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# FREQUENCY OF NEONATAL ATELECTASIS OF THE LUNG AND MORPHOLOGICAL CHANGES

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**Abstract**
The aim of this study was to address the problems of lung tissue changes in atelectasis. Primary atelectasis of the lungs enters the respiratory distress syndrome and appears as a separate nosological unit. In the article, specific pat morphological signs of primary lung atelectasis are studied. As a material, the lungs of babies who died of atelectasis in the neonatal period were studied microscopically. In the results of the microscopic examination, it was found that the lung tissue has an underdeveloped appearance at first glance.
It is observed that the tissue between the alveoli consist of dense tissue and cellular tufts, the blood vessels are wide and full, and it has a structure around which blood clots have appeared. Macrophages, neutrophils and migrated alveolocytes are detected in the alveolar space. After 7-10 days, it is determined that alterative – proliferative processes escalated and turned into atelectatic pneumonia. As a result, pneumosclerosis, bronchiectasis, and the transformation of bronchi into retention cysts are observed. Often, at the site of atelectasis, connective tissue grows and sclerosing develops.

**Keywords**: Newborn infants, atelectasis, respiratory failure syndrome.

## 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].
In the early neonatal period, pulmonary atelectasis is a manifestation of respiratory failure syndrome (RFS), which often leads to acute respiratory failure. Lung atelectasis in infants is a primary failure to open lung alveoli or alveolar collapse in the first two days of life due to the specific structure of the lungs and insufficient development of the central system of respiratory

regulation. Infantile atelectasis is a part of the "Respiratory Failure Syndrome". The relevance of this problem for paediatrics is due to the large number of causes of lung tissue deterioration in children of one month old. According to the World Health Organization (WHO), its incidence averages 1% of all newborns, and pulmonary atelectasis occurs in up to 14% of cases in premature infants with a body weight of less than 2500 g. Atelectasis occurs in 65% of babies born before 30 weeks gestation who did not receive prenatal prophylaxis with steroid hormones, 35% of those who received prenatal prophylaxis, 25% and 10% of those born at 30-34 weeks, respectively. RFS, including surfactant deficiency as a risk factor for the development of pulmonary atelectasis and all conditions leading to premature lung development, including: neonatal and fetal asphyxia, morphofunctional failure, impaired cardiopulmonary adaptation, pulmonary hypertension, metabolic disorders, i.e., acidosis, hypertension, metabolic disorders, etc. i.e. acidosis, hypoproteinaemia, hypofermentosis, electrolyte metabolism disorders; diabetes mellitus, haemorrhage in pregnant women, caesarean section, second births of twins.

**The aim of the research**

Analysis of risk factors for pulmonary atelectasis in infants, determination of pathomorphological changes developing in the focus of atelectasis depending on pregnancy age, life after birth, and evaluation of diagnostic value in "respiratory failure syndrome".

## MATERIALS AND METHODS

Autopsy data of 52 preterm and 34 preterm infants who died of pulmonary insufficiency during 2020-2022 were obtained as material. First, the medical history and autopsy report of the infants were analysed. The preterm infants were divided into the following groups according to weeks of gestation: Group 1, 12 (23.1%) 22-27 weeks; Group 2, 18 (34.6%) 28-32 weeks; Group 3, 22 (42.3%) babies born at 33-37 weeks who died of pulmonary insufficiency (Table 1). Of these, group 1, i.e., 22-27-week-old, profoundly premature infants with very low birth weight who died within 1 hour of birth, was taken as a control group. The aim was to identify the first morphological changes in the lungs of this group of children that led to the formation of atelectasis. The main cause of death in this group was postnatal asphyxia. Prenatal and intrapartum risk factors from the mother's side, pathologies from the placenta and umbilical cord were identified in all cases. Anthropometric parameters of the examined children are presented in Table 1.

**Table 1 Anthropometric indicators of premature infants, M±m**

| № | Group of premature infants | N | Time of pregnancy | Body weight, g | Height, cm |
|---|---|---|---|---|---|
| 1 | 22-27 weeks | 12 | 25,2±0,4 | 654±24,3 | 29,5±1,6 |
| 2 | 28-32-weeks | 18 | 29,8±0,6* | 1067±84,7* | 38,4±4,6* |
| 3 | 33-37-weeks | 22 | 35,3±0,7** | 1986±124,6** | 43,2±8,5** |

Appendix: * - P≤0.05 - reliability of the difference in pregnancy term, body weight and height in group 2 compared to group 1.

