MORPHOLOGICAL SIGNS OF ESOPHAGUS CONGENITAL ABNORMALITY

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Annotation. The critical type of preeclampsia considered as a unfavorable impact in pulmon development in the 2nd and 3rd trimester of pregnancy, at 20-36 weeks. The defects of GIT, CVC and respiratory systems of the baby also adversely affect the development of the pulmonary parenchyma. Hystomorphological signs of the pulmon of fetus born with atresia of esophagus have been studied in medical science. The most important symptom and sign was verified in pulmon in operative procedure of esophageal atresia in fetus: appearing of cellular inflammatory infiltrates - lymphocytes. In pulmon of fetus who undergo operative procedure for esophageal atresia, stillbirths with esophageal defects, babies who died, but aspiration pneumonia was comparable. Microscopical investigation showed each situation had its own features and signs. Respiratory problem is the most important type of diseases of newborns. The defects of pulmon considered for 1 in 6 deaths worldwide (1,5,10). Nowaday the issue of prevention and curation of respiratory pathology in childhood, fetus remains topical. The physiological development of the pulmon in infants, their pathological appearing is influenced by the following agents: polyhydramnios, diabetes mellitus, hyperthyroidism, chronic infections (2,5,7). The critical type of preeclampsia has a negative impact on pulmon development in the hestational 20-36 weeks. The defects of the infants GIT, CVC adversely damage the development of the pulmon (4,6,7). The main agents contributing to alveolar tissue injury, formation of hyaline membrane disease (HMD) in preterm fetus is intranatal hypoxia, amniotic fluid aspiration (2,3,6,8,9). The atresia of esophagus and pathology of GIT directly alterate the development of tissues of the pulmon.

Key words. Fetus, infant, pulmon, pulmonary, injury, alteration, atresia, defect, abnormality, bronchi, appear, repair, verification, accumulation, aspiration.

МОРФОЛОГИЧЕСКИЕ ПРИЗНАКИ ВРОЖДЁННОЙ ПАТОЛОГИИ ПИЩЕВОДА

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Аннотация. Тяжёлая форма преэклампсии рассматривается как неблагоприятный фактор, влияющий на развитие лёгких плода во II и III триместрах беременности (20–36 недель гестации). Пороки развития желудочно-кишечного тракта, сердечно-сосудистой и дыхательной систем также оказывают отрицательное воздействие на формирование лёгочной паренхимы. В медицинской науке изучены гистоморфологические особенности лёгких плодов, рождённых с атрезией пищевода.

Наиболее значимым морфологическим признаком, выявленным в лёгких плодов при атрезии пищевода, являлось появление клеточных воспалительных инфильтратов, представленных преимущественно лимфоцитами. В лёгких плодов, перенёсших оперативное лечение атрезии пищевода, у мертворождённых с пороками пищевода, а также у умерших новорождённых были обнаружены изменения, сходные с аспирационной пневмонией. Микроскопическое исследование показало, что каждая из указанных ситуаций имеет свои характерные морфологические особенности.

Респираторные заболевания остаются одной из наиболее значимых патологий периода новорождённости. Патология лёгких является причиной примерно одной шестой всех случаев смерти новорождённых в мире (1,5,10). В настоящее время проблема профилактики и лечения заболеваний дыхательной системы у плода и детей раннего возраста сохраняет свою актуальность.

На физиологическое развитие лёгких плода и новорождённого, а также на возникновение патологических изменений влияют такие факторы, как многоводие, сахарный диабет, гипертиреоз и хронические инфекции (2,5,7). Тяжёлая преэклампсия оказывает негативное воздействие на развитие лёгких в период 20–36 недель гестации. Пороки развития желудочно-кишечного тракта и сердечно-сосудистой системы плода также неблагоприятно влияют на формирование лёгочной ткани (4,6,7).

Основными факторами, способствующими повреждению альвеолярной ткани и развитию болезни гиалиновых мембран у недоношенных плодов, являются интранатальная гипоксия и аспирация околоплодных вод (2,3,6,8,9). Атрезия пищевода и другие заболевания желудочно-кишечного тракта непосредственно нарушают развитие тканей лёгких.

Ключевые слова: плод, новорождённый, лёгкие, лёгочная ткань, повреждение, патологические изменения, атрезия, порок развития, аномалия, бронхи, возникновение, репарация, верификация, накопление, аспирация.

