Brazilian Man With HIV Is No Longer in Remission

The São Paulo Patient is no longer HIV-free after more than 15 months off treatment, but was it a relapse or reinfection?

March 23, 2021 By Liz Highleyman

A Brazilian man who last summer had no evidence of remaining HIV after more than a year off antiretroviral treatment once again had detectable viral load a few months later, according to a report at the recent Conference on Retroviruses and Opportunistic Infections.

The case was first reported at the 2020 International AIDS Conference (AIDS 2020) last July. At the time, Ricardo Diaz, MD, PhD, of the Federal University of São Paulo, who presented the case, and other experts said it was too soon to say whether the man could be functionally cured and stressed that more investigation was needed.

So far, just two people appear to have been cured of HIV, both after bone marrow transplants to treat advanced cancer. They received stem cells from a donor with a rare genetic mutation that prevents most types of HIV from entering immune cells. Timothy Ray Brown, the famous Berlin Patient, had no evidence of viable HIV anywhere in his body for more than 13 years after stopping antiretrovirals; Brown died last September after his leukemia relapsed. Adam Castillejo, the London Patient, still has no detectable virus after more than three years off HIV treatment. But stem cell transplants are too dangerous for people who don't have life-threatening cancer, and it is unlikely the intensive and costly procedure could be scaled up to treat the millions of people living with HIV worldwide.

The anonymous São Paulo Patient was a participant in a clinical trial that evaluated intensified drug regimens in an effort to reduce the size of the viral reservoir—the latent virus sequestered in long-lived immune cells that makes it so difficult to cure HIV.

The man, who was 35 at the time of last summer's report, was diagnosed with HIV in October 2012. At one point, his viral load reached more than 20,000, showing that he is not a natural elite controller like Loreen Willenberg and a small group of other people who are able to maintain viral suppression despite never using antiretrovirals.

When the Brazilian man entered the trial in September 2015, he'd had viral suppression on treatment for more than two years using a standard three-drug regimen. He had never interrupted

treatment or experienced virological treatment failure. At that time, two additional antiretrovirals—the integrase inhibitor dolutegravir (Tivicay) and the HIV entry inhibitor maraviroc (Selzentry)—were added to his regimen. He also received nicotinamide, a form of niacin (vitamin B-3) that appears to prevent immune cells from entering an inactive state. He remained on this five-drug combination for 48 weeks and then went back to standard therapy.

In March 2019, he started a closely monitored treatment interruption. At AIDS 2020, more than 15 months later, Diaz reported that the man continued to have undetectable HIV RNA (the form of viral genetic material measured in a typical viral load test) and HIV DNA (the form that largely makes up the viral reservoir). In addition, his antibody level declined steadily, dropping low enough that a rapid antibody test became negative.

“These are exciting findings, but they’re very preliminary,” Monica Gandhi, MD, PhD, of the University of California, San Francisco, said during a media tele-briefing prior to AIDS 2020. “This has happened to one person and one person only. We need to watch the other participants in this important study.”

As Diaz would report, the São Paolo man was the only one of the five trial participants treated with this particular intensified regimen who had prolonged viral suppression after stopping treatment. But within months, this was no longer the case.

Diaz and his team continued to monitor the man’s viral load and examined stored blood samples to analyze cell-mediated immune responses and other parameters. While the man was on the intensified regimen, he showed increasing T-cell responses against HIV’s envelope and Gag proteins. But these cell-mediated responses “progressively disappeared” in parallel with the decline in his HIV-specific antibodies after the treatment interruption.

In September 2020, the man was diagnosed with secondary syphilis. At the time, his viral load was still undetectable. But in November, 72 weeks into the treatment interruption, he experienced symptoms suggesting a resurgence of viral replication, including fever, chills, headache and diarrhea. At that time, he was found to have a viral load exceeding 6,300. His HIV antibodies also started to rise. He restarted treatment in early December, and by January 1, his viral load was once again undetectable.

The emerging HIV strain had substantial genetic differences in its envelope and Gag proteins compared with the baseline strain—more variation than would be expected due to normal viral evolution within the man’s body, Diaz said.

Other possibilities, he suggested, include the reemergence of “ancient strains” from prior dual infection that were able to gain a foothold when the dominant virus was suppressed or perhaps reinfection with a new strain. Further genetic sequencing and analysis of antibody responses is underway in an effort to solve this mystery.

While understanding the need to determine whether this was a case of relapse or reinfection, some advocates have expressed concern that excessive probing of the São Paulo Patient’s risk

behavior or the viral status of his partners could make people hesitant to participate in HIV cure research.

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