Table 1. Species, type and clinical manifestations of Human Adenovirus isolates (patients under surveillance)

ID	Number of specimens retrieved from archive, by source									Species/Type (source)[£]	Transplant type	Disease	Days from detection to disease [¥]	Deceased (within 180 days post-transplant)
	BAL	GI	L	NP	P	S	Sp	U	O					
1					1					C2	Allo	Pneumonitis (prov)	0	N
												Hepatitis (pos)	5	
2					8					C2; E4	Auto	Hepatitis (pos)	5	N
3					8				1	C2 (P); B3 (O)	Allo	Hepatitis (pos)	6	N
4					6					C1	Allo	Hepatitis (prob)	7	N
6					2					C5	Allo	None	N/A	N
7				2	7	1				A31 (NP; S) C2 (P)	Allo	Pneumonitis (pos)	36	Y[†]
												Hepatitis (pos)	38	
8				1	9				0	C2 (P; NP)	Allo	Pneumonitis (prob)	25	N
												Colitis (pos)	43	
9				1	9					C2(P)	Allo	Pneumonitis (pos)	32	N
11					1	6		3	1	D20-15 (S; U)	Allo	Hepatitis (pos)	42	N
												Cystitis (pos)	63	
												Colitis (pos)	82	
12		1			4	2				C1 (GI; S)	Allo	Colitis (prob)	17	N
13		1			4		1	1		C2 (P)	Allo	Cystitis (pos)	0	Y[†]
												Hepatitis (pos)	0	
												Colitis (pos)	3	
												Pneumonitis (pos)	17	
14			1		9					C1 (P)	Allo	Hepatitis (pos)	23	N
15		1		4	7	1		1		C2 (NP; S)	Allo	Colitis (prob)	13	Y[†]
												Cystitis (pos)	36	
												Pneumonitis (prob)	90	
16					17	2		2		C1 (S)	Allo	Hepatitis (pos)	0	Y[†]
												Colitis (pos)	3	
												Cystitis (pos)	37	
												Pneumonitis (pos)	70	
17						2				B3	Allo	Colitis (pos)	0	N
18		1			2	2				C5 (GI; S)	Allo	Colitis (pos)	0	N
												Hepatitis (pos)	2	
19					5					C2	Allo	Hepatitis (prob)	0	N
21					2	1				A12 (S)	Allo	None	N/A	N
23					2	2		1		C2 (S) A31 (S)	Allo	Colitis (pos)	23	Y
												Hepatitis (pos)	23	
												Cystitis (prob)	38	
24		1				3				A12 (GI; S)	Allo	Colitis (prob)	2	N
25	1	2		1	3					C2 (GI; NP; BAL)	Allo	Colitis (pos)	100	Y[†]
												Pneumonitis (prov)	108	
26					1	2				A31 (S)	Allo	Colitis (prob)	2	N
27					19	2				C2 (S)	Allo	Colitis (pos)	0	N
												Hepatitis (pos)	1	
												Pneumonitis (pos)	8	
28					3					C2	Allo	None	N/A	N
29				2	14	1				C1 (P; S)	Allo	Pneumonitis (prob)	87	Y[†]
												Hepatitis (prob)	87	
												Colitis (prob)	87	
												CNS (prov)	87	
30						1				C2	Allo	None	N/A	N
31				1	7	1		2		B11-7 (S; U) B11 (P)	Allo	Hepatitis (prob)	0	Y[†]
												Cystitis (pos)	14	
												Colitis (pos)	49	
												Pneumonitis (prov)	55	

Abbreviations: P=Plasma/Blood; NP=Nasopharyngeal aspirate; S=Stool; U=Urine; BAL=Bronchoalveolar Lavage; GI=Gastrointestinal tissue; Sp=Sputum; L=Liver; O=Other; pos=possible; prob=probable; prov=proven

[£]The species and type were identified from at least one of the clinically positive specimens. When specimens from more than one source were sent, the source in which the molecular type was identified, is specified in the parentheses next to the type.

