A Guide to AIDS Research and Counseling

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Treatment of Cognitive Impairment
Penelope Zeifert, PhD, Mark Leary, MD and Alicia Boccellari, PhD

Through a variety of mechanisms, HIV disease can alter thought, emotion, and behavior, leading to symptoms of “cognitive impairment,” a loss in the ability to process, learn, and remember information. The result is a confusing array of changes that inspires fear and helplessness in both clients and providers.

Cognitive impairment occurs in 55 percent to 65 percent of people with AIDS.1,2 Severe cognitive impairment occurs in the later stages of HIV infection. There is disagreement about whether cognitive impairment occurs during the early stages of HIV infection; if it does, it is likely to be subtle and not discernible in activities of daily living.3 This article provides an overview of HIV-related cognitive impairment, focusing on the role mental health providers play in its treatment.

Treatment Approaches
Some aspects of cognitive impairment are treatable if addressed quickly; others are untreatable and rapidly progressive. When cognitive impairment occurs among people with HIV disease, more than one disorder may be present, a situation that complicates assessment and treatment. The response to cognitive impairment is four-fold, requiring: accurate diagnosis; coordination of treatment and education; aggressive treatment of the acute causes of cognitive impairment, for example, opportunistic conditions and delirium; and management through psychotherapy and psychopharmacology of impairment that is not otherwise treatable.

Accurate diagnosis is crucial to distinguishing among the variety of conditions that cause HIV-related cognitive changes.4 The most common cause is direct infection of the brain by HIV. Other causes include opportunistic conditions, organic affective disorders, and conditions—such as delirium and substance abuse—that lead to transient impairment.

When questions of cognitive or behavioral change arise, psychotherapists should encourage clients to contact their physicians to rule out or treat reversible causes of impairment. It is useful to lay the foundation for coordination with medical providers early in the therapeutic relationship by obtaining releases from clients.

Once an HIV-related cognitive disorder is diagnosed, education can make the diagnosis less overwhelming and frightening. It can also foster realistic expectations. Providers should seek to demystify impairment by explaining the physical, emotional, and cognitive changes that may occur, the extent to which these can be ameliorated, and the fact that these changes are organic and are not within the conscious control of the client.

When cognitive impairment cannot be eliminated through treatment, management focuses on psychotherapy and psychopharmacology. There are two psychotherapeutic models for managing HIV-related cognitive impairment.4,5 The first approach—“Adaptation and Compensation”—is used for mild cognitive impairment and aims at reinforcing remaining strengths while helping individuals compensate for changes in ability. The goal of this approach is to maintain independence and self-esteem by encouraging full participation in treatment and life decisions. Supportive psychotherapy including education and problem-solving is most appropriate at this stage.

The second approach—“Environmental Engineering”—best addresses HIV-associated dementia or cognitive impairment of at least moderate degree. Individuals with dementia lose the ability to deal with the changing demands of the world and

*See page 5 of this issue, “Diagnosis of Cognitive Impairment,” for a discussion of the causes of cognitive impairment and diagnostic techniques that can discern among them.
Late last year, when we published a monograph on the subject, it had become clear that cognitive impairment was one of the most frightening conditions associated with HIV disease. *AIDS and the Impact of Cognitive Impairment*, by Penelope Zeifert, Mark Leary, and Alicia Boccellari, set out to catalog the state of the art in terms of etiology, diagnosis, and treatment of the more than one dozen conditions that lead to loss of the ability to process and remember information.

Fears of “losing” one’s mind and the capacity to communicate run deep. For clients who have observed the effects of dementia, fears revolve around the loss of control; for clients with less experience, the term may conjure up images of violence and psychosis. Yet it appears that cognitive impairment is often one of the least likely issues to be spontaneously raised in psychotherapy, and for both clients and therapists, one of the least understood conditions in the pantheon of HIV-related distress.

