Table
Comparison of late aGVHD categories with classic aGVHD

	De-novo n=7	Persistent n=39	Recurrent n=29	Classic Acute n=268
Median time to onset, range	155 (101-262)	100	143 (102-342)	36 (7-180)
2 year overall survival after onset (95% CI)	83% (27-97)	45% (28-60)	74% (53-87)	50% (44-57)
1 year NRM after onset	0%	26% (12-40)	14% (4-26)	24% (19-29)
Progression to cGVHD within 2 years of onset	42% (16-75)	51% (36-66)	38% (23-56)	31% (26-37)

received PBSC and 8% marrow as the graft source. 64% received a reduced intensity conditioning. Median follow up was 4 years.

Results: The cumulative incidence of any late aGVHD was 14.7% (95% CI: 11.6-17.8) and grade II-IV late aGVHD was 11.7% (95% CI 8.7-14.7) at one year (Figure 1). 52% had persistent (defined as active aGVHD that persisted without resolution after 100 days), 39% had recurrent (aGVHD that recurred following resolution after 100 days) and 9% had de-novo late aGVHD. Median time to onset was 155 days for de-novo late aGVHD, and 143 days for recurrent late aGVHD (Table). Persistent late aGVHD was considered to have a time of onset of 100 days. Outcomes were estimated from onset of late aGVHD (Table). One and 2 year overall survival was 73% (95% CI 63-84) and 59% (95% CI 49-72). Figure 2 shows the cumulative incidences of outcomes after late aGVHD. Progression to cGVHD or overlap syndrome was seen in 45% (95% CI 35-57) by two years. Durable discontinuation of immunosuppression (IS) was achieved in 15%, 41% and 57% of persistent, recurrent and de-novo late aGVHD. In multivariate analysis, PBSC [HR 2.1, 95% CI: 1.2-3.5, p=0.01] and myeloablative conditioning [HR 1.8, 95% CI: 1.0-3.2, p=0.04)] were associated with higher risks of developing late aGVHD.

Conclusions: We present results of clinical presentation and outcomes in patients with late aGVHD. Amongst patients with late aGVHD, durable discontinuation of IS was seen in only 15% of those with persistent late aGVHD, indicating need for intensified therapy in this group. PBSC and myeloablative conditioning were associated with a higher risk of developing late aGVHD. Modulation of these risk factors (such as intensified or longer duration of GVHD prophylaxis in these higher risk groups) may allow reduced risks and improved transplant outcomes.

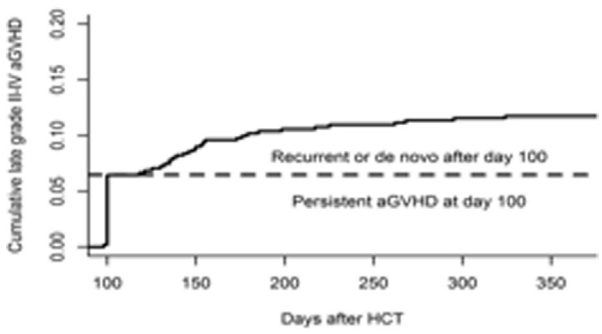

Figure 1. Cumulative Incidence of Grade II-IV late aGVHD

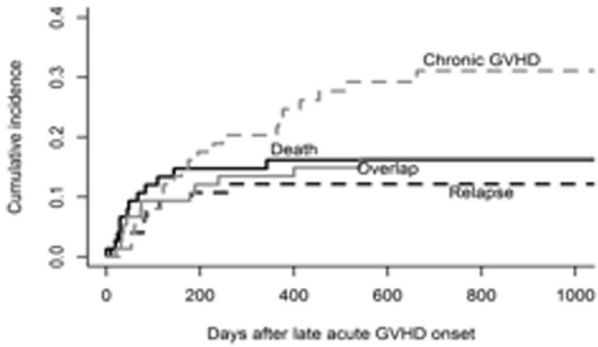

Figure 2. Cumulative incidence of outcomes following late aGVHD

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A Review of Radiographic Findings in GVHD

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Acute and chronic graft vs host disease remains one of the largest challenges in allogeneic stem cell transplantation. Diagnosis and staging of GVHD is largely clinically based: physical exam findings, laboratory data, combined with pathologic confirmation. Typical radiologic findings may aide in diagnosis as well. Recent radiographs of GVHD patients are reviewed. Notable findings in acute GVHD of the gut included edematous bowel wall thickening and fluid filled bowel. Some patients have exhibited hyper enhancing gut mucosa on contrast enhanced CT. PET scans may show diffuse increased metabolic activity in affected areas. Chest CT findings in chronic GVHD patients commonly include ground glass opacities in the lungs, both peribronchial and paraseptal. Later advanced findings may classically include bronchiectasis. MRI findings in sclerotic type GVHD may include edema and enhancement within deep fascial planes.

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Association of Polymorphisms in the HLA-C Locus with Disease Risk for Developing Acute Graft Vs. Host in Pediatric Patients with Allogeneic Hematopoietic Stem Cell

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The acute graft versus host disease (aGVHD) is the most common complication subsequent to allogeneic hematopoietic progenitor cell donor match HLA (Human Leukocyte Antigen), occurring in 30-50%. In hematopoietic progenitor transplantation (HSCT) there are other genetic factors such as minor histocompatibility antigens and cytokines. The HLA-C locus has been divided into two groups according to the amino acid at position 80; this variation is important for the type of KIR (killer cell Ig-like receptor). The C1 group contains a lysine at position 80 and includes Cw2, -4 alleles -, - 5, -6, -15. The group includes C2 Cw1, -3, -7, -8 alleles contains an asparagine at position 80 alleles of the C1 group for KIR2DL1 and KIR2DS1 receptor and group C2 alleles bind to KIR2DL2 receptors bind and KIR2DL3. The interaction of the different alleles of HLA-C locus with KIR receptors expressed on NK cells cytotoxic activity changes which influence the risk for development of aGVHD.