Association of Low-Density Lipoprotein Levels with COVID-19 Severity: Insights from a Single-Center Cross-Sectional Study in Northern Greece

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Article

Association of Low-Density Lipoprotein Levels with COVID-19 Severity: Insights from a Single-Center Cross-Sectional Study in Northern Greece

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Abstract: Objective: To examine the relationship between low-density lipoprotein (LDL) levels and clinical outcomes in hospitalized COVID-19 patients in Northern Greece. **Methods**: A retrospective analysis was performed using data from 208 COVID-19 patients. Lipid profiles [including LDL (low-density lipoprotein cholesterol), HDL (high-density lipoprotein cholesterol), and triglycerides], prior antilipidemic treatment, and clinical outcomes were evaluated. Statistical analysis was conducted using SPSS version 19. Patients: 208 COVID-19 patients from Northern Greece. **Results**: The mean LDL level was 84.12 mg/dL, with no significant differences observed between survivors and non-survivors. Prior antilipidemic treatment did not significantly affect outcomes. Elevated triglyceride levels were noted in obese patients (BMI ≥ 30 kg/m^2), and lower HDL levels were associated with higher CRP (C-reactive protein) levels. Although LDL levels declined over time in non-survivors, this decrease was not statistically significant. Longitudinal analysis showed normalization of LDL levels post-recovery, while HDL levels remained persistently low. **Conclusion**: Despite observable alterations in lipid profiles, their prognostic significance in this cohort was limited. These findings highlight the need for further investigation into the role of lipid metabolism in the pathophysiology of COVID-19.

Keywords: COVID-19 severity; low-density lipoprotein; lipid metabolism; dyslipidemia; inflammation; C-reactive protein

1. Introduction

Since its emergence in late 2019, COVID-19 has posed unprecedented global health challenges, leading to millions of infections and fatalities worldwide. The disease, caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), manifests across a broad spectrum of clinical presentations, from asymptomatic or mild symptoms to severe pneumonia, acute respiratory distress syndrome (ARDS), multi-organ failure, and death. The severity of COVID-19 is influenced by various host factors, including age, comorbidities such as diabetes, cardiovascular diseases, obesity, and dysregulation of the immune response [1].

Emerging evidence suggests that lipid metabolism plays a pivotal role in the pathophysiology of viral infections, including SARS-CoV-2. Lipids are essential components for viral entry, replication, and modulation of the immune response. Notably, LDL-C and HDL-C have been implicated in immune regulation, inflammatory responses, and endothelial function. Several studies have documented significant alterations in lipid profiles during viral infections, including reductions in total cholesterol, LDL-C, and HDL-C levels [1-5].

These lipid disturbances have been associated with systemic inflammation, liver dysfunction, altered lipoprotein metabolism, and direct viral interactions with host lipid pathways [1-6].

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In the context of COVID-19, the inflammatory cytokine storm—characterized by excessive release of interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and CRP may contribute to dyslipidemia. Studies have reported that LDL-C levels tend to decline during severe COVID-19, potentially due to increased lipid catabolism, hepatic dysfunction, or oxidative stress. Similarly, reductions in HDL-C have been linked to impaired anti-inflammatory and antioxidant defenses, which may exacerbate endothelial dysfunction and increase the risk of thrombosis [6]. Elevated triglyceride levels have also been observed in critically ill patients, possibly reflecting metabolic stress and altered lipid transport [7,8].

Despite these observations, the precise role of lipid alterations in COVID-19 severity and prognosis remains unclear. Some researchers propose that low LDL-C levels could serve as markers of disease severity, while others argue that lipid abnormalities may merely reflect the systemic inflammatory burden [9-13]. Given the potential implications for risk stratification and therapeutic interventions, further research is warranted to elucidate the relationship between lipid metabolism and COVID-19 outcomes.

In this study, we investigate the association between LDL-C levels and COVID-19 severity in a cohort of hospitalized patients in Northern Greece. By analyzing lipid profiles, antilipidemic treatments, and clinical outcomes, we aim to contribute to the understanding of lipid metabolism's role in COVID-19 and its potential as a prognostic biomarker or therapeutic target.

2. Materials and Methods

A retrospective study was conducted at a hospital in Northern Greece over a two-month period, including 208 COVID-19 patients. The study protocol was approved by the Scientific Board of AHEPA University General Hospital (Number 36002/29th/04.12.2024). Data were collected on demographics, lipid profiles (including total cholesterol, LDL, HDL, and triglycerides), and antilipidemic treatment, while excluding patients under 16 years old or those with in-hospital-acquired COVID-19 (Table 1). Statistical analyses were performed using SPSS v.19, with correlations evaluated using ANOVA, Mann-Whitney U tests, and Pearson's correlation coefficients, and significance was set at $p < 0.05$.

3. Results

The mean LDL level among the study cohort was 84.12 mg/dL, with no significant differences observed between survivors and non-survivor. Additionally, HDL, total cholesterol, and triglycerides were examined but showed no statistically significant correlation with clinical outcomes. Approximately 48.1% of the discharged patients had been undergoing antilipidemic treatment prior to hospitalization (Table 1, Figure 1). However, this treatment did not significantly impact their LDL levels or survival rates.

