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MONITORING AND MEASURES FOR PNEUMONIA IN CHILDREN

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Abstract
Globally, pneumonia is the leading cause of morbidity and mortality in children under 5 years of age. While the majority of pneumonia-related deaths in children are mostly in developing countries, the burden of the disease is large and there are significant health-related costs associated with pneumonia in developed countries. This article discusses the causes of pneumonia in children. Untreated pneumonia is aggravated by the development of pulmonary and extrapulmonary complications.

Keywords Children, pneumonia, infection, lungs, disease, virus, pathogenicity, toxin, respiratory tract, treatment.

INTRODUCTION

Pneumonia is an acute infectious disease of the pulmonary parenchyma, diagnosed by respiratory disorder syndrome or physical findings in the presence of focal or infiltrative changes on the radiograph. The presence of these radiological signs «gold standard», according to WHO, with a high degree of probability indicates the bacterial etiology of the process and makes it possible to exclude from the range of diseases defined as pneumonia most lesions of the lower respiratory tract: bronchitis, including obstructive ones, caused by respiratory viruses and not in need of antibacterial treatment[1].

Relevance: The widespread occurrence of acute pneumonia poses a great danger to children. Pneumonia is the leading single cause of death in children around the world. It kills approximately 1.1 million children under five years of age every year. It is the cause of all deaths of children under five years of age worldwide. Timely correct diagnosis of acute pneumonia in children, assessment of the severity of the disease taking into account concomitant diseases, correct choice of antibacterial therapy allows children to fully recover from pneumonia, reduce complications and mortality from pneumonia[2].

Pneumonia are divided into out-of-hospital and in-hospital. Community-acquired pneumonia occurs in a child under normal conditions, in-hospital pneumonia after 72 hours of hospital stay or within 72 hours after discharge from there. Neonatal pneumonia is also recognized [3].

Problem

Community-acquired pneumonia remains a critical medical problem due to its prevalence and high mortality. Streptococcus pneumoniae is the most common causative agent of community-acquired pneumonia. Along with this, atypical pathogens of pneumonia (Mycoplasma pneumoniae, Legionella sp., Chlamydia pneumonia) play a significant role, which together are responsible for the occurrence

of approximately 40% of cases of community-acquired pneumonia, as the main or co-pathogens, while mortality can increase and reach 25%. Laboratory methods for identifying atypical flora cannot be called routine and publicly available, pathogens are not detected during Gram staining, and the cultivation of these microbes using conventional microbiological methods is difficult. There are no significant differences in the clinical and radiographic manifestations of infections caused by typical and atypical flora. Therefore, empirical therapy for community-acquired pneumonia should be chosen taking into account the need to suppress both typical and atypical flora.

MAIN PART

It is important to distinguish between «typical» forms with clear, homogeneous appearance, focus or infiltrate on the radiograph and «atypical» with non-homogeneous, borderless changes. The severity of pneumonia is due to pulmonary heart failure, toxicosis and the presence of complications (pleurisy, pulmonary destruction, infectious-toxic shock). With adequate treatment, most uncomplicated pneumonia resolves in 2-4 weeks, complicated in 1-2 months; a prolonged course is diagnosed in the absence of reverse dynamics in the period from 1.5 to 6 months. Subject to hospitalization are: The child's age is less than 2 months, regardless of the severity and prevalence of the process, age is up to 3 years for the lobar nature of lung damage, age is up to 5 years for damage to more than one lobe of the lung, Leukopenia < 6 thousand, leukocytosis > 20 thousand, atelectases, children with severe encephalopathy of any origin, children of the first year of life with intrauterine infections, children with congenital malformations, especially hearts, children with concomitant bronchial asthma, diabetes mellitus, diseases of CCC, kidneys, oncohematology, children from poor social conditions, lack of guaranteed implementation of treatment measures at home. Pneumonia is diagnosed auscultately and percutaneously. Signs of pneumonia: fever, shortness of breath, cough, wheezing in the lungs. Temperature above 38.0°C for more than 3 days, shortness of breath in the absence of signs of bronchial obstruction (>60/min in children under 2 months, >50 aged 2 - 12 months and >40 in children 1 - 5 years), asymmetry of wet wheezing [4].

Hemograms for diagnosing pneumonia are not as significant as is commonly believed; clinical signs have higher diagnostic effectiveness. Leukocytosis below 15x109/l is observed in the first days of illness in 40% of patients with cocca and 96% with SARS, in fact, as often as with bronchitis. And only numbers above 15x109/l make it possible to exclude the viral etiology of damage to the lower respiratory tract with a moderate probability, since such numbers are also possible in bronchitis (RSviral in children aged 2-3 months). CRP levels >30 mg/l and PCT >2 ng/ml are more reliable for diagnosing pneumonia. Levels of leukocytosis above 15x109/l and procalcitonin (PCT) above 2 ng/ml exclude atypical pneumonia, however, at lower levels the differences are almost completely smoothed out[5].

