



The Relationship Between SpO₂ and Laboratory Diagnostic Markers of Cardiovascular Patients with COVID-19

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Abstract

Background: In December 2019, a new virus, the coronavirus disease 2019 (COVID-19) was identified as the cause of a cluster of cases of pneumonia of unknown etiology in Wuhan, China. The COVID-19 disease increases cardiovascular events both directly and indirectly. This study aimed to investigate the laboratory diagnostic markers of cardiovascular patients with COVID-19 in Ayatollah Taleghani hospital in Abadan from March 20, 2020 to March 19, 2021.

Materials and Methods: This study is a cross-sectional analytical study. Some demographic, laboratory, and clinical information of 200 cardiovascular patients with COVID-19 admitted to Taleghani hospital, Abadan, was received randomly by referring to the medical records section and medical records section and health information system (HIS) as well as searching in the patient records. Inclusion criteria included cardiovascular patients with COVID-19 who had been admitted to Ayatollah Taleghani hospital in Abadan with the diagnosis of a cardiologist and infectious disease specialist, and those whose information was available in HIS. The results were analyzed using SPSS software version 24.

Results: The mean age of cardiovascular patients with COVID-19 was reported to be 66.98 ± 18.14. The results revealed that the mean of fasting blood sugar (FBS), serum glutamic oxaloacetic transaminase (SGOT), *alkaline phosphatase* (ALP), lactate dehydrogenase (LDH), creatinine (Cr), blood urea nitrogen (BUN), and erythrocyte sedimentation rate (ESR) was higher than the normal level in these patients. In addition, the mean oxygen saturation (SpO₂) was observed to be lower than normal. Reducing the level of SpO₂ to less than 90% was significantly related to increasing age, death, patients with a history of lung disease, the duration of hospitalization in ICU, and intubation. This reduction also led to an increase in respiratory rate (RR), LDH, ESR, and C-reactive protein (CRP + 1) in cardiovascular patients with COVID-19.

Conclusion: In cardiovascular patients with COVID-19, FBS, some kidney markers, liver markers, and inflammatory markers were observed to be higher than normal, and a significant relationship was observed between the reduction of SpO₂ and some laboratory diagnostic markers, which requires extensive studies with larger sample size.

Keywords: SpO₂, COVID-19, Cardiovascular diseases

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Introduction

In December 2019, new viruses were identified as the cause of a cluster of cases of pneumonia of unknown etiology in Wuhan, China, and the World Health Organization (WHO) named the new virus as the official disease caused by the coronavirus disease 2019 (COVID-19) virus (1).

The COVID-19 disease increases cardiovascular events

both directly and indirectly. In direct incidents with the mechanism of myocarditis, it causes damage to the heart muscle. The indirect method causes hypoxia and ischemia in the body by reducing the oxygen saturation (SpO₂) caused by lung involvement. In addition, the inflammatory factors and cytokine storm caused by the COVID-19 disease lead to the instability of atherosclerotic plaques and increase the risk of clot formation in blood

vessels, causing the blockage of coronary arteries and damage to the heart (2).

Cardiovascular diseases are the most common cause of death in most countries of the world, including Iran. Cardiovascular diseases are one of the most preventable non-communicable human diseases. The occurrence of cardiovascular diseases can be prevented to a large extent by controlling risk factors such as high blood pressure and smoking, reducing blood cholesterol levels and weight, as well as changing and modifying lifestyles (3). Sinus tachycardia is the most common COVID-19 tachycardia, while ventricular fibrillation and atrial fibrillation are less common (4). The mortality rate in patients with a history of cardiovascular disease is more than four times that of people without a history of cardiovascular disease with COVID-19 (5). COVID-19 causes lymphopenia, thrombocytopenia, increased erythrocyte sedimentation rate (ESR), increased C-reactive protein (CRP), increased lactate dehydrogenase (LDH), and prolonged prothrombin time, which are known to be the most common laboratory abnormalities (6). Aspartate aminotransferase elevation is common in COVID-19, mirrors disease severity, and appears to reflect true hepatic injury (7). The blood urea nitrogen/creatinine (BUN/Cr) ratio may be related to disease severity, and the routine use of these parameters may be helpful in disease assessment. Elevated BUN/Cr ratios are independent predictors of the severity and survival of COVID-19 patients (8).

