Two More People May Be Cured of HIV After Stem Cell Transplants

One patient experienced viral rebound after an initial treatment interruption but remains in remission after stopping antiretrovirals a second time.

March 11, 2025 By Liz Highleyman

Two more people appear to be free of HIV after stem cell transplants for cancer treatment, according to a pair of posters presented at the Conference on Retroviruses and Opportunistic Infections (CROI 2025) this week in San Francisco. If the men remain in remission, they will be the ninth and tenth known cases of a functional cure after the procedure.

The first man, dubbed the Chicago Patient, is notable because he remains in remission 10 months after a second antiretroviral treatment interruption, suggesting that early viral rebound during an initial interruption does not preclude a cure. The second man, the Oslo patient, had a stem cell transplant from a sibling and received the immunosuppressive drug ruxolitinib to treat severe graft-versus-host disease (GVHD); he remains in remission two years after stopping antiretrovirals.

While stem cell transplantation is too risky for people without advanced cancer, each new case offers clues that could help scientists develop more widely accessible functional cure approaches.

“Every case is important to build the knowledge about how cure can be achieved,” Marius Trøseid, MD, PhD, of Oslo University Hospital, said at a conference media briefing. “It’s important now to compare all these cases to try to find similarities…to see if we can find some things in common that could be used in future cure studies.”

A Handful of Cures

Antiretroviral therapy (ART) can keep HIV suppressed indefinitely, but the virus inserts its genetic blueprints into host cells and establishes a long-lasting reservoir that is nearly impossible to eradicate.

The first person known to be cured of HIV—Timothy Ray Brown, the original Berlin Patient—received two transplants to treat acute myeloid leukemia (AML) from a donor with two copies of a mutation known as CCR5-delta32, which disables a receptor most strains of HIV use to enter cells. Prior to the transplant, he underwent intensive chemotherapy and radiation

conditioning therapy to kill off malignant immune cells and make room for healthy new ones. Afterward, he developed near-fatal GVHD, which occurs when donor immune cells attack the recipient.

As first reported in 2008, he stopped ART but his viral load did not rebound. Over the years, scientists tested his blood, gut and other tissues, finding no evidence of intact HIV anywhere in his body. At the time of his death in September 2020, Brown had been free of HIV for more than 13 years.

Four other people—Adam Castillejo (the London Patient), Marc Franke (the Düsseldorf Patient), Paul Edmonds (the City of Hope Patient) and a woman in Marseille (the French Patient)—were also cured after receiving stem cell transplants from donors with the double CCR5-delta32 mutation. All three remain off antiretrovirals without viral rebound.

Initially, experts thought Brown's cure was attributable to the double mutation. But in 2022, researchers described the New York Patient, a woman with leukemia who received a combination of umbilical cord blood cells with the CCR5-delta32 mutation and partially matched adult stem cells from a relative without the mutation. The following year brought news about Romuald (the Geneva Patient), who appears to have been cured after a transplant using so-called wild-type stem cells with no copies of the mutation. Finally, last summer, researchers presented the next Berlin Patient, a man with a single copy of the mutation who is in long-term remission after a transplant from a donor who also has a single copy.

The Chicago Patient

Paul Rubinstein, MD, of the University of Illinois at Chicago Medical Center, presented the first case, which involves a 67-year-old man who had been living with HIV for 14 years when he was diagnosed with AML. He underwent reduced-intensity conditioning therapy followed by a stem cell transplant from an unrelated donor with the double CCR5-delta32 mutation. A year later, his plasma viral load, HIV RNA and DNA in peripheral blood cells and HIV-specific CD4 and CD8 T-cell responses were undetectable and his HIV antibody levels were low, so he stopped antiretrovirals 15 months after the transplant.

The man's plasma viral load rebounded to nearly 800 copies after about two months, and he restarted treatment with Biktarvy (bictegravir/tenofovir alafenamide/emtricitabine). Despite the rebound, HIV RNA and DNA remained undetectable in blood cells derived from the donor, and HIV-specific T-cell responses and antibody levels did not increase. This suggests that the resurgent virus came from a residual reservoir of cells infected prior to the transplant, but his new cells "truly were protected," according to Rubinstein.

After nearly two years back on ART, the man tried a second treatment interruption, and he remains in remission 10 months later. This is the first known case of sustained remission after viral rebound during an initial treatment interruption following a stem cell transplant, indicating that early rebound does not rule out the possibility of a functional cure, the researchers concluded.

This case “demonstrates how profoundly difficult it is to get rid of the reservoir” and “shows how protective these CCR5-delta32 cells are,” Rubinstein told reporters. “If virus rebounds, remission is still possible.”

