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LONG COVID AS A RHEUMATOLOGICAL CHALLENGE: POST-INFECTIOUS AUTOIMMUNITY MECHANISMS AND CLINICAL PHENOTYPES

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ABSTRACT

COVID-19 has become an important factor modulating the autoimmune response, and its rheumatological consequences include both transient musculoskeletal complaints and full-blown autoimmune diseases. Patients experience activation of innate immunity, dysregulation of T and B lymphocytes, formation of autoantibodies, and NETosis. The rheumatological complications of COVID-19 cover a wide spectrum of phenotypes, ranging from joint pain and myalgia, through reactive arthritis, myopathies, and SLE-like phenotypes, to vascular changes associated with endothelial dysfunction and resembling vasculitis. In patients with rheumatic diseases, the course of infection is more often severe, and the risk of Long COVID is significantly increased. Rare cases of de novo autoimmune diseases have also been observed after COVID-19 vaccination. Diagnosis requires an integrated immunological and clinical assessment, and understanding the molecular basis of Long COVID is key to identifying risk factors and developing personalized therapies.

KEYWORDS

Long COVID; Post-Infectious Autoimmunity; Rheumatological Manifestations; Immune Dysregulation; SARS-CoV-2

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1. Introduction

The impact of SARS-CoV-2 on the immune system and cytokine profile remains unclear. Several reports have been published describing the immunological changes observed in COVID-19, suggesting that SARS-CoV-2 infection can trigger a wide spectrum of immune responses, ranging from mild disorders to severe cytokine storms resulting in multi-organ damage. The emergence of autoimmune phenomena such as Guillain-Barré syndrome, immune thrombocytopenia, rheumatological symptoms including arthritis (, reactive arthritis, chronic arthritis, rheumatoid arthritis), lupus-like syndromes, vasculopathies (Kawasaki-like disease, frostbite-like lesions, vasculitis), and chronic fatigue syndrome, indicating the possibility of these conditions being induced by SARS-CoV-2 [1].

Fedorchenko and Zimba also note that Long COVID is more common and more severe in people with rheumatic diseases, which indicates the need to pay special attention to these patients and monitor their condition for late immunological complications [2].

This review describes the mechanisms of autoimmunity in COVID-19, the rheumatological manifestations of the infection, and their diagnosis.

2. Methodology

This paper was created as a literature review regarding the mechanisms of autoimmunity and rheumatological manifestations of Long COVID syndrome. A literature search was conducted using Google Scholar. Combinations of keywords were used, including: Long COVID, post-COVID syndrome, SARS-CoV-2, autoimmunity, rheumatic diseases, arthritis, vasculitis, myositis, autoantibodies, NETosis, and molecular mimicry.

Review articles, research papers, observational studies, case reports, and publications on the immunopathogenesis of COVID-19 and Long COVID were included in the analysis. Only peer-reviewed scientific publications available in English were included. Selected articles were qualitatively analyzed for mechanisms of the immune response, autoantibody profile, clinical manifestations, and the impact of SARS-CoV-2 infection on the course of rheumatological diseases. Based on the collected data, a synthesis of the current state of knowledge regarding the rheumatological consequences of COVID-19 was performed.2. Mechanisms of autoimmunity in COVID-19

3. Results

An analysis of the available literature revealed that SARS-CoV-2 infection often leads to long-term immune dysregulation and autoimmunity. The most important mechanisms include excessive production of proinflammatory cytokines, NETosis, endothelial damage, lymphocyte dysfunction, and molecular mimicry.

Studies have detected the presence of autoantibodies such as ANA, ANCA, anti-dsDNA, and antiphospholipid antibodies. The most frequently described rheumatological manifestations of Long COVID included joint and muscle pain, arthritis, myositis-like symptoms, and vascular lesions resembling vasculitis.

Patients with previously diagnosed rheumatic diseases were at higher risk of severe COVID-19 and the development of Long COVID symptoms. These results indicate that Long COVID poses a significant rheumatological challenge and require further research into its mechanisms and diagnostic and therapeutic options.

4. Discussion

4.1 Cytokine storm and activation of non-specific immunity

The process of autoimmunization induced by SARS-CoV-2 infection is a complex phenomenon in which the virus acts as a trigger for the development of autoimmune and autoinflammatory diseases in genetically predisposed individuals, possibly through the abnormal activation of immune pathways associated with the non-specific or specific immune system.[main2] COVID-19 is associated with the activation of numerous components of the innate immune system via Toll-like receptors (TLRs), with particular emphasis on TLR-3, 4, 7, and 8 [3].

