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Immunopathology and therapeutic strategies for long COVID: mechanisms, manifestations, and clinical implications

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Abstract

Long coronavirus disease-19 (COVID-19) is a complex, multifactorial condition characterized by persistent symptoms lasting more than 12 weeks following acute severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection. The underlying mechanisms remain incompletely understood, but chronic inflammation, immune dysregulation, autoimmunity, and viral persistence are increasingly being implicated. This study investigated the immunopathological drivers of long COVID-19 and their associations with clinical manifestations and organ damage. A prospective, longitudinal cohort study was conducted on 200 COVID-19 survivors aged 18-65 years, in which immune markers, autoantibody profiles, lymphocyte dysfunction, and imaging findings were assessed over a 12-month period. Persistent inflammation was observed, with elevated interleukin-6 and tumor necrosis factor α ± levels correlated with lung fibrosis and cognitive impairment. Autoantibodies were detected in 40% of the participants, particularly those with cardiovascular and neurological symptoms. A significant reduction in CD8+ T-cell counts was associated with severe fatigue and cognitive dysfunction, whereas persistent SARS-CoV-2 RNA was identified in 10% of cases, primarily in individuals with gastrointestinal symptoms. Imaging studies revealed multiorgan involvement, with structural abnormalities in the lungs, heart, and brain. These findings highlight the interplay of immune dysfunction, chronic inflammation, and autoimmunity in long-term COVID-19, underscoring the need for targeted therapeutic strategies to address its long-term health impacts.

Keywords

Long coronavirus disease-19. Chronic inflammation. Autoimmunity. Cytokines interleukin-6. Tumor necrosis factor-α. CD8+ T-cell dysfunction.

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Introduction

The coronavirus disease-19 (COVID-19) pandemic has profoundly impacted global health, with over 800 million confirmed cases as of 2024. While most individuals recover from acute severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection, a substantial proportion experience lingering symptoms, a condition now recognized as long COVID-19 or post-acute sequelae of SARS-CoV-2 infection PASC World Health Organization (WHO), 2021. According to the WHO, long COVID-19 is defined as the persistence of symptoms for at least 12 weeks after the acute phase, often including fatigue, cognitive dysfunction, respiratory issues, and cardiovascular complications.

The pathophysiological mechanisms of long COVID-19 remain poorly characterized, complicating efforts to develop effective management strategies. Emerging evidence suggests that chronic immune dysregulation plays a pivotal role. Persistent inflammation, autoantibody production, lymphocyte abnormalities, and the potential for viral persistence have been implicated in ongoing symptomatology and organ dysfunction[1]. Elevated levels of proinflammatory cytokines, such as interleukin-6 (IL-6) and tumor necrosis factor-α (TNF-α), are linked to chronic inflammation and fibrotic changes in multiple organs[2]. In addition, autoantibodies against phospholipids, interferons, and nuclear antigens may contribute to cardiovascular and neurological sequelae through autoimmune mechanisms[3].

This study aimed to investigate the immunopathological mechanisms underlying long COVID-19 in a longitudinal cohort of survivors. By examining the relationships among immunological markers, clinical symptoms, and imaging findings, this research aims to provide a comprehensive understanding of the disease process. The insights gained may inform the development of targeted therapeutic strategies to mitigate the long-term health impact of long COVID.

Methods

Study design and population

A prospective longitudinal study involving 200 COVID-19 survivors aged 18-65 years was conducted between January 2023 and January 2024. Patients were recruited from hospitals and outpatient clinics in (Specify Region). The participants provided informed consent. Ethical approval was obtained from the Scientific Research Ethics Committee at Taif University HAO-02-T-105.

Inclusion and exclusion criteria

Inclusion criteria

- Adults aged 18-65 years with a confirmed diagnosis of COVID-19 by positive reverse transcription polymerase chain reaction or an antigen test.
- Presence of persistent symptoms lasting ≥ 12 weeks after initial COVID-19 diagnosis as per the WHO definition of long COVID-19.
- Willingness to participate for a 12-month follow-up period, with regular clinical assessments and laboratory tests.

