Short Communication

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Post-covid-19 patients who develop lung fibrosis have lower plasma levels of ifn-î² but higher levels of il-1î± and tgf-î²

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Abstract

Background. SARS-CoV-2 has caused a world-wide pandemic inducing a disease known as coronavirus disease 19 COVID-19, which may be associated to pulmonary fibrosis. A significant number of COVID-19 patients, although negative to the oral-pharyngeal swab, experience prolonged symptoms, herein called post COVID-19 (PC).

Aims and objectives. Based on the fact that the mechanism/s underlying SARS-CoV-2- derived pulmonary fibrosis are still not well defined, the aim of this study was to identify blood biomarkers to predict lung fibrosis-like changes in PC patients

Methods. We used blood samples collected from healthy subjects, anti-SARS-CoV-2 vaccinated (VAX) subjects, and PC patients who were stratified according to the severity of the disease and chest computed tomography (CT) scan data.

Results. We found that C reactive protein (CRP), complement complex C5b-9 and LDH, but not IL-6, were still higher in PC patients, independent of the severity of the disease and lung fibrotic areas. Interestingly, fibrotic PC was characterized by higher plasma levels of IL- 1α, CXCL-10, TGF-β, but not of IFN-β, compared to healthy and VAX groups. In particular, 19 out of 23 (82.6%) severe PC and 8 out of 29 (27.6%) moderate PC patients presented signs of lung fibrosis, associated to lower levels of IFN-β, but higher IL-1α and TGF-β.

Conclusions. Our data suggest that higher IL-1α and TGF-β, and lower plasma levels of IFN- β could predict an increased relative risk (RR = 2.8) of lung fibrosis-like changes in PC patients

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Biography

Chiara Colarusso is currently a Post-Doctoral Researcher in Pharmacology at the Department of Pharmacy, University of Salerno. Her actual scientific interest is on the molecular/cellular mechanisms involved in chronic lung inflammation at the basis of lung diseases, such as COPD andextensively in high impact medical journals, and is an Editorial lung cancer. In particular, she is being focusing on the inflammasome-dependent pathways puzzling from the process of lung cancer establishment up to progression that occurs after therapeutic treatment. In this regard, another goal she is trying to reach is to understand cellular and molecular mechanism/s at the basis of ICI's resistance in lung cancer patients...

