13 People Now Appear Cured of HIV After Stem Cell Transplants

The Essen Patient, who also has hepatitis B, and the Kansas City Patient remain in long-term remission after stem cell transplants to treat cancer, offering new clues for a functional cure.

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Two more people appear to be free of HIV after stem cell transplants for cancer treatment, according to case presentations at the 26th International AIDS Conference (AIDS 2026) this week in Rio de Janeiro. If they remain in remission, they will be the 12th and 13th cases of a cure after the procedure.

The Essen Patient is notable because he is the first to have hepatitis B coinfection. The Kansas City Patient, the youngest, offers more information about the biological markers of a cure. 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.

“It’s exciting to see another person join the ranks of those who have achieved sustained HIV remission without antiretroviral treatment,” International AIDS Society president Beatriz Grinsztejn, MD, PhD, said at a media briefing. “These transplants are high-risk procedures for life-threatening cancers—not scalable cures for HIV. However, these cases do provide important insights that help advance broader HIV cure research.”

The Essen Patient

Antiretroviral therapy can keep HIV suppressed indefinitely, but the virus inserts its genetic blueprints into host cells and establishes a long-lasting reservoir that makes viral eradication nearly impossible.

The first person known to be cured of HIV—Timothy Ray Brown, the original Berlin Patient—received two stem cell transplants to treat acute myeloid leukemia from a donor with double copies of a mutation known as CCR5-delta32, which disables a receptor most strains of HIV use to enter cells. As first reported in 2008, Brown stopped antiretroviral therapy, 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.

Carina Elsner, PhD, of University Hospital Essen in Germany, described the Essen Patient, a man who tested positive for HIV in 2003 with a CD4 T-cell count low enough for an AIDS diagnosis (146). In 2010, at age 51, he was diagnosed with chronic lymphocytic leukemia. In 2020, after conditioning chemotherapy and whole-body radiation, he received a stem cell transplant from an unrelated matched donor with two copies of the CCR5-delta32 mutation. At the time, he was on an antiretroviral regimen containing an integrase inhibitor and tenofovir alafenamide, which has dual activity against both HIV and hepatitis B virus (HBV).

The man shows evidence of mixed-tropic HIV, meaning he has some virus that uses the CCR5 receptor to enter cells and some that uses a different coreceptor known as CXCR4. A transplant of stem cells with the CCR5 mutation, therefore, might not offer protection against CXCR4-tropic virus.

After his transplant, the man achieved complete remission of leukemia and continued to have an undetectable plasma HIV viral load. At about two years post-transplant, traces of HIV DNA could still be detected in peripheral CD4 cells—indicating that the viral reservoir was not completely depleted—but researchers could find no replication-competent virus capable of producing new copies. Likewise, they found no intact proviral DNA in gut biopsy samples. What's more, the man had no evidence of HIV-specific T cells and waning HIV-specific antibody responses, suggesting there was no remaining viable virus to trigger his immune system.

At 51 months after the transplant, the man stopped his antiretroviral therapy in a closely monitored analytical treatment interruption. Now, about 14 months later, he still has undetectable HIV viral load. The presence of some CXCR4-tropic virus did not preclude long-term remission, Elsner noted.

However, the man did experience hepatitis B reactivation 25 days after stopping treatment. He was vaccinated for HBV repeatedly after the transplant but didn't respond well, Elsner said.

Like HIV, HBV can establish a latent reservoir in cells despite antiviral therapy. About 15 weeks after the treatment interruption, the man tested positive for HBsAg, indicating active HBV replication. At about 21 weeks, he started entecavir (Baraclude), an antiviral with activity against HBV but not HIV. At about 31 weeks, he showed a transient elevation of liver enzymes, a sign of liver inflammation. After that, at about 35 weeks, he experienced HBeAg seroconversion, which indicates remission. He does not yet have hepatitis B surface antibodies—a marker of immunity—but does have HBV-specific T cells fighting the virus, suggesting that his newly reconstituted immune system may enable a functional cure of hepatitis B as well as HIV.

The Kansas City Patient

Wissam El Atrouni, MD, an infectious disease physician at the University of Kansas Medical Center, described the second case. The Kansas City Patient was diagnosed with HIV in 2018 with a CD4 count of just 23. He was treated with antiretroviral therapy including an integrase inhibitor, a boosted protease inhibitor, tenofovir alafenamide and emtricitabine. His viral load became

undetectable and his CD4 count recovered somewhat, but it mostly remained under 300 before the transplant. In June 2020, he was diagnosed with acute myeloid leukemia at age 21, making him by far the youngest person to be cured after a stem cell transplant.

In October 2020, the man received a transplant from an unrelated matched donor with the double CCR5-delta32 mutation after conditioning chemotherapy, which put his leukemia into remission. The transplant was not completely effective, however, and he needed a boost of stem cells from the same donor four months later. During this time, he experienced several complications, including persistent low blood cell counts, E. coli and Pseudomonas aeruginosa infections, invasive fungal sinusitis, cytomegalovirus colitis and graft-versus-host disease (GVHD), which occurs when donor immune cells attack the recipient. At one point, El Atrouni said, they did not think he was going to make it out of the intensive care unit.

The man had three HIV viral blips after the initial transplant and before the stem cell boost, but his viral load has remained undetectable since January 2021. About 2.5 years after the transplant and again seven months later, intact proviral HIV DNA could not be detected in peripheral CD4 cells.

In February 2025, the man started an analytical treatment interruption. About a year after stopping antiretrovirals, his CD4 count stopped bouncing around and stayed above 500. Now, at 17 months post-transplant, plasma HIV RNA viral load, intact proviral HIV DNA and replication-competent virus remain undetectable in his blood; tissue biopsies have not yet been done. However, he does still have persistent HIV antibodies and HIV-specific T-cell responses. El Atrouni suggested that this may be due to low-level residual HIV antigens rather than deep, undetected viral latency.

Clues to a Cure

Researchers are still trying to determine why some 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.

In addition to Brown and the two newly reported cases, seven other people—Adam Castillejo (the London Patient), Marc Franke (the Düsseldorf Patient), Paul Edmonds (the City of Hope Patient), a woman in Marseille (the French Patient), the Chicago Patient, the Oslo Patient and the Toronto Patient—were also cured after receiving stem cell transplants from donors with the double CCR5-delta32 mutation.

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. In 2024, 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. And Romuald, the Geneva Patient, appears to be cured after receiving wild-type stem cells with no copies of the mutation.

Prior to his first stem cell transplant, Brown underwent strong chemotherapy and radiation conditioning therapy to kill off malignant immune cells and make room for healthy new ones. Afterward, he developed severe GVHD. Some of the other cured patients also underwent intensive conditioning therapy, but others received gentler regimens. Some experienced severe GVHD—which some experts think may have contributed to HIV eradication—but others did not.

As the Kansas City Patient demonstrates, stem cell transplantation is an arduous and risky—not to mention costly—procedure that is only appropriate for people with life-threatening cancer, so it's far from feasible for most people living with HIV. But the growing ranks of cured patients add to our understanding of how the immune system might permanently control, or even eradicate, HIV. Researchers are now exploring other ways to protect cells from the virus, including using CRISPR gene editing to disable or delete CCR5 receptors on susceptible immune cells.

"I'm thrilled that there are more cure cases. It gives everyone hope, but I'm not sure how much new we learned," Joseph Eron, MD, University of North Carolina at Chapel Hill, told POZ. "Can we do this without having to destroy someone's immune system and put them at risk? What we need to do is figure out how to produce the same result by making cells safe from reinfection by modifying receptors without the need for aggressive radiation and chemotherapy."

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