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Pathomorphological Features of Congenital and Acquired Tracheobronchomalacia

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Abstract: Tracheomalacia is a pathological condition characterized by increased flaccidity of the tracheal or bronchial walls due to underdevelopment or degeneration of the cartilaginous framework, resulting in dynamic airway collapse during respiration. It is classified into two main types: congenital and acquired. Congenital tracheomalacia arises due to developmental anomalies during fetal ontogenesis, while the acquired form may result from prolonged mechanical ventilation, chronic inflammation, or external compression, but importantly, can also originate from prenatal developmental disturbances. This ontogenetic link between congenital and acquired forms has been increasingly recognized in recent studies, particularly when the acquired variant emerges as a consequence of cartilaginous dysplasia during intrauterine development.

From a morphological perspective, tracheomalacia can manifest in various structural forms, including crescentic, bow-shaped, and concentric types. In all types, there is a common feature of anatomical underdevelopment of the tracheobronchial wall layers, especially affecting their cartilaginous and muscular components. In the crescentic and bow-shaped variants, the mucosal integrity is typically preserved, and the primary deficiency lies in the cartilaginous tissue. Histologically, this is expressed by a reduced number of chondrocytes, absence of continuous cartilaginous support along more than three-quarters of the airway circumference, and the presence of small, isolated cartilaginous islets. These anomalies lead to structural instability and dynamic collapse of the airways, exacerbated by contraction of the bronchial smooth muscle bundles.

Understanding the morphogenetic and structural characteristics of tracheomalacia is crucial for accurate diagnosis and appropriate therapeutic decision-making, particularly in neonates and infants presenting with airway obstruction symptoms.

Keywords: Tracheobronchomalacia, airway anomaly, cartilage dysplasia, congenital malformation, acquired malacia, bronchial wall deformation, airway collapse, ontogeny, chondrocyte deficiency.

Introduction: Tracheobronchomalacia (TBM) is a pathological condition characterized by weakness and excessive collapsibility of the tracheal and bronchial walls. It manifests in two main forms: congenital and acquired. Congenital TBM arises due to developmental defects in the formation of the tracheobronchial cartilage and connective tissue during the embryonic and fetal stages, whereas acquired TBM develops secondary to inflammation, prolonged mechanical ventilation, trauma, or other external factors affecting airway integrity [1, 2].

From a pathomorphological perspective, TBM is defined by structural abnormalities in the airway walls, primarily involving cartilage hypoplasia or dysplasia, thinning of the mucosal and submucosal layers, and disorganization of the elastic fibers that normally provide mechanical stability [3, 4]. Congenital TBM often presents as incomplete or weakened cartilage rings, with shapes varying from semicircular and

crescentic to concentric deformities. These alterations result in impaired airway patency and dynamic collapse during respiration [5].

Acquired TBM, on the other hand, is frequently associated with degenerative changes in the tracheobronchial wall components due to chronic inflammation, scarring, or external compression, leading to loss of structural support and similar clinical manifestations [6, 7].

Epidemiologically, congenital bronchopulmonary anomalies, including TBM, account for approximately 4–7% of all congenital malformations in newborns, with significant neonatal mortality rates worldwide. The pathomorphological understanding of TBM is essential for improving diagnostic accuracy and developing effective therapeutic interventions to reduce morbidity and mortality [8, 9].

Given the diverse etiology and complex morphological changes, detailed histopathological studies are critical to differentiate congenital and acquired TBM, to identify their severity, and to guide appropriate clinical management [10].

Research Objective

The objective of this study is to investigate the pathomorphological features of congenital and acquired tracheobronchomalacia, to identify the structural changes in the tracheobronchial walls, and to analyze their clinical significance in relation to neonatal and infant respiratory morbidity and mortality. The study aims to improve the understanding of the developmental mechanisms underlying tracheobronchomalacia and to provide insights for better diagnosis, management, and treatment of affected patients.

METHODS

Data and autopsy materials were collected from 113 cases referred to the Republican Center of Pathological Anatomy with confirmed congenital anomalies. Bronchial and lung tissues were obtained for analysis. Using morphological methods, the tissue samples were stained with hematoxylin and eosin (H&E), and the structural components of the tissues were examined and analyzed.

