Letter to the Editor

Fulminant myocarditis and COVID-19. Response

Miocarditis fulminante y COVID-19. Respuesta

To the Editor,

First of all, the authors would like to thank Joob et al. for the interest shown in our article. We consider that the questions they raise are highly interesting and deserve further analysis and debate. Unfortunately, the pathophysiology of this virus is still unknown. As described in the article, previous studies have highlighted that patients infected with coronavirus disease 2019 (COVID-19) have high concentrations of interleukin (IL)-1 beta, interferon (IFN)-gamma, IFN-inducible (IP)-10, and monocyte chemotactant protein (MCP)-1. It has been shown that patients with severe illness have higher concentrations of granulocyte colony-stimulating factor (GCSF), IL-10, MCP-1, macrophage inflammatory protein (MIP)-1A, and tumor necrosis factor (TNF) alpha; this might indicate that many of the pathological mechanisms of COVID-19 (such as respiratory damage) seem more related to an exaggerated immune response rather than to direct damage from virus, but in the case of myocarditis this (as in other viruses) has not been confirmed.

Recently, the first case has been reported of biopsy-proven myocardial localization of viral particles with morphology and size typical of coronavirus. The pathology study showed low-grade interstitial and endocardial inflammation. Pathology studies are especially advised for the characterization of acute myocardial injury in COVID-19 patients.

In the reported case, the diagnosis of myocarditis was based upon clinical data, imaging and biomarkers of acute cardiac damage. Regarding the etiology of myocarditis, myocardial biopsy was not performed due to hemodynamic instability, significant coagulopathy, and subsequent improvement in cardiac function. Polymerase chain reaction (PCR) for viruses in the nasopharyngeal swab tested was positive for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and negative for adenovirus and influenza A and B viruses, with a positive epidemiological environment. Therefore, when all this information was gathered together, the assumption of COVID-19 as the etiology of myocarditis was supported. We hope this adds some clarification to the comments by Joob et al.

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