Myocardial injury in hypertensive patients with COVID-19 is related to immune dysfunctions, reveals study

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Researchers from the D'Or Institute for Research and Education (IDOR), in partnership with colleagues from the Rede D'Or, the Evandro Chagas National Institute of Infectious Diseases/FIOCRUZ, the Federal University of Rio de Janeiro, the Bahia School of Medicine and Public

Health, the Federal Fluminense University, and the State University of Rio de Janeiro, have published a study in the *Journal of Clinical Immunology* that reveals alterations in the immune system that may be involved in cardiac muscle injury in hypertensive patients with COVID-19.

Cardiac injury, defined as significant cardiac troponin elevation, is the most reported cardiac abnormality in hospitalized patients with COVID-19. A previous study by the same group of researchers suggests that troponin testing is important for improving the prediction of cardiac risk in these patients.

Among the comorbidities associated with COVID-19 and myocardial injury, hypertension appears with higher prevalence and immune system dysregulation may be associated with this complication. The objective of the research was to investigate the immune system functioning in these patients and identify biological markers that could help better understand myocardial injury.

The study included 193 hypertensive patients with COVID-19, divided into two groups: 47 cases with myocardial injury and 146 without myocardial injury (control group). Comparative analyses were conducted between the groups, considering biological markers and subsets of immune cells. The results revealed significant differences in immune profiles between the two groups.

The research group had demonstrated in a previous study that IL-12 (p70) and IL-10 obtained at hospital admission, along with certain clinical comorbidities, can be biological markers associated with increased severity of COVID-19 in hypertensive patients. In general, cytokines are groups of proteins produced by various cell types that regulate the immune response in the body. Cytokine storm, which refers to the hyperactivation of the immune system due to SARS-CoV-2

infection, can lead to systemic inflammation. In the present study, cytokines IL-ra, IFN-γ, MIP-1α, MIP-1β, IL-17A, TNF-α, and TNF-β showed higher levels in the group of patients with myocardial injury compared to the control group.

Another important characteristic in the context of COVID-19 is lymphopenia, which is a reduction in lymphocytes, the cells involved in the immune response, and is strongly associated with disease severity progression. The researchers found a decrease in the total lymphocyte count, particularly in T lymphocytes, in myocardial injury patients. This result was consistent with a previous study that identified impairment of cell-regulated immunity. It is worth noting that there was also observed dysregulated activation of specific subsets of immune cells, such as NK cells and CD8+ cells.

In summary, the results of this study provide new insights into the possible mechanisms of myocardial injury in hypertensive patients with COVID-19. Immune dysregulation, characterized by altered levels of cytokines and activation of immune cell subsets, appears to play an important role in this complication. These factors can be useful in predicting myocardial injury in hypertensive patients with COVID-19. These findings contribute to a better understanding of the disease and may help in the development of more effective treatment strategies.

More information: Renata Moll-Bernardes et al, New Insights on the Mechanisms of Myocardial Injury in Hypertensive Patients With COVID-19, *Journal of Clinical Immunology* (2023). DOI: 10.1007/s10875-023-01523-6

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