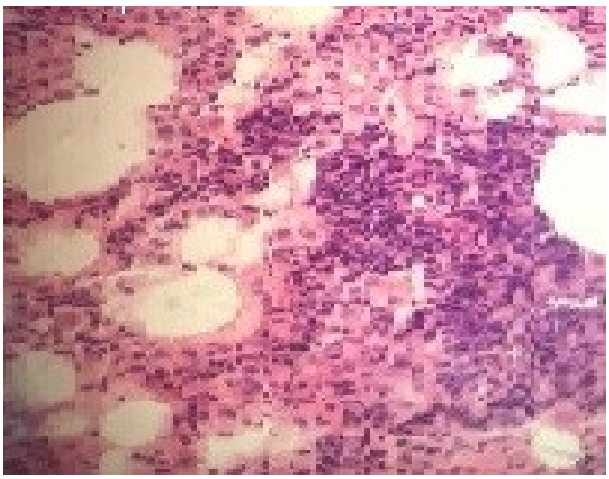
The goal of researching was to provide concept in the literature on morphological signs of pulmon of fetus with esophageal atresia.

Research sources and methods. In the Samarkand Regional Research Center for Pediatric Surgery in 2015-2017 (64 cases) infants born with esophageal atresia and died of surgical treatment, infants born with esophageal anomalies and deaths with complications such as aspiration pneumonia infants were screened. All cases were autopsied in the Department of Pathological Anatomy of the 1st Clinic of SamMI. Of these, 42 were male (73.12%) and 22 were female (26.88%). age was 1–1.5 months. During the autopsy examination, we obtained tissue fragments from different areas of the lungs and different parts of the esophagus, as well as unaltered parts of these organs. Autopsy revealed the development of aspiration pneumonia in 12 cases in girls, 30 cases in boys, esophageal-tracheal fistula (leakage) in 10 cases in girls and 12 cases in boys. In all cases, the leading causes

of death were progressive respiratory and heart failure, aspiration pneumonia, mediastinitis, pleurisy, and pericarditis.

In all groups of observations, attention was paid to the selection of materials for research to the extent that they allow a complete assessment of the morphofunctional status of the bronchi, respiratory parts of large, medium and small caliber. To do this, the lungs of the fetus and the dead baby were examined in whole or in part. Attention was paid to obtaining 5-7 pieces of tissue from different parts of the lungs of slightly older fetuses and dead infants from different areas. We used a light microscope to analyze these samples. Lung tissue fragments were treated with Buen's fluid and solidified. We took the frozen pieces in 3-4 parts, washed them in 800 alcohol, dehydrated them and then waxed them. From each block, we prepared 6-8 steps, 10 μm thick, 60-80 μm apart, and stained with hematoxylin-eosin dye. We also did morphometric studies.

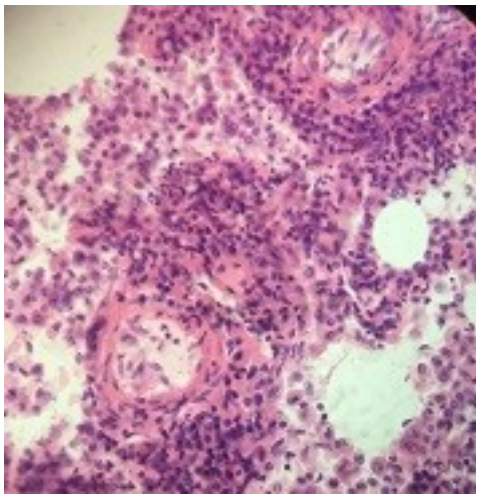
Research results. Macroscopical investigation of pulmon of fetus who underwent esophageal defects surgery showed a small amount of serous fluid in the large-caliber bronchial area, both side pulmon was full, pink, airy. By microscope we can see mucosal layer of respiratory bronchioles is covered with prismatic epithelium. Epithelial cells of some areas are alterated, desquamated, subcutaneous vessels are full, hypersecretion of mucous glands, myocytes are oedematous, sparsely formed fibrous tissue fibers are fibrocytes, cellular inflammatory infiltrates appear. All inflammatory cells are composed of lymphocytes, monocytes, macrophages, fibrocytes, fibroblasts, plasmoblasts, and segmented nuclear neutrophils.



Picture-1. **Infiltration of lymphocytes, plasma cells, and macrophages in the lungs of an fetus who died of esophageal atresia. Hematoxylin-eosin, ob. 40, ok. 10.**

