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# MORPHOLOGICAL FEATURES OF STENOSIS OF THE TRACHEA AND BRONCHI, BRONCHIOLECTATIC EMPHYSEMA, AND TRACHEOESOPHAGEAL FISTULAS

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**Abstract**

Congenital and early developmental bronchopulmonary malformations are frequently characterized by structural defects within the bronchiolar wall, most notably involving the smooth muscle layer. Hypoplasia or aplasia of these muscular elements results in both functional and morphological disturbances, manifesting as abnormal dilatation of the bronchioles. This pathological process plays a critical role in the formation of centrilobular emphysema, particularly in its Leschke variant. Moreover, insufficient maturation of the mucosal lining and smooth muscle framework reduces airway stability, thereby predisposing the respiratory bronchioles to emphysematous transformation.

Such anatomical and functional insufficiencies create a favorable environment for recurrent respiratory infections. Over time, secondary inflammatory changes become predominant, commonly progressing to bilateral, multisegmental bronchopneumonia. Pathological examination frequently identifies alterations in pulmonary segments 2, 6, 7, 8, 9, and 10 adjacent to the hilar region. These sites often demonstrate compact foci of mixed exudative inflammation, areas of atelectasis, and emphysematous changes. Recognition of these morphologic alterations is fundamental for elucidating the pathogenesis of post-bronchopneumonic complications, and they remain critical for precise diagnosis and optimized therapeutic strategies.

The present investigation focuses on the pathomorphological characteristics of bronchiolectatic emphysema, tracheoesophageal fistulas, and stenotic lesions of

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the trachea and bronchi, with particular emphasis on their clinical significance and diagnostic implications.

**Keywords:** Bronchiolectasis, Leschke emphysema, tracheal stenosis, bronchial stenosis, tracheoesophageal fistula, pathomorphology, airway deformation, respiratory bronchioles, bronchopneumonia.

**Relevance of the Problem**

Congenital and acquired anomalies of the trachea and bronchi, including stenosis, bronchiolectatic emphysema, and tracheoesophageal fistulas, remain one of the most urgent problems in pediatric pulmonology and neonatal surgery due to their high clinical significance and association with respiratory insufficiency and early mortality. These conditions account for a considerable proportion of neonatal respiratory pathologies, often complicating the course of the early postnatal period [1,2]. Morphological studies have demonstrated that hypoplasia or aplasia of the bronchial muscular layer, disturbances of the submucosal connective tissue, and persistent interstitial edema lead to bronchospasm, bronchiolospasm, and impaired airway patency [3,4]. Such changes contribute to polysegmental bronchopneumonia and secondary pulmonary hypertension, which represent the main causes of neonatal morbidity and mortality [5,6]. Bronchiolectatic emphysema, characterized by cystic dilatation of bronchioles, epithelial desquamation, and inflammatory exudation, has been identified as a key morphological substrate for chronic lung injury in neonates [7]. Similarly, tracheoesophageal fistulas and stenoses significantly increase the risk of aspiration syndrome, aspiration pneumonia, and long-term structural remodeling of the respiratory tract [8].

Recent evidence highlights that epithelial hypoplasia, squamous metaplasia, fibrotic changes, and edema of the lamina propria are crucial morphological determinants of functional respiratory impairment [9,10]. The identification of these features not only enhances diagnostic accuracy but also provides a rationale for timely surgical interventions such as lobectomy or resection, which are

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essential to preventing the progression of pulmonary hypertension, cor pulmonale, and fatal neonatal outcomes [11].

**Research Objective**

To investigate the pathomorphological characteristics of bronchioloectatic (Leschke-type) emphysema, tracheoesophageal fistulas, and congenital stenotic lesions of the bronchi and trachea in neonates.

**Materials and Methods**

This study was based on autopsy material from 113 cases of confirmed congenital anomalies obtained at the Republican Pathological Anatomy Center. Lung and bronchial tissue specimens were collected for detailed examination. Standard histological processing was performed, including hematoxylin and eosin staining, to evaluate the structural organization of tissue components. The acquired morphological data were systematically analyzed to identify characteristic pathological features.

