Role of Anticoagulation in COVID-19 Treatment
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ABSTRACT
Coronavirus Disease 2019 (COVID-19) is caused by a novel coronavirus (SARS-CoV2) and is a highly contagious disease that first appeared in Wuhan, Hubei province of China in December 2019. Many nations are battling to control the spread of the infection [1]. In the infected population, although most of the patients have mild symptoms of fever, fatigue and cough, in some severe cases, patients can progress rapidly and develop the acute respiratory distress syndrome, metabolic acidosis, septic shock, and coagulopathy, venous thromboembolism (VTE) and disseminated intravascular coagulation (DIC).

This article focuses on the emerging guidelines and various recommendations of different institutions on how to manage thrombotic risk, coagulopathy and DIC in patients with COVID-19

The data presented below is intended to provide guidance for anticoagulation prophylaxis and treatment in COVID-19 patients and should not supersede clinical judgement.

Keywords: Thromboembolism, coagulopathy, coronavirus

INTRODUCTION
Background
The coronavirus enter cells by binding to angiotensin-converting enzyme 2 (ACE2), which is found mainly on alveolar epithelium and endothelium apart from many other tissues. Severe COVID-19 disease is associated with features of VTE, disseminated intravascular coagulation (DIC) with coagulopathy. The large inflammatory response may be responsible for induction of this pro-thrombotic state. The exact mechanism of venous thromboembolism is unknown and is likely multifactorial attributed to systemic inflammatory response, stasis, and direct endothelium damage from viral injury and ACE 2 binding [2]. Pulmonary thrombosis appears to be common in severe Covid-19 pneumonia. The factors attributing to high rate of pulmonary thrombosis in severe COVID-19 are observed to be intense endothelial inflammation leading to local micro vascular thrombosis > disturbances in Virchow's triad within the lung due to altered pulmonary blood flow > classical DVT to PE transition which may have a minor role. There is emerging strong evidence from the autopsy studies from different countries that the extensive micro vascular thrombosis to be the leading cause of death in COVID-19. It has been shown in many studies that the highly elevated levels of D-Dimer with no other sites of thrombosis to explain the raised D-Dimer levels are more prone to death. A study of 449 confirmed COVID-19 patients observed that anticoagulation decrease the 28 day mortality by 20% in highly raised D-Dimer levels. Another study of 184 ICU patients with proven COVID-19 pneumonia observed a remarkably high 31% incidence of thrombotic complications. The study recommends thrombosis prophylaxis and to consider higher-dose prophylaxis in high-risk population even in the absence of randomized evidence [3].

Conclusion
All hospitalized adults with COVID-19 should receive pharmacologic thromboprophylaxis with LMWH over unfractionated heparin. A caution to keep in mind before starting the prophylaxis is the risk of bleeding outweighing the risk of thrombosis. In the setting of heparin-induced thrombocytopenia, fondaparinux is suggested. Dose adjustment for obesity could also be used per institutional guidance. In patients where anticoagulants are contraindicated or unavailable, use mechanical thromboprophylaxis (e.g. pneumatic compression devices). Combined pharmacologic and mechanical prophylaxis isn't generally recommended [4].

Disclaimer
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REFERENCES
2. https://covidprotocols.org/protocols/hematology/