RESULTS AND DISCUSSION

Tracheobronchomalacia (TBM) is characterized by partial or complete disruption of the integrity of the tracheal cartilage and occurs with an incidence of approximately 1 in 2100 to 1 in 2200 births. Congenital TBM accounts for 5–23% of all cases born with pneumonia, and it is detected in about 50% of autopsy examinations. This condition reflects not a loss of tracheal integrity per se, but rather developmental insufficiency and deformation of the anatomical layers of the tracheal wall. The incidence is significantly higher in males (82%) compared to females (18%), suggesting that TBM may serve as a predisposing factor for the development of chronic obstructive pulmonary disease (COPD) later in life.

Infants born with tracheomalacia often exhibit fewer severe clinicopathological manifestations, which can result in underdiagnosis of the disease. This underdiagnosis increases the risk of various forms of COPD development in the future and aggravates the severity of respiratory infections.

Based on etiology, TBM is classified into two types: congenital and acquired. There is an ontogenetic relationship between these types, with the acquired form also resulting from developmental dysplasia of the tracheobronchial wall during the intrauterine period. Morphologically, TBM can be classified into semicircular, crescent-shaped, and concentric types. All forms manifest as a deficiency of components within the anatomical layers of the tracheobronchial wall.

In semicircular and crescent-shaped tracheomalacia, the mucosal layer remains intact, while the main defect is observed in the connective tissue. Specifically, there is a numerical reduction of chondrocytes and failure of the cartilage wall to occupy approximately three-quarters of its normal structure. Small clefts are observed, accompanied by deformation due to contraction of the bronchial muscular bundles.

In crescent-shaped tracheobronchomalacia, the connective tissue within the bronchial wall shows a deficiency of chondrocytes, a sparse fibrous extracellular matrix, and the presence of coarse fibrous connective tissue around the periphery of the cartilage. Additionally, glandular structures are underdeveloped. These changes result in a marked reduction of the morphofunctional parameters of the ciliated epithelium lining the bronchial mucosa and cause distortion of the annular relief on the inner surface of the trachea and bronchi. Consequently, mucociliary clearance is disrupted, leading to asphyxia and aspiration syndromes.

Pathomorphological investigations detect these pathological changes in only 23–47% of cases. In most patients, areas of tracheal wall deformation flatten after death, which often leads to failure in diagnosing the cause of death (see Figure 1).

