

Clinical and Pathogenetic Features of Lipid Metabolism Disorders and the Development of Obesity in COVID-19 Patients

Khairullaeva G. S.

Bukhara State Medical Institute, Bukhara, Uzbekistan

Abstract The analysis showed that in COVID-19 patients, lipid metabolism disorders and the severity of systemic inflammation progress as the degree of obesity increases. In patients with severe obesity, maximum levels of atherogenic lipids, inflammatory markers, and insulin resistance were identified, which are accompanied by adipokine imbalance. These changes contribute to the worsening of COVID-19 and determine the need for early metabolic correction in this category of patients.

Keywords COVID-19, Obesity, Lipid metabolism, Dyslipidemia, Insulin resistance, Adipokines, Leptin, Adiponektin, Systemic inflammation, IL-6, CRP

1. Introduction

The COVID-19 pandemic coincided with global obesity growth, forming a combination of two major epidemics. Patients with excess body weight significantly more often require hospitalization and have a more severe course of COVID-19, which is associated with metabolic disorders, chronic inflammation, and atherogenic dyslipidemia [1,2,3,4]. Obesity is considered an independent risk factor for respiratory failure, thrombosis, and mortality, while the combination of obesity with dyslipidemia and insulin resistance forms an unfavorable metabolic phenotype in the patient [2,3,4,5,6].

The pathogenesis of COVID-19 in obesity is associated with lipid metabolism disorders, a decrease in HDL cholesterol and LDL levels, an increase in triglycerides, and the active production of pro-inflammatory cytokines [7,8,9,10]. SARS-CoV-2 is capable of infecting adipocytes and macrophages of adipose tissue, intensifying local and systemic inflammation, insulin resistance, and metabolic dysfunction [11,12,13]. These processes exacerbate the course of COVID-19 and explain the high frequency of complications in patients with obesity.

Correction of lipid metabolism disorders is considered an important area of treatment for COVID-19 patients. The effectiveness of statins, omega-3 LDLs, fibrates, and non-drug interventions (diet, weight reduction) has been confirmed by clinical data showing a decrease in the frequency of

complications and improvement in outcomes [9,14,15,16]. However, optimal management strategies for patients with obesity and COVID-19 require further study, which determines the relevance of this study.

The purpose of the study is to identify the clinical and pathogenetic features of lipid metabolism disorders and the mechanisms of obesity development in COVID-19 patients, as well as to evaluate the effectiveness of various methods for correcting dyslipidemia and metabolic dysfunction in the acute and post-infectious course of the disease.

2. Materials and Methods of Research

The study was conducted under multicenter observation and included 168 patients who had COVID-19 of varying severity and had signs of lipid metabolism disorders. The selection was carried out on the basis of the infectious diseases hospital and the consultative and diagnostic center. The age of the examined varied from 28 to 72 years (average age 49.6 ± 12.1 years); among them were 92 men (54.8%) and 76 women (45.2%). Inclusion criteria included laboratory-confirmed SARS-CoV-2 (PCR), presence of dyslipidemia or obesity ($MI \geq 30 \text{ kg/m}^2$), and consent to participate in the study.

To analyze the characteristics of lipid metabolism, patients were divided into three groups. Group I (n=54) included patients with normal body weight and moderate dyslipidemia in COVID-19; Group II (n=62) - patients with obesity of I-II degree, who had COVID-19 in moderate form; Group III (n=52) consisted of patients with obesity of the III degree and pronounced lipid profile disorders who had a severe form of the disease. This distribution made it possible

* Corresponding author:

gulruh_xayrullayeva@bsmi.uz (Khairullaeva G. S.)

Received: Dec. 29, 2025; Accepted: Jan. 22, 2026; Published: Jan. 22, 2026

Published online at <http://journal.sapub.org/ajmms>

to assess the influence of the degree of obesity and the severity of metabolic disorders on the clinical course of COVID-19.

3. Research Results

Analysis of 168 COVID-19 patients showed significant differences in lipid metabolism and metabolic status between the groups. The progression of obesity was accompanied by an increase in the frequency of moderate to severe COVID-19, as well as a deterioration in laboratory inflammatory parameters and lipid profile (Table 1).