Detailed subgroup analyses revealed several key findings:

1. ICU Admission: Patients requiring intensive care unit (ICU) admission (n = 62) had slightly lower mean LDL levels (82.14 mg/dL) compared to non-ICU patients (85.03 mg/dL). Despite this trend, the difference was not statistically significant ($p = 0.08$) (Table 2, Figure 2).
2. Obesity and Triglycerides: Obese patients (BMI ≥ 30 kg/m²) displayed significantly elevated triglyceride levels, with a mean of 142.78 mg/dL, compared to 121.32 mg/dL in non-obese patients ($p = 0.03$) (Figure 3).
3. CRP Levels and HDL: Among patients with higher baseline C-reactive protein (CRP) levels (≥ 10 mg/L), HDL levels were notably lower (mean 35.22 mg/dL) compared to those with lower CRP levels (<10 mg/L, mean HDL = 39.31 mg/dL; $p = 0.04$) (Figure 4, Figure 5).
4. LDL Trends in Non-Survivors: LDL levels in non-survivors declined from 86.41 mg/dL at admission to 76.25 mg/dL by day 7, suggesting a possible association with disease progression, although this change was not statistically significant ($p = 0.12$) (Figure 6, Figure 7).

Longitudinal Follow-Up:

- Among discharged patients, LDL levels tended to normalize within four weeks post-recovery, showing a mean increase of 5.3 mg/dL compared to admission levels ($p < 0.05$).

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- In contrast, HDL levels remained persistently low (mean 36.12 mg/dL) even at follow-up, indicating prolonged alterations in lipid metabolism post-infection (Table 3).

Visualization and Statistical Trends:

- Box plots demonstrated significant variability in triglyceride levels, particularly among ICU patients, highlighting a correlation with systemic inflammation and obesity.
- Bar charts emphasized the marginally improved outcomes in patients with prior antilipidemic therapy, although the differences were not statistically significant ($p = 0.07$) (Figure 8).

These findings underscore the complex interplay between lipid metabolism and COVID-19 outcomes, warranting further exploration into lipid profiles as potential prognostic markers or therapeutic targets.

4. Discussion

Our findings align with prior studies indicating lipid disturbances in COVID-19 patients. Reduced LDL and HDL levels likely reflect systemic inflammatory responses rather than direct viral effects. While alterations in lipid profiles are notable, their lack of prognostic significance in this cohort underscores the multifactorial nature of COVID-19 outcomes.

Comparative Analysis with Literature

Several studies have explored the relationship between lipid profiles and COVID-19 severity, supporting and extending our findings. For instance, Tanaka et al. observed reduced HDL-C and LDL-C levels among ICU patients, correlating with disease severity and inflammation markers such as CRP [14].

Similarly, Feingold and Li reported that low HDL and LDL levels at hospital admission were associated with increased mortality risk [15,16]. These studies highlight the dual role of lipid profiles as both markers and mediators of the inflammatory response.

Studies by Aung and Liang independently highlighted genetic predispositions linking elevated LDL levels to increased susceptibility to COVID-19 [17,18]. The Apo COVID study also demonstrated a dynamic interplay between lipid profiles and inflammation in critically ill patients, suggesting that lipid dysregulation may exacerbate the pathophysiological effects of SARS-CoV-2 [14].

These studies highlight the significance of monitoring lipid profiles in COVID-19 patients for both prognostic assessment and therapeutic decision-making.

Inflammation and Lipid Dynamics

COVID-19-induced cytokine storms significantly disrupt lipid metabolism. SARS-CoV-2 infection triggers systemic metabolic changes, including dyslipidemia and liver dysfunction. Studies have observed that LDL-C and HDL-C levels inversely correlate with CRP levels, with reductions reflecting inflammatory severity [19]. This inverse relationship underscores the role of inflammation in modulating lipid dynamics. Furthermore, alterations in triglycerides (TG) and lipoprotein particle distribution provide additional layers of complexity to the lipid-inflammatory nexus.

Several studies have indicated lipid metabolism alterations during COVID-19 infection, specifically a decrease in high-density lipoprotein (HDL) and low-density lipoprotein (LDL) concentrations and an increase in triglyceride (TG) levels during the infection [20-23].

However, a decline in triglycerides can also be observed in critical cases. A direct correlation can be observed between a decrease in serum cholesterol, HDL-C, LDL-C and TGs, and the severity of the disease; these laboratory findings can serve as potential markers for patient outcomes. The transmission of coronavirus increases proportionally with rising levels of cholesterol in the cell membrane. This is due to the fact that cholesterol increases the number of viral entry spots and the concentration of angiotensin-converting enzyme 2 (ACE2) receptor, crucial for viral penetration. Studies have found that lower HDL-C levels correspond with a higher susceptibility to SARS-CoV-2 infection and infections in general, while higher HDL-C levels were related to a lower risk of developing them [6,24].

The hyper-inflammatory state mediated by the cytokine storm disturbs several fundamental lipid biosynthesis pathways. Virus replication is a process that drastically changes the host cell's lipid metabolism program and overuses cell lipid resources. Lower HDL-C and ApoA1 levels are

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associated with higher severity and mortality rates and with higher levels of inflammatory markers [7,25].

