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Research Article

Assessment of the lipid profile in individuals infected with COVID-19

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Abstract

Background: The World Health Organization declared the coronavirus global on March 11, 2020. Lipid dysregulation may be a factor in COVID-19 infection-related morbidity and mortality. The dyslipidemias associated with COVID-19 are likely caused by immune system dysfunction, cytokine storm, and poor lipid metabolism following viral infection. Aim: To identify lipid alterations in COVID-19 patients and their connection to the course of their illness. **Methods:** Patients with confirmed COVID-19 infection who were admitted to the hospital between April and August of 2021 were the subjects of our prospective study. Within the first 24 hours after admission, all patients had their lipid profiles (LDL, HDL, Triglyceride, and Cholesterol) assessed. The patients were then monitored to ascertain how they fared. **Results:** The study comprised eighty patients; of them, twenty-five (31.2%) were diagnosed as having intermediate disease, and 68.2% of patients as having severe disease. Depending on their state of hospital discharge, the patients were split into two groups: group I, or the survivor group, consisted of 59 (74%) patients, and group II, or the non-survivor group, consisted of 21 (26%) patients. Low HDL was present in 100% of cases, high LDL was present in 47 (58.8%), high triglyceride in 46 (57.5%) and increased cholesterol was present in 40 (50 %) of cases. Triglyceride was significantly higher in non survivors than survivors (194.71±81.14 VS 149.78±50.68, p=0.004). While HDL, LDL and cholesterol levels showed non-significant difference between survivors and non survivors. **Conclusion:** Patients with COVID-19 infection experience disturbances in their lipid metabolism, which may be used as a predictor for their outcome and mortality.

Key words: COVID-19, Lipid dysregulation, LDL, HDL

Introduction

The World Health Organization declared the coronavirus to be a global pandemic on March 2020[1].

Lipids play a role in the pathophysiology of viral diseases as well as viral pathogenesis. Lipids constitute the viral envelope and play a role in both viral invasion and replication. When they enter and exit the host cell membrane, they participate in membrane fusion and replication[2].

The dyslipidemias associated with COVID-19 are likely caused by immune system dysfunction, cytokine storm, and poor lipid metabolism following viral infection. Lipid profile alterations in individuals with COVID-19 should be given more attention since dyslipidemias might result in cardiovascular disease, especially hypertension [3]. Patients

with COVID-19 have different lipid profiles than healthy people. Furthermore, the severe version of the disease has a different lipid profile than the

less severe type of COVID-19 [3]. Lipid dysregulation may be a factor in COVID-19 infection-related morbidity and mortality.
The current study aimed to identify lipid alterations in COVID-19 patients and their relation to patient outcome

Patients and Methods
The current study was a prospective study that carried out on individuals with a verified COVID-19 infection who underwent admission at the Minia Cardiothoracic University Hospital between April and August of 2021. Eighty patients were included in the study and followed up until their discharge to determine their outcome.
Every patient underwent a thorough history that included clinical presentation, risk factors, and a general and local chest examination. All patients underwent routine laboratory testing, including CBC, liver, and renal function tests. Also, Lipid profile (LDL, HDL, Triglyceride, Cholesterol) were measured within the first 24 hours of admission for every patient.
Lipid study was done by collect about 5 ml from venous blood and blood was left to clot then centrifuged. The supernatant serum was used for measurement levels of cholesterol, triglyceride, HDL and LDL. Cholesterol and triglyceride were measured by automated chemistry analyzer (Mindray – BS-800M, China). HDL was detected by enzymatic colorimetric method using (Human Diagnostics Kit, Wiesbaden, Germany) while LDL was calculated through a formula: (total cholesterol - (HDL + triglycerides /5) in mg/dl)

Ethical consent:
Every patient received an explanation of the purpose of the current study. There were no ethical dilemmas with the laboratory or radiological techniques because they were normal practice. Every patient consent to take part in the research. Approval No. 5:2/2021 was acquired with the consent of the Minia Faculty of Medicine Research Ethics Committee. The Declaration of Helsinki, the World Medical Association's code of ethics for human subjects research, has been followed in the conduct of this work.

