Table 1: Demographics, comorbidities, immunosuppression, and clinical data for patients with OIs

Variable	Very-Late Onset Opportunistic Infections (n = 16)
Median age at time of transplant (range)	43 (25-72)
Age at time of transplant	
18-29	2 (12.5%)
30-39	4 (25%)
40-49	5 (31.3%)
50-59	1 (6.2%)
60-69	2 (12.5%)
70-79	2 (12.5%)
Living (vs. deceased)	11 (68.8%)
Living donor (vs. deceased/unknown)	7 (43.8%)
Retransplantation	1 (6.2%)
Median Years From Transplant to Last Follow Up (range)	14.2 (10-37)
Mean Charlson Comorbitiy Index of Living Patients (SD)	5.6 (3.6)
Male sex (versus female)	10 (62.5%)
Race	
Asian	2 (12.5%)
Black	3 (18.8%)
Hispanic	3 (18.8%)
White	8 (50%)
Induction Methods	
Basiliximab	3 (18.8%)
Daclizumab	1 (6.2%)
Methylprednisone	7 (43.8%)
Thymoglobulin	3 (18.8%)
Unknown/Unspecified	2 (12.5%)
Maintenance Regimens	
Belatacept and prednisone	1 (6.2%)
Belatacept, mycophenolate, and prednisone	4 (25%)
Cyclosporine, mycophenolate, and prednisone	1 (6.2%)
Tacrolimus and prednisone	2 (12.5%)
Tacrolimus, mycophenolate, and prednisone	4 (25%)
Other	4 (25%)
Comorbidities at Time of Review	
Hepatitis C positive	2 (12.5%)
History of Diabetes	6 (37.5%)
Currently on Dialysis	2 (12.5%)
History of Cardiovascular Disease	5 (31.3%)
History of Lung Disease	0
History of Chronic Liver Disease	0
History of Cerebrovascular Injury	4 (25%)
History of Malignancy	4 (25%)
Number of Infections	
1	6 (37.5%)
2	5 (31.3%)
3	2 (12.5%)
4+	3 (18.8%)
Serologic Data	
CMV D+/R-	2 (14.3%)
CMV D-/R-	0
CMV D?/R-	4 (25%)
CMV D+/R+	0
CMV D-/R+	1 (6.2%)
CMV D?/R+	8 (50%)
Mean Absolute Lymphocyte Counts (10^3/µL)	0.78

