001-6843-9026*

Abstract

COVID-19 is an infectious disease triggered by the SARS-CoV-2 virus. Its first emergence was documented in December 2019 in Wuhan, China, leading to a global spread and a pandemic declaration. COVID-19 symptoms usually include fever, cough, headache, fatigue, respiratory problems, and loss of sensation. Diabetes is a medical condition characterized by insufficient insulin production from the pancreas. According to previous studies, there is a two-way relationship between diabetes and Covid-19. On the one hand, it was found that diabetes leads to a fourfold increase in the risk of infection with Covid-19 in the average person. On the other hand, infection with Covid-19 can cause diabetes in patients who do not have diabetes. Renal dysfunction is a condition in which the kidneys are unable to remove waste macromolecules from the blood serum and require medical support. According to previous studies, more than one-third of patients infected with COVID-19 had acute renal dysfunction, and 15% of those infected are known to be on dialysis. Samples were collected from patients, which numbered 160 samples, the number of normal samples was 60, and the number of abnormal samples was 60. The following analyses were performed for all samples (PCR. Ferriten, LDH, D.Dimer, S.Creatinine, B. Urea, Albumin, S, Glucose). In Covid 19, the following three analyses were chosen to diagnose the presence of the virus in the body: PCR, Ferriten, LDH, and D.Dimer. In diabetes mellitus, the following analyses were selected for this disease. These analyses are among the most accurate analyses for diagnosing diabetes which is: S, Glucose. In renal failure, the three most important analyses were chosen to diagnose the efficiency of the work and functions of the kidneys creatinine,

B.UREA, and Albumin. When samples were tested by Independent T-test regarding ferritin, LDH D-dimer, and FBS, there were no significant differences between samples that were affected by COVID-19 and samples that were not infected by COVID-19. While Creatinine, Urea, and Albumin, there are significant differences between samples that are affected by COVID-19 and samples that are not infected by COVID-19. Thus, there is a relationship between Covid 19, diabetes, and kidney function impairment, and this relationship whenever there is a strong infection with the Covid 19 virus may result in kidney function impairment or diabetes.

Keywords: COVID-19; Renal Function; Diabetes; PCR; CRP; LDH; D-dimer.

1. Introduction

The global public health emergency known as severe acute respiratory syndrome COVID-19 is caused by the coronavirus 2 (SARS-CoV-2) virus. The first symptoms of COVID-19 are known to have appeared in December 2019 in Wuhan, Hubei province, China. Since the first outbreak of COVID-19 pneumonia, it has spread rapidly and has been reported in several regions beyond China [1]. First, a group of viruses must exhibit distinctive characteristics to be classified under the family Coronaviridae of the order Nidovirales. Within this family, Coranaviruses are non-segmented, enveloped, positive RNA viruses in the coronaviridae family of the order Nidovirales. The International Committee on Taxonomy of Viruses is the primary body charged with classifying the virus family and all known viruses [2]–[4].

The coronavirus team, including COVID-19, a member of the virus family that we have recently encountered, is causing outbreaks affecting public health in East Asia and the Middle East. The first patient admissions of Severe Acute Respiratory Syndrome (SARS) in 2002 and Middle East Respiratory Syndrome (MERS) in 2012 have seen widespread public outbreaks, as well as the emergence of Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) in recent years, leading to the declaration of a global pandemic that continues to affect numerous countries and regions [5], [6]. SARS-CoV-2 is a highly virulent public health problem, transmitted through contact between individuals, as well as through contaminated objects and airborne atmospheric transmission. It is worth noting that personal protective equipment (PPE) could also serve as a source of airborne infections [7], [8]. As previously mentioned, the person-to-person transmission of SARS-CoV-2 primarily occurs through respiratory droplets generated when a patient coughs, sneezes, talks, or sings. Typically, these droplets have a limited range of about six feet (almost two meters) and linger in the air for a brief duration. Nevertheless, SARS-CoV-2 remains viable and contagious in smaller droplets (less than five microns in diameter), capable of remaining suspended in the air for up to three hours [9], [10].

