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PATHOMORPHOLOGY OF PRIMARY ATELECTASIS OF INFANT LUNGS

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ABSTRACT

Primary atelectasis of the lungs “enters the respiratory distress syndrome and occurs in the form of a separate nosological unit. This article examines the specific pathomorphological features of primary pulmonary atelectasis. As material, the lungs of infants who died of atelectasis in the neonatal period were studied microscopically. Microscopic examination revealed that the lung tissue had an underdeveloped appearance at a glance.

Alveolar interstitial tissue is composed of dense connective tissue and cell clots, the blood vessels are wide and full, surrounded by a hemorrhagic structure. If primary atelectasis develops 2-3 days before infant death, inflammation of the lung tissue is observed, i.e. macrophages, neutrophils, migrating alveocytes are detected in the alveolar cavity. After 7-10 days, it is determined that the alterative-proliferative processes are exacerbated and turn into atelectatic pneumonia. It results in pneumosclerosis, bronchiectasis, and the development of retinal cysts of the bronchi. Often, connective tissue grows in place of atelectasis and sclerosis develops.

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

INTRODUCTION

Respiratory disorders account for 8.8% of infantile diseases, ranking 2nd, and are more common in premature infants due to the morphofunctional characteristics of the respiratory system. In particular, respiratory distress syndrome in infants is generally 6-12%, in premature infants - 1-1.8%, in very low birth weight infants - 0.4-0.5% [2, 4]. The main reasons for the development of this disease are the lack

of internal surfactant in the lungs of infants, the weakness of the respiratory muscles and the inability to breathe independently. In the foreign scientific literature, the terms "respiratory distress syndrome" and "primary atelectasis of the lungs" are synonymous and develop in the form of a separate nosological unit. The clinical differential diagnosis of these infantile lung diseases is very difficult. Primary atelectasis from respiratory disorders is a direct cause of infant mortality in pathological examinations. Primary atelectases and hyaline membranes are the most common morphofunctional forms of respiratory disorders in preterm infants. The main risk factors are intranatal aspiration of amniotic fluid, damage to the alveolar epithelium and increased capillary wall permeability [1,3].

The results of the microscopic examination showed that when the lung tissue is seen under a small lens of the microscope, it is determined at a glance that it has an underdeveloped appearance. In lung tissue, only the bronchi and bronchioles are found to have a tubular structure of varying size, indeterminate shape. The reticular appearance of the alveolar tissue is indistinguishable. Respiratory alveoli and their interconnected cavities are not identified. The interstitial alveolar tissue consists of densely packed tissue and cellular tufts joined together (Fig. 1). In such a dense lung tissue, the blood vessels are wide and full, with a structure around which blood flows.

When viewed through a large lens of a microscope, it is determined that the wall of the bronchi consists only of immature tissue structures. It is found that only one and several layers of disordered epithelial cells form the wall of the bronchiole. Around it is found the presence of dense tissue consisting of dense young histiocytic and lymphoid cells. However, it is not known whether alveolar cavities appear in this thin and young cellular tissue. Thus, it is determined that the lung tissue is young and immature, the alveoli are not differentiated, dense and in the form of primary atelectasis consists of tissue without alveolar cavities (Fig. 2). In most cases, the fullness of the blood vessels in the lung tissue affected by the primary atelectasis process and the appearance of massive hemorrhages into the lung tissue are detected (Fig. 3). It is observed that these foci of hemorrhage are dense and the formation of hematomas of indeterminate hemorrhage in the tissue structure without alveolar cavity. As a result of the hemorrhage, it is determined that the lung tissue that underwent primary atelectasis is further compacted and deformed, leaving the spleen tissue resembling red pulp.

When viewed under a large microscope, the alveolar tissue affected by primary atelectasis is composed of dense unformed histiocytic and lymphoid cells. Tissue with such a structure has no alveoli, alveolar interstitial tissue, alveocytes, blood vessels and interstitial tissue also have an undifferentiated structure. The

blood vessels in such dense and unformed tissue are dilated, full, and massively transfused into most areas (Fig. 4).