**Results and Discussion**

Aplasia and hypoplasia of the muscular layer of the bronchiolar wall were shown to play a decisive role in the development of **centrilobular pulmonary emphysema.** The insufficient differentiation of myocytes within the bronchiolar musculature, in combination with underdevelopment of the mucosal lining, results in diminished airway stability. Consequently, the bronchiolar walls exhibit **ectatic dilatation**, giving rise to emphysematous transformation of the respiratory bronchioles. These alterations create favorable conditions for the onset of secondary infections, which predominantly manifest as **bilateral polysegmental bronchopneumonia.**

Autopsy examination frequently revealed **mixed exudative inflammatory foci, atelectasis, and emphysematous changes** in pulmonary segments 2, 6, 7, 8, 9, and 10, especially in perihilar regions. These findings reflect a close clinicopathological correlation between emphysema and subsequent bronchopneumonia.

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In cases of **cystic bronchial hypoplasia,** the terminal bronchioles were replaced by **dilated cyst-like cavities**. Their mucosal lining exhibited a markedly thinned structure resembling indistinct alveolar walls, while blood vessels were irregular and tortuous. Within these altered zones, **alveolar cells were absent**, and desquamation of the bronchiolar epithelium was commonly observed. The lumina of affected bronchioles contained **inflammatory exudates** with vascular dilatation, leukocyte diapedesis, macrophage accumulation, and a heterogeneous admixture of cellular components. These structural abnormalities ultimately promoted **bronchiolar obstruction,** which was pathognomonic for this condition.

Given these morphological criteria, the risk of **aspiration pneumonia** is substantial. Therefore, in advanced or complicated cases, **surgical interventions**-including lobar resection or pneumonectomy are considered necessary for effective management (see Figure 1 and 2).

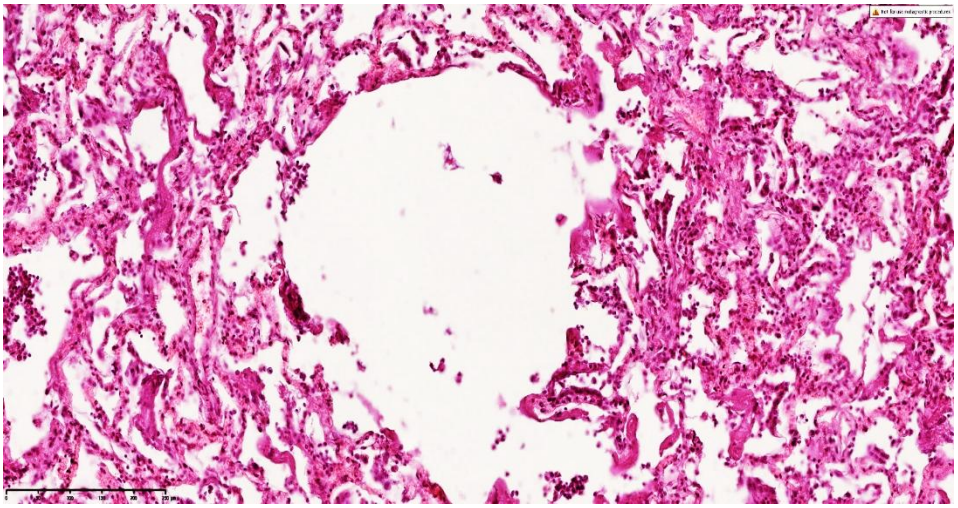

**Figure 1. In bronchioloectatic Leschke-type emphysema, a centrilobular cystic dilated area is visualized (1), surrounded peripherally by atelectatic zones within the alveolar spaces** (2). **The walls of small bronchioles demonstrate sclerotic and atrophic alterations (3). Along the margins of the cystic expansion, there is evidence of developing inflammatory exudation accompanied by features consistent with bronchopneumonia. Staining: H&E. Magnification: 100×40.**

