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Opinion Article

Immune-Related Genetic factors for Determining Imatinib Therapy Outcome in Persistent Myeloid Lymphoma

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DESCRIPTION

Chronic Myeloid Leukemia (CML) is a cancerous condition with uncontrolled proliferation that develops from hematopoietic stem cells. Tyrosine Kinase Inhibitors (TKIs), particularly Imatinib (IM), which has demonstrated efficacy, are frequently used in the treatment of CML. TKI use, however, can lead to adverse effects and drug resistance, which can diminish its efficacy in some patients. Therefore, it's crucial to comprehend how imatinib therapy works and find potential indicators. Tumor development depends heavily on the immune system.

The importance of anti-tumor immunity in cancer treatment has been underscored by recent developments in cancer immunotherapy, including the use of modified T cells with chimeric antigen receptors or immune regulation utilizing antibodies targeting the PD-1/PD-L1 pathway. It has long been known that the immune system may control CML, as seen by therapies like interferon alpha, donor lymphocyte infusion, and allogeneic hematopoietic stem cell transplantation. Consequently, leveraging immune regulation may present a viable strategy for totally curing CML.

Gene expression patterns have been investigated in several studies to distinguish between patients who benefit from imatinib treatment and those who do not. Immune-Related Genes (IRGs) in CML may or may not have clinical significance, although this is not yet established. Imatinib has a high level of clinical efficacy and safety as a first-line therapy for CML. One of the biggest clinical issues with imatinib resistance, nevertheless, continues to be CML therapy. A considerable number of patients in the historic phase 3 IRIS imatinib study achieved Complete Cytogenetic Remission (CCR), although 17% of patients developed resistance after 5 years of follow-up. In order to choose the best course of action for further treatments, clinical practice requires routine monitoring of treatment response in CML. Therefore, current objectives for research on tyrosine kinase inhibitor therapy include the identification of response or resistance biomarkers and examination of molecular pathways to direct individualized interventions.

In this study, the immune scores of CML patients were determined using the ESTIMATE algorithm, and it was discovered that both immune scores and IRGs can be used to predict how well an imatinib treatment will work. By comparing DEG analyses between groups with high and poor immunological scores, 10 hub genes using WGCNA has been identified. A prior study of CML patients with imatinib resistance found that one of these hub genes, PRKCH, had its levels altered. Another investigation into therapeutically targetable mechanisms found that PRKCH were controlled by increased SRSF1 levels, which in turn affected their ability to respond to the drug imatinib. The roles of *CD7* and *BCL11B* were also revealed in a number of researches.

Haplo insufficiency and acquired loss of *BCL11B*, which functions as a tumor suppressor, work with *p210BCR/ABL* to cause CML blast crisis. These pertinent research or bits of proof may offer justification for the potential utility of immunological score and IRGs as predictive biomarkers for medication response. The different endpoints could produce less-than-ideal validation results. The study's sample size, however, might not be sufficient to warrant the broad use of the predictors in clinical practice. In order to conduct additional research, a unified case cohort and a bigger sample size are required. Though clinically, the widespread use of next-generation TKIs like nilotinib or dasatinib raises the issue of drug resistance as well. Our study focused on imatinib response. The selection of effective clinical treatment plans for CML will be aided by further investigation into additional TKI resistance indicators.

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