[¥]Time from first positive to first disease designation

[†]Death was attributable to Adenovirus

Table 2. Species, type and clinical manifestations of Human Adenovirus isolates (patients not under surveillance)

ID	Number of specimens retrieved from archive, by source									Species/Type (source)[£]	Transplant type	Disease	Days from detection to disease [¥]	Deceased (within 180 days post-transplant)
	BAL	GI	L	NP	P	S	Sp	U	O					
5				2						C6	Allo	Pneumonitis (pos)	0	N
10					5					C2	Allo	Cystitis (prob)	0	Y
												Hepatitis (pos)	0	
20				1						C1	Allo	Pneumonitis (pos)	0	Y[†]
22						4				B3	Allo	Cystitis (pos)	0	N
												Colitis (prob)	14	

Abbreviations: P=Plasma/Blood; NP=Nasopharyngeal aspirate; S=Stool; U=Urine; BAL=Bronchoalveolar lavage; GI=Gastrointestinal tissue; Sp=Sputum; L=Liver; O=Other; pos=possible; prob=probable; prov=proven

[£]The species and type were identified from at least one of the clinically positive specimens. When specimens from more than one source were sent, the source in which the molecular type was identified, is specified in the parenthesis next to the type.

[¥]Time from first positive to first disease designation

[†]Death was attributable to Adenovirus

Disclosures. **All authors:** No reported disclosures.

1763. The Use of Haploidentical Donors Compared with HLA-Matched Unrelated Donors is Associated with Increased Risk of BK Viruria and Hemorrhagic Cystitis

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Session: 169. Transplant ID: Viral, Mycoplasma/Ureaplasma Infections

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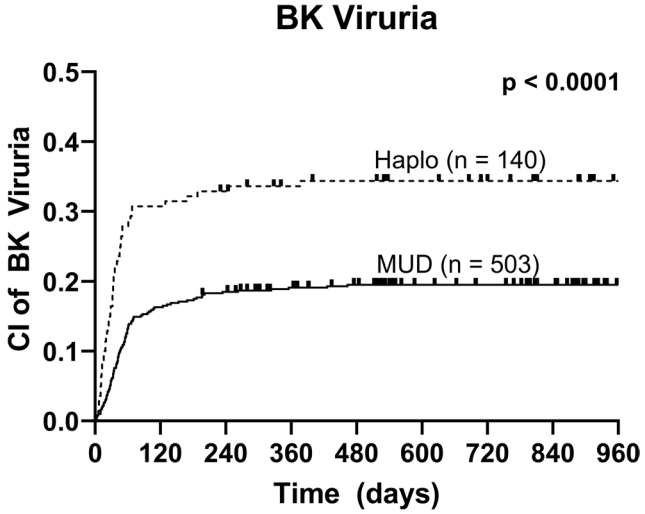
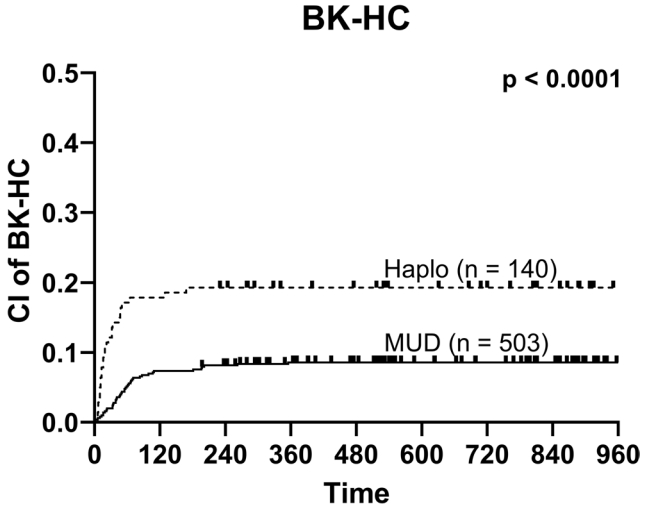
Background. BK virus-associated hemorrhagic cystitis (BK-HC) is a common and often serious complication of hematopoietic cell transplantation (HCT). Studies have suggested a higher incidence of BK-HC in patients receiving haploidentical (haplo) HCTs compared with those receiving matched unrelated donor (MUD) transplants.

Methods. We retrospectively identified all adult patients receiving HCT from MUD or haplo donors at Washington University School of Medicine between January 1, 2011 and January 1, 2016. Via informatics queries, we obtained the results of every urine BK test performed on these patients. Patients with BK viruria were then evaluated for BK-HC and graded according to established criteria. The last day of follow-up was April 31, 2017.

