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A Perspective of Pathological COVID-19

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Opinion Article

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INTRODUCTION

Coronavirus disease 2019 (COVID-19) is a major public health concern that can be fatal, particularly in the elderly. The SARS-CoV-2 virus causes COVID-19 disease. Although there is a lot of information about the clinical disease's mortality, there isn't much information about its pathobiology. Although the cellular responses to this virus are unknown, a likely sequence of events can be postulated based on previous SARS-CoV research. A cellular biology perspective can help frame research questions and explain the clinical course by focusing on the areas of the respiratory tract that are involved. COVID-19 can be divided into three phases that correspond to different clinical stages of the disease based on the cells that are likely infected.

SARS-CoV-2, when inhaled, most likely binds to epithelial cells in the nasal cavity and begins replicating. The main receptor for both SARS-CoV2 and SARS-CoV is ACE2. SARS-CoV *in vitro* data show that the ciliated cells are primary cells infected in the conducting airways. However, this concept may need to be revised because single-cell RNA shows a low level of ACE2 expression in conducting airway cells with no clear cell type preference. The virus is spreading locally, but there is only a limited innate immune response. Nasal swabs can detect the virus at this stage.

Despite having a low viral burden, these people are contagious. The viral RNA RT-PCR value could be used to predict viral load, infectivity, and clinical course. These researches may be able to identify super spreaders. The sample collection procedure would need to be standardized for the RT-PCR cycle number to be useful. Swabs taken from the nose may be more sensitive than swabs taken from the throat. The virus spreads and migrates down the respiratory tract along the conducting airways, triggering a more robust innate immune response. Nasal swabs or sputum should yield the virus (SARS-CoV-2) as well as early markers of the innate immune response. COVID-19 is currently clinically manifest. The level of CXCL10 (or another innate response cytokine) may be predictive of the subsequent clinical course. Viral-infected epithelial cells are a major source of beta and lambda interferon. CXCL10 is an interferon-responsive gene with a high signal-to-noise ratio in the alveolar type II cell response to SARS-CoV and influenza. CXCL10 has also been shown to be effective as a disease marker in SARS. Determining the host's

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innate immune response may improve predictions of the disease's subsequent course and the need for more aggressive monitoring. Around 80% of infected patients will have a mild disease that is mostly limited to the upper and conducting airways. Conservative symptomatic therapy may be used to monitor these patients at home. Unfortunately, approximately 20% of infected patients will progress to stage 3 diseases and develop pulmonary infiltrates, with some developing extremely severe disease. Initial estimates place the fatality rate at around 2%, but this varies significantly with age. Once the prevalence of mild and asymptomatic cases is better defined, the fatality and morbidity rates may be adjusted. The virus has now infected alveolar type II cells in the lung's gas exchange units. Type II cells are more likely to be infected by SARS-CoV and influenza than type I cells. Infected alveolar units are typically found in the peripheral and sub pleural regions. SARS-CoV multiplies within type II cells, releasing a large number of viral particles and causing the cells to die. As the released viral particles infect type II cells in adjacent units, the end result is most likely a self-replicating pulmonary toxin. I believe that areas of the lung will lose the majority of their type II cells, triggering a secondary epithelial regeneration pathway. Type II cells are normally the precursors of type I cells. The murine model of influenza pneumonia has demonstrated this proposed sequence of events.

CONCLUSION

SARS and COVID-19 cause diffuse alveolar damage, which is characterized by fibrin-rich hyaline membranes and a few multinucleated giant cells. Atypical wound healing can result in more severe scarring and fibrosis than other types of ARDS. Recovery will necessitate a robust innate and acquired immune response, as well as epithelial regeneration. Similarly to influenza, I believe that administering epithelial growth factors such as KGF may be harmful and may increase viral load by producing more ACE2 expressing cells. Because of their weakened immune systems and decreased ability to repair damaged epithelium, the elderly are especially vulnerable. The elderly also have lower mucociliary clearance, which may allow the virus to spread more easily to the gas exchange units of the lung.