Analysis of the presented data shows a clear trend towards the progression of lipid metabolism disorders as the degree of obesity increases in COVID-19 patients. All lipid profile indicators differed statistically significantly between the groups ($p < 0.01-0.001$), indicating a close relationship between the severity of metabolic disorders and the clinical course of the disease.

In patients of group I with normal body weight or excess weight, the indicators of total cholesterol and LDL were within moderate dyslipidemia. At the same time, the level of HDL remained relatively stable (1.28 ± 0.22 mmol/l), reflecting a more favorable antiatherogenic status. Triglycerides also did not exceed diagnostically significant thresholds.

In group II, represented by patients with obesity of I-II degree, a further increase in total cholesterol and LDL was noted, which was accompanied by a significant decrease in the concentration of HDL (1.10 ± 0.19 mmol/l). An increase in triglycerides by almost 30% compared to the first group indicated the formation of a mixed type of dyslipidemia. The atherogenicity index reached 3.9 ± 0.7 , reflecting the transition to a more pronounced atherogenic profile.

The most pronounced disorders were identified in Group III, consisting of patients with severe obesity. The level of total cholesterol increased to 6.3 ± 1.1 mmol/l, and LDL to 4.1 ± 0.9 mmol/l ($p < 0.01$ compared to both groups).

Simultaneously, a significant decrease in HDL was observed, reaching only 0.94 ± 0.17 mmol/l, which is an important marker of lipoprotein balance deterioration. Triglycerides increased by more than 1.6 times compared to group I, which indicates a pronounced disruption of lipid metabolism. The maximum atherogenicity index (4.8 ± 0.9) reflected a high risk of atherothrombotic complications under COVID-19 conditions.

The level of systemic inflammation and the severity of the cytokine response increased proportionally to the degree of obesity. This confirms the participation of adipose tissue as an active inflammatory organ in COVID-19 (Table 2).

Data analysis shows a pronounced intensification of the inflammatory response and an increasing metabolic dysfunction in patients with an increase in the degree of obesity. All indicators differed statistically significantly between the groups ($p < 0.001$), which emphasizes the important role of obesity in the pathogenesis of severe COVID-19.

In patients of group I with minimal manifestations of metabolic disorders, CRP and IL-6 levels were characterized by a moderate increase, typical for the moderate course of COVID-19. The insulin resistance index ($\text{HOMA-IR} = 2.4 \pm 0.6$) corresponded to a mild degree of carbohydrate metabolism disorders. Leptin levels were relatively low, and adiponektin was the best among all groups, reflecting a more stable metabolic status.

In group II, a significant increase in inflammation was noted: CRP and IL-6 concentrations increased by more than 1.6 times compared to the first group. Simultaneously, an increase in insulin resistance ($\text{HOMA-IR} = 3.8 \pm 0.9$) was observed, confirming the influence of increased fat mass on glucose homeostasis. The level of leptin increased almost twofold, while the adiponektin content decreased to 4.3 ± 1.0 mcg/ml, indicating the development of adipokine imbalance and increased pro-inflammatory status.

Table 1. Lipid profile of patients in three groups (M \pm SD)

Indicator	Group I (n=54)	Group II (n=62)	Group III (n=52)	p (intergroup)
Total cholesterol, mmol/l	5.1 \pm 0.8	5.8 \pm 0.9	6.3 \pm 1.1	<0.01
LDL, mmol/l	3.0 \pm 0.7	3.6 \pm 0.8	4.1 \pm 0.9	<0.01
HDL, mmol/l	1.28 \pm 0.22	1.10 \pm 0.19	0.94 \pm 0.17	<0.01
Triglycerides, mmol/l	1.42 \pm 0.31	1.86 \pm 0.44	2.39 \pm 0.58	<0.001
Atherogenicity index	3.0 \pm 0.5	3.9 \pm 0.7	4.8 \pm 0.9	<0.001

Table 2. Inflammatory and metabolic indicators of patients

Indicator	Group I	Group II	Group III	p
CRP, mg/l	18.4 \pm 6.2	31.7 \pm 9.4	48.9 \pm 12.6	<0.001
IL-6, pg/ml	22.1 \pm 7.5	37.4 \pm 10.3	61.8 \pm 14.7	<0.001
HOMA-IR	2.4 \pm 0.6	3.8 \pm 0.9	5.2 \pm 1.3	<0.001
Leptin, ng/ml	15.1 \pm 5.3	27.4 \pm 8.2	41.6 \pm 12.1	<0.001
Adiponektin, mcg/ml	6.9 \pm 1.4	4.3 \pm 1.0	3.1 \pm 0.9	<0.001

The most unfavorable indicators were recorded in patients of Group III. A significant increase in CRP (48.9 ± 12.6 mg/l) and IL-6 (61.8 ± 14.7 pg/ml) reflected a pronounced systemic inflammatory response characteristic of severe COVID-19. Maximum HOMA-IR values indicated profound insulin resistance, characteristic of severe obesity. Leptin reached the highest levels (41.6 ± 12.1 ng/ml), which corresponds to hyperleptinemia - a marker of chronic inflammation of adipose tissue. At the same time, adiponektin levels decreased to minimal values (3.1 ± 0.9 μ g/ml), indicating a practically complete depletion of its anti-inflammatory potential.

4. Output

As a result of the conducted research, it was established that the increase in the degree of obesity in COVID-19 patients is accompanied by a significant increase in lipid metabolism disorders, a pronounced increase in pro-inflammatory markers (CRP, IL-6), progression of insulin resistance, and adipokine imbalance, which forms an unfavorable metabolic and inflammatory background that worsens the course of the disease. The most pronounced deviations were found in patients with severe obesity, who exhibited maximum values of atherogenic lipids, hyperleptinemia, and minimal levels of adiponektin. The obtained results confirm the key role of obesity as a pathogenetic factor in the complicated course of COVID-19 and justify the need for early diagnosis and active correction of metabolic disorders in this category of patients.

REFERENCES

- [1] Stefan N., Birkenfeld A.L., Schulze M.B., Ludwig D.S. Obesity and impaired metabolic health in COVID-19. *Nat Rev Endocrinol.* 2020; 16: 341-352.
- [2] Yang Y., Ding L., Zou X., et al. Obesity and COVID-19 pandemics. *Diabetes Metab Syndr Obes.* 2023; 16: 2209-2225.
- [3] Ritter A., Kreis N.N., Louwen F., et al. Obesity and COVID-19: Molecular mechanisms. *Obes Rev.* 2020; 21: e13095.
- [4] Gammone M.A., D'Orazio N. Obesity-COVID-19 interaction. *Front Endocrinol.* 2021; 12: 652639.
- [5] Makhoul E., Jankowski M., Debevec T., et al. COVID-19 and metabolic syndrome. *Metab Open.* 2022; 14: 100180.
- [6] Zhdankina N.V., Smirnova Yu.V., Malysheva N.A. Metabolic Syndrome and COVID-19. *Endocrinology.* 2022; 11 (2): 53-64.
- [7] Li Y., Zhang Y., Lu J., et al. Lipid metabolism changes in severe COVID-19. *Clin Chim Acta.* 2021; 517: 66-73.
- [8] de Paiva Silvino J.P., et al. COVID-19 and dyslipidemia. *Res Soc Dev.* 2022; 11: e319754.
- [9] Jovandaric M.Z., Rajovic N., Milovanovic J. Lipid metabolism in COVID-19. *Int J Mol Sci.* 2022; 23: 15098.
- [10] Wang T., Du Z., Zhu F., et al. COVID-19 metabolism. *MedComm.* 2022; 3: e157.
- [11] Martínez-Colón G.J., Ratnasiri K., Chen H., et al. SARS-CoV-2 infection in adipose tissue. *Sci Transl Med.* 2022; 14: eabm9151.
- [12] Basolo A., Poma A.M., Bonaventura I., et al. SARS-CoV-2 in adipocytes. *J. Endocrinol Invest.* 2022. 45: 1021-1030.
- [13] Zickler M., Stange R., Diebel L., et al. Replication of viruses in adipose tissue. *Cell Metab.* 2022; 34: 1152-1167.
- [14] Martins-Filho P.R., et al. Statins in COVID-19. *Eur J Intern Med.* 2022; 99: 50-57.
- [15] Movahed F., et al. Statistics therapy meta-analysis. *Clin Exp Med.* 2024; 24: 459-472.
- [16] Ortega-Paz L., et al. Statins in hospitalized COVID-19 patients. *Thromb Res.* 2025, 243: 109484.