Effect of Statins and Flozins on Cardiomyocytes Contacted With COVID-19 Spike Protein

Adamkiewicz, Laura (School: Sevenoaks School)

Long Covid (LC) is a condition that can occur after Sars-Cov-2 infection or as a result of vaccinations (Post-Covid Vaccination Syndrome (PCVS)). There is a wide range of over 200 registered symptoms, and it is estimated that it affects 5-30% of the people who had COVID-19. This study examines mechanisms and potential interventions for LC or PCVS-induced myocarditis, a common symptom of both. The most prominent hypothesis for cardiac injury in LC and PCVS is that the COVID-19 S-Protein triggers the overproduction of cytokines, proteins secreted by the immune system which are involved in controlling inflammation responses. The aim of the study was to investigated the possibility of reduction of cardiac inflammation by statins and flozins, drugs with recognized pharmacological activity and safety, and the proposed mechanism of anti-inflammatory action. To investigate this, AC16 human cardiomyocyte cells were pre-treated with Rosuvastatin (ROSUVA), Simvastatin (SIMVA), Dapagliflozin (DAPA) and Empagliflozin (EMPA) and then treated with S-Protein or TNF-a. The effect of the drugs on cell survival, apoptosis, mitochondrial morphology, and the level of inflammation in the cells was investigated. However, no significant increase in the MILLIPLEX Human Cytokine Panel A of 12 cytokines was observed, indicating that the cytokine storm model is unlikely to be the direct mechanism responsible for cardiomyocyte damage by S-Protein. It was observed that S-Protein alters mitochondrial morphology, and the studied drugs prevent this effect to varying degrees. This illuminates the possible influence of the S-Protein on the energy metabolism of cells and the restoration of metabolic balance by the drugs. Further studies are necessary to understand the mechanism of this phenomenon.

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