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Morphological characteristics of brain structures in premature infants of different periods with atelectatic form of pneumopathy

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Abstract

Premature birth remains a pressing public health issue, with approximately one in every ten pregnancies in the United States ending in preterm delivery. Globally, an estimated 15 million infants are born prematurely each year, and tragically, nearly one million of these newborns die within the first day of life. Despite notable advancements in perinatal and neonatal care, respiratory distress syndrome (RDS) continues to be one of the leading causes of perinatal morbidity and mortality. RDS affects not only premature infants but can also occur in full-term neonates. One particularly severe and often fatal manifestation of RDS is the atelectatic form of pneumopathy—a non-inflammatory lung condition characterized by incomplete lung expansion and alveolar collapse. This form of pneumopathy leads to significant systemic complications, including profound effects on the central nervous system. The current study explores the morphological and morphometric alterations in the brains of 138 neonates who died due to the atelectatic form of pneumopathy. Through detailed histopathological and morphometric analyses, the research focuses on assessing the extent of neuronal degeneration, proliferation of glial cells (gliocytes), vascular irregularities, and expansion of the perivascular spaces. These findings underscore the detrimental impact of severe pulmonary dysfunction on the developing brain. By shedding light on the neuropathological consequences of neonatal RDS, the study offers valuable insights that could inform future therapeutic approaches and preventive strategies in neonatal intensive care.

Keywords: Distress syndrome, atelectasis, distelectasis, pneumopathy, hyaline membrane

Introduction

Respiratory distress syndrome (RDS) is more prevalent in preterm infants and ranks as the second most common perinatal pathology. The morphofunctional characteristics of the respiratory system are crucial in the development of this syndrome. Research indicates that genetic and multifactorial elements contribute to its manifestation [1,2]. Autopsy studies of neonates who succumbed to respiratory disorders reveal pulmonary alterations, including atelectasis foci and hyaline membranes, without an inflammatory response. This study aims to analyze the morphological and morphometric characteristics of brain structures in neonates affected by the atelectatic form of pneumopathy.

Material and Methods

Histopathological and morphometric examinations were performed on 138 neonates who died from the atelectatic form of pneumopathy at the Samarkand Regional Perinatal Center between 2019 and 2023. The study population included 39 infants born at 22-25 weeks, 34 at 26-28 weeks, and 22 at 29-31 weeks of gestation. Standard hematoxylin-eosin and Van Gieson staining methods were employed to assess neuronal and vascular alterations [3,4]. Morphometric analysis was performed to quantify neuronal body dimensions and perineuronal/perivascular space expansion. Statistical analysis was performed using SPSS software, and comparisons between different gestational periods were analyzed using ANOVA and student's t-tests. Data were expressed as mean ± standard deviation, with p-values <0.05 considered statistically significant. The study was conducted in accordance with the Helsinki Declaration. Data were collected with the approval of the Ethics Committee of the Samarkand Regional Perinatal Center. Due to the retrospective nature of the study, informed consent was not obtained from the patients or their relatives.

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Results

We studied the morphological and morphometric features of the structures of the brain of newborns by pathologist examination of the corpses of 138 newborns.

A total of 39 children were born in the period of 22-25 weeks, of which 26 (66.6%) were boys, and 13 (33.4%) were girls. During this period, macroscopical examination of the brains of newborns who died due to the atelectatic form of pneumopathy revealed fullness and swelling of the soft membrane. Neurons of the cerebral hemispheres do not have a specific pyramidal shape, the number of pyramidal cells is small, they are scattered far from each other. Neurons are characterized by dystrophic changes, i.e. eccentric enlargement of nuclei, pyknotic state, dystrophy with cytoplasm and vacuoles. Signs of bending, shortening of barriers and thickening were observed in neurons. In neuron atrophy, a small amount of satellitosis is detected. Weak swelling was observed in the atrophy of neurons. Neuropil sharply matured. It was observed that in neuron atrophy, gliocytes increased in number and grew close to each other. Nuclei are enlarged in the center, cytoplasm is bright, nuclei of some gliocytes are not aniculated. Their atrophy is determined by the widening of the perivascular space (Figures 1 and 2).

