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BIOCHEMICAL MARKERS OF CANCER: OPPORTUNITIES FOR DIAGNOSTIC APPLICATION

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Abstract

Cancer represents one of the most widespread and challenging diseases in contemporary medicine. Biochemical markers play a crucial role in the early detection of malignant processes, evaluation of disease prognosis, and the development of personalized therapeutic strategies. This article examines a broad range of biochemical markers associated with cancer, focusing on their classification, molecular origins, and clinical relevance. Particular attention is given to protein-based markers, including carcinoembryonic antigen, alpha-fetoprotein, and cancer antigen one hundred twenty-five, as well as genetic and epigenetic indicators such as mutations in tumor suppressor and oncogenes, and alterations in deoxyribonucleic acid methylation patterns. In addition, metabolic changes characteristic of malignant transformation, including increased lactate production and disturbances in glucose metabolism, are analyzed in relation to their diagnostic value. The sensitivity, specificity, and practical applicability of these markers are discussed, along with their role in current diagnostic systems and potential future developments. The analysis is based on data from international scientific literature and aims to provide an in-depth overview of modern biochemical approaches in oncology.

Keywords:

Biochemical markers, tumor markers, oncological diagnostics, carcinoembryonic antigen, alpha-fetoprotein, cancer antigen one hundred twenty-five, prostate-specific antigen, breast cancer susceptibility genes one and two, Kirsten rat sarcoma viral oncogene homolog, epidermal growth factor receptor, deoxyribonucleic acid methylation, epigenetic markers, genetic mutations, metabolic biomarkers, Warburg effect, lactate, glycolysis, micro ribonucleic acid, prognostic markers, predictive biomarkers, oncogenes, tumor protein fifty-three, biomolecular diagnostics, individualized therapy, liquid biopsy, oncological screening, immunodiagnostics, monitoring of treatment response, preclinical diagnostics, personalized medicine.

Introduction

Cancer is a complex pathological condition characterized by dysregulated cell division and the ability of malignant cells to invade surrounding tissues, and it represents one of the most pressing challenges for global healthcare systems. According to data from the World Health Organization, nearly twenty million new cases of cancer are diagnosed annually, and more than ten million patients die from this disease each year. Early detection and effective control of cancer largely depend on a thorough understanding of its molecular and biochemical foundations.

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In recent years, the rapid development of medical biochemistry and molecular oncology has led to extensive research on biochemical markers used in cancer diagnosis, commonly referred to as tumor markers. These markers are molecules that reflect alterations in biological processes within the body and are typically detected in blood, urine, or other biological fluids. They are produced either directly by cancer cells or as part of the host's biological response to malignant transformation.

Biochemical markers play an important diagnostic, prognostic, and predictive role in the early detection of cancer, determination of disease stage, evaluation of therapeutic response, and monitoring of recurrence risk. For instance, alpha-fetoprotein is considered a key marker for hepatocellular carcinoma, prostate-specific antigen for prostate cancer, and carcinoembryonic antigen for colorectal malignancies.

In addition, genetic and epigenetic markers, including mutations in breast cancer susceptibility genes one and two, Kirsten rat sarcoma viral oncogene homolog, and epidermal growth factor receptor, as well as ribonucleic acid–based biomarkers and metabolic indicators such as lactate, provide valuable insights into the molecular dynamics of cancer development. These advances enable oncological diagnostics to achieve significantly higher accuracy and sensitivity compared with traditional diagnostic approaches, thereby contributing to the progress of personalized and precision medicine.

Etiology

Cancer is a disease with a multifactorial etiology, arising from a complex interaction of genetic, epigenetic, and environmental factors. The development of cancer is initiated by alterations in the cellular genetic material, including mutations in deoxyribonucleic acid, chromosomal abnormalities, and epigenetic modifications. Such changes disrupt the normal mechanisms regulating cell growth, division, and apoptosis.

