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Measurement of Lipid Profile, LDH, Spo2 and Heart Rate in COVID-19 Patients Associated with Cardiovascular Problems in Thi Qar Province

Mohammed Mahdi abd[1*]
[1]Ministry of Education, Thi-Qar Education Directorate, Thi-Qar, Iraq

Email: biologist_mohammed@sci.utq.edu.iq

Abstract. The study aims to investigate the physiological characteristics of COVID-19 patients, considering demographic factors such as gender, age, infection severity, and associated cardiovascular complications. This is particularly relevant as several clinical trials have established a correlation between COVID-19 and cardiovascular disease, a grave illness that has impacted individuals globally. The study recruited 142 COVID-19 patients and 50 control individuals from hospitals and isolation centers in Thi Qar Province. Patients were categorized based on infection severity, with 54 moderate, 53 severe, and 35 critical cases. The findings reveal that 60.6% of patients were male, and the majorities (50%) were between 50-69 years old. Patients experienced various cardiovascular complications, including myocardial infarction (5.6%), arrhythmia (4.2%), heart failure (2.8%), and pulmonary embolism (2.8%), with 3.5% experiencing more than one adverse event.Compared to the control group, COVID-19 patients had significantly higher lactate dehydrogenase (LDH) concentrations and heart rates, but lower levels of total cholesterol (TC), high-density lipoprotein (HDL), low-density lipoprotein (LDL), triglycerides (TG), and oxygen saturation (SPO2). The study also identified gender and age-related differences in these parameters, highlighting the importance of considering individual characteristics when assessing the impact of COVID-19 on cardiovascular health.

Highlights:

1. COVID-19 patients showed increased LDH and heart rates but lower cholesterol and oxygen saturation levels.
2. Gender, age, and infection severity influenced cardiovascular health outcomes in patients.
3. The study emphasizes personalized approaches to manage COVID-19-related cardiovascular risks.

Keywords: COVID, LDH, Lipid Profile, Spo2, Cardiovascular Disease.

Introduction

Global Coronavirus Disease 2019 (COVID-19), commonly known as severe acute respiratory syndrome 2 (SARS-CoV-2), is a serious disease caused by coronavirus,

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affecting the lives of all people in the world. Several previous studies have shown that SARS-CoV-2 is similar in many biological features to SARS-CoV, including the way it enters host cells, by binding to a spike protein with angiotensin-converting enzyme 2. It is an animal virus that caused the outbreak of severe acute respiratory syndrome in 2002 [1].

Several clinical studies have reported an association between COVID-19 and cardiovascular disease. Also, COVID-19 can cause cardiovascular problems including acute coronary syndrome, arrhythmias, venous thromboembolism, and others. This is done through the host cell receptors of the virus, ACE2 (angiotensin-converting enzyme 2), which is found in the cells of the heart and blood vessels [2]. Therefore, COVID-19 can exacerbate underlying cardiovascular conditions and even precipitate new heart complications. This study aims to study of some cardiovascular diseases resulting from COVID-19 infection and investigate their causes through the following:

Assess the physiological and immunological condition of COVID-19 patients by monitoring key vital factors as lipid profile, LDH, Spo2 and heat rate. Also identify certain cardiovascular ailments resulting from COVID-19 infection.

1. Relationship between Cardiovascular Disease and COVID-19

People with cardiovascular disease (CVD) appear to be more vulnerable to getting COVID-19 and more likely to suffer a more severe version of the disease with negative clinical outcomes. Geographical area will determine the degree of this sensitivity. Scientific and medical research have confirmed that COVID-19 harms the cardiovascular system, hence aggravating the severity of the disease and causing death [3]. Acute myocardial damage—characterised by increased levels of cardiac enzymes, especially high-sensitivity cardiac troponin I (cTnI), and abnormal electrocardiographic findings—is the most often cited cardiovascular effect of COVID-19 [4]. Investigations illustrate that for most critical care patients, higher cardiac troponin levels are a negative sign. Though it may potentially directly or indirectly affect the cardiovascular system, COVID-19 mostly affects the respiratory tract, Tveit et al. [5] indicate COVID-19 directly affects the heart and blood vessel lining by binding to ACE2 receptors on their cell surfaces. This enzyme is essential for controlling various physiological processes in the heart and lungs, including blood pressure control in both healthy and sick conditions. The virus's attachment to these

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receptors alters their signal pathways, therefore generating problems in the impacted devices. Moreover, certain studies have found direct viral infection in non-respiratory organs including the brain, kidney, liver, and heart based on histological [6].

a) The occurrence of cytokine storm was triggered by systemic infections resulting from COVID-19 infection. raised levels of some inflammatory cytokines in the bloodstream, caused by hyperinflammation, have been identified in severe and critical instances. These raised levels might potentially result in cell death and malfunction of several organs. The cardiovascular system, along with other physiological systems, may undergo disturbance consequently.
b) The inflammation generated by a COVID infection leads to hypoxia of blood in the lungs, resulting in insufficient oxygen flow to the heart muscle. This leads to weakness and cardiac stress and can cause consequences such as acute myocardial damage.
c) The infection-induced inflammation triggers clotting of the capillary veins in the lungs, resulting in heightened blood flow to the heart and ultimately causing a myocardial infarction.
d) Several medicines employed to address difficulties arising from COVID infection, including corticosteroids, antivirals, and other medications, have adverse effects on the cardiovascular system.
e) Prior investigations have revealed that infections, namely COVID-19, might cause abnormalities in electrolytes, specifically hypokalemia, due to the interaction with the renin-angiotensin-aldosterone system. This results in arrhythmia.

