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Systemic Manifestations brought on by Oral Intake and the Renin-Angiotensin-Aldosterone System

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INTRODUCTION

One of the most intricate human systems is the Renin-Angiotensin-Aldosterone System (RAAS), which is a crucial regulator of arterial blood pressure. Since the actions of the major components were revealed, new chemicals and functions are continually being found. The basic pathway is initiated by the release of renin from the kidney and completes with the synthesis of angiotensin II (Ang II), a potent vasoconstrictor, thanks to the angiotensin-converting enzyme protein (Pro). In 2000, scientists found ACE2, a second enzyme that changes Ang II into Ang-(1-7), a heptapeptide with anti-inflammatory and vasodilating qualities that are the exact reverse of Ang II's actions. The need for this specific enzyme has increased dramatically as a result.

We have combined a number of studies on the relationship between ACE2 and human coronaviruses into a system to give an overview of the pathogenic mechanism. Two human coronaviruses, SARS-CoV and SARS-CoV-2, bind to ACE2 *via* their spike proteins (S), which causes the enzyme to be destroyed. A dysregulated inflammatory response results from ACE2 inhibiting the synthesis of Ang II by turning it into Ang-(1-7). The objective of this review is to elucidate the complex pathophysiological mechanisms behind the oral, cardiac, pulmonary, and systemic multiorgan problems caused by the interaction of the SARS CoV-2 virus with the angiotensin-converting enzyme type 2.

DESCRIPTION

Around the world, both peoples' lifestyles and healthcare systems saw a substantial change as a result of the coronavirus disease 2019 (COVID-19) pandemic. In order to expedite the clinical studies necessary to comprehend the pathophysiology of the SARS-CoV-2 infection, respiratory distress syndrome, hypercoagulation, and multiorgan failure, the international scientific community was compelled to act swiftly and effectively, engaging in unprecedented research efforts. In order to enter cells that have this molecule expressed on their membrane, SARS-CoV-2 interacts with its receptor, angiotensin converting enzyme 2 (ACE2). This review of the current literature summarises the proposed pathophysiological mechanisms that cause the host response to SARS-CoV-2 infection to become dysregulated and inflammatory. Methods: PubMed, Medline, and Web of Science were used to perform the literature search. We looked for the following words: COVID-19, SARS-CoV-2, ACE2, oral mucosa lesion, and oratitis.

The interaction between SARS-CoV-2 and ACE2 receptors causes ACE2 to be downregulated, inhibits RAAS' anti-inflammatory arm, and intensifies the pro-inflammatory effects of Ang II, leading to an increase in the production of inflammatory cytokines and a cytokine storm. Because the prevalence of oral manifestations and COVID-19 is still unknown, they continue to be significant topics for investigation.

CONCLUSION

Additional in-depth research and additional testing, such haematological tests and biopsies, must be carried out on these patients in order to identify the potential etiopathogenesis or factors that affect these oral lesions. Dentists must also be involved in the management of patients who have SARS-CoV2 infection because they may do a complete oral examination to detect, treat, and monitor the development of the lesions listed above. In conclusion, a better understanding of the connection between severe multi-organic complications and the SARS-CoV2 virus-type 2 angiotensin converting enzyme opens the door to research and the development of novel treatments for this infection, which, through the various illnesses that it causes, caused a pandemic and the emergence of a new disease.

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