III-IV after sUCBT (19%) compared to dUCBT (10%, p=0.06) but increased incidence of grade II aGVHD after dUCBT (28%) compared to 17% after sUCBT (p=0.05). CI of chronic GvHD at 2 years was 21% after dUCBT and 12% after sUCBT (p=0.15). At 2 years, CI of non relapse mortality (NRM) was 28% after dUCBT and 30% after sUCBT (p=0.87). CI of 2y RI was 21% after dUCBT whereas it was 38% after sUCBT (p=0.03). In a multivariate analysis adjusting for the differences between the 2 groups, dUCBT was associated with lower RI compared to sUCBT (HR=0.74, p=0.01). Therefore, there was an improved 2-y LFS after dUCBT (51%) compared to sUCBT (32%; p=0.03). This was confirmed in a multivariate analysis (HR=0.64, p=0.04).

Concerning pts transplanted in CR2 (n=148), there were no differences of outcomes after dUCBT (n=93) or sUCBT (n=55). At 2y, LFS was 40% after dUCBT and 48% after sUCBT (p=0.32). In a subgroup analysis of dUCBT (n=118) and sUCBT (n=51) recipients using the same conditioning regimen (CY+FLU+TBI2Gy), 2 y LFS were 54% and 33% respectively (p=0.05).

In this retrospective comparative based registry analysis, in AL pts transplanted in CR1, neutrophil recovery, GVHD and NRM were not statistically different after RIC-dUCBT or RIC-sUCBT, however, dUCBT recipients had decreased RI and improved LFS. For AL pts transplanted in CR2, there was no benefit of using dUCBT when compared to sUCBT.

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Incidence and Kinetics of CMV Infection After T-Cell Depleted and Unmodified Allogeneic Hematopoietic Stem Cell Transplantation: A 10-Year Experience at Memorial Sloan-Kettering Cancer Center

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Cytomegalovirus (CMV) is a major cause of mortality and morbidity in hematopoietic stem cell transplantation (HSCT). Transfer of CMV-specific T-cells from the donor is important for the control of CMV replication after HSCT. In this study, we compared incidence and kinetics of CMV infection and CMV disease between T-cell depleted (TCD) and unmodified (CONV) HSCT.

Methods: The cohort consisted of 714 adult HSCT recipients of bone marrow or peripheral blood stem cell allografts from September 1999 to March 2010 at Memorial Sloan-Kettering Cancer Center. Patients were followed until July 2012. TCD recipients did not receive any additional prophylactic medicinal immunosuppression for graft-vs-host disease (GvHD). CMV infection was monitored by PP65 antigenemia assay (CMV Ag) if recipient or donor were CMV seropositive and the information was prospectively stored in a computerized database. Prior to 2007, recipients of mismatched or unrelated allografts were eligible for CMV prophylaxis if recipient or donor were CMV seropositive. Anti-CMV agents were given to patients who had >= 2 cells per slide (cps) on 1 occasion or 1 cps on >= 2 consecutive occasions. Relapse, second transplant, death, and study termination (April, 2012) were considered as competing risk for CMV reactivation.

Results: Four hundred and three (56.5%) patients received TCD grafts and 311 (43.6%) received unmodified grafts (CONV). Recipient CMV seropositivity was 48.3% in TCD and 50.8% in CONV (p=0.5219). There are 221 (54.8%) TCD and 140 (45.0%) CONV patients received allograft from mismatched or unrelated donors (p=0.0092). Sixty-four (15.9%) TCD and 45 (14.5%) CONV patients received CMV prophylaxis (p=0.6031). CMV infections occurred in 135 (33.5%) TCD and 86 (27.7%) CONV patients. Two hundred and five (92.8%) of the 221 infections developed by day +100 post-transplant. CMV infections requiring antiviral treatment occurred in 111 (27.5%) TCD and 64 (20.6%) CONV patients (p=0.0319). Days from HSCT to first CMV infection were median 31 in TCD and 41.5 in CONV (p<0.0001). Maximum cps were median 5 (range 1 to 100) cps in TCD and 3 (1 to 100) cps in CONV (p=0.0159). Duration of reactivation was median 11 days in TCD and 8 days in CONV patients (p=0.0042). CMV disease was diagnosed in 4% in TCD patients and 2.3% in CONV patients (p=0.197).

Conclusion: 1) Rates of CMV infection were similar in TCD and CONV allogeneic HSCT; 2) In contrast, the kinetics of CMV replication were different between the 2 groups: In TCD, CMV infection occurred earlier, with higher peak level, and longer duration of viremia 3) Rates of CMV disease were low and similar between TCD and CONV (4% and 2.3% respectively) Our data suggests that preemptive treatment based on antigenemia is similarly effective for prevention of CMV disease in TCD and CONV allografts.

	TCD N=403	CONV N=311	p-value
CMV Serology			
R+/D+	109 (27%)	104 (33.4%)	0.1665
R+/D-	86 (21.3%)	54 (17.4%)	0.5219*
R-/D+	46 (11.4%)	41 (13.2%)	
R-/D-	162 (40.2%)	112 (36%)	
HLA			
MRD	182 (45.2%)	171 (55%)	0.0092
Mismatched or Unrelated	221 (54.8%)	140 (45.0%)	
Stem Cell Source			
Bone Marrow	76 (18.9%)	52 (16.7%)	0.4602
PBSC	327 (81.1%)	259 (83.3%)	
CMV Prophylaxis			
Yes	64 (15.9%)	45 (14.5%)	0.6031
No	339 (84.1%)	266 (85.5%)	
CMV reactivation, number of patients (%)	135 (33.5%)	86 (27.7%)	0.0939
Clinically significant reactivation, number of patients (%)	111 (27.5%)	64 (20.6%)	0.0319
Median days to first antigenemia (range)	31 (10 to 585)	41.5 (10 to 384)	<0.0001
Peak number of CMV-positive cells, median (range)	5 (1 to 100)	3 (1 to 100)	0.0159
Median duration, days (range)	11 (1 to 258)	8 (1 to 107)	0.0042
CMV Disease, number of patients (%)	16 (4%)	7 (2.3%)	0.197

TCD indicates, T-cell depleted transplant; CONV, unmodified transplant; CMV, cytomegalovirus; R+, recipient seropositive; R-, recipient seronegative; D+, donor seropositive; D-, donor seronegative; HLA, human leukocyte antigen; MRD, matched related donor; PBSC, peripheral blood stem cells;

* Comparing recipient seropositivity between TCD and unmodified graft