Could You Have HAD?

Stephen Gendin passes an early-warning test for HIV-associated dementia. He may be nuts, but he isn't crazy.

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This month, David Dorfman, PhD, neuropsychologist at the Mount Sinai School of Medicine's Neuro-AIDS Research Program in New York City, discusses his neuropsychological testing of POZ *contributing editor Stephen Gendin.*

Although Stephen has *not* experienced the mental-function problems that might normally lead people with HIV to fear harmful brain changes, his desire to participate in the research project called the Manhattan HIV Brain Bank led him to undergo a neuropsychological examination. This included a broad range of neurologic and neuropsychiatric tests aimed at evaluating his cognitive (thinking) function.

This type of exam is useful for HIVers since it can establish current mental status and provide a baseline for comparison with follow-up tests in later years to determine whether mental function has worsened. More than half of all HIV positive people experience nervous--system complications at some point, and many fear the possibility of developing HIV-associated dementia (HAD), a widely misunderstood condition.

It is not known precisely how the virus causes HAD. We do know that lower CD4 counts increase the risk, but that higher counts don't preclude the development of problems. Half of newly diagnosed HAD cases are in people with CD4s *below* 200, but a fourth are in those with CD4s *above* 400. Common symptoms are memory lapses, weak concentration, slowed thinking, apathy, altered mood, withdrawing from others, and slowed or clumsy movements.

It is crucial that a complete workup be carried out to assess the sources of such problems. Several different infections and cancers that affect the central nervous system, as well as psychological problems such as depression, can contribute to mental changes, and most are treatable. As part of such a workup, the type of neuropsychological exam that Stephen underwent can help by assessing how well the brain is functioning. The exam consists of a set of tasks, some of which are like the ones you might take in school. Others resemble puzzles and games.

Not all the mental changes that such tests might show add up to HAD. Determining the appropriate diagnosis and treatment requires overcoming three common misconceptions about

HIV-related cognitive problems:

Myth 1: Mental changes mean you're "losing your mind."

The truth is that in its early stages HAD resembles the mental changes that come with normal aging. Although unwelcome, these changes are easily overcome by simple strategies such as keeping lists, organizing medications and the like. Furthermore, in many cases the thinking problems may result from Minor HIV Cognitive Motor Disorder (MCMD). There is no clear evidence indicating that MCMD progresses to HAD, and its signs and symptoms are usually an annoyance rather than a handicap.

Myth 2: HAD is relentless, destined to get worse as time goes on.

The truth is that even without treatment, HAD is often stable for long periods, and sometimes for a lifetime. In many cases, treatment reverses the dementia.

Myth 3: HAD is untreatable.

This is the most destructive misconception. In fact, several treatments can be very helpful. Some antiretrovirals, such as d4T (Zerit) and AZT (Retrovir), readily cross into the brain, although others, including most protease inhibitors, do not. In many cases, all that's needed is to include in one's combo at least one drug that can enter the brain in sufficient amounts. This doesn't work for everyone, but when it does, the results can be dramatic. Some of our patients with disabling cognitive problems have improved so much that neuropsychological tests no longer detect impaired function. Another treatment possibility is cognitive remediation—teaching patients behaviors or practices that help to restore their concentration or compensate for handicaps. Finally, there are experimental options, including treatment with nimodipine, memantine, antioxidants and anti-inflammatory agents, all of which appear promising, although insufficiently studied.

In Stephen's case, there was no need to discuss treatment options since we found no evidence that he has HAD. There were some equivocal signs of MCMD, such as mild memory problems and possibly slowed and clumsy movements. But even if Stephen turns out to have MCMD, it probably will not affect his functioning noticeably.

Stephen's case shows the importance of having a baseline evaluation. First, in the future, the equivocal signs that we found on his exam won't be mistaken for the onset of HAD. Second, it is now far less likely that we will miss any subtle changes that might point to a treatable disorder. I recommend that Stephen—and all HIVers—repeat neuropsychological examinations every six months so that changes can be detected and appropriate treatments recommended quickly.