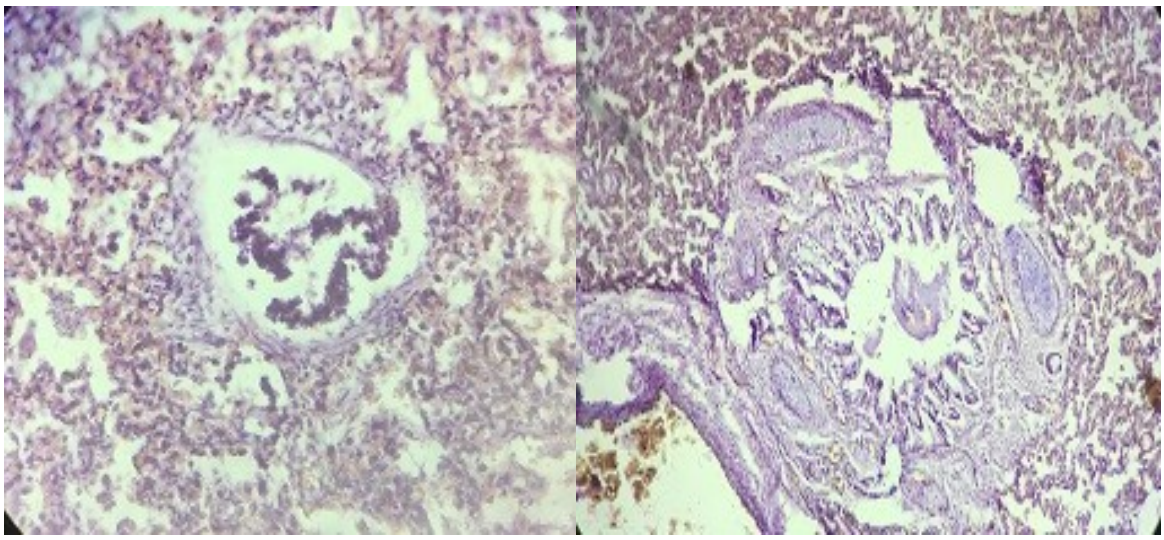
The lymphohistiocytic infiltrates are detected in the muscle layer of the terminal bronchioles. By microscope in pulmon of fetus - have a canalicular structure, component. The alveoli are covered with flat and cuboidal epithelium. A small amount of clear fluid, fibrin fibers and macrophages are found in the alveolar cavity. Lymphocytes and fibroblasts accumulate in the walls of the aerohematic barrier, and in the capillaries appear fullness and hemostasis. Transudate is detected in the alveolar cavity and aerogematic barriers due to increased permeability of capillary endothelial cells. substance, migrating mucous membranes and amniotic fluid, meconium residues are detected. The lung parenchyma is dark brown, airless, gives a positive result when tested with water, has a soft-elastic consistency when cut. On microscopic examination, the bronchial mucosa is covered with prismatic epithelium, and in most areas desquamation encountered, prismatic epithelial cells appear to be attached to the lashes and covered with an adhesive substance. The mucous membrane is sparsely formed connective tissue, myocytes are swollen, covered with lymphocytes, plasmoblasts, monocytes and fibroblasts. In small blood vessels we can see fullness and hemostasis. The fibrosis and the adventitial layers are unchanged. The small and terminal bronchial cavities are covered with a mucous mass. Microscopically, most

of the cubic epithelial cells are found to be desquamated in the mucosa. The alveoli of most segments of the lung are not open.



Picture-2. Infiltration of lymphocytes, plasma cells, and macrophages in the lungs of an fetus who died of esophageal atresia. At the same time sclerotic changes of terminal bronchioles are revealed. Hematoxylin-eosin, ob. 40, ok. 10.

In intrauterine period of fetal development, most alveocytes have uncorrected, displaced, airless, distillate areas. The alveolar barrier is thickened; infiltration of lymphocytes, monocytes, as well as hemosiderin granules are noted. an increase in the number of endothelial cells in the blood vessels, thickening of the middle membrane and the outer wall of the walls, stasis in the blood vessels were noted. The alveoli are covered with cubic and flat epithelium. In some cases, the lungs have a structure typical of the alveolar stage, the alveolar compartments are wide, the general capillaries of the lungs are not formed, the endothelium of the capillaries is swollen.



Picture-3. In the lungs of a stillborn baby, desquamative changes in the terminal bronchioles, accumulation of aspiration fluid in the lungs, and autolysis of the lung parenchyma can be seen. Hematoxylin-eosin, ob. 40, ok. 10.

In premature infants (1000-1200 g) the alveoli are small in size and covered with a cubic epithelium. The walls of the alveolar barrier are wide, and the network of capillaries in them is not located directly under the epithelium in all areas. Insufficient development of elastic fibers in interstitial tissues.

Pulmonary structure in the state of atelectasis appears to be partially flattened in each places.

Conclusion. Histological examination of the internal organs of infants who underwent surgery for respiratory pathology and esophageal atresia revealed that the morphological structure of the lungs was preserved. However, the morphological picture was found to be polymorphic, which in turn indicates the depth of the changes in the lungs: exudate and fibrin, segmented leukocytes in the cavity of the deformed bronchi and alveoli in a number of dead infants fallen alveocytes, fragmented and whole erythrocytes, hemosiderin granules were detected. The walls of the bronchioles are often intact, the cavity of the preserved terminal bronchioles contains dense exudate, numerous segmented nuclear leukocytes, migrated bronchiolar epithelial elements. In the peribronchial areas and

in the walls of the alveolar barrier, a large number of tumors, mixed cell infiltrates: segmented leukocytes, macrophages and lymphocytes were identified, depending on the course.

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