PATHOGENESIS OF NEUROBLASTOMA DEVELOPMENT IN CHILDREN

Sur’atova S.Sh.

Tashkent Pediatric Medical Institute, Republic of Uzbekistan,

Tashkent Department of Pathological Physiology

Scientific Advisor: Senior Lecturer G.A. Hakimova

Introduction: Neuroblastoma is among the most common malignancies in the pediatric population. The disease is often difficult to diagnose in its early stages, highlighting the need for advanced and effective treatment strategies. Neuroblastoma is one of the most extensively studied tumors in pediatric oncology, offering opportunities to improve outcomes and quality of life for affected children.
Objective: To investigate the pathogenesis of neuroblastoma in children, with a focus on identifying key cellular, genetic, and molecular abnormalities underlying tumor development.
Materials and Methods: This study is a literature review with elements of analytical evaluation. The research involved examining contemporary national and international scientific sources that explore the molecular, genetic, and cellular basis of neuroblastoma pathogenesis in children. Special emphasis was placed on publications describing the mechanisms of tumor transformation, the embryonic origin of the tumor, and factors influencing disease progression. Sources included peer-reviewed scientific articles, clinical guidelines, and authoritative medical reference materials.
Results: The literature review revealed that neuroblastoma is one of the most prevalent pediatric solid tumors, particularly in children under five years of age, accounting for approximately 14% of all childhood cancers in this age group. The tumor originates from embryonic neural crest cells of the sympathetic nervous system and is most commonly found in the adrenal glands, retroperitoneum, and mediastinum. A key pathogenic mechanism in neuroblastoma is the disruption of neuroblast differentiation, leading to genetic and epigenetic alterations. Among the most significant findings are MYCN gene amplification (present in around 30% of cases), and mutations in the ALK and PHOX2B genes, which are associated with aggressive tumor behavior. Additionally, loss of caspase-8 expression results in impaired apoptosis of tumor cells, contributing to resistance to chemotherapy and radiotherapy. An intriguing feature of neuroblastoma is its potential for spontaneous regression, especially in infants under one year of age. In some cases, the tumor may differentiate into a benign ganglioneuroma or even completely regress without medical intervention. However, the majority of cases are characterized by an

aggressive clinical course with early metastasis to the bone marrow, liver, lymph nodes, and skin. Diagnosis of neuroblastoma involves a comprehensive approach that includes histopathological examination, measurement of urinary catecholamines and their metabolites, and molecular genetic testing to identify prognostic markers. This information is essential for risk stratification and selecting the most appropriate therapeutic approach, which may include surgery, chemotherapy, and radiotherapy.

Conclusion: The pathogenesis of neuroblastoma in children involves a complex interplay of genetic and molecular abnormalities, including MYCN amplification, ALK and PHOX2B mutations, and defects in apoptotic pathways. Given the aggressive nature of the disease and its tendency for early metastasis, early detection is critical for effective treatment planning. Personalized therapy based on the tumor’s genetic profile improves treatment efficacy and contributes to better clinical outcomes and quality of life for pediatric patients.