distribution: comparison to $C^{15}O_2$ inhalation steady-state method measured by positron emission tomography. *J Cereb Blood Flow Metab* 1988;8:S52–S60.
20. Yonekura Y, Nishizawa S, Mukai T, et al. SPECT with [^{99m}Tc]-d,l-hexamethylpropylene amine oxime (HMPAO) compared with regional cerebral blood flow measured by PET: effects of linearization. *J Cereb Blood Flow Metab* 1988;8:S82–S89.
21. Gemmell HG, Evans NTS, Besson JAO, et al. Regional cerebral blood flow imaging: a quantitative comparison of technetium-99m-HMPAO SPECT with $C^{15}O_2$ PET. *J Nucl Med* 1990;31:1595–1600.
22. Iida H, Kanno I, Miura S, et al. A determination of the regional brain/blood partition coefficient of water using dynamic positron emission tomography. *J Cereb Blood Flow Metab* 1989;9:874–885.
23. Lammertsma AA, Martin AJ, Friston KJ, Jones T. In vivo measurement of the volume of distribution of water in cerebral grey matter: effects on the calculation of regional cerebral blood flow. *J Cereb Blood Flow Metab* 1992;12:291–295.
24. Koeppe RA, Hutchins GD, Rothley JM, Hichwa RD. Examination of assumptions for local cerebral blood flow studies in PET. *J Nucl Med* 1987;28:1695–1703.
25. Ohta S, Meyer E, Fujita H, Reutens DC, Evans A, Gjedde A. Cerebral [^{15}O] water clearance in humans determined by PET: I. Theory and normal values. *J Cereb Blood Flow Metab* 1996;16:765–780.

Cerebral Perfusion Scanning in Treating AIDS Dementia: A Pilot Study

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Acquired immunodeficiency syndrome (AIDS) dementia complex (ADC) is a common effect of the AIDS virus. We studied the regional cerebral blood flow of patients with early ADC and its response to atevirdine mesylate. **Methods:** Ten men with early ADC, who had failed or were intolerant to zidovudine or didanosine therapy, were treated with atevirdine mesylate for 12 wk. Cerebral perfusion SPECT using ^{99m}Tc-HMPAO was performed at Week 0 and Week 12. SPECT images were analyzed qualitatively and semiquantitatively. **Results:** The cerebral perfusion abnormalities in early ADC were usually mild and characteristically involved the cortices and periventricular regions bilaterally and symmetrically. Four patients were able to complete the protocol. Three of these patients responded to atevirdine clinically, two of whom showed improvement in their Week 12 SPECT images. The other responder had an essentially unchanged image. The patient who did not respond to atevirdine showed a definite deterioration in cerebral perfusion. **Conclusion:** Cerebral perfusion SPECT is useful in detecting and assessing therapeutic responses in ADC. The preliminary results of atevirdine in treating ADC are promising and need further investigation.

Key Words: AIDS; dementia; technetium-99m-HMPAO; atevirdine
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Acquired immunodeficiency syndrome (AIDS) is caused by the human immunodeficiency virus Type 1 (HIV-1). The development of opportunistic infections, neoplasms and a progressive subcortical dementing illness, termed AIDS dementia complex (ADC) is a common effect (*1*). ADC covers three spheres of abnormality: cognitive, motor and behavioral. The cognitive features of reduced concentration, forgetfulness and slowing of intellectual processing are almost always associated with motor dysfunction ranging from slowing of fine finger movements and abnormal eye movements (inaccurate pursuit and saccades) to limb ataxia and leg weakness with, at times, bladder and bowel incontinence. Behavioral features are less common in the early phases of ADC but, as it advances, progressive apathy and social withdrawal become prominent. In the terminal stages of the disease, patients are globally demented with paraparesis and virtual mutism (*2*).