Among genetic factors, the activation of oncogenes such as Kirsten rat sarcoma viral oncogene homolog and MYC, together with the inactivation of tumor suppressor genes including tumor protein fifty-three and retinoblastoma gene one, represents key events in carcinogenesis. Mutations affecting these genes lead to uncontrolled cellular proliferation and loss of growth control. Epigenetic alterations, particularly aberrant deoxyribonucleic acid methylation, histone modifications, and dysregulation of micro ribonucleic acid expression, alter gene activity without directly damaging the genetic sequence. Hypermethylation of tumor suppressor genes results in their functional silencing, thereby promoting abnormal cell growth and survival.

In addition, environmental factors such as ultraviolet radiation, chemical carcinogens including benzene and nitrosamines, oncogenic viruses, unhealthy dietary habits, tobacco use, and chronic inflammation are recognized as important contributors to cancer development. From a biochemical perspective, these etiological factors disrupt cellular metabolic homeostasis. Cancer cells differ markedly from normal cells in their energy metabolism, relying predominantly on glycolysis for energy production even under aerobic conditions, a phenomenon known as the Warburg effect. This metabolic shift leads to increased lactate production, alteration of the tumor microenvironment, and enhanced tumor invasion and progression

Thus, the etiology of cancer encompasses multiple interacting factors that disturb genetic regulation and metabolic activity of the cell, resulting in characteristic biochemical alterations

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that form the basis for the emergence and clinical application of cancer-associated biochemical markers.

Epidemiology

Cancer is a major global public health problem, and its early detection remains a key factor in reducing mortality and improving treatment outcomes. In this context, biochemical markers play an essential role in oncological diagnostics by enabling the identification of malignancy, assessment of disease stage, and prediction of prognosis. Biochemical markers reflect molecular and metabolic alterations associated with cancer development. Genetic and epigenetic markers are particularly important in epidemiological studies. Mutations in tumor protein fifty-three are frequently observed in various malignancies, including lung, skin, and esophageal cancers. Alterations in breast cancer susceptibility genes one and two are well-established risk factors for breast and ovarian cancers. Mutations in the Kirsten rat sarcoma viral oncogene homolog are commonly detected in colorectal, lung, and pancreatic cancers and are associated with disease progression.

Protein-based markers are widely used in clinical practice. Carcinoembryonic antigen is detected in colorectal, lung, and gastric cancers, while cancer antigen one hundred twenty-five is primarily applied in ovarian cancer. Alpha-fetoprotein serves as a key marker for hepatocellular carcinoma, and cancer antigen nineteen-nine is associated with pancreatic and other gastrointestinal malignancies. In addition, next-generation markers such as Ki-67 and human epidermal growth factor receptor two are increasingly used for prognostic evaluation and treatment stratification. Recent epidemiological studies emphasize the growing importance of biochemical markers in early cancer detection. Large-scale population studies have demonstrated that certain plasma proteins can be detected several years before the clinical manifestation of cancer. Moreover, an increasing incidence of cancer among individuals under fifty years of age has been reported, particularly for breast, colorectal, kidney, and uterine cancers. These trends are likely related to lifestyle, dietary habits, and environmental factors.

Pathophysiology

Cancer is characterized by uncontrolled cell proliferation, resistance to apoptosis, and the capacity for invasion and metastasis. These pathological features are accompanied by profound molecular and metabolic alterations that give rise to specific biochemical markers. One of the key metabolic changes in cancer cells is the preferential use of glycolysis for energy production, even under aerobic conditions, known as the Warburg effect. This shift leads to increased glucose consumption and elevated lactate production, resulting in higher levels of lactate dehydrogenase in the blood. Elevated lactate dehydrogenase levels are observed in many malignancies and are often associated with advanced disease and poor prognosis. Genetic alterations play a central role in cancer pathophysiology. Mutations in oncogenes and tumor suppressor genes, including Kirsten rat sarcoma viral oncogene homolog, BRAF, epidermal growth factor receptor, human epidermal growth factor receptor two, and breast cancer susceptibility genes one and two, disrupt normal cellular signaling pathways and promote malignant transformation. These mutations are clinically relevant, as they influence tumor behavior and therapeutic response. Cancer cells and the host organism produce specific molecules that serve as biochemical markers. Elevated carcinoembryonic antigen levels are observed in colorectal, lung, breast, and gastric cancers and correlate with tumor differentiation. Increased alpha-fetoprotein levels indicate hepatocellular carcinoma and certain germ cell tumors, while cancer antigen one hundred twenty-five reflects tumor burden and disease spread in ovarian cancer. Other markers, such as mesothelin and S100 proteins, provide additional information regarding tumor invasiveness and cellular origin. Furthermore, cancer-associated