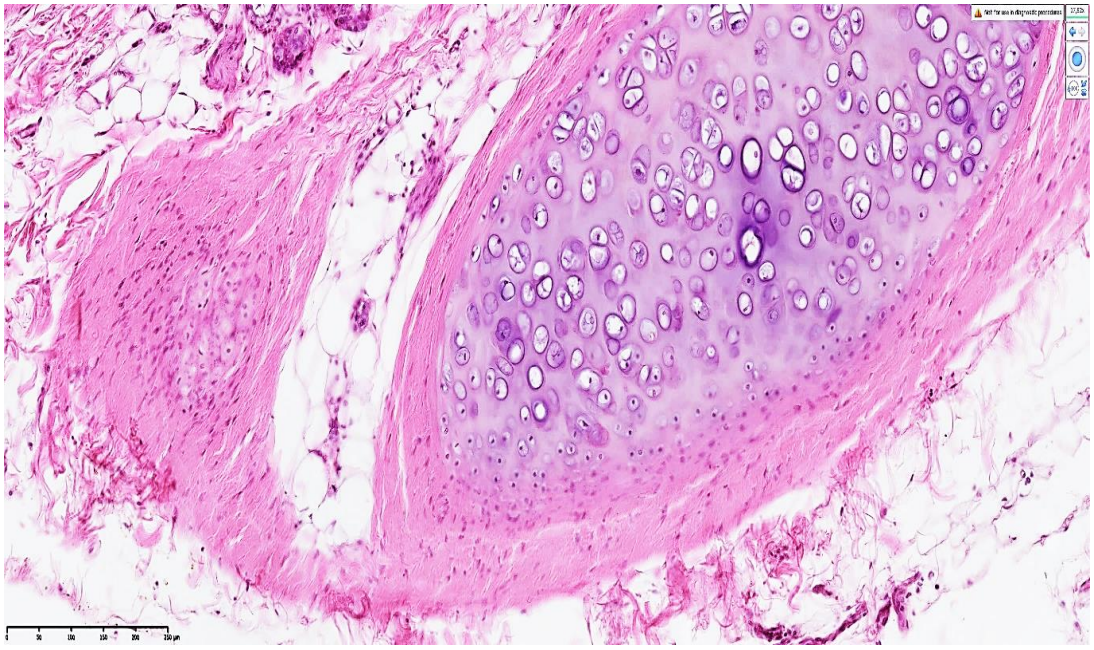

Figure 1. Perisclerosis and fibromatosis of the connective tissue in the bronchial wall in tracheobronchomalacia (1). Various degrees of morphological maturity of chondrocytes within the connective tissue (2). Irregular clustering of chondrocytes (3). Interstitial edema is preserved in the peribronchial area. Staining: Hematoxylin and Eosin (H&E). Magnification: 20x10.

In semilunar tracheobronchomalacia, the primary features manifest as symmetrical developmental dysplasia of the bronchial wall. One of the main characteristics is the underdevelopment of the connective tissue on either the right or left wing of the tracheobronchial wall, which leads to adhesion of the mucous membranes to each other, resulting in friction and erosive lesions on their surfaces. These changes clinically present as aspiration syndrome.

The dysplastic connective tissue contains chondrocytes that are smaller in size, with immature small-nucleated cells still in development. The stroma is filled with a homogeneous interstitial substance, and at the periphery, there is delayed development of the muscle layer and bronchial glands.

In areas of the bronchial wall lacking connective tissue, muscle bundles undergoing myosclerosis are observed. The mucous membrane is incompletely formed, and the epithelial lining on the mucosal surface shows metaplastic changes, characterized by cuboidal and semi-flattened covering epithelium with a marked reduction in cilia (see Figure 2).