** - P≤0.05 - reliability of the difference in pregnancy term, body weight and height in group 3 compared to group 1

According to the results of clinical and anamnestic analysis, the shortest-lived children and increasing cardiorespiratory failure as a cause of death were observed in the first group of children, and those who lived relatively long - in the 3rd group.

Pieces from different parts of both lungs were taken for histological examination at pathological autopsy. Macroscopic appearance of the lungs was assessed by examining their appearance at autopsy. Lung sections were fixed in 10% neutral formalin, denatured and embedded in paraffin. Histological sections were stained in hematoxylin-eosin, Periodic Acid - Schiff (PAS) reaction and by Van Gieson. The following morphometric calculations were performed.

## RESULTS AND DISCUSSION

Externally, areas of lung atelectasis look like flesh, lung lobes are significantly smaller than their normal size and differ in greyness compared to the surrounding healthy tissue. Foci of atelectasis have been found to have a small appearance, dark red in colour, resembling foci of infarction.

If primary atelectasis develops 2-3 days before the death of babies, the development of inflammation in the lung tissue is observed. As a result, macrophages, neutrophils and migrated alveolocytes are found in the alveolar space. After 7-14 days it is determined that the alterative-proliferative processes have aggravated and passed into atelectatic pneumonia. As a result, pneumosclerosis, bronchiectasis, transformation of bronchi into retention cysts are observed (Fig. 1). It is often determined that the pulmonary vascular system is restored due to connective tissue overgrowth and sclerosing at the site of atelectasis (Fig. 2). If atelectasis foci develop in the subpleural region of the lung, they develop in the posterior basal segments of the lung, and morphologically, partial recession of alveoli and surrounding pathomorphological changes develop to varying degrees.

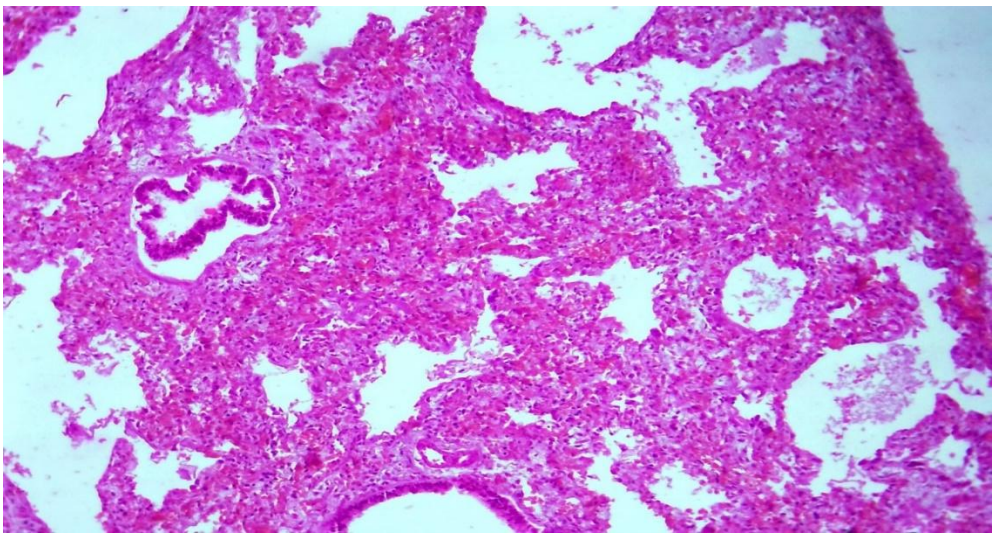

**Fig. 1.** Irregular formation of lung tissue, appearance of various cavities and bronchiectasis. Stain: van Gieson method. Size: 10x40.

