TUMOR PATHOPHYSIOLOGY: ETIOLOGICAL FACTORS, MECHANISMS OF CARCINOGENESIS AND EFFECTS ON THE ORGANISM

Jakhonov Azizbek Kholmirzaevich

(Tashkent Medical Academy, Alfraganus University)

Mamatkulova Dilrukh Fayzullayevna

(Center for Pediatric Hematology, Oncology and Clinical Immunology)

Abstract

Tumor development represents one of the most complex pathological processes in modern medicine. The aim of this article is to analyze the etiological factors, molecular mechanisms of carcinogenesis, and the pathophysiological effects of tumors on the organism. The study is based on the analysis of contemporary scientific literature on oncology, molecular biology, and pathological physiology. The article describes exogenous and endogenous carcinogenic factors, stages of carcinogenesis, and biological characteristics of tumor cells, including uncontrolled proliferation, evasion of apoptosis, metabolic reprogramming, and angiogenesis. The mechanisms of tumor metastasis and its clinical significance are also discussed. Understanding the pathophysiology of tumors is essential for improving early diagnosis, developing effective treatment strategies, and enhancing patient outcomes.

Keywords: tumor, neoplasia, carcinogenesis, oncogenes, metastasis, tumor cachexia, pathophysiology

Introduction Cancer remains one of the leading causes of morbidity and mortality worldwide. Tumor (neoplasia) is a pathological process characterized by uncontrolled cell proliferation resulting from genetic and epigenetic alterations in

the cellular genome. Unlike normal tissues, tumor cells lose regulatory mechanisms controlling cell growth, differentiation, and programmed cell death.

The pathophysiology of tumors involves complex interactions between genetic mutations, environmental carcinogens, immune responses, and metabolic changes within the tumor microenvironment. Over the past decades, advances in molecular biology have significantly improved our understanding of carcinogenesis, revealing the crucial roles of oncogenes, tumor suppressor genes, and signaling pathways in tumor development.

Tumor progression is typically accompanied by several hallmarks including sustained proliferative signaling, resistance to apoptosis, replicative immortality, induction of angiogenesis, and the ability to invade surrounding tissues and metastasize to distant organs. These biological characteristics distinguish malignant tumors from benign neoplasms and are responsible for the severe clinical consequences of cancer.

A comprehensive understanding of tumor pathophysiology is essential for early detection of cancer, development of targeted therapies, and improvement of patient prognosis.

Materials and Methods

This study is based on a comprehensive review and analysis of modern scientific literature related to tumor biology, oncology, and pathological physiology.

Scientific sources were selected from internationally recognized medical textbooks and peer-reviewed publications in the fields of pathology, molecular biology, and oncology. Key textbooks and reference materials included works by Robbins and Cotran, Guyton and Hall, Ganong, Weinberg, and other authoritative researchers.

The methodological approach included:

- literature analysis and synthesis
- comparative analysis of modern oncological theories
- systematization of data regarding etiological factors of tumors
- evaluation of molecular and cellular mechanisms of carcinogenesis

The collected information was analyzed to identify the main mechanisms involved in tumor development, progression, and metastasis.

Results

Etiological Factors of Tumor Development

Tumor formation results from the interaction of multiple carcinogenic factors that induce genetic and epigenetic changes in cells. These factors are generally classified as exogenous and endogenous.

Exogenous Factors

Exogenous carcinogens originate from the external environment and directly or indirectly damage cellular DNA.

Chemical carcinogens include nitrosamines, polycyclic aromatic hydrocarbons such as benzopyrene, and aromatic amines. These substances often require metabolic activation within the body before they can bind to DNA and induce mutations.

Physical carcinogens mainly include ionizing radiation and ultraviolet radiation. Ionizing radiation causes DNA strand breaks and chromosomal abnormalities, whereas ultraviolet radiation induces pyrimidine dimers in skin cells.

Biological carcinogens include oncogenic viruses that integrate into the host genome and alter cellular gene expression. Examples include human papillomavirus (HPV), Epstein–Barr virus (EBV), and hepatitis viruses (HBV and HCV).

Endogenous Factors

Endogenous carcinogenic factors originate within the organism itself.

Genetic predisposition plays an important role in tumor susceptibility. Mutations in tumor suppressor genes such as p53 and RB disrupt normal cell cycle regulation.

Hormonal imbalance also contributes to tumor development, particularly in hormone-dependent tissues such as the breast, prostate, and thyroid gland.

Additionally, impairment of immune surveillance may allow transformed cells to evade detection and proliferate.

Molecular Mechanisms of Carcinogenesis

Carcinogenesis is a multistep process involving the accumulation of genetic alterations that transform normal cells into malignant ones.

Three main stages are recognized:

Initiation

During this stage, irreversible genetic mutations occur in cellular DNA as a result of exposure to carcinogenic factors.

Promotion

Mutated cells undergo selective clonal expansion under the influence of promoting factors such as chronic inflammation or hormonal stimulation.

Progression

The final stage is characterized by increased genetic instability, anaplasia, invasive growth, and metastatic potential.

Activation of oncogenes and inactivation of tumor suppressor genes represent the central molecular events in carcinogenesis.

Pathophysiological Features of Tumor Cells

Tumor cells exhibit several unique biological properties that distinguish them from normal cells.

One of the most important characteristics is uncontrolled proliferation, resulting from dysregulation of the cell cycle.

Another key feature is evasion of apoptosis, which allows abnormal cells to survive despite genetic damage.

Tumor cells also demonstrate metabolic reprogramming, known as the Warburg effect, in which glycolysis predominates even in the presence of oxygen.

Furthermore, tumors stimulate angiogenesis, the formation of new blood vessels, to ensure sufficient nutrient and oxygen supply.

Immune evasion mechanisms also play an important role in tumor survival and progression.

Metastasis and Systemic Effects

Metastasis represents the most critical feature of malignant tumors and is responsible for the majority of cancer-related deaths.

The metastatic cascade involves several sequential steps:

1. invasion of surrounding tissues

2. intravasation into blood or lymphatic vessels
3. survival in circulation
4. extravasation into distant tissues
5. colonization and formation of secondary tumors

In addition to local tissue damage, tumors exert systemic effects on the organism. These include cancer cachexia, anemia, immune suppression, and paraneoplastic syndromes.

Discussion

The development of tumors is a complex biological process involving interactions between genetic alterations and environmental factors. Advances in molecular oncology have revealed numerous signaling pathways and regulatory mechanisms that contribute to tumor initiation and progression.

Understanding the molecular basis of carcinogenesis has led to the development of targeted therapies aimed at specific oncogenic pathways. Examples include inhibitors of tyrosine kinases, immune checkpoint inhibitors, and monoclonal antibodies targeting tumor-associated antigens.

Despite significant progress in cancer research, metastasis and tumor heterogeneity remain major challenges in oncology. Continued research is necessary to identify novel biomarkers for early detection and to develop more effective therapeutic strategies.

Conclusion

Tumor pathophysiology involves complex biological processes ranging from genetic mutations at the cellular level to systemic effects on the entire organism.

A deeper understanding of carcinogenic mechanisms provides essential insights for improving cancer prevention, early diagnosis, and treatment strategies. Advances in molecular medicine and targeted therapy offer promising prospects for more effective management of oncological diseases in the future.

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