Results. 503 MUDs and 140 haplos were identified for inclusion in the study. Haplo patients were significantly more likely to be nonwhite (21% vs. 5%, $P < 0.001$) and were younger (median age: 51.5 vs. 55, $P = 0.01$). Conditioning regimens were also significantly different; haplos were less likely to receive myeloablative conditioning (44% vs. 57%, $P < 0.001$) and busulfan-based conditioning (13% vs. 39%, $P < 0.001$), but were more likely to receive total body irradiation-based conditioning (83% vs. 26%, $P < 0.001$). Haplos were also more likely to have undergone previous allogeneic HCT (26% vs. 6%, $P < 0.001$). The cumulative incidence of both BK viruria and BK-HC were significantly higher in haplos (both $P < 0.001$). This was observed at 100 days, 180 and 365 days (Table 1).

Conclusion. We found a significantly higher incidence of both BK viruria and BK-HC in patients receiving haplo HCT compared MUD HCT. Significant demographic and clinical imbalances exist between our two cohorts and attribution of increased risk for BK-HC to donor type vs. other factors should be further explored.





	MUD	Haplo
BK Viruria (%, 95% C.I)		
At 100 days	16 (13 - 19)	31 (23 - 38)
At 180 days	17 (14 - 21)	32 (24 - 40)
At 1 year	19 (16 - 23)	34 (26 - 41)
BK-HC (%, 95% C.I)		
At 100 days	7 (5 - 9)	18 (12 - 24)
At 180 days	7 (5 - 10)	19 (13 - 26)
At 1 year	9 (6 - 11)	19 (13 - 26)

Table 1: BK viruria and BK-HC by donor type

Disclosures. **All authors:** No reported disclosures.

1764. Use of Intravesical BCG for Treatment of Bladder Cancer in a Renal Transplant Recipient, with Subsequent Resolution of Chronic BK Viremia

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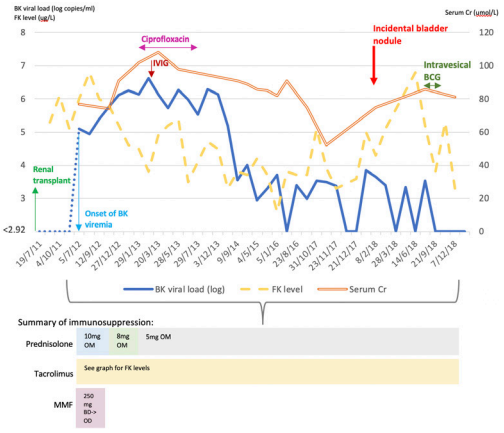
Background. BK virus-induced nephropathy in renal transplantation is well recognized but its oncogenic potential is less appreciated.

Methods. We report a case of high-grade urothelial tumor in a renal transplant recipient with chronic BK viremia in the absence of BK nephropathy successfully treated with intravesical Bacillus Calmette-Guerin (BCG) instillation. Following BCG therapy, there was also spontaneous clearance of BK viremia.

Results. In July 2011, Mr. LYY, a 57-year-old Chinese man with end-stage renal failure secondary to chronic glomerulonephritis underwent uncomplicated deceased donor renal transplantation. One year later, he developed persistent high-level BK viremia despite reduction in immunosuppression. He was also treated with a course of ciprofloxacin from January to October 2013, and intravenous immunoglobulin (IVIG) in February 2013, but high-level BK viremia persisted. In spite of this, his graft function was preserved. He was subsequently placed on BK surveillance alone. Chronic BK viremia persisted for the next 5 years with stable graft function. See Figure 1. In February 2018, an incidental urinary bladder nodule was picked up on computed tomography (CT) scan performed for evaluation of urosepsis. Follow-up flexible cystoscopy in May 2018 showed a 1.5 cm papillary tumor over the left superior bladder wall. Transurethral resection of bladder tumor (TURBT) was performed and histopathological examination revealed high-grade papillary urothelial carcinoma. In addition, the biopsy specimen was stained positive for SV40, suggesting the possible association between chronic BK virus replication in the bladder and oncogenesis. Relook TURBT in July 2018 revealed a persistent erythematous patch over the posterior bladder wall. Given the cystoscopic findings, Mr. LYY received a weekly instillation of intravesical BCG for 6 weeks. He tolerated the intravesical BCG treatment with no local or systemic complications. Interestingly, his serum BK viral loads dropped below lower limits of detection thereafter and continued to remain so for the next 4 months.

Conclusion. We postulate that the intravesical BCG therapy triggered an immune response that targeted not only the tumor but also the BK virus infection, resulting in successful clearance of chronic BK viremia.