According to clinical studies, the third phase of SARS-CoV-2 infection is characterized by massive production of cytokines and chemokines, primarily IL-2, IL-6, IL-7, and TNF-α, which ultimately leads to circulatory and respiratory failure, shock, and multiple organ damage [4]. In the course of COVID-19, the overproduction of IL-6 is one of the key elements of pathogenesis, promoting the induction of pathological B-cell clones and the formation of autoantibodies [5].

4.2 NETosis

One of the most characteristic immunological phenomena in the course of COVID-19 is the increased activation of neutrophils and NETosis, i.e., the process of ejecting extracellular neutrophil networks, chromatin decondensation in the nucleus, disintegration of cellular organelles and mixing of their components, followed by cell membrane lysis [6]. It has been shown that complement activation in COVID-19 activates extracellular neutrophil networks (NETs) [3]. NETs are an important source of hidden antigens and, by releasing nuclear and mitochondrial structures, promote the formation of antibodies, especially antinuclear antibodies (ANA) and antiphospholipid antibodies [7]. In addition, the persistent presence of NETs increases the exposure of autoantigens and subsequently intensifies antigen presentation by dendritic cells, which stimulates the activation of pathological B-cell clones. It has been reported that ANCA autoantibodies are more frequently present in the blood of patients with high NET activity and that they more often present phenotypes resembling vasculitis, suggesting a molecular similarity between COVID-19 and systemic vasculitis [5].

4.3 Endothelial dysfunction and damage

COVID-19 affects the epithelial barrier, endothelial cells, blood clotting, and fibrinolysis. SARS-CoV-2 can directly damage the vascular endothelium, which is one of the key mechanisms initiating autoimmunity. The endothelium is not only a physical barrier, but also an active regulator of the immune response, hemostasis, and vascular homeostasis. Its damage leads to the activation of the complement system, the release of von Willebrand factor, thrombin, and the inflammatory cascade, which promotes the formation of autoantigens and immune aggregates [5].

4.4 Lymphopenia and dysregulation of lymphocyte subpopulations

Lymphopenia is one of the key factors in the pathophysiology of COVID-19 and one of the elements driving the development of autoimmune disorders [2]. SARS-CoV-2 infection leads to dysregulation and damage to the immune system, resulting in lymphopenia, including a decrease in the number of T lymphocytes (Th CD3+, CD4+) and cytotoxic CD8+ T lymphocytes [4]. There is also a weakening of the function of regulatory T cells and a decrease in the number of NK cells. The observed changes may persist for many weeks after infection [2].

Lymphopenia compensatorily increases lymphocyte proliferation, which induces the formation of autoreactive clones. This phenomenon is known from the pathogenesis of systemic lupus erythematosus and other connective tissue diseases — such a sudden decrease in the number of lymphocytes results in an imbalance in the system and leads to the expansion of lymphocytes with a low threshold of reactivity to their own antigens.

Furthermore, SARS-CoV-2 has been shown to impair B lymphocyte function, leading to the formation of a B lymphocyte population with a phenotype similar to that observed in systemic lupus erythematosus, which promotes the production of autoantibodies against numerous nuclear and cytoplasmic structures [2].

4.5 Molecular mimicry

One of the main mechanisms inducing autoreactivity is the phenomenon of molecular mimicry, which consists of accidental similarity of peptide sequences between the SARS-CoV-2 spike protein (S protein) and human autoantigens [8]. Bioinformatic analyses have revealed extensive sequence homologies between the S protein and the human proteome, which may cause loss of peripheral tolerance and trigger autoimmune disease [9].

