COVID-19 AND THROMBOSIS: BEYOND A CASUAL ASSOCIATION.
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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, 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.
Bibliography: