American Journal Of Biomedical Science & Pharmaceutical Innovation
(ISSN – 2771-2753)
VOLUME 04 ISSUE 05 PAGES: 46-55
SJIF IMPACT FACTOR (2022: 5. 705) (2023: 6.534) (2024: 7.7)
OCLC – 1121105677
Publisher: Oscar Publishing Services

Journal Website: https://theusajournals.com/index.php/ajbspi

Research Article

PATHOMORPHOLOGICAL CHANGES IN THE BRONCHI AND LUNG PARANCHYMA IN RESPIRATORY DISTRESS SYNDROME DEPENDING ON THE GESTATION TIME IN NEWBORNS

Submission Date: May 20, 2024, **Accepted Date:** May 25, 2024,
Published Date: May 30, 2024
Crossref doi: https://doi.org/10.37547/ajbspi/Volume04Issue05-08

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ABSTRACT

The aim of this study was to identify the type of pathomorphological alterations in the lung tissue and bronchial wall of neonates that, based on gestational age, died from different types of pneumopathy. The study uses materials from the corpses of 74 newborns who passed away from various forms of pneumopathy in the Samarkand region's perinatal center. The bodies of the newborns were autopsied and studied in the pathoanatomical department of the Samarkand regional multidisciplinary hospital. Based on the distinct nosological forms of pneumopathy in neonates and their respective periods of maturity, the following groups were identified from the material: 1) Primary lung atelectasis by gestation: 22–28 weeks, 29–31 weeks, and 32–37 weeks. 2) Hyaline membrane disease, contingent on infant survival. Third, pneumopathy with an edematous-hemorrhagic form of pathology, which is also dependent on the period of neonatal life. An autopsy examining the lungs of infants whose primary diagnosis was primary pulmonary atelectasis revealed macroscopically reduced volume, cyanotic, inactive lungs with pointed, flat edges that did not fill the pleural cavities to capacity. Conclusions: Thus, hyaline membrane disease and primary atelectasis are the predominant morphological forms of pneumopathy in premature newborns.

KEYWORDS

bronchus, lungs, pneumopathy, atelectasis, gestation, newborns, submucosa.

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INTRODUCTION

Among the most urgent issues in neonatology today are respiratory disorders in infants. Due to the morphofunctional features of their respiratory system, respiratory disorders usually develop in premature infants, ranking second in the structure of morbidity among newborns at 8:8 percent [1]. 56.7 percent of newborn and early child deaths are related to respiratory diseases. Of the disorders of the newborn's respiratory system, pneumopathy has a unique place. A non-inflammatory lung lesion is the pathology that affects newborns. Etelectasis, edematous-hemorrhagic changes, and hyaline membranes are found during the pathological examination of newborns that died from pneumopathy [3,4]. In pneumopathy, the newborn's gestational age typically determines the incidence, prevalence, and degree of lung damage. The propensity to develop the disease increases with shorter gestational ages [2,5,6]. Pulmonary destress syndrome, a broad term for the clinical signs and symptoms of pneumopathy, is defined as varying degrees of critical respiratory distress. As soon as the baby is born, breathing becomes shallow and frequent due to this pathology. Severe cases have widespread swelling, quickly worsening cyanosis, heart failure, and foamy fluid leaking from the mouth and nose. Pneumopathy forms are very difficult to differentiate clinically [3].

Study goal: Depending on gestational age, identify the type of pathomorphological alterations in the lung tissue and bronchial wall of neonates that perished from pneumopathy.

Materials and methods of research: The perinatal center of the Samarkand region, along with the pathological department of the Samarkand regional multidisciplinary hospital, examined the bodies of 74 newborns who died from various forms of pneumopathy. The materials from these corpses are included in this study. There were 42 (56.8%) boys and 32 (43.2%) girls among the 74 newborn bodies accounted for. The following is a breakdown of the body weight of newborns at birth: 6 (8.2%) weighed between 500 and 999 g, 28 (37.8%) weighed between 1000 and 1459 g, 22 (29.7%) weighed between 1500 and 2000 g, 12 (16.2%) weighed between 2000 and 2500 g, 4 (5.4%) weighed between 2500 and 3000 g, and only 2 (2.7%) weighed more than 3500 g. Newborns are distributed according to gestational age: 12 (16.2%) were born between 22 and 28 weeks, 21 (28.4%) between 29 and 31 weeks, 32 (43.2%) between 32 and 34 weeks, and 12 (16.2%) between 35 and 37 weeks. Upon analyzing newborns based on their Apgar score at birth, it was discovered that 24 infants (32 %) had severe asphyxia at birth, 34 (46 %) had moderate asphyxia, and only 16 (21 %) had mild asphyxia.

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Four types of pneumopathy were identified from the pathoanatomical and additional histological examination of the lungs of the deceased newborns: hyaline membrane disease in 18 (7.3 percent) of the deceased, edematous-hemorrhagic pneumopathy in 8 (19.5 percent) of the newborns, and combined aspiration-pneumopathic syndrome in 2 (8.9 percent) of the infants. Primary pulmonary atelectasis was identified in 46 (47.7 percent) of the infants in the study. The Shor method was used to completely remove the organs from the infants' bodies before performing a pathoanatomical examination. Chest tissue from all lobules, including the main and lobar bronchi, was removed for histological analysis during the autopsy. The materials were dehydrated in 70, 80, 90, 96, and 100% alcohols and chloroform for 72 hours. They were then fixed in 10% neutral formaldehyde for another 72 hours, rinsed under running water for three to four hours, and then put into blocks by embedding them in paraffin with wax. We used a microtome to cut sections that were 3–5 μm thick. In a thermostat set to 57 o C, paraffin was melted and then removed from the sections using acid. Staining of objects in sections of lung tissue and bronchi was done using the Van Gieson method, which involved staining them with picrofuchsin to identify collagen fibers and hematoxylin-eosin solution to study their histological structure. Using the point counting method, a morphometric study of the lung and bronchus mucous membrane was conducted, as per G. G. Avtandilov (1984). This technique was used with pinpoint eyepiece inserts under a microscope. Ten different fields of view, roughly evenly spaced throughout the section, were examined. The volume fraction of the structural components under study was computed using the data that was gathered. Hematoxylin and eosin-stained histological preparations were used for a statistical analysis of the bronchial mucosa tissue. Software called Statistica 6.1 (Statsoft Inc. , R USA) was employed to analyze the statistical data.

Results of the study: The material was divided into the following groups according to the distinct nosological forms of pneumopathy in newborns based on their periods of maturity: 1) Primary lung atelectasis by gestation: 22-28 weeks, 29-31 weeks, and 32-37 weeks. 2) Hyaline membrane disease based on an infant's prognosis. 3) Pneumopathy exhibiting an edematous-hemorrhagic pathology, which is also contingent upon the neonatal life stage. Using a macroscopical approach, an autopsy on the lungs of infants diagnosed with primary pulmonary atelectasis showed reduced volume, airless, cyanotic, collapsed lungs with pointed, flat edges that did not fill the pleural cavities entirely. The pleura typically had a moderate sheen; subpleurally, there were occasionally multiple pinpoint hemorrhages. The lung tissue was doughy-elastic to the touch, and the parenchyma was dark, bloody, and reddish-blue in color in that area. Small foci of light red air parenchyma were seen in some newborns; these were thought to be distelectatic and were mostly

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located in the hilar segments. The information from a morphometric analysis of the structural elements of the bronchial walls' mucous and submucosal layers—which revealed an atelectatic form of pneumopathy—is presented below (Table №). 1, 2).

Table №. 1. Comparative morphometric indicators of the structural components of the mucous membrane of the bronchial walls with a diagnosis of primary lung atelectasis in %.

Gestational age	Vsqe	Vce	Vgc	Vbec
22-28 weeks	50,5±2,78	9,8±2,22	9,5±1,11	31,2±1,65
29-32 weeks	43,4±2,09	14.7±1,13	11,6±0,26	30,3±2,08
33-37 weeks	44,5±2,67	15,9±0,57	13,5±0,19	26,1±1,75

Notes: Vsqe - squamous epithelium of the bronchial mucosa; Vre - ciliated epithelium; Vgc - goblet cells; Vbec - basal epithelial cells.

Table №. 2. Comparative morphometric indicators of the structural components of the submucosal layer of the bronchial wall with a diagnosis of primary lung atelectasis in %.

Gestational age	Vsp	Vg	Vc	Vste
22-28 weeks	55,3±2,25	6,1±0,21	10,5±1,96	28,1±2,39
29-32 weeks	54,4±2,51	9,7±0,23	12,5±2,12	23,4±3,31
33-37 weeks	54,8±3,22	10,4±1,14	13,2±1,28	21,6±3,93

Notes: Vsp – submucosal layer of the bronchus; Vg - glands; Vc - cappillaries; Vste - stromal elements

Large areas of atelectasis, which are typically polysegmental and occasionally include lobar atelectasis zones, were discovered during a microscopic examination of the lungs of newborns who was died from primary atelectasis, with macroscopically visible complete atelectasis, at the light-optical level.

The histological appearance of the bronchial tubes, which house the intrapulmonary airways, matched the gestational age of the neonate. These anatomical characteristics included a lack of collagen and elastic fibers, underdeveloped smooth muscles, and a low density of protein-mucosal glands in the respiratory tract. The cells of the lining epithelium of the bronchial mucosa in newborns with low gestational ages appeared swollen, partially rounded, and there was fragmentary or massive desquamation into the bronchi lumen with a noticeable and extensive subepithelial edema (Fig. 1.) The mucous membrane in the interlobar bronchi was folded both finely and coarsely, and epithelial cell desquamation foci were visible (Fig. 2.)

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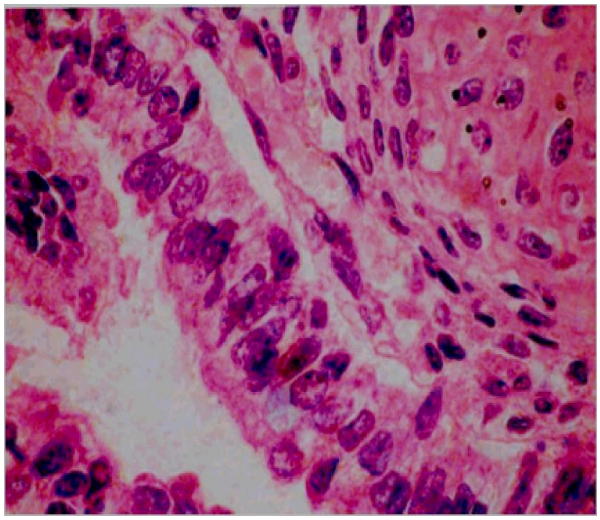

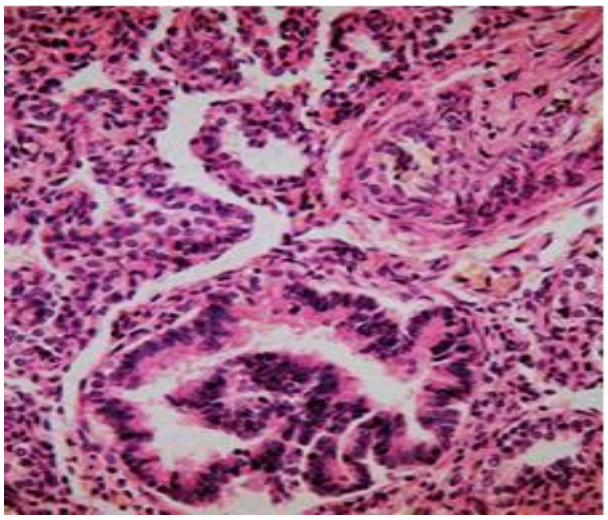

Fig. 1. Severe subepithelial edema of the bronchial wall with primary atelectasis. Hematoxylin and eosin staining. Ob.10x40.
Fig. 2. Desquamation of the epithelium in primary atelectasis. Hematoxylin and eosin staining. Ob.10x20.