5. Autoantibody profile in COVID-19 and long COVID

One of the best-documented mechanisms of COVID-19 immunopathology is the induction of autoantibodies, especially in patients with severe disease. Their profile observed in the course of COVID-19 infection and in Long COVID syndrome is diverse and is mainly associated with dysregulation and hyperreactivity of the immune system caused by the SARS-CoV-2 virus [3]. Significant data on this issue is provided by Vlachoyiannopoulos, who indicates that 68.7% of patients with severe COVID-19 tested positive for antinuclear antibodies (ANA), antineutrophil cytoplasmic antibodies (ANCA), anti-dsDNA antibodies, antibodies against β2 glycoprotein, or antiphospholipid antibodies (aPL). It is worth noting that none of the subjects had a previously diagnosed autoimmune disease [5]. These results suggest that COVID-19 can induce both antibodies specific to connective tissue diseases and autoantibodies that play a role in coagulation disorders, which is consistent with endothelial dysfunction and the prothrombotic phenotype of the disease. The presence of prothrombotic autoantibodies provides a biological explanation for the tendency toward thrombosis observed in some patients with COVID-19. Antinuclear antibodies (ANA) are typically detected at low titers and lack high specificity [3].

6. Rheumatological manifestations of Long COVID

The rheumatological consequences of SARS-CoV-2 infection constitute a very diverse group, which includes both mild and transient symptoms as well as fully symptomatic autoimmune diseases. Based on clinical observations and immunological data, we can assume that Long COVID triggers phenomena characteristic of immune system activation, loss of immune tolerance, and the development of autoimmune inflammation, thus acting as both an initiator and modulator of existing immune disorders.

6.1 Arthritis

Arthritis is one of the most common rheumatological manifestations after SARS-CoV2 infection. In many patients, transient joint pain is initially observed, which may be secondary to prolonged activation of non-specific immunity and elevated concentrations of pro-inflammatory cytokines.

In some patients, these symptoms may take the form of full-blown reactive arthritis with a clinical picture similar to classic reactive arthritis. These symptoms usually develop within a few weeks after infection, with typical involvement of large joints. Cases of arthritis with a clinical picture similar to early RA, symmetrically affecting small joints, with the presence of autoantibodies, have also been described [1].

6.2 Connective tissue diseases and SLE-like phenotypes

SARS-CoV2 infection may reveal previously latent autoreactivity or induce phenomena mimicking connective tissue diseases. Numerous studies have raised the issue of the appearance of autoantibodies, mainly ANA, but also anti-Ro/SSA, anti-La/SSB, and anti-dsDNA antibodies, both during acute SARS-CoV-2 infection and during the recovery phase after COVID-19. The literature also contains descriptions of cases of patients who developed full-blown systemic lupus erythematosus (SLE) after recovering from COVID-19. The immunological explanation for the SLE-like phenotypes observed in Long COVID is that the pathophysiology of severe COVID-19 resembles the B-cell responses observed in SLE, and there is a risk—as yet unconfirmed—that patients with COVID-19 will be more susceptible to developing SLE in the future [1].

6.3 Myositis

Myositis during or after SARS-CoV2 infection develops a variety of clinical manifestations. Some patients experience elevated creatine kinase levels, muscle pain, and proximal muscle weakness, which can be described as myositis-like. Among the autoantibodies characteristic of inflammatory myopathies, the literature indicates positive results for the presence of anti-MDA5 antibodies in some patients with COVID-19. In addition, there are many reports of rhabdomyolysis occurring in patients, probably secondary to viral myositis [1].

6.4 Vascular changes

Vascular changes are one of the best-known rheumatological manifestations of COVID-19. They may refer to small vessel inflammation, such as leukocytoclastic vasculitis, but also to phenotypes suggesting medium or large vessel inflammation.

Zacharias et al. demonstrated that severe COVID-19 is associated with endothelial damage, coagulation disorders, and thromboembolic phenomena. The authors of the study indicate that SARS-CoV-2 affects epithelial barriers, endothelial cells, coagulation, fibrinolysis, and the immune system, which may be the basis for the development of vascular sequelae, including vasculitis-like in Long COVID [5].

The presence of autoantibodies may also play an important role. Vlachoyiannopoulos noted the presence of p-ANCA and c-ANCA in 6.9% of patients with severe COVID-19 and the frequent occurrence of antiphospholipid autoantibodies (aCL, β2-GPI), in the absence of a history of autoimmune diseases ( ), thus pointing to SARS-CoV-2 as a potential trigger for vascular autoimmunity [5].

Also noteworthy is the ample evidence pointing to the role of NET-osis in the pathogenesis of vascular changes—extracellular neutrophil networks contribute to immunothrombosis in the course of COVID-19 ARDS, which may explain symptoms such as microangiopathies, livedo reticularis, perfusion disorders, and chronic vascular symptoms in some patients with Long COVID [7].