Exclusion criteria

- The background of existing autoimmune disorders, such as lupus and rheumatoid arthritis, is significant. In addition, the administration of long-term immunosuppressive treatments, including corticosteroids and biologics, within the 6 months leading up to enrollment is pertinent.
- The presence of serious chronic illnesses that are not associated with COVID-19, such as end-stage renal disease or advanced cancer, is also relevant.
- Furthermore, any inability to provide informed consent or adhere to follow-up protocols must be considered.

Data collection

Demographic and clinical data

Baseline demographic characteristics included age, sex, existing health conditions such as hypertension, diabetes, and obesity, and the severity of acute COVID-19 classified as mild, moderate, or severe.

Clinical symptoms

Assessment of fatigue, cognitive impairment, dyspnea, thoracic discomfort, and myalgia was conducted at 3, 6, and 12 months following the infection.

Immunological markers

The levels of IL-6, TNF-α, and C-reactive protein (CRP) were quantified through enzyme-linked immuno-

sorbent assay. In addition, the enumeration of CD4+ and CD8+ T cells was performed through flow cytometry.

Autoantibody detection

The assessment of ANA, antiphospholipid, and antineuronal antibodies was conducted through conventional immunoassay techniques.

Imaging

Imaging studies of the respiratory, cardiovascular, and neurological systems were performed as clinically warranted. The interpretations of the computed tomography (CT) and magnetic resonance (MR) images were carried out by radiologists certified by the board.

Statistical analysis

All statistical analyses were conducted using Statistical Package for the Social Sciences version 27.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were used to summarize demographic and clinical characteristics. Continuous variables were presented as means ± standard deviations or medians with interquartile ranges depending on data normality, assessed using the Kolmogorov-Smirnov test. Categorical variables were expressed as frequencies and percentages.

Comparative analysis

For comparisons between long COVID patients and healthy controls:

- Independent t-tests were used for normally distributed continuous variables (e.g., IL-6 and TNF-α levels).
- Mann-Whitney U tests were employed for non-normally distributed variables (e.g., CD8+ T-cell counts).
- χ^2 tests or Fisher's exact tests were applied to compare categorical variables (e.g., prevalence of autoantibodies, organ-specific damage).

Longitudinal analysis

Changes in immunological markers and symptom severity over time (3, 6, and 12 months post-infection) were analyzed using:

- Repeated measures analysis of variance for normally distributed variables with post-hoc Bonferroni correction for pairwise comparisons
- Friedman tests followed by Wilcoxon signed-rank tests for non-parametric longitudinal data.

Correlation and regression analysis

- Pearson's correlation coefficient (r) was used to assess the relationships between inflammatory markers (IL-6, TNF-α) and symptom severity scores (respiratory and neurological symptoms).
- Spearman's rank correlation was applied for non-normally distributed variables.
- Multivariable logistic regression models were constructed to evaluate associations between immune dysregulation (cytokine levels, autoantibodies, CD8+ T-cell counts) and clinical outcomes (lung fibrosis, myocarditis, cognitive impairment). Adjusted odds ratios with 95% confidence intervals were reported.

Survival analysis

Kaplan-Meier survival curves were plotted to evaluate the persistence of long COVID symptoms over time, with log-rank tests comparing symptom resolution between subgroups based on immune markers.

A $p < 0.05$ was considered statistically significant. All statistical tests were two-tailed. Missing data were handled using multiple imputation techniques, assuming data were missing at random. This robust statistical approach ensures reliable interpretation of immune dysregulation patterns in long COVID and their associations with clinical outcomes.

Results

This section presents the findings from the cohort study, highlighting associations between immune dysregulation and clinical manifestations of long COVID-19. The results are organized to address chronic inflammation, autoimmunity, and lymphocyte dysfunction and are correlated with organ damage.