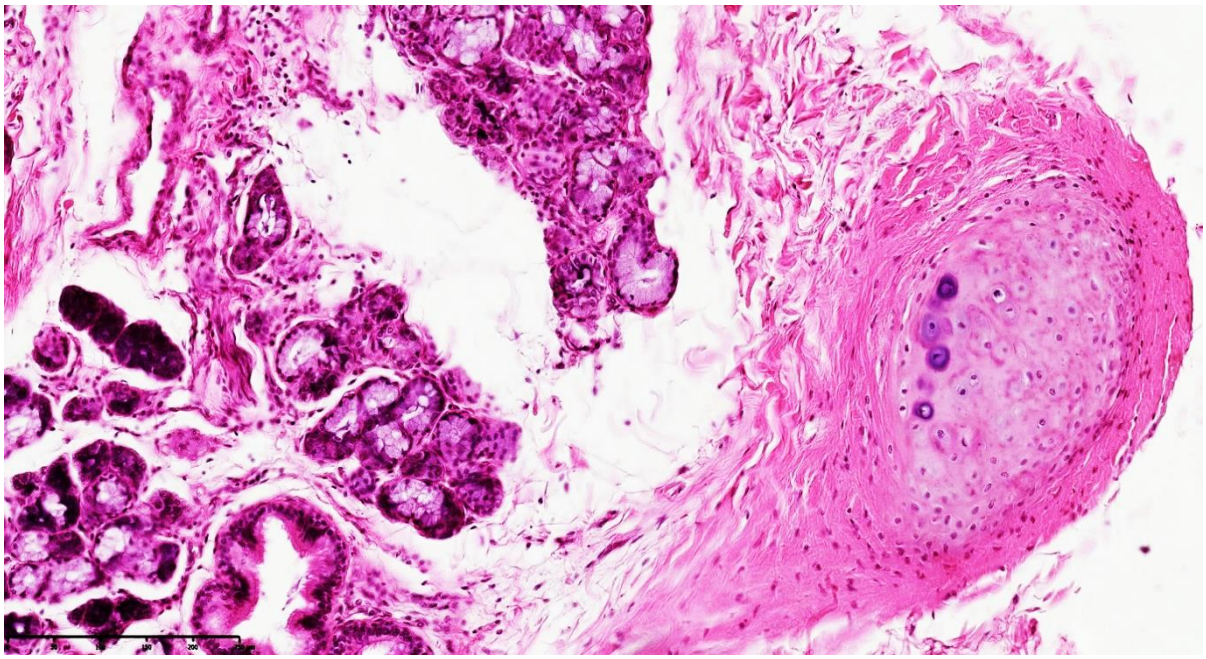

Figure 2. In tracheobronchomalacia, immature chondro-fibromatous and sclerotic connective tissue with morphological signs of dysplasia is observed (1). Bronchial glands exhibit various forms of hypersecretion with areas of calcification (2). There is an increase of sparse and coarse fiber connective tissue in the interstitium, with regions of interstitial edema undergoing organization (3). Staining: Hematoxylin and Eosin (H&E). Magnification: 40x10

This is manifested by the formation of interstitial edema beneath the mucosal layer and the appearance of irregular vascular fullness in the blood vessels.

In the concentric form of tracheobronchomalacia, developmental delay or aplasia is observed across all anatomical layers of the bronchial wall. Foci of multilayered squamous epithelium persist on the mucosal surface, with some elements resembling the esophageal tissue wall. Chondrocytes within the connective tissue are diffusely small in shape, and metachromasia is observed in the mucopolysaccharides of the extracellular matrix in the interstitial spaces (see Figure 3).

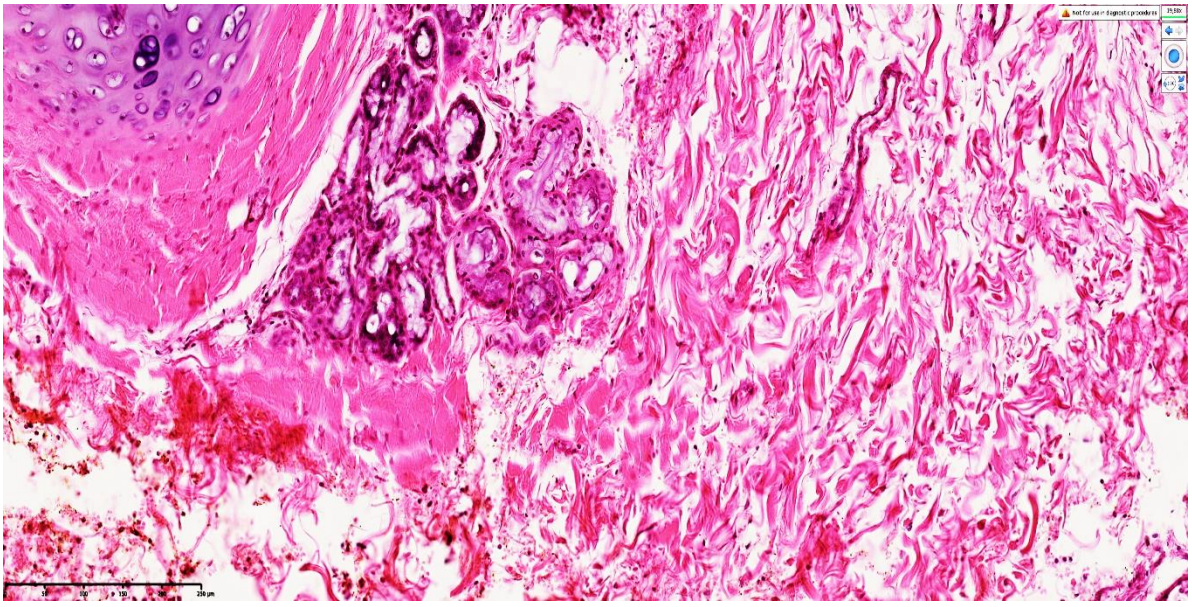

Figure 3. Areas of developing chondro-fibromatosis and sclerosis with morphological signs of dysplasia in the connective tissue are identified (1); hypersecretion of bronchial glands in various forms, with foci of calcification present (2); increased loose and dense fibrous connective tissue in the stroma, with regions of organized interstitial edema observed (3).

Staining: Hematoxylin and Eosin (H&E). Magnification: 20x10.