The most specific and sensitive cardiac biomarkers that indicate myocardial infarction are troponin I and troponin T, which are known as the gold standard and are measured quantitatively and qualitatively (9). This study was conducted to investigate the laboratory diagnostic markers of cardiovascular patients with COVID-19 at Ayatollah Taleghani hospital in Abadan, Iran. By examining laboratory diagnostic markers in cardiovascular patients with COVID-19, important information is provided to doctors for the proper treatment of these patients because there are numerous unknown features in these patients, which raise the necessity for conducting such research.

Materials and Methods

This is a cross-sectional analytical study. Some demographic, laboratory, and clinical information of 200 cardiovascular patients with COVID-19 admitted to the COVID-19 disease center in Taleghani hospital, Abadan, from March 20, 2020 to March 19, 2021 was received randomly by referring to the medical records section and HIS as well as searching in the patient records. Inclusion criteria were cardiovascular patients with COVID-19 who have been admitted to Ayatollah Taleghani hospital in Abadan with the diagnosis of a cardiologist and infectious disease specialist, and their information was available in HIS. Exclusion criteria included cardiovascular patients

who were not infected with COVID-19, or if they were infected, their tests were either not available or their laboratory markers were incomplete. After extracting information, the data were sorted based on age, sex, date of admission, disease code, and other laboratory and clinical diagnostic markers; afterward, duplicate data were removed and recorded in Excel software. The results were analyzed using SPSS software version 24.

The sample size was calculated after a literature review of previous similar studies using the formula for the estimate of linear correlation between two quantitative variables (10). Considering a bilateral α of 0.05 and β of 0.10, 193 patients were needed to ensure that a correlation coefficient of 0.35 was significantly different from the null hypothesis. This study hypothesized the existence of a correlation between the factors and SpO₂. Finally, a total of 200 cardiovascular patients with COVID-19 were recruited by convenience sampling from Taleghani hospital, Abadan.

Statistical Analysis

To describe the data, mean and standard deviation were used in quantitative variables, and frequency and percentage were used in qualitative variables. Data analysis was done by SPSS software and a *P* value less than 0.05 was considered statistically significant. Multiple linear regressions were also used to check the correlation and relationship of variables with the SpO₂ index.

Results

The mean age of 200 cardiovascular patients with COVID-19 was 66.89 ± 14.80 years. Most patients (51%) were 51 to 70 years, and half of the studied patients were women. Shortness of breath (52%), cough (41%), and fever (31%) were the most common symptoms observed in patients (Table 1).

The mean length of hospitalization of the patients was 12.43 ± 4.50 (range: 1-24 days). The number of intubated patients was 22 (11%). Hospitalization in the intensive care unit (ICU) and elevated troponin level was reported in 73 (36.5%) and 32 (16%) patients, respectively (Table 1). The mean (122.23 ± 49.05) of fasting blood sugar (FBS) was higher than the normal level. Among the renal markers, the mean of BUN (35.79 ± 20.63) and Cr (1.63 ± 1.70) was higher than the normal level (Table 1). Among the liver markers, the mean level of serum glutamic oxaloacetic transaminase (SGOT) 38.72 ± 1.42 , *alkaline phosphatase* (ALP) 217.36 ± 65.17 , and LDH (855.22 ± 385.01) were reported to be higher than the normal level (Table 1). Moreover, Table 1 indicates that among the inflammatory markers, ESR 60.25 ± 28.34 was higher than normal, and CRP was positive in 172 patients (69 CRP1+, 53 CRP2+, and 50 CRP3+). The mean of SpO₂ (85.06 ± 7.09) was reported to be lower than the normal level, while the mean of respiratory rate (RR) (23.15 ± 3.03) was reported

Table 1. Demographic, Laboratory, and Clinical Characteristics of Patients (N=200)

| Variable | | Mean or N | SD or % |
|--------------------------------|----------|-----------|---------|
| Age (y) | ≤ 50 | 21 | 10.5% |
| Mean ± SD: | 51–70 | 102 | 51.0% |
| 66.89 ± 14.18 | ≥ 71 | 77 | 38.5% |
| Gender | Male | 100 | 50.0% |
| | Female | 100 | 50.0% |
| Blood pressure | Yes | 82 | 41.0% |
| Diabetes | Yes | 103 | 51.5% |
| Renal disease | Yes | 25 | 12.5% |
| Heart disease | Yes | 53 | 26.5% |
| Lung disease | Yes | 23 | 11.5% |
| FBS (mg/dL) | | 122.23 | 49.05 |
| BUN (mg/dL) | | 35.79 | 20.63 |
| Cr (mg/dL) | | 1.77 | 1.68 |
| Na (mmol/L) | | 138.23 | 5.03 |
| K (mmol/L) | | 4.29 | 0.74 |
| ALP (U/L) | | 217.34 | 65.17 |
| LDH (U/L) | | 852.22 | 385.01 |
| Total bilirubin (mg/dL) | | 0.80 | 0.26 |
| Direct bilirubin (mg/dL) | | 0.40 | 0.39 |
| SGOT (IU/L) | | 38.75 | 15.42 |
| SGPT (IU/L) | | 27.52 | 12.10 |
| PT (s) | | 12.64 | 1.53 |
| PTT (s) | | 34.46 | 8.64 |
| INR | | 1.10 | 0.21 |
| ESR (mm/h) | | 60.25 | 28.34 |
| CRP | Negative | 28 | 14.0% |
| | 1 + | 69 | 34.5% |
| | 2 + | 53 | 26.5% |
| | 3 + | 50 | 25.0% |
| WBC (/mm ³) | | 11.02 | 5.50 |
| RBC (million/mm ³) | | 4.41 | 0.83 |
| MCV (μm ³) | | 85.14 | 8.04 |
| MCH (pg/cell) | | 28.22 | 5.50 |
| MCHC (Hb/cell) | | 31.64 | 2.21 |
| PLT (mm ³) | | 226.99 | 85.22 |
| RR | | 23.15 | 3.03 |
| SPO ₂ | | 85.16 | 7.09 |
| SBP | | 121.91 | 20.92 |
| Troponin | Negative | 178 | 84% |
| | Positive | 32 | 16% |

Note. N: Number; SD: Standard deviation; FBS: Fasting blood sugar; BUN: Blood urea nitrogen; Cr: Creatinine; Na: Sodium; K: Potassium; ALP: Alkaline phosphatase; LDH: Lactate dehydrogenase; SGOT: Serum glutamic oxaloacetic transaminase; SGPT: Serum glutamate pyruvate transaminase; PT: Prothrombin time; PTT: Partial thromboplastin time; INR: International normalized ratio; ESR: Erythrocyte sedimentation; CRP: C-reactive protein; WBC: White blood cell; RBC: Red blood cell; MCV: Mean corpuscular volume; MCH: Mean corpuscular hemoglobin; MCHC: Mean corpuscular hemoglobin concentration; PLT: Platelet; RR: Respiratory rate; SPO₂: Oxygen saturation; SBP: Systolic blood pressure.

higher than the normal level (Table 1).

How Is SpO₂ Related to Laboratory Markers and Clinical Findings of Patients?

As Table 2 illustrates, with each year of increase in age, the mean of SpO₂ decreased by 0.06 units, which had a significant relationship ($P=0.02$). The mean of SpO₂ in patients who died is 3.16 units less than that in patients who recovered, which had a significant relationship ($P<0.01$). In addition, the mean of SpO₂ in patients with lung disease was 2.79 units lower than that in patients who did not have lung disease, which had a significant relationship ($P<0.01$). Furthermore, according to Table 2, with each unit increase in LDH, the mean of SpO₂ decreased by 0.002 units, which had a significant relationship ($P=0.02$). Likewise, with each unit increase in ESR, the mean of SpO₂ decreased by 0.05 units, which had a significant relationship ($P<0.001$). The mean of SpO₂ in patients who had CRP1+ was 2.07 units lower than that in patients whose CRP was negative, which had a significant relationship ($P<0.01$). With each unit increase in mean corpuscular hemoglobin concentration, the mean of SpO₂ increased by 0.46 units, which had a significant relationship ($P<0.01$), and with each unit increase in RR, the mean of SpO₂ decreased by 1.26 units, which had a significant relationship ($P<0.001$). Moreover, with each unit increase in SBP, the mean of SpO₂ increased by 0.04 units, which had a significant relationship ($P=0.04$), as depicted in Table 2.

Furthermore, the mean of SpO₂ in patients who were intubated upon arrival to the emergency department was 6.12 units lower than that of the patients who were not intubated at the beginning of hospitalization, which had a significant relationship ($P<0.001$), as illustrated in Table 2. Finally, with each unit increase in the duration of hospitalization in the ICU, the mean SpO₂ decreased by 0.19 units, which had a significant relationship ($P=0.01$), as illustrated in Table 2.

Discussion

In the present study, the results showed that in the cardiovascular patients with COVID-19, ESR, CRP, and LDH levels increased; further, with the increase in ESR, LDH, and CRP levels, SpO₂ decreased, and a significant relationship was found between ESR, LDH, and CRP¹⁺ markers.