Chronic inflammation and correlation with symptoms

Longitudinal immunological assessments revealed that chronic inflammation persisted in a large proportion of long COVID-19 patients. Elevated levels of pro-inflammatory cytokines, including IL-6 and TNF-α, were observed at 6 and 12 months post-infection. The mean IL-6 level in long COVID-19 patients was 9.4 pg/mL at

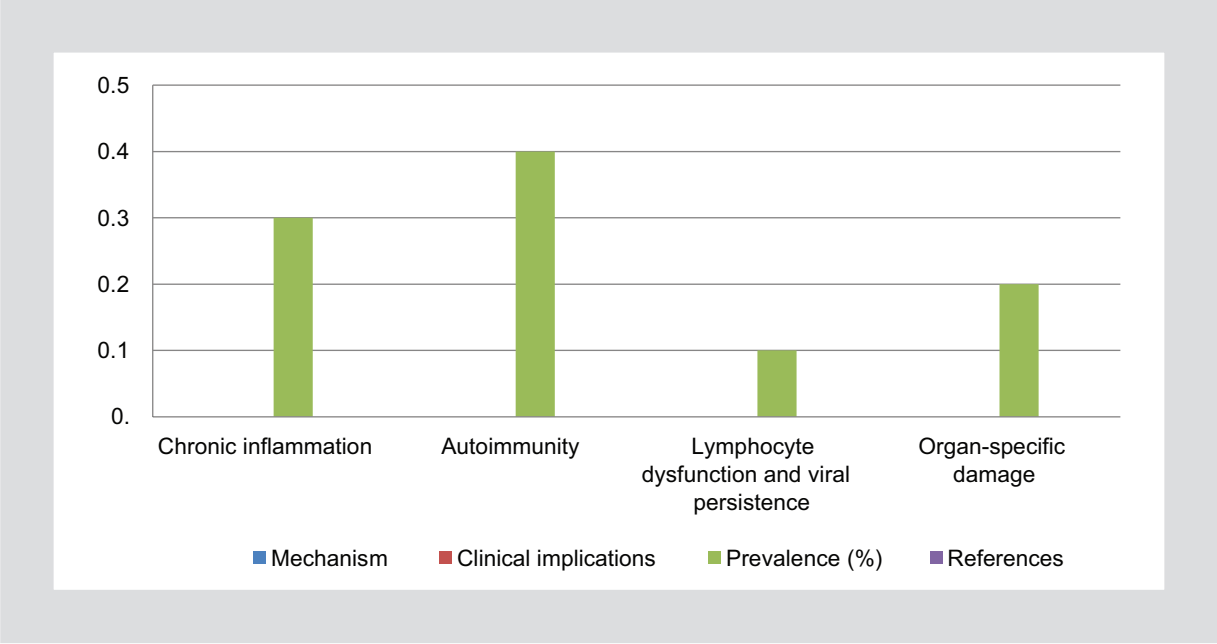

Figure 1. *Pathophysiological mechanisms and clinical correlates in long coronavirus disease chart title.*

6 months, whereas it was 2.1 pg/mL in the control group $p < 0.001$. Similarly, TNF-α levels were significantly higher, with a mean of 7.8 pg/mL versus 1.9 pg/mL in controls $p < 0.001$.

Patients exhibiting elevated IL-6 and TNF-α levels were more likely to experience persistent respiratory symptoms, including dyspnea and fatigue, and presented with imaging evidence of lung fibrosis on follow-up chest CT scans. Approximately 30% of the participants n = 60 had signs of interstitial lung abnormalities. These findings underscore the role of ongoing inflammation in the pulmonary sequelae of long COVID-19.

