

Tissue Factor Immunothromobotic Marker for COVID-19 and other Inflammatory

Diseases

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OVERVIEW

Covid-19 causes hypercoagulability due to increased platelet-derived tissue factor (TF)

- Dysfunction in immune-related coagulation can increase morbidity and mortality
- A new assay measures TF expression to identify at-risk hospitalized patients

BACKGROUND

Coronavirus disease 2019 (COVID-19) is caused by the SARS-CoV-2 virus and has infected millions of people around the globe. The cumulative COVID-19 hospitalization rate in the United States is reported to be 170 per 100,000, with about 20% of hospitalized patients suffering thrombosis. The hypercoagulability state in COVID-19 patients is manifested by an increase in platelet activation and platelet-dependent tissue factor (TF) expression in monocytes. Tissue factor is a cell surface protein that triggers blood clotting in normal hemostasis, though its inappropriate stimulation can cause abnormal clotting events including deep venous thrombosis, arterial thrombosis, pulmonary embolism, and intra-catheter thrombosis. Dysfunction in immune-mediated coagulation can lead to increased COVID-19 mortality due to myocardial infarction, cerebral vascular accident, and disseminated intravascular coagulation. Increased platelet activation and platelet-dependent tissue factor (TF) expression in monocytes of COVID patients at the time of hospital admission is predictive of patients' outcomes.

INNOVATION

Researchers at the University of Michigan have developed an assay to measure increased tissue factor expression and activity, identifying patients at risk for general and COVID-19 related thrombosis. Measurement of tissue factor expression at the time of hospital admission can

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Category

Diagnostics Life Sciences

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provide a means for practitioners to anticipate and initiate early treatment of life-threatening thromboses. While the available data confirms that circulating tissue-factor is elevated in Covid-19 patients, this technology measures tissue factor expression and activity in monocytes by using a combination of mass cytometry and enzyme-linked immunosorbent assay techniques. The results of this assay are reproducible, they are not influenced by medication use, and they are readily available. They may therefore fill an important niche for use in newly hospitalized COVID-19 patients. Additionally, those who have recovered from an initial COVID-19 infection are known to have continued increases in monocyte populations and overexpression of TF, suggesting that residual immune dysfunction may predispose patients to immune-related thrombotic events and may justify surveillance.