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PATHOMORPHOLOGY OF PRIMARY ATELECTASIS OF THE LUNGS OF INFANTS

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Abstract: Primary atelectasis of the lung " penetrates into the respiratory distress syndrome in the form of a separate nosological unit threeraydi. In this article, specific pathomorphological symptoms of primary atelectasis of the lung are studied. As a Material, the lungs of infants who died from atelectasis in the neonatal period were microscopically studied. The results of the microscopic examination revealed that lung tissue at first glance has an immature developed appearance.

When primary atelectasis developed 2-3 days before the death of infants, it is observed that inflammation has developed in the lung tissue, that is, macrophages, neutrophils, migratory alveolocytes are detected in the alveolar cavity. primary atelectasis occurs when the tissue of the alveolar cavity has a dense connective tissue and a pattern of cellular handles, blood vessels are wide and full, blood After 7-10 days, it is determined that the alterative-proliferative processes have turned into atelectatic pneumonia by outbreaks. As a result of it, pneumosclerosis, bronchoectasis and the transformation of the bronchi into a retentional cyst are observed. Often, in place of atelectasis, connective tissue grows and Sclerosis develops.

Key words: infant, lung, distress syndrome, primary atelectasis

摘要：“原发性肺不张”以单独的疾病分类单元threeraydi的形式渗透到呼吸窘迫综合征中。本文研究了原发性肺不张的具体病理形态学症状。作为材料，死亡婴儿的肺对新生儿期肺不张进行了镜检，镜检结果显示，肺组织乍看呈未成熟发育状态。

当婴儿死亡前2-3天出现原发性肺不张时，观察到肺组织出现炎症，即在肺泡腔内检测到巨噬细胞、中性粒细胞、游走性肺泡细胞。肺泡腔内有致密的结缔组织和细胞柄纹，血管宽而饱满，血 7-10天后，确定变异-增殖过程已转变为肺不张的肺炎爆发。结果，观察到肺硬化、支气管扩张和支气管转化为滞留性囊肿。通常，代替肺不张的是结缔组织生长和硬化症发展。

关键词: 婴儿, 肺, 窘迫综合征, 原发性肺不张

Relevance of the problem. In diseases of infants, respiratory disorders account for 8,8% of the cases, and in the 2nd place, and in most cases more than threeraydi in infantile infants, depending on the morphofunctsiional characteristics of the respiratory system members. In particular, if the respiratory distress

syndrome in infants is na 6-12%in general – in infantile births-1-1, 8%, the weight is 0,4-0,5% in very rare infants [2, 4]. The main reason for the development of this disease is the lack of an internal surfactant in the lungs of infants, the stagnation of the respiratory muscles and the state of inability to breathe independently on its

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own. In foreign scientific literature, the terms "respiratory distress syndrome" and "primary atelectasis of the lungs" are synonyms and develop in the form of a separate nosological unit. Clinical differential diagnosis of lung diseases in these infants is considered very difficult. Primary atelectasis from respiratory disorders is the main among the direct cause of death of infants in pathologic examinations. Primary atelectases as the main morphofunctional forms of respiratory disorders in infantile babies and a lot of hyaline membranes tripled. Intranatal aspiration of water of the capillaries as the main risk factors is injury of the epithelium of the alveoli and increased permeability of the wall of the capillaries [1,3]. Primary atelectasis of the lungs of infants, this is an opening or re – closing of the pulmonary alveoli, depending on the specific structure and central management of the Broncho-alveolar tissue for 2 days after birth. The literal translation of atelectasis is "complete openness", denotes the state of anatomical spread of the lungs. Infants atelectasis of the lungs is included in the "syndrome of breathing disorders (nbs)". Its overall survival rate is 1% of all infants, while in infancy it is threeraydi at 14% [2,4, 5, 6,7].. The urgency of the problem of atelectasis for pediatrics lies in the abundance of the reasons why babies are punctured lung alveolar tissue in a one-month period. Atelectasis is a manifestation of the syndrome of dyspnea, which is the main cause of respiratory failure in the neonatal period. The smaller the gestation period and body weight, the higher the degree of its occurrence. On average, 30% of babies born in the week 65 of gestasia, if prophylaxis with steroid hormones is performed in 35%, 34% of those born in the week 25 of gestasia, three in 10% when prophylaxis is performed inraydi [2,

6,7]. All cases that lead to a lack of surfactants are considered dangerous factors of NBS, including: incomplete development of the lungs, asphyxiation of the fetus, morphofunctional insufficiency, violation of pulmonary-cardiac adaptation, pulmonary hypertension, violation of metabolism, including: acidosis, hypoproteinemia, hypofermentosis, electrolyte disorders. The causes of primary atelectasis in infants are: slowness and slow excitation of the respiratory center, incomplete development of the respiratory system, conducted hypoxia or asphyxia, damage to the head or spine, as well as the syndrome of aspiration by paper water [2, 3, 4].

