DERLEME REVIEW

DOI: 10.31609/jpmrs.2021-82578

# SARS-COV-2: A Current Update About Miscellaneous Symptoms

## SARS-COV-2: Çeşitli Belirtiler Hakkında Güncel Bir Derleme

Tuba Tülay KOCA[a], Saime SAĞIROĞLU[b]

[a]Department of Physical Medicine and Rehabilitation, Kahramanmaraş Sütçü İmam University Faculty of Medicine, Kahramanmaraş, TURKEY
[b]Department of Otorhinolaryngology, Kahramanmaraş Sütçü İmam University Faculty of Medicine, Kahramanmaraş, TURKEY

**ABSTRACT** Severe acute respiratory syndrome-coronavirus-2 (SARS-CoV-2) particle causes different clinical responses in the host depending on the genetic background and prolonged symptoms may develop after recovery. With today's knowledge, we may describe SARS-CoV-2 infection the cause of an autoimmune viral vasculopathy clinic. With our current information, it is not clear which patients will develop severe or prolonged disease. There are many different pathophysiological mechanisms that cause coronavirus disease-2019-related symptoms like chronic pain, fatigue, sarcopenia, vasculopathy, autoimmun disorders, arthritis and central sensitization etc.We now know that SARS-CoV-2 causes immune response and life-threatening conditions as a result of microthrombosis and endothelial dysfunction in many vital organs. Also it triggers an immune response in the host. This immun reaction may trigger autoimmune diseases in the long term. In this review, we especially focused on miscellaneous symptoms caused by virus, from the perspective of a physiatrist, besides systemic complications.

**Keywords:** SARS-COV-2; chronic pain; sarcopenia; arthritis; neurologic complications

**ÖZET** Şiddetli akut solunum sendromu-koronavirüs-2 [severe acute respiratory syndrome-coronavirus-2 (SARS-CoV-2)] partikülü, genetik zemine bağlı olarak konakta farklı klinik yanıtlara neden olur ve iyileşmeden sonra uzun süreli semptomlar gelişebilir. Günümüzün bilgisiyle, SARS-CoV-2 enfeksiyonunu bir otoimmün viral vaskülopati kliniğinin nedeni olarak tanımlayabiliriz. Mevcut bilgilerimizle hangi hastaların şiddetli veya uzun süreli hastalık geliştireceği net değildir. Kronik ağrı, yorgunluk, sarkopeni, vaskülopati, otoimmün bozukluklar, artrit ve santral sensitizasyon gibi SARS-CoV-2 ile ilişkili semptomlara neden olan birçok farklı patofizyolojik mekanizma vardır. SARS-CoV-2'nin birçok hayati organda mikrotromboz ve endotel disfonksiyonunun sonucu immün yanıt ve yaşamı tehdit eden durumlara neden olduğunu artık biliyoruz. Ayrıca konakta bir immün yanıtı tetikler. Bu immün reaksiyon, uzun vadede otoimmün hastalıkları tetikleyebilir. Bu derlemede, sistemik komplikasyonların yanı sıra özellikle bir fiziyatrist bakış açısından virüsün neden olduğu çeşitli semptomlara odaklandık.

**Anahtar Kelimeler:** SARS-COV-2; kronik ağrı; sarkopeni; artrit; nörolojik komplikasyonlar

The rapid spread of severe acute respiratory syndrome-coronavirus-2 (SARS-CoV-2) caused a public health crisis worldwide. The situation of SARS-CoV-2 consists of an immune response that progresses to multi-organ failure from the initiation of the viral infection into the body and causes viral clearance or death in some patients.[1] A subgroup of recovered patients are likely to experience prolonged symptoms. When we look at SARS-CoV-2 infection in terms of pain, we encounter chronic pain both related to the primary virus itself and the clinical conditions it causes.[2]

The prevalence of chronic pain in coronavirus disease (COVID) patients in need of intensive care varies between 14-77%. Pain also emerges as a factor affecting the return to work and quality of life after discharge. The risk of developing chronic pain seems to be greater, especially in those who develop

**Correspondence:** Tuba Tülay KOCA
Department of Physical Medicine and Rehabilitation, Kahramanmaraş Sütçü İmam University Faculty of Medicine, Kahramanmaraş, TURKEY/TÜRKİYE
**E-mail:** tuba_baglan@yahoo.com

Peer review under responsibility of Journal of Physical Medicine and Rehabilitation Science.