In addition, neurological symptoms, particularly cognitive dysfunction and brain fog, are correlated with increased systemic inflammation markers. Elevated cytokine levels were significantly associated with reduced cognitive performance scores assessed by the Montreal Cognitive Assessment, with a mean score of 23.1 in patients with high inflammatory markers compared with 28.7 in those with lower cytokine levels $p < 0.05$.

Autoantibody prevalence and symptom association

A notable proportion of long COVID-19 patients present with autoantibodies. Approximately 40% n = 80 of the participants in the study cohort had detectable autoantibodies, with higher frequencies observed in individuals reporting neurological and cardiovascular symptoms. Autoantibodies targeting phospholipids are found in 25% of patients and are correlated with myocarditis and other cardiovascular complications. Autoantibodies against interferons were identified in 15% of the participants, primarily those with severe fatigue and multisystem involvement.

Patients with autoantibody-positive profiles had a greater prevalence of myocarditis, with 20% n = 40 having confirmed myocarditis on echocardiographic and cardiac MRI evaluations. These data suggest a potential autoimmune component underlying the cardiovascular manifestations of long COVID-19.

Lymphocyte dysfunction and viral persistence

A significant reduction in CD8+ T-cell counts was observed in long-term COVID-19 patients, particularly those with severe fatigue and cognitive symptoms. The mean CD8+ T-cell count in symptomatic individuals was 450 cells/μL, whereas it was 870 cells/μL in asymptomatic controls $p < 0.001$. This reduction suggests that a compromised adaptive immune response contributes to prolonged illness.

In addition, persistent SARS-CoV-2 RNA was detected in gut biopsies from 10% n = 20 of the participants, supporting the hypothesis that viral persis-

Table 1. The types of autoantibodies detected and their corresponding clinical presentations

Autoantibody type	Symptom correlation	Prevalence (%)
Anti-phospholipid	Myocarditis, thrombotic events	25
Anti-interferon	Fatigue, severe respiratory symptoms	15
Anti-nuclear antibodies	Generalized fatigue, joint pain	10

Table 2. Prevalence of organ-specific damage in long-term COVID-19 patients

Organ system	Type of damage	Prevalence (%)
Respiratory system	Lung fibrosis	30
Neurological system	Cognitive impairment	25
Cardiovascular system	Myocarditis	20

COVID-19: coronavirus disease-19.

tence is a potential driver of chronic immune activation. Patients with positive viral RNA findings reported more severe gastrointestinal symptoms, including abdominal pain and diarrhea (table 1).

Organ-specific damage and imaging findings

Imaging studies have provided insights into the structural impact of long COVID-19. The prevalence rates observed are shown in table 2.

Patients with lung fibrosis presented the highest inflammatory marker levels, with mean IL-6 levels exceeding 10 pg/mL. Neurological imaging through brain MRI revealed signs of microvascular injury in 15% of patients with cognitive symptoms, further linking chronic inflammation and vascular damage to cognitive dysfunction.

Figure 1 summarizes the interconnected pathophysiological mechanisms and clinical outcomes observed in long COVID-19 patients, emphasizing the need for multidisciplinary management.

Therefore, in summary:

1. Chronic inflammation, as indicated by elevated IL-6 and TNF-α levels, is strongly associated with respiratory and neurological symptoms in long COVID-19 patients.
2. Autoantibody production is prevalent, with significant correlations with cardiovascular and systemic manifestations.
3. Lymphocyte dysfunction, particularly reduced CD8+ T-cell levels and evidence of viral persistence, contributes to sustained immune dysregulation.
4. Imaging studies confirmed substantial organ damage, including lung fibrosis, myocarditis, and cognitive impairment.

These findings highlight the multifactorial nature of long COVID-19, which is driven by immune dysregulation, autoimmunity, and potential viral persistence. These results support the need for targeted immunomodulatory and antiviral therapeutic strategies.

