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Editorial

Comprehension of Thrombosis in COVID-19

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EDITORIAL

COVID-19 results in increased clotting abnormalities such as increased D-dimer, prolonged pro thrombin time, and thrombocytopenia, which are thought to lead to thrombotic complications. Consequently, venous and arterial thromboembolism has been reported and the true incidence of thromboembolic disease is unknown, with data from the case series indicating that thrombotic complications of COVID-19 are common. In Wuhan, China, 81% of 81 intensive care patients had higher rates of reported thrombotic complications with 25% reported venous thromboembolism, and 31%reported thrombosis of venous or systemic arteries, including 184 patients with ischemic stroke and myocardial infarction, 19 in India with COVID-19.

Subsequent studies from France and Italy also reported higher rates of thromboembolic incidence in severely ill patients with COVID-19 (17%-22%), despite the presence of immunosuppressant. In this issue, Ali and Spinler review the COVID-19 and thrombosis in a timely and comprehensive manner in relation to its underlying mechanisms. Subsequent studies from France and Italy have also reported high rates of thromboembolic events in critically ill patients with COVID-19 (17%-22%) despite prophylactic anticoagulation. Some authors have placed special emphasis on the potential for their exploitation of the proposed pathophysiological approaches for COVID-19 induced thrombosis as therapeutic targets. They coherently report a series of cases and studies that demonstrate an association between the regulation of the clotting system, elevated inflammatory markers, and poor prognosis.

Acute inflammatory response, hypoxia and direct viral mediating effects in patients with COVID-19 as well as increased ACE2 expression in endothelial cells are thought to contribute to the prothrombotic environment and lead to a higher rate of thrombotic complications. The mechanisms including. Deregulation of the renin-angiotensin-aldosterone system leading to oxidative stress, endothelial dysfunction and activation of von Will brand factor. Deregulation of the immune response involving complement activation, neutrophil extracellular traps and mitogen activated protein kinase pathways.

Vitamin C, which targets oxidative stress damage, and N-acetylcysteine-based synergistic pathways based on the mechanism of action of N-acetylcysteine intended to break down large von Wilfred factor millimeters activated on endothelial damage. In terms of current therapeutics, treatment dose anticoagulation has been suggested in critically ill patients with heparin and Low Molecular Weight (LMW) heparin having theoretical benefits over vitamin K antagonists and direct oral anticoagulants, as they are thought to possess anti-inflammatory properties beneficial in the context of COVID-19 infection this is, however, only supported by observational data. In a large study of 2773 patients hospitalized with COVID-19, 786 receiving treatment dose heparin during their hospital stay showed a significantly improved survival with the mortality benefit being more marked in the 234 patients requiring mechanical ventilation. However, disturbingly there have been reports of heparin resistance in COVID-19 and in the setting of ST Elevation Myocardial Infarction (STEMI) higher doses of heparin are needed to achieve therapeutic activated clotting times. Aside from anticoagulation, a New York case series has demonstrated the therapeutic benefit of tissue plasminogen activator in COVID-19 patients with refractory respiratory failure on mechanical ventilation which also suggests that pulmonary microvascular thrombi may in part drive the pathophysiology of certain phenotypes of acute respiratory distress syndromes in COVID-19.

It is interesting to note that there is now mounting evidence for thromboembolism, particularly arterial as the first manifestation of COVID-19 infection. A case series from New York reported on a high incidence of young patients presenting with large vessel occlusion and ischemic stroke as a first presentation of COVID-19. Similarly, an Italian series has reported increased incidence of acute limb ischemia presentation in COVID-19. Furthermore, in a large series from London of COVID-19 patients presenting with STEMI, the majority of cases acute coronary thrombosis was the first presentation of infection suggesting that COVID-19 can cause acute arterial thrombosis even in the absence of substantial systemic inflammation.

The second wave of COVID-19 highlights the need for further work on novel treatment options based on the mechanisms

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underlying the problem of thrombosis in COVID-19 patients. This suggests that these mechanisms may change with venous or arterial thromboembolism. The mechanistic difference between venous thromboembolism and atrial thrombosis in patients with systemically ill COVID-19 as the first manifestation of COVID-19 is not yet clear. In terms of current therapeutics, we look forward to the results of ongoing trials of anticoagulant in this patient cohort.