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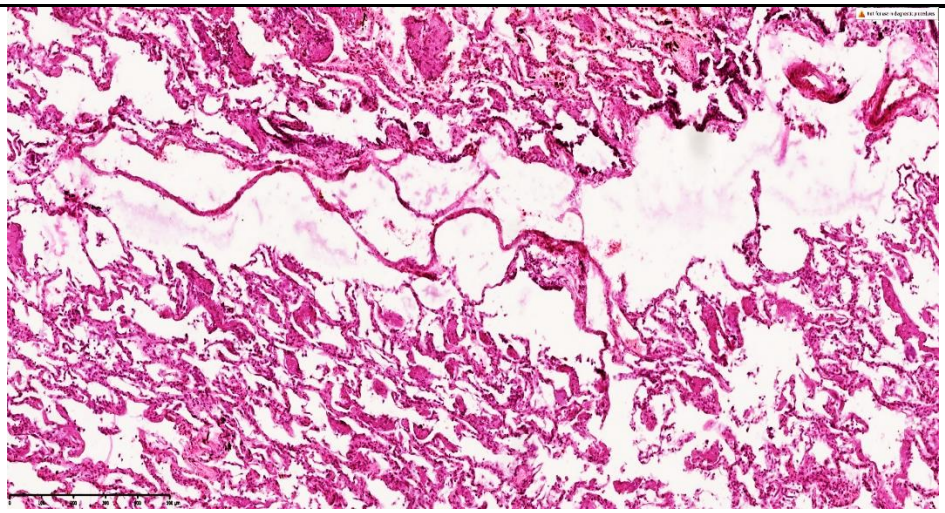

**Figure 2. In bronchioloectatic Leschke-type emphysema, segmental bronchiolar dilatation is evident, with a centrilobular cystic focus (1). Peripheral alveolar spaces demonstrate atelectatic areas (2). The walls of small bronchioles show sclerotic and atrophic changes (3). At the margins of the cystic dilatation, inflammatory exudate and histological features of bronchopneumonia are present. Staining: H&E. Magnification: 100×40.**

In bronchioloectatic Leschke-type emphysema, along with **cystically dilated centrilobular zones** within bronchiolar dilatation foci, there is a consistent presence of **markedly expanded bronchiolar segments** and **pronounced capillary congestion** in the alveolar wall vessels. These alterations collectively form inflammatory foci resembling **bronchopneumonia.** Furthermore, **atelectatic changes** are regularly identified around the cystically dilated alveolar spaces. In our material, **22.1% of cases demonstrated hyaline membrane formation,** which corresponds to the **clinical and morphological features of respiratory distress syndrome (RDS)** (see Figure 3 and 4).

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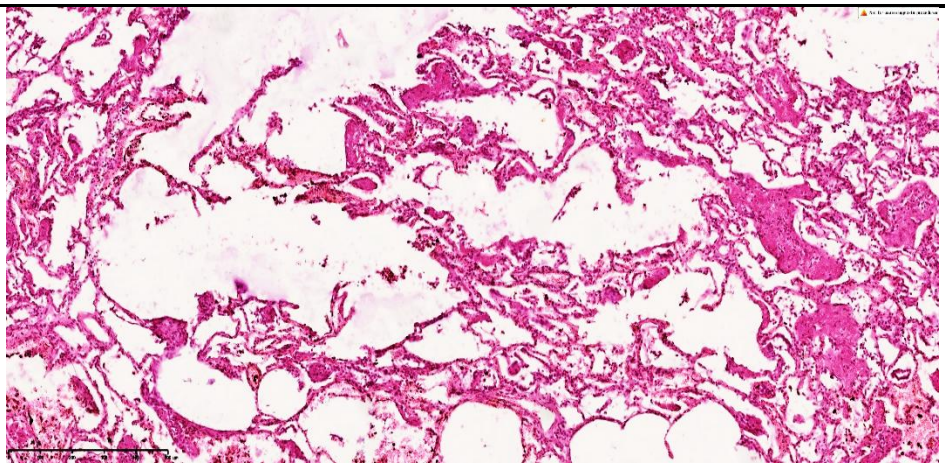

**Figure 3. In bronchioloectatic Leschke emphysema, multicentrilobular cystically dilated foci are distinguished (1), while atelectatic zones are noted in the peripheral alveolar spaces (2). Sclerotic alterations are detected in the walls of small bronchioles (3). Along the periphery of the cystically dilated regions, inflammatory exudation with features of bronchopneumonia is observed.**
**Staining: H–E. Magnification: 10×10.**