Discussion

Emerging research suggests that factors beyond direct SARS-CoV-2 infection may contribute to long-term COVID-19 pathophysiology. Pintos-Pascual et al. 2022[4] explored whether SARS-CoV-2 is the sole driver of long COVID-19 or whether other mechanisms, such as latent viral reactivation, for example, Epstein-Barr virus and human herpesvirus-6, immune dysregulation, and post viral syndromes, play a role. These findings indicate that persistent symptoms in some individuals may be linked to immune system alterations triggered by COVID-19 rather than to ongoing viral presence alone. These findings align with our findings of CD8+ T-cell dysfunction and autoantibody production, suggesting broader immune response dysregulation. Furthermore, their discussion of the role of preexisting autoimmune predispositions and environmental factors supports the notion that long COVID-19 may represent a spectrum of post viral syndromes rather than a single disease entity. These insights reinforce the importance of multifactorial diagnostic approaches and highlight the need for further studies investigating potential cofactors influencing long COVID-19 outcomes. Future research should assess whether targeted immunomodulatory strategies, including therapies addressing latent viral reactivation, could be beneficial in certain patient subgroups.

These findings provide critical insights into the complex pathophysiological mechanisms underlying long COVID-19, emphasizing the roles of chronic inflammation, autoimmunity, lymphocyte dysfunction, and viral persistence. This discussion explores these key aspects, integrating the current literature to contextualize the implications for patient management and future research.

Chronic inflammation and long-term symptomatology

The persistent elevation of proinflammatory cytokines, specifically IL-6 and TNF-α, observed at 6 and 12 months post-COVID-19 infection underscores the significant role of chronic inflammation in the pathogenesis of long COVID-19. The data demonstrate that patients with elevated cytokine levels experienced more severe respiratory symptoms and structural lung changes, with approximately 30% showing evidence of interstitial lung abnormalities. These findings align with studies[5,6] that identified chronic inflammation as a central driver of persistent respiratory dysfunction and lung fibrosis in post-acute COVID-19. Elevated IL-6, in particular, has been implicated in cytokine storm phenomena during acute infection, and prolonged IL-6 elevation may perpetuate tissue injury and fibrotic remodeling[7].

Neurological manifestations, including cognitive impairment and "brain fog," were also strongly correlated with systemic inflammatory markers. Patients with higher IL-6 and TNF-α levels had significantly lower cognitive performance scores, as measured by the Montreal Cognitive Assessment MoCA. This association suggests a potential link between chronic inflammation, endothelial dysfunction, and microvascular injury, which is consistent with findings from Guo et al. 2022[1], who reported similar cognitive deficits in long COVID-19 patients with elevated inflammatory markers. Neuroinflammation and impaired cerebral perfusion may contribute to these symptoms, highlighting the need for longitudinal neuroimaging and cognitive evaluations in affected individuals[8].

Autoimmunity and its clinical implications

The detection of autoantibodies in 40% of the cohort reinforces the hypothesis that autoimmunity plays a pivotal role in long-term COVID-19 pathophysiology. The most prevalent autoantibodies are directed against phospholipids, interferons, and nuclear antigens, with distinct clinical associations. Anti-phospholipid antibodies are linked to myocarditis and thrombotic complications and affect 25% of patients. This finding is consistent with research by Zuo et al. 2020[9], who reported elevated levels of antiphospholipid antibodies in COVID-19 patients with thrombotic events[9]. The prothrombotic state induced by these antibodies may exacerbate cardiovascular complications, necessitating careful monitoring for embolic phenomena in long-term COVID-19 patients[10].

Similarly, the presence of anti-interferon antibodies, detected in 15% of the participants, was associated with severe fatigue and respiratory symptoms. This finding supports previous work by Bastard et al. 2021[3], who demonstrated that anti-interferon autoantibodies contribute to impaired viral clearance and increased disease severity[3]. In the context of long COVID-19, persistent immune dysregulation driven by these antibodies may exacerbate systemic inflammation and chronic fatigue syndrome-like symptoms[11].