Diabetes mellitus encompasses a group of disorders, including autoimmune, metabolic, and genetic factors, all characterized by elevated blood glucose levels (hyperglycemia). The approach to measuring plasma glucose and the criteria for defining normal or abnormal levels have undergone various revisions in recent decades. Diabetes mellitus is classified into four main categories: type 1 diabetes, type 2 diabetes, various specific types, and gestational diabetes. Type 1 diabetes usually involves impaired pancreatic beta-cells, often due to autoimmune inflammatory mechanisms. Autoimmune markers in serum include antibodies to insulin, tyrosine phosphatases IA-2 and IA-2b, zinc transporter ZnT8, islet cell autoantibodies, and antibodies to glutamic acid decarboxylase (GAD). Although the rate of development might vary, this damaging process typically culminates in absolute insulin insufficiency, which is defined by undetectable levels of plasma C-peptide [11], [12].

Diabetes mellitus is a common coexisting condition and contributes to a more unfavorable prognosis in individuals with COVID-19. Certainly, when examining instances of pneumonia with unidentified origins documented in Wuhan and individuals with a history of exposure to the Huanan seafood market before January 1, 2020, it was observed that

20% of these cases were associated with diabetes. Data from Italy indicates that over two-thirds of individuals with COVID-19 who did not survive had a history of diabetes [13]

To sum up, diabetes is a common comorbidity, a risk element, and an independent prognostic factor among individuals with COVID-19 [14–17]. Strong evidence supporting the adverse impact of diabetes in COVID-19 patients is further supported by two meta-analyses. In individuals with COVID-19, the prevalence of diabetes is twice as high in severe/ICU cases compared to non-severe/non-ICU cases [18].

Certainly, the identification of diabetes in a group of patients with COVID-19 infection revealed a subset of individuals facing a 2.26-fold elevated risk of encountering unfavorable disease outcomes, as reported in analyses by [19]. Furthermore, individuals with obesity and/or glucose intolerance appear to be particularly susceptible to COVID-19 [20]. Diabetes is associated with an increased risk of COVID-19 infection and worse outcomes, including hospitalization and death Individuals with diabetes, both Type 1 and Type 2, have a higher risk of contracting COVID-19 compared to those without diabetes. Insulin treatment is associated with a greater risk of COVID-19 infection compared to non-insulin drugs or no treatment. Poor glycemic control, as indicated by higher hemoglobin A1c levels, is also associated with an increased risk of COVID-19 infection. Diabetes, especially when poorly controlled, is a risk factor for severe COVID-19 outcomes [1], [21], [22].

The phrase renal failure indicates the kidneys' incapacity to carry out the excretory function, resulting in the accumulation of nitrogenous waste products in the blood [23]. There are two forms of kidney function impairment: acute and chronic renal failure. The term used when a patient requires renal replacement therapy is end-stage renal disease (ESRD).

Acute renal failure (ARF) is a clinical syndrome characterized by a swift decrease in glomerular filtration function, disruptions in water and electrolyte balance, and the rapid accumulation of nitrogen wastes in the body [24] [25].

Recently, the term acute kidney injury (AKI) has been used instead of acute renal failure (ARF) because AKI encompasses the complete clinical spectrum from a minor elevation in serum creatinine to evident renal [26].

A determined glomerular filtration rate (GFR) of less than 60 milliliters per minute (or 1.73 millimeters) or excessively increased blood creatinine for more than three months are the hallmarks of chronic renal disease (CKD), Chronic renal failure (CRF), also known as chronic kidney disease, is characterized by a gradual deterioration in renal structure and function, eventually necessitating kidney transplantation therapy [27]. People with multiple sclerosis, especially those on immunosuppressants or immunomodulators, are more at risk for serious symptoms of SARS-CoV-2 infection and COVID-19 sequelae [28]. Research has sought to determine which MS patients are more vulnerable to SARS-CoV-2 infection, analyze the relationship between SARS-CoV-2 and MS, and assess the immune system's reaction to SARS-CoV-2 infection and vaccinations [29].

In COVID-19, kidney disease has been documented, with AKI observed in over 20% of severely ill or deceased patients, a proportion consistently reported in studies conducted in China, Italy and the United States [30], [31]. It is noteworthy that AKI, proteinuria, and hematuria have been individually linked to an increased risk of mortality in individuals with COVID-19 [32]. The occurrence of pre-existing chronic kidney disease was markedly higher among those experiencing severe COVID-19 disease [33].

