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Abstract. Infant mortality caused by incomplete differentiation of the lung parenchyma in newborns today reaches high percentages. This, in turn, requires a deeper study of pneumopathies and their pathomorphological aspects, as well as a new medical approach. The materials obtained from the immature structures of the lungs during pathological-anatomical autopsy investigation make it possible to understand in detail the macroscopic and microscopic symptoms [1, 2].

Keywords: fetus, surfactant, ischemiya, micropreparation, parenchyma, pulmon, dysphonia, apnoe, dyspnoe, bradypnoe.

Аннотация. Младенческая смертность, вызванная неполной дифференциацией паренхимы легких у новорожденных, сегодня достигает высоких процентов. Это, в свою очередь, требует более глубокого изучения пневмопатий и их патоморфологических аспектов, а также нового медицинского подхода. Материалы, полученные из незрелых структур легких при патологоанатомическом вскрытии, позволяют детально разобраться в макроскопических и микроскопических симптомах [1, 2].

Ключевые слова: фетус, сурфактант, ишемия, микропрепарат, паренхима, пульмон, дисфония, апноэ, диспноэ, брадипноэ.

INTRODUCTION: Despite the current advances in medicine, microscopic examination of the lungs of newborns who died from respiratory distress syndrome has revealed acute pulmonary failure caused by morphological changes such as primary widespread atelectasis and the presence of hyaline membranes. In addition, respiratory distress syndrome in newborns is considered one of the most common and still insufficiently studied severe pathological processes during the neonatal period, especially in premature infants [3,5].

In CIS countries, the pathomorphological types of pneumopathy include the formation of hyaline membranes, aspiration of amniotic fluid into the respiratory tract, diffuse atelectasis, and extensive hemorrhages into the lung parenchyma. According to literature sources, respiratory distress syndrome is divided into two types. The first type includes the presence of hyaline membranes. In this type, premature infants account for 50–70% of neonatal deaths in the United States. The second type includes aspiration syndrome, diffuse atelectasis, and massive hemorrhaging into the lung parenchyma. Pneumopathy is classified into nosological forms such as primary pulmonary atelectasis, hyaline membranes, extensive pulmonary hemorrhage, and aspiration syndrome [4].

RESEARCH OBJECTIVE: To study the morphological and morphometric changes in organs associated with pneumopathy in newborns.

MATERIALS AND METHODS: A total of 72 cases involving newborns weighing between 500–999 grams and born at gestational ages of 22–28 weeks were analyzed during autopsy. The study used morphological, autopsy, morphometric, and microscopic examination methods.

RESEARCH RESULTS: Structural changes in the cerebral cortex and medulla oblongata nerves

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and blood vessels were evaluated in newborns with the atelectatic form of pneumopathy who died at different time intervals after birth.

- The dynamics of structural changes in the cerebral cortex and medulla oblongata nerves and vessels were described in newborns who died at different time points with the atelectatic form of pneumopathy.
- Morphological and morphometric differences in the cerebral cortex and medulla oblongata based on the duration of postnatal survival were revealed.
- Based on the obtained results, the causes of death in pneumopathy cases were substantiated, a comparative assessment of the role of these changes in thanatogenesis was made, and preventive measures for pneumopathy cases were developed.

It was substantiated that the manifestation of thanatogenesis in the cerebral cortex and medulla oblongata of newborns who died with the atelectatic form of pneumopathy does not vary depending on gestational age. It was found that neuronal development in the atelectatic form of pneumopathy remains unchanged from early to late gestational periods. However, morphometric indicators of neurons in the medulla oblongata increase with gestational age, and as gestational age increases, destructive changes in the cerebral cortex and medulla oblongata—such as increased neuron numbers, vascular components, as well as perineuronal and perivascular spaces—are linked to postmortem duration.

It was substantiated that in the atelectatic form of pneumopathy, the increase in size of neurons and blood vessels in the medulla oblongata, the reduction in pericellular and perivascular spaces, and the increase in the number of altered neurons and blood vessels are all correlated with the duration of postnatal life. Ischemic-type changes in the neurons of the medulla oblongata, expansion of perineuronal spaces, increased permeability of blood vessel walls resulting in diapedesis hemorrhages and widening of perivascular spaces, represent constant features of thanatogenesis. However, it was shown that as survival time increases, ischemic changes in the medulla oblongata decrease, which supports the increased survival potential in cases of the atelectatic form of pneumopathy.

