

The Change in the Morphological Picture in Children with Immunodeficiency of Advanced Disease with Pneumonia

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Abstract--*The aim of the work was to study of the morphological changes in the organs in case of acute pneumonia in children with immunodeficiency. The object of the study was the internal organs of 43 children with immunodeficiency from the age of 10 days to 7 years who died from acute pneumonia. The etiology of pneumonia, the nature of the inflammatory process in the lungs, changes in other organs, complications and causes of death from acute pneumonia in children with immunodeficiency are described in the article. A study of the morphological structure of pneumonia showed that mononuclear infiltration of the pulmonary parenchyma with a pronounced violation of blood rheology prevailed in viral pneumonia. In viral-bacterial pneumonia, interstitial tissue is infiltrated with lymphocytes, polymorphonuclear leukocytes in the lumen of the alveoli.*

Keywords--*viral pneumonia, morphology, internal organs, children, immunodeficiency*

I. INTRODUCTION

Acute pneumonia is currently one of the urgent problems of pediatrics. This is due to its wide distribution among children and high mortality [2]. According to WHO data, about 155 million cases of pneumonia in children are recorded annually in the world, of which approximately 1.4 million die before the age of five. Thus, this disease is one of the leading causes of infant mortality [1].

Currently, there is no reason to argue that the overall mortality in viral pneumonia in children with immunodeficiency is higher than in traditional forms of viral pneumonia. Nevertheless, it is known that in severe cases of the course, viral pneumonia in children with immunodeficiency is often complicated and rapidly progresses with the development of severe respiratory failure and acute respiratory distress syndrome [9].

In Uzbekistan, acute pneumonia remains among the leading causes of death in children of the first year of life. According to various sources, the frequency of pneumonia as the main cause of deaths varies from 21.6 to 43.9% [4]. A retrospective analysis of mortality in pediatric wards of the over the past 5 years has shown that acute pneumonia (35.5%) is the main place in the nosological structure of the wards [3].

According this the aim of the work was to study of the morphological changes in the respiratory system in acute pneumonia in children with immunodeficiency.

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II. MATERIAL AND METHOD

The object of the study was the internal organs (lungs, thymus, brain, heart, kidneys, liver, adrenal glands, spleen and lymph nodes) of 43 children with immunodeficiency from the age of 10 days to 7 years who died from acute pneumonia. For bacteriological examination, the material was taken from the main bronchi, for histological examination, pieces of lungs were fixed in 10% formalin and embedded in paraffin according to the Lloyd method, histological sections were stained with hematoxylin and eosin.

Depending on age, the dead children were divided into the following groups: from 10 days to 1 month (5 children), from 1 to 6 months (15), from 7 months to 1 year (9), from 1 year to 3 years (9), from 3 to 7 years (5). The deaths on the first day were 23, on the 2-5th day - 9, on the 6-10th day - 6, after 10 days - 5. We took into account body weight at birth the nature of nutrition, the duration of the disease before admission to the clinic. Boys prevailed - 62%. Bilateral acute pneumonia predominated with the primary localization of the inflammatory process in the II, IV, VI, IX, X segments of the lungs on both sides. By etiology, acute pneumonia was divided into viral (27) and viral-bacterial (16).

III. RESULTS

It was established that in children of the first year of life with immunodeficiency, who died from viral pneumonia, the process had a bilateral focal or focal-drainage character. There was a multiple focal process in the lung tissue, severe circulatory disorders, lesions of the interstitial tissue of the lung, the rapid development of extrapulmonary complications. In viral pneumonia, morphological changes are found in the trachea, bronchi and lungs. Histologically, the lung tissue was with foci of hemorrhage. The foci of inflammation of the lung tissue of the alveoli contained serous hemorrhagic exudate, erythrocytes, alveolar macrophages, single polymorphonuclear leukocytes, and a large number of desquamated alveolocytes (Fig. 1a). In peribronchial and interalveolar septa, lymphocytic infiltration was determined (Fig. 1b).

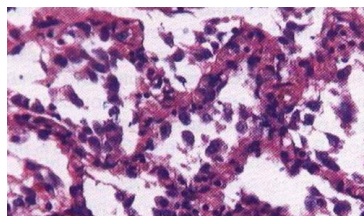


Fig 1a. Viral pneumonia. A lot of desquamated cells in the alveoli, edema of the interalveolar septa. Hematoxylin and eosin stain.

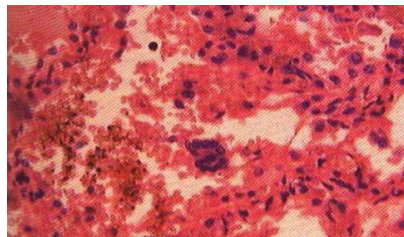


Fig 1b. Viral pneumonia

Among the erythrocytes in the lumen of the alveoli there are cell symplast, single lymphocytes, moderate lymphoid infiltration of the edematous walls of the alveoli. Hematoxylin and eosin stain.

Violations of blood rheology were observed not only in the lungs, but also in the central nervous system, internal organs, serous and mucous membranes. There were plethora, stasis, loosening of brain tissue around the vessels, various degrees of severity of perivascular and pericellular edema, retinal rarefaction of brain tissue. In 25% of cases, diapedetic hemorrhages were noted, sometimes significant. Secondary leptomeningitis occurred in 12.5% of cases. A histological examination of heart tissue in children who died from viral pneumonia revealed edema of the myocardial interstitium, degenerative changes in muscle fibers and inflammatory cell infiltration in 37.5% of cases (fig 3). All deceased observed discirculatory, isotrophic and inflammatory changes in the liver, kidneys and adrenal glands.

