### VZV Risk Factors in Heart Transplant Recipients: Multivariable Analysis

Factor	HR (95% CI)		
Age	1.1 (0.8-1.8)		
Gender (M)	0.8 (0.3-2.4)		
Race			
Black	Ref		
White	1.9 (0.4-9.4)		
Other	2.5 (0.4-17.4)		
Immunosuppression at 1 yr			
Prednisone	0.4 (0.2-1.2)		
Cyclosporine (vs. Tacrolimus)	1.4 (0.5-5.0)		
MMF/MPA (vs. Azathioprine)	1.6 (0.6-4.2)		
CMV	4.7 (1.7-13.5)		

**Conclusion.** HT recipients are at highest risk for VZV reactivation. CMV infection before 1 year is associated with increased risk of VZV reactivation after 1 year in HT. This information may help design clinical trials of the recombinant zoster vaccine.

Disclosures. All authors: No reported disclosures.

### 1555. A New Perspective About Disseminated Adenovirus Infection and Its Outcomes in Pediatric Solid Organ Transplantation

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**Background.** Adenovirus (AdV) in solid-organ transplants (SOT) was historically associated with increased morbidity and mortality. Detection of AdV at  $\geq 2$  sites is predictive of invasive disease in other immunocompromised populations; however, data is lacking for SOT.

*Methods.* All SOT in children  $\leq 18$  years from January 2005 to June 2017 (n = 1,024). We evaluated host and viral risk factors associated with disseminated AdV infection (defined as AdV from  $\geq 2$  sites or DNAemia alone) and the clinical spectrum of disease.

**Results.** Ninety-two patients had 116 AdV infections. Overall prevalence was 9% with one death. Thirty-nine percent of patients had disseminated infection and of those, 44% received cidofovir. Patients with disseminated infection were more likely to be  $\leq 2$  years compared with >2 years (P = 0.003), infected in first-year post-transplant compared with >1 year (P = 0.05), and to present with fiver compared with no fever (P = 0.02). No difference was observed for organ subtypes, presence of gastrointestinal or upper respiratory tract symptoms, peak DNAemia, mean viral load (mean 3.9log<sub>10</sub>vs. 4log<sub>10</sub>) between patients with dissemination compared with without dissemination. For patients who received a biopsy, dissemination was not different between patients with a positive biopsy vs. negative biopsy (46% vs. 54%). Cidofovir was given to 64% of the positive biopsy patients. No difference for age at infection or time to infection was observed between the treated and not treated groups.

**Conclusion.** Our data shows that younger age at infection, shorter time to infection and clinical fever are risk factors for disseminated adenovirus infection in pediatric SOT patients, supporting primary infection and enhanced immunosuppression as main factors that allow viral dissemination. Some patients with high viral loads and biopsy-proven disease were not treated with cidofovir with very low mortality, reflecting a broader spectrum of infection than previously recognized. Our data begins to define a high-risk clinical and viral phenotype for adenovirus dissemination, which can inform management strategies.

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# 1556. Infectious Disease Complications with Use of Checkpoint Inhibitors in Solid Organ Malignancies

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**Background.** Immune checkpoint inhibitors (ICIs) are innovative cancer immunotherapies used for solid-organ and hematologic malignancies. ICIs are known for their immune-related adverse events (irAE) but there are limited reports on infectious complications of immunosuppression for these complications. The purpose of this study was to describe the spectrum of infections in patients with melanoma, renal cell carcinoma or non-small cell lung cancer receiving ICI.

Methods. Retrospective review of City of Hope patients with melanoma, renal cell carcinoma or non-small cell lung cancer on nivolumab, pembrolizumab, and/or ipilimumab from January to November 2017 and received two or more doses of ICI. Pt characteristics assessed: age, sex, prior chemotherapy, steroid use, and type of immunosuppression for irAE. Microbiology records were used to identify infections.

