LETTER TO THE EDITOR
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Anticoagulation as prophylaxis of severe forms of COVID 19? A perspective

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COVID-19 leads to the involvement of the respiratory tract causing a severe acute respiratory syndrome, SARs-Cov-2[1,2]. This can predispose to thrombotic diseases, both arterial and venous, due to the excess of inflammatory reaction, platelet activation, endothelial dysfunction, and stasis[1,3]. Thrombotic events can be diverse: venous thromboembolism[4,5], pulmonary embolism[2,6], and even disseminated intravascular coagulation[2].

Thinking about the pathophysiological importance of these thrombotic events for the appearance of severe forms, we raised the following question: did anticoagulation have a role in the prevention of thrombotic events in SARS-Cov-2? Would this role be greater in patients with countless patients, who generally progress to more severe forms? Are patients who are already using anticoagulants better protected from these severe forms of this disease? The following is a clinical case that illustrates this hypothesis:

- Patient

Male patient, 66 years old, with diarrhea and prostration initially, later evolving with fever, headache, and dry cough. Comorbidities: obesity, systemic arterial hypertension, Diabetes Mellitus, heart failure, sleep apnea, and atrial fibrillation (AF). Using medications including Eliquis 5 mg twice daily to prevent thromboembolic events secondary to AF.

Due to symptoms and the current context of the pandemic, the patient sought emergency care, where an RT-PCR test was performed for COVID 19, which was positive. As the patient was stable from a respiratory and hemodynamic point of view, without the need for oxygen supplementation, symptomatic treatment was prescribed and guided observation at home. The patient evolved uneventfully with complete clinical improvement 15 days after the onset of symptoms.

DISCUSSION

In this clinical case, the patient had numerous comorbidities, which determine a greater predisposition to severe forms of acute respiratory syndrome[1]. However, it evolved well, without any serious hemodynamic, circulatory, or respiratory repercussions of COVID-19. Was this fact related to the use of anticoagulants?

There are still no reports on the use of anticoagulants for the prophylaxis of severe forms in patients with COVID-19. However, as some studies have verified the association between COVID-19 and thromboembolic events in critically ill patients, this hypothesis can be suggested[4,7].

The pathogenesis of thromboembolism in SARs-Cov-2 results from an exacerbated inflammatory response, cytokine storm, and elevation of mediators such as Von Willebrand factor and tissue factor, generating endothelial, hemostatic activation, and consequently the activation of the coagulation cascade[1,3,6]. There is also an increase in prothrombin time, an increase in fibrinogen degradation products, platelet consumption, and an increase in Dimero-D. In addition to these findings, the fact that in autopsies of patients with COVID-19 there is the presence of microthrombi in the pulmonary microvasculature, suggesting that the hypoxemia presented during the severe form may be associated with dissociation between ventilation and local perfusion[8]. Unlike Severe Acute Respiratory Syndrome, patients present good lung compliance in the initial stage, reinforcing the hypothesis of microthrombi[1,3].

Direct oral anticoagulants are an alternative to vitamin k antagonists (Warfarin) in preventing thrombotic events. Unlike warfarin, which prevents the coagulation process by suppressing the synthesis of vitamin k dependent factors, apixaban enters the coagulation cascade directly inhibiting the activated

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factor X, preventing thrombin activation and the consequent development of the thrombus[9].

CONCLUSION

The related patient had several risk factors for an unfavorable outcome, which surprisingly did not occur. Was this fact related to anticoagulation that, as discussed, could prevent the activation of the presented coagulation cascade? We suggested this possibility and suggested that cross-sectional and longitudinal studies be carried out to test this hypothesis. A practical application if this hypothesis is proven, would be the use of these substances in patients at risk who for some reason would not be able to maintain social isolation.

AUTHORS 'CONTRIBUTION

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