In the study by Tang et al in China, it was found that COVID-19 causes lymphopenia, thrombocytopenia, increased ESR, increased CRP, increased LDH, and prolonged prothrombin time (6).

Based on a cohort study by Yang et al in 2020, it was found that 50% of people who died due to the COVID-19 disease and 6% of people who were infected with the severe type of disease of COVID-19 had heart enzyme troponin I, and CRP increased significantly (11). In the

Table 2. Association between Spo2 and Laboratory Diagnostic Factors in the Linear Regression Model

| Variable | Unstandardized | | Standardized | t | P | 95.0% CI for B | |
|--------------------------------|----------------|------------|--------------|--------|--------|----------------|--------|
| | B | Std. Error | Beta | | | Lower | Upper |
| Age (y) | -0.067 | 0.03 | -0.13 | -2.26 | <0.05 | -0.12 | -0.009 |
| Gender (male) | -0.13 | 0.75 | -0.01 | -0.17 | 0.86 | -1.61 | 1.35 |
| Outcome (died) | -3.16 | 1.23 | 0.12 | 2.57 | 0.01 | 0.74 | 5.59 |
| Blood pressure (yes) | -0.62 | 0.84 | -0.04 | -0.73 | 0.46 | -2.29 | 1.04 |
| Diabetes (yes) | 0.81 | 1.00 | 0.05 | 0.80 | 0.42 | -1.17 | 2.79 |
| Renal problem (yes) | -1.21 | 1.57 | -0.05 | -0.77 | 0.44 | -4.31 | 1.88 |
| Heart problem (yes) | -0.62 | 0.94 | -0.04 | -0.66 | 0.50 | -2.49 | 1.24 |
| Lung problem (yes) | -2.79 | 1.04 | 0.13 | 2.69 | 0.01 | 0.74 | 4.85 |
| FBS (mg/dL) | -0.003 | 0.01 | -0.02 | -0.32 | 0.74 | -0.02 | 0.02 |
| BUN (mg/dL) | 0.045 | 0.02 | 0.13 | 1.76 | 0.08 | -0.006 | 0.09 |
| Cr (mg/dL) | 0.34 | 0.37 | 0.08 | .93 | 0.35 | -0.39 | 1.08 |
| Na (mmol/L) | -0.046 | 0.08 | -0.03 | -0.52 | 0.59 | -0.22 | 0.12 |
| K (mmol/L) | -1.31 | 0.69 | -0.12 | -1.90 | 0.06 | -2.68 | 0.05 |
| ALP (U/L) | -0.01 | 0.006 | -0.09 | -1.75 | 0.08 | -0.02 | 0.001 |
| LDH (U/L) | -0.002 | 0.001 | -0.13 | -2.29 | 0.02 | -0.004 | 0.00 |
| Total bilirubin (mg/dL) | -1.65 | 1.73 | -0.06 | -0.95 | 0.34 | -5.07 | 1.77 |
| Direct bilirubin (mg/dL) | 0.15 | 1.06 | 0.01 | 0.14 | 0.88 | -1.94 | 2.24 |
| SGOT (IU/L) | 0.04 | 0.03 | 0.09 | 1.40 | 0.16 | -0.02 | 0.10 |
| SGPT (IU/L) | -0.03 | 0.03 | -0.06 | -1.03 | 0.30 | -0.11 | 0.03 |
| PT (s) | 0.63 | 0.22 | 0.14 | 2.87 | <0.01 | 0.19 | 1.06 |
| PTT (s) | -0.02 | 0.05 | -0.03 | -0.48 | 0.62 | -0.12 | 0.07 |
| INR | 4.04 | 3.64 | 0.12 | 1.11 | 0.26 | -3.16 | 11.24 |
| ESR (mm/h) | -0.05 | 0.01 | -0.19 | -3.98 | <0.001 | -0.07 | -0.02 |
| CRP ^a = + | -2.07 | 0.73 | 0.14 | 2.84 | <0.01 | 0.63 | 3.51 |
| CRP ^a = ++ | 0.97 | 1.36 | 0.06 | 0.71 | 0.47 | -1.72 | 3.66 |
| CRP ^a = +++ | 0.35 | 1.37 | 0.02 | 0.25 | 0.80 | -2.36 | 3.06 |
| WBC (/mm ³) | -0.05 | 0.08 | -0.04 | -0.72 | 0.47 | -0.21 | 0.09 |
| RBC (million/mm ³) | 0.49 | 0.57 | 0.06 | 0.87 | 0.38 | -0.62 | 1.61 |
| MCV (µm ³) | 0.11 | 0.06 | 0.13 | 1.82 | 0.07 | -0.01 | 0.24 |
| MCH (pg/cell) | -0.15 | 0.06 | -0.12 | -2.35 | 0.02 | -0.28 | -0.02 |
| MCHC (Hb/cell) | 0.46 | 0.16 | 0.14 | 2.83 | <0.01 | 0.14 | 0.79 |
| PLT (mm ³) | 0.00 | 0.00 | -0.00 | -0.05 | 0.95 | -0.01 | 0.01 |
| RR | -1.26 | 0.12 | -0.54 | -10.25 | <0.001 | -1.51 | -1.02 |
| SBP | 0.04 | 0.02 | 0.13 | 2.00 | 0.047 | 0.00 | 0.09 |
| Fever (yes) | 0.34 | 0.82 | 0.02 | 0.41 | 0.68 | -1.29 | 1.98 |
| Cough (yes) | -0.63 | 0.81 | -0.04 | -0.77 | 0.43 | -2.23 | 0.97 |
| Shortness of breath (yes) | -1.20 | 0.88 | -0.08 | -1.36 | 0.17 | -2.96 | 0.54 |
| Sore throat (yes) | -0.76 | 1.35 | -0.03 | -0.56 | 0.57 | -3.44 | 1.91 |
| Intubation (yes) | -6.12 | 1.25 | -0.26 | -4.87 | <0.001 | -8.60 | -3.64 |
| ICU care (yes) | 0.02 | 1.19 | 0.00 | 0.02 | 0.98 | -2.34 | 2.39 |
| ICU duration (day) | -0.19 | 0.07 | -0.12 | -2.54 | 0.01 | -0.35 | -0.04 |
| Troponin (+) | -0.06 | 1.08 | -0.00 | -0.06 | 0.95 | -2.21 | 2.08 |

