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BJOG Exchange

Authors' reply re: Pre-eclampsia-like syndrome induced by severe COVID-19: a prospective observational study

Sir,

Thank you for the opportunity to respond to the letter from Leavitt and colleagues.¹ We would like to thank them for their interest in our study² and their valuable observations. We agree with Leavitt and colleagues that a similar underlying pathophysiology (endothelial damage) may exist in both pre-eclampsia (PE) and COVID-19, which may explain most of their common clinical manifestations.

Other authors have suggested that the signs and symptoms of PE present in some pregnant women with COVID-19 could be a consequence of the placental dysfunction due to intravascular inflammation associated with the infection, leading to a prothrombotic state in the placenta.³ This hypothesis is supported by the higher rates of maternal vascular malperfusion features observed from placentas of women with COVID-19.⁴

Although this hypothesis has biological plausibility, previous studies have shown a correlation between placental histological findings consistent with maternal underperfusion and anti-angiogenic status.⁵ By contrast, in our study we did not find increased soluble fms-like tyrosine kinase-1/placental growth factor values in most of the women with COVID-19 and signs and symptoms of PE. For this reason, we believe that the placental malperfusion due to COVID-19 is unlikely to be the main aetiology of the PE-like syndrome.

Thus, we agree with Leavitt and colleagues that the shared pathophysiology between COVID-19 and PE is probably related to immunothrombosis and decreased alpha-1-antitrypsin (AAT) and that further research is needed to better understand the causes of PE and PE-like syndrome in order to achieve appropriate treatments for these conditions. ■

References

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