

## MORPHOLOGICAL ASPECTS OF ESOPHAGEAL ATRESIA WITH DISTAL FISTULA IN NEWBORNS

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### ABSTRACT

Esophageal atresia is a pathological condition that manifests itself in children by the absence of any connection between the upper and lower segments of the esophagus. This anomaly is one of the relatively common developmental anomalies (in 1 case per 3,500 births). There are 5 different variants of esophageal atresia, which are the most common in terms of clinical morphology and anatomy. Atresia of the esophagus with distal attachment, that is, morphological changes in the 1st variant of this anomaly are not covered at all, the lack of morphological criteria shows that there is a high need for sound fundamental knowledge to propose a solution to the problem.

**Key words:** esophageal atresia, morphology, dysplasia, morphological immaturity.

### INTRODUCTION

**Relevance of the problem.** Esophageal developmental anomalies are a process in the embryonic period that continues the development of the esophagus and its structures with anatomical and histological changes. Worldwide the development of this pathology corresponds to 40 out of 100,000 babies. The detection of this pathology during early pregnancy screening in the United States and European countries, based on strict guidelines for termination of pregnancy, has an average incidence rate of 4-8 per 100,000 infants. While the incidence of these pathologies in the CIS countries, including the Russian Federation, is 20-25 cases per 100,000, in the Republic of Uzbekistan this figure averages 8-10 cases

per 1,000 infants, which is manifested by a high mortality rate - on average 60-78% in the period from birth to a month. Currently, this issue is a problem for both pediatricians and neonatologists, since the incidence rates in infants born between relatives marriages with a high genetic predisposition are 2.5 times higher according to foreign literature and medical history collections. It is the fact that mortality in combined bronchoesophageal adhesions and oesophageal malformations exceeds 85% that causes infants to die from aspiration pneumonia in the early days of the early neonatal period. This, in turn, requires focusing on the morphological aspects of finding a solution to the problem and developing methods for the expression of mutated genes and targeted therapy based on molecular genetic testing.

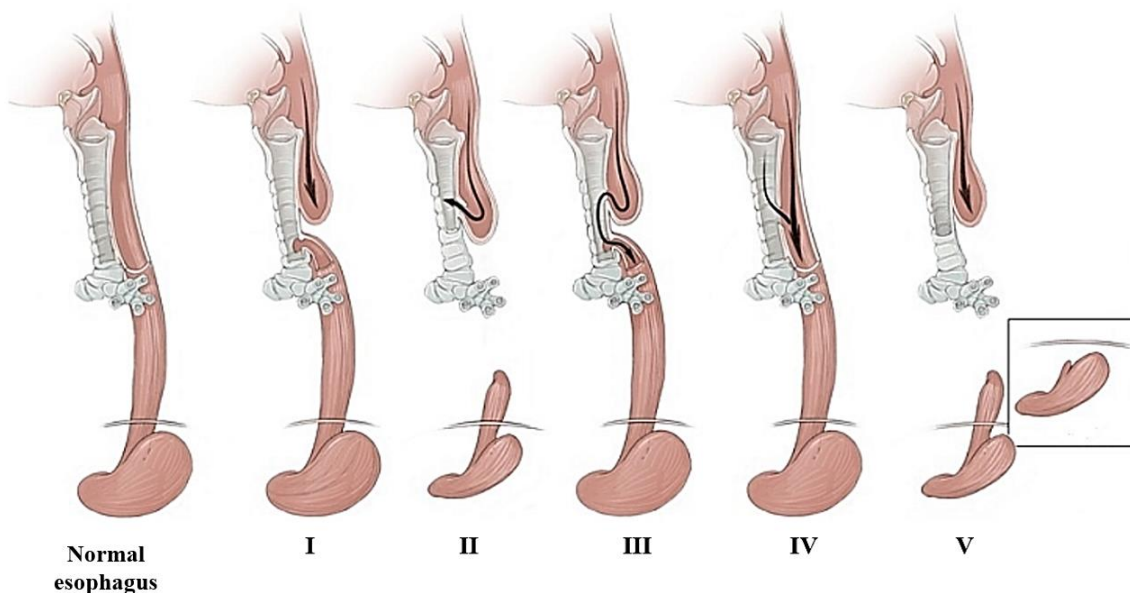
**Research Aim.** The study of specific pathomorphological changes that occur in the clinical and morphological variant #1 of the anomalies of the esophagus.

