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COVID-19 AND PREECLAMPSIA: ENDOTHELIAL DYSFUNCTION AS A COMMON PATHOPHYSIOLOGICAL LINK

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On 11th March 2020, COVID-19 was officially recognized as a global pandemic by the World Health Organization. This disease not only brought the entire world to a standstill but also debilitated the entire healthcare system across the world forcing numerous high-income countries like Italy to a medical, economic, and social collapse.

SARS-CoV-2 has been noted as one of the most contagious viruses affecting an array of organs including lungs, liver, kidneys, heart, etc. Patients with COVID-19 include mainly middle aged and older adults, and those aged 85 years or older stand a higher chance of developing life-threatening symptoms. This also puts pregnant women at risk of contracting the virus due to specific immunological and physiological adaptive remodeling occurring during the gestational period.

Pathogenesis of COVID-19 begins via droplet transmission, then the virus attaches to the angiotensin-converting enzyme (ACE-2) receptors present on the Type-2 pneumocytes. Via endocytosis the virus penetrates these cells and undergoes replication consequently damaging the pneumocytes in this process and causing inflammation (pneumonia).

Numerous recent articles have indicated the presence of ACE-2 receptors in abundance in human reproductive organs like placenta, uterus and maternal-fetal interface during pregnancy making it a potential site of attachment for SARS-CoV-2.Studies have drawn a statistically significant link between pregnant women with COVID-19 and such hypertensive disorders of pregnancy as preeclampsia and eclampsia. The incidence of preeclampsia among pregnant women with COVID-19 increased up to 8.1% in comparison with 4.4%, which shows an almost two-fold increase in the prevalence of preeclampsia.

Clinical findings of COVID-19 (hypertension, mild thrombocytopenia, proteinuria, increased level of liver enzymes, etc.) and COVID-19-induced placental damage have an overlap with clinical presentation of preeclampsia or preeclampsia-like syndrome making it difficult to accurately differentiate between these conditions without specific laboratory evaluation, such as levels of placental growth factor and soluble FMS-like tyrosine kinase-1: however, this evaluation is technically and economically unavailable in the majority of hospitals, and, therefore, is more widely used in research settings than in practical healthcare.

Studies have suggested a phenomenon of developing hypertensive disorders of pregnancy in patients who contracted COVID-19 early on in their pregnancy (during the first trimester), at the time when crucial processes of implantation, placentation and placental development occur. This has been known as COVID-19 modulation of placental ACE-2 expression.

The common pathophysiology between these two pathologies, however, remains the endothelial injury caused by disrupted placentation and SARS-CoV-2 mediated (direct or indirect) placental damage, and overall inflammatory microenvironment characterized by a significant increase in serum and placental levels of pro-inflammatory cytokines and a decrease of anti-inflammatory cytokines, presence of anti-phospholipid antibodies (aPLAs), etc. According to the results of the INTERCOVID multinational cohort study (2021), COVID-19 in pregnancy correlated with crucial increase in severe maternal morbidity and mortality, with mortality increasing 22,3 times (RR 22.3; 95% CI 2.88–172). Also, the risk of preeclampsia among pregnant women with COVID-19 was 1.76 higher than in those without COVID-19 (RR 1.76; 95% CI 1.27–2.43). Obviously, scientific research aimed at further evaluation of placental dysfunction in women with COVID-19 will contribute greatly to mitigating the risk of preeclampsia/eclampsia and, therefore, to decreasing the alarmingly high rate of severe maternal morbidity and mortality worldwide.