

SHORT COMMUNICATION

An immunotherapy approach to COVID-19

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ABSTRACT

Coronavirus disease 2019 (COVID-19) is a virus-based illness brought on by SARS-CoV-2, a member of the Coronaviridae family that causes severe acute respiratory syndrome. The World Health Organization (WHO) declared the recently discovered, swiftly spreading epidemic to be a pandemic on March 11, 2020. Along with risk-reduction strategies like immunisation and physical and social seclusion, a variety of therapy techniques have been created with the goal of curing the disease.

Regarding its function in eradicating the virus and in causing complications such as cytokine storm syndrome, the immune system is characterised as a double-edged sword in the pathophysiology of COVID-19. As a result, immune-based therapeutic strategies, such as corticosteroids, Intravenous Immunoglobulins (IVIG), interferon therapy, and more COVID-19-specific strategies like anti-SARS-CoV-2-monoclonal antibodies, have emerged as an intriguing area of COVID-19 study.

Key Words: COVID-19, SARS, Coronavirus, SARS-CoV-2, ARD; Pandemic, Pneumonia, Immune, Immunotherapy, Corticosteroids, IVIG, Interferon, Monoclonal antibody

INTRODUCTION

Corona viruses, a genus of the family *Coronaviridae*, are pleomorphic, enveloped, positive sense ssRNA viruses that cause infections of the respiratory system. From mild respiratory illnesses like those caused by HCoV-OC43, HCoV-229E, HCoV-NL63, and HCoV-HKU1 infections to more serious diseases like SARS-CoV-1, SARS-CoV-2, and Middle East Respiratory Syndrome Coronavirus (MERS-CoV), members of this genus are responsible for a wide range of respiratory complications.

The viral envelope encases the viral DNA and the nucleoprotein that surrounds it. Spikes, matrix proteins, and structural proteins are present in the envelope. The interactions between the spike protein and the angiotensin converting enzyme-2 (ACE-2) on the surface of the host cell are what allow SARS-CoV-2 to attach to the cell surface. The pathophysiology of COVID-19 is divided into four stages; asymptomatic stage, upper respiratory tract involvement, lower respiratory tract involvement, and ARDS/ Multi Organ Dysfunction Syndrome (MODS). The pathophysiological, clinical, and immunological characteristics of these stages are summarized in (Table 1).

TABLE 1
The pathophysiological, clinical, and immunological characteristics of these stages

Stage	Pathophysiology	Clinical manifestations	Immunological characteristics
Asymptomatic stage	Virus enters the nasal ciliated epithelial cells via ACE2 and TMPRSS	Asymptomatic	Mild innate response Strong innate response, higher levels of IP-10, MIG, IL-8, MCP
Upper respiratory tract involvement	Presence of virus in sputum Virus-associated damage in alveolar cells (mostly pneumocyte II), apoptosis and death in pneumocytes.	Cough, sore throat histological findings including hyaline membrane, alveolar damage, pneumocyte II hyperplasia, consolidation	Aggravated immune response (especially T cells), cytokine storm, higher levels of IL-6, TNF
Lower respiratory tract involvement	Alveolar macrophages are also targeted by viruses.	High cytokine levels, unremitting fever, high ferritin levels, cytopenia, multi-organ damage	Higher levels of ferritin, IL6, LDH, ddimer, CRP
ARDS/MODS	hemophagocytic lymphohistiocytosis-like cytokine storm		

Corticosteroid

A group of chemicals known as corticosteroids, which can be either artificially produced or naturally produced in the adrenal cortex, have a variety of impacts on the immune system, inflammation, stress

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response, metabolism, bodily fluids, and electrolytes. The inflammatory mediator-producing enzyme phospholipase A2 is inhibited by lipocortin, which is produced as a result of corticosteroids.

Mortality from COVID-19 is typically brought on by a heightened immunological response to the infection. Such responses are controlled by corticosteroids, which also reduce the mortality rate, length of stay in the intensive care unit (ICU), and time spent on mechanical ventilation in COVID-19 patients.

Challenges

High-dose corticosteroids can delay the viral clearance process, despite the fact that short-term corticosteroid treatment has been beneficial. According to the current recommendations, the use of corticosteroids should be restricted due to a variety of negative effects. Increased mortality rates, diabetes, avascular necrosis, femoral head osteonecrosis, psychosis, lung damage induction, and shock are a few of these.

