**Results.** Of 70 patients with detectable CMV DNA at randomization (48 LET, 22 PBO), CMV VL was 150 c/ml in 63 patients (range, 150–716). All patients had undetectable CMV VL ≤5 days before randomization. Baseline characteristics were similar to the PEP, except for more patients with myeloablative conditioning (62.9% vs. 48.3%) and longer median days post-HCT to start of study drug (15 days vs. 8 days). Median study drug exposure was 70 days (range, 1–113) in LET group and 14 days (range, 7–99) in PBO group. By HCT Week 14, CS-CMVi occurred in 15 (31.3%) LET-treated patients and 17 (77.3%) PBO patients; CS-CMVi with imputed events were 22 (45.8%) in LET group and 20 (90.9%) in PBO group (difference −44.8%; 95% CI, −64.7% to −24.8%; P < 0.0001). Median CMV VL at time of PET was 413 c/ml (range, 150–31,847) and was similar between groups. Eight patients had quantifiable CMV VL (range, 171–1,728 c/ml) 1 week after starting study drug; 6 did not receive PET (5 LET [10.4%], 1 PBO [4.5%]). CMV VL was undetectable subsequently; other 2 withdrew from study. One (2.1%) LET-treated patient developed breakthrough CMV viremia with a CMV VL was undetectable subsequently; other 2 withdrew from study. One (2.1%) LET-treated patient developed breakthrough CMV viremia with a CMV VL was undetectable subsequently; other 2 withdrew from study.

**Conclusion.** LET prevented CS-CMVi compared with PBO among patients with detectable CMV DNA at randomization.

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**Methods.** DNA was extracted from liver tissue followed by targeted amplification of the cestode COX1 gene. PCR products confirmed to be 134 bp, as expected for a CESTO C325W mutation. The sequence matched to *Versteria* sp. (larval stage) of a cestode (tapeworm). Treatment with praziquantel and albendazole led to improvement of symptoms and lesions. Disseminated cestode infections other than due to *Echinococcus* species are rare in humans. Sequencing was pursued due to the unusual findings.

**Results.** The sequence matched to *Versteria* sp. (*T. mustelae*) COX1 gene from a mink in Oregon (accession KT223034) with 98% identity.

**Conclusion.** Metacestodes have the propensity to proliferate and rarely disseminate. There is one reported case of *Versteria* sp. causing a lethal disseminated infection in a human and is singular because the patient survived. The patient likely accidentally ingested ova shed from a tapeworm in a mink or similar mammalian host. Histopathologic assessment is crucial in diagnosing cestode infection. COX1 gene sequencing is useful for cestode identification.
1732. Adenovirus Load Dynamics Are Consistently Correlated With Risk of Mortality in Pediatric Allogeneic Hematopoietic Cell Transplant Recipients: Findings From The Landmark AdVance Study

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Background. Adenovirus (AdV) infection is an important cause of mortality among allogeneic hematopoietic cell transplant (allo-HCT) recipients. Current European Conference of Infections in Leukemia (ECIL-4) guidelines support weekly AdV screening for those at-risk and pre-emptive antiviral treatment with off-label cidofovir when adenoviremia is detected. However, there is limited understanding of the relative prognostic strength of different dynamic AdV viral load measures. We examined the association between adenovirus viral load dynamics and mortality in pediatric allo-HCT recipients managed under the current standard of care.

Methods. AdVance was a multinational, multicenter study characterizing the current screening and treatment practices for AdV infection in allo-HCT recipients between January 2013 and September 2015. This analysis focused on pediatric (<18 years) patients who experienced AdV viremia ≥ 1,000 copies/mL within 6 months of HCT. Multivariate Cox Proportional Hazard models, controlling for factors including immune reconstitution, were used to examine the relationship between AdV viral load dynamics (Figure 1) and all-cause mortality in the 6 months after first AdV viremia ≥ 1,000 copies/mL.

Results. A total of 241 pediatric allo-HCT recipients had AdV viremia ≥ 1,000 copies/mL in the 6 months following allo-HCT. Among these, 43/241 (18%) died within 6 months of first AdV viremia ≥ 1,000 copies/mL. AdV viral load dynamics; whether measured by AdV AAUC0–16, peak viremia, 2-week change in viremia, or days of viremia > 1,000 copies/mL, were consistently correlated with all-cause mortality (Figure 2; hazard ratio [HR] range: 1.3–2.3). Most notably, patients with AdV AAUC0–16 in the highest quartile had an HR of 11.6 relative to those in the lowest (confidence interval: 4.7–24.0; Figure 3).

Conclusion. AdV infection is a significant risk for allo-HCT recipients. The AdVance study has identified several dynamic measures of AdV viral load that correlate with the risk of mortality in pediatric allo-HCT recipients. Results show for the first time, that AdV AAUC0–16 provides the optimal correlation with mortality in this population and serves as a clinically useful indicator of outcome in patients with AdV infection.