



Progress in Immune Research of Mycoplasma Pneumoniae Infection

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Abstract: Mycoplasma pneumoniae is one of the common pathogens that induce respiratory tract infection with diverse manifestations after infection. In addition to involving nasopharynx, lungs and bronchus, it can also cause a variety of complications, such as myocarditis, nephritis, meningitis, immune hemolytic anemia, etc. Since the continuous development of clinical testing technology, research on Mycoplasma pneumoniae has been gaining momentum, especially the immune mechanism. Therefore, this paper mainly discusses the immune research of Mycoplasma pneumoniae infection.

Keywords: Mycoplasma pneumoniae infection, immune research, pathological changes

Mycoplasma pneumoniae is the pathogen causing Mycoplasma pneumoniae, and its pathological manifestation is interstitial pneumonia. Some patients developed bronchopneumonia, mainly transmitted by droplets, with an incubation period of 2~3 weeks. Most patients have mild or even asymptomatic symptoms. Common respiratory symptoms include sore throat, cough, etc., which can occur all the year round, with a high incidence in autumn and winter[1]. Immune research of Mycoplasma pneumoniae infection can reveal its immune mechanism, so as to take effective prevention measures and reduce the harm and influence of the disease. Therefore, it is necessary to further understand the progress of immune research on Mycoplasma pneumoniae infection.

1. Immune mechanism of Mycoplasma pneumoniae infection

At present, the pathogenesis of Mycoplasma pneumoniae infection has not been clearly defined. In addition to the direct invasion of Mycoplasma pneumoniae and organ damage caused by toxin release, the immune mechanism of Mycoplasma pneumoniae during the pathogenesis has attracted more and more attention in clinical medicine. The symptoms of Mycoplasma pneumoniae infection in infants are not obvious, the symptoms of pneumonia in school-age children are more obvious, and the pathological changes of lung in people with lower immune function are not obvious. In animal experiments, it was found that even when Mycoplasma pneumoniae infection occurred in mice with thymectomy, it was difficult to develop pneumonia, suggesting that immune response plays an important role in the occurrence of Mycoplasma pneumoniae pneumonia[2]. Clinical findings showed that even the inoculation of inactive Mycoplasma could induce pneumonia in mice, but the inflammatory response was lower than that of active Mycoplasma, and the course of disease was relatively short. Therefore, immune response played an important role in the occurrence of Mycoplasma pneumoniae pneumonia.

1.1 Cellular immunity

Epidemiology points out that infants and young children are infected with Mycoplasma pneumoniae in a timely manner, and the symptoms are not obvious, only slight serum antibody response. While in children with recurrent infection, pneumonia symptoms are relatively more obvious, mainly because repeated infection can induce hypersensitivity reaction. At the same time, there were significant differences in the intensity of tuberculin test in patients with Mycoplasma pneumoniae infection, which suggested that different cellular immune responses after Mycoplasma pneumoniae infection would affect the way of lung injury. Research found that after pneumonia Mycoplasma infection, lung developed the rapid growth of the T lymphocyte and B lymphocyte growth speed is relatively slow, and CD4 / CD8 ratio increased significantly, thus the T lymphocyte is the main cell of Mycoplasma pneumoniae pneumonia, suggests cellular immunity plays an important role in the disease happening[3].

Mycoplasma pneumoniae can stimulate lymphocytes and monocytes, leading to increased cytokine expression levels, and there is a certain difference in the changes of different cytokines after infection. For example, both active and inactive Mycoplasma can lead to the release of cytokines, including significantly increased interleukin-6 and tumor necrosis factor- α , but not interleukin-4[4]. In addition, prostaglandin E2 can activate inflammatory cells, stimulate the accumulation of inflammatory cells in alveoli, and produce toxic reactions to alveolar endothelial cells, leading to lung injury.

T cells in peripheral blood can be divided into CD4 and CD8 subsets. CD4 cells can be divided into Th1 and Th2. The

former mainly secretes interleukin-2, interferon γ and tumor necrosis factor β , mainly mediating cellular immunity, while the latter mainly secretes interleukin-4, interleukin-5 and interleukin-6, etc. It mainly mediates humoral immunity[5]. Under normal circumstances, CD4 and CD8 can maintain a balance, but after the occurrence of *Mycoplasma pneumoniae* infection, CD4 cells significantly decreased, while CD8 cells significantly increased, leading to significant changes in CD4/CD8 ratio, and the more serious the disease, the more obvious changes, so it can be seen that *Mycoplasma pneumoniae* infection can lead to Th1/Th2 imbalance.

1.2 Humoral immunity

Antibody response plays an important role in the body's response to *Mycoplasma pneumoniae* infection. After *Mycoplasma pneumoniae* infection, the human body produces various antibodies, including IgM, IgG, IgA and other antibodies, which can be used as an important indicator of clinical diagnosis. Specific IgM usually appears 1 to 2 weeks after the onset of *Mycoplasma pneumoniae* infection, and generally peaks at 3 to 4 weeks and lasts for several months. Many studies have confirmed that the serum IgM level of children infected with *Mycoplasma pneumoniae* is significantly higher than that of healthy children[6]. IgA antibody plays an important role in the local immunity of respiratory tract, which can hinder the adsorption of *Mycoplasma* to respiratory cilia and enhance the phagocytosis of alveolar macrophages. Specific IgG appeared late, but maintained for a long time, can prevent the recurrence of the disease. Elevated levels of serum immune complexes are closely associated with extrapulmonary complications caused by *Mycoplasma pneumoniae* infection, such as meningitis and nephritis.

