Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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LETTER TO THE EDITOR

Approach to post-COVID-19 syndrome (PC19S): How long to maintain corticotherapy and heparin after discharge from intensive care?

Abordaje del síndrome post-COVID 19 (PC19S): ¿cuánto tiempo deben mantenerse la corticoterapia y la heparina tras el alta de cuidados intensivos?

Dear Editor:

SARS-CoV-2 is a pro-inflammatory and prothrombogenic virus with a high mutagenic profile, which produces active infection of variable duration in various organs and systems[1] and it has been observed that patients who have already suffered from the disease, especially in its more severe forms such as bilateral pneumonia or respiratory distress, present symptoms and signs of chronic multiorganic involvement. Given that there is a generalised chronic reactive endotheliitis with thrombosis markers (D-dimer, von Willebrand Factor...) elevated in patients after discharge from Intensive care and which persists for weeks or even months,[2,3] the possibility of chronic immunomodulatory treatment (corticotherapy) and anticoagulant treatment (with low molecular weight heparin) has been recommended in the medium term,[4] without being able to specify a specific time frame but in the light of the available evidence it seems prudent for at least 6 months to 1 year, evaluating clinical response every 2 months, especially in those patients who have had a high viral load at the beginning of the disease and who have maintained it for more than 4 weeks, since particularly in these patients, similar conditions to residual pulmonary fibrosis (up to 15%), neurological sequelae and even cases of fulminant myocarditis with a severe prognosis have been described. Immunomodulatory treatment would decrease oxidative stress and fibroblast activator factor production, thus minimizing global endothelial damage and especially at lung level, having already been proven in animal models.[5] Although the virus has already been eliminated from the organism, it can be demonstrated by repeated negative PCR, the pro-inflammatory and prothrombotic state at the endothelial level in the microvasculature remains in some patients due to the formation of immune complexes, generating a chronic reactive endothelitis with multiple vascular affectation, preferably in the lungs (up to 60%) and central nervous system (up to 59–65%)[6] that ends up in the lungs in approximately 2 or 3 months in a picture that mimics a fibrotic interstitial pneumopathy clearly different from classic pulmonary fibrosis,[7] conditioning an autoimmune hyperexcitability of the endothelium that remains after the acute clinical course of the disease and favours a progressive decrease in pulmonary compliance. This results in an urgent need to research into whether the prolong immunomodulatory treatment (corticotherapy) and anticoagulant treatment (low molecular weight heparin) in the long term in patients who have suffered severe SARS-CoV-2 infection will improve the prognosis and decrease the sequelae of this disease.

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