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.

Letters to the Editor

Fulminant myocarditis and COVID-19

Miocarditis fulminante y COVID-19

To the Editor,

We found the article titled "Fulminant myocarditis due to COVID-19" very interesting.[1] Irabien-Ortiz et al. report a COVID-19 patient and note that "the clinical picture was consistent with acute myocarditis, with no initial respiratory symptoms and with rapid clinical progression to cardiogenic shock and need for venoarterial ECMO support."[1] We would like to share ideas on this clinical issue. There are some sporadic reports on this cardiac problem in COVID-19.[2,3] In our country, Thailand, the second country to experience COVID-19,[4] there are still no patients with myocarditis due to COVID-19, although there are many severely ill patients and the infection has been in circulation for 4 months. An important clinical consideration is the pathophysiology of myocarditis in COVID-19. There may be direct viral invasion or the disease may be an immune disease.[2,3] Finally, there is a possibility that the patient reported by Irabien-Ortiz et al. may have had a concurrent medical disorder such as another viral infection that could have induced the myocarditis.

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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.[1] 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)-γ, IFN-inducible protein (IP)-10, and monocyte chemoattractant protein (MCP)-1. It has been shown that patients with severe illness have higher concentrations of granulocyte colony-stimulating factor (GCSF), IP-10, MCP-1, macrophage inflammatory protein (MIP)-1A, and tumor necrosis factor (TNF) alpha[2]; 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.

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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.[3] 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[4] 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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