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Molecular Mimetism Phenomenon in SARS Cov-2

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Mini Review

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Abstract

Background: Following the current SARS Cov2 pandemic, cross-reactivity was proposed as a mechanism that could explain the immunopathology associated with coronavirus infection. The rationale is that the exchange of peptide motifs between SARS Cov2 and human proteins could elicit immune responses capable of attacking not only the virus but also human proteins with the consequent autoimmune pathological sequelae in the human host.

Methodology: A mini narrative review was carried out through various databases from October 2020 to January 2021; the search and selection of articles was carried out in journals indexed in English and Spanish. The following keywords were used: Molecular mimicry, SARS-CoV-2, Covid-19.

Results: There is solid evidence that shows the existence of a link between SARS Cov2 infection and autoimmunity induced by the phenomenon of molecular mimicry.

Conclusions: This review offers information based on the available evidence about the phenomenon of molecular mimicry in SARS Cov2.

Keywords: Molecular mimicry; SARS CoV 2; Covid-19

Introduction

The covid 19 in recent months has put global public health in check, health systems worldwide have been facing this disease that has spread rapidly, in which approximately 15% of infected patients can reach developing a severe form of it, molecular mimicry has currently been proposed as a cause of the autoimmune phenomena observed in Covid-19, the syndrome associated with severe acute respiratory syndrome coronavirus 2 infection. There is very little information about this phenomenon that could explain many things about the current pandemic and even through its knowledge we could counter this disease.

Under the previous considerations, we propose this mini review whose primary objective is to provide information about the phenomenon of molecular mimicry in SARS Cov2.

Materials and Methods

A mini narrative review was carried out, in which the PubMed, Scielo and ScienceDirect databases, among others, were searched. The collection and selection of articles was carried out in journals indexed in first and second languages from 2020 to 2021. As keywords, the following terms were

used in the databases according to the DeCS and MeSH methodology: molecular mimicry; SARS-CoV-2; COVID-19. In this mini-review, 20 original and review publications related to the subject studied were identified, of which 10 articles met the specified inclusion requirements, such as articles that were in a range not less than the year 2020, which were articles full text and that reports on the phenomenon of molecular mimicry reported in SARS Cov2. As exclusion criteria, it was taken into account that the articles did not have sufficient information and that they did not present the full text at the time of their review.

Results

Molecular mimicry is defined as that similarity in the antigenic determinants of two specific molecules. It may happen that a microorganism exhibits molecular mimicry with a molecule of an immunocompetent host. In this case, the antibodies produced against the microorganism would react with the host molecule, causing an autoimmune disease [1,2].

Venkatakrishnan, et al. state that molecular mimicry could explain some autoimmune events observed in covid 19 disease, according to the authors, there are 33 different 8 mer / 9 mer peptides, which are identical between SARS Cov2 and the reference proteome Among human antigens that mimic SARS Cov2, there are four human helicases involved (MCM8, DNA2, MOV10L1 and ZNFX1) [3,4].

This information becomes more solid based on clinical findings such as the large number of reported cases of patients with SARS-CoV-2 infection and Guillain Barre Syndrome, which have existed since the beginning of the current pandemic to date [5].

This phenomenon could be explained by a stressor triggering the entire cell signaling cascade, most of the patients who had more severe complications from covid 19 were affected mainly by hypertension and diabetes. Both induce, among other problems, chronic stress in endothelial cells, which in turn can express molecules in their plasma membranes in an abnormal way as an effect of post-translational modifications of intracellular proteins, including some heat shock proteins. This condition can predispose cells and tissues to molecular mimicry phenomena that can occur during infection [6,7].

In other words, molecular mimicry could be the cause of the aggravation of COVID-19 patients through its participation in crucial steps of the pathogenic cascade. Since severe pneumonia causes a serious decrease in the partial pressure of oxygen, which, in turn, causes cellular stress and an increase in protein synthesis, especially in anti-stress proteins, which accumulate in the cytosol and are undergoing posttranslational modifications, the already modified anti-stress proteins migrate to the membrane of plasma cells [8,9]. Antigenic epitopes of antistress proteins that share molecular mimicry with SARS-CoV-2 proteins become accessible on the outer surface of cells to cross-reactive antiviral antibodies, which act as autoantibodies and cause autoimmunity. Mechanisms of autoimmunity in this way damage and kill host cells. This type of cell death takes place in many organs and tissues causing multiple organ failure, which has already been documented in many patients with severe Covid [10].

Conclusion

There is currently evidence, although little, that demonstrates the existence of a link between SARS Cov2 infection and autoimmunity induced by the phenomenon of molecular mimicry, which is why the scientific community should dedicate itself to investigating the implication of molecular mimicry in the pathogenesis of covid-19, especially through research that provides serological and in vivo data on this phenomenon.

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