The autoimmune hypothesis is further strengthened by the detection of antinuclear antibodies ANAs in 10% of patients, which are correlated with joint pain and generalized fatigue. ANAs are hallmark markers of systemic autoimmune diseases, and their presence in long-term COVID-19 raises concerns about the potential for post-viral autoimmune syndromes[12]. These findings warrant the integration of rheumatological assessments into long COVID-19 management protocols, as early identification and treatment of autoimmune complications could mitigate long-term morbidity[13].

Lymphocyte dysfunction and viral persistence

The reduction in CD8+ T-cell counts observed in symptomatic patients with long COVID-19 indicates significant adaptive immune impairment. CD8+ T cells are critical for cytotoxic responses against viral infections, and their depletion may hinder viral clearance, contributing to prolonged symptoms. The mean CD8+ T-cell count in affected individuals was significantly lower than that in asymptomatic controls, which is consistent with the findings of Rha et al. 2021[14], who reported that lymphopenia is a common feature of severe COVID-19 and its chronic sequelae[14].

The detection of persistent SARS-CoV-2 RNA in gut biopsies from 10% of participants provides compelling evidence that viral persistence is a potential driver of chronic immune activation. This phenomenon has been

previously documented in studies by Chertow et al. 2021[15], where viral RNA was identified in multiple tissues long after acute infection resolution[15]. The gastrointestinal tract may serve as a reservoir for viral persistence, explaining the frequent gastrointestinal symptoms reported by long COVID-19 patients. Persistent viral antigens can continuously stimulate the immune system, perpetuating inflammation and tissue damage[16].

These findings highlight the importance of developing diagnostic tools to identify viral reservoirs and tailor antiviral therapies for patients with evidence of persistent infection. Future studies should explore the efficacy of antiviral agents in reducing the viral load and mitigating immune dysregulation in long COVID-19 patients[17].

Organ-specific damage: imaging correlations

Imaging studies revealed significant structural damage in multiple organ systems, corroborating the systemic impact of long COVID-19. Lung fibrosis was the most prevalent finding, affecting 30% of participants, with mean IL-6 levels exceeding 10 pg/mL in this subgroup. Fibrotic lung changes are concerning due to their association with progressive respiratory decline and reduced quality of life. Similar findings were reported by Myall et al. 2021[2], who reported persistent radiological abnormalities in COVID-19 survivors with elevated inflammatory markers.

The neurological imaging findings included microvascular injury in 15% of patients with cognitive symptoms, further supporting the role of chronic inflammation and endothelial dysfunction in cognitive impairment[18]. This aligns with research by Puntmann et al. 2020[19], emphasizing the need for ongoing neurological surveillance in long COVID-19 patients.

Cardiac imaging revealed myocarditis in 20% of the participants, a complication linked to both autoimmunity and chronic inflammation. Cardiac MRI findings of myocardial edema and fibrosis have been reported by Puntmann et al. 2020[19], highlighting the need for cardiological follow-up and potential immunomodulatory interventions to prevent long-term cardiac sequelae[20].

Conclusion

This study highlights the multifactorial nature of long COVID-19, which is driven by immune dysregulation, chronic inflammation, autoimmunity, and viral persistence. The correlations between immunological markers and clinical outcomes emphasize the importance of targeted therapeutic strategies, including anti-inflammatory and immunomodulatory agents, to address the underlying pathophysiology. Future research should focus on identifying biomarkers for disease stratification, optimizing treatment protocols, and developing antiviral therapies to mitigate the burden of long COVID-19 on global health.

Funding

None.

Conflicts of interest

None.

Ethical considerations

Protection of humans and animals. The authors declare that no experiments involving humans or animals were conducted for this research.

Confidentiality, informed consent, and ethical approval. The study does not involve patient personal data nor requires ethical approval. The SAGER guidelines do not apply.

Declaration on the use of artificial intelligence. The authors declare that no generative artificial intelligence was used in the writing of this manuscript.

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