OPEN ACCESS Freely available online

Rheumatology: Current Research

ISSN: 2161-1149

Editorial

Effect of Coronavirus in Patients with Rheumatoid Arthritis

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DESCRIPTION

COVID-19 is an infectious virus caused by coronavirus acute respiratory syndrome, which mainly affects the lungs and, in some cases, leads to excessive or uncontrolled immune activation and response. The responses of alveolar structural and inflammatory cytokines induced in COVID-19 show similarities with those targeted in the treatment of rheumatoid arthritis. Several clinical studies have started to test the effects of inhibiting IL6, IL1 β, or TNF or targeting cytokine signaling through Janus kinase inhibition in the treatment of COVID-19. Despite these comparisons, COVID-19 and other coronavirus-mediated zoonosis do not cause clinical arthritis, suggesting that a local inflammatory function develops in alveolar structures and leads to sickness. COVID-19 poses a challenge for arthritis patients for several reasons, including the safety of immune interventions during the pandemic. However, we do not suggest that patients with arthritis are at higher risk for COVID-19.

PROBABILITY OF DEVELOPING RA

Musculoskeletal symptoms can develop with coronavirus infection and lead to other respiratory toxicities. In general, viral infections can cause arthritis, but the indications are predominant and range from joint pain to pseudo-and chronic arthritis. While chronic arthritis is caused by hepatitis C and some endemic alpha viruses (such as Chikungunya, Ross River, Barmah Forest, Sindbis, O'nyongnyong, and Mayaro viruses) and self-limiting arthritis occurs with parvovirus B19, rubella, or hepatitis B. In contrast, coronavirus is more likely to cause joint and myalgia than clinical arthritis. Joint and muscle pain are moderately rare (occurring in 1μg). Glucocorticoids do not appear to be beneficial in SARS, but they have the potential to relieve the musculoskeletal manifestations of COVID-19. In general, COVID-19 seems to be patent or be complicated by mild to moderate musculoskeletal symptoms that are indistinguishable from those associated with other respiratory viruses, such as influenza infection. In a large Korean study, they observed that co-infections with endemic human coronavirus, Para-influenza virus, and Delta viruses increased the probability of developing RA. However, there are no reports of people developing autoimmune arthritis, such as Rheumatoid Arthritis (RA), after being infected with SARSCoV2.

Treatment of rheumatic diseases

The COVID-19 disease has an intense effect on the treatment of rheumatic diseases. On the one hand, a large portion of the predictable operations of rheumatology units have been impacted by the developing need to care for COVID-19 patients; on the other hand, the need to maintain the high level of care required to treat rheumatic diseases. The reduction in the number of doctors currently in the rheumatology profession due to illness, isolation, or participation in COVID-19 units, which is difficult to address with targeted treatments, has now become serious. Oral analgesics such as acetaminophen oxycodone and hydrocodone used to treat rheumatic diseases. Rheumatologists must advance their work by demanding to postpone all non-emergency visits and weighing the potential harm of delaying an in-person visit against the potential harm of infection. Indeed, the COVID-19 pandemic has gradually disrupted the traditional model of care, both for rheumatic patients and for rheumatologists, accelerating the transition to distant health care.

Occurrence of rheumatoid arthritis effect by coronavirus

The effect of the COVID-19 pandemic on people with inflammatory or autoimmune rheumatic diseases is still unclear. Risk factors associated with severe COVID-19 outcomes include advanced age (>65 years), male sex, and pre-existing comorbidities (hypertension, diabetes, obesity, cardiovascular disease, and respiratory disease, chronic respiratory tract). In addition, immune-compromised persons, including those with systemic rheumatic diseases, have a higher risk of infection. This challenge has been undertaken in Europe and the United States, involving the use of hydroxychloroquine, chloroquine, and tocilizumab. Regulatory agencies and manufacturers should consider these drugs to avoid the unintended recurrence of rheumatic diseases due to treatment disruption. COVID-19 is characterized by an excessive or uncontrolled host immune

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Received: October 06, 2021; **Accepted**: October 20, 2021; **Published**: October 27, 2021

Citation: Ejeta T (2021) Effect of Coronavirus in Patients with Rheumatoid Arthritis. Rheumatology (Sunnyvale). S19: e001.

response, caused by the destruction of alveolar epithelial cells and activation of T cells in the lung, which induces excessive local production of proinflammatory cytokines which leads to the attraction of large numbers of neutrophils, macrophages to the lungs and mainly the occurrence of rheumatoid arthritis.