

Endothelial dysfunction and inflammation cell signaling triggered by preeclampsia versus COVID-19 in pregnancy

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Objective

To study in vitro alterations in endothelial cells exposed to sera from pregnant women with preeclampsia (PE) and COVID-19 in addition to assessing inflammation cell signaling pathway.

Methods

Sera samples were obtained from pregnant women with COVID-19 infection classified into mild (n=10) or severe (n=9) in addition to normotensive pregnancies as controls (n=10) and patients with PE (n=13). Endothelial cells were obtained from the human dermal microvascular endothelial cell line. Nuclei and deposits of ICAM-1 and Von Willebrand factor (VWF) staining was performed, and micrographs were captured by fluorescent microscopy. The area covered by fluorescent ICAM-1 or VWF labelling was calculated and expressed as the average fold increase compared to controls. The effect of sera samples on the inflammation cell signaling pathways was assessed by the quantification of phospho-P38MAPK.

Results

Both COVID-19 and PE induced an overexpression of ICAM-1 and VWF on endothelial cells although the effect of preeclampsia was less pronounced than the one triggered by severe COVID-19 ($p < 0.05$). Exposure of endothelial cells to the sera of severe COVID-19 and PE had the capacity to activate inflammatory signaling pathways. Immunoblots showed an increase in the degree of phosphorylation of p38MAPK being both severe COVID-19 and PE statistically different from controls ($p < 0.05$).

Conclusion

Both COVID-19 and PE can trigger endothelial damage and induce the activation of inflammation cell signaling pathways in endothelial cells. The effect on cell adhesion and prothrombosis of severe COVID-19 is more pronounced than PE.