

# Coagulation Disorder in COVID-19

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## Description

In December 2019, the new Coronavirus-induced severe acute respiratory syndrome (COVID-19) outbreak was first reported. Three months later, the World Health Organization's Director-General declared COVID-19 a global pandemic. A coagulation disorder is frequently seen in COVID-19, according to accumulated evidence, and the incidence is higher in severe cases [1]. Because data on COVID-19 coagulopathy is still scarce, it is necessary to synthesise data from infections caused by similar RNA viruses that frequently cause coagulopathy, such as Ebola virus (filovirus), Lassa virus (arenavirus), and Dengue fever virus (flavivirus) [2].

Uncontrolled virus replication and inflammatory responses are thought to promote vascular damage and coagulopathy in these viral hemorrhagic fevers [3] with 30% to 50% of cases showing hemorrhagic symptoms in Ebola virus infection [4]. On the contrary, despite belonging to the enveloped, single-stranded RNA virus family, coronavirus does not cause hemorrhagic complications.

For example, the coronavirus that caused SARS in 2002 (SARS-CoV-1) was linked to thrombocytopenia (55 percent), thrombocytosis (49 percent), and a prolonged activated Partial Thromboplastin Time (aPTT) (63 percent), but the incidence of bleeding was low [5]. With SARS-CoV-1 infection, 20.5 percent of patients had deep vein thrombosis and 11.4 percent had clinical evidence of pulmonary embolism. SARS-CoV-2, the virus that caused COVID-19, is a sister clade to SARS-CoV-1 and may have a similar ability to cause thrombotic complications. In this regard, Chinese experts stated that in severe cases, patients can develop Acute Respiratory Distress Syndrome (ARDS), which is characterized by coagulation predominant-type coagulopathy [6].

Found that 71.4 percent of non-surviving COVID-19 patients met the criteria for Disseminated Intravascular Coagulation (DIC), but only 0.6 percent of survivors did. Coagulation and fibrinolysis abnormalities in the pulmonary circulation and bronchoalveolar space are likely to be important factors in the pathogenesis of ARDS in COVID-19. This review will speculate on the pathophysiology and clinical implications of this new Coronavirus-associated coagulation disorder.

## Changes of Coagulation-Related Biomarkers

SARS is caused by both SARS-CoV-1 and CoV-2, with CoV-1 being the human coronavirus most closely related to CoV-2. While information on hemostatic features in CoV-2 infection is still being reported, we gathered data on CoV-1 coagulopathy to understand similarities and differences. Examined 94 CoV-2-infected patients' hemostatic parameters and found that Prothrombin Time (PT) activity was lower in the patients compared to healthy controls (81 percent vs. 97 percent; P-001). In D-dimer and Fibrin/Fibrinogen Degradation Products (FDP) testing, the differences were more pronounced (10.36 vs. 0.26 ng/L; P-001, and 33.83 vs. 1.55 mg/L; P-001, respectively). Notably, the rise in D-dimer value was greater in critically ill patients. Conducted research in D-dimer levels greater than 0.5 mg/L were found in 260/560 (46.4 percent) of the 1099 patients. In nonsevere patients, 43 percent had elevated D-dimer levels, whereas in intensive care, the incidence was around 60 percent unit (ICU) patients. A number of studies have found that nonsurvivors have significantly higher D-dimer and FDP levels, as well as longer PT and a PTT when compared to survivors at admission. They reported a link between coagulation disorder and the development of ARDS, a significant complication with poor outcomes, in this context [1]. These findings suggest that coagulopathy can develop in patients with COVID-19, with coagulopathy being more common in critically ill patients. As a result, monitoring D-dimer and PT levels will aid in patient triage and management. The International Society of Thrombosis and Haemostasis (ISTH) published guidelines for the management of coagulopathy in COVID-19, recommending routine hemostatic marker testing and monitoring in all cases.

## Conclusion

The information in COVID-19 about coagulopathy is still evolving, but evidence suggests that thrombotic coagulation disorder is quite common in severe cases. Thrombocytopenia is uncommon in comparison to septic shock, but D-dimer is more sensitive than other coagulation markers and more useful for determining severity. In comparison to the high incidence of thrombotic events, bleeding complications are extremely rare in COVID-19, and thus standard anticoagulant therapy is strongly advised.

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