Progress in the Pathogenesis of Pulmonary Fibrosis After Viral Pneumonia

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Abstract: Viral pneumonia is the main cause of adult infection and death. The mild symptoms of this kind of disease are self-limiting, but it is easy to cause other serious diseases and lead to death. Through the investigation of relevant pathological literature and imaging literature, we can know that, In our clinical work, many patients with COVID-19 or other viral pneumonia have left fibrous words after improvement "The data shows that the PPF of patients with severe pneumonia caused by COVID-19 infection is 100% when they are discharged from hospital. Some patients will retain corresponding fibrosis changes after suffering from the disease, which will directly affect the pulmonary function and quality of life of patients. However, at present, China is not clear about the pathogenesis of pulmonary fibrosis caused by the disease. This article mainly focuses on pulmonary fibrosis after viral pneumonia inflammation To analyze the progress of research on pathogenesis of lung injury, explore the epidemiological characteristics of pulmonary fibrosis in patients with severe viral infection, and form the pathogenesis of lung injury based on relevant pathological basic research, so as to provide diagnostic ideas and basis for the follow-up treatment of the disease in China.

Keywords: Viral Pneumonia; After Inflammation; Pulmonary Fibrosis; Pathogenesis

Introduction

Viral pneumonia can lead to pulmonary fibrosis, which is a kind of inhibitory disease caused by multiple injury factors. The main characteristics of pulmonary fibrosis are the proliferation and activation of myofibroblasts and the deposition and remodeling of extracellular matrix. Some patients will produce different degrees of pulmonary fibrosis after infection with the disease, which will have a relatively serious impact on the overall disease after treatment. The direct pathogenic relationship between pulmonary fibrosis and virus infection is not clear, so we should continue to study its contents, analyze the characteristics of pulmonary fibrosis after inflammation from the standpoint of pathophysiology, and summarize the relevant pathogenesis of lung injury after virus infection.

1. Pathogenesis of pulmonary fibrosis after viral pneumonia

Inflammatory mediators can regulate pulmonary fibroproliferative reaction by regulating pulmonary fibroblasts, alveolar macrophages, neutrophils, proteases, antiproteases, thromboxanes and cellulose. Inflammatory mediators include pro-inflammatory mediators and anti-inflammatory mediators. The pro-inflammatory mediators TNF-a.I1-13 and IL-6 were released by inflammatory cells such as macrophages and increased within 24 hours after the onset of acute lung injury. The anti-inflammatory mediators such as IL-4, 1L-10 and 1L-13 were continuously increased in the dead, and the pro-inflammatory mediators and pro-inflammatory mediators jointly regulated the pulmonary fibrosis response. The regulation of inflammatory reaction on pulmonary fibrosis The uncontrolled inflammatory reaction is the root cause of ARDS, and also an important mechanism to promote early pulmonary fibrosis in ARDS. At present, the mechanism of early pulmonary fibrosis in ARDS is still unclear. Research shows that the interaction of extracellular matrix metalloproteinase,

anti-protease and inflammatory mediators can promote the proliferation of lung fibroblasts and other interstitial cells, increase the destruction of basement membrane, make the pro-fibrosis effect exceed the anti-fibrosis effect, resulting in increased collagen synthesis, decreased degradation, accelerated collagen deposition, and pulmonary fibrosis.

Pulmonary fibrosis caused by different causes has different pathophysiological mechanisms, including damage and over-repair of alveolar epithelium, activation of myofibroblasts, collagen secretion and extracellular matrix deposition, and TGF-β、 Vascular endothelial growth factor receptor (VEGFR), fibroblast growth factor receptor (FGFR), platelet-derived growth factor receptor (PDGFR) and other related signal pathways are activated.

2. Pathological basis of pulmonary fibrosis after viral infection and inflammation

Generally, some patients can completely subside after being infected with the virus, but some patients still have the problem of pulmonary fibrosis, and explore the relevant pathological mechanism. In the field of medicine, scholars will agree that pulmonary fibrosis after inflammation will take alveolar epithelial injury as the starting point and diffuse alveolar injury as the pathological basis. Through a series of animal model observation and influenza autopsy, it can be found that the occurrence of pulmonary fibrosis will take the alveolar epithelial injury as the starting point, destroy the subcutaneous basement membrane under the uncontrolled inflammatory reaction, continue to activate fibrous cells, and finally lead to the phenomenon of fibrous changes. The lung pathology will appear in the proliferation phase, exudation phase and other periods of DAD, and there will be characteristic changes in different periods.