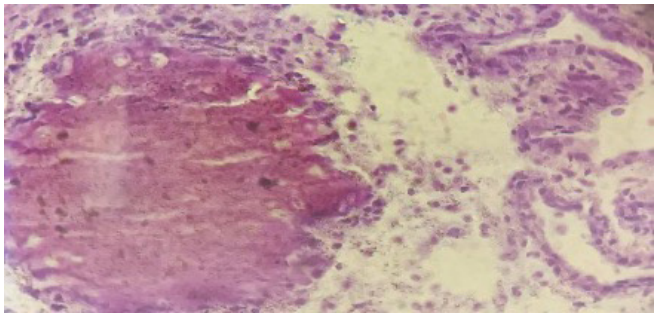

Figure 1. Fullness and swelling of the soft membrane of the brain in the atelectatic form of pneumopathy; Stained with hematoxylin-eosin method; The picture enlarged - 40x10

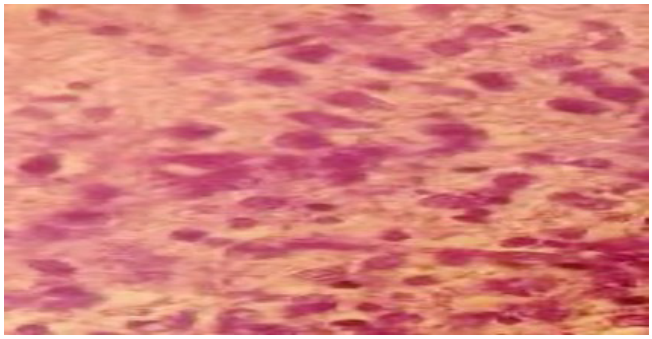

Figure 2. Pneumopathy in the form of atelectatic changes in neurons and glial cells of the cerebral cortex, satellitosis; Stained with hematoxylin-eosin method; The picture enlarged - 40x10

During this period (22-25 weeks) severe spasm and dystonia of cerebral blood vessels are detected. Elements of the blood form are present in the vascular cavity, and the shape of erythrocytes has not been lost. Signs of strong swelling are noted on the wall. Endotheliocytes are scattered, the kernels of some endotheliocytes are not anicized. The perivascular space is expanded on all sides of the blood vessel. Shaped elements were present in the cavity of microcirculatory veins, and a state of stasis was observed. The shape of individual erythrocytes is not defined, the capillary wall is completely broken, the transfer of erythrocytes to the intercellular space and interstitial tissue (diapedesis) is noted (Figure 3).

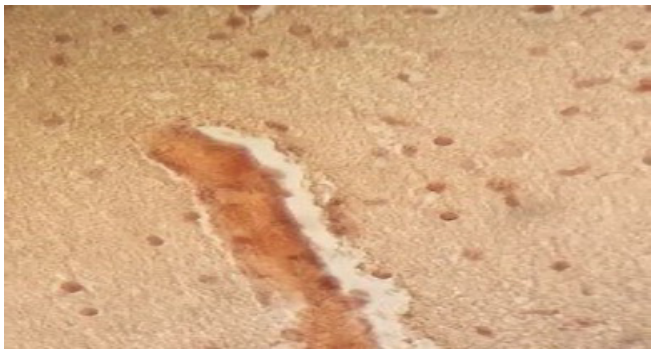

Figure 3. Expansion of the perivascular space in cerebral cortical cavity vascular atrophy in the atelectatic form of Pneumopathy; Stained with Van-Gison method; The picture enlarged - 40x10

As can be seen from the data of Table 1, the length of the body of the neurons of the cortex of the cerebral hemispheres of the newborns who died from the atelectatic form of pneumopathy was on average 0.34±0.01 mm, while its width was 0.16±0.01 microns. The average size of the length of neuron fences was 0.38±0.01 μm, and the width was 0.14±0.01 μm. The morphometric indicators of the areas occupied by neurons and blood vessels in the brains of the elderly who died from the atelectatic form of pneumopathy are presented in the table.

