At day +133 for evaluation of GVHD immunity may cause homeostatic proliferation of latently infected cells, decreasing the risk of reinfection. Reinstiitution of cART resulted in suppression of detectable viral replication after 4 wks.

Analytic Treatment Interruption

• At day +784 (Figure 1A), an ATI started according to a Mayo Clinic IRB approved protocol (15-001678).

• Plasma HIV RNA was monitored and undetectable Q2 wks for 12 wks, then Q4 wks .

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• Viral genotype revealed no resistance associated mutations. The patient denied any potential HIV exposure to explain a potential new infection. Reinstiitution of cART resulted in suppression of detectable viral replication after 4 wks.

Results (Figure 1)

• Plasma HIV RNA viral load was undetectable (<10 copies/mL) at day +4 post transplant, was detectable but below the LOQ (<20 copies/mL) on days +15 and +56, but again became undetectable by day +91 and remained undetectable through follow up (Figure 1A).

• Clinical peripheral blood HIV proviral DNA Test, which was positive pre-transplant, became undetectable by day +56 and remained undetectable through day +604, when the assay was no longer commercially available.

• HIV RNA was undetectable by ISH of 15 total colon biopsy sections obtained on day +133 for evaluation of GVHD-associated diarrhea (Figure 1B).

• The patient underwent leukapheresis on day +11 pre-transplant, and on days +142, +288, +436 and +888. Multiple measures of HIV reservoir size in peripheral blood cells revealed significant reductions in reservoir size (Figure 1C, E and F).

• Single genome sequencing and phylogenetic analysis identified identical clones at day +142 (Figure 1D), possibly consistent with homeostatic proliferation of latently infected cells in the post-transplant period.

Results (Figure 2)

• Microchimerism evaluation revealed 8% of DNA in circulating CD4 T cells at day +142 was of recipient origin, during the time when the patient was experiencing clinical GVHD (Figure 2A). By day +265, only 0.0013% of circulating CD4 T cell DNA was of recipient origin, or 13 per 10^6 cells.

• Consistent with the "Berlin patient" (Plos Pathog. 2013;9(5):e1003347.), our patient demonstrated devolution of anti-HIV antibodies, as demonstrated by decreasing number and intensity of anti-HIV bands on western blot from day -119 to day +888 (Figure 2B).

Conclusions

• Allogeneic PBST in the setting of HIV is associated with significant reductions in HIV reservoir size by multiple measures, including prolonged cART free remission.

• Allogeneic PBST in the setting of suppressed viral replication may be associated with loss of HIV-specific immunity. Further immunologic studies are underway.

• We hypothesize that immune activation in the setting of GVHD without anti-HIV specific immunity may cause homeostatic proliferation of latently infected cells, decreasing the chance of HIV eradication.