**Results.** Thirty-nine infectious episodes (35 bacterial, four viral) were identified among 111 patients. Four bacteremia (two *B. cereus, coagulase-negative staphylococcus,* 1 *S. aureus*), 12 urinary tract (10 Gram-negative rods, 2 Gram-positive cocci), one intra-abdominal, eight skin and soft-tissue infections (one *S. aureus,* one *Actinomyces radinge,* one *E. faecalis,* and one *E. cloacae*). There were two probable viral pneumonias (two rhinovirus, two enterovirus) and no fungal infections. Fourteen (12.6%) infections were defined as serious (requiring intravenous antimicrobials and/or hospitalization). There was no association between the specific malignancy or ICI used and risk of infection. Steroid use was significantly associated with serious infections: 12/14 (85.7%) vs. 27/95 (28.4%); *P* = 0.0003), and no patients had received infliximab or other immunosuppressant.

**Conclusion.** Bacterial infections were most common, and the only risk factor associated with serious infections in our study was steroid use. Type of ICI did not impact the rate of infection.

Disclosures. S. Dadwal, Ansun Biopharma: Investigator, Research grant.

### 1557. Acyclovir-Resistant (ACV-R) Herpes Simplex Virus (HSV) Disease in Patients with Hematologic Malignancies (HM) and Hematopoietic-Cell Transplant (HCT) Recipients

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**Background.** HSV reactivation is a challenging complication of HM and HCT. ACV prophylaxis effectively decreases the incidence of symptomatic HSV episodes, but may contribute to development of ACV-R HSV disease in this population. Outcomes in patients with ACV-R HSV disease remain poorly characterized.

**Methods.** We identified adult HM patients and HCT recipients treated at Dana-Farber Cancer Institute who developed clinically significant ACV-R HSV disease between January 1, 2006 and March 1, 2018. HCT recipients typically receive 1 year of ACV prophylaxis after HCT, or longer in those with graft-vs. host disease. Clinical, microbiological and treatment details were collected.

**Results.** Nineteen patients had 27 episodes of ACV-R HSV disease during the study. Median age was 50 years (range 31–77); 15 (79%) were men. Fifteen (79%) were allogeneic HCT recipients and 4 (21%) had HM (3 CLL, 1 NHL). Thirteen (68%) had oral ulcers (HSV1), four (21%) had perineal ulcers (3 HSV2, 1 HSV1), one had HSV1 vesicles on the trunk and one had concurrent oral HSV1 and perineal HSV2 ulcers. Three patients had recurrent ACV-R HSV; two had one recurrence each and one had six recurrences. Of 19 first episodes of ACV-R HSV, 15 (79%) were confirmed by culture-based phenotypic resistance testing.

Most episodes (20/27, 74%) were treated with foscarnet at clinical diagnosis or after failure of high-dose val-ACV; four of these episodes were also treated with topical cidofovir without success before foscarnet. Three episodes resolved on high-dose val-ACV or IV ACV alone and three were treated with cidofovir or brincidofovir initially. Coinfection was present in 19 episodes (70%), most often bacterial pneumonia or blood stream infection. Twenty-two episodes (81%) resolved completely after a median of 36 days (range 10–88) of treatment. No patient died of HSV disease but five (26%) died before resolution of ACV-R HSV, a median of 25 days (range 1–117) after treatment started. Eight patients died after ACV-R HSV resolved, a median of 111 days (range 27–382) after treatment started. Among HCT recipients, six (37%) died within 12 weeks of diagnosis.

*Conclusion.* ACV-R HSV disease is an uncommon complication of HM and allogeneic HCT. While ACV-resistant HSV did not cause death in this cohort, death within 12 weeks of infection was common.

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## 1558. Commensal *Neisseria* Species as a Cause of Disease in Patients Taking Eculizumab

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**Background.** Non-meningococcal and nongonococcal *Neisseria* spp. are usually commensal and rarely cause invasive disease in humans. Eculizumab, a terminal complement inhibitor, increases susceptibility to meningococcal disease, but data on atypical *Neisseria* spp. disease in persons receiving eculizumab are lacking. This case series describes postmarketing reports of disease by commensal *Neisseria* spp. in patients receiving eculizumab.

*Methods.* The FDA Adverse Event Reporting System (FAERS) database and the medical literature were searched for cases of disease by any nonmeningococcal and nongonococcal *Neisseria* spp. in patients receiving eculizumab. Included cases had a diagnosis of disease by any atypical *Neisseria* spp. with onset on or before January 31, 2018 and ≥1 dose of eculizumab in the 3 months prior to disease.