Approximately 20% of patients with AIDS have ADC to some degree, with the prevalence of ADC increasing with advancing immunodeficiency. The neuropathological features of ADC can be divided into three subsets: gliosis and white matter pallor, multinucleated-cell encephalitis and vacuolar change of spinal cord or brain. Many of these pathological abnormalities are found in the basal ganglia (*3*). Gliosis and pallor are characteristic of those patients with mild ADC, but can be found also in patients without dementia, presumably representing subclinical disease. Multinucleated-cell encephalitis is the hallmark of productive HIV-1 infection in the brain. Such changes are found characteristically in patients with moderate or severe ADC. Vacuolar change is a rare finding, the clinical correlate of which still has to be adequately defined. It is similar to the vacuolar change that may occur in the spinal cord and which has been termed "vacuolar myelopathy" (*4*). Neuronal loss is both rare and minor suggesting that ADC may be reversible.

There is reasonable evidence for the efficacy of the antiretroviral drug zidovudine (AZT) in ADC (*5–8*). However, not all patients tolerate AZT because of its myelotoxicity (*9*). Indeed, there is an association of greater toxicity with advancing stages of HIV-1 infection. In addition, some patients fail to respond to AZT or deteriorate after a period of improvement. There are conflicting data on the efficacy of didanosine (DDI) in ADC (*10,11*), but DDI also has been associated with toxicity (predominantly painful peripheral neuropathy and pancreatitis) (*12*). Consequently, patients who develop ADC while receiving AZT or in the context of intolerance to AZT have little in the way of therapy that can be offered to them.

Atevirdine mesylate, an arylpiperazine non-nucleoside reverse transcriptase inhibitor, has been shown to be effective against HIV-1 infection with studies showing equipotency to AZT (*13*). Moreover, the drug has significant central nervous system penetration (*14*) and has exhibited synergy with AZT as well as activity against AZT resistant viral strains in vitro. Furthermore, it has been shown to have lower toxicity than nucleoside analogs (*13*). As a consequence, atevirdine may be effective in the treatment of ADC.

When a variety of therapeutic choices exists for patients with ADC, a predictable and early test that also indicates the brain's response to therapy would be invaluable. Cerebral perfusion tomography (SPECT) has been shown to be frequently abnormal in patients with HIV infection even before detectable

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cerebral dysfunction is noted (*15–23*). Furthermore, abnormalities in regional blood flow have been shown to respond to antiviral therapy (*15,24*).

This preliminary study was performed to investigate the regional cerebral blood flow of patients with ADC and its response to atevirdine mesylate with clinical correlation.

MATERIALS AND METHODS

Ten HIV-1 infected homosexual men (age range 26–59 yr; mean age 41 yr) were recruited who met the following criteria: (a) ADC Stage 1 or 2, as defined by the Price-Brew Scale (*25*); (b) failure or intolerance to AZT or DDI; (c) combined neurological and neuropsychological impairment score > 4 (normal < 4) (*26*); (d) an estimated premorbid IQ of > 70 as determined by the Nelson Adult Reading Test (NART) score of the vocabulary subtest of the Wechsler Adult Intelligence Scale (WAIS); and (e) absence of confounding neurological illness by clinical assessment, hematological and biochemical screens, blood and urine analyses for illicit drugs, MRI brain scan and cerebrospinal fluid (CSF) analysis. After informed consent by the patient and his next of kin, each patient was enrolled in an open study of atevirdine 600 mg tds for 12 wk. Response to atevirdine was defined as an improvement to ADC Stage < 1 and an improvement in neuropsychological performance (patients who responded to the treatment were offered the option of continuing to receive the study drug). Blood count and biochemical profiles were checked every 2 wk. Neurological and neuropsychological assessments were performed every 4 wk. CSF analyses were scheduled at entry and at Weeks 4 and 12. Cerebral perfusion SPECT and MRI cerebral scans were scheduled at Weeks 0 and 12.

Subjects were given 600 MBq ^{99m}Tc-HMPAO through an intravenous cannula in a quiet, darkened room after an initial period of repose. SPECT scanning was performed approximately 45 min later using a single-head tomographic camera with a high-resolution collimator to collect 64 40-sec frames over 360° in a 64 × 64 matrix with a zoom of 1.5. SPECT reconstruction was performed by filtered backprojection using a Metz adaptive filter ($3 < q < 14$), processing transverse, sagittal and coronal slices (slice thickness 1 pixel). Attenuation correction was performed according to Chang's method ($m = 0.11\ cm^{-1}$).