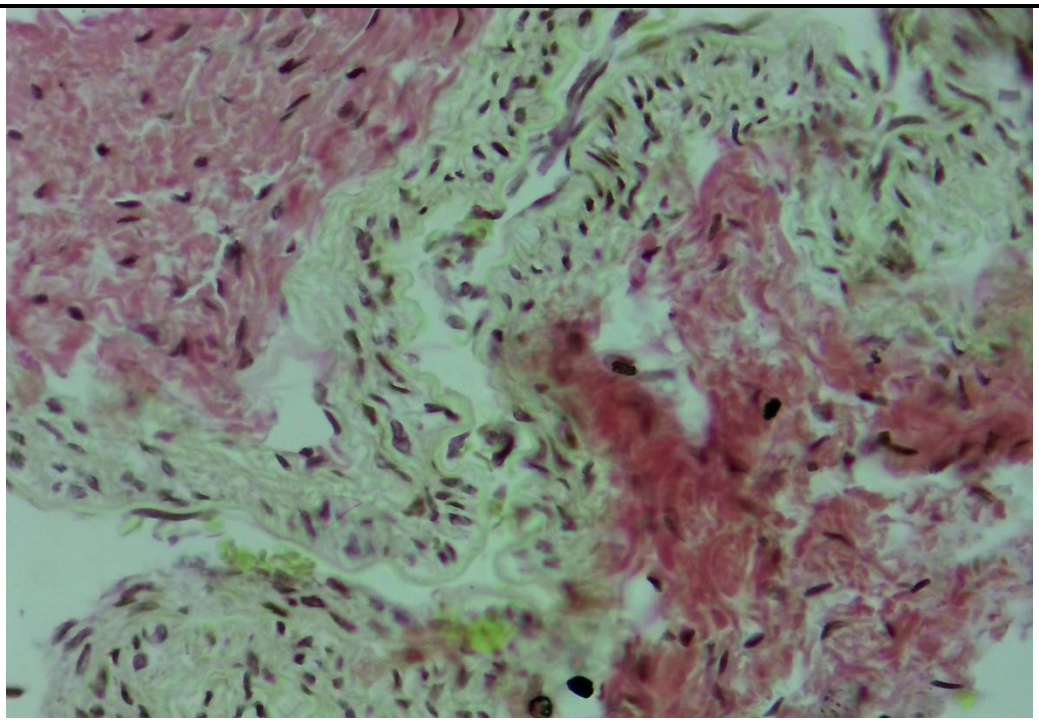

**Fig. 2.** Connective tissue overgrowth in the focus of atelectasis**.** Stain: van Gieson method. Size: 10x40.

The results of microscopic examination showed that the lung tissue appeared underdeveloped at first glance when examined through a small microscope lens. In the lung tissue only bronchi and bronchioles have tubular structures of various sizes and irregular shape. The reticular appearance of the alveolar tissue is not detectable. Respiratory alveoli and communicating spaces are not identified. Alveolar interstitial tissue consists of densely packed tissue and cell bundles (Fig. 3). In such dense lung tissue, wide and full blood vessels are observed, and it has a structure around which thrombi have formed.

When examined under a large microscope objective, it is determined that the bronchial wall consists of only poorly developed tissue structures. It is determined that only one and a few layers of disorderly epithelial cells make up the bronchiolar wall. It is determined that there is a thin tissue surrounding it consisting of dense young histiocytic and lymphoid cells. However, it is not determined whether an alveolar space has appeared in this delicate and young fibre. Therefore, because the lung tissue is young and immature, it is determined that the alveoli are undifferentiated and consist of dense tissue in the form of primary atelectasis and without alveolar space (Figure 4).

