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Editorial

Statin Therapy and COVID-19

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COVID-19 is an infection caused by a new coronavirus named sever acute respiratory syndrome (SARS-CoV-2). One of the endocytic route for the virus is via angiotensin converting enzyme (ACE2), which is vastly expressed in the lungs, heart, gastrointestinal tract and kidney, hence disturbing the cardiovascular system and the immune system. The overexpression of ACE2 was documented to improve viral entrance and intracellular replication. Moreover, COVID-19, may also use ACE2 as a receptor to initiate infection, foremost to various complications. It is also more probable to lead to poor consequences in obese patients and those with metabolic syndrome or cardiovascular disease. Due to the fact that viral infection often disturbs the cardiovascular system, causing myocardial infarction, viral myocarditis, tachyarrhythmia and stress cardiomyopathies, which have been recognized as risk factors for severe COVID-19 in various large case analysis from China, Italy, and the United States. It has been proposed that (3-hydroxy-3-methyl-glutaryl-CoA reductase) inhibitors (statins) can decrease the risk of cardiovascular complications among COVID-19 patients. Using of these drugs to treat hyperlipidemia and its pleiotropic effects have been revealed to decrease cytokines in several non-infective situations. Treatment with statin for long-term associates with enhanced outcome in the setting of bacterial pneumonia and influenza.

A randomized controlled trial estimating atorvastatin as a medication for influenza revealed significantly lesser concentrations of inflammatory cytokines using [NCT02056340] as treatment, which may be caused by direct and indirect mechanisms. In addition, statins are tolerated drugs, either by their immunomodulatory effect or by inhibiting cardiovascular damage. This hypothesis should justify consideration for phase III clinical trials. Although these useful effects, statin therapy may have side effects that should be deliberated, such as increased serum glucose levels, creatinine kinase, and liver enzymes, which are already increased in severe COVID-19 infection.

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