7. New rheumatic diseases after COVID-19 vaccination

There have been a few reports of new autoimmune diseases with a rheumatological phenotype, manifesting relatively soon after vaccination against COVID-19. The analysis by Matsuda et al. presents three such examples: rheumatoid arthritis, ANCA-associated vasculitis, and systemic lupus erythematosus, where symptoms appeared one to several days after vaccination [10]. In their literature review, the authors identified a total of 7 cases of rheumatoid arthritis, 37 cases of ANCA-associated vasculitis, and 18 cases of systemic lupus erythematosus temporally associated with vaccination, and the median time from vaccination to the onset of the first symptoms of the disease was approximately 11–12 days [10]. The cases described suggest that the patients met the applicable classification criteria and presented with typical laboratory abnormalities. For example, newly diagnosed rheumatoid arthritis after vaccination was characterized by high inflammatory activity and the presence of RF/ACPA, ANCA-associated vasculitis by positive MPO-ANCA and features of inflammation in the biopsy, and systemic lupus erythematosus by the presence of anti-Sm/anti-RNP antibodies and hypocomplementemia [10]. The authors suggest that the stimulation of innate immunity induced by mRNA vaccines could trigger or exacerbate previously latent autoimmunity in some individuals with specific genetic predispositions [10]. Despite the fact that the reactions described are extremely rare, these cases indicate the need for clinical vigilance and individual risk assessment in patients, especially those with a predisposition to autoimmune diseases.

8. Long COVID in patients with rheumatic diseases

Patients with rheumatic diseases are a group particularly vulnerable to severe COVID-19, which is associated with both disease activity and immunosuppressive treatment. Data from a large population analysis show that patients with rheumatic diseases are more likely than the general population to require hospitalization and treatment in intensive care units, especially if they have systemic vasculitis or connective tissue diseases [7]. Similarly, in a review of the literature from the first phase of the pandemic, the authors noted that active autoimmune disease and the use of biological therapies targeting T cells or NK cells increase the risk of infection and pulmonary complications in the course of COVID-19 [7]. Rituximab therapy is of particular clinical importance in the treatment of patients with rheumatic diseases. Clinical studies have shown that B-cell depletion weakens the antiviral response and increases the risk of severe SARS-CoV-2 disease, and patients treated with rituximab are more likely to require hospitalization and have delayed virus eradication [11]. This mechanism is confirmed by immunological studies showing that in patients with a low number of B cells, the decrease in antibody levels is most pronounced, which on the one hand weakens the humoral response and on the other hand increases susceptibility to the long-term effects of COVID-19 [2]. Analyses of the treatment of patients after COVID-19 have pointed out that rituximab therapy may predispose to persistent viremia and be associated with a more severe course of COVID-19, therefore requiring special vigilance in the course of COVID-19 [7].

Long COVID in patients with rheumatic diseases also has a clear immunological component. Observational studies have described persistent activation of monocytes and neutrophils after SARS-CoV-2 infection, prolonged cytokine production, and T-cell dysregulation, which may contribute to joint inflammation recurrence and worsening control of the underlying disease after infection [1]. This could be due to long-term immune system dysfunction leading to chronic activation of the IFN-I pathway and expansion of the pool of autoreactive B lymphocytes, which has also been described in immunophenotypic pathway studies in patients with COVID-19 with musculoskeletal long COVID [2].

9. Immunological basis for the diagnosis of long COVID with a rheumatological phenotype

Makowska notes that many patients after SARS-CoV-2 infection have persistent abnormal immunological test results, such as the presence of ANA antibodies, decreased complement levels, or elevated concentrations of inflammatory markers, which may indicate the early stages of a developing autoimmune disease. The author emphasizes the need to determine the autoantibody profile and systematically monitor patients whose symptoms persist for ≥12 weeks after infection, especially when new musculoskeletal or systemic unexplained symptoms appear [7].

Immunological studies have shown that some patients with COVID-19 have persistent chronic monocyte activation and impaired T and B lymphocyte function, which promotes the formation of autoantibodies and the development of immune tolerance disorders. Cell studies described by the authors of Cells indicate that an inappropriate cytokine balance and persistent activation of interferon pathways sustain

inflammation after infection, which justifies immunological testing in patients suspected of having Long COVID with a rheumatological phenotype [2].

