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# HEMIPARESIS IN HIV INFECTION

## Rehabilitation Approach<sup>1</sup>

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**ABSTRACT** O'Dell MW, Sasson NL: Hemiparesis in HIV infection: rehabilitation approach. *Am J Phys Med Rehabil* 1992;71:291-296.

Persons with acquired immunodeficiency syndrome (AIDS) and human immunodeficiency virus (HIV) infection demonstrate a wide array of central nervous system impairments and may be at a significantly increased risk for cerebrovascular disease. Cerebrovascular disease can be the first manifestation of HIV infection and may be associated with a treatable etiology. Anticipating more referrals for HIV-related physical disability, we detail the rehabilitation management of three persons with HIV infection and hemiparesis. Onset of hemiparesis ranged from just before to 24 months after an AIDS-defining illness. No specific underlying etiology was identified in two of three patients, consistent with previous observations. Rehabilitation interventions included lower and upper extremity orthoses, assistive devices to aid gait and activities of daily living, therapeutic exercise and use of antispasticity medication. All patients made at least mild, temporary gains in functional status. Survival ranged from 3 to >6 months from initial contact with rehabilitation services. Neurologic and nonneurologic considerations in the rehabilitation of persons with HIV infection are discussed. We conclude that selected individuals with HIV infection and hemiparesis can benefit from rehabilitation intervention. HIV infection should be considered in any young adult presenting with stroke.

**KEY WORDS:** AIDS, HIV, Disability, Stroke, Cerebrovascular Disease, Rehabilitation

As of September 1991 over 175,000 cases of acquired immunodeficiency syndrome (AIDS) had been reported in the United States.<sup>1</sup> The number of persons in the United States infected with the human immunodeficiency virus (HIV), the causative agent of AIDS, is estimated to be between 750,000 and 2 million.<sup>2</sup> The proportion of these individuals who will go on to manifest AIDS is probably quite high.<sup>3</sup> With numbers of this magnitude, the care of persons with AIDS poses a tremendous challenge to the healthcare system.<sup>4</sup> There is recent evidence, however, suggesting a moderate increase in life ex-

pectancy after an AIDS-defining event.<sup>5</sup> As this survival time continues to increase with improved treatment, the number of persons living with AIDS and a physical disability will increase. This is especially true when considering the number of neurologic,<sup>6</sup> neuromuscular,<sup>6</sup> cardiac<sup>7</sup> and pulmonary<sup>8</sup> complications associated with symptomatic HIV infection. Assessment and management of HIV-related physical disability by rehabilitation professionals will play a significant role in the long-term management of HIV infection and will influence the future practice of physiatry.<sup>9,10</sup>

Despite numerous reports of HIV-related neurologic disease and impairment, the medical literature has provided little information regarding the management of residual disability. This report will add to a growing clinical literature on rehabilitative management in HIV infection. We present three cases of hemiparesis (from cerebral infarction or infection) and subsequent rehabilitation.

### CASE 1

A 31-yr-old homosexual male experienced a single, AIDS-defining episode of *Pneumocystis carinii* pneumonia (PCP) 2 yr before evaluation. There were no major intervening complications. Hospitalization was prompted by a gradually worsening left hemiparesis, dysarthria and diplopia over a 2-month period. He denied headaches or fevers in conjunction with the new symptoms. Activities of daily

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living (ADL) and ambulation were becoming increasingly difficult. He was taking no medication on a regular basis at the time of admission.

Physical examination showed an afebrile, thin black male oriented to person, place and time. His speech was markedly dysarthric, but intelligible. He exhibited a marked, left-sided, spastic hemiparesis. He was globally hyperreflexic, more so on the left. Sensation was normal to light touch. Cranial nerve examination showed a marked weakness of the left facial musculature and abducens nerve. The patient demonstrated episodes of spontaneous, inappropriate laughing and crying. He required moderate to maximal assistance in all ADLs transfers and ambulation.

Cerebrospinal fluid analysis was normal; however, brain magnetic resonance imaging showed multiple, bilateral areas of increased signal intensity in the white matter consistent with progressive multifocal leukoencephalopathy (PML) (Fig. 1). Increased signal was also noted in the left pons and middle cerebral peduncle. Blood chemistries were essentially unremarkable.

