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Editorial

Special Issue "COVID-19 and Thrombosis"

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Since the pandemic began, an association among COVID-19 and venous thromboembolism has been reported, in particular for inpatients.

This association covers several items from pathophysiology to prognosis: from interactions between SARS-CoV-2 and respiratory cells by heparin/heparan sulphate, endothelial dysfunction, and prothrombotic cytokine storm to finding methods to perform early diagnosis of VTE in inpatients with COVID-19 and finding the right drugs to prevent and to treat VTE in patients with COVID-19.

The interaction between SARS-CoV-2, heparan sulphate, and ACE receptors 1 and 2 were described early on, and these interactions were reported as main actors of the following induced hypercoagulable state [1]. Furthermore, additional prothrombotic conditions were found between comorbidities and induced hypomobility for intensive or sub-intensive hospital care.

Endothelial dysfunction due to the respiratory damages and virally induced inflammation is responsible for the abnormal release of vWF [2]. Furthermore, endothelial dysfunctions are also able to induce other prothrombotic action because of platelets' activation and the release of other molecules, such as cadherins [3].

Endothelial dysfunctions remain for a long time due to cytokine storm, and this abnormality may induce a persistent prothrombotic state. Therefore, many other transient risk factors may induce further pathophysiological changes that are associated with a worsening prognosis [4–7].

Moreover, for these types of dysfunctions, the protective role of heparins was testified in inpatients with COVID-19, where it crossed its anti-thrombotic actions and took anti-viral and anti-inflammatory roles [1,8]. For this reason, a really complex clinical debate has been taken place for several months on the right prophylactic or therapeutic dosage of heparins in inpatients with COVID-19 [9–11].

On the other hand, prothrombotic conditions may also trigger thrombotic diseases different from VTE during COVID-19: atherothrombotic diseases such as coronary heart disease increased their incidence during infection by SARS-CoV-2, as well as other thrombotic diseases of small vessels [12].

From a clinical point of view, such as complex scenario associated with the use of antithrombotic drugs at different posologies is also associated with clinical bleedings [13].

Furthermore, as a leitmotif, thrombosis has also been described as the most dangerous complication of the COVID-19 vaccination campaign. VITT was reported for all types of anti-SARS-CoV-2 vaccines, and its pathophysiology and prevention have been debated for a long time [14,15]. However, cerebral vein thrombosis has not been the only type of venous thrombosis detected after vaccination, as reported in a large registry [16]

In summary, we can conclude that after 2 years of this pandemic and several studies on its pathophysiology and clinical thrombotic disease, some viral infections, such as COVID-19, are able to induce an associated life-threatening, pro-thrombotic condition as well as bacterial infection by mechanisms that are associated with prolonged inflammation.

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These experiences may play an important role in the field of prevention when other outbreaks occur.

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