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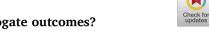
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Letter to the Editor

Endothelial dysfunction and COVID-19: What's the true impact on surrogate outcomes?



We have read with great interest the results of the study performed by Mejia-Renteria et al. [1], which demonstrated a deleterious effect of severe acute respiratory syndrome-coronavirus-2 (SARS-CoV-2) infection on endothelial function of enrolled subjects, further impairing during post-infectious stage. According to a recent meta-analysis, several endothelial biomarkers are significantly elevated in critically ill subjects with SARS-CoV-2 infection compared to non-critically ill patients, suggesting a prognostic role of endothelial dysfunction in coronavirus disease-19 (COVID-19) [2]. However, another prospective observational study performed in the Intensive Care Unit (ICU) setting showed that levels of angiopoietin 2:1 (Ang-2:1 ratio) and soluble tumor necrosis factor receptor 1 (sTNFR-1), markers of endothelial dysfunction, were significantly lower in COVID-19 patients compared to noninfected hospitalized patients in the ICU [3]. Therefore, the authors suggested that endothelial dysfunction might not be characteristic of severe COVID-19 early after ICU admission [3]. Based on the complex pathophysiology of COVID-19, it seems that endothelial dysfunction may not be pivotal for the development of severe disease, since several other pathways, such as chemotaxis, interleukin production and immunothrombosis also play a significant role [4].

Since Mejia-Renteria et al. [1] assessed logarithmic scaled reactive hyperemia index (LnRHI), a non-invasive but also not laboratory marker of endothelial function, we would be really interested to know how many subjects from the group 1 developed critical illness, were admitted to ICU or even died during disease course, and of course if there was a significant difference in LnRHI values compared to those that did not develop severe disease.

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Declaration of Competing Interest

None declared.

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