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Topic area: Disease-specific topics

Topic: 19. Acute leukaemia

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Effect of in vivo T-cell depletion with ATG on cytomegalovirus (CMV) induced antileukemic effect in patients with acute myeloid leukemia (AML) receiving grafts from unrelated donors.

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Introduction: Several studies provided evidence of a consistent antileukemic effect induced by CMV replication in AML patients receiving allogeneic hematopoietic stem cell transplantation (HSCT). It is conceivable that the “virus-versus leukemia” effect promoted by CMV reactivation requires a robust T- or NK-cell immune response to elicit a graft-versus leukemia effect. In vivo T-cell depletion is typically associated with a delayed T-cell reconstitution, and may potentially abrogate the protective effect of CMV infection. Nevertheless, the influence of antithymocyte globulin (ATG, Thymoglobulin) on the antitumor effect of CMV replication after HSCT is rather unexplored. To address this issue, we retrospectively analyzed a cohort of 101 patients with AML who received grafts from an unrelated donor in two italian institutions between 2004 and 2014, after a conditioning regimen including ATG.

Materials (or patients) and methods: Overall, 66 patients (65%) had early disease phase at the time of HSCT and 83 patients (82%) received myeloablative regimens. Prophylaxis of GVHD consisted of cyclosporine and short course MTX combined with ATG at a dose of 5-7.5 mg/Kg over two or three days. Real-time qPCR testing in whole blood for CMV reactivation was routinely monitored twice weekly. CMV viral load >2000 copies/mL was considered as CMV reactivation.

Results: The cumulative incidence of CMV reactivation was 59% at 12 months. The 5-year cumulative incidence of relapse in patients with CMV reactivation was 29% compared with 37% for patients without CMV reactivation (P=0.279). The only factor associated with a reduced 5-year cumulative incidence of relapse was an advanced disease status at HSCT. Similarly, CMV reactivation was not associated with different OS or NRM rates (p=0.850 and p=0.297 respectively). In the multivariable model adverse cytogenetics (HR 2.42, 95% CI 1.02-5.72; p=0.044) and acute GVHD (HR 3.36, 95% CI 1.32-8.54; p= 0.011) were independent risk factors for reducing OS, while the presence of chronic GVHD was associated with a better OS (HR 0.37, 95% CI 0.15-0.89; p=0.027).

Conclusion: The results of present study showed that patients with AML receiving T-cell depleted HSCT using ATG did not benefit from CMV reactivation. Larger studies are mandatory to confirm our preliminary data.

Disclosure of Interest: None Declared

Keywords: Acute Myeloid Leukemia, Cytomegalovirus (CMV) Reactivation, T-cell depletion