
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

## COVID-19 and thrombosis: Beyond a casual association

### *COVID-19 y trombosis: más que una asociación*

*Dear Editor:*

Despite various therapeutic schemes used since the onset of the SARS-CoV2 pandemic of COVID-19, mortality remains around 3–5% in the different countries that have reported cases.[1] After the knowledge that the virus enters the cell through the union of its protein S with the receptor for ACE2 (angiotensin converting enzyme type 2)[2] has been speculated with the suspension of certain pharmacological groups that due to their mechanism of action increase the presence of these receptors and therefore could increase the passage of virus into the alveolar cells, this point remaining in controversy. On the other hand, in a recently published retrospective series of cases, a frequent elevation of D-dimer has been observed, which has been related to acute pulmonary thrombosis, which has dramatically worsened the prognosis in this subgroup of patients.[3] It is striking that those patients with a higher D-dimer also show more marked desaturations even without observing pneumonia on CTPA (Computarized Tomography Pulmonary Angiography).

Unlike hemorrhagic viruses (Ebola, Marburg...), SARS-Cov-2 could be a highly prothrombotic virus that causes alterations in the coagulation cascade not well characterized at present that would lead to a progressive elevation of D-dimer in function of the severity and extent of microthrombosis. In turn, this hypothesis could explain that these patients have a clearly worse prognosis since in them, orotracheal intubation would provide oxygen to a lung with no microvascular perfusion due to disseminated microthrombotic disease, which would also only be seen in CTPA in very advanced stages and in which little can be done to reverse this situation.[4]

Gradually a therapeutic scheme is being established that would include hydroxychloroquine and azithromycin[5] (or in other cases lopinavir/ritonavir) in the early stages of moderate disease that does not require treatment in ICU (Intensive Care Unit) but given the analytical indication (elevation of D-dimer) and imaging (thrombosis in CTPA) in many cases, should be evaluated the early inclusion of low molecular weight heparin (LMWH) at doses of at least high-risk prophylaxis in all these patients without thrombopenia <20,000 platelets or acute bleeding and manifesting high D-dimer. Given the paucity of prospective studies, the need for urgent effective management, and the relative safety of these LMWH doses, the HAH (hydroxychloroquine–azithromycin–heparin) regimen could be tested in randomized clinical trials to improve the evolution of the disease in cases of torpid evolution.

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## Acute pericarditis due to COVID-19 infection: An underdiagnosed disease?

### *Pericarditis aguda secundaria a COVID-19: ¿una enfermedad infradiagnosticada?*

*Dear Editor,*

The 11th of March of 2020, the World Health Organization declared a pandemic caused by a novel coronavirus, named Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2). The infection mainly causes respiratory tract symptoms.

Acute pericarditis is the inflammatory condition that affects the sac surrounding the heart, which is most often due to viral infections.[1] Currently, to establish the diagnosis, it is essential the use of ultrasound.[1]

We herein report a case of a healthy 35-year-old woman who presented to the emergency department (ED) with dry cough, anosmia, malaise and low-grade fever. A nasopharyngeal swab for SARS-CoV-2 test was done, being positive. Lung Point-of-Care Ultrasonography (POCUS) was performed, showing a thickened pleural line with prominent B-lines and subpleural consolidations in posterior lower lobes. No pleural effusion was detected. Since she had no comorbidities but had lung abnormalities, she was discharged with hydroxichloroquine 200 mg bid during 7 days (off-label use).[2]

On the 6th day of home isolation, she reported a prolonged pleuritic centrothoracic chest pain that improved sitting forward and worsened with supine position. The physicial exam was unremark-