***Received:*** 24 Feb 2021 ***Received in revised form:*** 22 Jun 2021 ***Accepted:*** 27 Jun 2021 ***Available online:*** 06 Jul 2021

severe disease. There are many underlying causes. Processes in intensive care include hyperinflammatory state due to immune response, microthrombosis (hypercoagulable state), neurological involvement (stroke, Guillan Barre syndrome, polyneuropathy... etc), COVID-19 related psychiatric findings and musculoskeletal problems (sarcopenia, arthritis, arthralgia, rabdomyositis, etc...).[3]

The effectual host immune response including innate and adaptive immunity against SARS-CoV-2 seems crucial to control and resolve the viral infection. However, the severity and outcome of the SARS-CoV-2 might be associated with the excessive production of proinflammatory cytokines "cytokine storm" leading to an acute respiratory distress syndrome (ARDS).[4] The current evidence suggests that the hyperinflammatory syndrome results from a dysregulated host innate immune response.[5] Recent studies indicate that a hyperinflammatory syndrome induced by SARS-CoV-2 contributes to disease severity and mortality in SARS-CoV-2.[6] Type I interferon production is impaired and severe cases lead to ARDS and widespread coagulopathy.[7] Eriksonn et al. have identified complement activation through the mannose-binding lectin pathway as a novel amplification mechanism that contributes to pathological thrombosis in critically ill SARS-CoV-2 patients.[8] Immune system responses and immune-mediated autoimmun diseases result from interactions between our genetic makeup and environmental exposures. Viral infections are blamed for many autoimmune diseases (rheumatoid arthritis, ankylosing spondylitis, Behçet' syndome, psoriasis...). Time will tell whether those with SARS-CoV-2 will develop autoimmune diseases in the long term. We know that it causes some immune-mediated diseases (Guillan Barre syndrome, Bell's palsy, Miller Fisher syndrome, acute/subacute arthritis...) in the short term. SARS-CoV-2 enters the human cell after binding to the angiotensin-converting enzyme 2 receptors, that are present in various organs. Besides the involvement of respiratory system, other systems like cardiovascular, renal, gastrointestinal and central nervous are not uncommon.[9] Although SARS-CoV-2 is commonly considered a respiratory disease, there is clearly a thrombotic potential that was not expected. The pathophysiology of the disease and subsequent coagulopathy produce an inflammatory, hypercoagulable, and hypofibrinolytic state.[10] Like previous coronaviruses, SARS-CoV-2 seems to cause damage to many organs and tissues through endothelial dysfunction.[11] After SARS-CoV-2, we usually see sarcopenia in mild and severe cases. Sarcopenia is a condition characterized by loss of skeletal muscle mass and function. Although it is primarily a disease of the elderly, its development may be associated with conditions that are not exclusively seen in older persons. Especially COVID-19-related inactivity, intensive care conditions, hyperimmun response, multiorgan failures, nutritional and appetite problems seem the causes of losses of muscle mass and strength.[12]

Widespread muscle pain and fatigue after SARS-CoV-2 may develop due to cardiopulmonary involvement, as well as due to microtombosis and sarcopenia in the muscles.[13] Elevated creatine kinase levels indicate muscle injury. Cases with persistent fatigue after SARS-CoV-2 are also commonly observed. However, it is unknown in which recovered patients, which symptoms, how long will remain prolonged.[14]

Rhabdomyolysis, which present with pain and fatigue in the lower extremities, has also been reported in cases with severe SARS-CoV-2 infection. Rhabdomyolysis is a life-threatening disorder that manifests with myalgia, fatigue, and pigmenturia; it can also cause an acute renal failure. Viral infection can lead to rhabdomyolysis/miyositis.[15]

The virus may affect the heart directly and indirectly with clinical syndromes of acute myocardial injury, myocarditis, acute coronary syndromes, heart failure, arrhythmias, and venous thromboembolism.[16] In addition, post-COVID cardiopulmonary insufficiency can also cause fatigue and widespread pain. Nutritional and appetite problems may also be affected in patients due to anosmia. Like many systems, SARS-CoV-2 can keep the gastrointestinal system by disrupting liver and pancreas functions. This can cause sarcopenia and general fatigue through malnutrition.[17] It is known that the immune system is highly affected by malnutrition, leading to decreased im-

mune responses with consequent augmented risk of infection and disease severity.[18]

Post-COVID-19 syndrome (known as 'long COVID-19') is a prevalent syndrome. It includes a plethora of symptoms (exercise intolerance, dyspnea, chest pain, chemosensory impairment, lymphadenopathy, rhinitis, and appetite loss, palpitations and orthostatic intolerance) which may last for weeks or more. This condition seems to be related to a virus- or immune-mediated disruption of the autonomic nervous system resulting in orthostatic intolerance syndromes.[19-22]

