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Impact of COVID-19 on CRP, Lipid Profile and Relationship with The hormone Adiponectin in the Province of Al-Najaf Al-Ashraf

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Abstract:
It is seen as an essential element in explaining the connection between COVID-19 respiratory failure and obesity. But few studies have looked at hypoadiponectin, or low levels of adiponectin, in COVID-19 patients. Blood samples were taken from a total of (100) patients who were with COVID-19 and divided into two groups, blood samples for patients with mild infection (50) and blood samples for severely injured (50), at the same time they were also collected from healthy people (50). Blood sample for monitoring, July 2023 to December 2023 at Al-Najaf Al-Ashraf Hospitals. Adiponectin and adiponectin hormone controls were tested for patients with COVID-19, cholesterol - control cholesterol for patients with COVID-19, triglycerides controls – triglycerides patients with COVID-19, low-density lipoprotein controls - low-density lipoprotein patients with COVID-19, C-reactive protein controls - C-reactive protein patients with COVID-19 controls, high-density lipoprotein - high-density lipoprotein patients with COVID-19.

Introduction

The 2019 coronavirus disease (COVID-19) is a potentially fatal illness that has sickened a large number of people (Wu JT et. al 2019). SARS-CoV-2, the disease-causing virus, was initially identified in Wuhan, Hubei Province, China, in December 2019. It quickly spread throughout China and other developing countries (Guan et al., 2019). SARS-CoV-2 was present when the World Health Organization (WHO) declared COVID-19 a pandemic on March 11, 2020 becomes first coronavirus to cause pandemic affecting humans. COVID-19 was first identified as a national emergency for global concern by the WHO on January 30, 2020 [WHO. 2020]. As of May 24, 2021, there were more than 166 million confirmed instances of the COVID-19 pandem (WHO. 2019). The biggest issue currently influencing public wellness, social advancement, and economic expansion is really the fast transmission of the whole communicable disease (Xinhua. 2020). The most recent and seventh human-infecting member of the coronavirus family, Positive-sense single-stranded RNA virus called SARS-CoV-2 is the same as the Middle East Respiratory Syndrome coronavirus (MERS-CoV) and the severe acute symptoms coronavirus (SARS-CoV) (Andersen et.al. 2020). SARS-CoV-2 exhibited additional 80 sequence commonality with two SARS-like viruses produced from bats, according to a sequence analysis. Unknown processes may be at play here. Using an anti-inflammatory hormone called adiponectin may contribute to

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COVID19 breathing difficulty (Lockhart & O'Rahilly, 2020). However, a solitary study19 looked at adiponectin levels in COVID patients (Zelst et al., 2020). Lipids play a crucial role in lung biology and the pathogenesis of viral diseases since they are the architectural underpinnings the membranes of viruses and cells (Lorizate et al., 2020). In order to produce fats for their envelope, virus targeted the recipient cell' fat production and signaling alteration processes (Murillo et al., 2015). Lipids' functions in membrane fusion, encapsulation, and modification are essential for the spread of viruses. Viral entry and release require passage through a host cell membrane because the viruses replicate within the human host (Lorizate et al.,2020). Lipids have a number of functions in the viral invasion, including entrance cofactors, fusing cofactors, and directly and indirectly virus receptors (Murillo et al., 2015). Additionally, lipids play a crucial role in the innate and adaptive immune systems' response to infections (Wendel et al., 2007) Proteomics investigation utilizing mass spectrometry (MS) found that disruption about lipid metabolism may increase the development of COVID-19 (Shen et al., 2020). Furthermore, one study discovered suggests the onset of hypolipidemia in people with moderate COVID-19 and worsens with illness severity and duration (Wei et al., 2020).

