6. ETIOPATHOGENETIC FRONTIERS OF TROPHOBLASTIC NEOPLASMS: A COMPREHENSIVE REVIEW

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Introduction. Trophoblastic neoplasms, a heterogeneous group of gestational trophoblastic diseases, present intricate challenges in deciphering their etiological and pathogenetic underpinnings. While these rare gestational disorders arise from abnormal proliferation of trophoblastic cells, the precise factors initiating and sustaining this aberrant growth remain elusive.

Aim of study. Considering a high incidence (2.02 out of 1000 pregnancies) of trophoblastic neoplasms amongst women of reproductive age, this constitutes a pressing social problem. By studying the origins and intricacies of this pathology, we could contribute to the understanding and prevention of it. Previous studies have hinted at genetic mutations, hormonal imbalances, and environmental influences as potential contributors to the development of trophoblastic neoplasms. However, a comprehensive understanding of these etiologic factors is paramount for advancing diagnostic precision and therapeutic interventions.

Methods and materials. A systematic literature review was conducted, encompassing studies from databases such as PubMed, Elsevier, and Scopus as well as prominent oncology journals like JCO, JNCI and JGOHR. Emphasis was placed on molecular analyses, genetic studies, and epidemiological investigations to compile a comprehensive overview of the current state of knowledge on the subject.

Results. The etiopathogenesis of trophoblastic neoplasms is multifactorial, involving genetic, epigenetic, and environmental factors. Aberrations in genomic imprinting, particularly in genes associated with trophoblast development, contribute to the dysregulated growth observed in these neoplasms. The genetic factors include the transcription of Factor p63 and Y-Chromosomal Complements. Additionally, disruptions in signaling pathways, such as the transforming growth factor-beta (TGF-β) pathway, play a pivotal role in the pathogenesis. Environmental influences, including nutritional (folic acid deficiency) and hormonal factors (elevated levels of hCG and prolactin), further modulate the risk and progression of trophoblastic neoplasms.

Conclusion. This comprehensive review consolidates current knowledge on the ethiologic aspects of trophoblastic neoplasms. Genetic, molecular, and environmental factors collectively shape the landscape of these gestational disorders, providing potential targets for future research and therapeutic interventions. Understanding the intricate interplay of these factors holds promise for refining diagnostic approaches and developing targeted therapies, ultimately improving patient outcomes. As we unravel the complexities of trophoblastic neoplasms, this synthesis serves as a foundation for further exploration into the intricacies of their etiopathogenesis.