

A longitudinal case study: Persistent dyslipidemia in post-COVID-19 syndrome

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ABSTRACT

Post-acute sequelae of severe acute respiratory syndrome coronavirus 2 encompass multisystem disorders, with emerging evidence indicating metabolic disturbances such as dyslipidemia in recovered patients. This case study presents a 48-year-old male with no prior dyslipidemia or cardiovascular disease, hospitalized for moderate-to-severe COVID-19 pneumonia. Five months post-discharge, he reported fatigue and cognitive symptoms and was found to have a profoundly atherogenic lipid profile. Despite 3 months of structured lifestyle intervention (Mediterranean diet and graded exercise), his dyslipidemia persisted for over 6 months, characterized by elevated total cholesterol (242 mg/dL), triglycerides (285 mg/dL), low-density lipoprotein cholesterol (165 mg/dL), and reduced high-density lipoprotein cholesterol (32 mg/dL). Elevated high-sensitivity C-reactive protein (8.2 mg/L) indicated ongoing inflammation. Due to persistent abnormalities, atorvastatin 20 mg daily was initiated for primary prevention. This longitudinal case highlights that COVID-19 can induce significant and persistent atherogenic dyslipidemia even in individuals without traditional cardiovascular risk factors. The findings underscore the need for proactive metabolic screening, including a fasting lipid panel, as a standard component of post-COVID assessment, particularly after moderate-to-severe illness, to mitigate long-term cardiovascular risk.

Key words: Cardiovascular risk, Case report, Dyslipidemia, Lipid profile, Long COVID, Metabolic syndrome, Post-COVID syndrome, Severe acute respiratory syndrome coronavirus 2

The long-term sequelae of COVID-19, termed post-acute sequelae of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection, include a spectrum of multisystem disorders extending beyond the initial respiratory illness [1]. While fatigue and cognitive dysfunction are commonly reported, growing evidence reveals significant metabolic disturbances among recovered patients, including new-onset diabetes and dyslipidemia [2]. Recent cohort studies indicate that approximately 30–40% of hospitalized COVID-19 survivors exhibit abnormal lipid profiles during convalescence [3,4]. These profiles often feature hypertriglyceridemia, elevated low-density lipoprotein cholesterol (LDL-C), and decreased high-density lipoprotein cholesterol (HDL-C). The underlying pathophysiology is thought to involve persistent systemic inflammation, endothelial damage, and potential direct viral effects on lipid regulatory pathways [5].

This case report is presented to contribute a detailed longitudinal analysis of persistent dyslipidemia in a

patient without pre-existing metabolic disease, thereby highlighting an underrecognized metabolic complication of COVID-19 and reinforcing the rationale for systematic post-recovery metabolic screening.

CASE PRESENTATION

A 48-year-old male, a non-smoker with a sedentary office job, presented for post-COVID-19 evaluation. His past medical history was unremarkable for dyslipidemia, hypertension, diabetes mellitus, or cardiovascular disease. A routine annual health check in 2019 documented a normal lipid profile. The patient contracted PCR-confirmed SARS-CoV-2 infection, experiencing moderate illness with hypoxemia (SpO₂ 88% on room air), requiring hospitalization for 7 days. Management included supplemental oxygen, dexamethasone, and prophylactic anticoagulation. He was discharged after 10 days in stable condition.

At a 5-month post-discharge follow-up, the patient reported persistent fatigue, exertional dyspnea (mMRC grade 2), and subjective cognitive impairment

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(“brain fog”). Vital signs were stable: Blood pressure 128/82 mmHg, heart rate 76 bpm, respiratory rate 16/min, and temperature 36.8°C.

Despite a normal chest X-ray and pulmonary function tests, he was enrolled in a post-COVID rehabilitation clinic. Physical examination revealed no xanthomas, xanthelasmas, or other stigmata of dyslipidemia.

Diagnostic workup revealed a profoundly atherogenic lipid profile (Table 1) with elevated total cholesterol, triglycerides, and LDL-C, and reduced HDL-C compared to pre-pandemic baseline. High-sensitivity C-reactive protein (hs-CRP) was elevated at 8.2 mg/L. Fasting blood glucose and thyroid function tests were normal. Liver function tests showed a mild transient alanine aminotransferase elevation at month 5, normalizing by month 6. Cardiac echocardiogram at month 4 was normal.

