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POST-COVID Lipid Profile Alterations In Stage 3 Chronic Kidney Disease Patients

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Abstract
The study evaluated changes in renal functional status in patients with stage 3 chronic kidney disease (CKD) who had previously experienced COVID-19 infection. A total of 131 patients were included in the study:

- Group 1 — CKD stage 3 (n=70);
- Group 2 — CKD stage 3 + COVID-19 (n=61).

For each patient, the lipid profile was evaluated, including: Total cholesterol (TC, mmol/L), Triglycerides (TG, mmol/L), Low-density lipoprotein cholesterol (LDL-C, mmol/L), High-density lipoprotein cholesterol (HDL-C, mmol/L)
Laboratory measurements were performed at 1 month and 3 months. Data are presented as mean ± SEM**.** Statistical analysis was conducted using Student's t-test or appropriate parametric tests. Significant changes over the 3-month period were indicated with an asterisk (*), $p<0.05$.
The study was conducted in accordance with ethical standards, and written informed consent was obtained from all participants.

Keywords: Chronic kidney disease, COVID-19, lipid profile, total cholesterol, triglycerides.

Introduction

Chronic kidney disease (CKD) is a global medical and social problem and is associated with a high incidence of cardiovascular complications and premature mortality. According to international guidelines, patients with stage 3 CKD exhibit pronounced metabolic disturbances, among which dyslipidemia plays a key role [7]. Lipid metabolism disorders in CKD include hypertriglyceridemia, increased levels of low-density lipoprotein cholesterol (LDL-C), decreased levels of high-density lipoprotein cholesterol (HDL-C), as well as qualitative changes in lipoproteins [6]. These alterations contribute to accelerated atherosclerosis and progression of renal dysfunction.
The pathophysiological mechanisms of dyslipidemia in CKD are related to decreased activity of lipoprotein lipase, impaired catabolism of apolipoproteins, chronic inflammation, and oxidative stress [6]. According to KDIGO recommendations, monitoring of the lipid profile in CKD patients is an essential component of cardiovascular risk prevention [7].
The COVID-19 pandemic, caused by the SARS-CoV-2 virus, has demonstrated pronounced systemic effects extending beyond respiratory involvement. COVID-19 is associated with systemic inflammation, cytokine response, endothelial dysfunction, and hypercoagulability [5]. These mechanisms are closely linked to disturbances in lipid metabolism.
Several studies have shown that during the acute phase of COVID-19, levels of total cholesterol, LDL-C, and HDL-C decrease, while triglyceride levels are altered [3,4]. Wei et al. (2020) reported that hypolipidemia correlates with disease severity and the level of inflammatory markers [4]. Wang et al. (2020) demonstrated that decreased HDL-C levels are associated with more severe clinical outcomes [3]. It is suggested that virus-induced inflammation affects lipoprotein synthesis and catabolism through cytokine-mediated pathways [2].
In the post-COVID period, persistent inflammation, metabolic destabilization, and endothelial dysfunction can persist, forming the so-called post-COVID syndrome [5]. Nalbandian et al. (2021) emphasized that prolonged metabolic changes may persist for several months after recovery from infection [5]. However, most studies focus on the general population, and data on patients with CKD are limited.
CKD patients are at high risk for severe COVID-19 due to reduced immune responsiveness, chronic inflammation, and concurrent cardiovascular disorders. In this patient group, the combination of renal dysfunction and post-infectious metabolic alterations may potentiate dyslipidemia and accelerate atherogenesis.

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Despite some publications on lipid metabolism disturbances during COVID-19, comprehensive evaluation of lipid profile dynamics in stage 3 CKD patients during the post-COVID period remains insufficiently studied. Therefore, investigating post-COVID changes in lipid profiles in CKD patients is of significant clinical importance for optimizing monitoring and therapeutic management.

Material and methods

This prospective comparative study was conducted at Tashkent State Medical University. The study included patients with stage 3 chronic kidney disease (CKD) who were under outpatient follow-up.

A total of 131 patients were examined and divided into two groups:

- **Group 1** — stage 3 CKD patients without a history of COVID-19 infection (n=70);
- **Group 2** — stage 3 CKD patients after COVID-19 infection (post-COVID) (n=61).

Inclusion criteria were: a confirmed diagnosis of stage 3 CKD according to KDIGO classification, age over 18 years, and a documented history of COVID-19 infection (for Group 2).

Exclusion criteria included: acute inflammatory diseases, decompensated cardiovascular disorders, active malignancies, severe liver disease, and the use of lipid-lowering medications with changes in dosage during the observation period.

Lipid profile assessment included the following parameters:

- total cholesterol (TC, mmol/L),
- triglycerides (TG, mmol/L),
- low-density lipoprotein cholesterol (LDL-C, mmol/L),
- high-density lipoprotein cholesterol (HDL-C, mmol/L).