Note. CI: confidence interval; Std: Standard deviation; FBS: Fasting blood sugar; BUN: Blood urea nitrogen; Cr: Creatinine; Na: Sodium; K: Potassium; ALP: Alkaline phosphatase; LDH: Lactate dehydrogenase; SGOT: Serum glutamic oxaloacetic transaminase; SGPT: Serum glutamate pyruvate transaminase; PT: Prothrombin time; PTT: Partial thromboplastin time; INR: International normalized ratio; ESR: Erythrocyte sedimentation; CRP: C-reactive protein; WBC: White blood cell; RBC: Red blood cell; MCV: Mean corpuscular volume; MCH: Mean corpuscular hemoglobin; MCHC: Mean corpuscular hemoglobin concentration; PLT: Platelet; RR: Respiratory rate; SPO₂: Oxygen saturation; SBP: Systolic blood pressure; ICU: Intensive care unit.

^a Reference level: Negative.

present study, the obtained results indicated that the troponin level and the CRP level were positive in 16% and 86% of the cardiovascular patients with COVID-19, respectively.

According to a laboratory study conducted by Omer et al in 2020 in Sudan, it was found that decreased level of consciousness, SpO₂ less than 90, and LDH of more than 486 are independent risk factors for death during admission (12). In the present study, there was a significant relationship between the decrease in SpO₂ level and the increase in the death rate and the increase in the LDH level, and with the decrease in SpO₂, the death rate and the LDH level increase in cardiovascular patients with COVID-19.

A study by Huang et al in 2020 in China revealed that the cytokine storm that occurs in the wake of COVID-19 causes hepatotoxicity and an increase in liver aminotransferase, which is associated with the intensification of the inflammatory responses associated with the disease and the progression of the disease, eventually leading to the death of some ill patients (13). In the present study, the mean SGOT and ALP in cardiovascular patients with COVID-19 were higher than normal and had no significant relationship with the decrease in SpO₂ level.

In a study conducted by Liu et al in China, the findings indicated kidney damage in patients with COVID-19, and increased levels of BUN and serum Cr were associated with mortality due to kidney damage (14). In the present study, renal markers, including Cr and BUN increased in patients with COVID-19, but no significant relationship was found between the increase in BUN and Cr levels and the decrease in SpO₂.