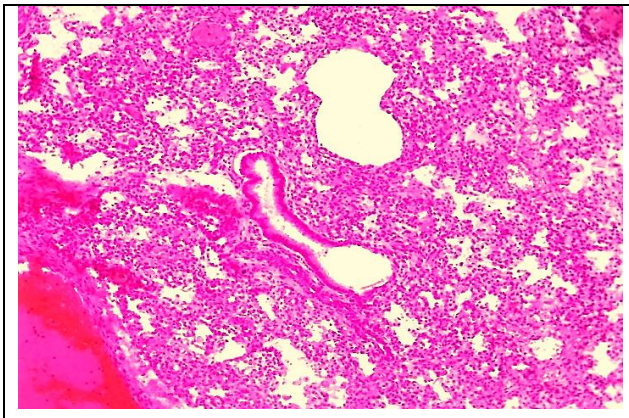

Figure 1. Alveolar interstitial tissue is formed from dense tissue and cellular tufts that are fused together. Paint: G-E. X: 10x10.

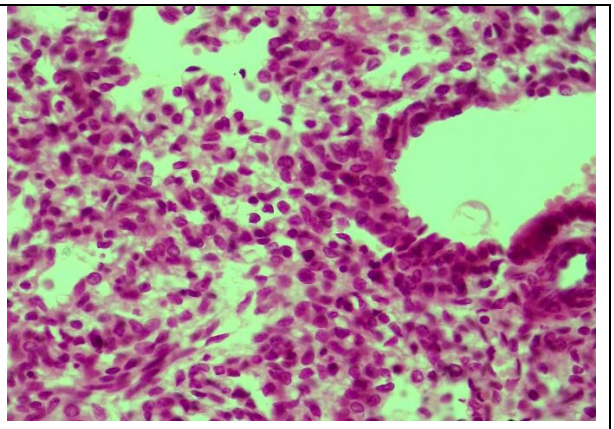

Figure 2. Primary atelectasis foci are found to be dense and composed of tissue without alveolar cavities. Paint: G-E. X: 10x40.

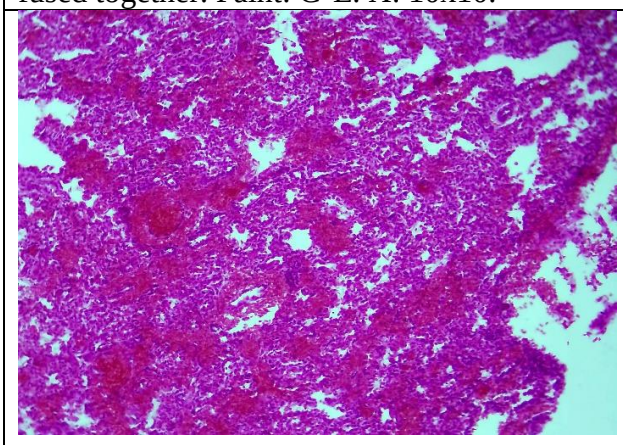

Figure 3. Primary atelectasis resulted in vascular occlusion and massive hemorrhage in the lung tissue. Paint: G-E. X: 10x40.

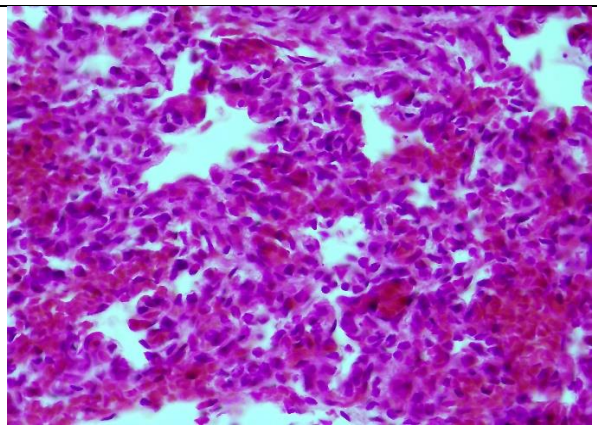

Figure 4. In the unformed lung tissue, the vessels dilate, and massive blood is infused into most areas. Paint: G-E. X: 10x40.