Chronic disorders or diseases caused by COVID infection, such as hypertension, myocarditis, arrhythmia, heart attack, angina pectoris, and blood clotting in the pulmonary, heart, and brain arteries, elevate the likelihood of mortality [7]. As previously mentioned, certain drugs, such as ACE inhibitors and ACE2 blockers, have potential negative effects that might impact an individual's vulnerability to contracting COVID-19. In addition, antiviral medications can lead to various clinical symptoms, including arrhythmias [8].

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2. Lipid Profile

Recent research has found a correlation between the severity of COVID-19 and the levels of total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C). These lipid profile values are associated with disease severity and mortality. In addition, it is recognized that infection and inflammation can lead to elevated levels of plasma triglycerides (TG). COVID-19 has been associated with dyslipidemias, which are characterized by a notable decrease in HDL values [9]. Patients with infected lungs develop pneumonitis and elevated levels of macrophage cytokines due to increased quantities of oxidized phospholipids. SARS CoV-2 modifies the ability of blood vessels to allow substances to pass through and affects the functioning of the liver. This leads to a decrease in the amounts of fats in the blood by reducing the production of LDL (low-density lipoprotein) and causing cholesterol and fats to leak into the small air sacs in the lungs. Statins can reduce a cell's immunity to infection, hence raising the probability of the patient experiencing a more severe form of COVID-19. The potential ways in which statins may worsen COVID-19 symptoms by enhancing the production of angiotensin-converting enzyme 2 [10].

The occurrence of dyslipidemias in COVID-19 is mostly attributed to a cytokine storm, compromised immune response, and disrupted lipid metabolism subsequent to viral infection. The presence of dyslipidemias in COVID-19 patients should be given greater consideration due to its potential to contribute to cardiovascular illness, particularly hypertension, as highlighted by Bellia et al [11].

3. The Level of Oxygen Saturation in the Blood

Pneumonia is a characteristic feature of COVID-19, which can advance in certain instances to acute respiratory syndrome (ARDS), requiring hospitalization in an intensive care unit (ICU), and has a significant fatality rate. Indications of respiratory failure in a clinical setting encompass reduced oxygen levels, heightened respiratory rate, or an escalated requirement for supplementary oxygen. In addition, patients may encounter chest constriction and difficulty in taking deep breaths. These indicators, along with feelings of anxiety and psychological discomfort, worsen the sensation of breathlessness and cause an increase in breathing rate, requiring prompt medical intervention [12].

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SpO2 is the recommended method of monitoring during COVID-19 due to its ease and speed. Given the presence of COVID-19, it is considered appropriate to have an average SpO2 (oxygen saturation level) ranging from 92 to 96 percent. The presence of underlying illnesses, such as fever and a decrease in SpO2, is widely recognised as a major contributing factor to the hospitalisation of COVID-19 patients. This serves as a clear indication of the likelihood of infection [13].

4. Heart rate (HR)

According to Raxmatjon o'g'li [14], an average adult's resting heart rate is 60–100 beats per minute. The ventricles contract strongly during ventricular beats, whereas the atrium contracts less forcefully during atrial beats; these are the two primary categories of heartbeats. Both the atrial and ventricular nodes contribute to the cardiac electrical system, which controls the heartbeat [15].

A person's general well-being is greatly influenced by their heartbeat, which in turn impacts the rate at which blood flows throughout the body. By employing various techniques, it is feasible to precisely gauge the heart rate and ascertain its regularity. These tests are crucial for evaluating cardiovascular health and identifying any irregularities in heart rhythm. Myocarditis has also been reported in certain patients infected with the COVID-19 virus, according to earlier research. The heart's ability to pump blood may be impaired as a result. Heart failure can develop from severe cases of carditis. In addition, people infected with COVID-19 have a higher chance of experiencing a heart attack, which could be because the infection causes inflammation and blood clotting. According to Rathore et al. [16] these issues tend to manifest more frequently in older individuals and those who already have heart conditions.

To discover issues like a high pulse or cardiac arrhythmia early on, it is crucial to monitor the heart rate of individuals with COVID-19 infection. As a result, possible health problems can be identified and treated at an earlier stage. Alterations to the heart rate might reveal how bad the illness is and how it's affecting the body. Doctors can assess the patient's condition and make therapeutic adjustments based on the results of the pulse monitor. More serious heart problems are common in those who already have heart disease. It is essential to closely observe these patients' heart rhythms in order to discover the disease early and treat it

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appropriately. The patient's progress and ability to resume normal activities can be assessed through pulse monitoring as well. The timing of the patient's return to everyday activities and the planning of their long-term care are both aided by this.

5. Lactate Dehydrogenase (LDH).

An integral part of the anaerobic metabolic process is the enzyme lactate dehydrogenase (LDH). In addition to catalysing the reversible conversion of lactate to pyruvate, the enzyme is responsible for reducing NAD+ to NADH and vice versa. The usual range for LDH is 140 to 280 U/L, according to Farhana and Lappin [17].

