OPEN ACCESS Freely available online

Rheumatology: Current Research

Editorial

Coronavirus Returning to Fiery Pathways of Joint Inflammation

Simon Roberto*

Department of Rheumatology, Konkuk University Medical Center, Seoul, Korea

DESCRIPTION

Covid Illness 2019 (COVID-19) is an irresistible infection, brought about by serious intense respiratory disorder Covid 2, which overwhelmingly influences the lungs and, under particular conditions, prompts an inordinate or uncontrolled safe actuation and cytokine reaction in alveolar constructions. The example of supportive of fiery cytokines instigated in COVID-19 has similitudes to those designated in the treatment of rheumatoid joint pain. A few clinical examinations are in progress that tests the impacts of restraining IL-6, IL-1β or TNF or focusing on cytokine flagging through Janus kinase hindrance in the treatment of COVID-19. Notwithstanding these likenesses, COVID-19 and other zoonotic Covid intervened sicknesses don't initiate clinical joint pain, proposing that a nearby incendiary specialty creates in alveolar constructions and drives the infection cycle. Coronavirus establishes a test for patients with incendiary joint inflammation for a few reasons, specifically, the wellbeing of invulnerable mediations during the pandemic. Starter information, notwithstanding, don't propose that patients with incendiary joint inflammation are at expanded danger of COVID-19.

Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) is the third zoonotic coronavirus to cross the species barrier infect humans and become transmitted between humans. Whereas the two other zoonotic coronaviruses, SARS-CoV and Middle East Respiratory Syndrome Coronavirus (MERS-CoV), could be contained at the regional level, SARS-CoV-2 has led to a global pandemic known as coronavirus disease 2019 (COVID-19). Although COVID-19 usually presents as a mild respiratory disease, like infections caused by the four endemic human coronavirus (HCoV-229E, HCoV-NL63, HCoV-OC43 and HCoV-HKU1), it can occasionally lead to severe alveolar inflammation. In a large study from China, ~15% of SARS-CoV-2-infected patients developed shortness of breath, radiological infiltrates in the lung and a drop in blood oxygen saturation (<93%), and 5% had critical illness requiring ventilation. Although such data might slightly overestimate the frequency of severe lung involvement, because testing for SARS-CoV-2 infection is not performed in milder cases and is therefore underestimated, COVID-19 undoubtedly constitutes a substantial risk factor for pulmonary failure. Histopathological findings of the lungs of deceased patients with COVID-19 showed extensive alveolar damage, fibrin deposits, widespread infiltration with immune cells and thrombosis of small and large pulmonary vessels3. Even though the clinical picture of severe COVID-19 resembles that of Acute Respiratory Distress Syndrome (ARDS), these lung histopathological findings suggest that COVID-19 creates a specific form of alveolar disease that is different from other forms of ARDS. The risk of severe lung disease in the context of COVID-19 depends on certain, as yet to be determined, susceptibility factors of the host. Higher age is one of the known risk factors and, as with the other zoonotic coronaviruses, the clinical course of COVID-19 is more severe in older individuals, whereas children and adolescents, who typically develop flu-like symptoms owing to the four endemic coronaviruses, are virtually spared from SARS and MERS. Reports suggest, however, that in very rare cases, Kawasaki-like disease can occur as a complication of COVID-19.

The current COVID-19 pandemic raises several interesting research questions, as well as clinical challenges in the context of rheumatic diseases. In this Perspective article, we address some of these points. We first consider the musculoskeletal symptoms elicited by COVID-19. We then describe how the mechanisms of alveolar inflammation in COVID-19 share similarities with those in Rheumatoid Arthritis (RA), in particular, with respect to the pattern of cytokines involved in driving the inflammatory process. On the basis of this concept, we discuss the potential of currently existing cytokine-blocking strategies to treat COVID-19, as well as their respective impact on the risk of developing viral infection. Finally, we critically discuss the effect of the COVID-19 pandemic on the treatment of patients with rheumatic diseases.

Covid and rheumatic indications

Musculoskeletal indications can create during Covid diseases, likewise with other respiratory contaminations. When all is said in done, viral diseases can cause joint pain, however the range of manifestations is wide, going from arthralgia to misleading and

Correspondence to: Simon Roberto, Department of Rheumatology, Konkuk University Medical Center, Seoul, Korea, E-mail: robermo3@nsi.Kr

Received: June 08, 2021; **Accepted**: June 22, 2021; **Published**: June 29, 2021

Citation: Roberto S (2021) Coronavirus Returning to Fiery Pathways of Joint Inflammation. Rheumatology (Sunnyvale). 11:e121.

ongoing arthritis. While ongoing joint pain can be brought about by hepatitis C and a few endemic alphaviruses, (for example, Chikungunya, Ross River, Barmah Forest, Sindbis, O'nyong-nyong and Mayaro infections), self-restricted joint inflammation happens with parvovirus B19, rubella or hepatitis B virus6. Conversely, Covid don't commonly cause clinical joint pain but instead arthralgia and myalgia. Joint and muscle torment are very uncommon (happening in <10% of cases) in the endemic Covid, while arthralgia is accounted for in 15% of patients with COVID-19, and myalgia is considerably more incessant (44%). Musculoskeletal manifestations, be that as it may, don't appear to be related with COVID-19 severity8. Myalgia is likewise normal in SARS-CoV and is found in 49-68% of cases; however arthralgia is less incessant (11%). Arthralgia and myalgia are additionally normal in patients with MERS-CoV (32%). Nonetheless, it should be viewed as that these predominance information depend on the foundation of high portions of glucocorticoids (total dosages of >1 g). Glucocorticoids don't appear to be of advantage in SARS or MERS, yet they likely hose the musculoskeletal signs of COVID-19. Generally, COVID-19 appears to give or be muddled by gentle to-unassuming musculoskeletal indications, which are indistinct from those related with other respiratory infections, like flu disease. At long last, another fascinating viewpoint with regards to the connection among Covid and joint pain is that the endemic human Covid are related with an expanded danger of creating RA. In a huge Korean examination, the creators saw that contaminations with endemic human Covid, parainfluenza infection and metapneumovirus agreed with an expanded pace of improvement of RA. Consequently, the COVID-19 pandemic might actually prompt an increment in instances of RA. In any case, until now, there is no report to show that individuals foster immune system fiery joint inflammation, like RA, subsequent to being tainted by SARS-CoV-2 or any of the other six known human coronaviruses.