**Results.** The search identified seven FAERS cases, including one case also reported in the literature. Patient ages ranged from 4 to 38 years. Five patients had positive blood cultures, of which three had an indwelling catheter for vascular access (n = 2, N. sicca/subflava) or hemodialysis (n = 1, N. cinerea). Two patients with bacteremia had N. cinerea septic shock with possible cholecystitis, and N. mucosa sepsis with concurrent *Streptococus* bacteremia after gastroenteritis. The remaining two cases in the series included one with N. sicca bacterial peritonitis associated with a peritoneal dialysis catheter (negative blood cultures, other cultures not specified), and one with a diagnosis of N. flavescens sepsis while neutropenic (specimen source not specified). All seven patients were hospitalized and three had sepsis or septic shock. All cases resolved with antibiotics and supportive care.

**Conclusion.** We identified seven cases of serious disease caused by atypical *Neisseria* spp. among eculizumab recipients. Since these organisms are typical inhabitants of the oropharynx and urogenital tract and are not skin flora, the source of disease was unclear. Our data suggest that eculizumab may confer increased risk for disease by usually commensal *Neisseria* spp. Healthcare professionals are encouraged to treat all *Neisseria* spp. isolated from sterile sites as pathogenic, and not as contaminants, in patients receiving eculizumab.

The views expressed are those of the authors and do not necessarily represent those of, nor imply endorsement from, the U.S. Food and Drug Administration, the Centers for Disease Control and Prevention, or the U.S. government.

Disclosures. All authors: No reported disclosures.

#### 1559. Hematopoietic Cell Transplantation with Post-transplant Cyclophosphamide: Impact of Donor Type on Pre-engraftment Blood-Stream Infections

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**Background.** The aim of the study was to estimate the cumulative incidence of pre-engraftment blood stream infections (PE-BSI), its predictive factors and the infection-related mortality (IRM) after hematopoietic cell transplantation (HCT) from any donor type, with post-transplant cyclophosphamide (PT-Cy).

*Methods.* Retrospective cohort study on 235 adults who underwent peripheral blood HCT from every donor type with PT-Cy platform, from 2013 to 2017 at San Raffaele Scientific Institute. The Poisson regression was used to estimate the crude incidence rate (IR) of PE-BSI. The Fine-Gray competing risk model was applied to estimate the cumulative incidence function (CIF) of the first PE-BSI and its predictive factors and of IRM.

Results. Patients' characteristics are reported in Table 1. During 5,316 person-days of follow-up (PDFU), 77 PE-BSI episodes occurred in 72 patients: IR = 1.45 per 100-PDFU [95% confidence interval (95% CI) 1.13-1.77]. The median time to PE-BSI was 13 days (IQR: 7-17) and the estimated CIF at 28 days was 32% (95% CI: 26-39%); no differences in CIF according to donor type [30% vs. 34% vs. 32% in match-related, match-unrelated and haploidentical donor, respectively; Gray's test: P = 0.968]. Among the 87 isolated pathogens, 60% were Gram-positive bacteria (GPB), 39% Gram-negative bacteria (GNB) and 1% nontuberculous mycobacteria. CIFs of GNB and GPB PE-BSI by type of donor are shown in Figure 1. By multivariate analysis (Table 2), after adjustment for age, sex, year of HCT, donor type and disease phase at HCT, the CIF of any PE-BSI was higher in subjects with absolute neutrophils count ≤500 for ≥7 days before HCT [adjusted hazard ratio (AHR) = 2.90] and in multi-drug resistant (MDR) GNB rectal carriers before HCT [AHR = 2.68]. These covariates were confirmed as independent factors also for GNB PE-BSI. Overall, IRM at 30 days was 5% (95% CI: 2-8%) with no differences by donor type (Gray's test: P = 0.106).

**Conclusion.** HCT with PT-Cy platform showed a 32% of cumulative incidence of PE-BSI at 28 days and donor type did not affect its occurrence, which was conversely increased by prolonged and severe neutropenia and MDR GNB rectal carriage before HCT. Haploidentical setting did not retain a higher IRM at 30 days than match-related and match-unrelated donors.

#### able 1. Characteristics of patients who underwent hematopoietic cell transplantation with PT-Cy platform (all patients received antibiotic prophylaxis with levofloxacin).