2.MATERIALS AND METHODS

Included all COVID-19 patients who agreed to provide blood samples to the intensive care unit (ICU) bio repository. Then chose healthy individuals who did not have COVID 19. Real-time SARS-CoV2 assays performed by a public health laboratory in Al-Najaf Al-Ashraf province verified COVID-19 diagnosis. Adipokine plasma samples were taken at ICU admission, as well as 24 and 72 hours later. Its Milliplex Humans Adiponectin Magnetic Bead Panel 1 kit (Millipore Sigma; Burlington, Massachusetts) used to measure the concentrations of humans adiponectin per plasma in accordance with the manufacturer's recommendations. The measurement distributions of the aforementioned set of outcome characteristics were compared between the COVID-19 cohort and other healthy cohorts as part of a hierarchical hypothesis test. The geometric mean of the COVID19 respiration failed cohort result interval scale spread was first checked using a worldwide null test for each outcome to see if it differed derived from the geometric means of the additional viral respiratory distress variable. Result measurement distributions are consistent regardless of when blood was collected. A time of blood draw when the geometric mean of the COVID-19 respiratory failure outcome variable measurement distribution differed from the geometric mean of the other infective respiratory distress conclusion variable distribution at the 0.05 level of significance was the subject of a second set of hypotheses to see if the null hypothesis was accepted globally at that level. Age, body mass index (BMI), prior diabetes mellitus, enteral nutrition, glucocorticoids, total insulin, APACHE II score, and Sequential Organ Failure Assessment (SOFA) scoring system were all taken into account while calculating the results. It was corrected across multiple comparison tests using the Tukey method. Aspartate aminotransferase (AST),Alanine aminotransferase (ALT), high-density lipoprotein (HDL),low-density lipoprotein cholesterol (LDL-C), triglycerides (TG) and total cholesterol (TC) were all measured using a Cobas analyzer. The classification COVID-19 was used to compare all parameters. Furthermore, the lipid profiles of the control and COVID-19 groups were compared. Blood samples taken at admission were used for all tests, which were performed while fasting.

3.Samples Collection

3.1 Samples

The study included 150 specimens patients , with 100 samples collected from patients with COVID-19 (mild, severe) and 50 samples collected as controls over a six-month period (from July 2023 to December 2023). The patient's age ranged from (40 years -70 years). Blood samples are taken from COVID-19 sufferers using a disposable sterilized EDTA and gel tube. To maintain this tube with specimen, they remain moist until taking to laboratory. Each sample was instantly made using CRP, a lipid profile, and an hourly ratio of the adiponectin hormone.

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3.2 Statistical study

The outcomes and case studies from the current study were examined using the Statistical Package for Social Sciences (SPSS version 26, Inc., Chicago, IL, USA). When the value is less than 0.05 determine the significance of the P-value.

4.RESULTS

In this study, 150 samples—50 mild, 50 severe, and 50 controls—from COVID-19 patients were used as samples. Compared to the COVID-19 cohort, these patients were noticeably older. Males predominated in both the COVID-19 and control conditions. The findings of this analysis show a relationship between the extent of the COVID-19 illness and changed lipid profile measures, with CRP being significantly elevated in all upper COVID-19 classes. A negative association between lipids and acute phase reactants generated as pro-inflammatory cytokines in COVID-19 patients was attributed to lower levels of LDL, HDL, and TG, suggesting the mechanism for immune upregulation by HDL and its metabolites. In severe instances compared to mild cases and controls, all lipid profiles dropped progressively and dramatically. Even after controlling for age, gender, BMI, and other variables, The correlation of lipid profile with C-reactive protein (acute phase reactants) and adiponectin levels was considerably lower in COVID-19 respiratory failure patients. When COVID-19 respiratory failure cases are compared to control specimens, adiponectin levels appear to be lower. The seriousness of COVID-19 sickness among all individuals was predicted using logistic regression analysis. The model proposed LDL, HDL, BMI, TG, and CRP as distinct risk factors for COVID-19.

5.Discussion

While acute phase reactants like CRP and acute phase reactants were predictive of severe COVID-19 disease, adiponectin, LDL, HDL, and TG levels all reduced dramatically having COVID-19 sickness that is severe. This is the first prospective study using lipid profiles from actual COVID-19 patients. The results demonstrate that lipid levels begin to decline in moderate instances and that lower lipid levels are inversely correlated utilizing acute phase reactants. Additionally, it shows how COVID-19's changed Lipidology's biochemistry and pathological development.