Table 2: Detailed characteristics of each patient with opportunistic infections

Patient	Age at Time of Transplant	Diagnosis For Kidney Transplant	CCI	H/x DM (1 = Yes; 2 = No)	On Dialysis at Time of Infection (1 = Yes; 2 = No)	CMV Status	Maintenance Immunosuppression	Total Number of Late Onset Infection Episodes	Treatment for Rejection Within 1 Year of Opportunistic Infection? (1 = Yes; 2 = No)	Opportunistic Infection Timing (years s/p tx)	Age at Time of Opportunistic Infection	Type of Opportunistic Infection	Pathogen	Absolute Lymphocyte Count at time of Infection (10^3/µL)	30-Day outcome
1	25	Analgesic Nephropathy	3	2	1	D+/R-	Tacrolimus + Mycophenolate + Prednisone	2	1	10 Years	35 Years Old	Pneumonia	Adenovirus	0.5	Alive
2	63	DM 2	7	1	2	D+/R+	Tacrolimus + Mycophenolate + Prednisone	2	2	11 Years	73 Years Old	Viremia	CMV	1.1	Alive
3	47	Hypertensive Nephropathy	4	2	2	D?/R-	Cyclosporine + Mycophenolate + Prednisone	1	2	15 Years	62 Years Old	Esophagitis	HSV + Candida	0.6	Alive
4	50	DM 1	7	1	2	D+/R+	Belatacept + Prednisone	3	2	12 Years	63 Years Old	Esophagitis	Candida	0.7	Alive
5	72	MPGN	N/A	2	2	D+/R+	Tacrolimus + Prednisone	5	2	15 Years	87 Years Old	Meningitis	Cryptococcus	0.2	Deceased
6	45	DM 1	15	1	2	D+/R+	Tacrolimus + Mycophenolate + Prednisone	10	1	13 Years	59 Years Old	Colitis	CMV	0.6	Alive
7	70	DM 2	8	1	2	D+/R+	Belatacept + Prednisone + Mycophenolate	3	2	11 Years	81 Years Old	Pneumonia	PJP	0.2	Alive
8	43	Glomerulonephritis	3	2	1	D+/R+	Belatacept + Prednisone + Mycophenolate	1	2	11 Years	54 Years Old	Pneumonia	PJP	0.6	Alive
9	31	DM 1	N/A	1	1	D?/R-	Tacrolimus + Prednisone	6	2	29 Years	59 Years Old	Pneumonia	PJP	0.2	Alive
10	33	Hypertensive Nephropathy	N/A	1	2	D?/R-	Belatacept + Prednisone + Mycophenolate	1	2	10 Years	43 Years Old	Pneumonia + Fungemia	PJP and Cryptococcus	0.2	Deceased
11	26	Hypertensive Nephropathy	N/A	2	2	D+/R+	Sirolimus + Prednisone	2	2	33 Years	59 Years Old	Colitis	Adenovirus	1.1	Alive
12	67	Hypertensive Nephropathy	N/A	2	2	D+/R+	Everolimus + Prednisone	1	2	12 Years	79 Years Old	Viremia	CMV	0.2	Deceased
13	41	Obstructive Nephropathy due to Neurogenic Bladder	4	2	2	D+/R-	Belatacept + Prednisone + Mycophenolate	1	2	10 Years	51 Years Old	Pneumonia	PJP	0.6	Alive
14	32	Polycystic Kidney Disease	2	2	2	R+	Tacrolimus + Mycophenolate + Prednisone	2	2	15 Years	46 Years Old	Pneumonia	PJP	0.6	Alive
15	43	HTN	4	2	2	R+	Sirolimus + mycophenolate + prednisone	1	2	10 Years	53 Years Old	Skin	VZV	1.4	Alive
16	34	Polycystic Kidney Disease	3	2	2	D?/R-	Sirolimus + prednisone	1	2	15 Years	50	Skin	VZV	1.4	Alive

Conclusion. OIs were infrequently observed beyond 10 years of transplant among long-term survivors of KT. However, OI incidence was associated with poor outcome. Low ALC and a higher burden of comorbidities were risk factors for very late occurrence of OIs in this population.

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1100. Outcomes of HIV-Associated Lymphoma Treatments: A Contemporary Single Center Cohort Study.

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Session: P-49. Infections in Immunocompromised Individuals

Background. There is a paucity of outcome studies on HIV-associated lymphoma treated with chemotherapy with or without autologous hematopoietic stem cell transplantation (autoHSCT) in comparison to HIV-uninfected individuals with similar histology.

Methods. In our retrospective matched cohort study, we enrolled adult HIV-positive patients with lymphoma treated with chemotherapy with (group 2) or without autoHSCT (group 1) between January 1, 2007 to December 31, 2018 at the University of Kansas Medical Center and followed until May 1, 2020. Group 1 were matched 1:1 to HIV-negative patients based on age, gender, lymphoma histology, stage at diagnosis, year of lymphoma diagnosis, and Group 2 were matched 1:2 to HIV-negative patients based on age at autoHSCT, gender, lymphoma histology, stage at diagnosis and year of transplantation. Overall survival (OS) and progression-free survival (PFS) at 2 years were calculated using Kaplan-Meier (KM) analysis, and adjustment for ECOG and IPI/IPS scores was done using multivariate Cox model.

