Neurological Problems Common Despite HIV Treatment

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Antiretroviral (ARV) therapy does not appear to prevent the development of mild neuropsychological impairment, at least in people who waited to start ARVs until their CD4s dropped below 200. These data, published December 7 in Neurology, appear to contradict other recent studies showing a neurological benefit from ARVs.

At one time, up to 15 percent of people with HIV went on to develop severe HIV-associated dementia. While the rate of dementia has fallen below 2 percent in more recent years, studies also suggest that at least 50 percent of people living with HIV still experience some degree of mild neurological impairment. Generally, the impairment is so mild that specialized testing is necessary to uncover it. Though this impairment appears to progress only slowly, if at all, it is unknown whether it could ultimately result in higher rates and earlier onset of age-related dementia.

To further add to this growing knowledge of neuropsychological (NP) deficits in people with HIV, Robert Heaton, PhD, from the University of California in San Diego, and his colleagues with the CNS HIV Antiretroviral Therapy Effects Research (CHARTER) study, conducted an analysis of the cohort's participants. More than 1,500 HIV-positive men and women are enrolled in CHARTER and being followed over time to determine the frequency and causes of HIV-associated neurocognitive disorders (HAND) and other neurological disorders that occur in people with HIV.

Most of the participants are taking ARVs. About 56 percent of those on ARVs have undetectable virus levels in the blood, and 84 percent have undetectable viral loads in the central nervous system (CNS). Complicating factors are common, with a significant percentage of the participants having a history of depression, substance abuse or both.

Previous reports on CHARTER indicate that about half of the participants have mild to moderate neuropsychological impairment. In the current study, Heaton and his colleagues analyzed how much a variety of factors—including ARV use, lowest ever CD4 count (nadir) and viral load—influenced the likelihood of a person having NP impairments.

Heaton's team found that, overall, ARVs did not protect against NP impairment. In fact, people using ARVs were actually more likely to have NP deficits than those not on ARVs—a finding that mirrors those of an AIDS Clinical Trials Group study reported last year. The authors make clear, however, that people taking ARVs are also far more likely to have been living with HIV for longer and to have had more severe disease progression.

In a deeper analysis, the researchers found that two factors were significantly predictive of NP

impairment in people who had no other complications (such as depression or substance abuse). These were a CD4 nadir under 200 cells and a detectable viral load.

Heaton and his team acknowledge that the nature of their study, which looked at a single snapshot in time, rather than multiple measures over time, means that their results cannot be broadly interpreted. This study design may also help explain why they did not find that ARV therapy had a protective effect, in contrast to studies that did follow study participants over time.

The authors state, however, that their data do lend weight to a growing consensus that NP impairment may be set in motion quite early in HIV disease, perhaps in the first months or years following infection, and that early treatment—especially before CD4s drop below 200—may be warranted to prevent its development.

“Randomized clinical trials targeting each of these mechanisms are needed to determine what will be the most clinically useful approach to the prevention and treatment of [HIV-associated neurological disorders],” the authors conclude.

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