Purpose of the work. To identify specific pathomorphological signs of primary atelectasis of lung of infants.

Material and methods.

Results and their discussion.

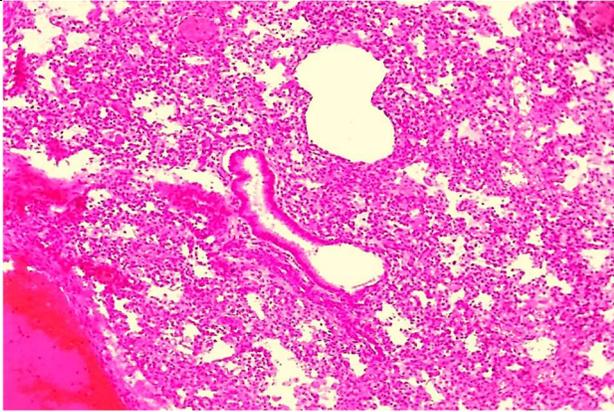
Morphological studies have shown that primary atelectasis of the lungs is most often observed in infants born dead, both developed in their lungs, and they have a small hearth in size, making a squeaky sound when cutting and fully drowned when thrown into the water. On the placement of primary atelectasis foci, it was often detected in I, II, IX, X segments of both lungs, IV and V-segments of the left lung, the cause of which is due to the low level of differentiation of these segments. In the cavity of the bronchi and alveoli, it was found that there was usually a paper broth, a mucous substance and a little blood. In terms of appearance, the atelectasis areas of the lungs are similar to meat, the pieces of the lungs are much smaller than their usual size, they differ in grayness compared to the surrounding soggy. It turned out that the atelectasis furnaces are small in appearance, dark red color, reminiscent of the inferkt furnace.

If primary atelectasis develops 2-3 days before the death of infants, it is observed that inflammation develops in the lung tissue. As a result, macrophages, neutrophils, migrating alveolocytes are detected in the alveoli cavity. After 7-14 days, it is determined that the alterative-proliferative processes have turned into atelectatic pneumonia by outbreaks. As a result of it, pneumosclerosis, bronchoectasis and the transformation of the bronchi into a retentional cyst are observed. Often, in place of atelectasis, it is determined that the connective tissue has grown and from the appearance of sclerosis the vascular system of the lungs has undergone reconstruction. If the foci of atelectases are developed in the subpleural area of the lungs, they develop in the posterior-basal segments of the lungs, morphologically partial puncture of the alveoli and the surrounding pathomorphological changes develop to a different extent.

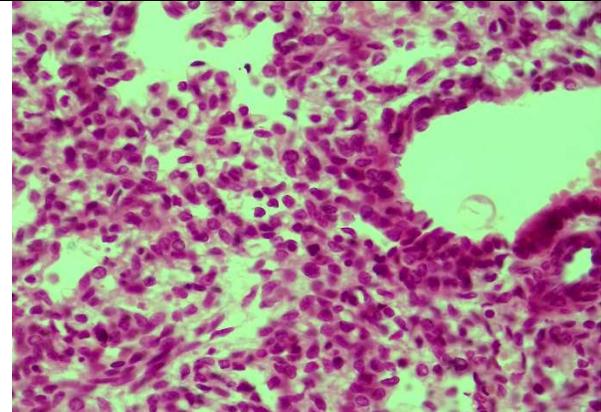
The results of the microscopic examination showed that when lung tissue is seen on a small lens of a microscope, it is determined from a single glance whether it has an immature developed appearance. In the lung tissue, only the bronchi and bronchioles are determined to have a different size, irregular-shaped tubular structure. The appearance of the sac of alveolar tissue is not noticed. The respiratory alveoli and their adjacent cavities are not detected. The tissue of the alveoli range is composed of dense connective tissue and cellular bunches (Figure 1). The tissue of the alveoli range consists of dense connective tissue and cellular bunches (Figure 1). Such a dense lung tissue has a structure in which blood vessels are wide and full, blood flows around them.