Under normal physiological conditions, LDH activity also increases during strenuous exercise to generate lactic acid. In the absence of oxygen, lactate dehydrogenase helps convert glycolysis's final product, pyruvate, to lactate [18]. The reason why serum usually has more LDH than plasma is because of coagulation. It is possible to get an inaccurately elevated LDH test if you take aspirin, alcohol, or anaesthesia [19]. Since RBCs already have their own LDH protein, the rise in haemoglobin levels caused by hemolysis is purely theoretical. Another factor that can increase LDH serum concentration is any type of cellular necrosis. Additionally, an increase in LDH-3 occurs because of the enormous loss of platelets that occurs during a pulmonary embolism. Because LDH is released during clotting, serum typically contains a higher level of LDH than plasma. Aspirin, procainamide, alcohol, some opioids, and anaesthetics can all lead to a falsely elevated LDH test [17].

Several medical illnesses can cause an increase in serum LDH levels, including liver and renal diseases, muscular injuries, trauma, heart attacks, pancreatitis, cancer, and anaemia. Haemolysis produces an artifactual rise, which leads to false-positive high results, because red blood corpuscles (RBCs) have their own LDH protein. Furthermore, elevated serum concentrations can be observed in cases of cellular necrosis. As in pulmonary embolism, a rise in LDH-3 is linked to the extensive hemolysis of platelets, severe cases of COVID-19 often exhibit elevated LDH activity, which could be attributed to cellular damage, as well as compromised blood circulation and inadequate oxygen supply [20]. Furthermore, it is important to consider that elevated LDH activity may be associated with various medical disorders, including cardiac ischemia and degenerative processes affecting the kidney, liver,

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muscle, and red blood corpuscles. Nevertheless, it is important to investigate LDH as it may have a significant correlation with the clinical progression of COVID-19 [21].

Materials and Methods

A. Lipid Profile, Cholesterol, Triglycerides, High Density, Lipoprotein, and Low-Density Lipoprotein

Lipid profile are a system fully automated quantitative test for use with Auto analyzer cobas e111 instruments were used for the determination lipid profile of in human serum using Cobas c111 analyzers.

B. Spo2, HR, SBS and DBS

Respiratory rate and heart rate were recorded based on the doctor's examination and recorded in the patient's records.

Results

A. Lipid Profile Levels in COVID-19 Patients

The In the present study, the concentrations of total cholesterol, high-density lipoproteins, low-density lipoproteins, and triglycerides were assessed in both the group of individuals with COVID-19 and the control group. A t-test analysis was conducted with a significance level of less than 0.05 to compare the means. The data shown in table (1) indicates that there were substantial variations in the average of all these parameters between the patients and the control group. The patient group exhibits a drop in the level of all criteria in comparison to the control group.

Table 1. Lipid Profile Levels in COVID-19 Patients and the Control Group

	Patients	**Control**	**p. value**
	Mean & Std.		
TC	186.8±21.59	169.0±14.94	< 0.001
HDL	64.56±178	30.76±6.018	< 0.001
LDL	113.0±16.98	98.69±13.03	< 0.001

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TG	171.4±34.01	155.1±28.85	0.001

B. Lipid Profile Levels in COVID-19 Patients According to Sex

The findings from table (2) demonstrate that there are statistically significant variations, with a significance level of less than 0.05, in the means of the indicated criteria, except for LDL, based on the Sex of the patients. The most significant decline in all these measures was observed in male patients.

Table 2. Lipid Profile Levels in COVID-19 Patients According to Sex

	Male	**Female**	**p. value**
	Mean & Std.		
TC	163.1±19.06	171.7±25.82	0.017
HDL	29.34±6.332	33.07±5.668	< 0.001
LDL	96.69±12.30	101.70±17.60	0.050
TG	150.2±23.19	162.6±29.04	0.009

C. Lipid Profile Levels in COVID-19 Patients According to Age Groups

The results of the current investigation indicate that there are statistically significant variations, with a significance level of less than 0.05, in the average values of the fats analyses in this study and presented in table (3) when comparing them based on age. The most significant reduction in TC (total cholesterol) and TG (triglyceride) levels occurred in the fourth age group, whereas the most substantial decrease in HDL (high-density lipoprotein) and LDL (low-density lipoprotein) was observed in the third age group.

Table 3. Lipid Profile Levels in COVID-19 Patients According to Age Groups

	Age groups	**Mean & Std.**	**p. value**	**LSD Sig**
TC	<30 Years	173.2±19.32	0.075	$NS^{1,2}$, $NS^{1,3}$, $NS^{1,4}$, $NS^{2,3}$, $NS^{2,4}$, $NS^{3,4,}$
	30-49 Years	171.2±29.35		
	50-69 Years	165.3±13.34		
	> 69 Years	157.1±28.29		
HDL	**<30 Years**	37.21±8.513	<0.001	
	30-49 Years	32.01±4.701		