Statistical analysis
The SPSS (Statistical Package for Social Sciences) version 22 for Windows® (IBM SPSS Inc., Chicago, IL, USA) was used to code, process, and analyze the data that were gathered. The Shapiro Walk test was used to determine if the data were normally distributed. Frequencies and relative percentages were used to display the qualitative data. To determine the difference between two or more groups of qualitative variables, the chi-square test ($\chi 2$) was used. The standard deviation (SD) of quantitative data was reported as mean ± SD. To compare two independent groups of regularly distributed variables (parametric data), the independent samples t-test was employed. A P value of less than 0.05 was deemed significant.

Results
The current study revealed low HDL in 100% of cases, high LDL in 47 (58.8%), high triglyceride in 46 (57.5%) and increased cholesterol in 40 (50 %) of cases.
Among COVID-19 patients, median age of non survivors was higher than survivors (59.67± 11.15 VS 56.85± 12.94 years). Also, non survivors had higher BMI than survivors (27.05±3.64 VS 25.78±2.90 kg/m2). As regard sex, there was no statistical significance (p=0.943) as shown in table.(1)
This study showed that non-significant difference regarding HDL, cholesterol, triglyceride levels or LDL among patients who needed NIV and those who did not need NIV, as demonstrated by table .(2)
It was found that LDL, cholesterol and triglyceride levels were higher among patients who needed IMV than those who did not need IMV (91.16±32.96 VS 90.77±34.89, 166.26± 44.94 VS 159.72±38.48and 182.63± 81.77 VS 156.02±54.74 mg/dL) and HDL level was lower among patients who needed IMV than those who did not need IMV (37.26±7.26 VS 38.22± 6.42), but without a statistical significance as shown in table.(3)

Table (4) showed that Triglyceride was significantly higher in non survivors than survivors (194.71±81.14 VS 149.78±50.68, p=0.004). While HDL, LDL and cholesterol levels showed non-significant difference between survivors and non survivors (38.08±6.58vs 38.05±6.81, 90.37±34.96 vs 90.33±33.21and 158.61±38.56 vs 166.62±44.29, p= 0.982, p= 0.966 and p=0.434 respectively).

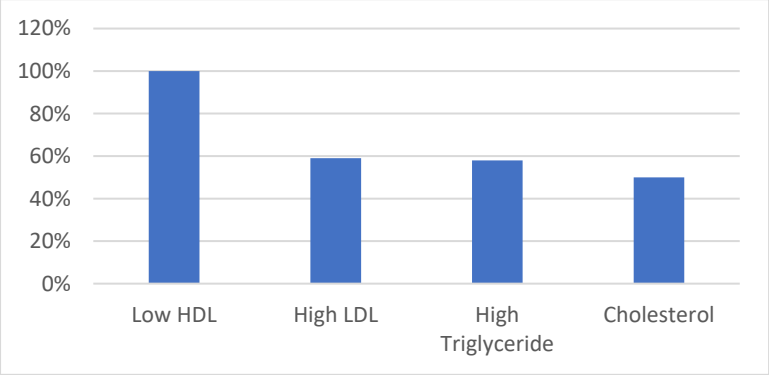

Fig (1) the prevalence of dyslipidemia among the whole studied group

Table (1): Demographic data among survivors and non survivors

Variable	Survivors (59)	Non survivors (21)	P value
Age (Mean ±SD)	56.85± 12.94	59.67± 11.15	0.378
BMI kg/m2 (Mean ± SD)	25.78±2.90	27.05±3.64	0.112
Gender No (%) Male	36 (61.0%)	13(61.9%)	0.943

(*) P <0.05 significant. SD= standard deviation BMI = body mass index

Table (2): Lipid profile in patients according to the need for noninvasive ventilation (NIV)

