Purulent Inflammatory Diseases of Lungs and Pleura in Children

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Abstract: This article describes in detail about the most common lung diseases in children, their etymology, clinical condition, causes and treatment methods. Moreover, this article is about Purulent inflammatory diseases of lungs and pleura in children.

Keywords: lungs, etymology of diseases, clinical condition, vaccine, COVID-19, protective equipment, mental stress, etc.

Several factors are causing the increase in lung diseases in children in Uzbekistan. First of all, the child does not fully follow the rules of personal hygiene and does not use protective equipment properly when there is an increase in airborne viral infections. Failure of parents to properly treat their child at home, sending them to kindergartens and schools even if they are sick, infecting other healthy children, not timely eliminating foci of chronic infection in the child, not feeding them properly, the child's Mental stress, lack of exercise, low mobility, sitting in closed rooms for a long time, and not being vaccinated during viral disease epidemics are one of the reasons for the increase in morbidity. To prevent this situation, parents should teach their child the rules of personal hygiene provide them with protective equipment and teach them to use them. It is necessary to feed properly, exercise, increase exercise and timely vaccination. First of all, parents should not send children with respiratory diseases to preschools and schools, because even if they are mildly infected, they are contagious. In such cases, the child must protect not only himself, but also the people around him and his peers from the disease. Scientific research shows that children are 17% adenovirus carriers and 18% mycoplasma carriers. During the period of illness, the child should drink more fluids, eat well, wear a permanent protective mask, follow the rules of hygiene, wash hands with soap or disinfect hands regularly with antiseptic agents, avoid using shared dishes, when coughing or He should cover his mouth and nose with a napkin or his elbow when he is sick, and he should not go to crowded places. Nowadays, parents try to protect their child from possible diseases by regulating their health before the birth of a child, before this important step. Nevertheless, a child can get many diseases from parents, and first of all, these are diseases that are passed on from the mother. Hereditary, infectious, viral and endocrine diseases can be transmitted to the child from the mother. For example, let's take bronchial asthma from pulmonological diseases. If the mother has bronchial asthma, the probability of transmission to the child is 40 percent. If both father and mother have this disease, the probability of the child getting sick is 80 percent. This is often observed in families with a high tendency to allergic diseases. Also, cystic fibrosis is a hereditary disease,
and in 25% of cases, a child is born to a parent who is a carrier of this disease gene. But it should not be forgotten that the most important factor in a child’s free breathing is proper care.

Pediatric interstitial lung diseases (ILD) are a heterogeneous group of diseases that primarily affect the interstitium, pulmonary capillaries, alveoli, and perialveolar tissue. The diseases are characterized by bilateral dissemination and progressive respiratory failure. The ILD group includes forms of the disease of various etiologies: alveolitis (exogenous allergic, toxic, fibrosing); granulomatosis (sarcoidosis, disseminated tuberculosis, etc.), dissemination of a malignant nature (lung carcinomatosis, etc.); rare forms of dissemination in the lungs (pulmonary hemosiderosis, Goodpasture’s syndrome, alveolar proteinosis, pulmonary angiomatosis, etc.); interstitial fibrosis of the lungs in systemic diseases (collagenosis, cardiogenic pneumosclerosis in chronic hepatitis, etc.). The initial pathogenic impulse triggers pathological immune responses involving various types of cells that produce proteases and oxidants that damage the interstitial and parenchymal structures of the lung tissue. Stereotyped changes in the pulmonary interstitium develop in the form of inflammatory infiltration of varying severity, productive alveolitis, and subsequently fibrosis forms (the “honeycomb lung” picture). It is believed that there is a genetic predisposition to the development of diffuse lung diseases due to excessive fibrosis formation in response to nonspecific damage to the pulmonary epithelium.

In children, respiratory symptoms may be vague and for a long time be regarded as manifestations of other diseases. The disease begins gradually. Clinical symptoms may be completely absent in the presence of radiological symptoms. Respiratory failure (RF) plays a decisive role in the clinical picture of the disease. Dyspnea is the main symptom of almost all IPD, occurs in most patients, especially in young children, and is the earliest sign of the disease. DN initially occurs or intensifies during physical activity and has a steadily progressive nature. In some patients, shortness of breath is accompanied by wheezing. These manifestations of the disease may be mistaken for bronchial asthma. Patients with IPD usually present with a nonproductive cough or with scanty mucous sputum. Cyanosis is a less constant and later sign of the disease; it occurs or intensifies with physical activity, in young children during feeding. Physical changes in the lungs with IPD are quite specific. When inhaling, patients hear soft, crepitating “cellophane” wheezing. They can be variable in their severity and localization. The discrepancy between severe shortness of breath and relatively minor physical changes in the lungs is one of the most important differential diagnostic signs that makes it possible to clinically distinguish IPD from other chronic diseases of the bronchopulmonary system. In the later stages of the disease, as a rule, there is progression of shortness of breath and the formation of pulmonary heart failure due to hemodynamic disturbances in the pulmonary circulation. As a result of a decrease in the diffusion capacity of the lungs and the development of ventilation-perfusion imbalance in patients, arterial hypoxemia is determined in the early stages of the disease only during physical activity. As the process progresses, hypoxemia is recorded at rest, accompanied by hypocapnia. Hypercapnia appears in advanced cases of the disease. When examining the function of external respiration, a predominantly restrictive type of ventilation impairment is detected, a decrease in the main lung volumes.

The most important diagnostic methods for IPD are chest x-ray methods. High-resolution computed tomography is becoming increasingly important. In the early stages of the disease, predominantly intensification and deformation of the pulmonary pattern, a decrease in the transparency of the pulmonary fields of the “ground glass” type, and small focal shadows are determined. As the process progresses, the deformation of the pulmonary pattern becomes more pronounced, signs of interstitial fibrosis and cavitary formations are revealed, and a picture of a “honeycomb lung” is formed. The most accurate diagnosis of most ILDs is
possible by assessing lung biopsy material. Lung biopsy is currently considered the gold standard in the diagnosis of ILD, allowing not only to establish a diagnosis, but also to predict the prognosis of the disease. However, a biopsy is not indicated and possible in all cases of ILD: it is currently performed only in 11-12% of patients with interstitial lung diseases.

If you stop contact with the antigen, complete recovery is possible in a few days or weeks. With repeated contacts, relapses of the disease develop, which can be subacute in nature, remain unrecognized, which leads, unexpectedly for the patient and the doctor, to the transition of the disease to a chronic form. The main symptom of the subacute form is shortness of breath, which persists for several weeks or months. In the chronic form, typical symptoms in the clinic are constant shortness of breath, cough with mucous sputum. With physical exertion, shortness of breath intensifies and cyanosis develops. On auscultation, constant crepitating rales are heard. The state of health gradually worsens, weakness, fatigue, loss of appetite, weight loss, and decreased physical activity appear. Upon examination, deformation of the chest is determined in the form of its flattening, changes like “drumsticks” and “watch glasses” develop.

References: