



Between inflammation and thrombosis: endothelial cells in COVID-19

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Elevated levels of several endothelial markers, including CD31, VEGFR-2, ICAM-1, VCAM-1, E-selectin, P-selectin and vWF, in lung tissue and circulation support an important role of the pulmonary endothelium in local and systemic COVID-19 pathology <https://bit.ly/3eQObIR>

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To the Editor:

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is causing the current coronavirus disease (COVID-19) pandemic [1]. Over recent months, a plethora of novel research articles has been published, dealing with multiple aspects and manifestations of the disease. Increasing evidence points to a central role of endothelial cells in SARS-CoV-2 infection [2–5]. Early studies have already indicated increased expression of vascular and inflammatory factors (such as vascular cell adhesion molecule (VCAM)-1, interleukin (IL)-8 or monocyte-chemoattractant protein (MCP)-1) in COVID-19 lung tissue [2]. Such markers of endothelial dysfunction and altered endothelial cell integrity are important predictors of a poor outcome in SARS-CoV-2 infections [6], and they are associated with pulmonary oedema, intravascular thrombosis and acute respiratory distress syndrome (ARDS). The pulmonary endothelium is crucial for regulation of vascular tone, inflammatory responses, coagulation/fibrinolysis and maintenance of vascular homeostasis and permeability. Disturbances of these tightly regulated processes may directly contribute to morbidity and mortality. However, the exact mechanisms leading to pulmonary vasculopathy in COVID-19 are still unclear. Here, we provide an analysis of several important vascular markers implicated in the inflammatory response (E-selectin, intercellular cell adhesion molecule (ICAM)-1, VCAM-1), maintenance of microvascular integrity (CD31, vascular endothelial growth factor receptor (VEGFR)-2), platelet activation and coagulation (P-selectin, von Willebrand factor (vWF)) in lung tissue and plasma samples of COVID-19 patients.

