https://www.proconference.org/index.php/usc/article/view/usc22-00-011
DOI: 10.30888/2709-2267.2024-22-00-011

УДК 616-053.31, 616.24.

MORPHOLOGY OF BRONCHOPULMONARY DYSPLASIA IN PREMATURE INFANTS

Serikbay Mereily Karmantaevna[1]
Esirgepova Sofya Richardovna[2]
Tazhimetov Bekzat Makhmutovich[2]
Ashirbekov Gamal Karimovich[2]
Kuzatbekova Eligay Bolatbekovna[1]
Stoilov Vladimir Vladimirovich[1]
KazNMU named after S.D. Asfendiyarova, Almaty, Kazakhstan[1]
KRMU, Almaty, Kazakhstan[2]

***Abstract**: A significant part of morphological studies of the lungs in newborns and premature infants is devoted to the study of respiratory distress syndrome, which in 80-82% of cases is the direct cause of death in children in the early neonatal period. A serious complication of SDR and mechanical ventilation with high oxygen concentrations in premature infants is bronchopulmonary dysplasia.*

***Keywords**. bronchopulmonary dysplasia, prematurity, respiratory distress syndrome, hyaline membranes, atelectasis.*

Prematurity is one of the most important health problems worldwide, due to the high mortality and disability of surviving children. Among various pathological conditions, one of the leading places is occupied by respiratory disorders, known as respiratory distress syndrome (RDS, neonatal respiratory distress syndrome), the frequency of which is 30-80%. Bronchopulmonary dysplasia (BPD) is a serious complication of SDR and artificial ventilation with high oxygen concentrations in premature infants (1).

In recent years, BPD has taken the leading place among bronchopulmonary iatrogenies in premature infants. Due to the expansion of indications for mechanical ventilation and growing interest in this pathology, the number of bronchopulmonary dysplasias in the diagnosis structure increases annually (2).

A.V. Bogdanova considers BPD as a chronic disease that develops mainly in premature infants as a result of lung damage during “hard” mechanical ventilation modes with high oxygen concentrations, manifested by tachypnea, dyspnea, hypoxemia, persistent obstructive disorders and characteristic radiological changes (3).

BPD (Bronchopulmonary dysplasia (BPD)) is a chronic lung disease that develops in newborns, predominantly premature infants, during the treatment of respiratory disorders using mechanical ventilation (ALV) with high oxygen concentrations and positive pressure ventilation (4).

Morphological changes in the lungs in BPD were described in 1967. In Norsveem in the form of 4 stages (cited from T.A. Shishko, Yu.A. Ustimovich 2000).

Stage 1 (during the first three days of life) includes interstitial and alveolar pulmonary edema, the presence of “hyaline membranes”, atelectasis, necrosis of the bronchiole epithelium.

Stage 2 (4-10 days of life) is characterized by more widespread atelectasis, alternating with areas of emphysema, widespread necrosis of the bronchiole epithelium, and the presence of necrotic masses in the airways.

In stage 3 BPD (11-30 days of life), widespread metaplasia and hyperplasia of the epithelium of the bronchi and bronchioles, areas of emphysema with areas of atelectasis, and massive interstitial edema are detected.

In stage 4 (2nd month of life), massive pulmonary fibrosis develops with destruction of alveoli, metaplasia of the respiratory tract epithelium, hypertrophy of the muscular layer of bronchioles, arterioles and venules (5).

One of the significant risk factors is respiratory distress syndrome (RDS), which is diffuse alveolar damage characteristic of premature newborns. It can be primary (various types of pneumopathy) and secondary at a later age of the child. The development of primary RDS is caused by the immaturity of the lungs, namely the inability of type II pneumocytes to synthesize and secrete sufficient amounts of surfactant into the lumen of the alveoli (6).

Purpose of the study: To study the morphological features of the lungs with bronchopulmonary dysplasia in very premature newborns at a gestation period of 22-28 weeks.

Materials and methods. We studied the lungs of 70 of our own observations of deceased newborns with extremely low body weight (ELBW) from 500 to 999 g at a gestation period of 22-28 weeks. The studies were carried out in the children's department of the city pathological-anatomical bureau of Almaty. All newborns from our own observations were on mechanical ventilation. Of these, in 12 newborns with primary atelectasis (5) and hyaline membranes (7), we identified manifestations of bronchopulmonary dysplasia. These newborns had risk factors: long-term mechanical ventilation, EMT, infusion therapy.

Results and discussion. Histological examination of the pulmonary parenchyma revealed signs of immaturity of the lung tissue, relating to both the structure and lining of the alveoli and the state of the vascular system. Severe interstitial and intra-alveolar pulmonary edema, atelectasis and distelectasis, alternating with multiple areas of acute emphysema, were detected in the lungs. Necrobiosis and necrosis of the epithelium of bronchioles and alveoli with massive desquamation of cells were revealed. These changes corresponded to stages 1 and 2 of bronchopulmonary dysplasia

Of the 72 observations, in 10 cases (7.2%) acute pulmonary emphysema with rupture of the interalveolar septa was morphologically diagnosed (Fig. 3, 4). In two cases, bullous emphysema was noted (Fig. 5).

Thus, in the lungs of very premature newborns who were on mechanical ventilation, complications occurred, which, according to the literature, are caused by;

1) a high percentage of oxygen in the inhaled air, which “burns” the child’s lungs, causing a chain of reactions: damage - inflammation - repair due to the proliferation of fibroblasts

2) high values of “peak” pressure (inspiratory pressure) during mechanical ventilation, while barotrauma can also be an inducer of the inflammatory response.