For this issue of *FOCUS*, we asked Zeifert, Leary, and Boccellari to take on the task of condensing their opus from 90 pages to fewer than 10. The result is a primer that defines the range of impairing conditions, the ways in which to distinguish among them, and most importantly, the variety of ways in which to manage and respond. HIV-related cognitive impairment is in many cases associated with immune decline and disability, but the important point of the book and these articles is that impairment is often treatable both with medication and through behavioral interventions. People with cognitive impairment can remain connected to their thoughts, behaviors, and feelings with the aid of caregivers and providers. By incorporating environmental and behavioral cues into their lives, by establishing routine, and by teasing equanimity and self-control out of a discombobulating situation, providers can help their clients transcend impairment and attain some measure of peace of mind. This understanding is a boon not only to clients, but also to providers themselves, who in learning about these tools, gain comfort in treating people with cognitive impairment.

**References**


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**Adaptation and Mild Impairment**

Mild impairment occurs most often in the presence of HIV-associated minor cognitive/motor impairment, which is caused by direct HIV infection of the brain and which may or may not progress to HIV-associated dementia. Mild impairment may also arise from lymphoma or in the early stages of progressive multifocal leukoencephalopathy (PML) (although both these conditions may cause much more severe impairment and death). Individuals with mild impairment have some slowness in their thinking, memory problems, and decreased problem-solving ability in the face of complex or novel tasks. Mild impairment, however, does not interfere with the ability to live independently or to interact meaningfully with others. Using the Adaptation and Compensation model, psychotherapy focuses on setting realistic goals, facilitating active problem-solving, and compensating for waning skills. In this context, psychotherapy can help client identify wishes for the future and make short- and long-term plans to meet their goals. It is notable that clients with mild cognitive impairment can participate fully in decision-making and that making realistic plans may increase a sense of control and competence.

Psychotherapists should encourage individuals with mild HIV-related cognitive impairment to be as independent as possible by capitalizing on strengths and compensating for limitations. Compensatory strategies are varied, but they include: performing one task at a time to decrease frustration and the tendency to make mistakes; getting enough rest and making appointments early in the day to minimize fatigue; developing routines and avoiding crowded public places to prevent overstim-
Psychotherapeutic treatment requires matching approach with degree of cognitive impairment and developing compensatory or environmental engineering strategies.


In the face of moderate cognitive impairment, individuals are no longer able to work and may need some assistance, but can still take an active part in their daily care and can continue to interact with family and friends. As dementia progresses, individuals may no longer be able to live independently, and they may no longer be able to make reasonable decisions.

In the face of moderate cognitive impairment, psychotherapy can no longer be reflective or psychodynamic. The therapist needs to take a pragmatic and direct approach, be more generous with suggestions, and at times even give advice. The emphasis of therapy shifts from processing feelings to negotiating the practical issues of day-to-day living. A session may consist of checking in on basic competencies like the ability to plan for meals or arrange transportation, or of discussing recent visits with doctors and current health status. The frame of the psychotherapeutic session may also change in response to the client's limitations. For example, the psychotherapist may suggest periodically including the client's partner or caregiver, meeting less often, or talking by telephone.

At the level of moderate impairment, therapy shifts towards Environmental Engineering—defining and matching the client's degree of cognitive impairment with the amount of structure necessary. As the need for assistance increases, the therapist can help the client explore both specific needs and personal and community resources. Discussing a client's reaction to increasing dependence and corresponding changes in interpersonal relationships can offer a sense of control and support.

As impairment worsens, therapy may serve to maintain a sense of continuity and support for the client and, increasingly, for caregivers. Therapists may take on the role of consultant to family and friends, and facilitator between caregivers and health providers. They may be particularly helpful in facilitating a home care plan that incorporates Environmental Engineering interventions.

Management of dementia occurs in three main areas: physical and cognitive changes; safety concerns; and emotional and personality changes. The key principle of intervention is to provide external support to the client in order to preserve the highest level of functioning possible. To achieve these goals, therapists should work with clients and their caregivers to develop and maintain a structured environment that includes the use of routine, physical cues, and safety devices.

