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The Role of Heparanase Activity and Endothelial Damage in COVID-19 Respiratory Illness

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Description

The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) causes the coronavirus disease-2019 (COVID-19) pandemic. Pneumonitis or acute respiratory distress syndrome (ARDS) are common symptoms of severe COVID-19. According to studies, 59% of COVID-19 patients had proteinuria upon hospitalisation, and 22% of non-ventilated patients and 90% of ventilated patients developed acute kidney injury. The function of the endothelial barrier is critical in the regulation of fluid and protein extravasation, particularly in the lungs and kidneys. Several studies have suggested that endothelial cell dysfunction plays an important role in the pathogenesis of COVID-19 complications. Because pulmonary edoema occurs when fluid leaks into the alveoli, endothelial dysfunction is likely to contribute to pulmonary edoema in COVID-19 [1-3]. Furthermore, it has been well established that proteinuria occurs when the glomerular endothelial barrier function is compromised.

The glycocalyx is a thick layer of negatively charged glycosaminoglycans (GAGs) that covers endothelial cells. In the glycocalyx, heparan sulphate (HS) is the most abundant sulfated GAG. Because of its negative charge, HS contributes to the endothelial charge-dependent barrier function. Heparanase (HPSE), the only known mammalian HS-degrading enzyme, disrupts the endothelial glycocalyx, resulting in loss of endothelial barrier function in ARDS and proteinuric kidney diseases. In addition to impairing barrier function, HPSE produces a pro-inflammatory glycocalyx that promotes chemokine, cytokine, and leukocyte binding to the endothelial cell surface. HPSE inhibition and/or deficiency are beneficial in experimental lung and kidney diseases. Notably, heparins and low molecular weight heparins (LMWH), which have been proposed to benefit COVID-19 patients, are potent inhibitors of HPSE activity.

SARS-CoV-2 was discovered in Wuhan, China, in 2019. It has been discovered to be the most pathogenic coronavirus. SARS-CoV-2 is linked to a wide range of respiratory syndromes, from mild airway symptoms to life-threatening viral pneumonia. Endothelial cells make up one-third of all lung cells and are linked to the severity of lung damage in patients. The surface layer of endothelial cells is coated with endothelial glycocalyx. Endothelial glycocalyx interacts with blood, regulating microcirculatory flow. In a variety of critical care conditions, the endothelium plays an important role in the innate immune response. Endothelial cells also play a role in barrier function and inflammation prevention by limiting their interactions with immune cells and platelets. Endothelial dysfunction is a significant contributor to the pathogenesis of organ dysfunction during viral infections [4,5].

Endothelial cells express angiotensin-converting enzyme 2 (ACE2), which has been found in a variety of arterial and venous endothelial cells. SARS-CoV-2 enters target cells and begins infection by binding to ACE2 on the host cell membrane. SARS-CoV-2 has been found in endothelial cells from a variety of organs. Most notably, endothelial cell injury has been confirmed by transmission electron microscopy in blood vessels obtained during autopsies of COVID-19 patients. Endothelial glycocalyx is made up of a variety of glycoproteins, proteoglycans, and glycosaminoglycans. One of these proteoglycans is heparan sulphate, which accounts for approximately 50-90% of the total proteoglycan content of the glycocalyx. Sheddeddases, such as heparanase, are activated as a result of infection. Heparanase cleaves heparan sulphate fragments from the proteoglycan, causing a loss of integrity and, as a result, endothelial dysfunction.

As a result, it is hypothesised that increased heparanase activity is one of the underlying causes of severe COVID-19 manifestations. Indeed, Buijsers and colleagues found that heparanase activity and heparan sulphate levels are significantly higher in the plasma of COVID-19 patients, which correlates with disease severity. Another study found significantly higher heparanase activity and higher levels of heparan sulphate in COVID-19 patients' plasma. Endothelial dysfunction is clearly implicated in the pathophysiology and clinical course of acute respiratory distress syndrome (ARDS). However, the role of heparanase activity and serum levels of heparanase and heparan sulphate in this context is unknown. As a result, the goal of this study was to look into the relationship between heparanase activity and heparan sulphate fragment formation and outcome in COVID-19 patients, as well as to assess the prognostic value of these potential new biomarkers in ARDS.

Conflict of Interest

None.

References

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