According to tissue cell immunohistochemistry analyses, ACE2 appears to be absent in renal endothelial cells. However, a study using single-cell analysis confirmed the presence of ACE2 and TMPRSS2 expression in human renal endothelial cells [5]. Recently, viral particles were observed in the endothelial cells of glomerular capillary loops in a COVID-19 patient using electron microscopy [34]. Histopathological analysis of the kidney of the patient with COVID-19 showed the presence of inflammation or endothelial tissue damage even in the absence of interstitial inflammatory infiltrates [35],[36].

Although there have been studies conducted on the impact of COVID-19 on diabetes and renal disease separately, there has not been any research on their combined effect in the same patient [37]. Therefore, our study aims to investigate the effect of COVID-19 on both diabetes and renal function in the same patient. We will analyze the data

to determine the correlation between the two conditions and how they may exacerbate each other in COVID-19 patients [38].

The samples were chosen from the Middle Euphrates region of Iraq, where 60 healthy samples were taken from individuals who were not infected with the COVID-19 virus and did not exhibit any symptoms of infection, and 60 abnormal samples were taken from individuals who were positively identified as having the virus and displaying symptoms of the illness. Males and females between the ages of 15 and 70 were chosen, and all 120 samples underwent the following analyses. The tests included (PCR, CRP, Ferritin, LDH, D. Dimers, Creatinine B. UREA, Albumin, and Glucose).

2.Material and Method

2.1.Collection of blood

Venous blood is drawn from the arm using a sterile syringe. The arm should be warm and the person should be in a comfortable position. A compressive bandage is applied gently, the skin is cleaned with alcohol, and then a needle is inserted into the vein to draw 5-10 ml of blood. After withdrawing the needle, the blood is placed in a test tube for separation.

2.2.Collecting samples for PCR analysis

as follows: 1. Blood test. 2. Nasal swabs. Nonetheless, nose swabs were utilized in this investigation, and there are three ways to collect nasal swabs: Frontal Nasal Swab: To perform this, place the swab in the front of your nostrils and hold it there for ten to fifteen seconds. Mid Nasal Swab: Here, the swab is inserted until resistance is felt, then it is rotated for 15 seconds before being withdrawn. This process is repeated in the second nostril. Oropharyngeal Nasal Swab: In this method, the swab is inserted into the nostril, reaching the nasopharynx or the back of the throat, and then rotated before being withdrawn. All specimens to be tested are infectious body fluids and must therefore be handled under the requirements of laboratory biosafety rules.

2.3. Polymerase chain reaction (pcr) test

Turn the 96-well plate upside down so that the liquid that sticks to the sealing film and the well walls falls to the bottom of the plate. To use, let them stand for three to five minutes. every part included in the package. In a 96-well plate, mix. After removing the sealing film, add 200 µL of sample and 15 µL of Proteinase K in succession to Extraction Reagent II in the 96-well plate. As directed, load plates into the instrument; once loaded, close the door. Select the program after turning on the nucleic acid extraction device. Launch the extraction software; it will take around 12 minutes. When finished, remove the 96-well plate.

2.4. Ferritin test

Transfer 30 µL of sample to a tube with detection buffer, mix thoroughly, and immediately pipette 75 µL of the mixture into a sample well on the cartridge. Insert the cartridge into an i-Chamber or incubator at 25 °C for 10 minutes. After incubation, scan the cartridge immediately using the instrument for ichroma™ tests. The normal range for women is 20-250 ng/mL and for men is 30-350 ng/mL, with a working range of 10-1,000 ng/mL.

2.5. C-reactive protein (CRP) test

In summary, the process involves adding undiluted serum and control samples to a slide and then adding CRP latex reagent to each sample. Agglutination is observed within 3 minutes, with positive results indicating a CRP concentration > 6 mg/l and negative results indicating a CRP concentration < 6 mg/l or no CRP present.