For example, to decrease confusion: maintain a familiar environment; provide frequent orientation cues; limit background noise; slow down speech and speak in simple sentences; and remove clutter. To increase physical mobility: use walkers or canes; use chairs with arms to aid in sitting and rising; and install a shower bench or raised toilet seat. To ensure safety: remove slip rugs and organize furniture to provide more open space; provide good lighting; safety proof the tub or shower with non-slip surfaces; install metal plates to cover stove burners; and have clients carry identification cards and wear identification bracelets.

As impairment progresses, clients are likely to undergo emotional and personality changes. Ironically, as cognitive impairment progresses and interferes with daily functioning, cognitively impaired individuals often become less anxious about their symptoms of progressive dementia. Emotions as well as thinking become simplified. The ability to distinguish the fine nuances of emotional states and to sustain emotional states decreases. In addition, organic denial may lead to a lack of awareness of deficits and poor judgment.

While people with moderate cognitive impairment tend to be apathetic, they are...
also overly responsive to stimulation. They can become easily distracted or agitated, and they have little tolerance for frustration. Emotions can be quick and intense, manifesting as sudden mood changes or as angry outbursts that resolve quickly. This changeability in emotions can be easily misunderstood by caregivers, who may see this behavior as volitional, and who may respond in kind with frustration or annoyance.

These changes cannot be addressed directly: confronting clients by challenging their reasoning or proposed course of action is likely to lead to outbursts and a more entrenched position. Redirecting or distracting clients from dangerous or inappropriate behaviors is likely to be more effective.

**Psychopharmacological Interventions**

Psychopharmacologic agents can provide valuable treatment of psychiatric and behavioral symptoms seen in clients with HIV-related cognitive impairment, particularly in response to psychosis, anxiety, and the affective disorders. Patients with HIV-related cognitive impairment, whatever the cause, are likely to be more sensitive than usual to the effects—including undesirable ones—of psychotropic medications. For this reason it is prudent to begin treatment with lower than usual dosages, to make changes gradually, to monitor for side effects regularly and carefully, and to limit the number of psychopharmacologic agents used.

Organic psychosis may develop during the course of HIV-associated dementia, often manifesting as organic mania, but also including auditory or visual hallucinations, paranoia, delusions, and confusion. Treatment of psychosis consists of prescribing moderate potency antipsychotic medications in low dosages, providing a routine and structured environment, and monitoring levels of stimulation.

Organic mania usually occurs with advanced HIV disease and is frequently associated with motor abnormalities and memory problems. Symptoms include hyperactivity, impulsivity, grandiose thinking, agitation, and delusions. Treatment consists of education, medication (lithium and Depakote), a structured environment to ensure safety and medication compliance, and in severe cases, hospitalization.

Cognitive symptoms associated with depression include generalized mental slowing and impaired attention and concentration, both resulting in apparent memory problems. Psychotherapy with depressed patients with mild cognitive impairment may include supportive individual therapy, as well as couples or group therapy. For patients with more serious cognitive impairment, depression is likely to be organic, and antidepressants or psychostimulants may be useful. Psychostimulants are particularly well-tolerated by clients with advanced HIV disease.

**Conclusion**

HIV-related cognitive impairment is manageable using a combination of measures. The fundamental tenets of treatment—accurate diagnosis, coordinated care, psychopharmacological intervention, matching psychotherapeutic approach with degree of impairment, and applying compensatory and environmental engineering strategies—place providers in a powerful position to mitigate cognitive impairment and to improve quality of life.

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**Clearinghouse: Cognitive Impairment**

**References**


Diagnosis of Cognitive Impairment
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HIV-related cognitive impairment is complicated because symptoms may vary only minimally across the many conditions that cause it. Treatment, however, varies dramatically, and so accurate diagnosis is crucial. This article delineates the causes of cognitive impairment and outlines the tools necessary to differentiate them.

