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Respiratory mycoplasmosis in children

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Abstract: Infectious and inflammatory diseases of the respiratory system caused by Mycoplasma are called respiratory mycoplasmosis (RM). The main pathogen is Mycoplasma pneumoniae. The importance of other mycoplasmas in the course of respiratory infections is still relevant, so the term "respiratory mycoplasmosis" is mainly associated with respiratory infection of M. pneumoniae.

Key words: Pneumonia, atypical pneumonia, respiratory diseases, mycoplasma, respiratory mycoplasmosis, children, IFA, IXLA, Immunoglobulin M, Immunoglobulin G.

INTRODUCTION

Infectious and inflammatory diseases of the respiratory system caused by Mycoplasma are called respiratory mycoplasmosis (RM). The main pathogen is Mycoplasma pneumoniae. The importance of other mycoplasmas in the course of respiratory infections is still relevant, so the term "respiratory mycoplasmosis" is mainly associated with respiratory infection of M. pneumoniae.[1]

In 1944, M. Eaton et al. isolated a pathogen from the sputum of patients with atypical pneumonia, initially called Eaton's agent. For almost 20 years it was considered a virus, and only in 1963 it was identified as Micoplasma pneumonia - a tiny bacterium that occupies an intermediate position between viruses and bacteria. It is similar to viruses in its ability to pass through filters, and to bacteria – in its ability to be cultivated in cell-free media [1, 2]. Currently, about 40 species of the family Mycoplasmataceae are known, but M. pneumoniae plays the main role in respiratory diseases in humans.[2]

M. pneumoniae is gram-negative, contains DNA and RNA, and is an aerobe and facultative anaerobe. In addition, it develops well in tissue cultures, while M. pneumoniae is not able to synthesize the sterols necessary for the formation of lipid layers of the cell membrane. As a result, mycoplasma fulfills the need for cholesterol and other sterols only by recycling them from infected tissues of the human macroorganism. The absence of a cell wall and a number of other metabolic features of M. pneumoniae determine its low survival rate outside the human body. Thus, it is known that mycoplasmas quickly die when heated, irradiated with ultraviolet rays and exposed to disinfectants [3].

Mycoplasmas reproduce extracellularly and intracellularly; their development cycle takes about 6 days. Intracellular localization protects mycoplasmas from antibodies and most antibiotics. In addition, mycoplasmas can multiply for a long time and persist in the macroorganism, change the metabolism of infected cells, and disrupt the normal regulatory mechanisms of stem, immunocompetent and other cells. In addition, mycoplasmas have the ability to have a cytotoxic effect on lymphocytes and thereby suppress the activity of the host's immune system. The ability of mycoplasmas to cause various chromosomal changes, including those affecting the chromosomal apparatus of human embryonic cells, is also known [2, 4].

The source of respiratory mycoplasmosis is a person suffering from mycoplasma infection, and infection occurs only through close contact, which is due to the instability of the pathogen in the environment. Therefore, family foci of infection are typical for M. pneumoniae, but unlike other respiratory infections, mycoplasma infection spreads slowly, over 1–3, sometimes 4 weeks, even within the same family. And the highest incidence rate is observed in organized groups, for example, if we talk about children, in schools. The maximum increase in incidence is observed in the autumn-winter period and March. At the same time, an epidemic surge in incidence is observed every 4–8 years [5]. Infection occurs aerationally, mainly by airborne droplets, but infection by airborne dust and rarely through household contact through hands or household items contaminated with sputum or saliva of a patient has also been described.

It is recognized that M. pneumoniae has a tropism for the mucous membranes of the respiratory tract, especially the bronchi, due to the fact that the surface antigens of the pathogen contain adhesins that provide ligand-receptor binding of M. pneumoniae to epithelial cells of the respiratory tract.

In this case, damage to the cell wall of epithelial cells is accompanied by disruption of intercellular connections, inhibition of mucociliary clearance and ultimately leads to the death of epithelial cells [6].

This is accompanied by dystrophy, destruction and metaplasia of some alveolar epithelial cells, as well as thickening of the interalveolar septa. At the same time, limited infiltrates are observed in the pulmonary interstitium, mainly peribronchial and perivascular, which are represented by lymphocytes, plasma cells, histiocytes, monocytes and single neutrophils [7]. In young children, hyaline membranes may develop. Cases of the development of generalized mycoplasma infection involving the circulatory system, nervous system, joints in the inflammatory process, as well as damage to the skin, mucous membranes and blood cells have been described [8].

The incubation period of the disease ranges from 1 to 4 weeks. The patient becomes infectious to others on average within 7–10 days from the onset of the disease. Susceptibility to the disease is observed in all age groups of the population, but most often acute respiratory infections of mycoplasma etiology occur in children, adolescents and young people [9], and the frequency of mycoplasma respiratory infection increases with the age of the child.