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metabolic reprogramming affects lipid, amino acid, and nucleotide metabolism. Alterations in glutamine and serine metabolism support rapid tumor growth, while changes in lipid metabolism contribute to invasion and metastasis. In summary, the pathophysiological mechanisms of cancer involve complex genetic and metabolic disturbances that lead to the formation of characteristic biochemical markers. These markers are of great value for early diagnosis, monitoring of treatment response, and prognostic evaluation, particularly when used in combination with other diagnostic methods.

Treatment

Current cancer treatment strategies are increasingly based on personalized and biomarker-driven approaches. Therapeutic decisions are guided by the patient's genetic profile, tumor histology, and molecular characteristics, allowing for more precise and effective interventions. The integration of genomic testing with histopathological analysis has significantly improved treatment selection and clinical outcomes.

Personalized and Targeted Therapy

Personalized therapy involves the identification of specific molecular alterations, such as epidermal growth factor receptor, anaplastic lymphoma kinase, and breast cancer susceptibility gene mutations, to guide targeted treatment selection. This approach enables the development of highly individualized drug regimens, improves therapeutic efficacy, and reduces unnecessary toxicity.

Immunotherapy

Immunotherapy enhances the ability of the immune system to recognize and eliminate cancer cells. Immune checkpoint inhibitors targeting programmed cell death protein one, programmed death-ligand one, and cytotoxic T-lymphocyte-associated protein four have demonstrated substantial clinical benefit in multiple malignancies. Biomarker-based selection of patients is essential for optimizing treatment response and minimizing adverse effects.

Nanotechnology and Innovative Drug Delivery

Nanotechnology-based therapies facilitate selective delivery of anticancer drugs to tumor tissues, improving targeting accuracy and reducing systemic toxicity. These approaches also enable real-time monitoring of therapeutic response through integrated biosensors, further enhancing treatment precision.

Recent Advances and Future Perspectives

Recent developments in cancer therapy emphasize the growing role of immunotherapy, personalized medicine, and novel technological approaches. Innovations such as rapid immunotherapy formulations, messenger ribonucleic acid–based cancer vaccines, advanced radiotherapy techniques, and molecularly targeted drugs have improved treatment effectiveness and patient quality of life.

In summary, modern cancer treatment relies on the integration of biochemical markers, molecular profiling, and advanced technologies. These approaches enable individualized therapy, improve treatment outcomes, and represent a key direction in the future of oncology.

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Conclusion

Biochemical markers play a pivotal role in modern oncology, providing critical insights into cancer diagnosis, prognosis, and therapeutic management. Genetic, epigenetic, protein-based, and metabolic markers not only facilitate early detection but also guide personalized treatment strategies, including targeted therapy, immunotherapy, gene editing, and RNA-based interventions. Advances in nanotechnology and artificial intelligence further enhance the precision and effectiveness of cancer care.The integration of molecular profiling with clinical and histopathological data allows for individualized treatment plans, improving patient outcomes and reducing unnecessary toxicity. Emerging therapies, including cancer vaccines, novel drug combinations, and advanced radiotherapy techniques, highlight the rapid evolution of oncology toward more personalized and effective approaches. In summary, the continued development and application of biochemical markers and molecular technologies hold the promise of transforming cancer management, enabling earlier detection, more precise interventions, and improved survival for patients worldwide.

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