SARS differs little in terms of marker levels from ARVI and bronchitis. In pneumonia in children of the first half of the year caused by C. trachomatis, very high leukocytosis (30-40*109/l) and eosinophilia of more than 5% are often found%. In non-hospital pneumonia. Atypical forms caused by Chlamidia trachomatis are common at 1 - 6 months of age. In more than half of the patients, typical pneumonia is associated with food aspiration, cystic fibrosis, primary immunodeficiency, their pathogens gram-negative intestinal flora, and staphylococci. SARS with the identified pathogen occurs in 7–20% of community-acquired pneumonia[6].

Symptoms of respiratory tract damage: Cough from dry, hysterical to productive with light sputum. Cough – is the most common symptom of damage to the respiratory tract. In patients with mycoplasma infection, cough is always present, but among coughers only 3–10% of patients with pneumonia.

Dyspnoea – is a rather rare symptom; if it occurs, it is poorly expressed.

Fever – the characteristic symptom of mycoplasma

infection – does not reach a high degree of severity. Basically there is no correspondence between fever and radiographic images and blood tests. Symptoms of pharyngitis in 6–59%. Rhinorrhea in 2–35%. Ear pain (myringitis) in 5%. Asymptomatic sinusitis. Physical examination reveals unexpressed wheezing (dry or wet finely bubbly); percutaneous changes are most often not detected. Extrapulmonary manifestations of mycoplasma infection: Hemolysis accompanied by increased titers of cold agglutinins, catarrhal pancreatitis, catarrhal meningitis, meningoencephalitis, neuropathy, cerebral ataxia, maculo–papular skin lesions (an association with Stevens–Johnson syndrome is described), myocarditis (not often), glomerulonephritis (not often), myalgia, arthralgia (without a picture of true arthritis)[7].

Objective examination methods: X-ray examination most often reveals an increase in the pulmonary pattern characteristic of peribronchial infiltration, but there may be focal infiltrates, discoid atelectases, enlarged lymph nodes of the lung root, pleurisy.

Laboratory findings: haemolytic anaemia with increased cold agglutinin titres and reticulocytosis. Leukocytosis is not noted. Thrombocytosis is possible as a response to anemia.

Immunological diagnosis: determination of antimycoplasma antibody titers (IgM, IgG). Positive result: initial increase in antibody titers >=1:32 or 4–x multiple increase in dynamics. The appearance of antibodies is observed by 7– 9 days, and a maximum of – by 3–4 weeks of the disease. Determination of antigens (maximum reliable results within one week of the onset of the disease).

The polymerase chain reaction is based on the determination of specific DNA from the mycoplasma of pneumonia. Sensitivity of the – method 93%, specificity – 98%. Pneumonia caused by pneumococci and Haemophilus influenzae type b occur in 10% of children; usually these are children who fell ill as a result of contact with a patient with acute respiratory infections. In children 6 months - 6 years old, the most common causative agent of pneumonia is pneumococcus; at the age of 7-15 years, the main bacterial causative agent of typical pneumonia is pneumococcus. The selection of antibiotics for the treatment of pneumonia is optimal when deciphering its etiology, but express methods are not always reliable and accessible.

If there is no certainty about etiology, a drug or combination of two drugs with a wider range may be used. Indications for replacement of the drug are the absence of a clinical effect for 36-48 hours for mild and 72 hours for severe pneumonia, as well as the development of side effects. For complicated pneumonia, treatment begins with parenteral drugs, replacing them with oral ones when an effect occurs (stepped method)[8].

For atypical pneumonia, macrolides and azithromycin are the drugs of choice. Since they also act on coccus flora, these agents can be used in individuals allergic to b-lactams, but their widespread use is undesirable due to their stimulation of the drug resistance of the flora.

The effectiveness of treatment is assessed after 24, 36 and 48 hours of treatment. The full effect is recorded when the temperature drops below 38.0°C (without antipyretics) and the general condition improves, appetite appears, and the radiological picture may improve or remain the same. This indicates the susceptibility of the pathogen to the product, therefore treatment with this medicinal product should be continued. A partial effect is recorded with an improvement in general condition and appetite, as well as the absence of negative dynamics in the lesion, but while maintaining febrile temperature, this picture is observed with a purulent focus (destruction) or an immunopathological process (metapneumonic pleurisy). In this case, the antibiotic is not changed; the full effect occurs later - when the abscess is emptied or anti-inflammatory drugs are prescribed. If the patient maintains a febrile temperature, lung infiltration or general disorders increases, it is generally accepted that there is no effect; in these cases, an immediate change of antibiotic is required.

CONCLUSION

If the effect of antibiotics occurs quickly, other types of therapy are not needed. Antipyretics for pneumonia are not prescribed, as this may make it difficult to assess the effectiveness of treatment. Ventilation of the room is mandatory. Before the effect occurs, bed rest, with fast reverse dynamics, the child is transferred to semi-bed rest, and from the 6th-10th day to general rest. Tempering can be resumed after 10-14 days, but heavy physical activity (sports) is permissible after 6 weeks, for mild and 12 weeks after complicated pneumonia. During this time, pulmonary blood flow is restored. The appetite decreased in the first days is quickly restored, which makes the prescription of vitamins unnecessary. Physiotherapeutic procedures for the chest (ionophoresis, microwave, etc.), including during the repair period, are ineffective.

With early detection and timely hospitalization in hospitals of sick children with acute pneumonia in pediatric areas by pediatricians, it reduces mortality and prescribes adequate therapy.

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