Table 1. Morphometric indicators of brain neuron bodies and bundles of newborns who died from the atelectatic form of pneumopathy, μm

No.	Status	Structure	Measurements (μm)
1	Pneumopathy	The height of the neuron body	0.34±0.01
2		The width of the neuron body	0.16±0.01
3		The height of the neuron growth	0.38±0.01
4		The width of the neuron growth	0.15±0.01

It can be seen from the data of Table 2 that the area occupied by neurons in the cortex layer of the brain of the dead from the atelectatic form of pneumopathy is 39%, and the area occupied by the perineuronal space is 61%. The area occupied by the vessel was 63%, and the percentage of perivascular space was 37%. This shows that the gap in neuron atrophy is more widening than the gap in vascular atrophy.

Table 2. The ratio (%) of brain neuron and perineuronal spaces (PNS), vascular and perivascular space (PVS) in newborns who died from the atelectatic form of pneumopathy

Status	Nerve tissue		
	Neuron	PNS	Neuron+PNS
Pneumopathy	1.56±0.08	2.48±0.08	4.04±0.16
	Mine vein structure		
	Mine vessel	PVS	Mine vessel+PVS
	1.59±0.08	2.69±0.1	4.28±0.18

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There are 34 babies born in the period of 26-28 weeks, of which 22 (64.7%) are boys, and 13 (35.3%) are girls. During this period, the cerebral hemispheres have a number of pyramid-shaped cells, which are close to each other. Dystrophic changes (eccentric enlargement of nuclei, pyknotic state, vacuolar dystrophy in cytoplasm) are detected in neurons. Neurons showed signs of fragmentation, shortening and thickening of their few barriers. In neuron atrophy, a small amount of satellitosis is detected. Weak bending of neurons was observed in atrophy.

Neuropil sharply matured. It was observed that in neuron atrophy, gliocytes increased in number and spread close to each other, and a group of gliocytes spread side by side at a uniform distance. The nuclei are enlarged in the center, the nuclei of some gliocytes are pushed aside, the cytoplasm is lightened, the kernels of some gliocytes are not differentiated. In their atrophy, it was noted that the perivascular space was fully expanded (Figure 4).

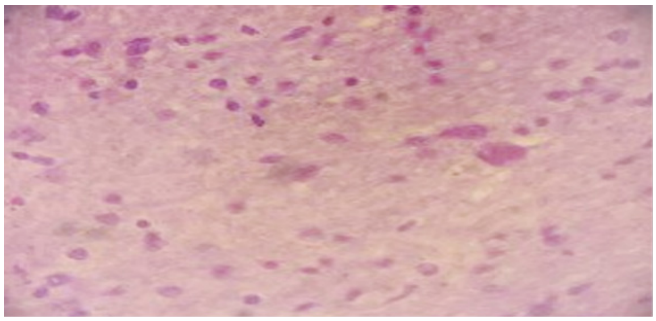

Figure 4. The changes in atelectatic form of Pneumopathy in neurons and glial cells of the cerebral cortex, satellitosis; Stained with hematoxylin-eosin method; The picture enlarged - 40x10

During this period (26-28 weeks) severe spasm and dystonia of blood vessels of the brain were preserved. There are elements of the blood form in the vascular cavity, and the shape of erythrocytes is specific. Signs of severe swelling were noted on the wall (Figure 5).

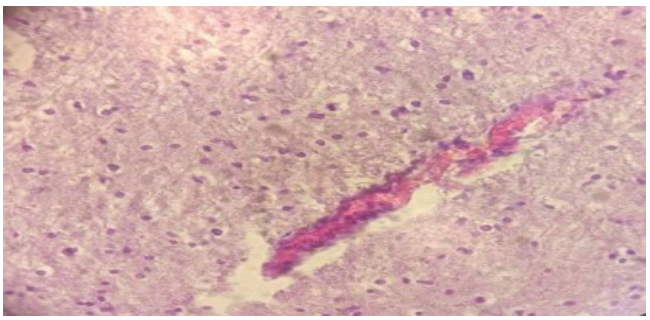

Figure 5. Expansion of the perivascular space in cerebral cortical cavity vascular atrophy in the atelectatic form of Pneumopathy; Stained with hematoxylin-eosin method; The picture enlarged - 40x10

Endotheliocytes are scattered, the cytoplasm is lightened (vacuolar dystrophy), karyopyknosis cusatild in many keratoses. Karyorrhexis was noted in some endotheliocytes. Core is not detected in some cells. Perivascular swelling in the space of blood vessels of the microcirculatory system there are shaped elements, and a state of stasis is observed. The shape of individual erythrocytes is not determined, the integrity of the capillary wall is broken, it is noted that erythrocytes move to the intercellular space and interstitial tissue.