In the study conducted by Tersalvi et al in 2020, it was concluded that troponin level has a direct relationship with the mortality of a patient with COVID-19 in such a way that with the increase in troponin level, the mortality rate due to COVID-19 increases (15). In the present study, the relationship between a decrease in SpO₂ level and positive troponin was not significant in cardiovascular patients with COVID-19.

The findings of the study of Chen et al showed that LDH is related to the severity of the disease in patients with COVID-19 with and without the previous disease. CRP, LDH, age, D-dimer, and lymphocytopenia in patients were independent risk factors in patients with comorbidities. CRP was also an independent risk factor and was associated with rapid death in patients with hypertension, which is in line with the present findings (16). In the present study, SpO₂ level had a significant relationship with the number of deaths of patients, LDH, and RR levels in cardiovascular patients with COVID-19, and with a decrease in SpO₂ levels, the death rate, LDH, and RR levels increased.

Based on a laboratory study conducted by Covino et al

in Italy, it was found that low numbers of lymphocytes and high levels of CRP and LDH are associated with the most severe type of COVID-19 disease and with a decrease in SpO₂ levels (17). In the present study, LDH and CRP levels increased and a significant relationship was found between SpO₂ and LDH and CRP¹⁺ levels. Moreover, with the increase in LDH and CRP¹⁺ levels, SpO₂ levels decreased in cardiovascular patients with COVID-19.

Furthermore, in a laboratory study carried out by Taj et al in the United States, acute liver damage was observed and increased liver enzymes were significantly associated with the most severe type of COVID-19 (18). In the present study, the level of SGOT and ALP was higher than normal, but no significant relationship was found between the level of SGOT and ALP and the SpO₂ of the cardiovascular patients with COVID-19.

Xie et al conducted a study in 2020 on 140 patients with COVID-19 in China, reporting that patients had an SpO₂ of less than 90%, and 36 patients died with a mean of 14 days after hospitalization (19). In the present study, a significant relationship was found between the SpO₂ level and the number of cardiovascular patients with COVID-19 who died, and the decrease in the SpO₂ level significantly increased the number of deaths.

Based on a study conducted by Buekers et al in the Netherlands and based on the continuous monitoring of cross-sectional oxygen levels in patients with underlying lung disease, it was concluded that patients with chronic obstructive pulmonary disease suffer from a decrease in blood SpO₂ levels more than others, and this decrease in SpO₂ level in simultaneous respiratory disease, activity, and exercise, etc. decreases more (20). In the current study, patients with chronic lung disease suffer from a decrease in SpO₂ more than others, and lung disease and oxygen level have a significant relationship in cardiovascular patients with COVID-19.

Mohammadi et al conducted a study in 2021 and concluded that severe respiratory distress decreases the level of consciousness, and decreased SpO₂ levels are among the most important outcomes of intubation in COVID-19 patients (21). In the present study, with the reduction of SpO₂ levels in cardiovascular patients with COVID-19, the intubation rate of patients increased significantly.

A study by Plotnikoff et al in 2021 in Canada indicated that the time spent by patients in the ICU depends on various factors, including SpO₂ level, consciousness level, and respiratory availability, which decreases with SpO₂ level. Moreover, consciousness and increased respiratory distress increased the duration of stay in the ICU (22). In the current study, the duration of hospitalization in the ICU in cardiovascular patients with COVID-19 increased with the decrease in SpO₂ level, and a significant relationship was found between them.

According to the study by Wang et al in China

on the FBS level and its relationship with death and the hospitalization rate of patients in the ICU, it was concluded that an increase in the FBS level by more than 126 mg/dL is directly related to the increase in the hospitalization rate of ICU duration and death of patients (23). In the present study, the mean of FBS was higher than the normal level in cardiovascular patients with COVID-19, but no significant relationship was found between the level of FBS and the SpO₂ of the patients.

Conclusion

In cardiovascular patients with COVID-19, FBS, kidney markers such as BUN and Cr, liver markers (e.g., SGOT, ALP, and LDH), and inflammatory markers (e.g., ESR) were observed to be higher than normal, while the mean SpO₂ was observed to be lower than normal. Reducing the level of SpO₂ to less than 90% was significantly related to increasing age, death, patients with a history of lung disease, the duration of hospitalization in ICU, and intubation. This reduction also led to an increase in RR, LDH, ESR, and CRP⁺ in cardiovascular patients with COVID-19.

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Competing Interests

There is no conflict of interests.

Ethical Approval

This study was approved by the Ethics Committee of Abadan University of Medical Sciences (Ethical Approval ID: IR.ABADANUMS.REC.1399.184).

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