The patient was referred to inpatient rehabilitation for further management of his physical disabilities. A resting splint was fabricated for his left wrist to help decrease tone and preserve range of motion. A "stock" molded ankle-foot orthosis was obtained to assist in left limb clearance during gait. After initiation of baclofen (Lioresal) at 10 mg three times daily, family members noted transfers required less assistance. Physical therapy focused on right-side strengthening, tone reduction and increasing endurance. At time of discharge, there was enhanced verbal intelligibility, ambulation of 15 feet with a quad cane and assistance and minimal assistance in going from sit to stand. ADLs required moderate assistance secondary to spasticity and decreased coordination. Initial discharge was delayed because of unresolved conflicts with the family pertaining to patient's lifestyle and homosexuality. He was even-

tually discharged home with extensive home care services and died there several weeks later (Table 1).

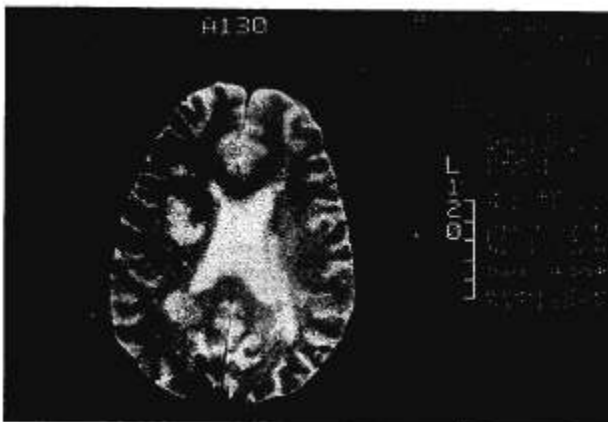
## CASE 2

A 37-yr-old right-handed homosexual male was referred to physical medicine and rehabilitation for evaluation of a possible peripheral neuropathy. He had been HIV seropositive for 2 yr and had experienced oral candidiasis, chronic diarrhea and moderate weight loss, but no opportunistic infections or cancers. His chief complaint was progressive right-sided "heaviness" over a 3-wk period. His handwriting skills and right upper extremity fine motor coordination had deteriorated considerably, as had his gait. In addition, he had noted slurring of speech and mild word-finding difficulties. Medications included zidovudine (AZT, 800 mg/day) and amitriptyline (100 mg at bedtime).

Physical examination showed a thin black male oriented to person, place and time and in no apparent distress. His affect was noted to be rather flat. He was afebrile and vital signs were stable. There was a mild, yet readily discernable, dysarthria. Strength was 5/5 on the left. On the right, both the upper and lower extremity demonstrated a moderate distal weakness. Reflexes were 2+ on the left and 3+ on the right except for the right ankle jerk reflex, which was 4+ with 5 to 6 beats of clonus. There were no Hoffman's or Babinski's signs. Decreased pain sensation was noted on the right side, but vibration and light touch were intact. A mild right facial paresis was also noted. Fine motor coordination and rapid alternating movements were decreased in the right upper extremity. The patient walked with an unsteady, circumducted gait with decreased right arm swing.

Suspecting a central nervous system process, immediate brain computed tomography was obtained and read as normal by neuroradiology. Cerebrospinal fluid and blood chemistries as well as cultures were unremarkable. Magnetic resonance imaging of the brain performed 2 wk later showed no evidence of infection, tumor or infarction.

Three weeks after the initial evaluation, the patient began an outpatient rehabilitation program including physical therapy and occupational and speech therapies. Physical therapy focused on lower extremity strengthening and gait retraining with a "stock" right molded ankle-foot orthosis and quad cane. Occupational therapy issued a variety of equipment to maximize independence in eating, bathing and handwriting. Work simplification and energy conservation techniques were stressed. Speech therapy proceeded with instruction in articulation techniques for greater intelligibility. After a few weeks of therapy, the patient ambulated independently and safely with a quad cane, required only set-up in dressing and feeding and demonstrated improved intelligibility in speech. Despite



**Figure 1.** Magnetic resonance image of the brain of Case 1. The image demonstrates multiple, bilateral areas of increased signal intensity consistent with progressive multifocal leukoencephalopathy.