Disclosures. **All authors:** No reported disclosures.

1765. BK Polyomavirus Reactivation Outcomes After Renal Transplantation in Association With Adherence to a Standardized BK Polyomavirus Screening Protocol: A Multi-Center Collaboration

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Background. Reactivation of BK polyomavirus (BKPyV) due to immunosuppression after renal transplantation can lead to allograft nephropathy (BKAN) or even allograft loss. Many transplant centers implement screening protocols in an attempt to detect BKPyV reactivation before progression to BKAN, although the frequency and duration of screening vary widely among centers.

Methods. The New England BK Consortium (NEBKCON), a collaboration of 12 transplant centers in the northeastern United States, has adopted a standard BKPyV screening protocol (screening monthly for the first 6 months followed by screening every 3 months until 2 years after transplantation). Participating members implemented this screening protocol at their centers, and later measured adherence to the protocol as part of a NEBKCON quality improvement project. This study retrospectively analyzes BKPyV-specific outcomes in association with adherence to this protocol.

Results. Six centers reported data on 472 subjects who received a renal transplant between January 2016 and December 2017. Adherence to the screening protocol during the first 12 months (7.1–76.7%, mean 56.1%) and 24 months (2.9–52.5%, mean 36.8%) after transplant varied between centers. Rates of BKPyV viremia (3.6–28.2%, mean 20.6%) as well as BKAN (0–4.5%, mean 3.2%) also varied among centers. Adherence to the screening protocol was associated with a decrease in the magnitude of the initial viral load detected (3.29 vs. 3.74 log10 copies/mL, $P = 0.065$), but was not associated with peak viral load (3.95 vs. 4.14 log10 copies/mL, $P = 0.47$), viremia duration (179 vs. 196 days, $P = 0.74$), or incidence of BKAN among viremic subjects (15.3 vs. 16.0%, $P = 0.91$).

Conclusion. Even with a uniform screening protocol for BKPyV in place, adherence to this protocol varied widely among centers. More research is needed to determine patient-level and center-level barriers to adherence, as well as to determine optimal screening practices to further reduce the incidence of BKAN.

Disclosures. **All authors:** No reported disclosures.

1766. Osimertinib-Associated Progressive Multifocal Leucoencephalopathy

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Background. Progressive multifocal leukoencephalopathy (PML) is a rare demyelinating disease of white matter in the central nervous system (CNS) caused by reactivation of John Cunningham (JC) virus. Drug-induced PML is increasingly reported with the widely used biological immunosuppressant drugs and molecular targeted antineoplastic agents. Monoclonal antibodies were the pioneer drugs to be associated with PML including the prototypical natalizumab.

Methods. Epidermal growth factor receptor (EGFR) tyrosine kinase inhibitors (TKI) have been rarely described in this context with few case reports of ibrutinib-associated PML. Osimertinib, a third-generation EGFR TKI, was recently FDA-approved for the first-line treatment of metastatic non-small-cell lung cancer (NSCLC), and to the best of our knowledge has never been associated with PML. We describe a case report of a rapidly progressive PML likely associated with osimertinib therapy.

Results. A 85-year-old female with history of NSCLC, on osimertinib, was admitted with progressively worsening left hemiparesis, facial palsy, unsteady gait, recurrent falls, and episodic confusion over a period of month. Brain magnetic resonance imaging revealed foci of non-enhancing increased T2 and fluid-attenuated inversion recovery (FLAIR) signal intensity in the periventricular and bilateral cerebral subcortical white matter. MRI cervical spine was unremarkable for acute enhancing lesions. Cerebrospinal fluid (CSF) was unremarkable for infectious etiology, oligoclonal bands, and cytology. The patient was readmitted 2 weeks later with worsening neurological deficits and new lesions in the bilateral middle cerebellar peduncles, pons, midbrain, and cerebral white matter. Positive CSF JC virus PCR lead to the final diagnosis of "probable" PML. Biopsy was deferred for high clinical suspicion of PML and procedural risks outweighing benefits. Osimertinib was likely contributing to PML in the absence of other immunosuppression.

Conclusion. Inhibition of tyrosine kinase-dependent pathways can potentially aid in the replication of JC virus per previously reported ibrutinib-associated PML. Clinicians should be aware of PML risk in patients on osimertinib and TKI therapy, especially those with positive serum JC virus serology.