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	50-69 Years	30.05±5.614		$0.002^{1,2}$, $0.001^{1,3}$, $0.001^{1,4}$, $NS^{2,3}$, $0.004^{2,4}$, $NS^{3,4,}$
	> 69 Years	29.96±5.818		
LDL	**<30 Years**	101.2±15.42	<0.001	$0.001^{1,2}$, $0.020^{1,3}$, $0.014^{1,4}$, $NS^{2,3}$, $NS^{2,4}$, $0.010^{3,4}$
	30-49 Years	94.81±12.36		
	50-69 Years	93.01±15.29		
	> 69 Years	114.2±15.03		
TG	**<30 Years**	159.9±27.02	<0.001	$0.001^{1,2}$, $0.001^{1,3}$, $0.001^{1,4}$, $0.004^{2,3}$, $0.004^{2,4}$, $NS^{3,4}$
	30-49 Years	197.8±23.78		
	50-69 Years	147.7±22.30		
	> 69 Years	139.9±24.55		

D. Lipid Profile Levels in COVID-19 Patients According to Severity

An analysis of variance (ANOVA) was performed to examine the target lipid profile in relation to the severity of the disease in the current investigation. The findings presented in table (4) indicate a consistent decline in lipid levels in patients with COVID-19 as the severity of the illness increases.

Table 4. Lipid Profile Levels in COVID-19 Patients According to Severity.

	Severity	**Mean & Std.**	**p. value**	**LSD Sig**
TC	**Moderate**	178.3±23.24	<0.001	$0.012^{1,2}$, $0.001^{1,3}$ $NS^{1,4}$, $0.022^{2,3}$, $0.001^{2,4}$, $0.001^{3,4}$
	Severe	164.2±22.68		
	Critical	150.9±7.654		
	Control	184.6±24.44		
HDL	**Moderate**	34.81±4.342	<0.001	$0.001^{1,2}$, $0.001^{1,3}$ $0.001^{1,4}$, $NS^{2,3}$, $0.001^{2,4}$, $0.001^{3,4}$
	Severe	30.01±4.213		
	Critical	24.03±1.134		
	Control	60.88±12.56		
LDL	**Moderate**	109.1±14.77	<0.001	$0.001^{1,2}$, $0.001^{1,3}$ $NS^{1,4}$, $NS^{2,3}$, $0.001^{2,4}$, $0.001^{3,4}$
	Severe	93.99±11.13		
	Critical	90.29±7.112		
	Control	114.0±13.44		
TG	**Moderate**	160.9±18.34	<0.001	$0.010^{1,2}$, $0.001^{1,3}$

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	Severe	149.01±16.11		$NS^{1,4}$, $NS^{2,3}$, $0.001^{2,4}$, $0.001^{3,4}$
	Critical	141.04±13.23		
	Control	170.8±22.87		

E. Lipid Profile Levels in COVID-19 Patients According to Complications

The findings of the present study demonstrate a discrepancy in the lipid standards level based on the presence of pathological consequences associated with COVID-19 infection, within the group of diseases diagnosed in this study compared to the control group. The findings demonstrated a notable reduction in the levels of all parameters in COVID-19 patients with obstructive lung illness due to the infection, except for HDL, which exhibited the most substantial decline in patients with myocardial infarction, the table (5) shows this.

Table 5. Lipid Profile Levels in COVID-19 Patients According to Complication.

	Complications	**Mean & Std.**	**p. value**	**LSD Sig**
TC	**Non**	170.2±22.49	0.075	0.0321,2, 0.0381,3, NS1,4, 241,5, 0.0121,6, 0.0181,7, NS2,3, NS2,4, 0.0362,5, NS2,6, 0.0012,7, NS3,4, 143,5, 0.0343,6, 0.0063,7, 0.0164,5, 0.0204,6, 0.0084,7, NS5,6, 0.0015,7, 0.0016,7
	MI	155.8±21.43		
	HF	158.8±34.07		
	Arrhythmia	166.1±18.12		
	PE	144.2±8.563		
	MIX	148.1±13.76		
	Control	185.9±23.44		
HDL	**Non**	32.01±4.987	<0.001	$NS^{1,2}$, $NS^{1,3}$, $NS^{1,4}$, $NS^{1,5}$, $NS^{1,6}$, $0.001^{1,7}$, $NS^{2,3}$, $NS^{2,4}$, $NS^{2,5}$, $NS^{2,6}$, $0.001^{2,7}$, $NS^{3,4}$, $NS^{3,5}$, $NS^{3,6}$, $0.001^{3,7}$,
	MI	25.92±4.346		
	HF	26.78±5.654		
	Arrhythmia	27.40±6.121		
	PE	23.98±2.754		