In CIS countries, the morphological manifestations of pneumopathy most frequently include the presence of hyaline membranes, aspiration of amniotic fluid into the airways, diffuse atelectasis, and massive hemorrhaging into the lung tissue. According to literature, respiratory distress syndrome is divided into two types. The first type includes hyaline membranes and accounts for 50–70% of neonatal deaths in the U.S. due to unviable preterm births. The second type includes aspiration syndrome, diffuse atelectasis, and massive pulmonary hemorrhages. These forms of pneumopathy are classified as non-infectious pathological processes in the lungs.

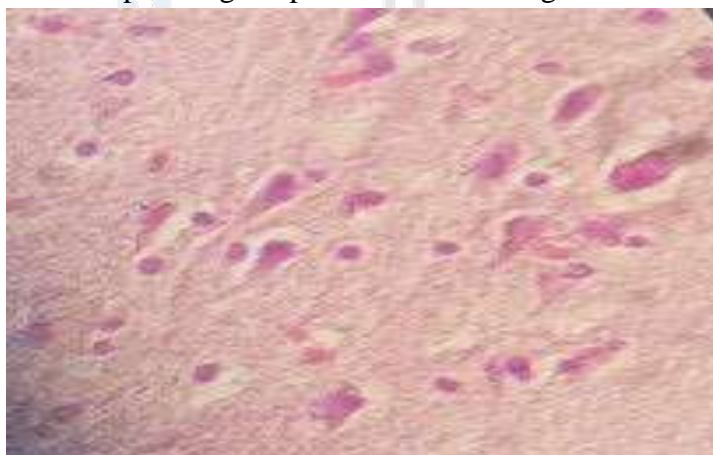


Figure 1. Condition of brain cells and blood vessels in the atelectatic form of pneumopathy. Stained with hematoxylin and eosin.

The research results indicate that in cases of neonatal death from the atelectatic form of pneumopathy, morphological and morphometric examinations of cranial structures revealed a ratio of vascular to perivascular space involvement.

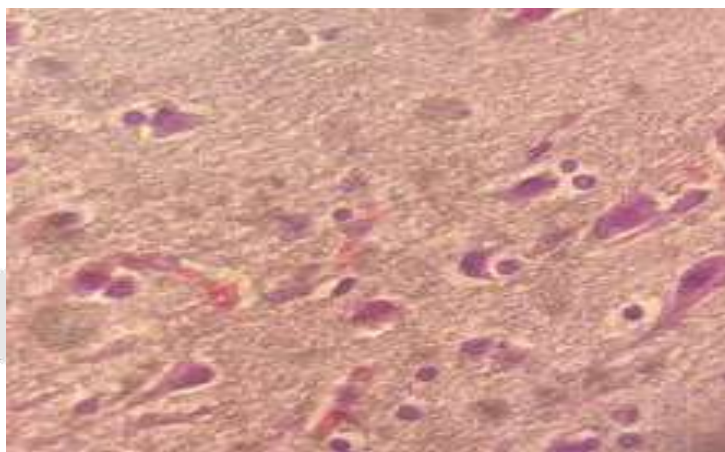


Figure 2. Condition of brain cells and blood vessels in the atelectatic form of pneumopathy. Stained with hematoxylin and eosin.

Morphometric indicators of cortical neurons in the brains of infants born and deceased at different stages of gestation due to the atelectatic form of pneumopathy varied. Morphological changes of the ischemic type and glial reactions were more prominently expressed in later stages of gestation. Compared to blood vessels, ischemic changes were more frequently observed in neurons.

In the atelectatic form of pneumopathy, the degree of expansion in perineuronal and perivascular spaces in the cerebral cortex and medulla can serve as a metric to assess the progression of thanatogenesis over time, as their dimensions increase in parallel. The dynamics of destructive changes in neurons and blood vessels of the brain and medulla also provide an additional criterion for evaluating the extent of pathological progression.

CONCLUSION: In the context of the atelectatic form of pneumopathy, the use of the ratio of perineuronal and perivascular space sizes in the cerebral cortex and medulla as indicators for assessing the progression of thanatogenesis over different survival periods has been improved. The final conclusion of the study explains the contribution of morphological changes in brain structures to the death of newborns with the atelectatic form of pneumopathy, by providing a comparative analysis based on their duration of survival. These findings expand on existing knowledge and highlight the critical role of these morphological markers in understanding compensatory mechanisms during cerebral hypoxia. Moreover, the study emphasizes that respiratory distress syndrome predominantly manifests in all cases of pneumopathy [1,6,7,8].

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