A study by Caterino et al. conducted a targeted lipidomic analysis in conjunction with measurements of proinflammatory cytokines and alarmins in serum samples from COVID-19 patients with varying disease severity [26].

The severity of COVID-19 is linked to an imbalanced immune response. The dysregulated metabolism of small molecules and bioactive lipids has also been associated with disease severity.

According to Zhang Z et al., LC-MS technology was employed to examine lipidomic and inflammatory profiles in plasma samples from COVID-19 patients with different severity levels, incorporating data from over 30 immune markers to enhance understanding of disease biochemistry and identify potential targets for intervention [27].

These findings suggest that monitoring lipid profiles in COVID-19 patients could provide valuable insights into disease progression and potential therapeutic targets.

Clinical Implications

While our study did not identify LDL as a prognostic marker, the broader literature underscores the potential of lipid profiles, particularly HDL-C, as indicators of disease severity. The therapeutic benefits of lipid-lowering agents, such as statins, are also under investigation for their anti-inflammatory properties. Statins, by modulating endothelial function and reducing cytokine release, may offer adjunctive benefits in mitigating severe COVID-19 outcomes. Zhang et al. emphasized the TG/HDL ratio's utility in risk stratification, further supporting the clinical relevance of lipid parameters [27].

Integrated Perspectives

The interplay between lipid metabolism, comorbidities, and COVID-19 outcomes warrants a comprehensive evaluation. Recent studies have highlighted the significant role of lipid metabolism in COVID-19 severity and prognosis. Evidence suggests that dyslipidemia, obesity, and other cardiometabolic conditions are closely linked to infection susceptibility and worse clinical outcomes.

Scalsky et al. analyzed data from the UK Biobank and found that elevated LDL-C and triglycerides increased COVID-19 infection risk, while higher HDL-C levels had a protective effect. Their findings emphasize the importance of baseline cardiometabolic health in determining COVID-19 outcomes [28].

Ochoa-Ramírez et al. investigated hospitalized COVID-19 patients and reported that low LDL-C and HDL-C levels were associated with higher mortality, prolonged ICU stays, and severe disease progression. Some critically ill patients exhibited hypertriglyceridemia, suggesting a complex interplay between lipid metabolism and inflammation [29].

Hua et al. provided a comprehensive review of the metabolic disruptions caused by SARS-CoV-2, discussing how inflammation, oxidative stress, and lipid metabolism dysregulation contribute to disease severity. They also highlighted the potential of lipid-lowering therapies (e.g., statins, metformin, and anti-inflammatory agents) in improving patient outcomes [30].

Together, these studies reinforce the critical role of lipid metabolism in COVID-19 pathophysiology and support the need for further research into metabolic interventions to improve patient outcomes. These findings highlight the importance of integrating personalized interventions to address metabolic derangements in COVID-19 patients. Addressing lipid dysregulation through targeted therapies and lifestyle modifications may hold promise in reducing morbidity and mortality associated with COVID-19.

Future Directions

Further research should focus on elucidating the mechanisms driving lipid perturbations in COVID-19 and their interactions with other metabolic pathways. Longitudinal studies examining the impact of lipid-lowering therapies on inflammation and clinical outcomes are particularly warranted. Additionally, the integration of lipidomics with other 'omics' technologies may uncover novel biomarkers and therapeutic targets in the context of COVID-19.

5. Conclusions

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This is the first and only study to date examining the relationship between lipid profiles and COVID-19 outcomes specifically in Northern Greece. Our findings provide a unique perspective on the regional epidemiology of lipid metabolism in SARS-CoV-2 infections.

This study contributes to the growing body of evidence on lipid metabolism's role in COVID-19. Although LDL levels did not predict clinical outcomes, understanding these dynamics may inform future therapeutic strategies. Further research is needed to elucidate lipid metabolism's potential as a therapeutic target or biomarker in COVID-19 and other viral infections.

Supplementary Materials: Table 1. Demographic and clinical data of the study population. Table 2. Lipid profile and statistical analysis. Table 3. Statistical summary of P-values and significance across analyses. Figure 1. Proportion of patients on antilipidemic treatment. Figure 2. LDL levels in ICU vs. non-ICU patients: a comparative bar chart. Figure 3. Comparison of triglyceride levels in obese and non-obese patients. Figure 4. Comparison of HDL levels in high and low CRP groups. Figure 5. Relationship between LDL and CRP levels, with each point representing an individual patient. Figure 6. Declining LDL trends over time in non-survivors. Figure 7. Comparison of LDL levels in patients with high (≥10 mg/L) and low (<10 mg/L) CRP levels. Figure 8. Mean lipid profile values.

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Informed Consent Statement: Informed consent was obtained from all subjects involved in the study.

Conflicts of Interest: The authors declare no conflict of interest.

Abbreviations

The following abbreviations are used in this manuscript:

LDL	low-density lipoprotein cholesterol
HDL	high-density lipoprotein cholesterol
CRP	C-reactive protein
ARDS	acute respiratory distress syndrome

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