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Abstract (oral session)

Hepatitis B reactivation among 137 recipients of allogeneic haematopoietic stem cell transplant with a resolved HBV infection (HBsAg-/HBcAb+): the analysis of risk factors and the impact on outcome

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Objectives: Hepatitis B virus (HBV) reactivation can occur in hematopoietic stem cell transplant (HSCT) recipients even in case of a previously resolved (called also potentially occult) HBV infection. The aim of the study was to identify HBV reactivation rate, its risk factors and outcome in a population of allogeneic HSCT recipients. Methods: Patients who underwent allogeneic HSCT at S. Martino Hospital in Genoa in years 2000-2011 and had a complete HBV serology available were included. Resolved HBV infection was defined as HBsAg- and HBcAb+. HBsAg+ patients were excluded. Data from a prospectively collected electronic database and chart review were retrospectively analysed. Baseline and post-HSCT variables were assessed with chi-square and Mann-Whitney tests. Cox regression model was applied to assess the impact on cause-specific hazard. Cumulative incidence of reactivation in presence of competing risks was analysed with Fine and Gray model and landmark analysis. Results: Overall, 137 of 764 patients (18%) had a resolved HBV infection. For 65 of them serum HBV-DNA at transplant was available and was negative in all. HBV reactivation occurred in 14 patients (10%) in median 19 months after HSCT (range: 9-77). Among 54 HBcAb+ patients alive at 7 years after HSCT, 14 (26%) developed HBV reactivation. In univariate analysis, cause-specific hazard for reactivation was decreased in case of an HBV exposed donor (HR=0.12; CI95%:0.02-0.89; p=0.039) and increased in patients who received rituximab treatment post-HSCT (HR=4.5; 95%CI:1.21-16.8; p=0.044). Multivariate analysis confirmed the protective role of donor HBV immunity (HR_{adjusted}=0.12, 95%CI:0.02-0.96; p=0.045) (fig1). An increased probability of reactivation was also associated with the length of treatment with cyclosporine, even after adjusting for the length of follow-up (p<0.001). In the multivariate model for evaluating competing risks and landmark analysis the probability of HBV reactivation was decreased with an HBV-exposed donor (p=0.041) and increased after treatment with rituximab (p<0.001). No significant difference in overall survival and non-relapse mortality were found between patients with and without HBV reactivation. Conclusion: In patients with a resolved HBV infection, donor immunity was independently and consistently associated with a decreased risk of HBV reactivation, while rituximab treatment and longer administration of cyclosporine increased the probability.