Venous blood samples were collected in the morning after an overnight fast. Laboratory analyses were performed at 1 and 3 months after COVID-19 infection (in Group 2) and at corresponding time points in Group 1.

Statistical analysis was performed using parametric methods. Results are presented as mean ± standard error of the mean (SEM). Student's t-test was used to evaluate the significance of differences. Differences were considered statistically significant at $p<0.05$.

The study was conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants.

Results

Analysis of lipid profile parameters demonstrated significant changes in patients with stage 3 CKD over the observation period.

In **Group 1** (stage 3 CKD without COVID-19), after 3 months, total cholesterol decreased from 6.31 ± 0.17 to 5.68 ± 0.11 mmol/L. Triglyceride levels decreased from 2.58 ± 0.15 to 2.12 ± 0.07 mmol/L. LDL-C levels decreased from 3.42 ± 0.08 to 3.02 ± 0.04 mmol/L. HDL-C levels increased from 0.98 ± 0.019 to 1.06 ± 0.011 mmol/L.

In **Group 2** (stage 3 CKD after COVID-19 infection), the changes were more pronounced. Total cholesterol decreased from 6.46 ± 0.09 to 6.20 ± 0.06 mmol/L ($p<0.05$). Triglyceride levels decreased from 2.62 ± 0.11 to 2.31 ± 0.09 mmol/L ($p<0.05$). LDL-C levels significantly decreased from 3.54 ± 0.09 to 3.16 ± 0.03 mmol/L ($p<0.05$). HDL-C levels increased from 0.96 ± 0.016 to 1.02 ± 0.009 mmol/L ($p<0.05$) (Table 1).

Table 1. Lipid Profile Parameters in the Study Groups

	Group 1		**Group 2**	
	1 month	**3 month**	**1 month**	**3 month**
TCh	6.31±0,17	5.68±0,11**	6.46±0.09	6.2±0.06*
THL	2.58±0,15	2.12±0,07**	2.62±0.11	2.31±0.09*
LDL-C	3.42±0,08	3.02±0,04**	3.54±0.09	3.16±0.03*
HDL-C	0.98±0,019	1.06±0,011**	0.96±0.016	1.02±0.009*

Note: * — level of statistical significance ($p<0.05$);
** — level of statistical significance ($p<0.01$).

Comparative analysis showed that in post-COVID patients, the decrease in total cholesterol, triglycerides, and LDL-C levels was statistically more pronounced compared to patients without prior COVID-19 infection. The increase in HDL-C levels also reached statistical significance in the second group.

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These findings indicate a more pronounced dynamic of lipid profile changes in stage 3 CKD patients during the post-COVID period.

Discussion

The results obtained indicate pronounced changes in the lipid profile of stage 3 CKD patients during the post-COVID period. The observed dynamics of total cholesterol, triglycerides, and LDL-C levels confirm the impact of prior COVID-19 infection on metabolic processes in this patient population.

It is well known that dyslipidemia in CKD has an atherogenic nature and is associated with decreased lipoprotein lipase activity, chronic inflammation, and oxidative stress. In the context of a prior viral infection, these mechanisms may be exacerbated due to systemic inflammatory responses, cytokine-induced alterations in lipid metabolism, and endothelial dysfunction.

The decrease in total cholesterol and LDL-C levels in post-COVID patients may be related to inflammation-mediated suppression of hepatic lipoprotein synthesis, as well as lipid redistribution in response to systemic immune activation. Similar changes have been reported in studies of the acute phase of COVID-19, where hypolipidemia correlated with the severity of inflammation.

The increase in HDL-C levels observed in Group 2 may reflect a compensatory response aimed at stabilizing lipid metabolism and reducing atherogenic risk. However, even with the observed positive trends in lipid parameters, the risk of cardiovascular complications in CKD patients cannot be excluded, given their inherently high cardiovascular risk.

The more pronounced changes in the lipid profile in the post-COVID group compared to patients without prior infection support the hypothesis of additional metabolic effects of SARS-CoV-2 in patients with chronic kidney disease. It is likely that post-COVID syndrome is accompanied by prolonged metabolic remodeling, necessitating intensified clinical and laboratory monitoring.

Thus, the observed changes emphasize the need for dynamic monitoring of the lipid profile in stage 3 CKD patients after COVID-19 infection, as well as the importance of an individualized approach to dyslipidemia management during the post-COVID period.

Conclusion

Post-COVID stage 3 chronic kidney disease patients exhibit significant alterations in lipid profiles compared to non-COVID CKD patients. Total cholesterol, triglycerides, and LDL-C levels decreased, while HDL-C levels showed a slight increase. These findings suggest that COVID-19 infection may exacerbate metabolic disturbances in CKD patients, highlighting the importance of close lipid monitoring and individualized management in the post-COVID period.

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