**Materials and methods of research.** As a material, autopsy materials were collected from infants who died with abnormalities of the esophagus and infants who underwent surgery and died without surgery at the multidisciplinary clinic of the Tashkent Medical Academy (MCTMA), during which esophageal substance was obtained in 43 cases. The finished products are studied morphologically. Morphology: stained with hematoxylin and eosin dye.

**Discussion and conclusion.** In case of esophageal atresia, the esophageal sac remains closed on one side in the form of a sac consisting of fibrous tissue. It is in the first month that the esophageal tube, which has come to this appearance, remains in the form of a bag-shaped shape, the complete formation of its anatomical walls, the absence of development of mucous and submucosal membranes and the appearance of retarded morphological immaturity with the development of esophageal aplasia. As a result, the surface of the mucous membrane takes on the appearance of a sac-like organ consisting of a cluster of serous secreting cells resembling single-layered mesothelial cells. It is precisely because the mucous membrane of the esophagus, mainly during embryonic development, initially has a single-layered prismatic epithelium, which mainly persists for up to 7-8 weeks, followed by absolute closure of the esophageal cavity, which leads to the development and desquamation of vacuolar dystrophy in the prismatic epithelium. epithelium and the re-evolution of the esophageal cavity. In a 3-month period, these detached epithelium should be replaced by the appearance of a multilayer epithelium, but with exogenous effects (infection, toxic substances, drug exposure, etc.), a congenital defect occurs, and anomalies form in the form of atresia, stenosis, adhesions. It is in our study that the role of pathoanatomical studies of the esophagus in intrauterine malformations is such that, under the

influence of factors influencing the formation of this esophagus, the formation of anatomical floors of the esophageal wall at different stages, morphological features of mucous membranes covered with a single layer of prismatic epithelium and multilayer. There are 5 different variants of esophageal atresia, which are most commonly used in clinical and anatomical practice. Esophageal atresia occurs morphologically, under the influence of various exogenous and endogenous factors in the first 3 months of intrauterine development. Esophageal atresia is mainly caused by a violation of the relationship of the fetus with the stomach, under the influence of exogenous and endogenous factors, during the tachymatic location of the trachea and thoracic tubes during the formation of endodermic integument at 4-12 weeks of fetal development. Clinically and morphologically, this is manifested in a child within 72 hours after birth, as a result of complete closure of the esophageal tube, accumulation of food in the esophageal cavity in the form of a cystic sac and the occurrence of periodic relapses in infants. From a morphological point of view, with esophageal atresia, the following changes occur in the normal histioarchitectonics of the esophageal wall.

#### Esophageal atresia



**Figure 1.** Graphic images of esophageal atresia. For comparison, an image of a normal esophagus is presented.

Option 1. An option in which the proximal part of the esophagus has the shape of a sac, and the distal part ends with a trachea. 86%

Option 2. Atresia of the esophagus without an isolated end. 7%

Option 3. Isolated esophageal-tracheal fistula. 4%

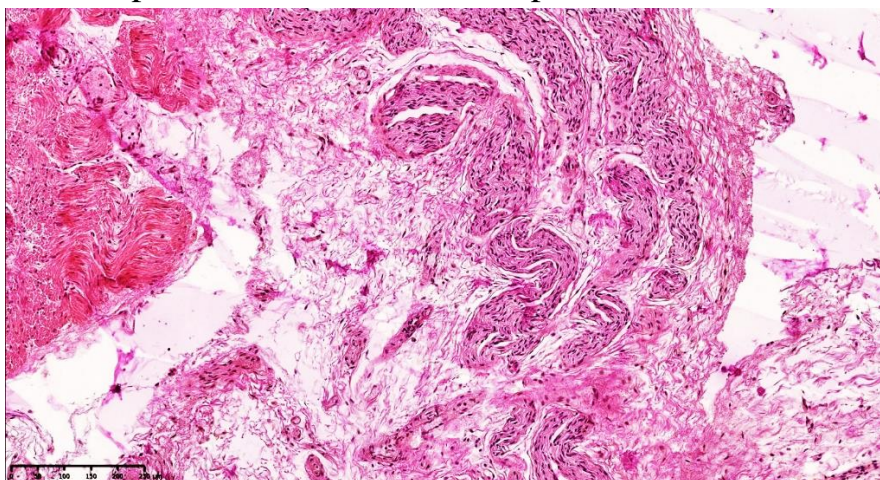
Option 4. Articular atresia of the esophagus in the proximal trachea. 2%

