International Journal of Medicine and Medical Sciences and ISSN 2167-0404 Vol. 13(1), pp.001, March, 2023.
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Perspective

Ethnic Considerations in Cancer Treatment: Finding Specific Biomarkers for Treatment Response

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Received: 01-Mar-2023, Manuscript No. IJMMS-23-92157; Editor assigned: 03-Mar-2023, Pre QC No: IJMMS-23-92157 (PQ); Reviewed: 17-Mar-2023, QC No: IJMMS-23-92157; Revised: 24-Mar-2023, Manuscript No: IJMMS-23-92157 (R); Published: 31-Mar-2023

DESCRIPTION

Certain molecules that are often overexpressed or mutated in cancer cells are targeted by molecular-targeted medicines. As a result, these medications are often thought to target cancer cells precisely, leading to fewer Adverse Drug Reactions (ADRs). Nonetheless, molecularly focused medications can still result in typical ADRs that, while uncommonly severe, can be fatal. Hence, it is critical to be able to identify individuals who are likely to have adverse events after receiving molecularly targeted medication. Pharmacogenetics is a new area that seeks to better separate the genetic variations linked to medication toxicity and effectiveness in order to enhance the selection of treatment approaches for each genetic profile.

A rapidly expanding number of cancer treatment options are flooding the medical sector, including the new class of cytotoxic medications, molecularly targeted therapies, and immune checkpoint inhibitors used in conjunction with chemotherapy and radiation. Although chemotherapy regimens have significantly improved in recent years and are still a popular treatment option, there is still a lot of variation in their effectiveness and toxicity among different patients, as well as physical and mental distress, a reduction in patient Quality Of Life (QOL), and a wide range of common Adverse Drug Reactions (ADRs). Although it is obviously preferable to choose medications that have the greatest therapeutic effect with the least amount of adverse drug reactions (ADR), stratified cancer treatment is still in its infancy, and what is often referred to as "personalised or precision medicine" is still largely based on trial and error.

Adverse drug reactions and molecularly targeted therapy in oncology

A more recent form of anticancer medication is known as a molecularly targeted medicine. The notion of therapy selection among cancer patients has undergone a significant change as a result of the more recently produced molecular-targeted medications, which are based on tumour molecular profiling. As these medications are made to interfere with the production of genes (proteins) that are often overexpressed or mutated in cancer cells, they are thought to selectively target cancer cells, resulting in fewer adverse drug reactions.

A humanised monoclonal antibody called Trastuzumab, often known as Herceptin, is used to treat tumours that express the EGFR type 2 (HER2) genes. Trastuzumab binds to the extracellular domain of HER2, and blocks the activation of HER2 signalling, producing Antibody-Dependent Cellular Cytotoxicity (ADCC). Nonetheless, cardiotoxicity is one of the most dangerous adverse effects of trastuzumab, with around 5% of patients seeing a reduction in left ventricular ejection fraction. The HER2-encoding gene, ERB-B2 receptor tyrosine kinase 2 (ERBB2), has so received a lot of attention in the search for polymorphisms linked to trastuzumab-induced cardiotoxicity. Particularly in White people, trastuzumab-induced cardiotoxicity is related with the germline polymorphism.

Sunitinib is a small-molecule multikinase inhibitor that targets a number of receptor tyrosine kinases, such as the Kit receptor, Fms-Like Tyrosine kinase-3 receptor (FLT3), platelet-derived growth factor receptors (PDGFR and PDGFR), and the receptor encoded by the ret proto-oncogene. 41 ADRs associated with multikinase inhibitors, such as sunitinib, include liver damage, hypertension, diarrhoea, mucositis, myelotoxicity, and hand-foot syndrome.

The discovery of genetic variations that could serve as biomarkers for severe ADRs has been considerably aided by candidate gene- and genome-wide association studies. There is a need to evaluate and corroborate the connections between these genetic variations and ADRs because the data to far on the possible use of ADR-related biomarkers in cancer treatment is conflicting. Moreover, it is crucial to find ethnically specific biomarkers for treatment response.

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