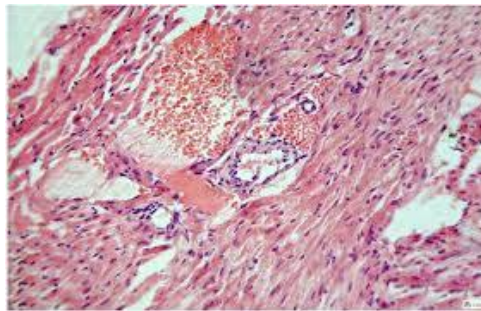


Fig 3. Viral pneumonia. Varicose veins, narrowing of arterioles with perivascular edema and hemorrhage. Gentian-eosin staining (magnification: eyepiece 10, lens 20).

The morphological rearrangement that occurs in the organs of immunogenesis is of particular importance. In case of viral pneumonia in the thymus, various phases of precipitating involution were found. In the lymph nodes, an expansion of the paracortical zone and the cerebral layer with diffuse mononuclear infiltration was noted. Similar changes were also found in the spleen, with increased light reactive centers and red pulp hyperemia. The most common immediate cause of death by reason of viral pneumonia was respiratory and acute cardiovascular failure.

In case of viral-bacterial association, changes in the lungs were different. Microscopic examination revealed pronounced circulatory disorders and severe dystrophic changes in the epithelial cells of the bronchi, swelling and desquamation of the epithelial cells of the mucosa of the trachea and bronchi. In the lungs, bilateral confluent pneumonia was determined, which was often localized in the lower lobes of the lung.

The lung on the incision was colored with dark red, the boundaries of inflammation are fuzzy. Foamy-bloody liquid drained from the cut surface. Histologically, there was a pronounced circulatory disorder, the alveoli were filled with a large number of desquamated alveolar epithelium, the interalveolar septa were thickened, edematous, with dilated full-blooded capillaries, infiltrated by lymphocytes and polymorphonuclear leukocytes, sometimes hyaline membranes were found (7). On the part of the internal organs, dystrophic and discirculatory changes were mainly determined. In one case, focal inflammatory infiltration by polymorphonuclear cells was

detected in the myocardium. In the thymus, in all cases, various phases of the incidental transformation were observed.

The abscess form was found in 18.5% of all viral-bacterial pneumonia, and was always accompanied by purulent pleurisy. In the liver, centrallobular fatty degeneration of hepatocytes developed. In the heart, epicardial edema with foci of hemorrhage, plethora of myocardial vessels, granular and vascular impairment of myocytes, foci of hemorrhage and accumulation of polymorphonuclear cells. Of extrapulmonary complications, leptomeningitis occurred in 18.5% of cases, sepsis in 14.8%, myocarditis in 44.4%, and nephritis in 14.8%. The results of the study of the thymus showed that among children who died from viral-bacterial pneumonia, 63% of patients in the thymus experienced an incidental transformation of mainly grade III, thymomegaly in 29.7%, hypoplasia in 7.4%.

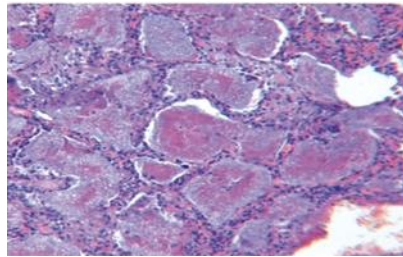


Fig 4. Viral bacterial pneumonia.

In the lumen of the alveoli, foamy eosinophilic masses. Inter-alveolar septa and stroma of the lung with diffuse inflammatory infiltrate, mainly from lymphocytes, macrophages, inflammatory hyperemia (acute interstitial pneumonia).

The analysis showed that viral and viral-bacterial pneumonia were more often observed at the age of 1 to 6 months, viral pneumonia - at the age of 7 months to 1 year. Among those who died from acute pneumonia in 24.3% of cases, children were born premature with a weight of less than 2500 g. An analysis of the nature of the nutrition revealed that the highest mortality in acute pneumonia was observed in children who were breast-fed (78.38%). Most children who died from acute pneumonia before being admitted to the hospital were ill from 2 to 7 days. On the first day of the hospital stay 16 children (43.3%) died, on the 2-3rd day - 4 (10.8%), on the 4th day - 3 (8.1%), on the 6th day or more - 14 (37.8%).

IV. CONCLUSION

Thus, viral infections predominate in the etiological structure of acute pneumonia in children. Children in the first half of the year have higher mortality from viral pneumonia. A study of the morphological structure of pneumonia showed that mononuclear infiltration of the pulmonary parenchyma with a pronounced violation of blood rheology prevailed in viral pneumonia. In viral-bacterial pneumonia, interstitial tissue is infiltrated with lymphocytes, polymorphonuclear leukocytes in the lumen of the alveoli. Among the pre-morbid factors, in addition to the immunodeficiency of children, low body weight at birth and artificial feeding should be noted, and among the factors contributing to an unfavorable outcome, immunodeficiency states and late hospitalization were important. Immunodeficiencies in 10 (27%) cases were primary, in 27 (73%) secondary.

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