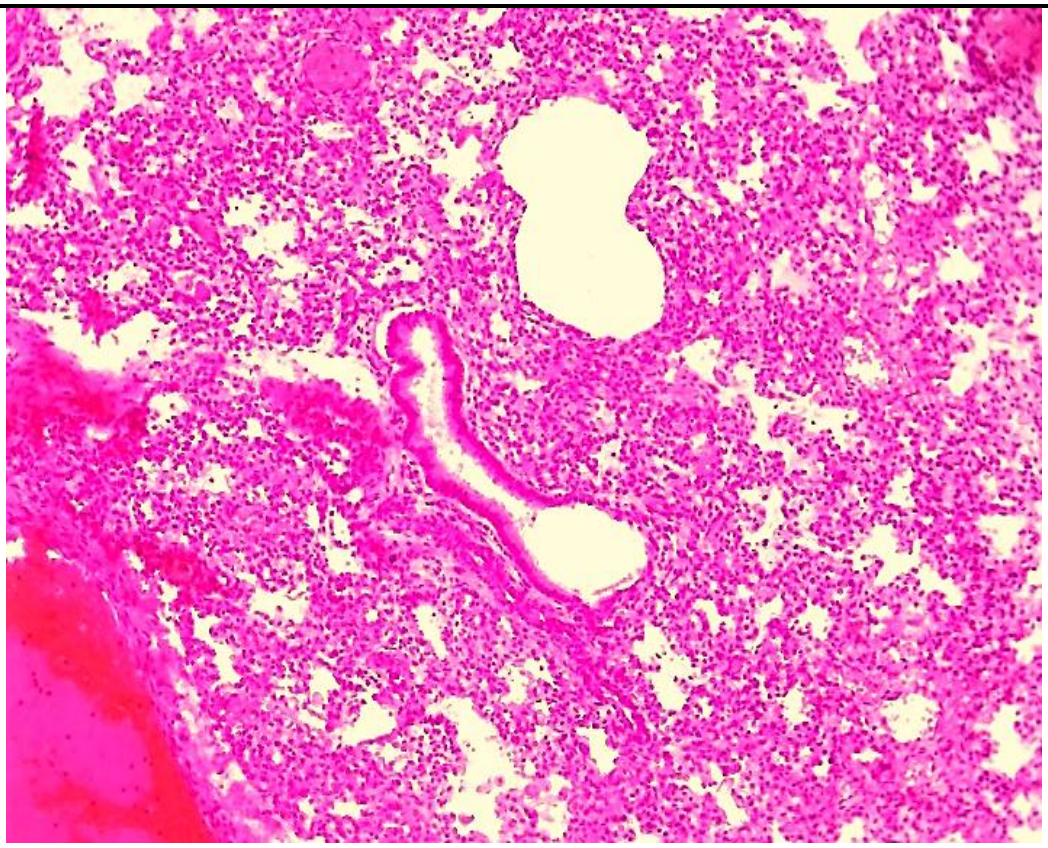

**Fig.3.** Alveolar interstitial tissue consists of densely packed tissue and cell bundles. Stain: van Gieson method. Size: 10x40.