3. The mechanism of lung injury after virus infection

After suffering from the disease, the patient is affected by the virus to destroy the epithelial and basal membrane of the alveolus. Combined with the uncontrollable inflammatory reaction, the two together constitute the core knowledge of ARDS, leading to the formation of fibrosis in his patients, and even leading to death and lung injury. It occupies a high position in the pathogenesis of pulmonary fibrosis, and the pathogenesis related to lung injury will also be more discussed. It will include immune mediation and the exposure of the virus itself. The virus will form adaptive immunity after five days, so the inherent immune injury will be more important.

3.1 Direct damage of virus

For example, after novel coronavirus infection, it will colonize the epithelial cells, endothelial cells and smooth muscle cells of respiratory organs, and damage the host cells, leading to alveolitis. The damaged lung tissue will release inflammatory mediators, such as interleukin, prostaglandin, epidermal growth factor, etc., and recruit platelets, leukocytes, etc. to support. The leukocytes will clear the pathogens and necrotic cells in the damaged parts, and platelets will gather to repair the damaged parts. The standby fibroblasts in the stroma are transformed into fibroblasts, which proliferate through mitosis and begin to synthesize and secrete a large number of collagen fibers and matrix components to fill the wound tissue defect, creating a foundation for the division and regeneration of tissue cells and covering the injury. On the fibrous reticular scaffold, the new tissue cells continue to divide and grow, and move on the fibrous reticular scaffold to regenerate the damaged tissue. When this process is completed, fibroblasts and extracellular matrix will gradually fade away, leaving only normal tissue, and the repair of injury will be completed. When the condition is serious, the pathogen stimulation persists or the damage repair is abnormal, the lung tissue cell necrosis and alveolar structure destruction are serious, the alveolar epithelium falls off, the alveolar capillaries expand, a large number of inflammatory cells infiltrate, and the alveolar wall widens. Subsequently, type II alveolar epithelial cells and fibroblasts remained active and extracellular matrix was deposited. When this situation continues, it will lead to serious fibrosis of the pulmonary interstitium, thickening of the alveolar and capillary space, destruction of the alveolar structure, and inability to restore normal function. This is the process of pulmonary fibrosis.

3.2 Inherent immune injury

The first line of defense of human body against virus infection is innate immunity, which uses its immune response to eliminate the virus. However, if the immune response is in failure, it will cause lung injury, especially in highly pathogenic virus infection. Inherent immunity will recognize receptors and pathogen related molecular patterns through patterns, which will form a large number of effector molecules, including lipids, type I and III interferon (IFN), pro-inflammatory cytokines and chemokines. Type I IFN can directly activate immune cells and indirectly activate immune response. Excessive infiltration of pro-inflammatory factors leads to uncontrolled tissue damage, while chemokines can recruit more immune cells. The activation of PRRs affects the recruitment of immune cells and the release of pro-inflammatory mediators. Studies have shown that the up-regulation of PRRs results in an increase in the expression activity of downstream nuclear factors and mediates the occurrence of lung injury.

Conclusion

To sum up, lung injury is a kind of disease phenomenon that is extremely easy to occur after viral pneumonia and pulmonary fibrosis is infected by virus. Its disease will be related to the severity of the patient's disease, and the patient will form pulmonary fibrosis after the lung injury is repaired. The regeneration failure of upper alveolar epithelial cells is an important link in its production. Although glucocorticoids have been widely used in clinical medicine, there is no relevant data to prove that glucocorticoids have supportive clinical benefits, and the side effects of their drugs will limit their drug application. It is necessary to take reducing the risk of fibrosis as the benchmark. In different links of the pathogenesis of fibrosis, the selection of appropriate drugs has become the key point of successful treatment of the disease. At this stage, the disease has been controlled, but other related problems will still arise. Therefore, it is important to carry out safe and effective diagnosis and treatment for patients with pulmonary fibrosis after infection. Clinicians should closely follow up relevant patients and carry out comprehensive supervision and care for them.

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Project: Shaanxi Province key research and development plan projects (Project No.: 2020SF-100)

Project name: Liin gene in early diagnosis and targeted therapy of lung cancer