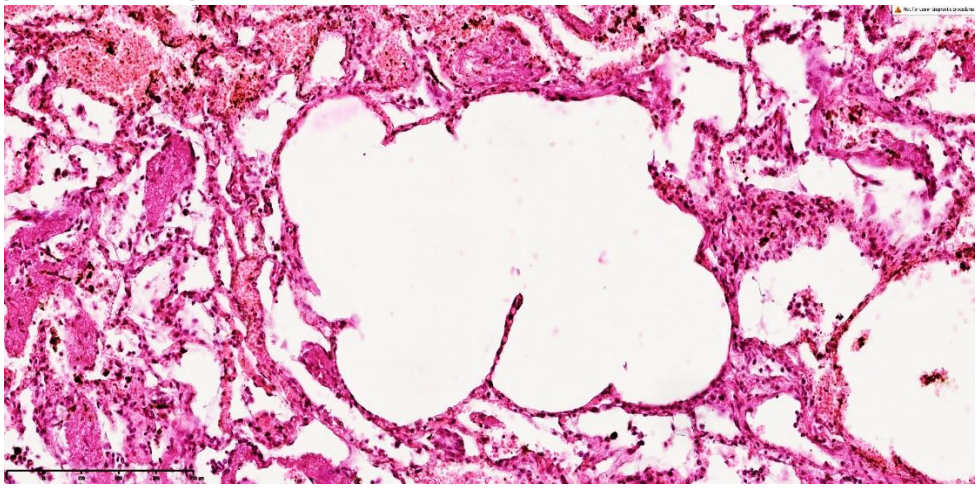

**Figure 4. In bronchioloectatic Leschke emphysema, a centrilobular cystically dilated focus is identified (1), with atelectatic changes in the peripheral alveolar spaces (2). Sclerotic and atrophic alterations are present in the walls of small bronchioles (3). Along the periphery of the cystic dilated focus, inflammatory exudation and features of bronchopneumonia are observed. Staining: H–E. Magnification: 10×10.**

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Thus, the most distinctive morphological characteristic of Leschke emphysema is bronchiectasis predominantly affecting the bronchial walls. From both clinical and morphological perspectives, when polysegmental bronchopneumonia develops—most often in patients aged 12 to 15 years—serious complications such as cor pulmonale, pulmonary hypertension within the small circulatory system, and cardiac asthma are frequently observed. Microscopic examination reveals multiple respiratory bronchioles in the centrilobular zones undergoing cystic emphysematous transformation. Due to pronounced vascular congestion, varying degrees of polysegmental bronchopneumonia are formed, accompanied by atrophic and sclerotic changes in the bronchiolar walls, inflammatory exudation of different intensity, and areas of epithelial desquamation within the mucous membrane of small-caliber bronchioles. In contrast, no essential pathological alterations are detected in medium- and large-caliber bronchi.

These findings suggest that hypoplasia and, in some cases, aplasia of the muscular layer in the walls of small bronchioles are a key pathological substrate, as evidenced by the frequent occurrence of centrilobular cystic dilatations. The main objective of our research is to study the morphological traits of individual bronchial developmental anomalies and, drawing from the obtained histological results, to elaborate practical recommendations.

Tracheoesophageal fistula, as well as congenital stenosis of the bronchi and trachea, generally result from diverse adverse influences acting during the 9th to 12th weeks of intrauterine development, when the esophagus and trachea undergo separation. Disruption of this process may cause partial or complete loss of continuity, producing fistulas or atresias localized proximally, in the middle portion, or distally. Clinically, newborns present with severe manifestations within minutes after birth, which carry a high risk for survival. Reported mortality reaches around 34% within the first 24 hours, increases to 61.3% after one day, and exceeds 75% by the third day, while by the fourth or fifth day it may rise to 90% or higher. A key complication is the onset of aspiration syndrome accompanied by aspiration pneumonia.

From a morphological point of view, tracheoesophageal fistula boundaries are defined by transitional areas where stratified and simple cuboidal columnar

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epithelium converge, frequently exhibiting metaplastic changes. Esophageal contents in such cases may include gastric juice and remnants of maternal milk. Aspirated material is often retained along the inner surface of the tracheal wall, as well as within the lumina of bronchi and bronchioles, where it appears as a homogeneous mass with intense eosinophilic staining. The tracheal mucosa typically demonstrates varying degrees of ciliated epithelial hypoplasia, alongside underdevelopment of the submucosal layer and stromal tissue, with the presence of small glandular alveoli of irregular sizes, reflecting developmental immaturity and congenital structural anomalies (see Figure 5).