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	MIX	24.97±4.452		$NS^{4,5}$, $NS^{4,6}$, $0.001^{4,7}$, $NS^{5,6}$, $0.001^{5,7}$, $0.001^{6,7}$
	Control	60.55±18.23		
LDL	**Non**	102.7±14.59	<0.001	$0.001^{1,2}$, $NS^{1,3}$, $NS^{1,4}$, $0.024^{1,5}$, $0.041^{1,6}$, $0.001^{1,7}$, $NS^{2,3}$, $NS^{2,4}$, $NS^{2,5}$, $NS^{2,6}$, $0.001^{2,7}$, $NS^{3,4}$, $NS^{3,5}$, $NS^{3,6}$, $0.002^{3,7}$, $NS^{4,5}$, $NS^{4,6}$, $0.003^{4,7}$, $NS^{5,6}$, $0.001^{5,7}$, $0.001^{6,7}$
	MI	82.03±11.75		
	HF	90.08±4.171		
	Arrhythmia	95.11±7.893		
	PE	84.76±11.43		
	MIX	87.56±9.413		
	Control	112.7±15.72		
TG	**Non**	160.9±26.23	<0.001	$0.014^{1,2}$, $NS^{1,3}$, $NS^{1,4}$, $0.005^{1,5}$, $NS^{1,6}$, $0.020^{1,7}$, $NS^{2,3}$, $NS^{2,4}$, $NS^{2,5}$, $NS^{2,6}$, $0.001^{2,7}$, $NS^{3,4}$, $NS^{3,5}$, $NS^{3,6}$, $0.011^{3,7}$, $NS^{4,5}$, $NS^{4,6}$, $0.039^{4,7}$, $NS^{5,6}$, $0.001^{5,7}$, $0.010^{6,7}$
	MI	133.7±16.76		
	HF	133.1±23.11		
	Arrhythmia	144.8±23.01		
	PE	119.2±8.231		
	MIX	134.9±9.121		
	Control	169.8±33.89		

F. SPO2, Heart Rate, and LDH in COVID-19 Patients

The factors listed in table (6) are linked to respiratory issues in individuals with COVID-19. The current study's findings indicate a notable and statistically significant reduction in blood oxygen saturation levels among patients compared to the control group. Additionally, a substantial increase in heart rate and LDH levels was seen in the patients. These results are statistically significant at a level <0.05.

Table 6. SPO2, Heart Rate, and LDH in COVID-19 Patients and the Control Group.

	Patients	**Control**	**p. value**
	Mean & Std.		
SPO2	97.12±1.143	86.79±6.372	< 0.001
HR	75.88±3.142	95.46±23.75	< 0.001
LDH	155.67±50.01	222.1±53.12	< 0.001

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G. SPO_2, Heart Rate, and LDH in COVID-19 Patients According to Sex

At a significance level of <0.05, the male group of COVID-19 patients experienced a greater decline in SPO2 level compared to the female group. The table (7) illustrates this.

Table 7. SPO_2, Heart Rate, and LDH in COVID-19 Patients According to Sex.

	Male	Female	p. value
	Mean & Std.		
SPO2	86.12±6.625	88.07±6.635	0.090
HR	95.75±22.27	92.75±20.45	0.422
LDH	226.0±56.80	214.2±45.74	0.197

H. SPO2, Heart Rate, and LDH in COVID-19 Patients According to Age Groups

The table's findings indicate that there is a negative correlation between age and SPO2 levels in COVID-19 patients, however there is a positive correlation between age and heart rate as well as LDH levels. The results exhibit statistical significance at a level of <0.05.

Table 8. SPO2, Heart Rate, and LDH in COVID-19 Patients According to Age Groups.

	Age groups	Mean & Std.	p. value	LSD Sig
SPO_2	<30 Years	94.11±2.131	<0.001	$0.014^{1,2}$, $0.001^{1,3}$, $0.001^{1,4}$, $NS^{2,3}$, $0.004^{2,4}$, $0.029^{3,4}$
	30-49 Years	88.22±5.432		
	50-69 Years	87.89±6.414		
	> 69 Years	83.01±7.530		
HR	**<30 Years**	77.95±13.24	0.001	$NS^{1,2}$, $0.025^{1,3}$, $0.001^{1,4}$, $NS^{2,3}$, $0.001^{2,4}$, $0.003^{3,4,}$
	30-49 Years	92.07±19.27		
	50-69 Years	95.01±20.01		
	> 69 Years	112.3±24.55		
LDH	**<30 Years**	218.9±36.13	0.169	$NS^{1,2}$, $NS^{1,3}$, $NS^{1,4}$, $NS^{2,3}$, $NS^{2,4}$, $NS^{3,4}$
	30-49 Years	220.9±51.77		
	50-69 Years	221.3±54.65		

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	> 69 Years	248.02±50.48		

I. SPO2, Heart Rate, and LDH in COVID-19 Patients According to Severity

The disease severity impacts the criteria indicated in table (9), with statistically significant differences seen at a significance level of <0.05. The current study observed a significant decline in SPO2 levels in patients with Critical cases of COVID-19 infection, along with an elevated heart rate and LDH levels in the same group.

Table 9. SPO2, Heart Rate, and LDH in COVID-19 Patients According to Severity.

