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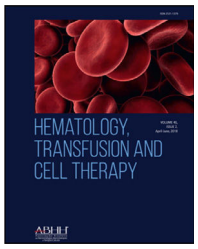

SPEAKER PRESENTATIONS

ADULT SPEAKER PRESANTATION

Sp01

WILL IMMUNE THERAPY CURE ACUTE MYELOID LEUKEMIA?

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There is considerable recent progress in using immune therapy to treat lymphoid including monoclonal antibodies, antibody-drug and -radionuclide conjugates, bi-specific antibodies and chimeric antigen receptor T-cells (CAR-T-cells). Targets of these therapies are B-cell lineage-specific antigens such as CD19, CD20 and BCMA, not cancer-specific antigens. Given these immune therapy advances in lymphoid cancers one might expect similar success using immune therapy to treat acute myeloid leukemia (AML). However, this is not so. There is only one FDA-approved therapy of myeloid cancers, gemtuzumab ozogamicin (Myelotarg®) for AML approved > 10 years ago. Why this discordance? The answer lies in two considerations: (1) lack of a robust AML-specific target antigen(s); and (2) unacceptable adverse effects resulting from non-specificity of lineage-specific antigens such as CD33 and CD124. Also, most data suggest less immune surveillance against myeloid cancers compared with lymphoid cancers. For example, AML cells have an average of 0.28 mutation *per* megabase of DNA compared with 8.15 mutations for lung cancer, 40-fold less. The exception is the anti-AML effect associated with haematopoietic cell transplants, so-called graft-*versus*-leukaemia (G*v*L). However, this effect occurs only in an allogeneic setting and is difficult or impossible to distinguish from graft-*versus*-host disease (G*v*HD). We can envision potential anti-AML immune therapy using two strategies: (1) antibodies; and (2) cell therapies. Synthetic biology may offer a solution to the problem of the lack of an AML-specific target antigens. I discuss the current state of immune therapy of AML and potential future directions. So, will immune therapy cure AML? Stand by.

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Sp02

BRENTUXIMAB VEDOTIN VERSUS CHECKPOINT INHIBITORS: WHICH ONE? WHEN? WHY SHOULD BE PREFERRED?

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About 15% of classical Hodgkin lymphoma (cHL) patients remain refractory to first-line therapy and about one third of the responding patients relapse[1]. The standard of care for relapsed or refractory (R/R) cHL is salvage chemotherapy followed by high-dose chemotherapy (HDCT) and autologous stem cell transplantation (ASCT)[2]. Three novel agents effective in R/R cHL were introduced; brentuximab-vedotin (BV), anti-CD30 antibody-drug conjugate[3] and the programmed-death-1 (PD-1) blocking antibodies, nivolumab and pembrolizumab[4, 5] has been approved. The optimal line to incorporate these agents is an actual dilemma. BV and PD1-blockers are effective in R/R cHL after ASCT. KEYNOTE-204 study reported that pembrolizumab treatment was associated with significantly longer PFS compared with BV (median:13.2 vs 8.3 months)[6]. In case of durable responses with PD1-blockers, cessation of the treatment may be an individualized decision and high response rates to re-treatment with PD1-blockers is an important advantage[7]. There is not obvious differences in the efficacy and toxicity of nivolumab and pembrolizumab[8]. We can conclude that PD1-blockers could be preferred over BV in patients who relapse following ASCT and who are naïve to BV and PD-1 blockade. For patients relapsing after ASCT with prior BV or PD1-blocker exposure, selection of the agent