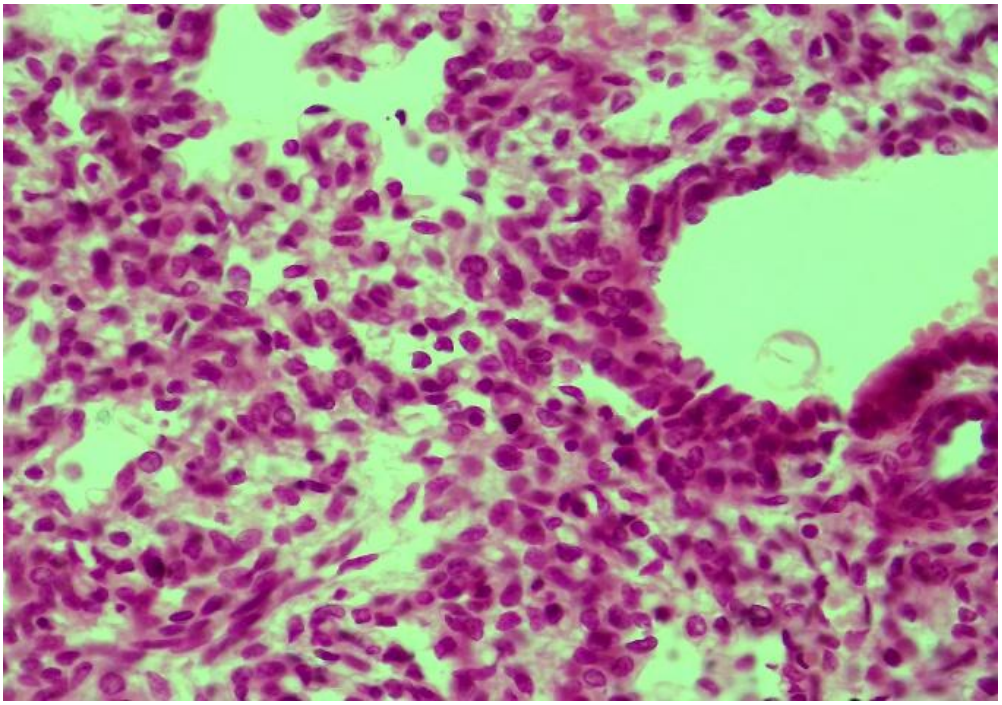

**Fig. 4.** Foci of primary atelectasis are defined as dense tissue without alveolar space. Stain: van Gieson method. Size: 10x40.

In infants who developed primary atelectasis in the lung tissue and died after 2-4 days, microscopic examination of the lung tissue revealed that the respiratory part of the lung and the alveolar space appeared in the form of small fissures. In the structure of the tissue between these spaces, it is determined that histiocytic cells characteristic of proliferative inflammation are activated and hypertrophied, and a dense proliferative inflammatory infiltrate is formed (Fig.5). Macrophages, giant cells and lymphoid cells appear in this inflammatory infiltrate. In the respiratory bronchioles and alveolar part of the lung, the overgrowth of epithelium, including alveolocytes, and the formation of a number of structures are determined.

After the development of primary atelectasis in the lung tissue of young children who died in 7-10 days, microscopic examination of lung tissue revealed that the alveolar tissue thickens and resembles the spleen tissue without voids. Only in places is determined the appearance of spaces of indeterminate shape, in the space of which migrated epithelial and inflammatory cells. In fact, it is determined that the tissue giving rise to the lung tissue, i.e. the tissue of the alveolar space, is sharply thickened and consists of unformed connective tissue (Fig. 6).

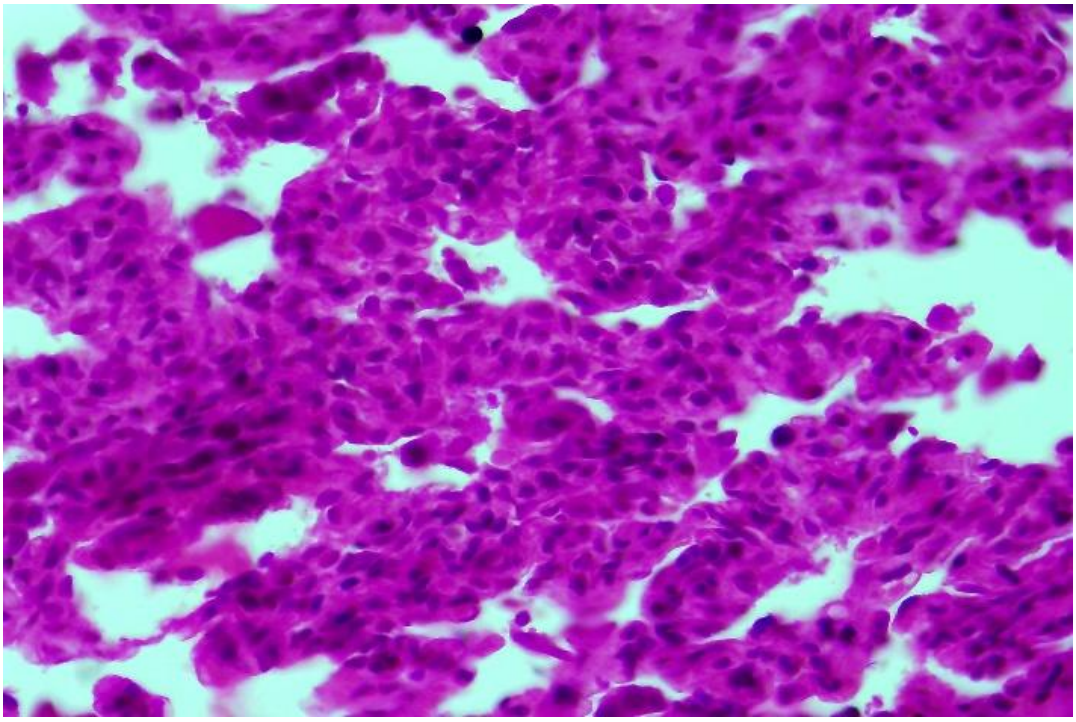

**Fig. 5**. Hypertrophy of proliferative-inflammatory histiocytes, formation of dense proliferative-inflammatory infiltrate between alveoli. Stain: van Gieson method. Size: 10x40.