	Severity	**Mean & Std.**	**p. value**	**LSD Sig**
SPO_2	**Moderate**	91.01±2.151	<0.001	$0.001^{1,2}$, $0.001^{1,3}$ $0.001^{1,4}$, $0.001^{2,3}$, $0.001^{2,4}$, $0.001^{3,4}$
	Severe	88.02±3.233		
	Critical	80.04±8.343		
	Control	94.99±1.234		
HR	**Moderate**	84.76±15.11	<0.001	$0.003^{1,2}$, $0.001^{1,3}$ $0.007^{1,4}$, $0.001^{2,3}$, $0.001^{2,4}$, $0.001^{3,4}$
	Severe	94.33±16.07		
	Critical	108.3±21.04		
	Control	76.06±3.715		
LDH	**Moderate**	196.8±39.22	<0.001	$0.022^{1,2}$, $0.001^{1,3}$ $0.001^{1,4}$, $0.001^{2,3}$, $0.001^{2,4}$, $0.001^{3,4}$
	Severe	226.4±50.11		
	Critical	254.6±50.17		
	Control	155.7±48.98		

J. SPO_2, Heart Rate, and LDH in COVID-19 Patients According to Complications

An analysis of variance (ANOVA) was performed on the data presented in table (10) based on the type of pathological problems arising from COVID-19 infection. The results indicated statistically significant differences among the various groups of patients, with a significant level of less than 0.05. Patients diagnosed with pulmonary embolism exhibit significantly reduced arterial oxygen saturation (SPO2) levels and elevated lactate dehydrogenase (LDH) levels in comparison to patients experiencing

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other comorbidities. COVID-19 individuals with several comorbidities experience elevated heart rate because of the infection.

Table 10. SPO_2, Heart Rate, and LDH in COVID-19 Patients According to Complication.

	Complications	Mean & Std.	p. value	LSD Sig
SPO_2	**Non**	88.57±4.591	<0.001	$0.001^{1,2}$, $0.001^{1,3}$, $NS^{1,4}$, $0.001^{1,5}$, $NS^{1,6}$, $0.001^{1,7}$, $NS^{2,3}$, $0.034^{2,4}$, $0.005^{2,5}$, $0.037^{2,6}$, $0.001^{2,7}$, $0.008^{3,4}$, $NS^{3,5}$, $0.009^{3,6}$, $0.001^{3,7}$, $0.001^{4,5}$, $NS^{4,6}$, $0.001^{4,7}$, $0.001^{5,6}$, $0.001^{5,7}$, $0.001^{6,7}$
	MI	80.10±11.20		
	HF	77.01±6.181		
	Arrhythmia	84.67±3.194		
	PE	72.15±8.434		
	MIX	84.91±4.123		
	Control	95.02±1.102		
HR	**Non**	91.76±20.68	<0.001	$NS^{1,2}$, $NS^{1,3}$, $NS^{1,4}$, $NS^{1,5}$, $0.014^{1,6}$, $0.001^{1,7}$, $NS^{2,3}$, $NS^{2,4}$, $NS^{2,5}$, $NS^{2,6}$, $0.001^{2,7}$, $NS^{3,4}$, $NS^{3,5}$, $NS^{3,6}$, $0.022^{3,7}$, $NS^{4,5}$, $NS^{4,6}$, $0.001^{4,7}$, $NS^{5,6}$, $0.001^{5,7}$, $0.001^{6,7}$
	MI	103.44±28.25		
	HF	97.90±28.12		
	Arrhythmia	104.8±26.26		
	PE	108.6±18.65		
	MIX	113.4±23.18		
	Control	76.18±3.484		
LDH	**Non**	357.3±75.42	<0.001	$0.022^{1,2}$, $NS^{1,3}$, $NS^{1,4}$, $0.001^{1,5}$, $0.016^{1,6}$, $0.001^{1,7}$, $NS^{2,3}$, $NS^{2,4}$, $NS^{2,5}$, $NS^{2,6}$, $0.001^{2,7}$, $NS^{3,4}$, $NS^{3,5}$, $NS^{3,6}$, $0.001^{3,7}$, $0.023^{4,5}$, $NS^{4,6}$, $0.001^{4,7}$, $NS^{5,6}$, $0.001^{5,7}$, $0.001^{6,7}$
	MI	415.2±98.18		
	HF	395.1±111.3		
	Arrhythmia	385.9±64.22		
	PE	491.2±35.49		
	MIX	434.6±65.37		
	Control	155.9±50.13		

Discussion

The The most prevalent causes of cardiac damage seem to be direct myocardial harm via viral involvement of cardiomyocytes and the influence of systemic inflammation [22]. COVID-19 individuals with pre-existing and/or infection-induced CVD have consistently showed a considerably worse result [23].

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Remarkably, thrombosis-related problems like pulmonary embolism, strokes, and multi-organ failure account for almost 70% of deaths connected to COVID-19 [24]. Activation of hypoxia-related coagulation factors like tissue factor (a key player in triggering the coagulation cascade), cytokine storm, neutrophil activation and release of neutrophil extracellular traps, immobility and ICU-related risk factors could explain coagulation abnormalities during COVID infection. Normally, the pro- and anticoagulant systems are tightly controlled. In some patients, SARS-CoV-2 infection seems to disturb this fragile balance and cause a blood-clotting condition. The reasons of the prothrombotic state in COVID patients seem to contrast with typical disseminated intravascular coagulation with raised plasma fibrinogen levels and D-dimers in patients [25]. Some evidence supports the existence of complex interactions between the innate immune response, coagulation, fibrinolytic pathways, the vascular endothelium, and a prothrombotic condition. Hyperinflammation seems generally connected with a number of mechanisms comprising alveolar epithelialisation, endothelial dysfunction, complement activation, monocytes/macrophages, cytokine storm, dendritic cell toxicity, platelets, and coagulant autoantibodies promoting microthrombus generation [26].

