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MINI REVIEW

COVID-19 and Stress Response

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ABSTRACT

The Coronavirus Disease 2019 has become a global public health emergency. The novel coronavirus is becoming more contagious through continuous evolution, it is difficult to prevent transmission. The patients of COVID-19 suffer from severe pneumonia and complex systemic manifestations. Reducing the damage of COVID-19 to the human body becomes particularly important. In this review, we reviewed the similarity and particularity of viral infectious diseases to understand the pathogenic mechanisms of COVID-19 and to explore optimal therapy for COVID-19.

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- Therapy
- Healthy carriers

INTRODUCTION

The Coronavirus Disease 2019 (COVID-19) has become a global public health emergency with many unprecedented challenges as well as important economic, political, and epidemiological implications [1–6]. The novel coronavirus is becoming more and more contagious through continuous evolution, it is difficult to prevent transmission of COVID-19. The patients of COVID-19 suffer from severe pneumonia and complex systemic manifestations [7–14], particularly in those with comorbidities [10–16]. It becomes particularly important to reduce the damage of COVID-19 to the human body. To evaluate current therapy and to find future optimal therapy for COVID-19, the pathogenic mechanisms of viral infectious disease must be understood, as optimal therapy should be directed primarily against these pathogenic mechanisms. Therefore, we reviewed the similarity and particularity of viral infectious diseases to understand the pathogenic mechanisms of COVID-19 and to explore optimal therapy for COVID-19.

Similarity and particularity of viral infectious diseases

Viruses have no vitality *in vitro*; they only replicate in living cells. Viruses have a specific tissue affinity, different viruses choose different tissue cells to replicate, such as mumps virus replicates in parotid cells, hepatitis virus replicates in liver cells, influenza virus replicates in upper airway cells, and novel coronavirus replicates in alveolar cells.

The viral infection triggers organism immune response to the viruses. Immune cells synthesize antibodies of virus through genetic recombination to capture free virus and destroy the cells virus replicated in leading to cell necrosis and local inflammation. The tissue affinity of viruses results in proprietary symptoms of different viral infection. If the organism immune cells cannot synthesize antibodies of the virus through genetic recombination allowing the virus to replicate, the people become asymptomatic and healthy carriers of the virus.

The viral infection also induces organism stress response meanwhile. The stress response coordinates the functions of various systems of organism to ensure that the body is able to survive dangerous period. In stress response, the locus

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coeruleus sympathetic adrenal medulla system is excited firstly, followed by the excitation of the hypothalamus-pituitary-adrenal cortex axis. Stress response can mobilize and integrate body functions, and inhibit the excessive inflammatory response. When intense stress response exceeds potential of endocrine, endocrine is exhausted. Patients with well potential of endocrine are mild and recover quickly. Patients with poor potential of endocrine are severe and recover slowly. Endocrine failure results in deterioration of the disease, inflammatory storms and multiple system organ failure. Nonspecific symptoms of viral infectious diseases, such as fever, headache, diarrhea, fatigue, etc., are manifestations of disorder and failure of endocrine.

Manifestations of COVID-19

COVID-19 is acute respiratory syndrome spanned from a mild influenza-like illness to life-threatening complications [17-19]. The novel coronavirus uses the ACE2 receptor to facilitate viral entry into the target cells to replicate. Approximately 83% of the ACE2 receptors are expressed on the luminal surface of alveolar epithelial type II cells, other ACE2 receptors are distributed in extra-pulmonary tissues, including heart, kidney, endothelium, and intestine. The viral invasion initiates immune response to destroy the cells that contain viral particles and lead to lung inflammation. Additionally, the multi-organ dysfunction observed in these patients can be attributed to the wide distribution of ACE2 receptors in extra-pulmonary tissues [20]. The pro-inflammatory cytokines released by the CD4 T-cells and cytotoxic granules in CD8 T-cells contributed to the severe immune injury in the patient. Diffuse alveolar immune damage with cellular fibromyxoid exudates and pulmonary edema, desquamation of pneumocytes, lymphocytes infiltration and hyaline membrane formation led to Acute Respiratory Distress Syndrome (ARDS) [21]. The sequelae of pulmonary fibrosis and dyspnea due to lung immune damage.

Older age group (65 years old) is at higher risk of developing severe infection because of higher proportion of established co-morbidities [22]. Children are less prone to develop symptomatic infection and severe disease [23].

The common nonspecific complaints of COVID-19 are fever, cough, and dyspnea, and less frequently gastrointestinal symptoms like diarrhea [24,25]. Furthermore, there are many phenomena not related to inflammation in the lungs occurred during the course of COVID-19, such as, taste alterations and olfactory disturbances, cutaneous manifestations such as erythematous rashes and urticaria, neurological manifestations such as headache, altered conscious state, and dizziness, etc. [26-28]. COVID-19 also associated with the involvement of the cardiovascular cerebrovascular system and autoimmune diseases [28-32]. Patients of COVID-19 usually have sequelae of taste alterations, loss of appetite, weakness and fatigue for a long time after recovery. Those manifestations are difficult to explain in terms of pneumonia. Those manifestations are the result of disorder and failure of endocrine.

Myths and difficulties in the prevention and treatment of COVID-19

The original host of the novel coronavirus is still unclear. Whether the novel coronavirus can coexist with humans depends on whether there are healthy virus carriers in the crowd. There are a large number of asymptomatic carriers in COVID-19 pandemic [33]. The asymptomatic carriers are different from healthy carriers. The asymptomatic carriers should be defined as patients that there are immune responses to the virus in the body but no clinical symptoms, and the healthy carriers should be defined as healthy persons that the virus is replicated continually in the cells but no immune responses to the virus in the body. So far, there are no studies to confirm the existence of healthy carriers of novel coronavirus.

Stress response plays an important role in prognosis of COVID-19. The current therapies of COVID-19 are mainly antiviral therapy and adjuvant supportive care [34]. There are less therapies for the stress response in course of COVID-19. Evidences shown that low dose dexamethasone can reduce the death rate by one-third for patients of COVID-19 who require ventilation [35], and timing of the steroid therapy will prevent ARDS [34]. World Health Organization (WHO) now strongly recommends systemic corticosteroids for the treatment of patients with severe and critical COVID-19 based on moderate certainty evidence.

Traditional Chinese medicine is a medical system different from western medicine. Western medicine treats diseases the pathogen. Traditional Chinese medicine treats diseases according to stress responses. The drug target of traditional Chinese medicine in the treatment of viral diseases is to calm the excessive stress response and maintain the endocrine function. Appropriate use of traditional Chinese medicine will reduce the severe case and death rate, and help patients to recover from sequelae.

CONCLUSION

Viruses only replicate in living cells. The different viruses choose different tissue cells to replicate. Virus infection triggers immune response and stress response. Immune cells synthesize antibodies of virus to capture free virus and destroy the cells the virus replicate in leading to cell necrosis and inflammation. The stress response coordinates the functions of various systems of organism to ensure that the body survives the critical period. Intense stress response depletes the potential of endocrine resulting in deterioration of the disease, inflammatory storms and multiple system organ failure. Coexistence of viruses and humans depends on whether there are healthy virus carriers in the crowd.

The novel coronavirus uses the ACE2 receptor to facilitate viral entry into alveolar cells to replicate. Immune cells produce antibodies to capture free virus and destroy the cells the virus replicate in leading to a respiratory tract disease ranged from mild to severe. Patients with well potential of endocrine are mild and recover quickly. Patients with poor potential of endocrine are severe and recover slowly.

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