2.6. Elevated lactate dehydrogenase (ldh) test

Pipette the reagents into a 1 cm long thermostat cuvette and bring the reagent media to 37°C 1000 µL after allowing the samples to stand at room temperature, then add the collected sample. Once the media is calibrated, collect 20 µL of sample, mix, and add again for measurement. Record the first absorbance at 340 nm (or 334 nm) after 30 seconds, then record the absorbance again after 1 minute and 2 minutes. Obtain results by calculating the change in absorbance per minute (Abs/min.). Calculations: LDH activity (IU/L) = (ΔAbs/min)Assay / (ΔAbs/min)Calibrator × Calibrator Activity. Normal Range Adult LDH activity: at 37° C: 200-400 IU/L (SFBC method).

2.7. D-dimer test

The process involves taking 150 µL of detector diluent and adding it to the detector tube containing a granule to form the detection buffer, then adding 10 µL of sample and mixing it thoroughly. After that, 75 µL of the sample mixture is dispensed into the sample well of the cartridge, which is then left at room temperature for 12 minutes. The cartridge is then scanned immediately using the instrument for ichroma™ tests, and the test result is read on the display screen. The normal range for the test is 500 ng/mL, with a working range of 50-10,000 ng/mL. It's important to ensure the cartridge is scanned immediately after incubation to avoid inaccurate results.

2.8. Creatinine blood test

Measuring against air at rising absorbance, measure mercury at wavelength 492 nm (490-510 nm), optical path (1 cm), and temperature 25°C/37°C. The cuvette and reagents should be heated to the appropriate temperature (±0.5°C) and maintained there during the test. Procedure for Assay Sample/STD 100 ul and working reagent 1000 ul are collected from the first tube, and Sample/STD 200 ul and working reagent 2000 ul are obtained from the second tube. When the mixing process begins, start the timer. Read the absorbance value A after 30 seconds, followed by the absorbance value A2 after 2 minutes. The keratin blood test's normal serum range is 0.5 _ 0.9 mg/dl for women and 0.6 _ 1.1 mg/dl for males (Eq.1).

$$A2\_ A1 = AA \text{ sample or } AA \text{ STD.} / \text{ Calculations } C = 2.0 \text{ X } \Delta A \text{ sample} / \Delta A \text{ STD [mg/dl].} \quad (1)$$

2.9. Albumin blood test

The sample to be measured is specific at a wavelength of 546 nm. The measurement is taken against the reagent blank at a cuvette length of 1 cm, temperature 20-25°C. Three tubes were taken into the Test Procedure and a Blank (Albumin Reagent 1000 µL, Standard 0 µL, Sample 0 µL) was taken in the first tube. Standard (Albumin Reagent 1000 µL, Standard 10 µL, Sample 0 µL) was taken in the second tube. Mix the sample (Albumin Reagent 1000 µL, Standard 0 µL, Sample 10 µL), mix and incubate at 20-25°C for 5 minutes. Measure the absorbance of the sample (As) and standard (Astd) against the reagent blank within 30 minutes.

Calculation Serum Albumin (g/dL) = ΔA sample /ΔA standard X 4 (Std.conc.) Normal Range serum albumin 3.8_5.1g/Dl

2.10. Blood urea nitrogen (bun) test

For the BUN test, optimum conditions for temperature (20-25°C or 37°C), wavelength (570-600 nm, 546 nm) 578 nm for Hg, Path 1 cm, and reagent should be provided. For each series, just one reagent blank is needed. Assay Methodology Sample/STD 0 µl, Enzyme reagent (R1) 1000 µl are placed in the first tube, and Sample/STD 10 µl and Enzyme reagent (R1) 1000 µl are placed in the second tube. After mixing and incubating for 5 minutes at 20–25°C or 3 minutes at 37°C, add 1000 µl of RGT2/R2 to each of the two tubes that came before it. Combine, then let it sit for 10 minutes at 20–25°C or 5 minutes at 37°C. Within 60 minutes, compare the absorbance of the sample (As ample) and the STD (ASD) to the reagent blank. Worksheets C = urea