Option 5. Atresia of the upper and lower segments of the esophagus with termination. 1%

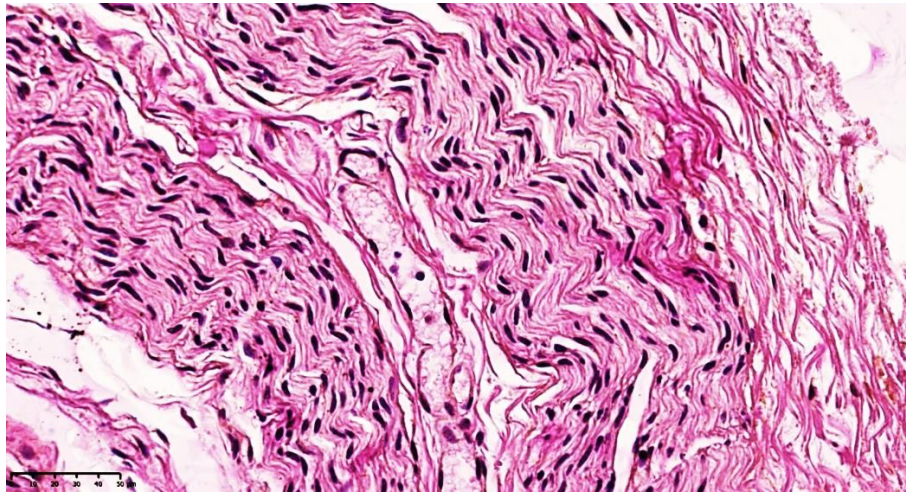
From the point of view of the specificity of this cited esophageal atheroma, the following was found in one of the variants of morphological changes. In particular, dysplasia of the esophageal tube is characterized by the fact that at the same time all anatomical floors lag behind in development, the presence of substrates resembling a picture of mucoid protrusion in the cytoplasm, vacuoles, hyperchromic nuclei covering the mucous flat surface of the epithelial cell. At the same time, one of the age-related aspects of the mucous membrane is the presence of 1-3 layers of flat epithelial coating, which should be 3-5-8 layers, in a flat and semicubic shape, due to the fact that food remains on the mucous membrane, an acute bulge is detected by an accumulation of epithelial cells. Signs of the formation of intermediate edema on the mucous membrane, uneven filling of blood vessels, erosive and ulcerative esophagitis on the surface of the mucous membrane are revealed. Of course, these signs are considered specific morphological signs caused by exposure to the mucous membrane of foods that remain in the esophageal cavity.

It is with esophageal atresia that myocyte pairs of short-term muscle fundus hypertrophy and various nodular manifestations are detected due to the fact that food remains stagnant in the esophageal cavity and frequent reflux occurs. One of the most interesting aspects is the appearance of edema and hypertrophy of the muscular tendons along the perimeter of the circularly located muscular fascia, while in the longitudinal muscles the appearance of zigzag contracture fibers is a morphological substrate confirming a violation of the peristalsis of the esophagus.

As a result, there is an asynchronous uneven thickening of the walls of the esophagus, a change in the texture of tissues, and uneven landscapes of different sizes are detected in places close to the cardiac part.



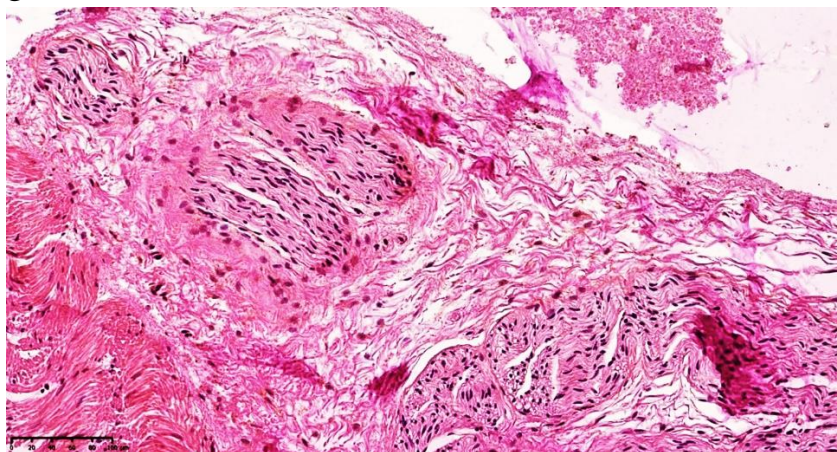
**Figure 2.** Variant 1 of esophageal atresia. The anatomical layers of the esophageal wall are partially formed. There is developmental dysplasia on the walls. There is a distinction between the serous membrane, the muscular membrane and the inner layer (mucous membrane) of trembling, sparse fibers. The dye G.E. Is 4x10 in size.