When viewed on a large lens of a microscope, it is determined that the wall of the bronchi is only a phrase from tissue structures that have not found a good takomil. It is determined that only one and several layers of malignant epithelial cells form the bronchial wall. It is determined that around it there is a delicate tissue consisting of dense young histiocytic and lymphoid cells. But it can not be determined that a cavity of alveoli appears in this delicate and young cell tissue. This means that since lung tissue is young and immature, alveoli are not differentiated and organized, a dense and alveolar cavity in the form of primary atelectasis is determined by the pattern of the tissue without it (Figure 2). In most cases, it is determined that the blood vessels in the lung tissue, which undergo the primary atelectasis process, are full and massive blood clots appear in the lung tissue (Figure 3). It is observed that the foci of blood transfusion are dense and without alveolar cavities, hematomas with an unclear form of blood transfusion appear in the tissue structure. As a result of the blood transfusion, the lung tissue, which suffered a primary atelectasis, becomes more dense and deformed, it is determined that the spleen tissue resembles a red pulp.

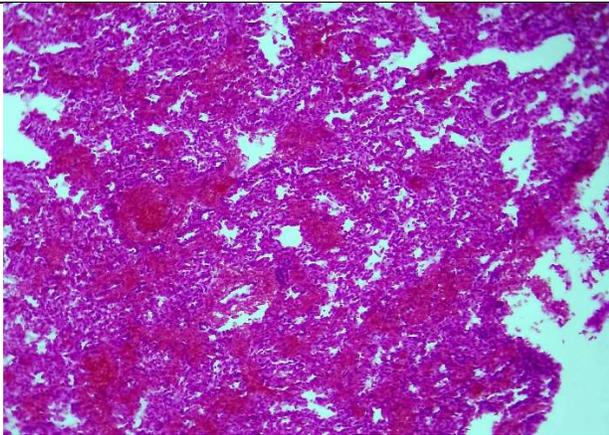
When viewed in a large lens of a microscope, it is determined that the alveolar tissue, which undergoes primary atelectasis, is composed of a tissue composed of non-densely formed histiocytic and lymphoid cells. In the tissue with such a structure did not form alveoli, in the tissue of the alveoli range, alveolacids, blood vessels and intermediate tissue also have a non-differentiated structure. Such dense and not formed tissue contains blood vessels, full-fledged, most areas of which are massively drained of blood (Figure 4).



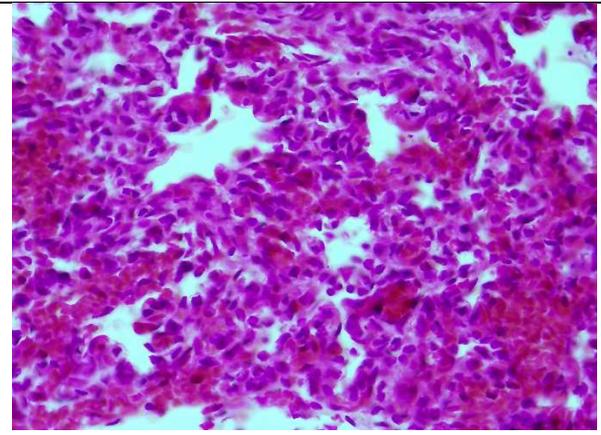
1-picture. Alveoli have emerged from dense connective tissue and cellular bunches in which the intermediate tissue is joined with each other. Paint: G-E. X: 10x10.



2-picture. Primary atelectasis furnaces are dense and alveolar cavity-free tissue pattern is determined. Paint: G-E. X: 10x40.



3-picture. Primary atelectasis is the fullness of blood vessels in lung tissue and the emergence of massive blood transfusions. Paint: G-E. X: 10x40.

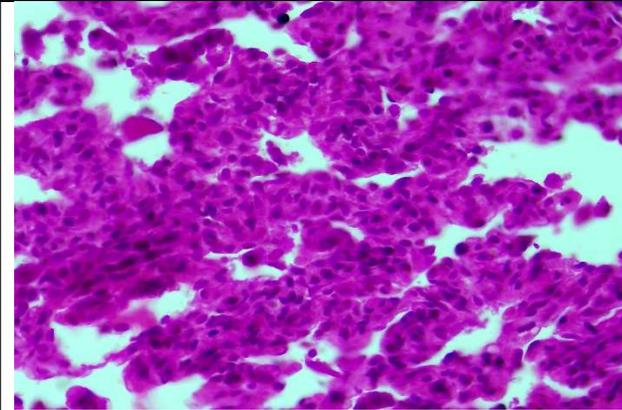


4-picture. In the non-formed lung tissue, veins are clogged, blood is poured into most areas of the body massively. Paint: G-E. X: 10x40.