1.3 Immune evasion

Because *Mycoplasma pneumoniae* has no cell wall, it mainly adheres to ciliated epithelial cells when invading the airway, and binds to host cells through aggregation of P1 and P30 proteins. *Mycoplasma pneumoniae* is adsorbed on epithelial cells, which can not only absorb nutrients, but also avoid the scavenging effect of ciliary movement and avoid cell phagocytosis. At the same time, because *Mycoplasma pneumoniae* and host cell membrane have similar antigen components, so it can avoid immune clearance, resulting in long-term parasitism. Therefore, *Mycoplasma pneumoniae* can survive in cells, which is one of the important mechanisms for it to evade immune surveillance and cause respiratory tract infection.

1.4 Immunosuppression

Since the infection of *Mycoplasma pneumoniae* can induce hypoglobulinemia, neutropenia and decreased resistance to other pathogenic bacteria, it can be seen that *Mycoplasma pneumoniae* can cause immunosuppressive response.

2. Immunological surveillance of *Mycoplasma pneumoniae* infection

Mycoplasma pneumoniae is a kind of microorganism between virus and bacteria, which is mainly transmitted by droplet and mainly induces respiratory tract infection. In recent years, with the increase of the incidence of *Mycoplasma pneumoniae* pneumonia, it has been widely concerned in clinical medicine. *Mycoplasma pneumoniae* infection develops in infants and preschoolers, extrapulmonary symptoms redundant immune factors, with pneumonia *Mycoplasma* antigens and body tissues and organs more common antigen, so easy to form immune complex after infection, combined with antibodies and immune complex may induce arthritis, thrombocytopenia, the symptom such as myocarditis[7]. For patients with negative bacterial and viral infection, *Mycoplasma pneumoniae* infection should be considered as soon as possible, and serum antibody testing should be carried out in time, so as to diagnose and treat it as soon as possible.

Due to the diverse clinical manifestations of *Mycoplasma pneumoniae* infection, it is difficult to rely on symptoms and signs to confirm the diagnosis, so it is necessary to select appropriate test methods. In the past, laboratory culture was mainly used as the gold standard in clinical medicine, but it was time-consuming and sensitive, so there were great limitations in practical application. At present, serum immunoassay is mainly used in clinical screening for *Mycoplasma pneumoniae* infection. The common methods are as follows: ① Complement binding test: It mainly uses glycolipid antigen to determine the antigen titer, but due to its low sensitivity and specificity, its practical application value is not high. ② Indirect hemagglutination test: This method mainly detects serum IgG antibody, which has high sensitivity, but low specificity, so it also has certain limitations. ③ Condensation set test: This method mainly uses condensation set elements for detection, but due to low specificity, it is gradually replaced by other methods. ④ ENZYme-linked immunosorbent assay (ELISA) : This method mainly uses specific antigen and monoclonal antibody for testing, with high sensitivity and specificity, and has been widely applied in clinical practice.

3. Immunotherapy of *Mycoplasma pneumoniae*

Mycoplasma pneumoniae is highly sensitive to antibiotics that act on DNA RNA or those that destroy cell membranes, including macrolides, tetracycline, quinolones, etc. Since the occurrence of *Mycoplasma pneumoniae* pneumonia is closely related to immune response and will lead to the decline of immune function after the onset, immunotherapy has become an important link in the treatment of *Mycoplasma pneumoniae* pneumonia [8].

The immune inhibitors and more immune modulators were adopted, some scholars think that severe pneumonia *Mycoplasma pneumoniae* caused by injury, lung function and cell immunity enhancement has certain relevance, so for patients with severe pneumonia *Mycoplasma pneumoniae* can use steroid hormone therapy, adrenal cortical hormone is to inhibit the action of the immune response. Therefore, there is a certain effect in the treatment of the disease, for patients with acute respiratory distress syndrome, dexamethasone shock treatment can be used. At present, most scholars believe that adrenal corticosteroids can be used to treat patients with acute severe *Mycoplasma pneumoniae* pneumonia, and patients with recurrent pulmonary lesions complicated with atelectasis, pulmonary interstitial fibrosis, and extrapulmonary complications can also be treated with adrenal corticosteroids. For patients with low immune function after *Mycoplasma pneumoniae* infection, if complicated with gamma globulinemia, the appropriate use of gamma globulin treatment can effectively improve the clinical symptoms of patients. At present, there are also studies pointing out that Chinese traditional medicine has certain effects in the treatment of this disease. For example, peach kernel can inhibit cellular immunity, thus reducing lung function injury.

4. Immunological prevention of *Mycoplasma pneumoniae* infection

As can be seen from the above, *Mycoplasma pneumoniae* infection can lead to the generation of specific antibodies, which have a good protective effect on the recurrence of *Mycoplasma pneumoniae* infection. Therefore, the recurrence rate of *Mycoplasma pneumoniae* infection can be reduced through vaccination. In the past, inactivated vaccines or live attenuated vaccines were mainly used for immunization prevention, but the protection was not high in terms of practical application effect. Although oral heat inactivated vaccine has a certain protective immune effect, great progress has not been made, so the research on P1 protein is mainly focused on at present. Currently, there are few clinical studies on P1 protein-related vaccines, and more scholars are still needed to participate in order to promote the development of immune prevention and reduce the incidence of *Mycoplasma pneumoniae* infection.

5. Conclusion

Mycoplasma pneumoniae infection is a common pathogen of lower respiratory tract infection, which has been widely concerned in clinical medicine. Many immunological studies have found that *Mycoplasma pneumoniae* infection can induce a variety of cytokines, but due to the complex role of cytokines, how to find a more accurate serum immune examination method and how to make a more effective vaccine need to be further explored.

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