2.11. Fasting blood sugar (fbs) test

For sample measurement, set the wavelength to 505 nm (490-550), temperature to 37°C/15-25°C, and cuvette length to 1 cm. Zero the instrument cavity with distilled water and then place the sample in the cuvette. Three tubes are taken for the test procedure. The first tube is blank (WR(ml) 1.0, standard grade 1.2(ul) 0 ul, sample (ul) 0 ul); the second tube is standard (WR(ml) 1.0, standard grade 1.2(ul) 10 ul, sample (ul) 0 ul); the third tube is sample (R(ml) 1.0, standard grade 1.2(ul) 0 ul, sample (ul) 10 ul). Mix the sample before placing it in the instrument and incubate for 10 minutes at 37°C or 20 minutes at room temperature (15-25°C). Read the absorbance (A) of the samples and standard against the blank. However, the color in the sample is stable for at least 30 minutes. Normal Range Serum or plasma should be 60_110 mg/dL, 3.33_6.10 mmol/L. Calculations (A) Sample - (A) Blank / A) Standard- (A) Blank x 100 (Standard conc.) = mg/dL glucose in the sample,

3.Sample Grouping

A total of 120 samples were collected and divided into two groups. The first group consisted of 60 samples infected with Corona virus and the second group consisted of 60 samples not infected with Corona virus. The following analyzes were performed on the samples; PCR test (Polymerase Chain Reaction Test), Ferritin test, Dimer Neo test, LDH test (Lactate dehydrogenase test), CRP test (C reactive protein), Creatinine Blood test, Blood Urea test, Glucose test.

4.Statistics Analysis

We carried out the statistical study based on patient tests transcribed in the form of an Excel table. To test the normality of the data, we used the Kolmogorov-Smirnov test and an independent t-test to compare the two groups (COVID and non-COVID). The chosen threshold of statistical significance was $p < 0.05$.

5.Results and Discussion

A total of 120 samples were tested using PCR and CRP for COVID-19, with negative results being placed in the non-COVID-19 group and positive results being placed in the COVID-19 group

5.1. Descriptive statistics

The mean and standard deviation values of the minimum, maximum test analyses for the groups without COVID-19 are given in table 1.

Table 1. Descriptive statistics, and values for Non-COVID groups.

Test Name	Minimum	Maximum	Mean	Std. Deviation
Ferritin Test	108.00	213.00	195.7833	19.45276
D.DIMER Test	108.00	213.00	96.13333	8.784552
LDH Test	108.00	213.00	242.3333	18.10952
ALBUMIN Test	3.40	5.40	4.355	0.595299
S.CREATININE Test	108.00	213.00	0.839333	0.186828
B.UREA Test	8.00	24.00	17.835	4.527012
FBS Test	80.00	125.00	99.4667	10.67147

The mean and standard deviation values of the minimum, maximum test analyzes for COVID-19 are given in table 2.

Table 2. The Descriptive statistics, and values for COVID groups.

Test Name	Minimum	Maximum	Mean	Std. Deviation
Ferritin Test	391.00	433.00	412.5932	10.99382
D.DIMER Test	391.00	433.00	617.5254	11.28876

LDH Test	391.00	433.00	524.6441	13.15677
ALBUMIN Test	2.40	8.90	4.040678	1.268901
S.CREATININE Test	391.00	433.00	1.978305	5.643343
B.UREA Test	59.00	901.00	76.28367	9.657102
FBS Test	145.00	208.00	183.5085	13.52771

A total of 120 samples were tested using PCR and CRP for COVID-19, with negative results being placed in the non-COVID-19 group and positive results being placed in the COVID-19 group. Subsequently, all samples were tested for LDH, Ferritin, and D-dimer, which, in contrast to the non-COVID-19 group, demonstrated an increase in the COVID-19 group. The diabetes test indicated that samples in the COVID-19 group had a higher mean value than those in the non-COVID-19 group. In terms of renal function tests, Creatinine and Urea increased, whereas the COVID-19 group's mean albumin levels dropped in comparison to the non-COVID-19 group. When samples were tested by Independent T-test regarding ferritin, LDH D-dimer, and FBS, there were no significant differences between samples that were affected by COVID-19 and samples that were not infected by COVID-19. While Creatinine, Urea, and Albumin, there are significant differences between samples that are affected by COVID-19 and samples that are not infected by COVID-19 (Figures 1,2 and 3).

