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Lymphoma updates-What's new in 2016?

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Malignant lymphomas are a heterogeneous group of diseases that can be divided into 2 major subtypes—Hodgkin's lymphoma (HL) and non-Hodgkin's lymphomas (NHL). The incidence of NHL has been rising over the past 3 decades. Diffuse large B-cell lymphoma (DLBCL) is an aggressive lymphoma and the most common form of adult NHL. It can be further subdivided into the favorable germinal center B-cell-like (GCB) subtype or the unfavorable activated B-cell-like (ABC) subtype. Unlike aggressive lymphomas, indolent NHL (iNHL) remains largely incurable with current therapies. Several lymphoma subtypes such as Mantle cell lymphoma (MCL), T-cell lymphoma, ABC-DLBCL, and double-hit DLBCL are associated with poor outcome. Our understanding of the intracellular machinery and the signaling pathways involved in lymphoma cell growth and proliferation has allowed the identification of key targets for new agent combination. Currently, several novel therapies are being investigated either as single agents or in combinations with novel agents or standard chemotherapy. In addition, current clinical trials are designed to tailor therapy to the patient's tumor molecular characteristics. We will review the current therapies of NHL and HL, and discuss challenging cases about how lymphoma patients should be treated in 2016.

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Chimeric antigen receptor–modified T cells for acute lymphoid leukemia

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Chimeric antigen receptor–modified T cells with specificity for CD19 have shown promise in the treatment of chronic lymphocytic leukemia (CLL). It remains to be established whether chimeric antigen receptor T cells have clinical activity in acute lymphoblastic leukemia (ALL). Two children with relapsed and refractory pre-B-cell ALL received infusions of T cells transduced with anti-CD19 antibody and a T-cell signaling molecule (CTL019 chimeric antigen receptor T cells), at a dose of 1.4×10^6 to 1.2×10^7 CTL019 cells per kilogram of body weight. In both patients, CTL019T cells expanded to a level that was more than 1000 times as high as the initial engraftment level, and the cells were identified in bone marrow. In addition, the chimeric antigen receptor T cells were observed in the cerebrospinal fluid (CSF), where they persisted at high levels for at least 6 months. Eight grade 3 or 4 adverse events were noted. The cytokine-release syndrome and B-cell aplasia developed in both patients. In one child, the cytokine-release syndrome was severe; cytokine blockade with etanercept and tocilizumab was effective in reversing the syndrome and did not prevent expansion of chimeric antigen receptor T cells or reduce anti leukemic efficacy. Complete remission was observed in both patients and is on-going in one patient at 11 months after treatment. The other patient had a relapse, with blast cells that no longer expressed CD19, approximately 2 months after treatment. Chimeric antigen receptor–modified T cells are capable of killing even aggressive, treatment-refractory acute leukemia cells *in vivo*. The emergence of tumor cells that no longer express the target indicates a need to target other molecules in addition to CD19 in some patients with ALL.

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