COVID - 19 infection: the perspectives on Angiotensin II role

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Abstract

Since who characterized COVID-19 as a pandemic, all scientists and clinicians studied the viral effects on the various organs and tissues of the infected patients. The virus irreversible binding to the ACE2 in the target cell in the lungs is the first step of the viral deleterious pathway to invade the target tissues. As the virus replicates and its number increase, the ACE2 in lung type II alveolar cells become inactive. ACE2 is also located in the arterial and venous endothelial cells and arterial smooth muscle cells in most organs. ACE2 is the enzyme catalyzing the conversion of angiotensin II to angiotensin (1–7).

Keywords: **Potassium, angiotensin (1, 7) , nitric oxide, covid 19**

The manuscript

The Novel Coronavirus 19, was first reported on in Wuhan, China in late December 2019. According to the CDC, the rate of American patients admitted to the ICU with a cardiovascular disease was 20% among US coronavirus patients. The patients admitted to the ICU with pulmonary issues (chronic lung disease, smokers and former smokers) was more than 39% As the CDC statistical analysis below.

Angiotensin-converting enzyme 2

The virus infects the target cell through the binding of the viral spike (s) glycoprotein with the ACE2. A drop in the ACE2 level, increase in the Ang II (Angiotensin II) level and decrease in the Angiotensin (1, 7) level was reported as the protein is degraded in the cell(1).

Angiotensin II

Angiotensin II is reported to increase the sympathetic activity (2) and decrease the potassium level in blood (3, 4), which may have a role in the increase of mortality rate. Therefore, the treatment should contain potassium as a component of the therapeutic course.

Fig 1 : The covid 19 effect on Ang 2 and lungs.

The pulmonary renin-angiotensin system

The paracrine expression of renin-angiotensin-system (RAS) components play an increasingly recognized as an important role in the lung injury. Activation of a local RAS within the pulmonary circulation and lung parenchyma could influence the pathogenesis of lung injury via a number of mechanisms including an increase in vascular permeability, vascular tone and fibroblast activity, and by reducing alveolar epithelial cell survival (5, 6). As the virus irreversibly bind to the pulmonary ACE2 cells, the angiotensin II could be increased (7, 8) and the mean pulmonary arterial pressure and total pulmonary vascular resistance (9). Nicin et al., (2020) reported that an increase in the vasoconstriction in the covid – 19 patients was observed(10).

In addition, treatment with angiotensin (1, 7) may be studied as a possible treatment of covid 19 infection. Angiotensin-(1-7) downregulates the angiotensin II type 1 receptor in vascular smooth muscle cells (7). Angiotensin (1, 7) is a truncated form of Ang II, lacking

phenylalanine in the eighth position. Its functional role in counterbalancing Ang II actions (11).

The paracrine effect on Ang II in lungs

The use of low-level inhaled Nitric oxide could be tested, as a vasodilator, it has a good effect on the lung (12). Inhaled nitric oxide has been reported to improve oxygenation in acute lung injury as it has minimal systemic effects (13). Nitric oxide simultaneously increases PaO₂. In addition to macrophages, a large number of other immune-system cells produce and respond to NO. It also regulates the functional activity, growth and death of many immune and inflammatory cell types including macrophages, T lymphocytes, antigen-presenting cells, mast cells, neutrophils and natural killer cells (14).

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