As can be seen from the data presented in Table 3, the body size of the neuron bodies of the cerebral hemispheres of the newborns who died from the atelectatic form of pneumopathy is on average 0.34±0.01 μm, and its e is on average 0.16±0.01 microns. The average size of the length of the neuron fences was 0.38±0.01 mm, and the width was 0.15±0.02 mm. The morphometric indicators of the areas occupied by neurons and blood vessels in the brains of obese people who died from the atelectatic form of pneumopathy are presented in the table.

Table 3. Morphometric indicators of neuron bodies and bundles of medulla oblongata of newborns who died from the atelectatic form of pneumopathy, μm

No.	Status	Structure	Measurements (μm)
1	Pneumopathy	The height of the neuron body	0.34±0.01
2		The width of the neuron body	0.16±0.01
3		The height of the neuron growth	0.38±0.01
4		The width of the neuron growth	0.15±0.02

From the data presented in Table 4, it can be seen that the area occupied by neurons in the hollow cavity of the cerebrum of jackals who died from the atelectatic form of pneumopathy is 38%, and the area occupied by perineuronal bush is 62%. The area occupied by the vessel is 38%, and the percentage of perivascular space is 62%. This shows that the gap in neuron atrophy is more widening than the gap in vascular atrophy.

Table 4. The ratio (%) of brain neuron and perineuronal spaces (PNS), vascular and perivascular space (PVS) in neonates who died from the atelectatic form of pneumopathy

Status	The structure is nerve		
	Neuron	PNS	Neuron+PNS
Pneumopathy	1.68±0.08	2.79±0.12	4.47±0.2
	Mine vein structure		
	Mine vessel	PVS	Mine vessel+PVS
	1.73±0.07	2.88±0.11	4.61±0.18

There are 22 children born in the period of 29-31 weeks, 21 of them (63.6%) are boys, and 11 (36.4%) are girls. During this period, the number of pyramidal cells in the cerebral hemispheres, the distance between them, is reduced. Changes such as eccentric nucleus enlargement, pyknotic condition, karyorrhexis condition in some neurons, dystrophy with vacuoles in the cytoplasm were noted.

Discussion

Our findings indicate a progressive pattern of neuronal and vascular degeneration in neonates with atelectatic pneumopathy, which aligns with previous studies highlighting the impact of hypoxia and respiratory distress on brain structures [5,6]. The most pronounced neuronal damage was observed in neonates born at 22-25 weeks, while those born at later stages exhibited slightly improved cellular organization but persistent dystrophic changes.

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The degree of vascular pathology correlated with the severity of neuronal damage, suggesting a link between hypoxic-ischemic injury and pneumopathy [7,8]. Gliocyte proliferation and satellitosis may represent early neuroprotective responses, as suggested in other neuropathological studies [9-11]. Additionally, the expansion of perineuronal and perivascular spaces is indicative of chronic hypoxic damage, which has been extensively documented in neonatal neuropathology research [12,13].

Despite these findings, certain limitations should be considered. The study is limited by the retrospective nature of the data and the relatively small sample size. Further research with larger cohorts and advanced imaging techniques may provide additional insights into the pathophysiological mechanisms involved in neonatal brain damage associated with pneumopathy.

Conclusion

Dystrophic changes in cortical neurons were evident across all gestational periods studied. Gliocyte proliferation and satellitosis suggested ongoing attempts at neuroprotection. Expanding perineuronal and perivascular spaces indicated worsening atrophic processes. Severe vascular dystonia, endothelial swelling, and microcirculatory stasis were hallmarks of pneumopathy-induced cerebral damage. Understanding these neuropathological alterations provides insight into the effects of respiratory distress on neonatal brain development and may guide future therapeutic strategies.

Conflict of Interests

The authors declare that there is no conflict of interest in the study.

Financial Disclosure

The authors declare that they have received no financial support for the study.

Ethical Approval

The study was conducted in accordance with the Helsinki Declaration. Data were collected with the approval of the Ethics Committee of the Samarkand Regional Perinatal Center.

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