Patients with COVID-19 have fairly quickened rapid heart rates [27]. One of the main problems brought on by COVID-19 is arrhythmia [28]. COVID-19 could mechanically impair many ion channels, which would alter calcium handling, cardiac conduction and/or repolarisation characteristics, hence possibly causing arrhythmias [29].Many antimicrobials utilised as possible therapy drugs for COVID-19, such as chloroquine, hydroxychloroquine, and azithromycin, may cause QT prolongation on electrocardiography with possible proarrhythmic consequences [30]. Apart from aggravating cardiomyopathy and prior conduction anomalies, and generating arrhythmic episodes. Under several different pathways, SARS-CoV-2 might also induce electrophysiological anomalies in people without previous history of heart disease [31]. Furthermore, elevated temperatures during COVID-19 infection could affect heart rate [32].

A frequent disease state that can be found at various phases in the course of a COVID-19 patient presentation is heart failure. New or pre-existing heart failure in the context of COVID-19 can provide distinct difficulties that could affect presentation, care, and prognosis. The correct triage and treatment of these individuals depend on a careful

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knowledge of the haemodynamic and diagnostic consequences. Common in COVID-19, abnormal cardiac biomarkers can result from several different processes involving the viral entrance itself via the ACE2 receptors, direct heart damage, higher thrombotic activity, stress cardiomyopathy, and others. Many of the seen processes and manifestations may be caused by the cytokine storm seen in this epidemic. Optimal care will follow from a proper knowledge of the two-way interaction between heart failure drugs and the infection as well as the suggested COVID-19 medications and heart failure.

Pulmonary embolism is a serious complication associated with COVID-19 infection [33]. It is a condition that occurs when a blood clot forms in one of the pulmonary blood vessels and blocks blood flow to part of the lung. Where in the event of a blood clot in the deep blood vessels, such as in the legs and pelvis (called deep vein embolism), and when it separates from the place of its formation and travels through the blood circulation to the lung, where it blocks the small pulmonary blood vessels and causes a pulmonary embolism. He points out that the very high D-dimer levels observed in COVID-19 patients are not only secondary to systemic inflammation, but also reflect true coagulopathy, possibly due to virus-induced cellular activation. A prothrombotic state can lead to pulmonary embolism and then acute right ventricular failure [34]. The results of the current study showed the presence of pulmonary embolism accompanying COVID-19 infection in 4 (2.8%) of patients. While a study by Safiriyu et al [35] reported the occurrence of pulmonary embolism in 12.8% of infected patients required hospitalization. There is some difference between this study and previous studies, perhaps due to demographic factors and the sample size of each study population. Pulmonary embolism during COVID-19 infection is a serious indicator and there is a high probability that it will lead to death due to respiratory failure [36]. There are many reasons for the occurrence of pulmonary embolism in patients with COVID-19, including: the induced state of clotting resulting from severe inflammation that accompanies the infection, as well as the exaggerated immune response that causes a type of polythrombotic vasculitis [37], as well as prolonged clinical rest and lack of movement during illness can be a potential cause of blood clotting and thus pulmonary embolism, in addition to the inflammation of blood vessels caused by the virus, which increases the possibility of hypercoagulability [38].

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1. Lipid Profile in COVID-19 Patients Dependent on Sex, Age, Chronic Disease, Severity and Complications

This study examined the levels of fats in the blood of COVID-19 patients and their relationship to the severity of the disease and its resulting complications. Because fats participate in the pathogenesis and pathophysiology of viral disease, they are involved in the structure of the virus's envelope and are related to its invasion, reproduction, and release from cells, in addition to their presence primarily in the host cell membranes [39]. The results of the current study showed that lipid levels in COVID-19 patients were significantly lower compared to the control group, and the relationship is inverse with the severity of the disease [40]. This study is consistent with many previous researches, including a study [38]. While this result is not consistent with what was mentioned in the study [41], which reported that lack of movement causes an increase in fat levels in patients, and that maintaining or improving physical fitness is associated with a reduced risk of cardiovascular disease, while a decrease in Cardiorespiratory fitness increases the risk of myocardial dysfunction and cardiac mortality. The current study found a greater decrease in these lipid parameters in males compared to females. This result is consistent with the previous study by [42]. One study also reported a relationship between a low level of these parameters and the occurrence of atherosclerosis in males more than in females, which is an inflammation of the inner layer of large arteries and is the main cause of myocardial and cerebral infarction [43]. Depending on the age of the patients, the results of this study showed an inverse relationship between the level of lipid profile and age. Moreover, the current study reported that the levels of TC, HDL, and LDL decreased more in patients who had chronic cardiovascular diseases and problems and cardiovascular resulte complications from COVID-19. This result is consistent with [9]. While it is not consistent with [44].

Many viruses, including COVID-19, have the ability to affect blood fat levels, as they can affect metabolic programming and fat synthesis and modify them through their proteins such as protein S [39]. This protein has an important role in processing fats and proteins in the endoplasmic reticulum, and there may be another protein other than S and N that has the ability to stimulate the synthesis of fats and modify them [45]. Furthermore, maintaining or improving physical fitness is

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associated with a reduced risk of cardiovascular disease, while decreased cardiorespiratory fitness is associated with an increased risk of myocardial dysfunction and cardiac mortality [41]. Low lipid levels may be caused by an immune response, or by leaky blood vessels in the lung parenchyma as a result of damage to the vascular lining. An increased concentration of pro-inflammatory cytokines may be responsible for a sharp decrease in plasma lipid levels during the acute phase response.

