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Endothelial to mesenchymal transition: a precursor to post-COVID-19 interstitial pulmonary fibrosis and vascular obliteration?

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Endothelial to mesenchymal transition (EndMT) could lead to post-COVID-19 pulmonary fibrosis and vascular remodelling https://bit.ly/2QqSKxT

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To the Editor:

We read with interest the recent editorial by Huertas *et al.* [1], highlighting the importance of endothelial cell dysfunction in patients with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection. The authors have made some very fascinating insights, and we would like to take this discussion further, especially emphasising the role of endothelial cells in initiating post-infection pulmonary fibrosis and vascular remodelling. The angiotensin-converting enzyme 2 (ACE2) has been suggested as the primary receptor for mediating SARS-CoV-2 entry into the host cells. Apart from ACE2, the other key players that facilitate SARS-CoV-2 entry, includes transmembrane serine protease 2 (TMPRSS2), furin, sialic acid and the extracellular matrix metalloproteinase inducer (CD147) [1]. In their article, the authors provide compelling comprehensions from a study comparing *post mortem* lung tissues from patients who died from coronavirus disease 2019 (COVID-19), acute respiratory distress syndrome due to influenza A (H1N1) infection and those from age-matched, uninfected control lungs [1, 2]. A crucial inference was the connections between the significant increase in ACE2 positive endothelial cells and the substantial change in endothelial cell morphology, disruption of intercellular junctions, cell swelling, and the breakdown of the underlying basement membrane, all indicative of vascular structural modification in tune to the process of endothelial to mesenchymal transition (EndMT) [3]. Considering the implications for post-COVID-19 pulmonary fibrosis and vascular destruction seen in this infectious pathology, we believe that the role of EndMT in disease manifestation could be consequential.