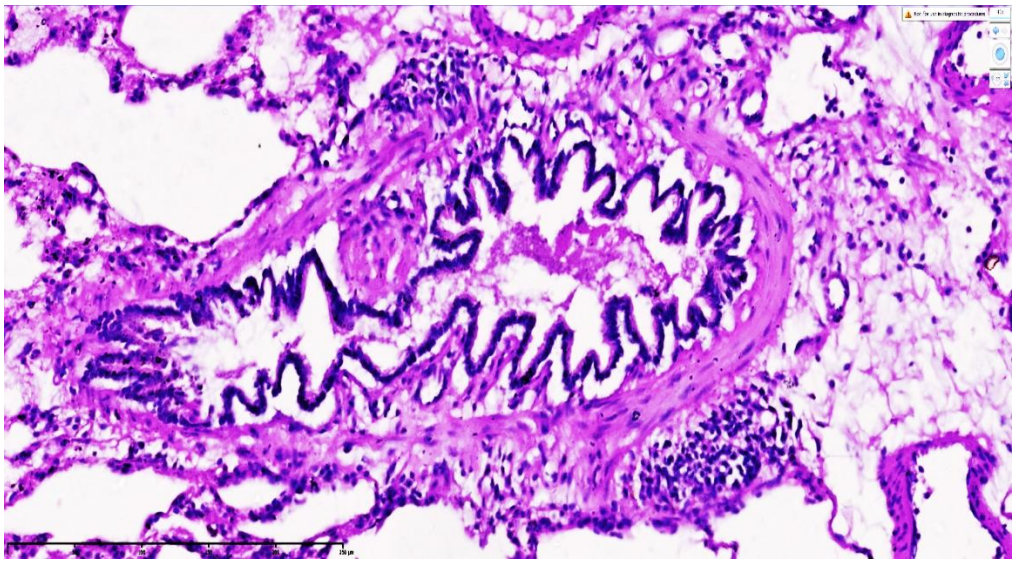

**Figure 5. In hypoplasia of the tracheobronchial tree (TBT), multiple sclerotic and fibrotic areas are observed in the peribronchial regions (1), while the bronchial wall demonstrates a relatively homogeneous and uniform structural pattern (2). Staining: H-E. Magnification: 10×10**.

The ciliated epithelium of the mucous membrane is represented by low cuboidal cells with poorly developed cilia, accompanied by vascular congestion and perivascular edema. Hypoplasia of the tracheobronchial tree (TBT) is evident within the mucosal layer, where germinative centers are absent. The connective tissue in the mucosa does not consist of delicate fibers but instead comprises coarse, irregularly oriented fibers arranged in a disorganized manner, reflecting a clinicomorphological deformity of the bronchi.

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In tracheoesophageal fistula cases, the mucosal layer of the bronchial wall demonstrates retarded development, characterized by epithelial cells of reduced height and volume, and a diminished population of intraepithelial lymphocytes of varying subtypes. Persistent congestion of small blood vessels within the interstitium, interstitial edema, and proliferation of coarse fibrous structures are consistently observed, along with foci of fibroblast and histiocyte proliferation. The submucosal layer is thickened beyond normal, while the glandular alveolar structures are diffusely enlarged. Extensive epithelial metaplasia and focal erosive defects in areas of epithelial migration are also noted (see Figure 6).

