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Letters

Challenges Before Considering Full-Dose Anticoagulation in Noncritically Ill Patients With COVID-19

I read with great interest the recent paper by Stone et al.[1] Among noncritically ill patients hospitalized with COVID-19, therapeutic anticoagulation did not reduce a 30-day composite endpoint; however, it was associated with a reduction in secondary outcomes of all-cause mortality and intubation compared with prophylactic anticoagulation. Major bleeding was observed rarely (0.1 vs 0.4% of the prophylactic and therapeutic groups, respectively; $P = 0.18$). We should pay special attention to the observations indicating that hospitalized patients with COVID-19 display wide physiological and biological heterogeneity.[2] The coagulopathy of COVID-19 seems to be a complex interplay of several biological systems and factors. The study excluded patients who require intensive care unit care.[1] Bleeding in COVID-19 patients remains a real threat, particularly for the critically ill patients requiring intensive care unit care.[3] Understanding the balance between coagulation, fibrinolysis, and hemostasis will help clinicians to develop optimal approaches to thrombosis prophylaxis in COVID-19. Similar to the coagulation factors, fibrinolytic parameters, including plasminogen activator inhibitor (PAI)-1 and tissue plasminogen activator (t-PA), are significantly dysregulated in COVID-19-related coagulopathy.[4] Both t-PA and PAI-1 are elevated among COVID-19 patients, yet extreme elevations of t-PA are commonly observed in critically ill COVID-19 patients with increased mortality.[5]

We should be cautious in recommending full-dose anticoagulation in COVID-19. Coagulopathy in COVID-19 is complex and potentially dynamic. A subset of COVID-19 patients display hyperfibrinolytic state and increased bleeding risk.[5] Novel surrogate biomarkers for poor clinical outcomes in COVID-19 (eg, t-PA) can help us in predicting the course of disease progression.

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