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Opinion Article

The Effectiveness of Imatinib in B-Cell Acute Lymphoblastic Leukemia

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DESCRIPTION

One high-risk subtype of B-ALL is Philadelphia chromosome-positive B-cell Acute Lymphoblastic Leukemia (Ph+ B-ALL). About 30% of instances of Acute Lymphoblastic Leukemia (ALL) in young adults and more than 50% of ALL cases in people over 50 are Ph+ B-ALL cases. The clinical prognosis of B-ALL patients has greatly improved due to advancements in allogeneic transplantation, immunotherapy, chemotherapy, and Tyrosine Kinase Inhibitors (TKIs), such as imatinib mesylate, dasatinib, and ponatinib. Even with these advancements in treatment, relapse still occurs in certain Ph+ B-ALL patients, and relapse management remains difficult. To prevent Ph+ B-ALL recurrence, it is imperative to identify Ph+ B-ALL medication targets or Tyrosine Kinase Inhibitors (TKI) combo approaches. According to recent research, the pathophysiology and chemo resistance of hematological malignancies are significantly influenced by niche microenvironments that have been altered by malignant cells. Leukemogenesis, cell proliferation, and treatment resistance are supported by the Bone Marrow (BM) niches that are remodeled by immune mediator imbalances.

These niches also activate survival pathways, defend against excessive Reactive Oxygen Species (ROS), induce metabolic reprogramming, and promote immunosuppression. Therefore, these leukemic niches offer a chance to find therapeutic targets to raise the effectiveness of TKI therapy in Ph+ B-ALL patients.

T helper type 17 cells are important in the development of cancer, autoimmune illnesses, and chronic inflammatory conditions because they secrete Th17-associated cytokines such TNF, IL-17, IL-21, and IL-22. In many autoimmune and chronic inflammatory illnesses, IL-17A appears to be a prospective therapeutic target, according on experimental and clinical data. Two anti-IL-17A monoclonal antibodies, secukinumab and ixekizumab, are authorized for the clinical management of psoriasis, psoriatic arthritis, and ankylosing spondylitis. According to recent studies, IL-17 is involved in creating host defense in physiological settings, preserving barrier integrity, and promoting the progression of cancer in pathological settings. Prior research has demonstrated a favorable correlation between treatment resistance and cancer development in B-cell ALL (B-ALL), Acute Myeloid Leukemia (AML), and Multiple Myeloma (MM) patients with elevations in the Th17 cell population. Thus, focusing on Th17 cells may enhance Ph+ B-ALL therapy results and lower medication resistance. Nevertheless, the precise functions of Th17 cells and cytokines linked to Th17 in the Ph +B-ALL niche microenvironment and the advancement of the disease remain unclear. The ability of IL-17 to produce pro inflammatory mediators, the mitogenic effects in tissue progenitor cells, and the capacity to change cellular metabolism are all necessary for the pathogenic roles of Th17 cells and Th17-associated cytokines. The inflammatory response in cancer cells raises the expression of immunological mediators in tumor microenvironments, and this is important for tumor metastasis, migration, proliferation, and cancer stem cells. Membrane-bound chemokine C-X-C motif ligand 16 binds to and ligandizesX-C chemokine receptor type 6 (CXCR6). It is implicated in the development of numerous chronic inflammatory disorders, such as cancer, atherosclerosis, fibrosis, and nonalcoholic fatty liver disease. Th17 cells have been shown to express CXCR6, and effector CD4+ cells with a CCR6+CXCR6+ phenotype mostly produced RORC, IL-23R, and IL-17A, which are conventional Th17 markers. In experimental autoimmune encephalomyelitis, cytotoxic Th17 cells can be identified using CCR6 and CXCR6. Moreover, CXCL16 has the ability to chemo attract tumor-infiltrating T cells, thereby generating a microenvironment that promotes the advancement of cancer. CXCL16's precise involvement in Th17 cell development and function, particularly in the leukemia niche milieu, is still unknown. In this study the Th17 cells in the bone marrow function contribute to Ph+ B-ALL formation because the Th17-associated cytokine IL-17A stimulates the production of different pro inflammatory mediators that can modify the location. Th17 cells concentrate in particular tumors, suggesting that the tumor microenvironment has recruited these cells on purpose. Through the release of Th17-associated cytokines such IL-17A, IL-21, IL-23, and TNF46, Th17 cells are essential for both inflammation and tumor immunity.

Research has shown that increased frequencies of Th17 cells are present in haematological malignancies, such as multiple myeloma, B-cell lymphoma, AML, and ALL. Th17 cell overpopulation, in conjunction with elevated IL-17 and other pro inflammatory cytokine levels, promotes B-cell lymphoma cell proliferation, treatment resistance, apoptosis inhibition, and immune response suppression. These investigations demonstrate the critical functions of IL-17A and Th17 cells in leukemia.

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