**TABLE 1**  
*Summary of cases*

	Case 1	Case 2	Case 3
Age (yr)	31	37	27
HIV risk group <sup>a</sup>	H/B	H/B	H/B
Symptoms	L-sided weakness, garbled speech, double vision	R-sided heaviness, decreased handwriting	R-sided hemiplegia, dizziness, slurred speech
Onset	24 mo after AIDS diagnosis	24 mo after found HIV positive	1 mo after AIDS diagnosis
Signs	L spastic hemiparesis, dysarthria, CN VI palsy	R hemiparesis, hyperreflexia, hypesthesia	R flaccid hemiplegia, hyporeflexia, R facial weakness
Brain imaging	Bilateral increases signal intensity in white matter	Normal	L internal capsule and basal ganglion infarction
Etiology	PML	Unknown	Unknown
Interventions	Baclofen, splinting, assistive devices, PT, OT, ST	Ankle orthosis, assistive devices, PT, OT, ST	Adaptive equipment, PT, OT, ST

The table summarizes the demographic and medical information and rehabilitation interventions in three persons with hemiparesis and HIV infection.

<sup>a</sup> H/B, homosexual or bisexual male; R, right; L, left; CN, cranial nerve; PML, progressive multifocal leukoencephalopathy; PT, physical therapy; OT, occupational therapy; ST, speech therapy.

these gains, however, the patient's weakness and spasticity worsened and mental status deteriorated over the next 3 months. He died 4 months after evaluation of unknown causes. Post mortem was not performed (Table 1).

### CASE 3

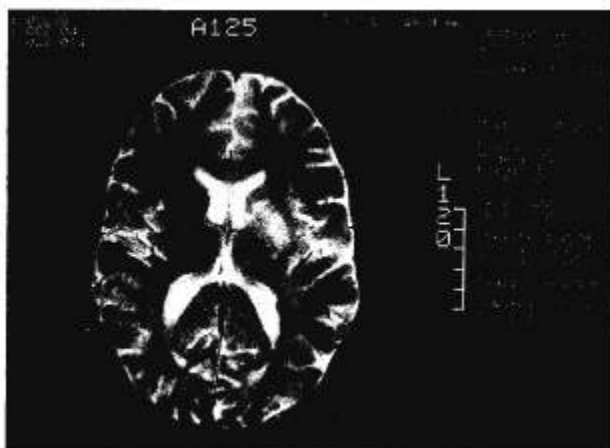
A 27-yr-old, right-handed homosexual male, known to be HIV seropositive for 3 yr before admission, was admitted to the acute care hospital with a 1-day history of progressive right-sided weakness, light-headedness and slurred speech. He had been hospitalized 3 wk previously with his first, AIDS-defining episode of PCP. He had experienced oral candidiasis and herpetic keratitis within the previous 12 months. He denied syncope, fever, nausea or vomiting, shortness of breath or headache. Medications included zidovudine (800 mg/day), acyclovir (1000 mg/day) and ketoconazole (400 mg/day).

Physical examination showed a young white male lying quietly in bed, slightly lethargic, but arousable. He was markedly dysarthric when attempting to speak. He exhibited a flaccid right hemiparesis, facial weakness and hyporeflexia. He also had a non-fluent, expressive aphasia in addition to the dysarthria.

Laboratory studies included a hemoglobin of 10.4

g/dl with normal white blood cell and platelet counts. Erythrocyte sedimentation rate was 67, but antinuclear antibodies, rheumatoid factor, rapid plasma reagin, Lyme titer and all urine studies, cerebrospinal fluid and blood cultures were negative. Chest radiograph was unremarkable. Brain computed tomography was read as normal but a subsequent brain magnetic resonance imaging was consistent with an infarction of the left internal capsule and basal ganglia with evidence of mass effect (Fig. 2). Echocardiogram and carotid doppler studies were negative, but cerebral angiogram showed an occlusive lesion in the middle cerebral artery without evidence of vasculitis.

He was transferred to another acute medical care facility where rehabilitation services were continued. Therapies focused on left-sided strengthening and active-assisted range of motion on the right. Change of hand dominance activities and a "rocker-knife" helped to facilitate independence in ADLs. Three months after discharge, there were continued mild deficits in naming and comprehension at the paragraph level, but expressive language was normal. He continued to have difficulties with gait at increased speed and demonstrated slightly increased tone on the right side, but strength returned to nearly normal. He was lost to follow-up several months after presentation (Table 1).