**Figure 3.** Variant 1 of esophageal atresia. The anatomical layers of the esophageal wall are partially formed. Muscle fundus myocytes are similar to embryonic cells with a large nucleus with hyperchromia, it was found that there is a wave-like appearance in the muscles, intermediate tumors have developed, fullness is also observed in the blood vessels, perivascular tumors have also developed. The dye G.E. Is 40x10 in Size.

This confirms the fact that the esophagus was left motionless and cystically dilated as a result of the lack of exit through all anatomical layers in the wall of the esophagus (see Fig. 4).

In tissues originating from areas of the esophagus where there is a sharp deficiency of alveolar glands located on the mucous membrane and submucous membranes, and from the enlarged areas of the cyst show sharp atrophic and sclerotic changes in the muscle tendons.



**Figure 4.** Variant 1 of esophageal atresia. In the anatomical layers of the esophageal wall, muscle bundles are preserved in the form of buds. The myocytes of the muscular fundus are similar to embryonic cells with a large nucleus with hyperchromia, the muscles are preserved in the form of sparse fibrous connective tissue that does not occupy the full perimeter and does not form in fibrosed tissue,

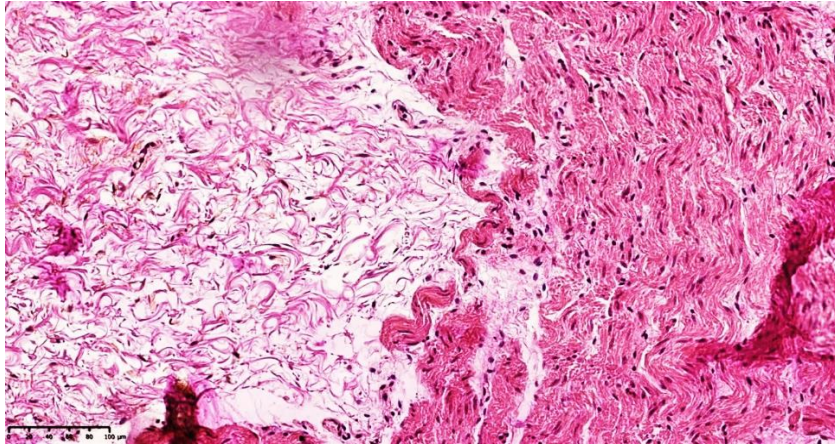
intermediate tumors, histiocytes, fibroblasts are found. The dye G.E.Is 20x10 in Size.

This, in turn, was characterized by the fact that esophageal motility was not developed at all, and infants were at high risk of mainly causing reflux and causing aspiration. As for the composition of intermediate tumors formed between all anatomical floors of the esophagus and inflammatory infiltrates formed to varying degrees, it was found that it consists of histiocytes, lymphocytes, plasmocytes, and cells. This confirms that the process was chronically caused by intrauterine infection of the fetus. With acute fullness and diapedesis, foci of blood clots are detected in the blood vessels of the muscular fundus. This confirms that it is precisely due to the effects of the food product and the mucous membranes that remain with esophageal atresia that secondary manifestations occur in the form of esophagitis.

