COVID-19: Coagulopathy, Risk of Thrombosis, and the Rationale for Anticoagulation

Miesbach W, Makris M.

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Author of the commentary: *Dr Raquel Ferrandis Comes.* Medical Specialist in Anaesthesiology and Reanimation. University and Polytechnic La Fe Hospital, Valencia. Associate Professor Faculty of Medicine, Valencia

This year has seen multiple publications on the coronavirus SARS-CoV2, cause of the COVID-19 pandemic. This study focuses on one of the aspects of this disease, the hypercoagulable state in patients with COVID-19. The risk of thrombotic complications in patients with COVID-19 is higher in those with additional risk factors, such as advance age, male sex, hypertension, cardiovascular morbidity and immobilisation. The risk is also higher in those with pneumonia requiring intensive care unit (ICU) stay, central vein catheters or with increase of hypoxia-inducible factor, or in SARS-CoV2-related infections with increased levels of angiotensin, cytokines, tissue factor and plasminogen activator inhibitor-I. A higher incidence of thrombotic events was described in patients who did not receive thromboprophylaxis, as well as in patients receiving low-molecular-weight heparin (LMWH), which has been associated with poor prognosis in COVID-19 patients. Furthermore, data also suggest that localised pulmonary microvascular thrombosis may play a role in the progressive respiratory failure. However, evidence is limited by small retrospective studies and the true prevalence of thrombosis in patients with COVID-19 is still unknown.

The high risk of thrombosis associated with COVID-19 is demonstrated by the increase d-dimer levels, suggesting increased thrombin production and activation of fibrinolysis. Elevated d-dimer levels are associated with pulmonary embolism in critically ill COVID-19 patients. However, other bleeding events or a significant reduction in other coagulation parameters such as platelets, fibrinogen or antithrombin (which are most frequently associated with disseminated intravascular coagulation) have not been frequently described in patients with COVID-19.

There is therefore a need to identify the increased risk of thrombotic events at an early stage of infection and to initiate thromboprophylaxis with LMWH as soon as possible, not only to prevent thrombotic events, but also because of the anti-inflammatory effect of LMWH that result in reduced IL-6 levels. Nevertheless, since thrombotic events have been reported despite thromboprophylaxis, studies on the appropriate antithrombotic prophylaxis and treatment duration are needed. The authors suggest that risk stratification according to d-dimer values may be an option to individualize treatment, with dose escalation of LMWH on high risk cases; however, given the complexity of the disease, further evidence is necessary.