Thus, respiratory mycoplasmosis was thought to be rare in children under 5 years of age [2], but more recent studies have shown that M. pneumoniae is a fairly common cause of hospitalization in children aged 3–4 years [10]. But still, the incidence among children is highest at 5–14 years of age, when M. pneumoniae is detected as the etiological agent of respiratory infections in 20–35%, and in adolescents and people 19–23 years old – in 16–30% of cases [11]

The clinical manifestations of PM are highly variable; it can be characterized by both subclinical and manifest course. Manifest forms of RM in children most often manifest themselves as acute inflammatory changes in the upper respiratory tract, with pharyngitis becoming the leading clinical variant. Rhinitis, sinusitis, otitis media, myringitis (which can be bullous) and laryngitis develop less frequently.[12]

It should be noted that the symptoms of mycoplasma pharyngitis and other mycoplasma lesions of the upper respiratory tract have few specific features, practically no different from similar diseases of other etiologies. The disease begins acutely, with a rise in body temperature to febrile levels and malaise; in some cases, headache and other symptoms of intoxication are noted. There is a sore throat, sore throat, and nasal congestion. Less common are a runny nose, ear pain and manifestations of conjunctivitis. Fever usually resolves within 3–5 days, but low-grade fever may persist for another 1–2 weeks. Catarrhal symptoms of the disease in the vast majority of cases regress within 7–10 days, however, the release of the pathogen from the nasopharyngeal secretion can be observed for a long time - up to several weeks.[13]

Mycoplasma infections of the lower respiratory tract include mycoplasma bronchitis and mycoplasma pneumonia. The more common clinical form of the disease is bronchitis, but with an epidemic rise in incidence, the frequency of mycoplasma pneumonia increases significantly, and during this period, about 40–60% of all pneumonia in school-age children have mycoplasma etiology.

Clinically, the onset of mycoplasma pneumonia resembles mycoplasma infection of the upper respiratory tract, but the fever persists for a longer period at a febrile level. Moreover, despite hyperthermia, the symptoms of intoxication are usually mild, which is one of the few specific signs of mycoplasma pneumonia. In addition, a few days after the onset of the disease, a dry, obsessive or paroxysmal cough appears, which can persist for a long time - from several weeks to several months. In older children and adolescents, the cough gradually becomes productive. Scattered dry and varied moist rales may be heard in the lungs. X-ray examination reveals bilateral foci of inhomogeneous infiltration in the lungs. In some cases (up to 10%), a transient maculopapular rash is noted with mycoplasma pneumonia.

Mycoplasma pneumonia, as a rule, is not severe, characterized by a smooth course and the absence of respiratory failure (or its mild severity). At the same time, children with immunodeficiencies, sickle cell anemia, severe heart or lung disease, as well as patients with Down syndrome have an increased risk of developing complicated forms of mycoplasma pneumonia.[14]

If respiratory mycoplasmosis is suspected, nasopharyngeal smears, sputum, lavage fluid, bronchial washings are used, and for pathological and anatomical examination - smears of tissues and organs.

In pediatrics, cultural methods are rarely used. Their disadvantage is low sensitivity due to the inadequacy of nutrient media, the inability of some mycoplasma strains to grow in the absence of living cells, as well as the duration of cultivation.

At present, such reactions as RIF, RSK and RNGA, which were widely used in previous years to verify mycoplasmosis, have practically lost their relevance.

Laboratory diagnosis of respiratory mycoplasmosis is considered optimal if a combination of methods is used aimed at identifying pathogen antigens in the test materials using ELISA and RAGA (aggregate agglutination reactions) or its genome using PCR [7].

Determination of a specific antigen is carried out in RAGA - diagnostic titer 1:8 or 0.001-0.0001 μg/ml for protein.

The most sensitive methods for detecting antibodies to mycoplasmas include RPGA (diagnostic titer 1:32), ELISA and indirect immunofluorescence reaction (IRIF), in which antibodies of the IgM and IgG classes are detected [10].

In modern conditions, PCR is used to diagnose this infection, the sensitivity and specificity of which is estimated at 92-98%. [15]

The use of PCR is possible to determine mycoplasma in smears during a local inflammatory process. Polymerase chain reaction has a number of advantages over serodiagnosis, but the high cost of this test does not allow its widespread use in practical healthcare.

With an acute onset and severe intoxication, mycoplasmosis is differentiated from influenza. In contrast, intoxication during mycoplasma infection is protracted with a maximum increase after 4-5 days of illness. When the process spreads to the lower respiratory tract, mycoplasmosis is differentiated from MS infection, whooping cough, pneumonic form of psittacosis and acute pneumonia of other etiologies.

Diagnosis of mycoplasma infection is difficult due to the polymorphism of clinical symptoms, therefore, great importance is primarily attached to the results of laboratory diagnostics.

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