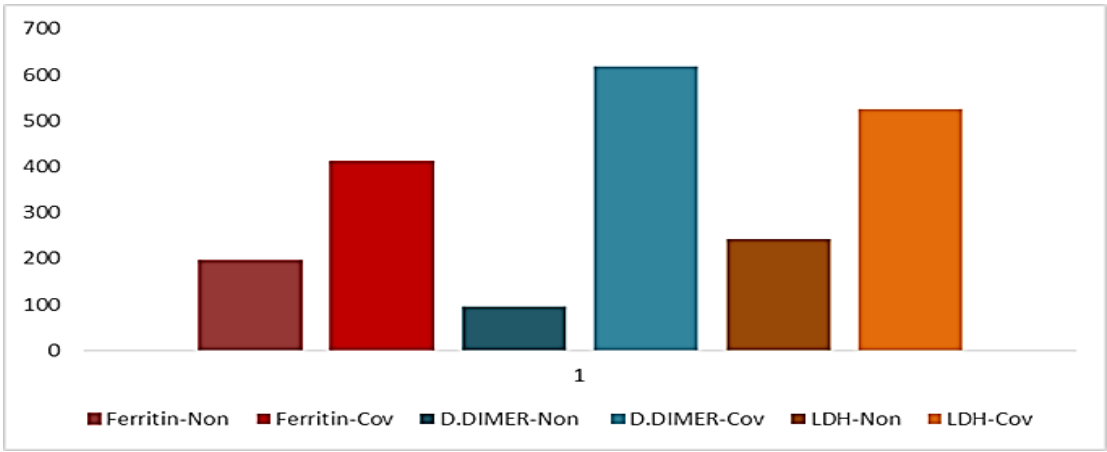

Fig. 1. The bar chart represents the non-significant increase in ferritin, LDH, and D-dimer levels in the COVID-19 group when compared to the non-COVID-19 group.

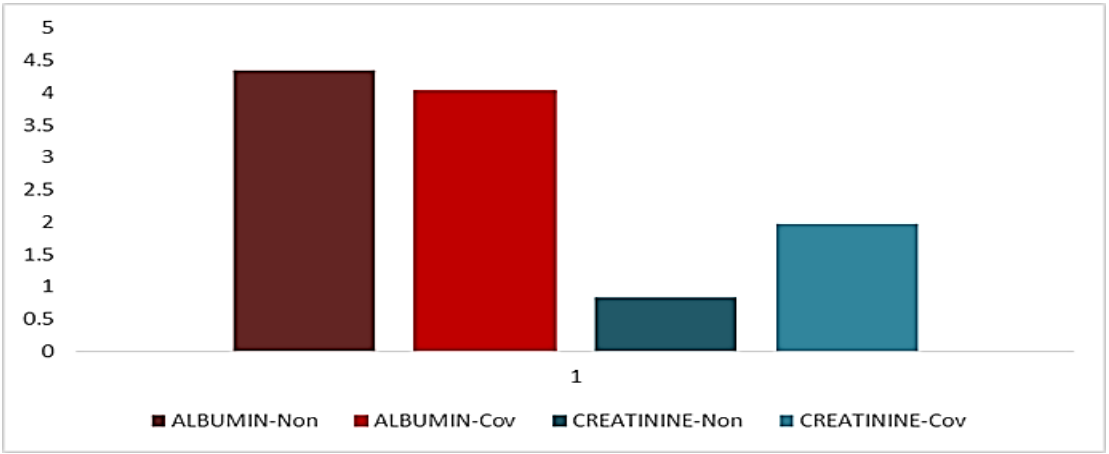

Fig. 2. The bar chart represents a significant increase in Creatinine, while Albumin appears significant decrease in levels in the COVID-19 group when compared to the non-COVID-19 group.

