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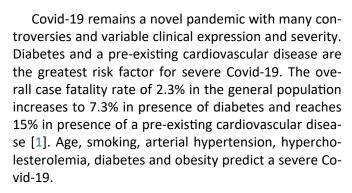
LETTER TO EDITOR

Can Statins Be Beneficial in COVID 19 Patients?

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MyD88 gene seems to be highly induced in SARS-CoV infection. It's hallmark triad activates NFkB pathway, reduces type 1 interferon and increases inflammation [2,3]. Modified LDL cholesterol is absorbed via scatter receptors into cells. It forms crystals that activate NLRP3 inflammasome - caspase-1 activity increasing the release of IL-1b and IL-18 [4]. Oxidized LDL cholesterol binds to Toll-like family receptors which activates inflammation via MyD88 and NFkB pathway. This increases transcription of inflammatory cytokines such as IL-6, IL-12, IL-27 & TNF α [4].

Statins inhibit the MyD88 pathway maintaining normal levels during hypoxia or under stress [2]. The attenuation of NFkB increased the survival of SARS-infected transgenic mice [3]. The ability of statins to maintain MyD88 at normal levels may be protective for Covid-19 patients as the attenuation of NFkB inflammatory pathway reduces IL-6 and other inflammatory cytokines transcription. Furthermore, mortality was lower in COPD and influenza patients on moderate doses of statins compared to patients without statin therapy [5].

In our prospective cohort study, 65 patients hospitalized for severe Covid-19 received Pitavastatin known for few drug interaction, less muscular toxicity and bet-

ter liver tolerability. Mean age was 53.1 ± 18.3 years and 60% were male. At database lock date, 80% of the patients were discharged with a median length of stay of 8 days, five remained in an isolation unit, one in ICU and only two patients were dead.

The ACE receptor seems to have also an important role in this disease. Thus, controlling cardiovascular risk factors tightly and exploring more the pathophysiology of Covid-19 will lead to a better comprehension of disease severity and variability and assure a better management.

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