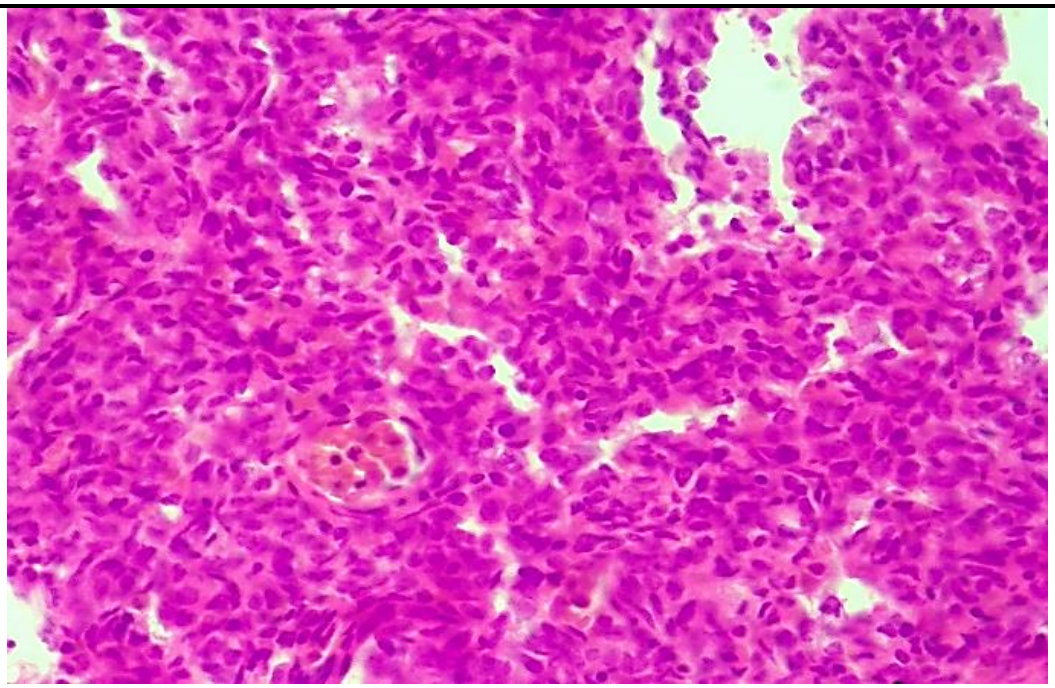

**Fig. 6.** Connective tissue overgrowth in the alveolar tissue, i.e. the appearance of sclerotic strands. Stain: van Gieson method. Size: 10x40.

## CONCLUSION

1. neonatal pulmonary atelectasis is part of the "respiratory distress syndrome (RDS)" and has an overall incidence of 1% of all newborns and 14% in preterm infants. The relevance of atelectasis to paediatrics is that there are many reasons why alveolar lung tissue deteriorates at one month of age.
2. On microscopic examination, the lung tissue at first glance has an underdeveloped appearance. Only bronchioles and bronchioles of tubular structure of various sizes appear in the lung tissue. The reticular appearance of the alveolar tissue is not detected. Respiratory alveoli and communicating spaces are not identified.
3. It is observed that the tissue between alveoli consists of dense tissue and cell bundles, blood vessels are wide and full blooded, has a structure with blood clots around.
4. If primary atelectasis develops 2-3 days before neonatal death, there is inflammation in the lung tissue, i.e. macrophages, neutrophils, migrated alveolocytes are found in the alveolar cavity.
5. After 7-10 days, it is determined that the alterative-proliferative processes have developed into atelectatic pneumonia. As a result, pneumosclerosis, bronchiectasis, transformation of bronchi into retention cysts are observed. Often at the site of atelectasis connective tissue overgrows and sclerosis develops.

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