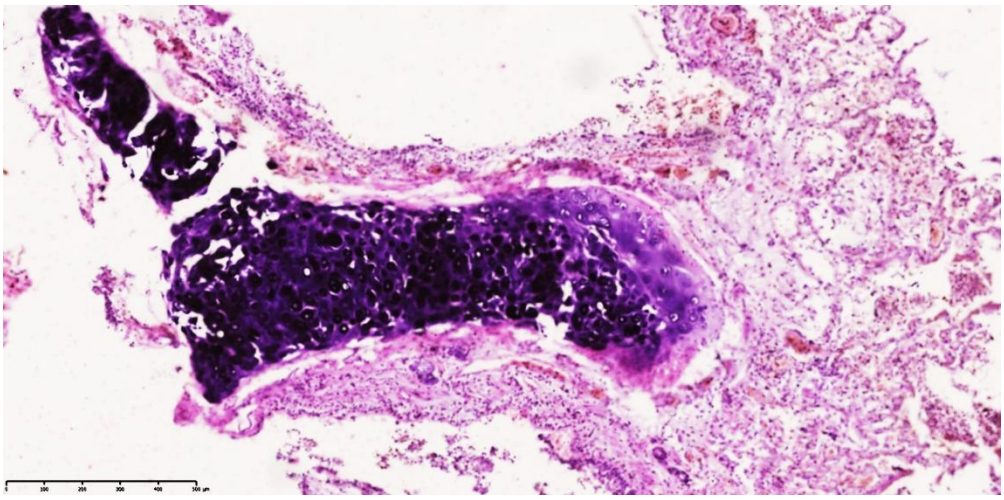

**Figure 6. Fibromatous formations and desquamative areas with scarring extending across all layers of the bronchial wall and mucosal surface (1). Inflammatory infiltrates of mixed cellular composition and interstitial edema of variable intensity are observed (2). The epithelial lining of the mucous membrane demonstrates focal erosive defects (3). Staining: H-E. Magnification: 40×10.**

As a consequence of mucoid degeneration within the submucosal layer, fragmentation of fibrillar components is observed. The connective tissue in the tracheal and bronchial walls demonstrates hypoplasia, accompanied by a morphofunctional reduction in the size of chondrocytes. These chondrocytes exhibit cytoplasmic vacuolization, with insufficient surrounding intercellular matrix and an increased accumulation of acidic mucopolysaccharides. In small-

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caliber bronchioles, a marked proliferation of Clara cells indicates the presence of nonspecific inflammatory foci. Overall, the majority of structural alterations are defined by underdevelopment of the mucosal, submucosal, and muscular layers. Variability in the size and number of smooth muscle cells in the muscular coat corresponds to diminished contractile function of the bronchial wall. The connective tissue is also characterized by low cellularity, persistent vascular congestion, interstitial edema, and ongoing plasma exudation within the interstitial compartment.

With respect to lethality, bronchospasm and bronchiolospasm are evidenced by distortion of the bronchial mucosal architecture, irregular pathways of the respiratory bronchiolar lining, desquamated cellular elements within the lumen, excessive tissue fluid retention, and deposition of homogeneous proteinaceous material. Such pathological changes contribute to the onset of pneumopathy or secondary polysegmental bronchopneumonia in neonates during the early postnatal period, frequently progressing to pulmonary edema. Hence, identifying these distinct morphological features of bronchial developmental anomalies is essential for improving early neonatal diagnosis, guiding prognosis, and reducing mortality.

## Conclusion

The findings of this study underscore the profound pathomorphological disturbances observed in the development of bronchioles and bronchi, particularly in cases of Leschke-type bronchioloectatic emphysema and congenital tracheoesophageal malformations. Structural anomalies such as hypoplasia and aplasia of the bronchiolar muscular layer, formation of cystically dilated centrilobular regions, mucosal desquamation, and inflammatory exudative processes collectively promote the progression of polysegmental bronchopneumonia. From both clinical and morphological perspectives, these alterations are closely associated with the onset of pulmonary hypertension and the development of cor pulmonale.

Congenital defects of the trachea and esophagus, including fistulas and stenotic changes, represent key contributors to upper airway pathology and play a pivotal

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role in the pathogenesis of aspiration syndrome and aspiration pneumonia. Morphological examination has revealed epithelial hypoplasia, zones of metaplasia, erosive changes, fibrosis, and stromal edema within the connective tissue of the lamina propria. These features reflect severe disruption of the structural integrity of the tracheobronchial wall, explaining the high frequency of respiratory dysfunction and elevated neonatal mortality associated with these anomalies.

Furthermore, hypoplasia and aplasia of both the muscular and connective tissue layers, combined with marked vascular congestion and interstitial edema, provide the basis for bronchospasm and bronchiolospasm, culminating in significant impairment of pulmonary ventilation and gas exchange. The pronounced proliferation of Clara cells within small bronchioles further highlights the presence of nonspecific inflammatory reactions and early microscopic evidence of progressive pulmonary injury.

Taken together, these results establish a crucial morphological foundation for the early diagnosis, prognosis, and targeted prevention of bronchial and bronchiolar developmental anomalies. Implementation of timely, morphologically guided surgical interventions—including lobectomy and pneumonectomy—may prevent the transition to recurrent polysegmental bronchopneumonia, mitigate the risk of pulmonary hypertension and cor pulmonale, and ultimately contribute to lowering neonatal morbidity and mortality rates.

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