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Opinion

The Cancer Treatment is not based only on Histology: The Genetic Mutation Pathway

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Abstract

Cancer treatment is primarily based on histological examination and related to the organ in which it develops (e.g. colon adenocarcinoma or squamous cell carcinoma of the lung). While this is a starting point, it cannot be considered the sole basis for treatment. The discovery of genetic mutations in neoplastic cells, whether somatic or germline, is a valuable aid in the development of innovative drugs. These drugs can be used regardless of the organ, facilitating clinical research and application.

Kew Words: cancer; histology examination; genetic mutation; innovative drugs; clinical research

Introduction

The study of tumour characteristics can no longer be limited to histology. While histology remains a fundamental reference, it is not the only useful parameter for defining treatment. Integrating new evaluation criteria, such as genetic alterations, is paving the way for more targeted and effective therapies [1].

Identifying genes associated with the differentiation, growth, proliferation, spread and metastasis of cancer cells has revealed new potential therapeutic targets. Understanding the genetic mechanisms underlying mutations that cause neoplastic transformation or uncontrolled proliferation will guide us towards developing drugs that can interfere with these processes and lay the foundations for truly personalised treatment. [2-3-4]

The alterations in gene expression that underlie tumour transformation are not random. The notion that a neoplasm is the result of 'bad luck' is being replaced by an understanding of the role played by intracellular and extracellular factors, known as epigenetic factors [5-6]. Over time, toxic substances, viruses or bacteria can contribute to the onset of cancer. In such cases, the resulting mutations can be targeted by therapy [7-8].

Traditionally, the therapeutic approach has focused on the morphological aspect of cells, bypassing the cause of transformation. However, a mutation in an oncosuppressor gene may not alter the appearance of the affected organ despite being the real driver of the disease.

In such cases, targeting the mutation directly rather than the organ may offer greater clinical benefit.

Currently, the use of targeted drugs is often still decided on the basis of the affected organ, even when the target mutation is present in tumours in different locations. A paradigm shift is needed, whereby tumours are classified based on all mutations present, regardless of the organ of origin. [9]

This approach would enable different tumours to be treated with the same drugs where they share the same molecular alterations. It would also speed up the development and validation of drugs by making it easier to enrol patients with the same mutation, rather than creating subgroups based on organ histology. [10]

At the heart of precision oncology is a gene mutation-targeted therapy, independent of tissue type. Rather than testing every drug on every histological tumour type, it will be sufficient to demonstrate its efficacy on mutations common among different neoplasms.

Discussion

This article explores multiple solutions for defeating cancer, with a focus on the potential evolution of personalised cancer therapy that considers genetic mutations affecting various organs, including those based on epigenetics.

Conclusions

In conclusion, the evolution of cancer therapy must involve personalised treatment. It is essential to recognise the epigenetic mechanisms that alter normal cell proliferation in order to use truly effective drugs that target

not only the morphological aspect of the tumour, but also the root causes of neoplastic transformation.

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