Results. We had 37 HIV+ patients with lymphoma in our cohort: 9 Hodgkin's disease (HD), 28 Non Hodgkin's Lymphoma (NHL). Eleven underwent autoHSCT (3 HD, 8 NHL). The majority were white (76.2%), non-hispanic (92.9%), males (90.5%) and mean age was 46 years. Median CD4 was 172.5, HIV viral load was < 50 copies/mL in 43..2%, and 76.2% were on antiretroviral therapy (ART) at diagnosis. ART was interrupted in 14.6% and adjusted in 40.5% of patients. After excluding rare histological types, 22 in group 1 and 9 in group 2 were included in the matched analysis. On KM survival at 2-years, group 1 had worse OS (75% vs 95%, p=0.02), and a trend for worse PFS (75% vs 90%, p=0.07) than the matched referent group, while group 2 had similar OS (100% vs 94%, p= 0.47) and better PFS (100% vs 70%, p=0.02) than the matched referent group. On Cox models adjusting for ECOG and IPI/IPS, HIV status was no longer independently associated with OS in group 1 or PFS in group 2.

Group 1 HIV lymphoma cases and controls characteristics

Group 1 HIV with lymphoma cases and controls characteristics			
Variable		case	control
Number		22	22
Age at diagnosis	Mean(sd)	46.3 (10.4)	47.9 (10.4)
Sex	Male, n (%)	21 (95.5%)	21 (95.5%)
Ethnicity	Non-Hispanic	20 (90.9%)	20 (90.9%)
Race	Black, n (%) White, n (%) Other, n (%)	3 (13.6%) 16 (72.7%) 3 (13.6)	3 (13.6%) 17 (77.3%) 2 (9.1%)
ECOG status	Mean(sd)	1.2 (1.4)	0.7(0.6)
Type of lymphoma	NHL-DLBCL, n (%) NHL-primary CNS, n (%) NHL-Burkitt's, n (%) HL-mixed cellularity, n (%) HL-nodular sclerosis, n (%) HL-lymphocytic rich, n (%)	12 (54.5%) 1 (4.5%) 4 (18.2%) 2 (9.1%) 2 (9.1%) 1 (4.5%)	12 (54.5%) 1 (4.5%) 4 (18.2%) 2 (9.1%) 2 (9.1%) 1 (4.5%)
Stage of lymphoma at diagnosis	Stage II, n (%) Stage III, n (%) Stage IV, n (%)	2 (9.1%) 4 (18.2%) 16 (72.7%)	2 (9.1%) 5 (22.7%) 15 (68.2%)
IPI	IPI-high, n (%) IPI-low, n (%) IPI-mid, n (%) IPI-n/a, n (%)	5 (22.7%) 4 (18.2%) 3 (13.6%) 10 (45.5)	1 (4.5%) 4 (18.2%) 6 (27.3%) 11 (50%)
IPS	IPS-high, n (%) IPIS-low, n (%) IPS-mid, n (%) IPS-n/a, n (%)	3 (13.6%) 1 (4.5%) 1 (4.5%) 17 (77.3%)	1 (4.5%) 2 (9.1%) 2 (9.1%) 17 (77.3%)
Number of relapse	0, n (%) 1, n (%)	22 (100%) 0 (0%)	21 (95.5%) 1 (9.1%)

Sd= standard deviation, n/a= not applicable, ECOG= Eastern Cooperation Oncology Group, NHL= Non Hodgkin's lymphoma, DLBCL = Diffuse large B-cell lymphoma, CNS= Central nervous system, HL= Hodgkin's lymphoma, IPI= International Prognostic Index, IPS= International prognoctic score