Patients' Characteristics		Overall	MRD	MUD	haplo	p-value	
		(n =235)	(n =40)	(n =50)	(n =145)		
BASELINE							
Age at HCT, yr, median (IQR)		49.6 (37.0-62.0)	48.1 (40.9-59.4)	50.6 (37.4-57.0)	51.6(36.4-63.1)	0.863	
Male gender, n (%)		147 (63%)	25 (63%)	33 (66%)	89 (62%)	0.844	
Year of HCT, median (IOR)		2016 (2014-2017)	2016 (2015-2017)	2016 (2016-2017)	2015 (2014-2016)	<0.000	
ANC \$500 for \$7 days before HCT		66 (28%)	10 (25%)	5 (10%)	51 (35%)	0.003	
Diagnosist, n (%) Acute myeloproliferative diseases		157 (67%)	27 (68%)	29 (58%)	101 (70%)	0.260	
Acute and chronic lymphoproliferative diseases		65 (27%)	11 (27%)	15 (30%)	39 (27%)		
Chronic myeloproliferative diseases		12 (5%)	2 (5%)	5 (10%)	5 (3%)	i .	
	-mediated diseases	1 (196)	0	1 (2%)	0	i	
Disease phase at HCT, n (%)	>CR1	40 (17%)	1 (2)	9 (18%)	30 (21%)	0.001	
	CR1	63 (27%)	16 (40%)	20 (40%)	27 (19%)		
	Active disease	131 (55%)	23 (58%)	20 (40%)	88 (61%)	i	
	Not applicable	1 (196)	0	1 (2%)	0		
Conditioning regimen, n (%)				- ()		0.286	
Myeloat	blative conditioning	184 (78%)	35 (88%)	39 (78%)	110 (76%)		
Reduced intensity conditioning		51 (22%)	5 (12%)	11 (22%)	35 (24%)	i i	
MDR-GNB rectal carrier within 30 days before HCT, n (%)		18 (8%)	3 (8%)	1 (296)	14 (10%)	0.214	
Number of HCT, n (%) Fir	st allogenic HCT	201 (87%)	39 (98%)	50 (100%)	115 (79%)	0.001	
	cond allogenic HCT	27 (12%)	1 (296)	0	26 (18%)		
	Third allogenic HCT	4 (3%)	0	0	4 (3%)	i	
	sirolimus/(MMF)	231	40 (100%)	50 (100%)	141 (98%)	0.387	
	closporine A/MMF	3	0	0	3 (2%)		
FOLLOW-UP							
Follow-up, days, median (IQR)		276 (137-580)	289 (197-577)	316 (174-531)	259 (114-618)	0.579	
ANC engraftment, n (%)		225 (96%)	39 (98%)	50 (100%)	136 (94%)	0.144	
Time to engraftment, days, median (IQR	9 1	20 (17-24)	20 (16-24)	22 (19-29)	19 (17-24)	0.046	
PE-BSI, n (%)	none	164 (70%)	28 (70%)	34 (68%)	102 (70%)		
	Single BSI episode	72 (31%)	12 (30%)	16 (32%)	44 (30%)	0.972	
	Two BSI episodes	5 (2%)	0	1 (2%)	4 (3%)	0.563	
At least 1 BSI due to Gram-positive bacteria, n (%)		46 (20%)	7 (18%)	10 (20%)	29 (20%)	0.936	
At least 1 BSI due to Gram-negative bacteria, n (%)		30 (13%)	7 (18%)	6 (12%)	17 (12%)	0.615	
Time to the first BSI after HCT among su	bjects who	13(7-17)	13 (12-15)	13 (7-20)	10(7-18)	0.549	
developed 21 BSI, days, median (IQR)		15(1-17)	15(12-15)	15(7-20)	10(/-18)	0.545	
Antimicrobial resistance score, n (%)	i					0.051	
Susceptible to 1 <sup>st</sup> line anti	biotic therapy (PTZ)	13 (18%)	1 (8%)	7 (44%)	5 (11%)		
Susceptible to 2 <sup>nd</sup> line antibiotic t	herapy (MEM, VAN)	49 (68%)	9 (75%)	8 (50%)	32 (73%)	1	
Resistant to 2 <sup>rd</sup> line antibiotic therapy (MEM.VAN)		10(14%)	2 (17%)	1 (6%)	7 (16%)	i i	
Septic shock, n (%)		12 (17%)	2 (17%)	2 (17%)	8 (18%)	0.873	

Abbreviations: HCT, hematopoietic cell transplantation; MRD, match-selated donor, MUD, match-urrelated donor; Fagio, papiolatenical donor; AME, atsociate neutropi count; CR, complete esponse; MDR-GNB, multi-drug resistant Gram-negative bacteris; GVHO, parti-versus host disease; PT-Cy, post-transplant cyclophosphamide; MM moleciell imyophenoiaae; PE-BSI, pe-negraftmare blocksteam Infection; PTZ, piperacilin/tabboktum; MM, metopener VAA, varacomyon.