Infected by viruses change the hosts' lipid biomarkers, which disrupts cholesterol rafts. It benefits them get through defenses of the host. In COVID-19, there is malfunction of LDL-C and HDL particles in responses of acute inflammation circumstances known as cytokines storm. Additionally, an increase in pro-inflammatory cytokines can result in reduced lymphocytes, consumption of TC, TG, albumin, ApoA1, LDL-C and HDL (Kočar et al., 2021). Therefore, our research implies that the lowered lipid characteristics might be used to predict the severity of COVID-19 disease (Wei et al., 2020). Similar research from China revealed COVID-19's lipid profile and other clinical characteristics (Hu et al., 2020). Patients had considerably lower levels of LDL-C, HDL, and TC, and this change was gender-specific. Males more frequently showed lower lipid levels in the examination, in contrary to our study, which was conducted on females. In only COVID-19 critical cases could High density lipoprotein levels significantly decline, even though all COVID-19 classes reported significant declines for LDL-C, TC, and TG (mild, severe, critical). Only the crucial COVID-19 cohort showed significantly decreased HDL, LDL-C, TG, and TC values, according to our research. Preprint, in contrast to previous studies, reveals considerably increased LDL-C levels compared to control groups, as well as a favorable link with COVID-19 severity and enhanced lipid profile modifications (Wei et al., 2020). Similar results have been shown in HIV patients, who had high plasma triglycerides and reduced HDL and LDL-C (Grunfeld et al., 1992). Additionally, individuals with HIV are more prone to experience lipid abnormalities such hypocholesterolemia and lower levels of free fatty acids (Shor et al., 1993). A 12-year study of lipid metabolism in SARS-recovered individuals discovered Very-low-density lipoprotein (VLDL) cholesterol is increased and triglycerides are high (Wu et al., 2017). These results may be explained by the components of HDL, which include esterified cholesterol, apo-lipoproteins, and triglycerides. These lipid particles are involved in a variety of processes, including small-vessel vasodilation, oxidation and the generation of free radicals,

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Apoptosis, thrombosis, infection, and inflammation are all possible outcomes (Taborda et al., 2019). Apart from being an anti-inflammatory lipoprotein, HDL also suppresses inflammatory mediators by inactivating T-cells and macrophages (Thacker et al.,2016). A surge in pro-inflammatory cytokines via COVID-19 shows the existence of systemic inflammation. HDL can limit monocyte as well as neutrophil activation while also acting as an antioxidant, allowing for the elimination of oxidized lipids and the neutralization of oxidative agents. As a result, its inflammatory response via host cells is reduced (Kaji ., 2013). Oxidation of HDL and LDL-C during a cytokine storm in COVID-19 raises immunological activation (Kelesidis et al., 2011). Given that HDL has an inhibitory impact, we may infer that immunological modulation in COVID-19 is the root cause of the reduced lipid levels seen in this study. There were several restrictions on this inquiry. First, the group had a range in the period of time between both the beginning of symptoms and specimen collection. The study may thus depict inconsistent outcomes of the COVID-19 illness course. The precise change in lipid profile along the illness course of the COVID-19 cohort may have been skewed towards lower cholesterol levels in these patients since many patients already were receiving statin medication. Third , the control group patients were healthy adults with no severe or critical acute illness. This may have resulted on sample selection bias, which would have produced unusual findings in this experiment. Fourth, no further information on the changes with in lipid profile was acquired to follow the dynamics of COVID-19. For a more exact explanation of these events in the COVID-19 cohort, this will be necessary.

Paired Samples Test									
		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	Age control – age covid19	.580	6.383	.903	-1.234-	2.394	.643	49	.524
Pair 2	Gender control – gender covid19	-.040-	.570	.081	-.202-	.122	-.496-	49	.622
Pair 3	Adiponectin control Adiponectin covid19	2.46460	1.78379	.25227	1.95765	2.97155	9.770	49	.000
Pair 4	Cholesterol control – cholesterol covid19	-.40960-	.87283	.12344	-.65766-	-.16154-	-3.318-	49	.002
Pair 5	TG control – TG covid19	-.15300-	.29384	.04156	-.23651-	-.06949-	-3.682-	49	.001
Pair 6	LDL control – LDL covid19	.40022	.71382	.10095	.19735	.60308	3.965	49	.000
Pair 7	CRP control – CRP covid19	-39.90640-	10.12145	1.43139	-42.78289-	-37.02991-	-27.879-	49	.000

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Pair 8	HDL control – HDL covid19	.42980	.22023	.03114	.36721	.49239	13.800	49	.000
Pair 9	BMI control – BMI covid19	.46520	2.60693	.36867	-.27568-	1.20608	1.262	49	.213

6. Conclusions

CRP was found to be considerably greater in all COVID-19 upper strata, indicating a correlation between the severity of the disease and various lipid profile data. Our findings showed that HDL and its metabolites regulate the immune system by associating low levels of LDL, HDL, and TG with a negative correlation between lipids and acute phase reactants, which are generated as pro-inflammatory cytokines in COVID-19 patients. Compared to moderate cases and controls, acute patients' symptoms are more severe and progressive. Despite controlling for age, gender, body mass index, and other variables, patients with COVID-19 respiratory failure had a considerably weaker correlation between their lipid profile and levels of adiponectin and C-reactive protein (acute phase reactants).

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