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Short Report

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Caution in the association between helminths and severe COVID-19

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It has been argued that in helminth-endemic regions, the intensity of SARS-CoV-2 infection and the severity of the disease it produces (COVID-19) can be satisfactorily reduced, due to helminth-motivated modulation of the immune system, since that, it is pointed out that part of the host's immune response against SARS-C0V-2 includes the uncontrolled elevation of Th2-type cytokines, that is, IL-3, IL-4, IL-5, IL-10, IL-13, IgE and eosinophils, thus infection by pre-existing helminths (their molecules) can reduce the risk of cytokine storm and consequently of COVID-19 [1].

Because unlike protozoa, helminths stimulate Th2-type immune responses, characterized by the secretion of IL-4, IL-5 and IL-13 as a regulatory mechanism of the host's immune response, which takes place more slowly and with the participation of protozoa. Innate and acquired immune systems, which act by mechanisms other than those mediated by cytokines produced by the subpopulation of Th2 helper T cells and which also modulate the activity of the latter [2,3].

In precise terms, the host-helminth interaction has, in regulatory terms, two additional actions. The first is the classic or inhibition of Th1 and Th17 responses (and their corresponding cytokines IL-12, IFN-γ, IL17, IL-23, TNF-α) by Th2 cytokines. The second is the limitation of type 1 and 2 responses by the activation of FOXP3+ regulatory T cells, regulatory B cells, and M2 macrophages, which together cause the release of regulatory cytokines such as IL-10 and, above all, TGF-β [4].

It is also mentioned that the IgG4 isotype, different from IgE, cannot bind Fc receptors on basophils, mast cells and eosinophils and does not activate the complement system or act as opsonin. Then, there is a modified Th2 state (non-inflammatory) in which cytophilic antibodies such as IgE are blocked and the possibility of damage due to inflammation is decreased [3].

From the foregoing, it can be deduced that the ability of helminths to modulate the immune responses of their respective hosts, in order to survive in them, can also attenuate the inflammatory effects of the defensive mechanisms of the parasites and, with this, reduces the immunopathological damage associated with severe infection by SARS-CoV-2 [4].

This hypothesis should be taken with caution because studies in experimental animals on viral-parasite coinfection are inconclusive, due to the possible inadequate interpretation of data from endemic areas due to delays in the notification of COVID-19 cases, and because the information Epidemiological research, especially in the Amazon basin in South America, does not suggest a decrease in the severity of COVID-19 in helminth-endemic regions, because they show prevalences of geohelminths that exceed 70% in some regions without a decrease in the number of cases of people with Severe COVID-19 [1,5].

In this sense, some authors point out that instead of being beneficial for those affected with COVID-19 in relation to their morbidity or mortality, the canonical Th2 immune response driven by helminths could be harmful. It is important to point out that there are several factors that can contribute to the uncertainty regarding the immunoprotective role of helminths against severe COVID-19, among them are the variation of innate immunity of virus response genes, polymorphism of cytokine genes and mutations in the Human Leukocyte Antigen (HLA-DQA1) [1].

Similarly, little is known about the influence of the human virome/microbiome on the immune response to new infections. In addition, it is expected that the risk of severe COVID-19 will increase due to the negative effects that helminth infections have on the human host, such as anemia caused by blood loss, competition for nutrients, intestinal malabsorption syndrome, and dehydration due to severe diarrhea or dysentery, therefore, it is advisable to carry out more and in-depth studies to measure the potential impact of these parasites on the severity of COVID-19 [1].

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