Allogeneic hematopoietic stem cell transplantation (HSCT) has been shown to lead to associated HIV RNA (CA-US HIV RNA) in cells from peripheral lymph node tissue (Fig 1).

The patient underwent HSCT with RIC in April 2010. He had full donor Δ32 mutation was tested by PCR.

HIV specific analyses and post-HSCT

Fig 1
Timeline, HIV RNA and DNA analyses

In October 2014 the patient had an unexpected viral rebound with >10^5 HIV RNA copies/ml and subsequent immunological exacerbation of graft vs host disease, or undisclosed poor adherence.

The reasons for viral rebound are unclear given that no analytic treatment interruption had been performed. Possible reasons for viral rebound are poor drug absorption due to an exacerbation of graft vs host disease, or undisclosed poor adherence.

The patient underwent HSCT with RIC in April 2010. He had full donor Δ32 mutation was tested by PCR.

Patient clinical baseline characteristics

Table 1

The phylogenetic tree shows clustering of the patient sequences with bootstrap value of 100 phylogenetically. Phylogenetic analysis of pre- and post HSCT sequences from the protease and reverse transcriptase regions, amplified from plasma RNA samples from the studied patient (Patient A). The phylogenetic tree shows clustering of the patient sequences with bootstrap value of 100 phylogenetically. Phylogenetic analysis of pre- and post HSCT sequences from the protease and reverse transcriptase regions, amplified from plasma RNA samples from the studied patient (Patient A).

There was a marked reduction in antibody detectability by western-blot and CMIA assays post RIC HSCT. CD4+ T cell responses to HIV-1 antigen were also absent post-HSCT.

Similarly, the assessment of HIV reservoir related measurements showed undetectable HIV RNA (both routine and single-copy assays) and HIV DNA in peripheral blood and undetectable CA-US HIV RNA in lymphoid tissue cells.

Together with other published reports on HIV infected individuals undergoing HSCT, these results describe the profound changes to the HIV immune response and marked decay of HIV RNA and DNA in blood and tissue to undetectable levels.

Despite these profound changes, the patient experienced HIV-1 rebound over 4 years after the HSCT, and despite being treated with ART throughout the transplant and post-transplant period.

The reasons for viral rebound are unclear given that no analytic treatment interruption had been performed. Possible reasons for viral rebound are poor drug absorption due to an exacerbation of graft vs host disease, or undisclosed poor adherence.

HIV viral rebound following Allogeneic Hematopoietic Stem Cell Transplantation

HIV specific analyses and post-HSCT

Table 2

The patient entered a phase of disease control after ART was initiated in February 2014. However, ART was stopped in May 2014 due to lack of adherence. Thereafter HIV RNA rebounded with >10^5 copies/ml. The patient then entered an infection phase where he was treated with ART.

In October 2014 the patient had an unexpected viral rebound with >10^5 HIV RNA copies/ml and subsequent immunological exacerbation of graft vs host disease, or undisclosed poor adherence.

The reasons for viral rebound are unclear given that no analytic treatment interruption had been performed. Possible reasons for viral rebound are poor drug absorption due to an exacerbation of graft vs host disease, or undisclosed poor adherence.

Viral rebound after HSCT, despite almost absent immune responses against HIV and undetectable levels of HIV RNA and DNA in blood and tissue, is an important observation for the future design of studies investigating HSCT in HIV patients, studies targeting the HIV reservoir and considerations relating to analytic treatment interruptions in these patients.

Summary & Conclusions

- There was a marked reduction in antibody detectability by western-blot and CMIA assays post RIC HSCT. CD4+ T cell responses to HIV-1 antigen were also absent post-HSCT.
- Similarly, the assessment of HIV reservoir related measurements showed undetectable HIV RNA (both routine and single-copy assays) and HIV DNA in peripheral blood and undetectable CA-US HIV RNA in lymphoid tissue cells.
- Together with other published reports on HIV infected individuals undergoing HSCT, these results describe the profound changes to the HIV immune response and marked decay of HIV RNA and DNA in blood and tissue to undetectable levels.
- Despite these profound changes, the patient experienced HIV-1 rebound over 4 years after the HSCT, and despite being treated with ART throughout the transplant and post-transplant period.

- The reasons for viral rebound are unclear given that no analytic treatment interruption had been performed. Possible reasons for viral rebound are poor drug absorption due to an exacerbation of graft vs host disease, or undisclosed poor adherence.
- Viral rebound after HSCT, despite almost absent immune responses against HIV and undetectable levels of HIV RNA and DNA in blood and tissue, is an important observation for the future design of studies investigating HSCT in HIV patients, studies targeting the HIV reservoir and considerations relating to analytic treatment interruptions in these patients.

Phylogenetic analysis of pre- and post HSCT sequences from the protease and reverse transcriptase regions, amplified from plasma RNA samples from the studied patient (Patient A). The phylogenetic tree shows clustering of the patient sequences with bootstrap value of 100 phylogenetically. Phylogenetic analysis of pre- and post HSCT sequences from the protease and reverse transcriptase regions, amplified from plasma RNA samples from the studied patient (Patient A). The phylogenetic tree shows clustering of the patient sequences with bootstrap value of 100 phylogenetically. Phylogenetic analysis of pre- and post HSCT sequences from the protease and reverse transcriptase regions, amplified from plasma RNA samples from the studied patient (Patient A). The phylogenetic tree shows clustering of the patient sequences with bootstrap value of 100 phylogenetically. Phylogenetic analysis of pre- and post HSCT sequences from the protease and reverse transcriptase regions, amplified from plasma RNA samples from the studied patient (Patient A). The phylogenetic tree shows clustering of the patient sequences with bootstrap value of 100 phylogenetically. Phylogenetic analysis of pre- and post HSCT sequences from the protease and reverse transcriptase regions, amplified from plasma RNA samples from the studied patient (Patient A). The phylogenetic tree shows clustering of the patient sequences with bootstrap value of 100 phylogenetically.