Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection is associated with increased risk of arterial and venous thrombotic complications. In a US registry of patients with coronavirus disease 2019 (COVID-19), thrombotic complications occurred in 2.6% of 229 non–critically ill hospitalized patients and in 35.3% of 170 hospitalized critically ill patients. The risk of thromboembolism in SARS-CoV-2 infection in nonhospitalized patients is not known.

Although the pathophysiology behind increased thromboembolism is not fully defined, COVID-19 infection is associated with abnormalities in all 3 parts of Virchow’s triad and hence there exists a pathophysiological rationale for an increased risk of VTE.

• First, endothelial dysfunction may develop due to direct viral invasion of endothelial cells via Angiotensin Converting Enzyme-2 (ACE2), or as a result of the subsequent marked inflammatory response and tissue hypoxia.  

• Second, COVID-19 induces a pro-coagulant state with an increase in factors V, VII, VIII and X and von Willebrand factor and a reduction in ADAMTS13 levels. High levels of antiphospholipid antibodies have also been reported, although their clinical significance is uncertain. Furthermore, reduced fibrinolyis resulting from increased plasminogen activator inhibitor 1 has been observed in intensive care unit (ICU) and non-ICU patients. In addition, platelet activation may also increase the risk of VTE.

• Third, immobility and resultant venous stasis is common, especially in more severe COVID-19 disease.

D-Dimers levels are frequently elevated in patients with COVID-19 and are prognostic. High levels may arise as a result of thrombosis or inflammation. Current data do not support the routine use of high D-Dimer levels in isolation to guide decisions regarding investigation and anticoagulation; levels should be assessed within the overall clinical context.

Pulmonary thromboembolic disease should be considered in patients with hypoxaemia disproportionate to X-Ray changes or sudden worsening of blood pressure, heart rate or oxygen requirements.

All patients admitted with COVID-19 should be assessed for, and the majority receive, thromboprophylaxis. Although multiple trials testing interventions to prevent thrombotic complications in COVID-19 are underway, current clinical guidelines have relied on previous studies of VTE prophylaxis in acute non–COVID-19 medical illness. Therapeutic LMWH should be considered for in-patients with Covid-19 disease who are managed on general wards and require supplemental oxygen. Patients with no evidence of VTE or other indication for therapeutic anticoagulation who require high-flow oxygen, CPAP, NIV for severe ventilatory failure or invasive ventilation should receive less than therapeutic dosing. The published evidence would suggest no benefit of intermediate over standard dose thromboprophylaxis in these patients. Bleeding risk should be considered when making decisions regarding intensity of anticoagulation.

There are no specific RCT data to guide the optimal duration of thromboprophylaxis in patients recovering from moderate or severe COVID-19. A number of observational studies have reported low incidences of acute VTE following hospital discharge of 0-0.6% which do not appear to be greater than in non-Covid-19 patients. ACCP does not recommend post discharge thromboprophylaxis. In contrast, the ISTH recommends post discharge thromboprophylaxis with LMWH or a DOAC for all high-risk hospitalized patients with COVID-19 who have a low risk of bleeding. The ISTH suggests a duration of 14 to 30 days for post discharge thromboprophylaxis, although optimal duration remains unclear.

REFERENCE:


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11. BTS Guidance on Venous Thromboembolic Disease in patients with COVID-19V4.0 31 August 2021