Direct Infection of the Brain
Most HIV-related cognitive impairment is caused by direct infection of the brain. HIV appears to damage the "myelin" and reduce electrochemical conduction of nerve impulses. Two conditions result from infection: "HIV-associated minor cognitive/motor disorder" at the mild end, and "HIV-associated dementia," which is an AIDS-defining condition, at the severe end. Dysfunction caused by direct infection is neither progressive, orderly, nor predictable.1 People with HIV-associated minor cognitive/motor disorder do not necessarily develop dementia,2 and it is the degree of impairment in activities of daily living that distinguishes between the two conditions.

Among the functions affected by direct HIV infection of the brain are: concentration, memory, emotion, behavior, ability to perform complex tasks, coordination and balance, and muscle strength. Diagnosis is achieved by confirming the presence of HIV and symptoms of impairment, and excluding other causes through testing.

Opportunistic Diseases
Four HIV-related opportunistic conditions commonly cause cognitive impairment:

toxoplasmosis, cryptococcal meningitis, progressive multifocal leukoencephalopathy, and lymphoma. Toxoplasmosis is caused by a parasite, which when active, typically produces lesions in various parts of the brain. This results in symptoms ranging from motor weakness to seizures to changes in mental status including confusion and memory loss. Common presenting symptoms of toxoplasmosis include fever and constant headache. Ongoing antibiotic treatment can suppress the symptoms of toxoplasmosis but not cure it. Toxoplasmosis is diagnosed in three ways: blood tests, magnetic resonance imaging (MRI), and lumbar puncture ("spinal tap").

Cryptococcal meningitis, a fungal infection of the outer covering of the brain, typically presents with fever and severe headache. Mental status changes range from mild behavioral and personality changes to severe memory loss and confusion. Antifungal drugs can clear infection. For an accurate diagnosis, clients must undergo lumbar puncture.

Progressive multifocal leukoencephalopathy (PML) is a neuropsychiatric disease caused by the JC virus. It usually results in severe dementia and death within several months. However, small but encouraging reports suggest that experimental treatments may stabilize progression. Definitive diagnosis of PML requires a brain biopsy. It is most frequently diagnosed on the basis of MRI scans and marked neurological symptoms (for example, one-sided weakness), which are not present in HIV-associated dementia.

Lymphoma, a cancer originating in the lymphatic system, can invade the brain and cause impairment. Some clients with brain lymphoma present similarly to those with

References


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See also references cited in articles in this issue.
dementia, but significant neurologic symptoms often differentiate the two conditions. While CT or MRI scans are critical for diagnosis, at times a brain biopsy may be necessary to distinguish lymphoma from toxoplasmosis. Radiation therapy is the most effective form of treatment.

Other Causes

“Organic affective disorders” are disturbances of mood that result from specific biological causes. HIV-related organic affective disorders emerge late in the course of HIV disease as immunosuppression increases. Organic mania, similar to the mania of bipolar disorder in symptoms and treatment, occurs four times more often in people with HIV disease when compared to the general population. Organic depression, best identified using neuropsychological assessment, is treated with antidepressants or psychostimulants.

Some cognitive impairment is “transient,” that is, short-lived and reversible. Delirium is a common medical syndrome with a variety of symptoms and underlying causes, including opportunistic infections and some HIV-related medications. It is signaled by abrupt changes in mental status, and clients may present as highly aroused or withdrawn. To accurately assess delirium, it is necessary to conduct “serial” mental status examinations (several times daily and over consecutive days).

While chronic and heavy substance abuse may cause permanent impairment, drug use generally causes transient problems with attention, memory, orientation, agitation, and confusion. Substance abuse may also result in secondary psychiatric disorders such as depression.

Diagnostic Tools

There are five procedures used in the diagnosis of cognitive impairment: mental status examination, neuropsychiatric interview, neurologic examination, laboratory and radiologic testing, and neuropsychological testing. The diagnostic process begins with a mental status examination and neuropsychiatric interview to assess overall level of neuropsychiatric functioning. These procedures may be conducted by a psychotherapist or primary health care provider.