In the morphological study of deceased infants one day later at a gestation period of 22-28 weeks, primary pulmonary atelectasis was most often recorded. The epithelium of the bronchi was partially exfoliated, their lumens were free, and in some infants the bronchi looked asleep. The proper layer of the bronchial mucosa is built from a delicate network of collagen fibers; smooth muscle cells were detected in the muscle layer. Collagen fibers were detected in the submucosa of the bronchi. Cartilaginous plates were identified in large bronchi; they had a rounded shape and were connected to each other by collagen fibers. Respiratory bronchioles were dilated, immaturity of the pulmonary parenchyma was revealed with the presence of unstraightened alveolar ducts, multiple glandular and tubular formations, lined with cuboidal epithelium, separated by wide layers of loose connective tissue, with a sharp congestion of microcirculation vessels. In isolated cases, fragments near the amniotic fluid, amniothelial scales and meconium bodies were detected in the bronchi.

In newborns who died after 6-7 days, the mucous membrane of the bronchi had uneven edges, and some had no cilia. The submucosa of the large bronchi contained fuchsinophilic collagen fibers. The cartilaginous plates in the large bronchi were defined as round in shape and were connected to each other by collagen fibers, forming a fibrocartilaginous sheath. In small and terminal bronchioles, the mucosal epithelium was cubic.

The lumens of the respiratory bronchioles contained desquamated epithelial cells and eosinophilic masses.

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A tiny number of single segmented leukocytes and flakes of amniotic fluid were found in the acinar alveoli and terminal alveolar ducts. The epithelium was flattened, and the alveoli had a round or unevenly oval shape. There were wide interalveolar septa. There were perivascular accumulations of red blood cells, the veins were somewhat dilated and full of blood, and the capillaries appeared deserted. The veins were somewhat full of blood, the lymphatic vessels were dilated, the pulmonary artery lumens were free, and some of the veins had blood clots in their lumens. The following histological picture of the bronchi was found in newborns with a gestational age of 29–32 weeks who died within 24 hours of birth: hyaline cartilaginous plates, a small number of elastic and collagen fibers, a small number of mucous glands, and insufficient expression of smooth muscles. The bronchi exhibited a slit-like lumen and a deformed appearance when atelectasis was present. The interlobar bronchi's mucous membrane folded in medium and large sizes, with large foci of desquamation and columnar epithelial cells lining them. Desquamated epithelial cells were found in layers and in groups within the lumens of terminal bronchioles and small bronchi. The morphological characteristics of the newborns in this group's lungs included edematous fluid in numerous alveoli and dilated alveolar ducts, which contain trace amounts of hyaline-like masses. The pulmonary artery branches appeared somewhat clogged, and the lymphatic vessel lumens were dilated. Subpleural hemorrhages were seen, and the pleural tissue appeared loose.

In children who died 6-9 days after birth, dystelectasis, hemorrhage, and bleeding foci in the alveoli were identified. Bronchi of different calibers maintained a free luminescence; attention was drawn to a small amount of dense epithelium and the presence of a large amount of mucus. Collagen fibers were found in the submucosa of the large bronchi. Cartilaginous plates were identified in the large bronchial structures in round form, connected by collagen fibers, and formed the fibrocartilaginous membranes of the large bronchial structures. In small and terminal bronchioles, the mucous membrane epithelium forms as a cubic one. The alveolar ducts looked large, and the inner walls of many of them were covered with hyalin membranes.

In alveoli without hyalin membranes, epithelial cells had homogeneous eosinophilic cytoplasms, with a flat appearance, and were partially exfoliated in the lumen. The vessels of the interalveolar septa had enlarged endothelium protruding into the lumen. They are located along the periphery of the septa and contain aggregated red blood cells. The lymphatic vessels were dilated and the peribronchial and interlobar connective tissues appeared loose. In some observations of newborns born at 33-37 weeks of gestation and dying 7-14 days later, primary atelectasis was complicated by the development of small-sided pneumonia. In two cases, it was pulmonary parenchyma combined with

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aspiration and pneumopathology. The mucous membranes of the large and medium bronchi were lined with prismatic epithelium in several rows; in the small bronchi, epithelium was single-layer, cubic in type. In the wall of the bronchi, compact muscle cells and fibers of the connecting tissue layers have been detected.