Intravenous Immunoglobulin (IVIG)

Intravenous Immunoglobulin (IVIG), a combination of human immunoglobulin's against microbial infections acquired from recovered patients, is used as a replacement therapy for immunodeficiency's as well as an immunomodulatory drug for autoimmune illnesses and infections. IVIG treatment prevents viral entry into the host cell by blocking viral surface proteins and modifies inflammatory responses after blocking FCR IIa and FCR IIIb on leukocytes in viral infections. IVIG treatment induces Antibody-Dependent Cellular Cytotoxicity (ADCC) by binding to viruses and phagocytosis by binding to FCR receptors.

Challenges

Numerous major side effects, including hemolytic anemia, acute lung injury, thrombosis, cardiac arrhythmia, meningitis, and renal impairment, have limited the adoption of this therapeutic approach in COVID-19 patients; hence, further research is required in this area.

Interferon's

Following viral attachment to cell surface receptors and stimulation of pattern recognition receptors, expression of the interferon-I (IFN-I) family, comprising IFN- and IFN-, is increased (PRRs). The initial line of defense against viruses is provided by the Janus Kinase (JAK) signaling pathway, which is induced by higher levels of IFN- and IFN-. SARS-CoV-2 exhibits better efficiency in proliferation and infection compared to other coronaviruses like SARS-CoV-1, with shorter peak-to-peak times and a higher number of viral particles at the peak, which may be caused by an insufficient IFN-I response. Insufficient innate IFN-I levels can also contribute to disease severity by reducing the expression of IFN-stimulated genes. Consequently, the prognosis of patients with low IFN-I signaling levels is poor.

Challenges

Interferon's cannot be administered to COVID-19 patients due to the immunopathology caused by the immune system imbalance; in these patients, vigilant patient monitoring is essential throughout the course of treatment.

Monoclonal antibodies

Monoclonal antibodies target a variety of molecules that are involved in the pathogenesis of COVID-19, including the spike (S) protein, a viral surface protein, and molecules that cause the cytokine storm syndrome, such as IL-6, TNF, and IL-1. The S protein consists of two subunits: S1 and S2. S1 is involved in viral particle fusion, while S2 is important in attachment to ACE2 with the aid of the N-Terminal Domain (NTD) and a receptor binding domain (RBD) that recognizes sugar moieties. Any of these targets can be neutralized to help prevent viral infection. In serious COVID-19 patients in particular, CSS is an unchecked inflammation that causes fever, ARDS, multiple organ failure, and death. Taking aim at the inflammatory components of CSS can lower the COVID-19 mortality rate.

Challenges

Development of antibodies against the new epitopes and techniques for prompt prediction of the emergence of new variants are required due to the rapid generation of several SARS-CoV-2 new variants.

CONCLUSION

Over the past few years, the COVID-19 pandemic has significantly increased morbidity and mortality rates. Immune-based treatments to treating the condition, such as corticosteroids, IVIG, interferon therapy, and monoclonal antibodies, are a strong contender since the immune system plays a dual role in producing complications like CSS and limiting the disease. Each of these therapy modalities is useful in particular clinical contexts and illness stages to get the greatest clinical benefit with the fewest side effects. Due to the wide range of potential adverse effects, corticosteroids are advised to be taken into consideration in severely or critically ill patients. Despite being seen as helpful in treating a number of infectious disorders, including COVID-19, interferon therapy can lead to a number of immune system abnormalities. A focused therapy approach, monoclonal antibodies have produced encouraging outcomes in individuals with a range of illness stages. A few treatment drugs are recommended in each clinic biological stage of the COVID-19 pathogenesis overall. Monoclonal antibodies can be employed in the asymptomatic stage since bamlanivimab has been used as a prophylactic drug by medical professionals. Depending on the severity of the condition, various medications may be suitable during the propagation phase. Monoclonal antibodies like bamlanivimab work well for patients with mild illness. Monoclonal antibodies, such as tocilizumab, itolizumab, bamlanivimab, and sotrovimab, interferon treatment, Corticosteroids, IVIG, interferon's, and monoclonal antibodies can be helpful in individuals with severe illnesses and those who are going through a complicated phase of their condition.