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Pathophysiological and clinical findings of Covid-19 virus infection in respiratory system: A anatomist's view point

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A new corona virus, Covid-19, is expanding, and this world is facing the pandemic.[1,2] There are many patients who have been infected by this virus. While some experience no symptoms, some show upper respiratory infection symptoms either in moderate or in serious symptoms. Thus, the biggest problem is that there are many patients who fall into respiratory dysfunction and die.[3]

Combined binding and activation mechanism, allow the virus to get into host cells. It's "spike" protein binds to angiotensin-converting enzyme 2 (ACE2) of goblet cells (in the nasal), type II pneumocytes (in the lung), and enterocytes (in the intestine). In addition, an enzyme, called Transmembrane protease serine2 (TMPRSS2), helps the activation of Covid-19 virus.[4,5]

Several initial symptoms of the Covid-19 virus infection can be found in the respiratory system. Fever and cough are seen in 60-90 % cases. Common cold symptoms, like nasal discharge, cough, fever, and sore throat can be found. Nasal discharge is caused by goblet cell activation. If goblet cells are disabled, and the mucus is not secreted, this symptom often cannot be seen.[6] If an upper airway disorder occurs, the virus is carried to the whole body that can infect others such as digestive tracts.

Infected type II pneumocytes by Covid-19 virus can cause pneumonia. A relatively early period of Covid-19 can cause interstitial pneumonia in almost half of asymptomatic patients (CT scan examinations). This condition is affected by Covid-19 virus infection in type II pneumocytes. Type II pneumocytes, also called large great alveolar cells, have a very important rules in the respiration. It produces pulmonary surfactants and progenitor/stem cells for type I pneumocytes. When type II pneumocytes become compromised, pulmonary surfactant cannot be secreted, and symptoms of respiratory distress syndrome (RDS) may occur. Furthermore, if type II cells are selectively attacked by the virus, replenishment of type I cells and additional structural recovery of alveolar septa may be difficult. Therefore, a prognosis of life can be adversely affected.[7]

KL-6 is known as a marker that indicates a progress of interstitial pneumonia (pneumonitis), and it is often clinically applied. The KL-6 can be found in type II pneumocytes cytoplasm. The increase of KL-16 reflects the number of type II cells. The rise of KL-6 is not so high with Covid-19 infection pneumonia. In a report by Kurahsima et.al, KL-6 is 163.5 U/ml in asymptomatic and mild cases, 217.3 U/ml in cases with pneumonia, and 211.[6,8] U/ml in severe cases (it is more than 1.000 U/mL with normal interstitial pneumonia). These results seem to reveal that damages of alveolar septa structure in Covid-19 infection are not easy to regenerate.

Although infected patients are treated with various conventional treatments, including use of Extracorporeal Membrane Oxygenation (ECMO) to improve the infection itself, regeneration of alveolar

septa is not easy to develop. In addition, a steroid treatment is suggested to be not effective for this novel pneumonia. This is because this pneumonia is not principally caused by immunoreaction disorder but by selective type II pneumocytes damaged by Covid-19. The rapid progress of respiratory symptoms and dyspnoea symptoms may be influenced by reduction of pulmonary surfactants synthesized and released from Type II cells.[8]

CONFLICT OF INTERESTS

The authors have declared that no competing interests and financial disclosures exist.

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