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Risk assessment and prognostic aspect of coagulopathy in COVID-19

To the Editor

Coronavirus disease 2019 (COVID-19) primarily being considered as a respiratory illness has been showing a highly diverse and anomalous array of symptoms since its origin in December 2019. The death toll has already surpassed 545,481 with more than 11.87 million confirmed cases worldwide [1], while the pathophysiology of COVID-19 is still obscure. At present, concerns are mounting over the increasing reports of blood coagulation accompanied by organ dysfunction among the COVID-19 patients. The disease tends to cause a hyper- and rapid coagulable state allegedly leading to pulmonary embolism and deep vein thrombosis, especially among the severe cases of COVID-19 [2]. However, predisposition to both venous and arterial thromboembolism causing acute pulmonary embolism (PE), deep vein thrombosis (DVT), ischemic stroke, myocardial infarction and systemic arterial embolism has also been reported. So, the experts are claiming that COVID-19 causes an eminent change in coagulation function, which is directly associated with disease severity [3]. Even a study showed that 71.4% of COVID-19 non-survivors meet the diagnostic criteria for disseminated intravascular coagulation (DIC) compared to only 0.6% of the survivors [4].

However, that study also reported an elevated level of D-dimer protein among COVID-19 cases, which is produced as the result of fibrinolysis following a thrombotic event and associated with the risk of Acute Respiratory Distress Syndrome (ARDS) [5]. Besides, D-dimer protein was reported

significantly higher among the severe cases and the patients requiring intensive care compared to those with mild symptoms [6]. A meta-analysis on 1015 cases showed a notable difference in D-dimer level along with prothrombin time between severe and mild cases, but not in case of platelet count (PLT) and activated partial thromboplastin time (aPTT) [3]. Most importantly, the level of D-dimer and other fibrin degradation protein of non-survivor cases significantly differ from survivors [4] and is considered the major cause of mortality. Therefore, the spike in the level of D-dimer protein provides evidence of abnormal coagulation with the prognostic value, which can be used to evaluate the severity and adverse outcome among patients with community-acquired pneumonia as well as COVID-19 [7]. Moreover, the level of D-dimer has been reported to be dependent on the ethnic groups, which may explain the differential racial susceptibility to COVID-19 severity evident across the world [8].

However, the patients' immobilization during treatment, presence of cardiovascular disease and damage of endothelial cells by viral infection/mechanical procedure has been reported to cause a higher incidence of venous thromboembolism among hospitalized subjects [9], which is even more threatening to COVID-19 susceptible individuals with underlying comorbidities, including cardiovascular diseases. Because COVID-19 patients with pre-existing comorbidities have higher risk of developing disease severity leading to hospitalization, which may result in hospital-induced thrombotic complications and vice versa [10].

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So, the differences in coagulopathy, especially D-dimer level among severe and non-severe cases surely urge immediate attention to the current diagnosis and treatment strategy. Besides, both thrombotic complications of COVID-19 and its risk factors need to be addressed to adapt a more effective management strategy.

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Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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