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Opinion

SARS-CoV-2 Infection: Pathophysiological Changes and Potential Role of Indomethacin as Antiviral and Anti-Inflammatory Agent

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COVID-19 is an emerging coronavirus with global health threat that has huge medical, social and economic impact; proven effective antiviral agent is not yet available. Genomic analysis studies have shown that both SARS-CoV-2 and SARS- CoV belong to the same family which is β-coronavirus. The genomic sequencing showed around 79% similarity between the two viruses [1] and they share the same human cell receptor which is the angiotensin-converting enzyme 2 (ACE2) [2].

The SARS-CoV-2 utilize its envelope spike glycoprotein to enter into a cell through binding to the cellular receptor, ACE2 in infected cells [3].

ACE2 receptors are widely distributed in the tissues of different organs including respiratory tract mainly lower part, gastrointestinal, heart, kidney [4] and vascular endothelium [1]. This distribution explained the clinical features of SARS-CoV-2 infection.

SARS-Cov-2 infection has three district clinical phases: the first one is viremia phase, the second one is the pneumonia phase and the third phase is the sever or critical phase [5]. The third stage occurs in around 20% of infected patients with marked pulmonary involvement [6].

Once the virus reaches lower respiratory tract; it infects alveolar type II cells [7]. It is well documented that the SARS-Cov-2 mainly infects peripheral and sub-pleural parts of the lung. The clinical hypoxia with grand glass appearance in CT chest noted in patients with severe SARS-CoV-2 infection [8] can be explained by the cell death of type II alveolar cells [9] with subsequent certain pathological changes take place like: diffuse alveolar damage with formation of hyaline membranes [10].

On the other aspect, SARS-CoV infection of the cells leads to down regulation of the ACE2 expression witch then leads to worsening of lung failure [11].

This will subsequently will cause renin-angiotensin system (RAS) dysfunction that will affect blood pressure and fluid electrolyte balance and results in more inflammation [12].

Currently; there is no proven effective antiviral agents against COVID-19 infection. Indomethacin (INDO) was found to have a potent antiviral activity against SARS-CoV by inhibiting virus replication and it also has a protective effect on host cell from virus induced damage [13].

As one of the NSAIDs agents, indomethacin has an anti-inflammatory effect which leads to inhibition of synthesis of prostaglandin. This effect achieved by inhibition of cyclooxygenase (COX) enzyme through competing with arachidonic acid substrate for the enzyme active site [14].

Indomethacin as an aerosolized option is showed to have a protective effect against alveolar tissue permeability in an animal module [15].

Taking into consideration the genetic similarity and the similar pathological changes caused by both SARS-CoV and SARS-CoV-2; indomethacin is a potential drug for treating SARS-CoV-2 infection. It has both antiviral and anti-inflammatory effects that works at two different phases of the infection: the viremia and inflammatory phase. This drug should be considered for in vitro studies to assess its ability to be used as an agent to treat SARS -CoV-2 infection.

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