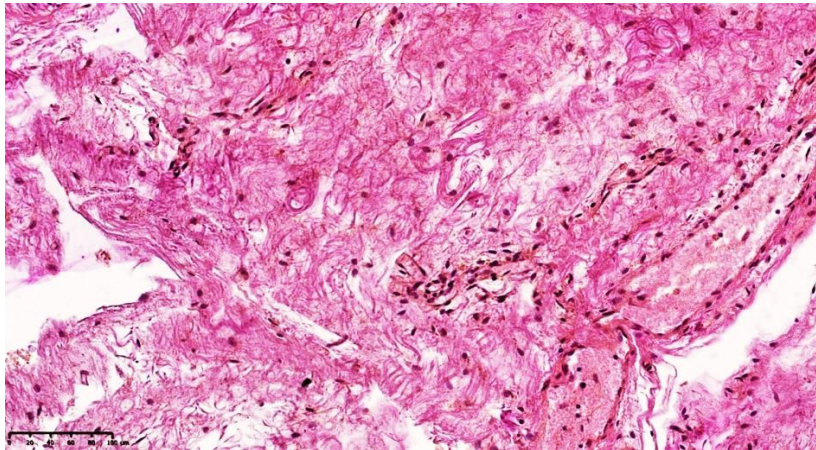
With anomalies of the esophagus, the formation of the endoderm leaflet at the age of 4-12 weeks of fetal development occurs during the development of the trachea and thoracic tubes, under the influence of exogenous and endogenous factors, due to a violation of their relationship with the stomach. Clinically and morphologically, this is manifested in the baby by the fact that within 24-72 hours after birth, due to the complete overlap of the esophageal tube, food accumulates in the esophageal cavity in the form of a cystic sac and infants experience periodic relapses. From a morphological point of view, with esophageal atresia, the following changes occur in the normal histioarchitectonics of the esophageal wall.

In variant 1, microscopic examination of the atresia of the proximal esophagus showed that the anatomical floor of the wall was not formed, while in 11 out of 13 cases a muscular fibrous sac was detected. In particular, it was found that the serous curtain of the esophageal wall, modified by the proximal sac, consists of normally visible sparse fibrous connective tissue, edema formed in the intervening tissue, and signs of uneven filling remained in the blood vessels. At the same time, the main part of the wall is characterized by the appearance of tumors in the intervening tissue and dysplasia, in which the muscle ligaments are not fully formed.

Myocytes in smooth muscle bundles in the wall of the isolated proximal sac are characterized by incomplete formation, large nuclei, dark eosinophilic cytoplasm, interstitial tumors along the perimeter of the thin fibrous curtain of the outer fascicular membrane of the muscle bundles are characterized by the presence of mucoid protrusion, histiocytes, fibroblasts in the intermediate stroma.



**Figure 5.** Variant 1 of esophageal atresia. The surface of the esophageal mucosa is not fully formed. Mucous membrane dysplasia, in which the surface of the mucous membrane is not formed by sparse fibers. The main volume of the stroma is made up of an intermediate liquid. Dye G.E. Is 40x10 in ize



**Figure 6.** Variant 1 of esophageal atresia. The anatomical layers of the esophageal wall are partially formed. The bundle of myocytes, which has not yet fully formed on the muscular floor, apparently has a hyperchromic nucleus, and the main composition of the stroma consists of unformed sparse fibrous connective tissue.

Dye G.E. Is 20x10 in size.

It was found that the violation of the ratio of anatomical floors in the wall of the esophagus continued with a change in the histioarchitectonics of the muscle bundles, in which the main floor was formed in different volumes. The bottom after the serous veil is a wave-like arrangement of the bundles of the muscular fundus in different irregular directions, the muscle bundles are not yet fully formed, the presence of connective tissue components with sparse fibers in their gaps, fullness of blood vessels and foci of erythrocyte slag formation is determined. Active foci of histiocyte and fibroblast proliferation are found in the area of muscle bundles and in large numbers in perivascular areas.

These changes are due to the fact that hypoxia has developed in the atresized esophagus, and the wall is morphofunctionally abnormal during fetal development,

and sac-shaped muscle bundles are found mainly in the neck, that is, in the proximal region. At the bottom and body of the atresized esophagus, the muscle bundles have undergone aplasia, and myocytes are found in the form of a single bundle. Especially considering the fact that the main composition of the atresized esophageal tuber consists of fibrosis and a small amount of muscle tissue, in clinical treatment this is the reason for the appointment of plastic surgery from other areas, such as the small intestine, when considering the occurrence of clinical and morphological signs in the form of akinesia, manifested in the form of postoperative complications in plastic surgery of the esophagus.

**Conclusion.** It was found that variant 1 of esophageal atresia morphologically consists of an atresized proximal part of the esophagus in the neck from connective tissue in the form of various levels of unformed fibrous structures in the form of dysplasia of the remaining serous membrane and mucous membrane in the presence of completely unformed muscle bundles on the surface of the esophagus. the muscular floor. It was found that the mucous membrane had undergone aplasia, in which the epithelial layer had not formed. This means that in tactical treatment, the use of this tissue leads to unexpected severe complications during postoperative rehabilitation, taking into account the fact that in clinical morphologically practical surgery, the muscle layer and epithelial layer are not formed during plastic surgery of this layer.

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