Group 2 HIV lymphoma with HSCT cases and controls characteristics

Group 2. HIV Lymphoma with HSCT,cases and controls characteristics			
		case	control
Number		9	18
Age at HSCT	Mean (sd)	45.8 (10)	43.4 (11.7)
Sex	Male, n (%)	9 (100%)	18 (100%)
Ethnicity	Non-Hispanic, n (%)	8 (88.9%)	17 (94.4%)
Race	Black, n (%)	0 (0%)	4 (22.2%)
	White, n (%)	8 (88.9%)	12 (66.7%)
	Asian, n(%)	0 (0%)	1 (5.6%)
	More than one, n (%)	1 (11.1%)	1 (5.6%)
ECOG	Mean(sd)	0.4 (0.7)	0.8 (0.7)
Type of lymphoma	NHL-DLBCL,n (%)	4 (44.4%)	8 (44.4%)
	NHL-Burkitt's, n (%)	2 (22.2%)	4 (22.2%)
	HL-mixed cellularity, n (%)	2 (22.2%)	4 (22.2%)
	HL-nodular sclerosis, n (%)	1 (11.1%)	2 (11.1%)
Stage of lymphoma at diagnosis	Stage III, n (%)	1 (11.1%)	2 (11.1%)
	Stage IV, n (%)	8 (88.9%)	16 (88.9%)
IPI	IPI-high, n (%)	0 (0%)	5 (27.8%)
	IPI-low, n (%)	3 (33.3%)	2 (11.1%)
	IPI-mid, n (%)	2 (22.2%)	3 (16.7%)
	IPI-miss, n (%)	4 (44.4%)	8 (44.4%)
IPS	IPS-high, n (%)	2 (22.2%)	3 (16.7%)
	IPIS-low, n (%)	0 (0%)	0 (0%)
	IPS-mid, n (%)	1 (11.1%)	3 (16.7%)
	IPS-n/a, n(%)	6 (66.7%)	12 (66.7%)
Number of relapses	0, n (%)	9 (100%)	16 (88.9%)
	1, n (%)	0 (0%)	1 (5.6%)
	2, n(%)	0 (0%)	1 (5.6%)

n=number, sd= standard deviation, n/a= not applicable, HSCT= Hematopoietic stem cell transplant, ECOG= Eastern Cooperation Oncology Group, NHL= Non Hodgkin's lymphoma, DLBCL = Diffuse large B-cell lymphoma, CNS= Central nervous system, HL= Hodgkin's lymphoma, IPI= International Prognostic Index, IPS= International prognoctic score.

Conclusion. In patients with HIV and lymphoma treated with chemotherapy with or without autoHSCT, the outcomes are comparable to those without HIV in our single center contempory cohort.

Disclosures. **Wissam El Atrouni, MD**, **ViiV** (Advisor or Review Panel member)

1101. Pulmonary Aspergillosis Complicating Non-Influenza Respiratory Virus Infections Among Solid Organ Transplant Recipients

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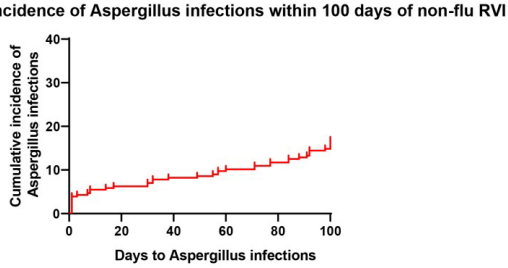
Session: P-49. Infections in Immunocompromised Individuals

Background. Invasive pulmonary aspergillosis (IPA) complicating influenza (flu) has been increasingly recognized. We have shown that IPA occurred in 22% of solid organ transplant (SOT) patients (pts) with flu. Associations between IPA and non-flu respiratory infections (non-flu-RVI) in SOT are unknown.

Methods. Retrospective review of consecutive pts transplanted from Jan 15, 2010-Dec 19, 2017. Pts who died within 100 days of SOT were excluded. Non-flu-RVI IFI was defined according to revised EORTC/MSG criteria. IFI had to occur within 100 days of non-flu-RVI. Colonization (COL) was defined as recovery of mold from airways in absence of IFI.