Microscopic examination of the lung tissue of infants who develop primary atelectasis in lung tissue and die 2–4 days later reveals that the respiratory portion of the lung and the alveolar cavity appear in the form of small cracks. In the tissue structure between such cavities, histiocytic cells, which are characteristic of proliferative inflammation, are activated and hypertrophied, forming a dense proliferative inflammatory infiltrate (Fig. 5). Such inflammatory infiltrates are characterized by the formation of macrophages, giant cells and lymphoid cells. Enlargement of the epithelium, including alveocytes, in the respiratory bronchioles and alveoli of the lungs is found to be located in a series of structures.

Microscopic examination of the lung tissue of infants who died 7–10 days after the development of primary atelectasis in the lung tissue revealed that the alveolar tissue became more dense and resembled splenic tissue without cavities. It is only in some places that indeterminate-shaped cavities appear, in which there are

migrating epithelial and inflammatory cells. In fact, it is found that the tissue that forms the lung tissue, i.e., the alveolar interstitial tissue, is abruptly thickened and composed of unformed connective tissue (Fig. 6). Such growth of connective tissue, i.e., the formation of sclerotic tufts, results from the proliferation of histiocytic cells and the formation of fibrous structures between them. The blood vessels in it also proliferated, manifested by the proliferation and proliferation of endothelial and pericytal cells that form the wall.

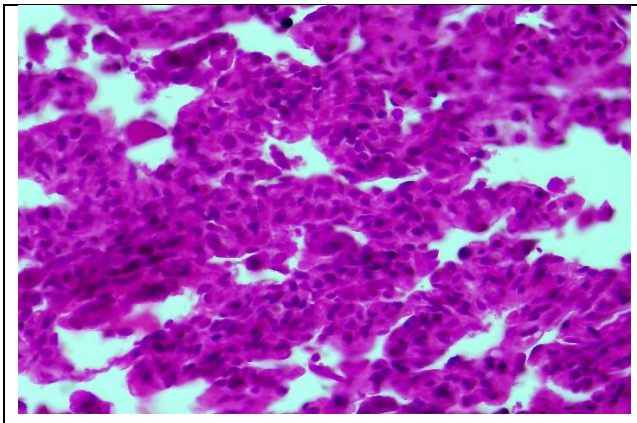

Figure 5. Hypertrophy of proliferative inflammatory histiocytic cells between the alveoli, the formation of dense proliferative inflammatory infiltrate. Paint: G-E. X: 10x40.

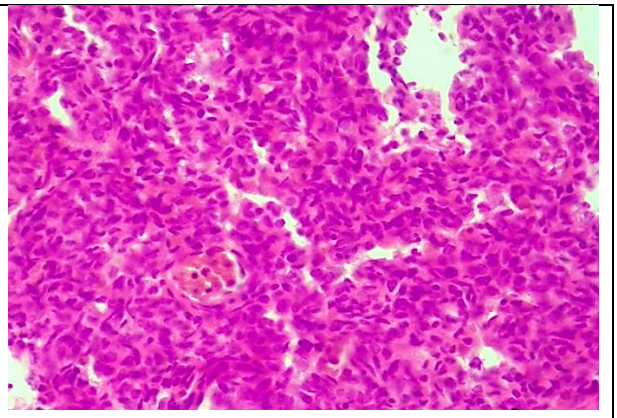

Figure 6. Growth of connective tissue in alveolar tissue, i.e., the appearance of sclerotic tumors. Paint: G-E. X: 10x40.

Conclusion

1. Infantile pulmonary atelectasis is referred to as “respiratory distress syndrome (NBS)” and its incidence is 1% of all infants and 14% of preterm infants. The urgency of the problem of atelectasis for pediatrics lies in the large number of causes of pulmonary alveolar tissue collapse in infants at one month of age.

2. Microscopic examination reveals that the lung tissue has an underdeveloped appearance at a glance. In lung tissue, only the bronchi and bronchioles appear in a tubular structure of varying size, indeterminate shape. The reticular appearance of the alveolar tissue is indistinguishable. Respiratory alveoli and their interconnected cavities are not identified.

3. It is observed that the interstitial tissue of the alveoli consists of dense tissue and cellular tufts connected to each other, the blood vessels are wide and full, having a structure around which blood flows.

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

5. After 7-10 days it is determined that the alterative-proliferative processes are exacerbated and turn into atelectatic pneumonia. It results in pneumosclerosis, bronchiectasis, and the development of retinal cysts of the bronchi. Often, connective tissue grows in place of atelectasis and sclerosis develops.

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