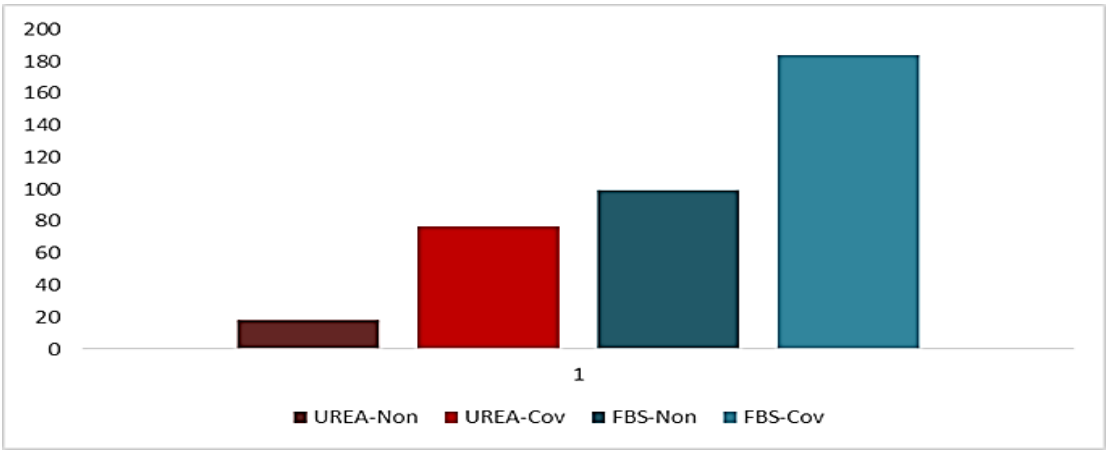

Fig. 3. The bar chart represents a significant increase in Urea while FBS appears non-significant levels in the COVID-19 group when compared to the non-COVID-19 group.

5.2. Polymerase Chain Reaction (PCR) and C-reactive Protein (CRP) Technique

PCR and CRP tests appear to aid in the early diagnosis and management of COVID-19, which is crucial in preventing the spread of the disease. In addition, the collected body fluid results provide evidence that PCR and CRP are effective testing and diagnostic tools for identifying COVID-19 cases. The findings highlight the importance of common tests that can be established to accurately identify and isolate infected individuals in the context of a

pandemic. PCR and CRP assays are diagnostic and in addition to investigating their efficacy in different populations and disease settings, there is a need for further research to explore the potential of new emerging potential diseases.

5.3. Ferritin Test

The test kits show that ferritin levels are elevated in individuals with the disease, but there is no statistical difference compared to patients without COVID-19. The results clearly show that a systematic review did not lead to significant results when compared to patients without elevated ferritin levels. The increase in ferritin levels may be due to cytokine storm and secondary hemophagocytic lymphohistiocytosis. The findings are similar to previous meta-analysis and meta-regression analysis studies [39], [40].

5.4. D-dimer Test

Tests have shown an increase in D-dimer levels in COVID-19 patients, but there is no statistically significant difference compared to patients without COVID-19. Tests have shown that D-dimer levels are commonly elevated in COVID-19 patients. The test results corroborate each other, suggesting that COVID-19 may be a useful tool in determining mortality risk, given the risk of disease transmission [7]. Increased D-dimer levels in COVID-19 patients are known to have several clinical implications, including its role in disease progression, its ability to guide in monitoring the course of treatment. These results suggest that elevated D-dimer levels may be associated with more severe COVID-19 outcomes such as death and syndrome in individuals suffering from COVID-19. Near future studies suggest that D-dimer levels, when used in combination with other testing factors, may help predict favorable COVID-19 outcomes [41].

5.5. Elevated Lactate Dehydrogenase (LDH)

The research findings reveal that among COVID-19 carrier individuals, there was a significant increase in Elevated Lactate Dehydrogenase (LDH) levels among those whose disease course did not progress. In addition, individuals without COVID-19 did not show a statistically significant increase in LDH levels compared to COVID-19 carriers, consistent with findings from previous studies. In addition, recent studies show that LDH levels in the blood of a patient with COVID-19 are an independent risk factor for the severity of COVID-19 and mortality in the population. Therefore, it suggests that a high LDH/Lymphocyte ratio is an independent risk factor for population mortality in COVID-19 patients. This finding demonstrates the potential use of LDH/lymphocyte ratio as a valuable diagnostic tool and prognostic marker in the clinical management of COVID-19 [42], [43].