The SPECT images were analyzed qualitatively and semiquantitatively. Qualitative analysis was performed by three experienced, blinded observers. The transverse, sagittal and coronal slices were normalized to the cerebellum, and perfusion was scored according to abnormalities of the cortex (frontal, parietal, temporal and occipital regions) and subcortical matter (basal ganglia and periventricular regions). Cerebral perfusion was scored as 0 (severe generalized hypoperfusion or large focal defects), 1 (moderate generalized hypoperfusion or medium focal defects), 2 (mild generalized hypoperfusion or small focal defects) or 3 (normal). Differences in opinion were settled by consensus.

The images were also analyzed semiquantitatively by selecting a transverse slice through the midcerebral cortex that best demonstrated the basal ganglia and thalamic structures. Regions of interest (ROIs) were drawn over the right anterior, middle and posterior cerebral cortex, right cerebellum and right basal ganglia regions. These ROIs were then flipped over, generating identical mirror-image ROIs for the left cerebral hemisphere. Cortex to cerebellum and basal ganglia to cerebellum ratios were then calculated by dividing the average counts per pixel of each ROI by the average counts per pixel found in the cerebellar hemisphere with the higher activity.

RESULTS

The SPECT results at entry are summarized in Table 1 (qualitative) and Table 2 (semiquantitative). On qualitative analysis, two patients were judged to have normal images (Patients 1 and 2). In the other eight patients (80%), cerebral hypoperfusion was generally bilateral and involved the cerebral cortices and periventricular regions diffusely (only three patients appeared to have normal perfusion in the periventricular white matter). The basal ganglia were less commonly involved, being spared in six of the eight patients with abnormal scans. The MRI cerebral images of all 10 patients before treatment were normal, thereby excluding atrophy as a potential cause for cerebral hypoperfusion.

TABLE 1
Qualitative Analysis of SPECT Images at Entry

Patient no.	Frontal		Parietal		Temporal		Occipital		Basal ganglia		Periventricular	
	R	L	R	L	R	L	R	L	R	L	R	L
1	3	3	3	3	3	3	3	3	3	3	3	3
2	3	3	3	3	3	3	3	3	3	3	3	3
3	2	2	1	2	2	2	2	2	3	3	2	2
4	2	2	2	2	2	2	2	2	1	1	2	2
5	2	2	2	2	2	2	2	2	3	3	3	3
6	1	1	2	2	2	2	2	2	3	3	2	2
7	2	2	2	2	2	2	2	2	1	2	2	2
8	1	1	1	1	1	1	1	1	3	3	2	2
9	2	2	1	1	2	2	1	1	3	3	1	1
10	2	2	2	2	2	2	2	2	3	3	2	2

R = right; L = left.
0 = severe hypoperfusion; 1 = moderate hypoperfusion; 2 = mild hypoperfusion; 3 = normal.

Five patients were able to complete the protocol, four of whom responded to atevirdine clinically. Unfortunately, one of the responders committed suicide before the Week 12 assessments (see Table 3 for impairment scores). Those patients who responded at Week 12 had shown clinical improvement by the combined impairment score at Week 4. No consistent changes were seen between the MRI brain scans at entry and at Week 12 (in both the responders and the patient who did not respond). In the patient who did not respond, both ADC stage and combined impairment score worsened.

