Identification of two specific transcriptomic clusters of COVID-19 acute respiratory distress syndrome patients with different immune profiles and different outcomes

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Transcriptomic clustering of patients with ARDS due to COVID-19 identified different immune profiles and outcomes. This demonstrates heterogeneity among COVID-19 ARDS patients and may help to establish personalised therapies. https://bit.ly/3h61sCj

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Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is the cause of the respiratory illness COVID-19 (coronavirus disease 2019). The virus was first identified in December 2019 in Wuhan, China and has since then spread globally, resulting in the ongoing SARS-CoV-2 pandemic, causing more than 615 million confirmed cases of infection (https://covid19.who.int/). Although the largest proportion of SARS-CoV-2 infections in humans is characterised by a mild course of disease, about 5% to 20% of patients are hospitalised with COVID-19 due to a more severe course of disease, and require admission to the intensive care unit for diffuse lung infiltrates and severe hypoxaemia [1]. This in turn can lead to the development of acute respiratory distress syndrome (ARDS). Multiple and heterogenous factors are known to cause ARDS, which is pathophysiologically characterised by acute diffuse damage to the alveolar–capillary barrier of the lung, resulting in overflooding of the alveolar space as well as severely limited gas exchange [2, 3]. Among the contributing factors to ARDS are surfactant dysfunction along with a reduction of the capacity of alveolar type I and II (ATI and ATII) epithelial cells to absorb excess fluid from the airspaces into the lung interstitium. Normally, fluid is eliminated from the lung interstitium *via* lymphatic capillaries by vectorial ion transport. The hypoxaemia seen in ARDS results from ventilation/perfusion mismatch and increased shunt perfusion, clinically detected by bilateral opacities on chest radiographs, associated with a decrease in lung compliance and increases in venous admixture of insufficiently oxygenated blood and physiological dead space. Also, acute exudative and proliferative phases are often followed by barrier repair and/or by lung fibrosis.