COMPARATIVE ANALYSIS OF THE ETIOPATHOGENESIS OF CONGENITAL DIAPHRAGMATIC HERNIA IN CHILDREN: UPDATED CLINICAL OBSERVATIONS

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ABSTRACT

This study provides a comparative analysis of the etiological and pathogenetic mechanisms of congenital diaphragmatic hernia (CDH) in newborns. Clinical data from 75 patients treated in an inpatient setting between 2000 and 2025 were analyzed. The study elucidates the complex interplay between visceral dislocation, pulmonary hypoplasia, and pulmonary arterial hypertension based on contemporary neonatal surgical frameworks.

Introduction and Significance. Congenital diaphragmatic hernia (CDH) remains one of the most challenging anomalies in neonatal surgery, characterized by the protrusion of abdominal viscera into the thoracic cavity through a diaphragmatic defect. Despite advancements in high-technology intensive care and surgical techniques, the mortality rate remains significantly high (35-50%). Epidemiological data indicate an incidence of 1 in 2000–2500 live births, with approximately 80% of cases being left-sided. Understanding the precise pathogenetic pathways is crucial for improving survival outcomes.

Objectives. To perform a comparative evaluation and analysis of the specific etiological and pathogenetic features of CDH in pediatric patients to identify key determinants of clinical outcomes.

Materials and Methods. A combined retrospective and prospective analysis was conducted on 75 patients diagnosed with CDH at the specialized neonatal surgery departments between 2000 and 2025. The cohort consisted of 41 girls (55%) and 34 boys (45%). Diagnostic evaluation included clinical, radiological, and statistical modeling.

Results and Discussion. The study confirmed that the embryonic formation of CDH occurs between the 8th and 11th weeks of gestation. The timing of teratogenic impact is a critical factor: exposure during weeks 8–10 leads to "false" hernias (lacking a sac), while exposure after week 11 results in "true" hernias (with a peritoneal sac).

Key findings in the pathogenetic chain include:

Mechanical Compression: Dislocated viscera compress the pulmonary parenchyma and cause a mediastinal shift, compromising contralateral lung development.

Aerophagy Factor: Respiratory distress induces aerophagy, leading to gastrointestinal gaseous distention, which further exacerbates pulmonary compression—a "vicious cycle" in CDH pathophysiology.

Vascular Remodeling: Hypertrophy of the distal muscular layer in hypoplastic pulmonary arteries increases vasoconstrictive reactivity, leading to persistent pulmonary hypertension, hypoxia, and eventually right ventricular failure.

Conclusions. The primary determinant of adverse outcomes in CDH is the degree of pulmonary hypoplasia. Our findings suggest that "pulmonary hypoplasia" should be defined not only by reduced lung volume but as a systemic developmental failure of the terminal bronchioles and alveolar apparatus. Future improvements in treatment outcomes rely on integrating advances in genetic engineering and experimental embryology into clinical practice.

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