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Perspective

The Role of Leukemia-Specific Immunotherapy in Targeting Non Metastasis Protein 2 (NME2)

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DESCRIPTION

A reciprocal translocation t(9;22) (q34;q11) fusing the *BCR* and *ABL1* genes on the Philadelphia chromosome (Ph+) is the hallmark of Chronic Myeloid Leukemia (CML). A constitutive-active chimeric non-receptor tyrosine kinase is encoded by the ensuing *BCR::ABL1* oncogene. Understanding the molecular pathomechanisms of CML has facilitated the development of particular *BCR-ABL1* Tyrosine Kinase Inhibitors (TKI), which has transformed the disease and is currently recognized as the gold standard for first-line CML treatment. Still, some patients fail to receive the intended response in spite of these remarkable advancements. After a year of 400 mg of imatinib treatment, the rates of Major Molecular Response (MMR) varied from 18% to 58%. During the 8-year follow-up period, 45% of the patients included in the IRIS stopped taking imatinib therapy. Mutations in the *BCR::ABL1* gene, which are seen in about 30% of patients who experience treatment failure in the chronic phase, frequently lead to resistance to TKI. Treatment with imatinib has few side effects. Even among responding patients, there is a great deal of interest in stopping treatment early in order to facilitate pregnancy or just to save money on the expensive long-term TKI medication. There are published trials showing that stopping imatinib can result in a long-lasting molecular remission. TKIs, however, are unable to eradicate leukemic cells that remain after a short to medium period of time, and multicenter trials continue to maintain that imatinib should only be stopped in the context of clinical trials and only in patients who have established and maintained a sustained deep molecular response for two or three years. More than 50% of patients who stopped using imatinib experienced an early molecular recurrence, even in this patient group. These factors make allogenic Hematopoietic Stem Cell Transplantation (HSCT) a viable and effective treatment option for a minority of CML patients. Leukemia reactive cytotoxic T-lymphocytes, which target tumor antigens or minor histocompatibility antigens and may be able to remove residual illness, are the primary means by which HSCT in CML produces a Graft-*Versus*-Leukemia (GVL) effect. In clinical practice, Donor Lymphocyte Infusion (DLI) can be utilized to enhance this effect. This implies that cancer vaccination techniques, such as the delivery of particular antibodies, antigenic peptides, peptide-loaded dendritic cells, Chimeric Antigen Receptor (CAR) T-cells, CAR-NK cells, or dead tumor cells, may enhance both TKI treatment and HSCT. The capacity of cytotoxic T lymphocytes to identify Tumor Associated Antigens (TAA) is a critical factor in the effectiveness of an anticancer immune response. Theoretically, vaccination strategies can be used to specifically activate and amplify these cells, and efforts are now underway to stimulate T cell responses specific to TAA against Wilms Tumor Protein 1 (WT1) and the PR1 epitope from proteinase 3 (PR3). Another tactic for CML therapy could be to target newly discovered TAAs in CML, such as telomerase, Hyaluronic Acid Mediated Motility (RHAMM), CML-66, CML-28, CML-Ag165, PPP2R5C, ELA2, PRAME, MPP1, and Aur-A. Studies using a peptide vaccination generated from *BCR::ABL1* (e14a2) have demonstrated some benefit.

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