The Oslo Patient

Trøseid described the second case, which involves a 58-year-old man who was also HIV-positive for 14 years before being diagnosed with myelodysplastic syndrome, a precursor to AML. The patient, who had a single copy of the CCR5-delta32 mutation, received a transplant from a brother with a double mutation.

After the transplant, the man developed severe and prolonged GVHD, which was treated with immunosuppressive drugs, including the JAK inhibitor ruxolitinib (Jakafi), which the Geneva Patient also received. Testing showed that the donor cells completely replaced his immune system.

The man’s plasma viral load remained undetectable, and he stopped antiretrovirals two years after the transplant. Two years into the treatment interruption, his plasma viral load is still undetectable, HIV-specific CD4 and CD8 T-cell responses are absent and his HIV antibody levels are waning, suggesting there may be “no remaining virus left to trigger the immune system,” Trøseid said. Although traces of HIV DNA were detected in gut lymphoid tissue, no intact HIV DNA was found in his blood or gut, and no replication-competent virus was detected in a viral outgrowth assay.

“The Oslo Patient adds valuable evidence to understanding HIV remission after hematopoietic stem cell transplantation,” the researchers concluded.

[Update: The Oslo Patient case was published in Nature Microbiology on April 13, 2026.]

Clues to a Cure

Researchers are still trying to figure out why these people were cured after stem cell transplants while other attempts have failed—and there does not seem to be a single decisive factor common to all cases.

Most of the patients received transplants from donors with a double CCR5-delta32 mutation, but some had donors with only one or no copies of the mutation. Some underwent intensive conditioning therapy, while others received gentler regimens. Similarly, some experienced severe GVHD but others did not. The size of the pre-existing viral reservoir may play a role, and the effects of ruxolitinib remains unclear.

“So far, there has been a lot of focus on the CCR5 mutation, but I think after the Geneva Patient, there is now more focus on reducing the reservoir,” Trøseid told reporters. “At the end of the day,” Rubinstein concurred, “we’re going to have to look at all of these cases together to get some kind of mechanistic answer as to how these things are happening.”

Stem cell transplantation is an arduous and costly procedure limited to people with advanced cancer, so it's far from a feasible solution for most people living with HIV worldwide. Researchers are therefore exploring other ways to protect cells from HIV, including using CRISPR gene editing to disable or delete CCR5 receptors on immune cells.

But there is an urgent need for strategies that expand gene therapy beyond CCR5 modification to target multiple stages of HIV replication, according to Timothy Henrich, MD, of the University of California San Francisco, and colleagues.

In another study presented at CROI, Henrich's team modified stem cells from people with HIV who received autologous transplants to treat lymphoma. All of the cured patients described above received so-called allogeneic stem cell transplants from a donor. An autologous transplant, in contrast, involves collecting an individual's own stem cells before they undergo chemotherapy or radiation and later returning them to the body.

In this Phase I study, sponsored by the National Cancer Institute's AIDS Malignancy Consortium, a lentivirus vector was used to deliver three HIV-resistance genes to autologous stem cells: a CCR5 shRNA to block viral entry, a chimeric TRIM5alpha gene to prevent capsid uncoating and reverse transcription, and a TAR decoy to prevent transcriptional activation.

Three cohorts, totaling 11 patients, received combinations of gene-modified and unmodified stem cells in different ratios. All participants experienced "successful and steady" engraftment of transplanted cells over the course of one to two years, and the modified peripheral blood cells persisted long-term. Several patients had intermittent or sustained low-level detectable plasma viral load after their transplants.

One person with an undetectable viral load underwent an optional antiretroviral treatment interruption at 42 months post-transplant. Testing showed that the gene-modified cells were around 75% protected from HIV infection during the treatment interruption. This person experienced viral rebound, resumed treatment after eight weeks and again achieved an undetectable viral load.

"Gene modification of hematopoietic stem cells in people with HIV requiring autologous transplant for lymphoma targeting three unique steps in the HIV life cycle led to long-term persistence of modified lymphocytes," Henrich and colleagues concluded.

This research is still very early. Here, too, it only applies to people who need a stem cell transplant for cancer treatment, though autologous transplants don't necessitate finding a suitable donor, and they're generally better tolerated than allogeneic transplants, without the risk of GVHD. But the study further adds to the evidence that could one day lead to a more broadly applicable functional cure.

(Editor's note: this report was updated to include an omitted case, making the two cases reported at CROI the ninth and tenth known HIV cures after a stem cell transplant.)

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