**Figure 2.** Magnetic resonance image of the brain of Case 3. Infarction of the left internal capsule and basal ganglia is shown.

## DISCUSSION

Although stroke is uncommon in persons under 40 yr of age,<sup>11</sup> the vast majority of persons with AIDS are, in fact, young adults.<sup>1</sup> Common nonhemorrhagic etiologies in the non-HIV infected young adult with stroke are cardiac disease,<sup>12,13</sup> ethanol intoxication<sup>14</sup> and migraine headaches.<sup>12-14</sup> A significant proportion have no etiology identified, even after an exhaustive search.<sup>12,13</sup> The prognosis of stroke in young adults is generally good to excellent.<sup>11,12,14</sup>

In general, central nervous system complications are extremely common in AIDS.<sup>6,15</sup> Neurologic signs are seen in up to 90% of hospitalized patients.<sup>16</sup> Up to 20% of persons with AIDS will present with neurologic symptoms as the initial manifestation of the disease.<sup>17</sup>

The incidence of cerebrovascular disease, in HIV-infected persons ranges from 8 to 34% in pathologic studies<sup>18-20</sup> and from <1% to nearly 7% in clinical studies.<sup>6,15,21</sup> As in the non-HIV-infected young adult stroke, many will not have an underlying etiology identified.<sup>21</sup> A thorough diagnostic search is warranted because some etiologies of HIV-associated stroke are relatively treatable. The minimal evaluation should include at least neuroimaging studies, echocardiography, complete blood and platelet counts, coagulation studies, lupus and anti-cardiolipin antibody assays and blood and cerebrospinal fluid cultures.<sup>19,22</sup>

The thrombotic,<sup>15,19</sup> embolic<sup>15,19,20</sup> and hemorrhagic<sup>6,15,19,23</sup> events have all been associated with HIV-cerebrovascular disease. In general, ischemic events are more frequent than hemorrhagic.<sup>18,19</sup> Embolic events are commonly associated with concomitant cardiac disease, both bacterial and nonbacterial.<sup>19</sup> Cerebral infarctions or necrosis can also occur from a multitude of central nervous system opportunistic infections including aspergillosis,<sup>24</sup> *Toxoplasma gondii*,<sup>25</sup> *Treponema pallidum*<sup>26</sup> and progres-

sive multifocal leukoencephalopathy caused by papovavirus.<sup>27</sup> Stroke in children with HIV infection has also been noted.<sup>28</sup>

Only recently has attention been directed to issues of physical disability and rehabilitation in persons with HIV infection.<sup>29-31</sup> The general approach to HIV disability will parallel that taken in cancer rehabilitation.<sup>9</sup> As in the cancer patient, overall prognosis, current medical condition and patient/family needs will determine the specific functional goals and interdisciplinary team planning. The heterogeneity and complexity of medical conditions and prognosis in the HIV-infected patient will necessitate a thorough medical evaluation by the referring physician before initiation of rehabilitation therapies. These cases demonstrate several important considerations in the rehabilitation approach to hemiparesis and other HIV-related physical disabilities.

The diagnostic role of the physiatrist in AIDS has been noted previously<sup>31</sup> and is demonstrated in Case 2. Originally referred for peripheral neuropathy, the psychiatric evaluation showed right-sided hemiparesis of a central nervous system origin and appropriate diagnostic workup was immediately undertaken. Considering the number of neurologic complications in HIV infection,<sup>6,15</sup> a thorough neurologic examination will be critical in the evaluation of any HIV-related disability.

