Crazy? Not at All

Techniques for diagnosing and treating HIV dementia

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In each issue, POZ *publishes a different standard of care, a guide by which people with HIV and their care providers can make personal choices about health care regimens. HIV standards of care differ by region, treatment philosophy and patient population. The following guide to diagnosing and treating dementia was prepared by* ***Justin C. McArthur****, an associate professor of neurology and epidemiology at Johns Hopkins University in Baltimore and a principal investigator with the Dana Consortium for the Therapy of AIDS Dementia. McArthur based these guidelines on a study of more than 300 patients with HIV dementia.*

Dementia appears in less than one percent of asymptomatic people with HIV, but after the onset of AIDS, 15 to 20 percent of people will eventually develop dementia; 20 to 30 percent more will have cognitive symptoms that fall short of full-fledged dementia. The most common symptoms are problems with memory (especially immediate recall), difficulty in walking, mental slowing, depression and psychomotor slowing (in both speech and other movements). In someone with advanced AIDS, these symptoms should raise a red flag about possible dementia. Deficits in language function, attention and recognition memory, while rare in early dementia, can appear as the condition progresses. (Limited research on women has yet to find any discernible gender differences in dementia's manifestations.)

Measuring mental functioning with standardized neuropsychological (NP) tests helps distinguish a patient who is depressed, anxious or using recreational drugs from one who is developing HIV dementia. This is the first step if a patient has mild forgetfulness and slowing that are not progressing rapidly. Also, a vitamin B12 measure is recommended; even without deficiency, B12 supplements may help.

If symptoms are more severe -- such as functional limitation due to memory impairment, motor slowing or apathy -- a workup should be done, including NP tests plus an MRI (a test using powerful magnets to form images of brain structures), a neurology evaluation and an exploration of other possible causes. Most opportunistic infections, such as CMV (cytomegalovirus), PML (progressive multifocal leukoencephalopathy) and herpes, can be ruled out with imaging studies, preferably an MRI. Cryptococcal meningitis can be ruled out with serum cryptococcal antigen; a spinal tap may not be necessary. To screen for possible syphilis, an RPR blood test should be done; a measure of more than 1:16, or a history of untreated syphilis, indicates the need for a spinal tap. If conditions other than direct HIV infection of the brain are discovered, they should be treated with the

appropriate medications.

In severe dementia, particularly if it is rapidly progressive and associated with delirium or seizures, other conditions may be contributing and should be explored. CMV encephalitis probably tops the list. Diagnosing it calls for a more thorough workup, including a spinal tap and a test to screen the fluid for CMV. Ironically, delirium can also be caused by various medications taken by PWAs.

Some antiretrovirals probably offer protection against dementia, although it is not clear what dose is optimal. Early studies that showed protective effects used high doses of AZT, 1,500 mg a day. It is not proven what level of protection results from standard doses of 500 to 600 mg. Still, the incidence of dementia may have stabilized with the more widespread use of antiretrovirals.

Thus, if a patient has confirmed HIV dementia, treatment depends on whether he or she is AZT-intolerant or has been on AZT for many months (and thus may have developed resistance). If the patient is not AZT-intolerant, we prescribe 800-1,000 mg a day of AZT as tolerated (in combination with any already used antiretrovirals), following a complete blood count. Even patients with severe dementia can improve greatly on high-dose AZT. If the patient is AZT-intolerant, we usually prescribe d4T.

If the dementia continues to progress, as it often does, we would either substitute or add d4T with 3TC; d4T is well-tolerated and seems to cross the blood-brain barrier better than ddI or ddC. However, no clinical data have proven d4T's effectiveness against dementia. Most available protease inhibitors (with the exception of the Glaxo experimental drug, Vertex) do not cross the blood-brain barrier and so would probably be ineffective for dementia therapy.

Several experimental treatments are now in clinical trials, including memantine (a blocker of a specific neuron receptor), OPC 14117 (a potent antioxidant drug) and thioctic acid (an antioxidant nutrient).

There will probably never be just one drug that is useful for HIV dementia. In the future, we will most likely use combination therapies -- for example, antiretrovirals plus cytokine blockers such as thalidomide and pentoxifylline, plus new drugs to prevent neurons from dying.

The diagram shown here is based on our clinic's standard of care for diagnosis and treatment of dementia.

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