



## FEATURES OF THE COURSE OF CLINICAL AND MORPHOLOGICAL SIGNS OF PNEUMONIA IN YOUNG CHILDREN

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### Abstract

Today, the problem of pneumonia development in young children remains one of the most pressing in medical practice. Although there are achievements in the prevention and treatment of this disease, it is still highly prevalent among young children and is one of the main causes of infant mortality among the population.

**Keywords:** pneumonia, early age, clinical presentation, morphology.

### Introduction:

Pneumonia is an infectious disease associated with the penetration of microorganisms into the respiratory system. The resulting inflammatory reaction in the lung parenchyma depends on the number and virulence of microorganisms, the state of the defense mechanisms of the respiratory tract and the body as a whole. The incidence of pneumonia is between 15 and 20 per 1000 children in the first year of life. According to the conditions of infection, pneumonia is divided into community-acquired (home) and nosocomial (hospital, intra-hospital), in newborns - into intrauterine (congenital), intranatal and postnatal (acquired), the latter can also be community-acquired and nosocomial. Ventilation pneumonia - pneumonia developing in individuals on artificial ventilation (ALV) - is divided into early (first 4 days on ALV) and late (more than 4 days on ALV). Pneumonias associated with immunodeficiency states are also distinguished. Community-acquired pneumonias are those that occur in a child under normal home conditions, while nosocomial pneumonias are pneumonias that develop after 48 hours of the child's stay in the hospital or within 48 hours after discharge. Intrauterine pneumonias are those that appear in the first 72 hours of the child's life. According to the nature of the clinical and radiological picture, focal, focal-confluent, segmental, lobar (croupous) and interstitial pneumonias are distinguished. In addition, according to the severity of the course, extremely severe, severe, moderate and mild pneumonias are distinguished. The severity of the clinical course is determined by the presence and degree of expression of pulmonary-cardiac insufficiency and toxicosis, as well as the presence of complications. In turn,





complications are divided into pulmonary - pleurisy, pulmonary destruction (abscess, bullae, pneumothorax, pyopneumothorax) and extrapulmonary - septic shock, otitis, meningitis. In children over 6 months, the main causative agents of community-acquired pneumonia are viruses: respiratory syncytial, parainfluenza (type 3 and 1), influenza A and B and, less often, adenoviruses. Of the bacterial pathogens, *Streptococcus pneumoniae* predominates in children over 6 months, causing about half of all community-acquired pneumonia. Less common are pneumonias caused by *H. influenzae* type B (up to 10%), *Mycoplasma pneumoniae* (less than 10% of cases), and even less common are pneumonias caused by *Chlamydia pneumoniae*. In the etiology of hospital pneumonia, both the hospital microflora, usually resistant to antibiotics, and the patient's automicroflora play a role. Among the pathogens most often encountered are *E. coli*, *K. pneumoniae*, *Proteus* spp., *Enterobacter* spp., *Pseudomonas aeruginosa*, and less often *S. aureus*. Often, infection occurs during therapeutic and diagnostic procedures (sputum suction, catheterization, bronchoscopy, thoracocentesis). The nature of the microflora depends on the hospital profile and the anti-epidemic regime. Focal pneumonia (bronchopneumonia) is more often observed in young children and currently accounts for 30-40% of the total number of pneumonias [2, 3]. In the overwhelming majority of cases, focal pneumonia develops acutely against the background of ARVI already in the first days or on the 4-7th day from its onset. Viral infection disrupts the protective mechanism of the lung, suppresses phagocytosis, changes the bacterial flora, affects the functioning of the ciliated epithelium and contributes to the emergence of inflammatory foci. The severity of pneumonia that develops against the background of ARVI is determined by the nature of the viral infection, bacterial flora, and the characteristics of the child's individual reactivity. Focal pneumonia is characterized by a deep, wet cough (a sign of bronchial damage). Intoxication is usually moderate. The body temperature rises to 38 degrees, lethargy or anxiety, pale skin, moderate cyanosis of the nasolabial triangle, increased respiratory rate with retraction of the intercostal spaces and tension of the wings of the nose are noted. Percussion of the chest reveals the presence of pulmonary sound, sometimes with a tympanic tint or with a slight shortening. An important diagnostic sign of focal pneumonia is the characteristic clinical picture in the lungs: persistent local fine-bubble wheezing or crepitation, mainly on one side. With concomitant bronchitis, widespread dry and wet wheezing of various sizes are heard.

**Objective of the study:** To study the features of clinical and morphological signs of pneumonia in young children.





### **Material and Methods:**

The study analyzed 6 case histories of acute pneumonia in young children and 3 autopsy reports performed in the pathological anatomy bureau in 2016-2017 in order to assess the extent of lung damage and the features of morphological manifestations.