After 3 months of structured lifestyle intervention (Mediterranean diet and graded exercise) without lipid improvement, pharmacological therapy was initiated. At 6 months post-COVID, after a detailed cardiovascular risk discussion, the patient was started on atorvastatin 20 mg daily for primary prevention. The treatment plan included a repeat lipid profile in 8 weeks to assess response, continued lifestyle counseling, and multidisciplinary monitoring for other Long COVID symptoms. The outcome at 6-month follow-up confirmed persistent dyslipidemia requiring statin therapy.

DISCUSSION

This case illustrates a significant metabolic complication of COVID-19: The emergence of persistent, atherogenic dyslipidemia in an individual with no prior history of lipid disorders. The observed pattern – marked by high triglycerides, elevated LDL-C, and low HDL-C — aligns with findings from larger cohort studies of post-COVID patients [4-6]. The persistence of this dyslipidemia for over 6 months, despite lifestyle modification, suggests the disturbance extends beyond a simple acute-phase response or deconditioning after severe illness.

The pathophysiological links between SARS-CoV-2 infection and dyslipidemia are multifaceted and appear to be driven by sustained inflammation and immune dysregulation. The elevated hs-CRP in our patient

indicates persistent low-grade inflammation, a known catalyst for metabolic dysfunction. Chronic inflammation can stimulate hepatic overproduction of very-low-density lipoprotein, leading to hypertriglyceridemia, while simultaneously impairing the synthesis and function of cardioprotective HDL particles [7,8]. Furthermore, SARS-CoV-2 is known to cause endotheliitis, which may disrupt the activity of endothelial-bound lipoprotein lipase, a key enzyme responsible for clearing triglycerides from the bloodstream [9].

Emerging research also points to more direct viral mechanisms. Some studies suggest that the SARS-CoV-2 spike protein may interact directly with HDL particles, potentially altering their anti-inflammatory and cholesterol efflux capabilities [10]. The inflammatory milieu post-COVID can also induce or exacerbate insulin resistance, a central driver of the characteristic “high triglyceride, low HDL” dyslipidemic pattern [11]. The lack of metabolic normalization in our patient for over half a year hints at a potential sustained reprogramming of metabolic homeostasis, possibly mediated through epigenetic modifications or chronic immune activation triggered by the initial viral infection [12]. This persistence is clinically significant, as it aligns with epidemiological data showing an increased incidence of diabetes and major adverse cardiovascular events among COVID-19 survivors within the first year after infection [2,13].

Our findings advocate for a paradigm shift in post-COVID care, from a solely symptom-focused approach to one that proactively screens for and manages metabolic complications. This case supports the integration of routine fasting lipid profile assessment at 3–6 months post-infection, particularly for patients who experienced moderate-to-severe acute illness. Early identification and intervention for dyslipidemia should be integral components of holistic post-COVID rehabilitation programs. Such measures are crucial not only for managing Long COVID but also for mitigating the potential long-term burden of cardiovascular disease in this rapidly expanding patient population [14,15]. Future research should focus on elucidating the precise molecular mechanisms driving these changes and defining optimal long-term management strategies for post-COVID metabolic syndrome.

Table 1: Longitudinal lipid profile and inflammatory marker

Parameter	Baseline (2019)	Month 3 Post-COVID	Month 6 Post-COVID	Reference range
Total Cholesterol (mg/dL)	178	242	235	<200 Desirable
Triglycerides (mg/dL)	120	285	270	<150 Normal
LDL-C (mg/dL)	110	165	160	<100 Optimal
HDL-C (mg/dL)	48	32	35	>40
Non-HDL Cholesterol (mg/dL)	130	210	200	<130
TC/HDL Ratio	3.7	7.6	6.7	<5.0
hs-CRP (mg/L)	1.8	8.2	Not repeated	<3.0

Serial assessment of lipid parameters and inflammatory marker (hs-CRP) in a post-COVID patient demonstrating persistent atherogenic dyslipidemia. LDL-C: Low-density lipoprotein cholesterol, HDL-C: High-density lipoprotein cholesterol, TC: Total cholesterol, hs-CRP: High-sensitivity C-reactive protein

CONCLUSION

This longitudinal case study reinforces that COVID-19 can act as a metabolic disruptor, inducing significant and persistent atherogenic dyslipidemia even in individuals without traditional cardiovascular risk factors. The case underscores the necessity for clinicians to adopt a proactive, surveillance-based approach in the follow-up of COVID-19 survivors. We recommend that metabolic screening, including a fasting lipid panel, become a standard component of post-COVID assessment protocols. Early detection and management are essential for comprehensive patient care and may play a critical role in reducing long-term cardiovascular morbidity associated with SARS-CoV-2 infection.

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