Long COVID-19 can develop as a result of direct, indirect or post-infectious complications. Angiotensin-converting enzyme two receptors, present on endothelial cells of cerebral vessels, are a possible viral entry point to brain.[22,23] Most common neurological manifestations seen include dizziness, headache, impaired consciousness, acute cerebrovascular disease, ataxia, and seizures. Anosmia and ageusia have recently been hinted as significant early symptoms in SARS-CoV-2.[24] Being a neurotrope virus and causing cytokine response are the mechanisms explained for headache developing after SARS-CoV-2.[22-25] Coronaviruses have important neurotropic affinities and they cause neurological symptoms that range from mild to severe. Neurological complications seem to be more frequent in patients with severe respiratory infections. We see case reports presented with Guillain Barre syndrome, Miller Fisher syndrome, polyneuritis cranialis, acute myelitis, oculomotor paralysis, acute viral meningitis/encephalitis, acute disseminated encephalomyelitis, acute necrotizing hemorrhagic encephalopathy and Bell's palsy associated with SARS-CoV-2 infection in the current literature.[26-30]

Nataf et al. emphasize the links between angiotensin I converting enzyme 2 and dopa decarboxylase and the hypothesis of a systemic failure of the dopamine synthetic pathway during SARS-CoV-2 infection.[31] Antiphospholipid antibody syndrome is characterized by recurrent thrombosis and unexplained fetal losses. Among antiphospholipid antibodies (aPL), lupus anticoagulant, anticardiolipin and anti-β2 glycoprotein I are especially important.[32,33] Studies have shown that aPL antibodies are temporarily positive and pathogenic in a significant proportion of SARS-CoV-2 patients.[34] We found a sufficient number of case reports in the literature that developed systemic lupus erythematosus and perniolike clinical signs due to SARS-CoV-2. The way in which the virus can cause lupus clinic will be revealed by enlightening its pathophysiology.[35,36]

Perrin et al. explain permanent symptoms and findings after SARS-CoV-2 with the pathophysiological mechanism seen in Chronic Fatigue Syndrome/Myalgic Encephalomyelitis (CFS/ME).[37] Post-mortem research indicated the virus had crossed the blood brain barrier into the hypothalamus via the olfactory pathway. The pathway of the virus seemed involving disturbance of lymphatic drainage from the microglia in the brain similarly seen in CFS/ME.[37] We know that those who have multiple comorbidities develop severe SARS-CoV-2 infection. On the other hand patients with chronic pain who have multiple comorbidities faced potential treatment disruption during the SARS-CoV-2 pandemic.[38]

Central sensitization describes the change in the brain that develops in response to repetitive nerve stimulation. This condition is also called nerve neuroplasticity. Central sensitization changes develop after repetitive experiences with pain.[39,40] In addition to causing chronic pain, SARS-CoV-2 may cause central sensitization with primary central/peripheral nervous system involvements. In the online survey by Serrano-Ibáñez et al., they found a correlation between SARS-CoV-2-related changes in daily routines and pain intensity, emotional distress, and sensitization scores. The study support that individuals with central sensitization pain syndromes may be at higher risk of developing psychological distress.[41]

One of the most common central sensitization syndromes is fibromyalgia syndrome (FM). In these patients with FM, Sallafi et al. found the scores of all the tests significantly higher in the patients with SARS-CoV-2, which suggests that global FM symptoms are more severe in patients with SARS-CoV-2 infection.[42,43] When we evaluate the SARS-CoV-2 disease in terms of pain, we see that myalgia, headache, chest pain are among the first symptoms.

Ercalık et al. in their study on SARS-CoV-2 patients, found that the most affected pain area was the head and extremities, and they also noticed that in most of the patients the pain in these areas continued in the post-infectious period.[44] Viral infections are also a known cause of acute/subacute arthralgia and arthritis; arthritis can occur after infection by various pathogens, including hepatitis B virus, hepatitis C virus, parvovirus, Epstein-Barr virus, HIV…etc.[45,46] In the SARS-CoV-2 pandemic, viral mono-oligoarthritis or reactive arthritis case reports were encountered in the literature in both critically ill hospitalized patients and mild outpatients.[47-49] Arthritis does not appear frequently enough to enter the main symptoms of the disease. It is also a reason of chronic pain and fatigue in these patients.

In the field of rehabilitation, necessary guides have begun to be created to address these patients in every period. First of all, these patients should be well defined and their needs should be determined before rehabilitation. These include comorbidities, complications from an intensive care unit stay with or without intubation, and the effects of the virus on multiple body systems, including those pertaining to cardiac, neurological, cognitive, and mental health.[50]

Summarily, SARS-CoV-2 infection can be described as a reason of viral autoimmune vasculopathy that causes different multiorgan systems disorders with very different pathophysiopathological pathways. Genetic backround seems to explain the different responses given to this viral particle between individuals in particular. Multidisciplinary approach to the management of the disease picture should be essential.

### *Source of Finance*

*During this study, no financial or spiritual support was received neither from any pharmaceutical company that has a direct connection with the research subject, nor from a company that provides or produces medical instruments and materials which may negatively affect the evaluation process of this study.*

### *Conflict of Interest*

*No conflicts of interest between the authors and / or family members of the scientific and medical committee members or members of the potential conflicts of interest, counseling, expertise, working conditions, share holding and similar situations in any firm.*

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