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COVID-19 infection: Is the outcome time-dependent?

Coronavirus disease 2019 (COVID-19) is an infectious disease that can progress rapidly from asymptomatic to acute respiratory distress syndrome (ARDS), refractory coagulopathy and multiorgan failure [1]. The pathogenesis in human remains unclear and despite several research, there is not still common agreement about the management of the disease.

Currently available data [2] have shown that the immune system plays a vital role in defense against COVID-19 infection. Viremia stimulates the immune function and, in addiction, it leads to prolonged proinflammatory responses as demonstrated by an increase of inflammatory cytokines such as Interleukin 6 and D-Dimer [3]. At the same time, the pathways that cause thrombosis is activated by inflammatory mediators; thrombin can trigger an inflammatory response in endothelial cells, platelets, and smoots muscle cells.

Clinical studies [4,5] have suggested that anticoagulant therapy, heparin, reduces the inflammatory response because thrombosis and inflammation are strictly interconnected.

A recent pathological anatomical study [6], starting on emerging observations that suggest that COVID-19 has clinical features distinct from typical ARDS, has recognized the role of an intense complement activation that could lead to diffuse thrombotic microangiopathy and end organ dysfunction; a complement inhibitor (i.e. a humanized anti-C5 monoclonal antibody, eculizumab) might be considered in severe COVID-19.

In line with these considerations, further treatment against COVID-19 seems to be represented by an other recombinant humanized monoclonal antibody, named tocilizumab, which binds the human IL-6 receptor, inhibiting its signal transduction. Chinese researches [7] have achieved encouraging results in 21 patients with severe COVID-19 pneumonia.

Currently, COVID-19 infection does not have specific antiviral drug treatment, so the treatment of the disease must be focused on check the progression of the inflammatory cascade. The “time factor” seems to be important in both the development and treatment of this disease: it appears necessary to block early the “cytokine storm”.

We feel like stating that the oxygen therapy and the mechanical ventilatory support in critically ill COVID-19 patients are not intended “to cure” but to “take time”, as a bridge therapy while the immune system faces the “cytokine storm”.

Based on these considerations, we can hypothesize that an early diagnosis and an early blockage of inflammatory and thrombotic cascades than at the end-stages, with an immunological treatment and an anticoagulant therapy, are fundamental elements to improve the outcome of patients with COVID-19 infection.

Conflict of interest statement

The authors declare that they have no financial or personal relationships with other people or organisations that could inappropriately influence this work.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.mehy.2020.109902.

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