Protective immunity after COVID-19 has been questioned: what can we do without SARS-CoV-2-IgG detection?

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Severe acute respiratory syndrome corona-virus 2 (The SARS-CoV-2) induces a severe acute respiratory syndrome that is called named as COVID-19. Clinical manifestations of COVID-19 include diarrhea, pneumonia, lymphopenia, exhausted lymphocytes, and pro-inflammatory cytokine production. Immunology is part of the process of clinical evolution, but there are some questions around immunity-based protection: (1) why How some infected people have only mild symptoms of the disease or are asymptomatic; (2) why delayed and weak antibody responses are associated with severe outcomes; and (3) why the positivity ion molecular tests does not represent protective antibodies IgG. Perhaps T cell responses may be the key to solving those questions. The SARS-CoV-2-specific memory T cells persist emerging in peripheral blood and may be capable of providing effective information about protective immunity. The T cells studies can be helpful into elucidating the pathways for development of vaccines, therapies, and diagnostics for COVID-19 and for filling these immunology knowledge gaps.
Currently, the world is experiencing the novel and highly transmissible coronavirus (SARS-CoV-2) outbreak, which also causes high transmissibility and high mortality [1,2]. The SARS-CoV-2 induces a severe acute respiratory syndrome, termed COVID-19, in which immunology is part of the process of clinical evolution consisting of lung tissue damage induced by an inflammatory response, such as a cytokine storm, macrophages and neutrophils activation [1,2].

During infection, a few studies have presented some information about the immune response during this infection attempting to control the infection, which involves the antibody production and lymphocytes T cell activation, but the information is restricted to those hospitalized patients who were hospitalized because they had the virus and were symptomatic. Over the natural course of the disease in the history of the hospitalized patients who recovered, antibody production was shown to increase increasing after the first weeks of symptoms onset, which is in recovery patients with hospitalization history, with a suggestive positive correlation with disease severity [3,4], while T cells were also activated; it seems that memory phenotype also showed an increase has also risen after 14 days of hospitalization [5,6]. However, there are some questions around immunity-based protection with respect to who does and does not need hospitalization, and the non-hospitalized population is considered as viral hosts by carrying the virus around, and contributing to the spread of the virus spread. Also, the other barrier in this outbreak is related to asymptomatic cases, mainly ion health care professionals in the hospital, which could contribute to increase the increase in the number of the cases. The solution to stopping the viral spread appears to have been be direct to social distancing and massive testing, mainly for antibodies detection. Surprisingly, some part of people who are presented positivity ion results from the
molecular test does not have detectable levels of protective antibodies IgG; furthermore, even more, neutralizing antibodies were lower or not at all present, even though in hospitalized patients [3,4]. This situation raised questions about protective immunity and about the time needed for quarantine. Given that, a few studies have already shown that T cells might be the key to solving this dilemma. Despite the finding that the virus can induce lymphopenia and cause a delay in T cells pathway activation during the first days of infection, after two weeks of symptoms, SARS-CoV-2-specific memory T cells phenotypes (central memory for CD4 and effector memory for CD8 lymphocytes) start to emerging in the peripheral blood. This process is capable of providing useful effective information about protective immunity [6]. The data that are needed to describe about how the memory phenotypes of T cells can differentiate on a follow-up study is not been elucidated yet, and the minimal amount of few information available is restricted to preprinted manuscripts, but it is enough to start a have some discussion about how the immune response should be evaluated. Nowadays, we have some vaccines targeting only T cell activation, thus providing robust memory T cell response, but these studies are still ion the preclinical phase. Actually, we have seen a change in the protective immunity status of viral diseases during vaccination, in which where no antibodies detection does not relate to protective status because memory T cells can be activated and protect people from subsequently reinfection [7,8].

Regarding respiratory infections, it also should be noted that viruses are constantly changing via the induction of viral mutations that can contribute to the viral escape of the host immune system. One of our hypotheses concerning around the novel coronavirus suggest it has its power to reduce B cell activity, and this pathway should be further explored. There is urgent need for solutions addressing around the time needed for quarantine in order to prevent shutting the economy down. There to do not shut down the economy may be have an answer to this problem in cellular response assays, in which the cost is similar compared to neutralizing antibodies tests. Once we can evaluate a small
cells after disease, ("in vitro" assays), this will be enough to guarantee the immunity protection. Lymphocytes T cell assays have as Elispot tests (e.g.), has high specificity and sensitivity and T cell immunity after infection, such as proliferation assays using viral particles as stimulators [9,10] and also by optimizing the assays in Biosafety Level 2 labs. The T cell assays could help to estimate the population’s (hospitalized or not) immunity, hospitalized or not, and will be feasible for developing countries with specialized immunology laboratories. Adding to that, the cellular assays will provide information that is useful for vaccine development to prevent and control this viral disease.

References


