T lymphocytes are reduced and depleted as a result of serological alterations.

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Abstract

A defining aspect of autoimmune disorders is the failure of mechanisms that ensure the identification of self and non-self. In the past, there has been a growing interest in a subset of regulatory T cells that suppresses T cells in vitro in a contact-dependent manner and preferentially expresses high levels of CD25 and the fork head and winged-helix family transcription factor fork head box P3 (FOXP3). TREGs appear to have a distinctive role in autoimmune disorders, according to recent studies of changing prevalence's and functional efficiencies. The involvement of FOXP3 as a 'master control gene' in the creation and function of TREGs is also supported by clinical results in individuals with mutant FOXP3 genes and a particular polymorphism in the promoter region of FOXP3. In autoimmune disorders, both aberrant TREG production and insufficient inflammatory suppression are thought to be important for illness start and continuation. TREG-related somatic cell therapy is being touted as a promising new treatment option for autoimmune disorders. The current COVID-19 epidemic started in Wuhan (China) in December 2019 and quickly spread to become a global sanitary and economic emergency. The coronavirus SARS-CoV-2 is the etiological agent. COVID-19 has a wide range of clinical symptoms, ranging from asymptomatic infection to severe pneumonia accompanied by multisystem failure, which can result in mortality. All components of the immune system that appear to be responsible for viral elimination and recovery from SARS-CoV-2 are known to be involved in the immunological response to SARS-CoV-2 infection.

Keywords: Autoimmune Disease, FOXP3, Regulatory T lymphocyte, Somatic Cell Therapy, Suppressor Cells.

Introduction

A defining aspect of autoimmune disorders is the breakdown of mechanisms ensuring the immune system's identification of self and non-self. 'Recessive tolerance,' which is created by the thymic deletion of autoreactive T cells, has lately been coined as the primary process leading to self-tolerance. However, even in healthy people, thymic selection is imperfect, and self-reactive cells can develop [1]. On the other hand, 'dominant tolerance,' which is mediated by regulatory T cells actively controlling immune responses, provides a further method for preserving peripheral self. The current COVID-19 pandemic, which was started in Wuhan, China, in December 2019 and is caused by the coronavirus SARS-CoV-2, has spread rapidly over the world. Due to its contagiousness and the huge number of patients presenting with serious infections and a high risk of mortality, its expansion has had devastating impacts in many nations, necessitating specialist medical care in intensive care units (ICU). As a result, on January 30, 2020, the WHO designated it a Global Sanitary Emergency. During this crisis, it's important to emphasise how quickly research studies have progressed, leading to a better understanding of the epidemiology, clinical manifestations, risk factors, and transmission dynamics, as well as the identification of the etiological agent, including its genome, morphological structure, and molecules, its relationship with other coronaviruses, and its entry into host cells by binding the Angiotensin II Conv. All these studies aim at developing diagnostic tests, strategies for clinical management, effective antiviral agents, and eventually, production of a protective vaccine [2,3].

The purpose of this review is to look at the key features of the immune response to SARS-CoV-2, as well as the link between protective and inflammatory responses and the COVID-19 clinical spectrum, which ranges from asymptomatic to severe clinical manifestations. COVID-19 pandemics represent significant immunological research hurdles, according to the assessment. The immunological response to SARS-CoV and MERS-CoV infection in humans and experimental animals has been widely investigated, with numerous great reviews. However, because to the parallels between COVID-19 and SARS and MERS, it will be required to mention research on those infections at crucial points [4,5].

Conclusion

The start of the COVID-19 pandemic in China, which

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quickly spread around the world and had a significant impact on public health and economies, the amount of data gathered on all aspects of the infection, and the speed with which the international scientific community shared that data is truly remarkable. However, because of the rush to publish results, many publications are currently sitting in repositories, awaiting peer review. If this knowledge is to be employed in the development of novel diagnostic, therapeutic, or preventive protocols, a word of caution is in order. It's also worth remembering how little time has passed since the start of the pandemic, during which time there haven't been enough data from in vitro and experimental animal models to assure a better knowledge of COVID-19's biology. Even with these limitations in mind, the evidence presented in the publications reviewed here strongly indicates quantitative and qualitative differences in immune responses among persons infected with SARS-CoV-2, which appear to be linked to COVID-19 clinical symptoms.

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