§ Acute myeloproliferative disease: scute myeloid laukamia, myelodyaplastic syndrome; Acute and chronic lymphoproliferative disease: scute lymphoblastic laukaemia, Hodgin lymphoma, non-Hodgin hymphoma, mutbige myeloma; Ornoris myeloproliferative disease: chronic myelogenous leukemia, kilopathic myelofibrasis, myeloproliferative neoplasm; Benign/immune-mediated diseases: chronic granubmatous disease.

\*1 patient died because of ESBL-producing Escherichia coli BSI before receiving GVHD prophylaxis

gure 1 - Cumulative incidence function (CIF) of the first pre-engrafiment BSI due to Gram-negative and Gram-positive bacteria according to the type of donor IF estimated according to the Fine-Gray method, with engrafitment, pre-engrafitment death or second HCT as competing events].

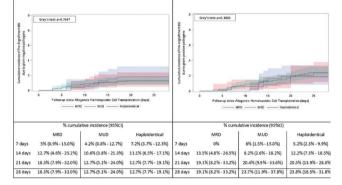


Table 2 - Multivariate Fine-Gray models to assess baseline factors associated with the incidence of any or Gram-negative bacteria (GNB) preengraftment BSI (PE-BSI)

Characteristic at HCT	Risk categories	Adjusted HR of any PE-BSI (95%CI)	p-value	Adjusted HR of GNB PE-BSI (95%CI)	p-value
Age	per 3-years older	1.010 (0.959-1.063)	0.716	0.943 (0.866-1.027)	0.178
	>50 vs ≤50 years				
Gender	Female vs Male	0.877 (0.524-1.467)	0.616	0.767 (0.340-1.730)	0.523
Year of HCT	per 2 more recent years	0.942 (0.628-1.411)	0.770	1.024 (0.515-2.036)	0.947
	>2015 vs ≤2015				
ANC ≤500 for ≥7 days before HCT	Yes vs No	2.895 (1.542-5.435)	0.0009	4.865 (1.992-11.89)	0.0005
MDR-GNB rectal carrier within 30 days before HCT	Yes vs No	2.683 (1.253-5.749)	0.011	3.885 (1.288-11.72)	0.016
Type of donor			0.496		0.367
	Haploidentical vs MRD	0.929 (0.480-1.801)	0.828	0.656 (0.255-1.688)	0.382
	MUD vs MRD	1.493 (0.758-2.944)	0.387	1.307 (0.417-4.099)	0.646
Disease phase					
	Active disease vs >CR1/CR1	0.886 (0.483-1.624)	0.694	1.074 (0.432-2.674)	0.877

bbreviations: HCT, hematopoletic cell transplantation; MRD, match-relazed donor; MUD, match-umelazed donor; hapio, hapioidentical donor; ANC, absolute neutrophils Junt; CR, complete response; MDR-GNB, multi-drug resistant Gram-negative bacteria; PE-851, pre-engraftment blood-stream infection.

The multivariate model considered engraftment and pre-engraftment death as competing events; it was constructed by considering the main exposure of interest (type of donor), opriori factors known to have a potential effect on the incidence of PE-BSI (age and sea) and other covariates with a p-value-0.2 at univariate analysis.

Disclosures. All authors: No reported disclosures.

### 1560. Clinical Presentation of BK Virus-Associated Hemorrhagic Cystitis (HC) After Hematopoietic Cell Transplantation (HCT)

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**Background.** BK polyoma virus (BKPyV) has been associated with hemorrhagic cystitis after HCT. Prior studies have examined risk factors for BKPyV-associated HC, but the characteristics of disease, including duration, common presentations, and the spectrum of clinical outcomes, have not been well described. Precise estimates of major clinical endpoints are critical to design clinical trials of novel prevention and treatment agents.