Small amounts of elastic fibers were detected, as well as mucous glands. The cartilage plates of large bronchis consist of a basophile interstitial material and individual chondrocytes. The medium-sized bronchi do not contain cartilage plates. In atelectasis, bronchial areas often had deformed appearances and lumens similar to slits. There was a medium and large folding of the mucous membrane of the interlobal bronchi, surrounded by cylindrical or glass cells with large dequamation foci. The bronchial tree's inner epithelium was significantly desquamated. There was an accumulation of neutrophil leukocytes in the lumen of the alveoli, the presence of alveolar macrophages, and a sharp confluence of microvasculature with edema and focal hemorrhages. The accumulation of myeloid cells was observed in the respiratory bronchioles. In newborns with hyaline membrane diseases who died before 24 hours of life, the lungs were macroscopically significant airless, dark red, and densely matched. The lumens of the bronchi have a star-shaped shape, many of them are cystically dilated, and the bronchioles were identified as being transformed into cystically dilated alveoli, forming bizarre cavities (Figure 3). In these formations, lamellar decamination of the epithelium was observed, and in the dilated bronchioles, the epithelium was preserved and flattened. Table No. 3 and 4 show morphological indicators of the structural components of the mucous membrane and submucous membrane of the bronchial wall in newborns who died of hyalin membrane disease.

Table №. 3. Comparative morphometric indicators of the structural components of the mucous membrane of the bronchial wall with a diagnosis of hyaline membrane disease in %.

Gestational age	Vsqe	Vce	Vgc	Vbec
22-28 weeks	51,5±2,78	10,4±2,22	9,2±1,11	28,9±1,65
29-32 weeks	44,7±2,09	13,2±1,13	12,4±0,26	29,7±2,08
33-37 weeks	46,5±2,67	14,9±0,57	13,2±0,19	25,4±1,75

Notes: Vsqe - squamous epithelium of the bronchial mucosa; Vre - ciliated epithelium; Vgc - goblet cells; Vbec - basal epithelial cells.

Table No. 4. Comparative morphometric indicators of the structural components of the submucosal layer of the bronchial wall with hyaline membrane disease in %.

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Gestational age	Vsp	Vg	Vc	Vste
22-28 weeks	54,7±2,25	6,4±0,21	10,7±1,96	28,2±2,39
29-32 weeks	54,4±2,51	9,7±0,23	12,5±2,12	23,4±3,31
33-37 weeks	54,8±3,22	10,4±1,14	13,2±1,28	21,6±3,93

Notes: Vsp – submucosal layer of the bronchus; Vg - glands; Vc - cappillaries; Vste - stromal elements

During microscopic examinations, bronchial lumens were freely preserved in newborns who died of hyalin membrane disease, regardless of the gestational age. The lumens of the bronchi contained a small amount of desquamated epithelium and mucus, and segmented leukocytes were observed. The alveolar epithelium submersion membrane thickened, was loose in some places, the alveolocytes looked thickened and larger, many of which had hypochromic nuclei in a state of lysis and caryorrhexis, and pericapillary edema was noted. In premature newborns with hyalin membrane diseases, a microscopic examination of respiratory parenchyma revealed all signs of immature lung tissue (Fig. 4).

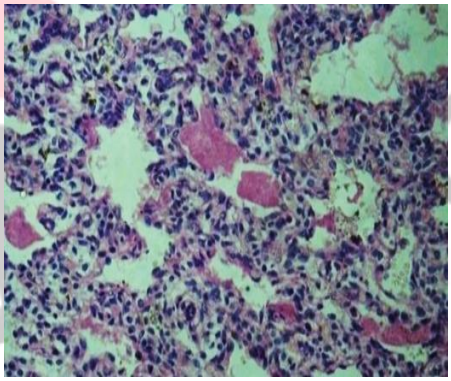

Fig. 3. Lung of a 34-week newborn with hyaline membrane disease. The alveolar ducts and alveoli contain hyaline membranes. Hematoxylin and eosin staining. 10x40.