2. SPO2, Hart Rate, LDH in COVID-19 Patients Dependent on Sex, Age, Chronic Disease, Severity and Complications

When conducting a blood oxygen saturation test for patients in this study, the results showed clear significant differences between COVID-19 patients and the control group. As the rate of SPO2 in patients clearly decreased, this decrease certainly occurred due to complications of infection that cause a disturbance in gas transport as a result of inflammation, damage to lung tissue, etc. This result is consistent with previous studies, including the study [46]. An imbalance in blood oxygenation leads to many clinical consequences in various organs of the body [47]. Maintaining cellular oxygen levels is crucial because insufficient or excess oxygen leads to increased levels of reactive oxygen species (ROS), and thus, both oxygen delivery and consumption are finely regulated by many different molecular mechanisms [48]. Therefore, doctors and health care workers resort to compensating for the deficiency in blood oxygen through artificial ventilation.

In the results of the current study, it was found that the heart rate increased in COVID-19 patients, which usually occurs as a condition accompanying a lack of blood oxygen as a mechanism to accelerate blood movement and thus compensate the tissues with the oxygen needed. Acceleration in the heart rate also usually occurs in the event of an increase in body temperature as well. This is the result. It is consistent with a study by [49]. Under conditions of lack of oxygen, the level of LDH increases, and this is what was recorded in this study as in many previous studies. LDH is an enzyme that participates in energy production by converting lactate into pyruvate. It is present in almost all cells of the body, with its highest levels in the heart, liver, lungs, muscles, kidneys, and blood cells, and its increase may be related to intravascular erythrocyte and tissue cell damage [50]. The enzyme participates

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in the anaerobic metabolism of glucose when oxygen is absent or when it is available in a limited way. Elevated LDH level is a general indicator of acute or chronic tissue damage and is considered an inflammatory marker, usually increasing during acute and severe lung damage [51]. When cells are exposed to anaerobic or hypoxic conditions, ATP production is disrupted by oxidative phosphorylation. This process requires cells to produce energy via alternative metabolism. As a result, LDH is upregulated in such conditions to meet the need for energy production. However, the lactate produced during anaerobic conversion of glucose hits a metabolic dead end. It cannot undergo further metabolism in any tissue except the liver. Hence, lactate is released into the blood and transported to the liver, where LDH performs the reverse reaction to convert lactate to pyruvate through the Cori cycle [17].

It was noted during the test results of the current study that there is a significant negative relationship between the severity of the injury and the level of SPO2 in COVID-19 patients, as well as the existence of a positive, significant relationship between the severity of the injury and the level of LDH, the heart rate of the patients. This result is consistent with what was recorded in a study by [52]. Moreover, the current study recorded a greater decrease in blood oxygen saturation, irregular heartbeat and an increase in its rate in COVID-19 patients who suffer from chronic heart problems, as well as patients who had cardiovascular problems and pulmonary embolism as complications of infection, in addition, the results of the current study recorded that the level of LDH in COVID patients is higher when there are chronic cardiovascular problems, and it is also higher in patients who have had cardiovascular problems as a complication of the infection. These data are consistent with the study of [49], as their study stated that the high level of LDH associated with cardiovascular problems is a good indicator of the risk of infection and a predictor of negative outcomes. A study [50] also reported that as the disease worsens, there will be increasing levels of D-dimer, lactate dehydrogenase (LDH), and IL-6, indicating a higher risk of death. LDH was previously used for the first time to diagnose myocardial infarction, because it reflects necrosis of cardiac myocytes, as LDH can reflect damage to lung cells due to acute respiratory failure and damage to the myocardium and other organs [52].

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The current study concluded the following:

COVID-19 patients exhibited markedly elevated indicators, LDH and Spo2, and dicrease in lipid profile and Spo2 in comparison to the control group. Lipid profile, LDH, heart rate and Spo2 are influenced by factors such as increasing age and in males. Moreover, there exists a signifecant direct correlation between the intensity of COVID-19 infection and the concentrations of indicators.

Hypoxia resulting from respiratory illness, systemic inflammation, increased coronary blood flow, and direct effects of the virus on the heart can also cause acute myocardial injury. Patients who already have cardiovascular disorders are at an even greater risk of experiencing complications related to COVID-19.

Conclusions

The study highlights a significant association between COVID-19 infection and alterations in lipid profiles, heart rate, and oxygen saturation, with variations influenced by age, gender, and the severity of infection. Notably, these parameters were found to be critically altered in patients with pre-existing cardiovascular conditions, exacerbating the risk of complications such as myocardial infarction and pulmonary embolism. The findings underline the importance of early monitoring and management of cardiovascular indicators in COVID-19 patients to mitigate adverse outcomes. This research implicates the need for integrated clinical strategies that address both the respiratory and cardiovascular implications of the disease. Future studies should explore the molecular mechanisms linking COVID-19 to lipid metabolism and cardiovascular dysfunction, as well as the long-term outcomes of affected patients to enhance therapeutic interventions.

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