### **Results and Discussion:**

Among the children whose pneumonia ended fatally, there were 2 girls (63.6%) and 1 boy (36.4%). Of these, 2 children were from large families and 1 from small families. When analyzing the age structure, it was found that pneumonia was diagnosed 4 times more often (81.8%) in deceased children under one year of age than in children aged 1-3 years (18.2%). The children were admitted to hospital in severe or extremely severe condition. From the medical history, the first symptoms of pneumonia in children were usually a rise in temperature to 38.5-39.0, cough, runny nose. The temperature lasted for 3 or more days. According to the mother, all sick children had a change in behavior - excitement, anxiety or, conversely, lethargy, apathy, decreased appetite, sometimes to complete anorexia. Almost all children with pneumonia upon admission had tension or distension of the wings of the nose, often retraction of the intercostal spaces, a change in the normal ratio between the respiratory rate and heart rate (1:2, 1:2.5 instead of 1:3). In the peripheral blood, changes were found in the form of leukocytosis and an increase in ESR. Acute pneumonia acted as the underlying disease and the only cause of death in 2 children (27.3%). Much more often, pneumonia was diagnosed as part of a combined underlying disease - in 4 children (62.7%). By the nature of inflammation, fibrinous-purulent pneumonia was predominant - in 1 child (55.7%), and in all children it was abscessing. Interstitial pneumonia was diagnosed less often - in 1 child (18.3%). Among other pathologies, against which pneumonia developed, were: congenital defects of the cardiovascular system - in 5 children (45.5%), congenital malformations (CM) - in 4 children (36.4%), diseases of the nervous system - in 3 children (27.3%), generalized infectious diseases (sepsis) - in 2 children (63.6%). The study of the premorbid state of children showed that 3 children (54.4%) had PEM (protein-energy malnutrition), and in 5 children (36.4%) PEM was of grades II-III. Rickets - in 2 children (18.2%). In 6 children (81.8%), deficiency anemia of varying severity was recorded as a concomitant disease. In the lungs of all deceased children, there was a uniform picture of inflammation: the lungs were poorly airy, of dense consistency, and full-blooded. Among the cells, lympholeukocytic infiltration, desquamation and proliferation of the bronchial epithelium, thickening of the interalveolar septa due to edema and hemorrhages were mainly visible, in the lumens of the alveoli and bronchi there was desquamated epithelium, mucus,





leukocytes, and fibrin masses. In some areas, the lung tissue was diffusely infiltrated by leukocytes, with decay. In some sections, serous-hemorrhagic exudate was found in the lumen of the alveoli, with massive lympholeukocytic infiltration of the interalveolar septa, and impregnation of the parenchyma with fibrin. In the lumen of the vessels there are stasis, erythrocyte sludge, erythrocyte-fibrin thrombi. All the children who died from acute pneumonia that we studied had immune deficiency of varying severity. This is evidenced by atrophy and accidental involution of the thymus gland. The mass of the thymus gland in patients with acute pneumonia was smaller, the longer the child was ill. The stroma is loosely fibrous, edematous. Edema and plethora of the vessels, focal lipomatosis, collapse of lobules, diffuse loss of lymphoid elements in the lobules are noted. The border between the cortex and medulla is not expressed, Hassall's corpuscles are medium and small, few in number, layered, located mainly in the center of the lobules, individual ones merging with each other. Hyalinosis and focal fibrosis of the ethmoid stroma are visible. In the lumen of the vessels there are stasis, erythrocyte sludge, erythrocyte-fibrin thrombi. From the side of internal organs in all children from large and small families who died from pneumonia, various changes were found depending on age and concomitant diseases. Most often, pathomorphological changes were found in the myocardium, liver, kidneys. They were characterized mostly by signs of cardiomyocyte dystrophy, parenchymatous protein and fatty dystrophy.

### **Conclusions.**

1. Community-acquired pneumonia was characterized by a gradual onset of the disease with gradual development of severe intoxication, prolonged cough, and fever.
2. The hemogram of children with community-acquired pneumonia revealed reliable changes in the form of leukocytosis with lymphocytosis and an accelerated ESR.
3. Histological changes in the lungs are characterized by lympholeukocyte infiltration of the lung parenchyma, which, in our opinion, can be regarded as an acute inflammatory process.
4. Atrophy and accidental involution of the thymus gland is a morphological marker of immunodeficiency. This is evidenced by lipomatosis, lobular collapse, a decrease in the number of thymocytes, single, small Hassall's bodies.
5. Given the instability of the immune system in children, it is necessary to continue studying the features of the course of pneumonia in young children in order to correct IDS.





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