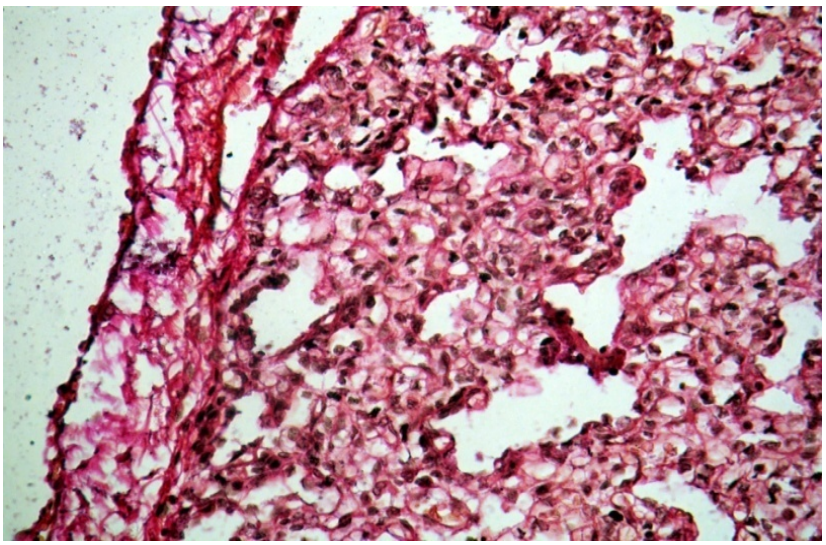

Fig. 1 Interstitial and intra-alveolar edema, Van Gieson stain x 200

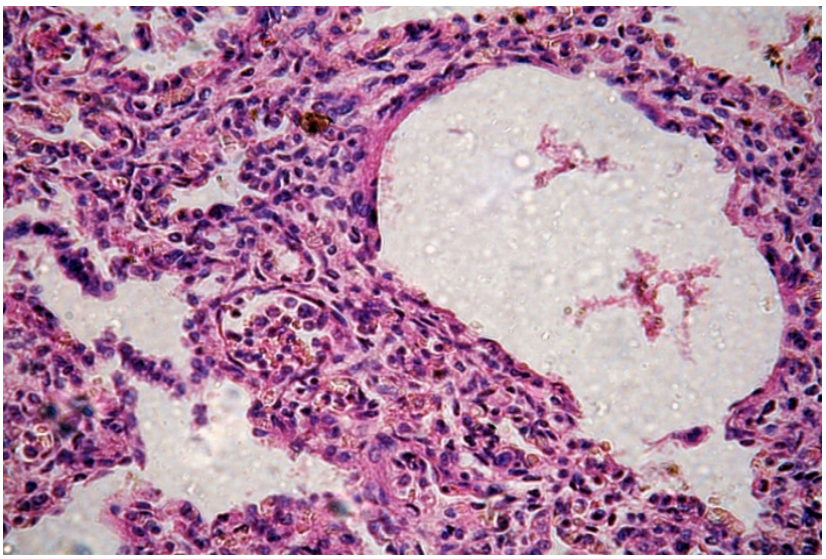

Fig. 2 Dystrophy, necrosis and desquamation of the epithelium of bronchioles, alveoli, staining HE x 200

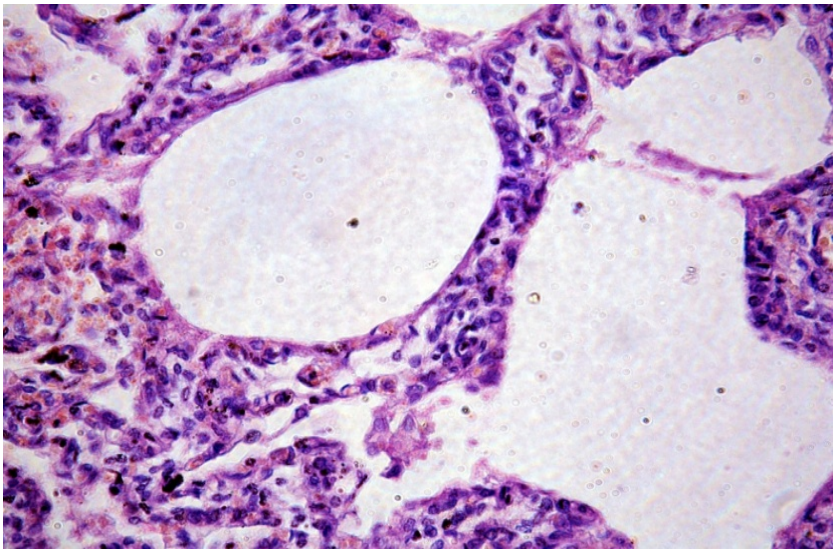

Fig. 3 Acute emphysema, investment edema, rupture of interalveolar septa, color HE x 200