Variable (Mean± SD)	Patients needed NIV. (n=45)	Patients did not need NIV (n=35)	P value
HDL (mg/dL)	38.61±6.86	37.42±6.33	0.44
LDL (mg/dL)	89.41±33.09	90.85±36.60	0.86
Cholesterol (mg/dL)	163.65±39.90	155.73±40.60	0.39
Triglyceride (mg/dL)	170.83±74.08	147.97±41.20	0.11

SD= standard deviation , HDL= high density lipoproteins, LDL= low density lipoproteins.
*Significant if P <0.05

Table (3): Lipid profile in patients according to the need for invasive mechanical ventilation (IMV)

Variable (Mean± SD)	Patients needed IMV (n=19)	Patients did not need IMV (n=5)	P value
HDL (mg/dL)	37.26±7.26	38.22± 6.42	0.586
LDL (mg/dL)	91.16±32.96	90.77±34.89	0.966
Cholesterol (mg/dL)	166.26± 44.94	159.72±38.48	0.537
Triglyceride (mg/dL)	182.63± 81.77	156.02±54.74	0.108

SD= standard deviation , HDL= high density lipoproteins, LDL= low density lipoproteins.
*Significant if P <0.05

Table (4): Lipid profile among survivors and non- survivors

Variable (Mean± SD)	Survivors (59)	Non survivors (21)	P value
HDL (mg/dL)	38.08±6.58	38.05±6.81	0.982
LDL (mg/dL)	90.37±34.96	90.33±33.21	0.996
Cholesterol (mg/dL)	158.61±38.56	166.62±44.29	0.434
Triglyceride (mg/dL)	149.78±50.68	194.71±81.14	0.004

SD= standard deviation , HDL= high density lipoproteins, LDL= low density lipoproteins.
*Significant if P <0.05

Discussion

The severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) that is causing the coronavirus disease 2019 (COVID-19) pandemic is a global public health emergency. Improving our comprehension and treatment of corona virus disease (COVID 19) requires defining the clinical traits and related outcomes of people with the illness.

Disturbance of lipid metabolism has been observed in the course of COVID 19 infection [4] and it was reported that the use of statins had a positive effect in the prognosis of patients with COVID 19 infection [5].

In the current study; Low HDL was present in 100% of cases, high LDL was present in 47 (58.8%), high triglyceride in 46 (57.5%) and increased cholesterol present in 40 (50%) of cases.

In the current study, the median age of non survivors was higher than the survivors (59.67± 11.15 VS 56.85± 12.94 years old). Also, non survivors had higher BMI than survivors (27.05±3.64 VS 25.78±2.90 kg/m2). However, Age, sex, and body mass index (BMI) did not significantly differ between survivors and non-survivors (p value=0.38,0.11and 0.94 respectively). This is in agreement with Goicoechea et al study and Wang et al study[6].While Almazeedi et al study and Gayam et al study showed significantly higher BMI (33.0 ±4.7 vs 26.8 ±5.9 p<0.000 and 31.8 vs 28.3 ,p= .002) and older age (55.0 ± 10.1vs 38.7 ± 15.1 p<0.000 and 71 vs 63 years p<.001respectively) among dead patients due to COVID-19 than alive but they did not find significant difference between alive and dead as regard sex [7, 8]. The difference could be due to racial variation and different sample size.

This study showed that there were no notable variations across the patients who needed mechanical ventilation (NIV and IMV) and patients who did not need mechanical ventilation regarding their lipid profile. It was found that LDL, cholesterol and triglyceride levels were higher among patients who needed IMV than those who did not need IMV (91.16±32.96 VS 90.77±34.89, 166.26± 44.94 VS 159.72±38.48and 182.63± 81.77 VS 156.02±54.74 mg/dL) and HDL level was lower among patients who needed IMV than those who did not need IMV(37.26±7.26 VS 38.22± 6.42) , but without a statistical significance .Also, simmonet et al observed no significant relation between dyslipidemia and the need for IMV in multivariate logistic regression analysis[10].

Another study demonstrated that dyslipidemia did not predict the need for mechanical ventilation in a multivariate analysis model [11].