When the primary atelectasis develops in the lung tissue and 2-4 days later the dead baby is examined microscopic of lung tissue, this condition is determined that the respiratory part of the lung and alveolar cavity appear in the form of cracks that are not so wide. Between such spaces, it is determined that the histiocytic cells, which are sensitive to proliferative inflammation in the tissue structure, are faecal, hypertrophied, in a dense state, proliferative inflammatory infiltrate appears (Figure 5). It is observed that in

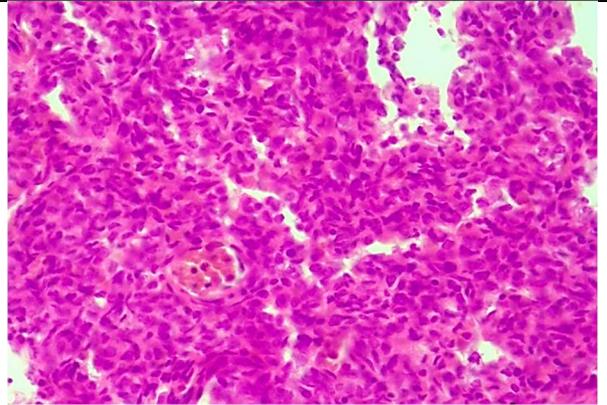
the composition of such inflammatory infiltrates appear macrophages, giant cells and lymphoid cells. It is determined that in the respiratory bronchioles and alveoli part of the lungs there is a rupture of the epithelium, including alveocytes, in which a number of ridges appear. It is known that babies who die 7-10 days after the development of primary atelectasis in lung tissue, when lung tissue is microscopically studied, it is observed that alveolar tissue becomes more dense and resembles spleen tissue

without cavities. Only in some places it is determined that there are cavities of an indefinite form, in which there are epithelial and inflammatory cells that migrate in the cavity. The fact is that it is determined that the tissue that forms the lung tissue, that is, the tissue of the alveoli range, is sharply thickened and consists of a connective tissue that is not formed (Figure



5-picture. Hypertrophy of proliferative inflammatory histiocytic cells in the range of alveoli, the emergence of a proliferative inflammatory infiltrate in a dense state. Paint: G-E. X: 10x40.

6). The growth of such connective tissue, that is, the emergence of multiple sclerosis, the proliferation of histiocytic cells, and the emergence of fibrous structures in between. The blood vessels in its composition were also exposed to proliferation, manifested by the proliferation of endothelial and pericytic cells that formed their walls.



6-picture. In alveolar tissue, the growth of connective tissue, that is, the appearance of sclerosis bunches. Paint: G-E. X: 10x40.

Conclusion

1. Infants' pulmonary atelectasis has been linked to "respiratory disorder syndrome (nbs)" and its overall incidence rate is 1% of all infants, with three-raydi in 14% in infantile births. The urgency of the problem of atelectasis for pediatrics lies in the abundance of the reasons why babies are punctured lung alveolar tissue in a one-month period.

2. In a microscopic examination, the lung tissue is determined at first glance by the presence of immature developed appearance. In the tissues of the lungs, only the bronchi and bronchioles are manifested in a tubular structure of different sizes, irregular shape. The appearance of the sac of alveolar tissue is not noticed. The respiratory

alveoli and their adjacent cavities are not detected.

3. The pattern of dense connective tissue and cellular bunches, in which the alveoli interleaved tissue is joined with each other, has a structure in which blood vessels are wide and full, blood clots form around them.

4. When primary atelectasis develops 2-3 days before the death of infants, inflammation is observed in the lung tissue, that is, macrophages, neutrophils, migratory alveolocytes are detected in the alveolar cavity.

5. After 7-10 days, it is determined that the alterative-proliferative processes have turned into atelectatic pneumonia by outbreaks. As a result of it, pneumosclerosis, bronchoectasis and the transformation of the bronchi into a

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