



Coagulopathy Main Factor Covid 19

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ABSTRACT

Coronavirus infection (COVID-19), an acute infectious disease caused by the SARS-CoV-2 virus, is characterized by activation of the hemostasis system, which in the most severe cases can lead to the development of consumption coagulopathy. Coronavirus disease 2019 (COVID-19) in severe cases leads to a cytokine storm, a systemic inflammatory response, and coagulopathy, which is one of the worst prognostic factors. Coagulopathy in COVID-19 is accompanied by a pronounced increase in the level of D-dimer and fibrin/fibrinogen breakdown products, while the deviation of other indicators (prothrombin time, activated partial thromboplastin time, platelet count) at the onset of the disease is relatively rare. This article will consider the role and intensive care of coagulopathy against the background of coronavirus infection.

Keywords:

coronavirus infection, coagulopathy, anticoagulant therapy, D-dimer, pro-inflammatory cytokines

Introduction. COVID-19 is associated with a wide range of phenotypes ranging from asymptomatic to complicated multiple organ failure and death. The calculated mechanisms for the formation of multiple organ failure are multifaceted, they contain hypercoagulability with the formation of blood clots in both micro- and macrocirculatory channels. The formation of DIC is a serious predictor of death (among patients with a fatal outcome, it occurred in 71.4% of patients and was observed in only 0.6% of surviving patients) [1,4,24].

There is also a significant increase in D-dimer and prothrombin with a parallel decrease in the number of fibrinogen by 10-14 per day in patients with a fatal outcome [5]. This indicates the need to track these results. A significant level of D-dimer (above 1 µg/ml) is expressed as one of the solid independent risk factors for death in this population [6].

The purpose of this work: To determine the incidence, clinical manifestations of

coagulopathic disorders and to identify risk factors for the development of coagulopathic disorders in patients with COVID-19.

Materials and Methods: Analysis of conducted studies and research literature published in scientific publications.

Results: Thrombocytopenia and endotheliopathy that form during coronavirus infection lead to the so-called thromboinflammation, which, as it turned out, is most pronounced in the pulmonary vessels. Scientists from Italy were the first to describe this syndrome as MicroCLOTS (microvascular COVID-19 lung vessel obstructive thromboinflammatory syndrome) - a microvascular obstructive thromboinflammatory syndrome of pulmonary vascular disease. They also noted that this condition has significant differences from the

classic acute respiratory distress syndrome [8,9,14].

Coagulopathy is expressed by increased fibrinogen levels, elevated D-dimer levels, and lesser changes in prothrombin time, aPTT, and platelet count in the early stages of infection. An increase in the level of IL-6 correlates with an increase in the level of fibrinogen. Coagulopathy is associated with the severity of the disease and the resulting thrombosis, and not with its own viral activity. An increased D-dimer at enrollment is associated with an elevated mortality rate, and an increase in D-dimer after hospitalization precedes multiple organ failure and overt DIC [7,22,23].

Conclusion. The choice of treatment regimen for severely ill COVID-19 patients is compounded by ever-changing survey and global episode data. As a result, as before, for all medical professionals treating patients with COVID-19, the priority is to make the most informed decisions. Based on a significant number of studies included in the peer review, we come to these conclusions that:

All patients with coronavirus infection, respectively, are required to undergo examinations for the level of D-dimer, platelets, as well as prothrombin time (PTT) [10,12,15,18].

- Prophylactic anticoagulants, preferably low molecular weight heparin, are recommended for all patients with coronavirus infection, unless there are contraindications, such as acute kidney injury (AKI), where unfractionated heparin is appropriate.[13,16,17]

- The use of anticoagulants at therapeutic doses is likely to be considered only for patients at high risk for coagulopathy, showing symptoms of organ dysfunction caused by microthrombi formation, or with confirmed or strongly suspected large vessel thromboembolism. [19,21,25,26] Identification of patients at significant risk through laboratory testing for coagulopathy includes: measurement of platelets, fibrinogen, fibrinogen breakdown products, D-dimer levels, thromboelastography, and prothrombin time. It should be noted that some medical

institutions prescribe anticoagulants in therapeutic doses to all patients, unless there are direct contraindications.

- The use of acetylsalicylic acid should be considered in cases with elevated troponin and cardiac dysfunction.

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