This, in turn, confirms that the thickening of the bronchial wall, along with the irregular and chaotic formation of fibrous tissue components around the connective tissue, leads to adhesions between the

muscular layer and the basal membrane of the perimysial fascicular membrane. This process results in various forms of muscle bundle atrophy and a reduction in their morphofunctional characteristics. Consequently, it contributes to the macroscopic formation of deformed bronchi. The increase of loose and dense fibrous connective tissue around the perimeter of the connective tissue ring leads to the development of interstitial edema and deformities (see Figure 3).

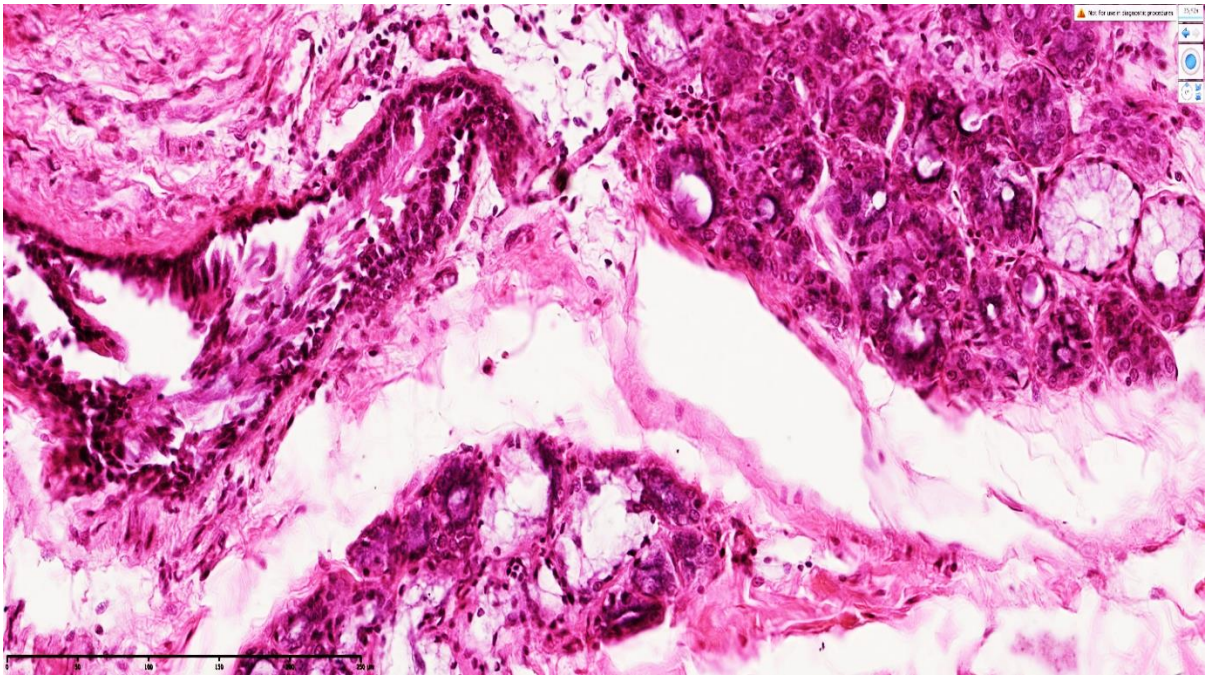

Figure 4. In tracheobronchomalacia, glands exhibit varying degrees of morphofunctional development (1). Interstitial edema and focal inflammatory infiltrates are observed around the glandular perimeter (2). In the interstitial space, mucoid degeneration of loose and dense fibrous connective tissue and accumulation of homogeneous mucus substances are detected (3).

Staining: Hematoxylin and Eosin (H&E). Magnification: 40x10.

Primarily, in areas where the connective tissue of the cartilage is underdeveloped and merges with fibrous connective tissue, foci of hypercellular tissue with small basophilic inclusions and hyperchromatic cells—remnants from the embryonic period—are identified, confirming morphofunctional hypoactivity.

Clinically and morphologically, this manifests as incomplete bronchial dilation, a predisposition to bronchospasm, friction between mucosal layers, and stimulation of asphyxia processes. Consequently, in neonates born during the early neonatal period, this leads to the development of asphyxia and aspiration syndrome, along with impaired mucociliary function of the bronchi.

