Review Article

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Therapy Options for COVID-19 from the Rheumatologist Angle

The clinical progression of the severe acute respiratory pattern coronavirus- 2 (SARS- CoV- 2) to critical illness is associated with a systemic and unbridled seditious response of the ingrain and adaptive impunity with the release of a plethora of proinflammatory cytokines nominated "cytokine storm". In the absence of an effective treatment, numerous off- marker agents from the armamentarium of rheumatology are used. Then, from the perspective of a rheumatologist, we will bandy the current remedial strategies in critically ill cases with SARS- CoV- 2 pneumonia. Therefore, we will bandy the agents that aim to target viral entry and its replication into the host cell and those fastening and targeting the seditious response. In this setting, numerous agents have been used with promising results but, not all have been approved by the International Authorities and Institutions. In the first step(viral entry), SARS- CoV- 2 monoclonal antibodies and remdesivir have been approved to be used and, in the alternate step, corticosteroids along with interleukin- 6 impediments, or Janus Kinase impediments are presently used.

Keywords: COVID- 19 • SARS- CoV- 2 mAbs • Remdesivir • Colchicine• DMARDs• Dexamethasone

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Introduction

Severe acute respiratory pattern coronavirus- 2(SARS- COV- 2), the cause of coronavirus complaint 2019 (COVID- 19), surfaced in China at the end of 2019 and has developed into a epidemic. The complaint has a variety of clinical instantiations ranging from asymptomatic, or flu like pattern (low grade fever, sore throat, myalgias, arthralgias, fatigue), but also to the development of bilateral pneumonia that can progress to hypoxia, dyspnea, respiratory failure, thrombotic diathesis, multiorgan failure and death. The host's vulnerable response is allowed to play a cardinal part in the complaint pathophysiology and multiorgan dysfunction. Indeed, the clinical progression of the infection to critical illness is associated with a systemic unbridled seditious response of the ingrain and adaptive impunity leading to inflated inflammation named cytokine release pattern (CRS). There's an original weak response to interferon (IFN) α, β and macrophage (MΦs) activation, that results in delayed polymorphonuclear (PMN) cell reclamation leading to lowered viral concurrence. This causes prolonged vulnerable cell stimulation and the release of proinflammatory cytokines similar as, excrescence necrosis factor- nascence (TNFα), interleukin (IL) - 1, IL- 6, IL- 12, IL- 18, chemokines and numerous others. As a result, high situations of seditious labels similar as D- dimers, C- reactive protein (CRP), ferritin and fibrinogen are produced. Latterly, a dysregulation of the adaptive impunity with drop of lymphocytes, substantially CD 4 and CD 8 may do. All the below may contribute to the pathological features of severe COVID-19 pneumonia expressed with seditious infiltrations, verbose alveolar damage and microvascular thrombosis.

Material and Methods

SARS- CoV- 2 infection

SARS- CoV- 2 is an enveloped contagion with a globular morphology and a single- stranded RNA (ssRNA) genome. The SARS- CoV-2 genome encodes four structural proteins shaft(S), envelope (E), membrane (M), and nucleocapside (N), as well asnon-structure and

appurtenant proteins. The shaft protein contains two subunits S1 and S2 that intervene host cell attachment and irruption. Through its receptor binding sphere (RBD), S1 attaches to angiotensin converting enzyme receptor- 2 (ACE2) on the host cell. This initiates a conformational change in S2 subunit that results in contagion host cell membrane emulsion and viral entry. The viral entry can also be through endocytosis [1]. Once outside, the contagion patches are uncoated and its genome enters into the host cell cytoplasm. Through its ssRNA the contagion can directly produce its proteins and new genome in the cytoplasm by attaching to the host's ribosomes, which translates the viral RNA to make proteins that will make RNA polymerase. Through the RNA polymerase, small RNA beaches are made, which will be read by the host's ribosomes in the endoplasmic reticulum to help make up new structural factors of the contagion. Therefore, new viral forms are made which are released from the host cells by exocytosis and can infect other cells. Also the contagion propagation causes towel injury and activation of the vulnerable system. Therefore, signals driven by the SARS- CoV- 2(viral RNAs), pathogen associated molecular patterns (PAMPs) and damage associated molecular patterns (DAMPs) (cellular debris), act on resident towel cells. Indeed, the contagion through ACE2, Risk- Like Receptors(TLRs) and Node- Like Receptors(NLRs) leads to host cell activation and product of pro-inflammatory cytokines, like IL- 1, IL- 6, TNFα and others. These cytokines act on their own receptors in conterminous cells, with farther cell activation and an inflated product ofpro-inflammatory cytokines takes place, performing in complaint progression and deterioration [2, 3].

