Letter to the editor

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Characterizing severe SARS-CoV-2 pneumonia in young adults: Reflections around the literature

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The infection by the *severe acute respiratory syndrome-related coronavirus 2* (SARS-CoV-2) originated in Wuhan City, China at the end of December 2019 and it turned into a rapidly evolving pandemic. It represents the most serious public health threat of the current century since it affects all spheres of global functioning, to the extent that today its name is being reconsidered at the level of syndemic. The SARS-CoV-2 betacoronavirus generates outbreaks of acute respiratory infection similar to those documented for SARS-CoV that emerged in Hanoi, Vietnam at the beginning of 2003. A considerable proportion of patients with 2019-nCoV disease (COVID-19) evolves into severe forms that may require support in intensive care units with high morbidity and mortality in the short and medium term (1).

The comprehensive approach to SARS-CoV-2 infection requires adjustments regarding viral pneumonia known to date and exhibits very particular characteristics such as the involvement of the olfactory bulb, the effect of silent hypoxemia and the radiological involvement evidenced by tomography, even in mild cases, therefore, it is a particularly novel infection from several points of view (2). Although data is clear regarding the distribution of severity by age groups, the clinical characteristics that differentiate elderly from young adults have not yet been well characterized and understood (3).

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The development of controlled and randomized clinical trials with the best scientific evidence has allowed to establish therapies with a substantial impact on the survival of critical patients, such as the use of steroids and anticoagulants, as well as to suppress the use of rushed therapies that have failed to prove its efficacy such as hydroxychloroquine and some antiretrovirals (4).

Similarity is known in comorbidities of survivor and non-survivor groups of young adults with severe COVID-19 (3).

Lymphocyte counts less than 0.5×10^9 / L, d dimer greater than 21 μg / mL, high-sensitivity cardiac troponin I greater than 15.6 pg / ml, and ultrasensitive C-reactive protein greater than 100 mg / L have been observed to be independent predictors of mortality rate in young adults diagnosed with COVID-19. Individuals with two or more of the above findings were more likely to die; it should be noted that the four predictors had the same effect when analyzed in the other age groups (3).

SARS-CoV-2 infection has taught us that it is better to cite the specific fatality ratio by age groups rather than mention the overall fatality, which turns out to be highly skewed due to the heterogeneity in the distribution of deaths according to this variable (5).

Individuals with clearly defined comorbidities such as those over 60 years, ischemic heart disease, diabetes mellitus, and immunosuppression states are especially vulnerable to disease progression in the form of severe pneumonia, acute respiratory distress syndrome, septic shock, and multi-organ failure; furthermore, low viral control replication, prolonged pro-inflammatory response, excess of interleukin 2, amid with defects in B and T cell function are regarded as responsible for the worst outcomes in older population (6).

The age-related change in innate and adaptive immunity against SARS-CoV-2 infection has not yet been investigated in detail, but its deterioration and dysregulation in older adults can be inferred. The senescence of the interferon type 1 (IFN-1) response accounts for increased influenza viral replication in cell cultures, and the elderly show an altered IFN-1 response to influenza virus vaccination. Several SARS-CoV nonstructural proteins shared by SARS-CoV-2 suppress the IFN-1 response, and this suppression has shown to lead to a poor CD8 + T cell response to viral infection (6).

In the middle of the described scenario, a particularly striking population of critically ill patients emerges; young adults, defined by some studies as those between 18 and 39 years old and represent to approximately 5% of patients admitted to intensive care units. Although several hypotheses have been formulated, the reasons why this minority of young adults become critically ill when infected with SARS-CoV-2 are not yet clear (7).

Firstly, these patients may have been infected with a higher viral load or a more virulent strain of SARS-CoV-2. Secondly, particular environmental conditions; global contamination has recently been singled out, but there is clinical heterogeneity in both contaminated and uncontaminated areas. Alternatively, previous infections with related viruses can be harmful, as reported for dengue, but detailed serological studies are required to investigate this aspect of

immune amplification in COVID-19. Finally, severe COVID-19 in previously healthy children and adults can result from a monogenic predisposition to innate errors in immunity (7).

Currently there are limited data on the epidemiological characteristics, time course, and survival of young adults hospitalized with severe COVID-19. It is also necessary to identify, through higher level studies, the risk factors associated with complications in this population and to characterize those that recover quickly versus those who suffer from severe forms of the disease with prolonged hospitalizations.

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