The mental status examination aims at detecting deficits in basic cognitive functions by looking at a variety of indicators including: general appearance, coordination, memory, language, affect, orientation, and concentration. The neuropsychiatric interview expands upon the mental status examination, adding a comprehensive medical and psychological history and assessing the client’s current ability to function at work and in activities of daily living.

Neurologic examination and laboratory and radiologic testing are critical to clarify the cause of impairment and are best performed by health practitioners familiar with HIV disease. A neurologic examination focuses on indicators that may signify central nervous system disease such as neurological functioning, including walking, coordination, and muscle tone. Laboratory tests include: routine blood tests, which indicate the general functioning of organ systems and immunity; more specialized tests, which indicate conditions that can cause cognitive impairment; brain scans; electroencephalograms (EEG), which identify seizure disorders; and lumbar puncture, which assesses acute brain infection.

If the diagnostic picture remains unclear, for example, if there are no biological indicators of cognitive impairment, it may be necessary for a neuropsychologist to undertake neuropsychological testing. Neuropsychological testing uses standardized measures, primarily paper-and-pencil tests, to assess suspected cognitive and emotional dysfunction that may not be evident through other procedures.

Conclusion

The complexity of diagnosing cognitive impairment may be daunting, but it is important to remember that diagnostic approaches are tools. No single provider need understand the intricacies of all of these tools, and psychiatrists, neurologists, and neuropsychologists can be useful resources in negotiating this process. Most significantly, accurate diagnosis is the gateway to the effective treatment of many of the conditions that cause cognitive impairment.

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Recent Reports

Mapping Brain Infection in Dementia Patients

Autopsies of 55 AIDS patients revealed that HIV infection of the brain was more limited than expected in patients with late-stage AIDS Dementia Complex (ADC) and absent in some cases of mild to moderate ADC. Researchers theorized that this evidence suggests that brain infection may be a late event in the course of HIV progression, and that brain infection alone cannot explain the clinical symptoms of ADC.

Researchers mapped HIV infection both pathologically and virologically in the brains of 54 men and one woman. The subjects ranged from 21 years old to 60 years old. Autopsy included an evaluation that mapped the frequency and regional distribution of the p24 antigen among sections of the brain.

Twenty-one brains contained detectable HIV p24 antigen. Instead of a homogeneous or random distribution, the antigen tended to concentrate in certain sections of each brain. Although different sections were infected in different brains, HIV infected the globus pallidus in almost two-thirds of the subjects and the corpus callosum (deep white matter) in more than half, suggesting that these two subcortical areas may be particularly susceptible to active infection.

Overall, p24 antigen was detected more often in late-stage ADC patients than in early-stage ADC patients. But the amount of detectable brain infection was surprisingly small, even nonexistent, in several cases of severe clinical ADC. Two of the six cases of stage 3 ADC (the second highest severity level) had no brain infection at all; researchers noted that in these cases, ADC diagnosis had been made based on impaired ambulation rather than cognitive dysfunction. But there was no explanation for one of the six cases of end-stage ADC in which there was cognitive impairment but no detectable brain infection. In milder stages of ADC, p24 antigen was also undetectable or disproportionately minor in relation to the symptoms of infection. It is notable, however, that there were no cases of productive brain infection without symptomatic ADC.

Neurological Impairment in Children

A small but comprehensive study found that children with severe symptomatic HIV disease also experience severe cognitive dysfunction and high levels of neurological abnormalities. Unlike adults, who may develop cognitive impairment as a result of opportunistic infections, children seem to develop neurologic deficits as a result of direct HIV infection of the brain.

The study comprised 44 boys and 31 girls with moderate to severe symptomatic HIV disease. Ages ranged from less than a year old to almost 14 years old. Evaluation included brain scans, psychological measurement of cognitive function, and T-helper cell and p24 antigen testing.