COVID- 19 treatment

COVID- 19 is a new epidemic complaint with fatal issues, in some cases, and significant worldwide health consequences. To treat COVID- 19 is a grueling task for croakers and rheumatologists, since there are no specific medicines to combat SARS- CoV- 2 infection. Therefore, numerous off- marker medicines, from the armamentarium of rheumatic conditions, are now used in COVID- 19 and a large number of trials have been published and numerous others are in progress so far. As depicted in. following the viral entry into the host cell and its way of viral lifecycle, as well as the cell activation, there are several points, as implicit targets of SARS- CoV- 2 infection. More specifically Steps 1 to 3 comprise antiviral curatives aiming to reduce the viral replication and its cargo. In way 4 to 8, the medicines used are concentrated to inhibit cytokine cell receptors, its signaling and eventually cell activation, leading to reduced cytokine product. In this setting, natural medicines and corticosteroids play a cardinal part to dwindle the seditious response [4, 5].

The question which arises then's why to use these natural and otheranti-inflammatory agents in an contagious complaint? The answer comes from other well- known diseases like viral hepatitis B and C associated with vasculitis, especially polyarteritis bumps ' and mixed cryoglobulineamia independently. In this setting, except of antiviral agents, anti-inflammatory and immunomodulatory curatives are used. On the other hand, cases with autoimmune rheumatic conditions (ARDs) are also characterized by a dysregulation of the vulnerable system, where severalpro-inflammatory cytokines, similar as TNFα, IL- 1, IL- 6, IL- 17 and others play a significant pathogenetic part in cases with rheumatoid arthritis (RA), spondyloarthopathies (Gym), and seditious bowel complaint (IBD) [6]. Treatment wise, a large number of medicines has been developed and great progress has been achieved over the last two decades with the use of targeted curatives. The use of TNFα impediments, IL- 6 and Janus kinase (JAK) impediments has revolutionized the treatment of these conditions. Likewise, cases with ARDs treated with cs, b and/ or tsDMARDs, appear not to have an increased threat of COVID- 19, compared to the general population. also, cases with ARDs treated with the below agents, when contract SARS- CoV- 2 infection, the complaint is expressed with lower hospitalization, good issues and seems that these medicines may alleviate the clinical course of COVID- 19 [7, 8].

Conclusions

In the last two times there has been a significant progress of scientific knowledge, as respects to COVID- 19 immunopathology and its treatment. Therefore, in the early stages of COVID- 19 and innon-hospitalized cases there's no substantiation to use any antiviral, or immunomodulatory agent. Still, in named sub groups of cases with threat factors of developing severe COVID-19, anti-SARS-CoV-2 mAbs may be considered. In rehabilitated cases with SARS- CoV- 2 infection, which don't need oxygen remedy, no immunomodulatory, or antiviral remedy is needed. In contrary, in rehabilitated cases taking supplemental oxygen, on-invasive, or mechanical ventilation the use of remdesivir in combination with DX, with or without the use of TCZ, or JAK impediments, especially BARI is obligatory. The application of the below immunomodulatory curatives from rheumatology perspective, opens new ways of how to treat severe acute infection conditions, which may

profit from these immunomodulatory treatments. On the other hand, vulnerable response to SARS- CoV- 2 differs among individualities, with different clinical phenotypes. Therefore, it's an imperative to interpret better the vulnerable response against SARS- CoV- 2 in order to further define new remedial ways and strategies [9, 10].

Conflict of Interest

The authors declare that they've no conflict of interest.

Acknowledgment

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