In the systematic review of Zaki et al., revealed that COVID-19-infected group had consistently lower levels of total cholesterol, HDL cholesterol, and LDL cholesterol compared to the control group. Notably, individuals with a high severity of disease had significantly lower HDL cholesterol levels. [12].

Based on both Hariyanto and Kurniawan metanalysis and Choi et al umbrella review, Dyslipidemia appears to be linked to a higher chance of developing a serious COVID-19 infection. [13,14]. This outcome can be explained by a number of theories. People who suffer from dyslipidemia exhibit elevated levels of low-density lipoprotein (LDL). In atherosclerotic plaques, this LDL may interact with macrophages to raise the expression of inflammatory genes. Cytokines and chemokines are expressed at elevated quantities in human atherosclerotic plaquesLDL buildup causes cholesterol crystals to develop in macrophages, which triggers the activation of inflammasomes. Proinflammatory cytokines like IL-1B and IL-18

will therefore be secreted more often as a result of activation of the inflammatory cascade [15] . Through cytokine storm syndrome, elevated levels of proinflammatory cytokines are linked to catastrophic outcomes in COVID-19 infections [16]. Moreover, individuals with high levels of LDL, usually have low levels of HDL. The innate immune response is regulated by HDL itself. Consequently, insufficient HDL will lead to dysregulation of the innate immune response, the body's initial line of defense against infection, including COVID-19 infection.[17]. Lastly, endothelial dysfunction is brought on by the buildup of LDL and triglycerides in dyslipidemia patients [18]. And because endothelial cells also express the ACE2 receptor, which is the receptor for SARS-CoV-2, endothelial dysfunction may be exacerbated in COVID-19 infections [19]. Combining these will result in the emergence of cardiovascular issues, which may have a serious effect on the patient's prognosis.

The heterogenicity of the previous results may be due to different factors. There were not much research that provided information about the correlation between dyslipidemia and COVID-19 infection. Since the COVID-19 pandemic is evolving so quickly and with such dire ramifications, a lot of reports are released without a thorough assessment of the data quality. The existence of confounding variables that may alter the association between dyslipidemia and COVID-19 severity, such as patient age and nutritional status, must still be taken into account. No study specifies the precise cut-off number or when to measure lipids in order to diagnose dyslipidemia.

In the current study, we found that triglyceride is significantly higher in non survivors than survivors (194.71±81.14 versus 149.78±50.68, P value =0.004), while high-density lipoprotein, low-density lipoprotein and total cholesterol were not significantly different between survivors and non survivors (38.08±6.58vs 38.05±6.81 , 90.37±34.96 vs 90.33±33.21and 158.61±38.56 vs 166.62±44.29, p= 0.982,p= 0.966 and p=0.434 respectively). Grasellie et al study found that hypercholesterolemia was significantly prevalent with increased mortality at univariable and multivariable analysis[20]. Also, Liu et al observed that individuals with dyslipidemia were 2.13 times more likely to die than those without the condition. (95% CI 1.84–2.47, P = 0.001, I2 = 66.4%)[19].

While other studies showed that the COVID-19 morality and dyslipidemia did not significantly correlate. [6]

This conflict between studies can be explained by many obstacles facing them. Since most of the investigations were retrospective, additional well-planned studies with more prospective research are needed to confirm our findings. The adjusted variables are not completely consistent across the included studies. The majority of research does not specifically identify the kind of dyslipidemia, such as elevated levels of total cholesterol, triglycerides, high-density lipoprotein, and low-density lipoprotein. When more information is available, research on the connection between a particular kind of dyslipidemia and COVID-19 mortality should be the main focus.

Conclusion

Patients with COVID-19 infection experience disturbances in their lipid metabolism, which could influence their prognosis and mortality.

Study limitation

The small patient population and dearth of information regarding comorbidities and drugs given by study participants limit the study.

Abbreviation

CBC : complete blood count
LDL : low density lipoprotein
HDL : high density lipoprotein
BMI : body mass index

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