Significant correlations were found between high T-helper cell levels and severe brain atrophy and white matter abnormalities. Brain lesions were associated with both cognitive and social-emotional dysfunction. Since opportunistic infections and lymphoma of the CNS are uncommon in children, these findings suggest that both neurological abnormalities and the resulting dysfunction are symptoms of direct infection. Researchers concluded that the progression of HIV-infection continuously compromises CNS functions, placing seropositive children at increased risk for developmental impairment.

No Evidence for Continuum of Impairment

A national study found that an AIDS diagnosis predicts only a modest decline in fine motor skills, providing no evidence for a continuum of cognitive decline in people with AIDS.
Researchers studied 138 HIV-infected individuals, including 52 with T-helper cell counts of less than 200 but no AIDS-defining illness, 57 with AIDS-defining illnesses but no central nervous system involvement, and 29 with a clinical diagnosis of HIV-associated dementia. The mean age of diagnosis was approximately 40 years, and the mean number of years of education was 16. The groups received two neuropsychological evaluations before and after AIDS diagnosis, and the Center for Epidemiological Studies Depression (CES-D) scale at each visit.

Following an AIDS diagnosis, the only cognitive impairment that researchers observed was a slight decline in psychomotor speed. As expected, the group with dementia showed the most significant decline after an AIDS diagnosis, with a less pronounced decline in the AIDS group, and a barely perceptible decline in the immunosuppressed group. These results suggest that dementia may be a discrete diagnosis with a specific pathogenesis rather than the end to a continuum of cognitive decline.

Disability and Neuropsychologic Impairment

Among people with HIV disease, defective neuropsychological performance is a chronic condition that leads to increased incidence of work disability, according to a prospective study of 185 gay and bisexual men.

Researchers evaluated 44 asymptomatic seropositive men and 72 seronegative men for HIV-related symptoms, neuropsychological performance (NP), and work disability (defined as having worked less than half-time for two or more years). Over a period of 4.5 years, work disability was consistently predicted by cognitive deficit. The incidence of disability was 70 percent among eight seropositive men with “defective” NP scores, 30 percent among 36 seropositive men with normal scores, 25 percent for 12 seronegative men with “defective” NP scores, and 15 percent for 60 seronegative men with normal NP scores. About the same proportion of seronegative and asymptomatic seropositive subjects scored in the defective range of neuropsychologic performance. But the 17 percent of seronegatives who scored in the defective range did so sporadically, while the 18 percent of asymptomatic seropositive subjects were more likely to do so consistently over a period of several visits.

Chronic Drug Use and Cognitive Decline

The decline of cognitive function among HIV-infected drug users is due to the chronic use of toxic substances rather than HIV infection, according to a small Italian study. An evaluation of cognitive function found that slower reaction time and poor memory occurred more often among the drug users regardless of serostatus.

The sample consisted of 56 seropositive drug users, 19 seronegative drug users, and 27 seronegative non-drug users. Researchers employed 13 neuropsychological tests.

Seronegative non-drug users scored highest, with statistically significant better scores in visual-spatial orientation, attention capacity and psychomotor speed, story recall, and abstract thinking. However, there were no significant differences in test results between the two drug-using groups. In addition, active drug users scored significantly worse than ex-drug users, also regardless of serostatus, which may suggest that cognitive deficit is reversible when drug use is discontinued.

Next Month
Several studies in the past few years have heralded the growth of HIV disease in rural areas that had been free of large caseloads. It is not news that rural social service systems are not able to meet the increasing and the complex mental health needs of people with HIV disease and their families. The March 1996 issue of FOCUS looks at two approaches to delivering care to these underserved areas.

Michael Shernoff, LCSW, one of the pioneers in HIV-related mental health care, describes the psychosocial issues faced in rural areas and an innovative strategy to providing support to people with HIV disease who are moving back from adult lives in urban areas to their rural families of origin. Alexander Tartaglia, DMin, a therapist at a South Carolina pastoral counseling center, looks at the role of rural clergy and congregations in supporting the families of people with HIV disease.
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