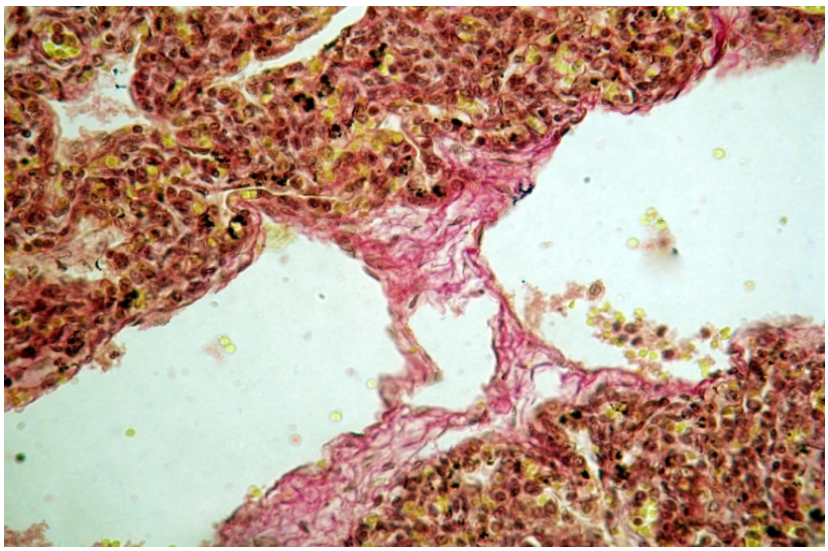

Fig. 4 Intermediate emphysema, Van Gieson stain x200

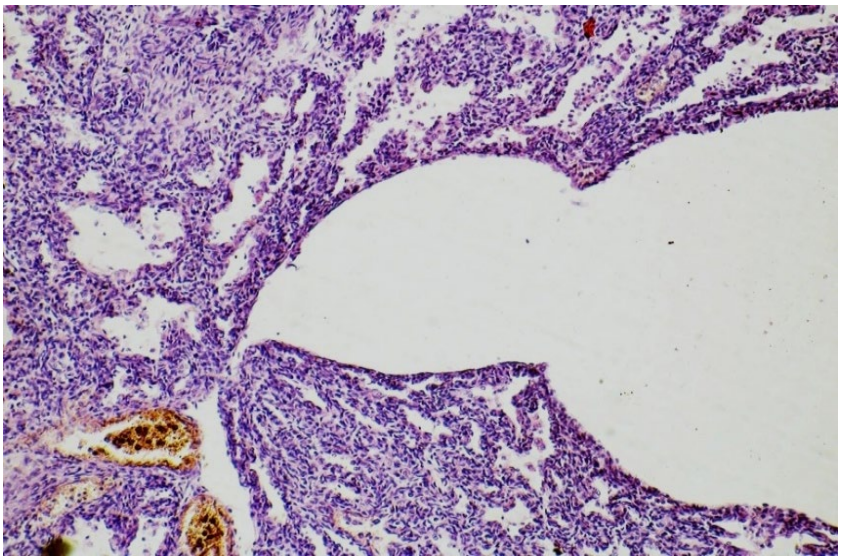

Fig.5 Bullous emphysema, color HE x 100

Therefore, it is necessary to carry out anti-inflammatory therapy in children on mechanical ventilation with strict parameters in the early stages of BPD.

References

1. Ovsyannikov D. Yu. Bronchopulmonary dysplasia in children of the first three years of life: Abstract of thesis. diss. ... doc. honey. Sci. M., 2010. 48 p. [Ovsyannikov D. Yu. Bronkholegochnaya displaziya u detei pervykh trekh let zhizni: Avtoref. dis. ...doct. med. nauk. M.; 2010. 48 s. (in Russian)]

2. Bogdanova A.V. BOYTSOVA S.V. Clinical features and course of BPD. Pulmonology 2002 No. 1 S 28-32.

3. Norhway W.H. Bronchopulmonary dysplasia: then and now // Am. J. Dis. Cild.-1990. – 65. – 819-823

3.Bassler D., Plavka R., Shinwell E. S., Hallman M. et al.; NEUROSIS Trial Group. Early inhaled budesonide for the prevention of bronchopulmonary dysplasia // N. Engl. J. Med. 2015. Vol. 373. N 16. P. 1497–1506.

4. Panchenko A. S., Gaymolenko I. N., Tikhonenko O. A., Ignatieva A. V. Bronchopulmonary dysplasia: causes of formation and morphology of lung tissue // Sib. honey. magazine (Irkutsk). 2013. T. 117. No. 2. P. 61–64. [Panchenko A. S., Gaimolenko I. N., Tikhonenko O. A., Ignat'eva A. V. Bronkholegochnaya displaziya: prichiny formirovaniya i morfologiya legochnoi tkani. Sib. med. zhurn. (Irkutsk). 2013; 117(2): 61–4. (in Russian)]

5. 3. Northway W. H. Bronchopulmonary dusplasia: Then and now. Am. J. Dis. Child.1990; 65:1076-1081

6. Boytsova E. V., Bogdanova A. V., Ovsyannikov D. Yu. Consequences of bronchopulmonary dysplasia for the respiratory health of children, adolescents and young adults // Issues. diagnostics in pediatrics. 2013. T. 5. No. 1. P. 5–11. [Boitsova E. V., Bogdanova A. V., Ovsyannikov D. Yu. Posledstviya bronkholegochnoi displazii dlya respiratornogo zdorov'ya detei, podrostkov i molodykh vzroslykh. Vopr. diagnostiki v pediatrii. 2013; 5(1): 5–11. (in Russian)]