Rapid Response:

”Long CoViD-19" and SARS-CoV-2 reinfections

Dear Editor,

With reference to this interesting article, I think we should also pay attention to another emerging issue of concern, namely that of SARS-CoV-2 reinfections, which are increasingly reported from several Countries, with severe health impacts being observed in some individuals.

In this respect, it would be important to assess whether (and to what extent) "long CoViD-19"-affected patients have developed such condition alongside or secondarily to a SARS-CoV-2 reinfection.

Noteworthy, the genetic "make-up" of the coronaviral strains/isolates recovered from SARS-CoV-2 "reinfected" people is (more or less) diverging from that of the virus strains/isolates originally infecting those individuals, with this arguing in favour of a "SARS-CoV-2 reinfection".

Notwithstanding the above, which appears to be largely plausible, I wonder to what extent we have adequately taken into account the additional possibility that, following a "primary" infection, the viral pathogen could hide itself "somewhere" within the host's body tissues, thereby giving rise to a "mutation process" leading to a virus with a genetic structure different from that of the original viral strain/isolate.

Human immunodeficiency virus (HIV) is a very remarkable example within such context, with the viral strains/isolates primarily entering the human body undergoing a number of mutations, so that the HIV strains/isolates subsequently shed into the surrounding environment and/or transmitted to uninfected hosts will be characterized by a different genetic background.

In the course of HIV infection patients will develop, in their turn, a range of "antibody waves", with the aim of counteracting the various "viral mutants" progressively showing up, with this contributing to the immune deficiency especially seen in Th2-dominant, HIV-infected individuals.

Many question marks are still open regarding the anti-SARS-CoV-2 host's immune response, with "cytokine storm" (otherwise called "cytokine release syndrome"), "macrophage activation syndrome", "Kawasaki-like disease", "antiviral antibody-dependent (immune response) enhancement" and "autoantibody production" (et cetera) being all pieces of an identical "puzzle" encompassing the most severe CoViD-19 phenotypes.

Should viral persistence - and its associated viral genomic mutations - provide an explanatory key to the currently termed "SARS-CoV-2 reinfections", then the hyperactive immune response put in place by the host through consecutive antiviral "antibody waves" could represent, in my opinion, a largely plausible biologic determinant for the severe CoViD-19 forms experienced by some "SARS-CoV-2 reinfected" patients.

As a concluding remark, this is a particularly relevant issue in SARS-CoV-2 infection's pathogenesis, warranting further investigation.

Competing interests: No competing interests

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