Among these cases, onset of disability occurred from just before to 24 months after an AIDS-defining event. Hemiparesis occurred at both the initial (Case 2) and terminal (Case 1) phases of the disease. Survival ranged from 3 to, at least, 6 months. Engstrom et al.<sup>21</sup> also noted a wide range of onset for HIV-cerebrovascular disease. In his study, cerebral infarction or transient neurologic deficit was the presenting symptom in 9 of 23 cases. In the remainder, onset of impairment occurred 3 wk to 12 months after an AIDS-defining event (mean = 5.5 months). Follow-up was obtained in ~60% of patients with a mean survival of 4 to 5 months and a range of 1 day to 24 months. Mean survival was longest among those persons with cerebral toxoplasmosis (mean survival = 9 months).<sup>21</sup>

Other concurrent neurologic conditions must be considered during the rehabilitation process. When orthoses or bracing are a consideration (as in Cases 1 and 2), the presence of HIV or drug-induced peripheral neuropathy must be established. At least 35% of persons hospitalized with AIDS will have symptomatic peripheral neuropathy.<sup>32</sup> The sensory deficits in Case 2 were in a hemidistribution rather than a stocking and glove distribution and were mild. We choose to use stock orthoses in our patients rather than expend extra time and resources for customization. Certainly, a more favorable prognosis and available financial resources may influence this decision. AIDS dementia complex will also be an important consideration in any rehabilitation intervention. Significant cognitive impairment can

occur in up to 70% of patients before death and may be the only manifestation of HIV infection.<sup>33</sup> The typical picture is one of rapidly progressing deficits in motor control, cognition and behavior invariably leading to a vegetative state and death. Depending on the severity, AIDS dementia complex could render the patient unable to effectively participate in the rehabilitation process because of poor carry-over, initiation and impaired new-learning ability. The fluctuations in emotion and mental status seen in Case 1 may have been partly due to AIDS dementia complex, but such changes are also seen in PML.<sup>27</sup> Therapy was only mildly disrupted by cognitive and behavioral problems.

There were few concomitant medical conditions in these cases, but this will probably be the exception rather than the rule. Cardiac<sup>7</sup> and particularly pulmonary<sup>8</sup> complications are common in HIV infection. Case 3 had experienced recent PCP, but his pulmonary status did not significantly hamper performance in rehabilitation therapies. Depending on the disease stage, fatigue and general deconditioning may greatly limit therapeutic exercise as an intervention for more specific physical disabilities.<sup>9</sup> There were no specific medical treatments initiated for underlying pathology in our series. No etiology was identified in Cases 2 and 3. As mentioned above, this is not uncommon in either HIV or non-HIV related cerebrovascular disease in young adults. In Case 1, the hemiparesis was caused by PML, a white matter viral infection caused by papovavirus. PML is also seen in other immunodeficient states besides AIDS. Treatment with cytosine arabinoside has been generally disappointing and, in Krupp's series, survival ranged from 4 to 37 wk.<sup>27</sup>

Baclofen (Lioresal), 10 mg three times daily, decreased spasticity and resulted in a mild functional improvement in Case 1. Because PML affects primarily the white matter of the brain, the therapeutic effect may be similar to that seen in multiple sclerosis.<sup>34</sup> In other cases of nonspinal cord HIV spasticity, baclofen may be of limited benefit.

Case 3, and to a lesser degree Case 2, responded to an outpatient strengthening program. Undoubtedly, resolution of local cerebral edema contributed to the functional improvement in Case 3. Spence et al.<sup>35</sup> have recently reported good improvement in strength and arthropometric measurements in persons with AIDS (1 month after the first episode of PCP) in the outpatient setting. The neurodevelopmental techniques as described by Brunstrom and the Bobaths will play a role in the therapeutic approach.<sup>36</sup>

The social ramifications for persons with AIDS and HIV infection, especially gay men, have been well documented.<sup>37</sup> The issue of life-style was a major consideration in the discharge plans of Case 1. Every person with HIV infection will face the new onset of physical disability, a loss of independence and a great uncertainty about the future. Gay men will also have the added stresses of life-style

disclosure to family members and facing the distinct possibility of rejection and discrimination based on both HIV status and sexual preference. When caring for persons with HIV infection and a physical disability, the health care professionals must exercise great sensitivity to minimize the stigmata surrounding the patient.

## CONCLUSIONS

1. Selected persons with hemiparesis and HIV infection can benefit from rehabilitation intervention in both the inpatient and outpatient settings.
2. The overall philosophy should parallel that of cancer rehabilitation.
3. Cerebral vascular disease can be either the presenting or terminal event in AIDS.
4. A thorough neurologic and medical evaluation must occur to rule out relatively treatable causes of HIV-related cerebrovascular disease.
5. HIV infection should be considered in any young adult with stroke.

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