It is noteworthy that macroscopically in tracheobronchomalacia, bronchial lumen narrowing by one-third, stenosis to half, and contraction to five-fourths of the normal diameter are observed. In our study, nearly 46.6% of examined tracheobronchomalacia cases showed lumen narrowing to half, 31.3% to one-third, and 22.1% to five-fourths of the normal bronchial diameter. Directly related to asphyxia, 68.7% of these cases resulted in death.

The above findings in tracheobronchomalacia are characterized by delayed development of bronchial cartilage, muscle, and mucosal layers during intra- and postnatal ontogenesis, presence of focal chronic inflammatory lesions, abundant loose and dense fibrous connective tissue surrounding cartilage plates, areas of chondro-fibromatosis, and hypercellular foci with embryonic small cells.

Clinically and morphologically, this suggests a high likelihood of aspiration syndrome during the postoperative rehabilitation period following palliative surgical interventions, emphasizing the importance of relying on morphological criteria for treatment planning.

CONCLUSION

Tracheobronchomalacia (TBM) represents a significant pathological condition characterized by the partial or complete loss of structural integrity of the tracheal and bronchial walls, resulting in airway collapse. Its prevalence, as evidenced by epidemiological data, ranges approximately from 1:2100 to 1:2200, with congenital forms accounting for 5–23% of neonatal pneumonia cases and being identified in up to 50% of pathological autopsies. This underscores the frequent underdiagnosis of the condition during life, often due to its subtle clinical manifestations and morphological variability.

The pathogenesis of TBM is multifactorial, involving both congenital and acquired etiologies. Congenital TBM arises from developmental dysplasia of the connective tissue and cartilage components of the airway wall, often leading to incomplete maturation of chondrocytes and aberrant formation of the tracheobronchial framework. Acquired forms are linked ontogenetically to prenatal insults and postnatal inflammatory or mechanical injuries. Morphologically, TBM manifests in several distinct patterns—including semilunar, crescentic, and concentric types—each defined by specific histological changes such as hypoplasia or aplasia of cartilage, peribronchial fibrosis, and muscular atrophy.

Histopathological examination reveals a spectrum of alterations: from immature chondro-fibromatous tissue with varying degrees of fibrosis and sclerosis, to irregular clustering and depletion of chondrocytes within the connective tissue matrix. These changes are frequently accompanied by hyperplasia and hypersecretion of bronchial glands, mucosal epithelial metaplasia, and marked reduction in ciliary function, contributing to impaired mucociliary clearance and predisposing patients to recurrent infections, aspiration syndromes, and potentially fatal asphyxia.

The thickening of the bronchial wall observed macroscopically results from disorganized fibrotic remodeling of connective tissue, which disrupts the normal architecture and induces muscular layer atrophy and functional decline. This fibrosis, particularly around the perimucosal regions, leads to airway stenosis and deformation. These pathological features contribute to the clinical picture of airway obstruction, chronic inflammation, and respiratory insufficiency frequently observed in affected neonates and infants.

Moreover, our findings highlight the significant association between TBM and obstructive pulmonary disease development later in life, notably chronic obstructive pulmonary disease (COPD), especially in male patients where incidence reaches up to 82%. The high prevalence of bronchial narrowing—documented as a reduction in lumen diameter to one-half or less in nearly half of cases—correlates strongly with increased mortality due to asphyxia and respiratory failure.

Given these complex morphofunctional changes, early recognition and accurate morphological assessment of TBM are imperative for guiding clinical management. The persistent hypoplasia and dysplastic changes of the connective tissue, muscular, and mucosal layers underscore the necessity of tailored therapeutic approaches. These may include palliation, surgical intervention, and vigilant post-operative care to mitigate aspiration risk and improve respiratory function.

In summary, tracheobronchomalacia represents a multifaceted developmental and acquired disorder of the airway, where pathological remodeling and structural deficits lead to significant morbidity and mortality in neonatal and pediatric populations. Detailed morphopathological evaluation remains essential for diagnosis, prognosis, and optimizing therapeutic strategies to improve patient outcomes.

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