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COVID-19 Associated Coagulopathy in Beta Thalassemia Patients: Letter to the Editor

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Dear Editor

Some evidence has shown that emerging COVID-19 can be the origin of several serious health problems, including the increased possibility of multiple microvascular thrombotic events (1). COVID-19–associated coagulopathy (CAC) may occur due to endotheliopathy, endothelial cell infection, and endotheliitis induced by COVID-19 after inflammatory cell infiltration and endothelial cell apoptosis (1).

The hypercoagulable state is a multifactorial phenomenon, particularly among non-transfusion-dependent thalassemia patients (NTDT) (2). Some causes of hypercoagulability in thalassemia can be attributable to raised platelet activation and aggregation, short platelets survival, enhanced levels of prostacyclin I2 and thromboxane A2, raised level of reactive oxygen species, increased generation of thrombin concentrations, declined levels of protein C and protein S and also cardiac, liver and endocrine dysfunctions (3).

A recent review underscored insufficient data regarding an increased risk of CAC in beta thalassemia patients affected by COVID-19. However, the results indicated a concern about an upcoming rise in mortality and morbidity in beta thalassemia cases with COVID-19 (4). Another study presented four pediatric non-splenectomized red-cell dependent-thalassemia affected by COVID-19 (mean hemoglobin level 6.7 g/dL, mean ferritin level 2524 ng/mL). 75% of cases witnessed the prolongation of activated partial thromboplastin time (APTT) or prothrombin time (PT) without clinical thrombosis. Moreover, 25% of the patients experienced a rise in D-dimer levels, representing less severity in developing a hypercoagulable state, likely to occur secondary to a dysregulation of the coagulation cascade. That study also illustrated partial coagulation-associated complications in these cases (5). Furthermore, a small study (n= 61) recently depicted that the COVID-19 pandemic could increase serum ferritin levels above 1000 ng/mL in 16.6% of patients with beta-thalassemia major (6).

Current evidence has not propounded thalassemia as a significant risk factor for poor clinical outcomes after COVID-19. Health care and treatments should be implemented more cautiously for beta thalassemia patients with COVID-19 due to the large variety of clinical problems related to beta thalassemia, particularly in severe cases (7). Beta thalassemia may change adverse effects associated with COVID-19, such as iron overload and hypercoagulation. Nevertheless, more robust studies are required to elucidate beta thalassemia's role as a paramount underlying disease in exacerbating CAC.

More studies are needed to precisely unveil the intensity of CAC in hospitalized beta thalassemia cases with COVID-19 infection, particularly in splenectomized, elderly, and NTDT patients. Performing coagulation tests needs to be encouraged in this population after contracting COVID-19, including D-dimer, APTT, PT, fibrinogen level, and platelet count.

Conflict of Interest

The authors declare no conflicts of interest.

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Authors' Contribution

The idea was provided by H.D.K. and H.K. The manuscript was drafted by M.N. Critical revisions were performed by H.D.K. and H.K. All authors approved the final version.

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