Clinical studies on the rheumatological consequences of COVID-19 indicate that chronic joint pain, myalgia, and inflammatory symptoms may resolve with both nonsteroidal anti-inflammatory drugs and short courses of glucocorticosteroids. but the decision to use immunomodulatory drugs should be preceded by the exclusion of active infection and an assessment of the autoantibody profile. A review of joint manifestations after COVID-19 emphasized that the diagnosis should include testing for RF, ACPA, ANA, ENA, ANCA, and inflammatory markers to distinguish reactive inflammation from newly revealed rheumatic disease [7].

The importance of thorough immunological diagnostics is also emphasized by reports of new cases of myositis and vasculitis following COVID-19. A study on rheumatological manifestations of COVID-19 describes cases of ANCA-associated vasculitis and myositis in which the development of ANCA autoantibodies or an increase in creatine kinase activity preceded full-blown disease, indicating the need for early diagnosis of laboratory abnormalities in patients with persistent symptoms after infection [5].

Recommendations for the clinical evaluation of patients with rheumatic diseases indicate that tests after COVID-19 should include organ function tests (with particular emphasis on the respiratory system), immunological tests with antibody panel determination, and assessment of the activity of the underlying disease. In a study for patients with rheumatic diseases in the context of COVID-19, particular caution is recommended in interpreting positive antibody test results, as they may occur temporarily after infection and do not always indicate a fully symptomatic autoimmune disease. In such a situation, clinical and serological evolution in the following months is important [7].

10. Conclusions

COVID-19 and Long COVID syndrome are a significant challenge in rheumatology due to the ability of SARS-CoV-2 to both initiate and modulate the autoimmune response. Current immunological and clinical data indicate that infection with this virus can cause a wide range of immune system disorders, including: activation of the innate response with cytokine overproduction, TLR activation, lymphopenia, and increased NETosis, as well as dysregulation of the specific response, including B-cell activation, T cell dysfunction, and autoantibody formation. Furthermore, due to molecular mimicry between the SARS-CoV-2 S protein and human autoantigens, the risk of developing autoimmunity is increased. Numerous studies have detected the presence of ANA, ANCA, antiphospholipid autoantibodies, anti-dsDNA, anti-SSA/Ro, and antibodies characteristic of myositis, which may mean that COVID-19 has the potential to break immune tolerance. The spectrum of rheumatological manifestations includes mild symptoms such as chronic pain and myalgia, but also severe autoimmune diseases such as reactive arthritis, connective tissue diseases, myositis, and vasculitis-like phenotypes resulting from vascular endothelial damage and immunothrombosis. SARS-CoV-2 may also reveal latent autoreactivity, leading to SLE-like phenotypes or full-blown SLE. It is worth mentioning that COVID-19 vaccinations, although extremely safe, have in isolated cases preceded the development of de novo rheumatic diseases such as rheumatoid arthritis, ANCA-associated vasculitis, or systemic lupus erythematosus, which is likely associated with excessive activation of the innate immune system in predisposed individuals. Patients with rheumatic diseases are at particular risk of severe COVID-19 and the development of long-term post-infectious complications. Immunosuppressive therapies, particularly B-cell depletion, increase the risk of complications, prolonged viremia, and the Long COVID inflammatory phenotype. Clinical data indicate that chronic symptoms after COVID-19, including joint pain, fatigue, muscle weakness, as well as exacerbations of autoimmune diseases such as rheumatoid arthritis or systemic lupus erythematosus, are more common in patients with previously diagnosed rheumatic diseases than in healthy individuals. Persistent abnormalities in the immune profile, including positive ANA, decreased complement components, the presence of ANCA, and elevated inflammatory markers, may reflect the early phase of the autoimmune process and require ongoing monitoring. Available evidence suggests that COVID-19 acts not only as a trigger for autoimmunity but also as a modulator, therefore all rheumatological consequences of infection should be subject to chronic clinical supervision. Effective diagnosis of patients after COVID-19 requires the cooperation of many specialists and is based on a combination of immunological assessment, organ function monitoring, and regular monitoring of disease activity. It is particularly important to recognize early abnormalities in laboratory tests, which may precede the onset of full clinical symptoms. Long COVID is currently one of the most rapidly developing fields in rheumatology, still requiring in-depth research into its mechanisms, predisposing factors, and therapeutic strategies that will allow for more effective patient care.

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