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CMV serostatus was positive in 51 patients. Overall 23 (24.2% overall, 45.1% in seropositive patients) patients had CMV reactivation of which 11 had reactivation after first transplant, 8 after second and 4 after both transplants. 13 patients received treatment with foscarnet, valganciclovir and/or ganciclovir. CMV symptoms were fever (4 patients), pneumonitis (2) and possible pericarditis (1) and esophagitis (1). No CMV reactivation was seen in seronegative patients.

Seven patients were found to have HHV-6 reactivation, 4 after first and 3 after second transplant. Two patients needed treatment with foscarnet and ganciclovir. HHV-6 symptoms were fever (1 patient) and possible encephalitis (1). With early treatment, no death occurred due to CMV and HHV-6 reactivation.

**Conclusions:** Our experience shows that CMV reactivation is very common (almost 50%) in CMV seropositive patients whereas HHV-6 reactivation is relatively rare. CMV surveillance should be performed routinely after APBSCT.

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### Acute Exacerbation and Reactivation of Chronic Hepatitis C Virus Infection in Hematopoietic Cell Transplant Recipients

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**Background:** Acute exacerbation (AcEx) and reactivation of chronic hepatitis C virus (HCV) infection is known to occur after systemic chemotherapy in cancer patients, which often leads to discontinuation of chemotherapy. However, data on occurrence of these phenomena in hematopoietic cell transplant (HCT) recipients are limited.

**Methods:** We studied HCV-infected HCT recipients seen at MD Anderson Cancer Center from 1/2008 to 8/2015 (1/2008-10/2011 retrospectively, 11/2012-8/2015 prospectively). AcEx was defined as a  $\geq 3$ -fold increase in serum alanine transaminase (ALT) from pre-HCT level in the absence of liver infiltration by cancer, use of hepatotoxic medications, recent blood transfusions, other systemic infections, and GVHD of the liver. HCV reactivation (HCV-R) was defined as an increase in HCV RNA of  $\geq 1 \log_{10}$  IU/mL over pre-HCT level within 1 year after HCT. HCV-associated malignancies were non-Hodgkin lymphomas such as diffuse large B cell (DLBCL) and marginal zone lymphomas (Peveling-Oberhag J, et al. *J Hepatol* 2013). Categorical variables were compared using  $\chi^2$  test and  $p < 0.05$  were considered significant.

**Results:** Fifty four HCV-infected patients underwent 59 HCTs, majority of them being autologous HCT (n=45, 76%). Most patients were under 65 years of age (n=50, 93%), male (n=40, 74%), white (n=33, 61%), and had genotype 1 infection (n=32, 68%). Most common cancer was lymphoma (n=23, 43%) [Mainly DLBCL (n=12)], multiple myeloma (n=19, 35%), and acute leukemia (n=9, 17%). Most common conditioning regimens used were reduced-intensity for allogeneic HCT (n=9, 64%), and melphalan (n=20, 48%) or BEAM  $\pm$  rituximab (n=14, 33%) for autologous HCT. Post-HCT immunomodulatory therapy was given in 41 (69%) patients, mostly corticosteroids (42%) and tacrolimus (26%). GVHD was observed in 11 patients. Of 52 patients with pre and post-HCT ALT data available, 15 (29%) had AcEx [median peak ALT (interquartile range, IQR), IU/mL, 389 (257-554)] with a median time to AcEx of 54 days (IQR, 11-214). Compared to those without AcEx, more patients who experienced AcEx

had an HCV-associated cancer (60% vs 16%;  $p=0.002$ ) or received BEAM  $\pm$  rituximab as conditioning regimen (60% vs 24%;  $p=0.05$ ). Pre and post-HCT HCV RNA data were available for 45 patients. HCV-R was seen in 5 (11%) patients; median time to HCV-R was 41 days (IQR, 35-132). Three patients with HCV-R (60%) had simultaneous AcEx. AcEx and HCV-R occurred more frequently after autologous HCT (67% and 60% respectively). Occurrence of either AcEx or HCV-R did not change the pre-established oncologic plan for patients. Neither AcEx nor HCV-R were associated with increased risk of cancer relapse or all-cause mortality.

**Conclusions:** AcEx and HCV-R may occur in HCV-infected cancer patients who undergo HCT but these phenomena do not seem to affect the oncologic care of infected recipients. Frequent virological monitoring post-HCT may not be required.

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### Community Acquired Respiratory Viral Infections (CARV) in Patients with Acute Leukemia and Hematopoietic Stem Cell Transplant (HSCT) Recipients

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**Background:** CARV infections are significant causes of morbidity and mortality in adult HSCT recipients. In 2008, the FDA approved the use of multiplex PCR assay to detect respiratory viruses. CARV infections have been studied mostly in the HSCT population where lymphopenia has been identified as a risk factor for poor outcomes. These infections have been less well studied in the acute leukemia population. This report, however, encompasses both patient populations. **Study Design:** This is a retrospective cohort study of adult patients ( $\geq 18$  yrs) with acute leukemia and/or after HSCT diagnosed with laboratory documented CARV infections.

**Results:** Between Sep 1st 2009 and Nov 1st 2014, there were 493 episodes of CARV infections in 291 patients. Out of these, 228 patients were post-HSCT and 63 patients had acute leukemia. In these episodes, rhinovirus was found in 193, coronavirus in 80, parainfluenza in 62, influenza in 56 (IFV), RSV in 52, metapneumovirus in 29, adenovirus in 19, and 1 each of bocavirus and enterovirus. In 29 episodes, more than one virus was documented. 450 episodes were initially diagnosed as an upper respiratory tract infection (URI). There were 67 episodes (13.6%) of LRI; of these 24 had a prior URI with rhinovirus (n=9), IFV (n=4), parainfluenza (n=4), metapneumovirus (n=3), coronavirus (n=2), RSV (n=2) while 43 episodes were LRI at initial presentation (10 RSV, 9 parainfluenza, 7 rhinovirus, 6 metapneumovirus, 5 coronavirus, 3 IFV, 2 adenovirus, 1 enterovirus). Out of the 291 patients there were 23 deaths (7.9%). Per univariate analysis, only lymphopenia ( $< 300$ /dl) at the time of diagnosis was associated with death ( $p$ -value = 0.01) and lymphopenia was also a risk for LRI and progression from URI to LRI  $p$ -value = 0.03.

**Conclusion:** In patients with acute leukemia and HSCT recipients lymphopenia at the time of RVI is an independent risk factor for LRI and death.