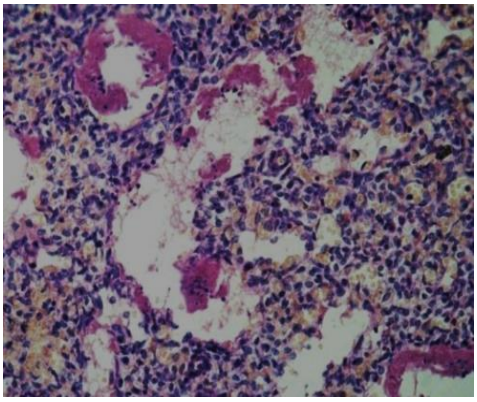

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Fig. 4. Lung of a 34-week newborn with hyaline membrane disease. Accumulation of segmented leukocytes and emphysematous areas. Hematoxylin and eosin staining.10x40.

The lumens of the vessels of the microcirculatory bed were dilated, full of blood, in the lumens there was a reaction of blood cells sludge, diapedesis of erythrocytes and a strong confluence of capillaries that erupted in the lumens of the alveoli. When death occurred 25-48 hours after birth, the bronchial lumens were enlarged and formed large cavities, partly covered with sharply flat epithelium. These cavities are often combined with areas of varying degrees of severe alveolar emphysema (Fig. 5), which are observed in all infants, and blood cranial foci have also been observed in the interalveolar septa, under the pleura, around the vessels and in the bronchial walls.

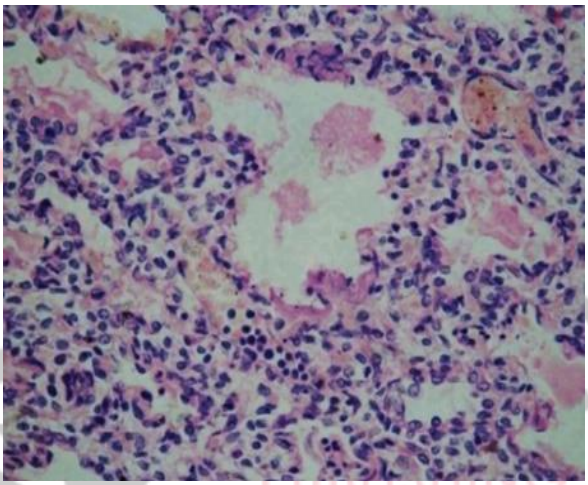

Fig. 5. Lung of a 36-week newborn with hyaline membrane disease. In the alveoli there are fibrin-like masses and segmented leukocytes. Hematoxylin and eosin staining. About. 10x40.

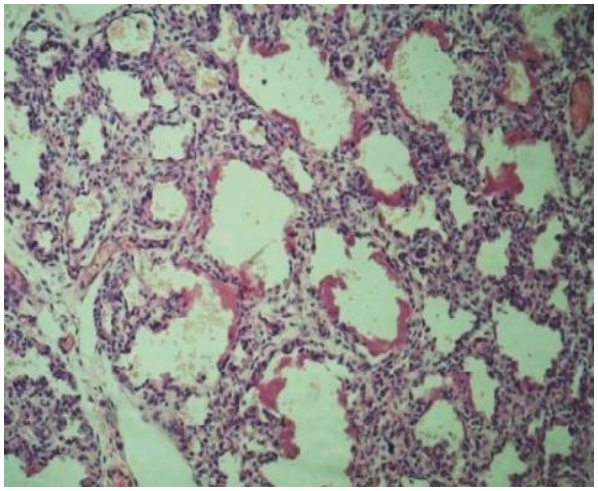

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Fig. 6. Lung of a 36-week newborn with hyaline membrane disease. alveolar emphysema of varying degrees. Hematoxylin and eosin staining. 10x40.

The alveoli had a polygonal or square shape, and in some children they had an extended shape, the shape of a process. In the lumen of the alveoli, amniothelial scales, fibrin-like masses and segmented leukocytes were detected (Figure 6). Severe alveolar emphysema was observed. Arteriovenular anastomoses and lymph vessels were dilated. Bowel arteries were detected around vessels of the bronchial walls and under the pleura.

CONCLUSION

Thus, the most important morphological forms of pneumopathy in premature newborns are primary atelectasis and hypoglycan membrane disease. The nature and severity of morphological changes in bronchial and lung walls depend on the patient's life expectancy and the gestational age of the newborn.

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