Results. 3,077 pts were included. 256 cases of non-flu-RVI were identified in lung (28%), multi-organ (16%), heart (6%), liver (1.3%) and kidney (1%) SOT pts. Parainfluenza (PIV) was most common (44%), followed by Respiratory Syncytial Virus (RSV, 60%) and Adenovirus (ADV, 15%). Median time to non-flu-RVI infections was 18.1 mos. 24% of pts with non-flu-RVI had lower tract disease. ADV was associated with longer hospital stay (median 14.5 days) than PIV (6.5 days) or RSV (6 days) (p=0.004). 59% of pts with non-flu-RVI required admission, and 64% received augmented steroids. *Aspergillus* was recovered from respiratory culture in 17% of non-flu-RVI pts. No other fungi were identified. Median time from non-flu-RVI to + culture was 29 days (Figure). 23% of pts with + culture had proven (7) or probable IPA (3), respectively; 77% had COL. 8% (3/37), 5% (6/114) and 7% (1/15) of pts with ADV, PIV, RSV infections developed IPA, respectively. 36% of pts were treated with a mold-active azole after + culture. Multivariate analysis identified lung transplant (p=0.02), PIV infection (p=0.02) and cumulative steroid dose in preceding 7 days (p=0.015) as independent risk factors for *Aspergillus* culture positivity. Cumulative steroid dose in preceding 7 days was an independent risk factor for IPA (p=0.03).

Cumulative incidence of Aspergillus infections within 100 days of non-flu RVI



Conclusion. IPA and COL occurred in 4% and 13% of non-flu-RVI in SOT recipients. Routine antifungal prophylaxis is not recommended for SOT pts with non-flu-RVI. The value of prophylaxis at time of PIV infection for lung transplant pts with recent steroid augmentation should be studied.

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1102. Reconstitution of CMV-specific cell-mediated immunity during letermovir prophylaxis in hematopoietic stem cell recipients

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Maheen Z. Abidi, Jonathan A. Gutman, Adriana Weinberg

Session: P-49. Infections in Immunocompromised Individuals

Background. Patients who are cytomegalovirus (CMV) seropositive (R+) prior to hematopoietic cell transplant (HCT), have 30% incidence of clinically significant CMV reactivation in the absence of prophylaxis. At our institution, letermovir prophylaxis through Day 100 is used in CMV R+ high-risk (HR) (cord blood, haplocord, haploidentical) HCT recipients. We hypothesized that clinically nonsignificant CMV reactivation during letermovir prophylaxis may lead to reconstitution of CMV specific cell mediated immunity (CMV CMI), which may protect the host against CMV disease after letermovir discontinuation.

Methods. Blood samples from CMV R+ HR HCT recipients on letermovir were tested by dual color CMV specific IL2/IFNg FLUOROSpot pre-transplant and on Days 100, 182 and 360 post-transplant. Clinical and virologic information were obtained from medical records.

Results. Among 35 participants enrolled to date, 19 were eligible for this analysis, which included only participants with CMV CMI defined as ≥20 spot-forming cells/10^6 PBMC pre-transplantation and follow up ≥180 post-transplantation. Median age was 51.5 years (range 22-75), 9 were women, 9 were white non-Hispanic, 8 were Hispanic and the most common underlying malignancy was acute myeloid leukemia (n=10). 14 participants had CMV CMI reconstitution at Day 100; including 5 with and 9 without low level CMV DNAemia, defined as <5000 international units/ml in whole blood quantitative polymerase chain reaction assay, while on letermovir prophylaxis. Among the 14 participants, 11 remained free of clinically significant CMV reactivation for a median (range) of 260 (80; 260) days post-letermovir discontinuation, while 3 developed acute graft vs. host disease (aGvHD) followed by clinically significant CMV reactivation. 5 participants did not reconstitute CMV CMI at Day 100 and none of them had DNAemia while on letermovir. 1 of 5 participants without CMV CMI reconstitution or aGvHD developed CMV disease after letermovir discontinuation.

Conclusion. High-risk patient populations can reconstitute CMV CMI while on letermovir. Ongoing investigation will help establish predictive parameters for CMV CMI that may allow risk stratification for CMV monitoring and letermovir usage.

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1103. Respiratory Virus Infections In Solid Organ Transplant Recipients: A Single Center Experience

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