5.6. Albumin Blood (ALB)

Tests show that serum albumin levels are significantly higher in patients with non-severe COVID-19 than in patients without COVID-19. A study by Gulam Rabbani and Saeyoung Nate Ahn reveals that low serum albumin levels are associated with an increased risk of severe COVID-19 and mortality. This is due to albumin's role in protecting the lungs from inflammation [44– 46].

5.7. Creatinine Blood

The test kit performed reveals that the Creatinine levels of individuals with COVID-19 are significantly higher than those who do not carry the virus. This difference is statistically significant compared to other test kits. The explanation

for high creatinine is a sign of November muscle damage. The results are in line with previous research by Deniz Ok MD et al. Blood urea nitrogen (BUN) levels of COVID-19 patients have been found to be significantly higher. The increased risk of death and the severity of the disease appear to be strongly associated with high Creatinine levels. High Creatinine values indicate acute kidney injury caused by COVID-19 treatment or the virus itself. This study is also in line with his study, which suggests that an increase in creatinine is the first presentation of coronavirus disease 2019 (COVID-19). Therefore, the increase in keratin levels can be caused by various factors, including rhabdomyolysis, injuries, certain medications, and cardiovascular disease [47– 49]

5.8. Blood Urea Nitrogen (BUN)

In our study, urea levels were significantly increased in COVID-19 patients, and the difference was statistically significant compared to patients without COVID-19. These findings are consistent with previous studies Fesih Ok MD et al., 2020 We found that blood urea nitrogen (BUN) levels were significantly higher in COVID-19 patients. Elevated BUN levels were positively associated with increased disease severity and risk of death. Elevated blood urea nitrogen (BUN) levels may be a sign of acute kidney injury caused by the virus or by the treatments used to treat COVID-19 [50].

Additionally, a study found that BUN levels were significantly higher in the deceased group compared to the survivor group. Specific BUN threshold values can be used to identify patients at high risk and initiate appropriate therapeutic interventions early [51].

5.9. Fasting Blood Sugar (FBS)

A significant increase is observed in glucose level studies conducted on non-severe COVID-19 patients. This increase gives statistically significant results, that is, the probability of it being due to chance is quite low. In cases of COVID-19, the increased glucose level in combination with diabetes disease is of importance in past, present and future studies. Therefore, he suggests that high blood sugar levels pose a risk factor along with severe cases of COVID-19. In October, an increase in glucose levels is also associated with an increased risk of COVID-19 complications, such as acute respiratory syndrome (ARDS) and respiratory failure. There are several possible explanations for high blood sugar in people with COVID-19. The first possibility is that infection with the COVID-19 virus can damage the cells of the pancreas that produce insulin. This can lead to a decrease in insulin production, and as a result, high blood sugar levels are observed. Another possibility is that, on the contrary, infection with the COVID-19 virus can lead to the release of insulin from the pancreas. This can lead to a decrease in blood sugar levels, which in turn leads to an increase in the production of glucose from the liver [52]. In addition, this study by Zohair Jamil Gazzaz confirms the high blood sugar studies in the tests for the 2021 COVID-19 cases.

6.Conclusion

Various studies are being proposed against the COVID-19 virus, disease markers, monitored methods, infection risks and high plasma levels. Also as a result of this study, it is known that COVID-19 patients have higher LDH, Ferritin, D-dimer and diabetes markers, as well as kidney dysfunction compared to non-COVID-19 patients. Along with these findings, the symptoms encountered in COVID-19 cases cause various organ and tissue damage. In addition, it shows that it can damage many organs and systems, including the kidneys and pancreas, leading to potential long-term health consequences. Therefore, it is very important to monitor various biomarkers such as LDH, Ferritin December, D-dimer, BUN and glucose in COVID-19 patients, to perform their tests at regular intervals and to develop appropriate treatment methods.

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Author Contribution

Hussein Ali Mashkoor Shubbar, Ebru Halvacı, Nour Elhouda Tiri, Aysenur Aygun, Nihal Yigir Ertas, Saadet Celikozlu; wrote, edited, drew figures and developed the article. Fatih Sen; supervisor and responsible person.

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