Results of SPECT scans in four patients at Week 12 (the three surviving responders and the one nonresponder) are summarized in Table 4 (qualitative) and Table 5 (semiquantitative). Of the responders (Patients 4, 8 and 9), the SPECT images of

TABLE 2
Semiquantitative Analysis of SPECT Images at Entry

Patient no.	Cortex/cerebellum ratios						Basal ganglia/cerebellum ratio	
	Anterior		Middle		Posterior			
	R	L	R	L	R	L	R	L
1	0.98	0.96	0.95	0.94	1.04	1.00	0.90	1.05
2	0.93	0.74	0.91	0.89	0.96	0.87	0.89	1.05
3	0.83	0.76	0.86	0.96	0.89	0.92	0.78	0.95
4	0.91	0.92	0.90	0.90	1.06	1.01	1.07	0.92
5	0.99	0.91	1.01	0.92	0.99	1.00	1.03	1.02
6	0.83	0.71	0.88	0.92	0.81	0.91	1.00	1.00
7	0.93	0.94	0.92	0.93	0.95	0.95	0.91	0.89
8	0.80	0.83	0.80	0.82	0.87	0.75	0.91	0.95
9	0.86	0.86	0.97	0.94	0.95	0.88	1.02	1.07
10	1.01	0.93	0.99	0.88	0.99	0.95	0.99	1.13

R = right; L = left.

TABLE 3
Clinical Response to Atevirdine

Patient no.	ADC stage	Neurological impairment score	Neuropsychological impairment score
Patient 1*			
Week 0	1	2	3
Week 4	0.5	3	7
Patient 2*			
Week 0	2	9	13
Week 4	2	7	12
Week 8	2	8	–
Patient 3*			
Week 0	2	12	16
Patient 4			
Week 0	2	7	15
Week 4	0.5	7	10
Week 8	0.5	2	5
Week 12	0	1	5
Patient 5			
Week 0	1	3	6
Week 4	0.5	1	7
Week 8	0.5	3	11
Week 12	2	8	13
Patient 6†			
Week 0	2	9	14
Week 4	2	7	13
Patient 7‡			
Week 0	1	7	8
Week 4	1	8	6
Week 8	0.5	3	5
Week 12	0.5	4	–
Patient 8			
Week 0	1	7	12
Week 4	0.5	3	8
Week 8	0.5	3	2
Week 12	0.5	2	3
Patient 9			
Week 0	2	8	10
Week 4	0.5	3	3
Week 8	0.5	2	1
Week 12	0	1	3
Patient 10§			
Week 0	2	9	14
Week 4	2	9	14

*Died of sepsis.
†Died of progressive multifocal leukoencephalopathy.
‡Suicide.
§Withdrawn due to rash.

Patients 8 and 9 revealed a trend toward improvement in both the qualitative and quantitative analyses (Fig. 1). The SPECT image of Patient 4 at Week 12 was essentially unchanged compared with his scan at entry. In the patient who did not respond to atevirdine (Patient 5), his SPECT scan at Week 12 showed a definite deterioration in cerebral perfusion qualitatively (Fig. 2), with the corresponding semiquantitative analysis revealing decreased ratios in all regions analyzed. In the qualitative analysis for Patient 5, deterioration in cerebral perfusion was most marked in the subcortical matter.

There were several reasons for five of the 10 patients not completing the protocol. One patient deteriorated at Week 7 and, on further investigation, was found to have developed progressive multifocal leukoencephalopathy. Review of the MRI brain image at entry into the trial did not reveal any evidence of progressive multifocal leukoencephalopathy. One patient died of hypotension possibly on the basis of sepsis at Week 6. Two patients died of sepsis at Weeks 2 and 8 and one patient developed a generalized rash at Week 4. Four of these patients were able to be evaluated at Week 4 of the protocol and none of them had improved (Table 3).

TABLE 4
Qualitative SPECT Results at Week 12

Patient no.	Frontal		Parietal		Temporal		Occipital		Basal ganglia		Peri-ventricular	
	R	L	R	L	R	L	R	L	R	L	R	L
Patient 4												
Week 0	2	2	2	2	2	2	2	2	1	1	1	1
Week 12	2	2	2	2	2	2	2	2	1	1	1	1
Patient 5*												
Week 0	2	2	2	2	2	2	2	2	3	3	3	3
Week 12	1	1	1	1	1	1	1	1	1	1	1	1
Patient 8												
Week 0	1	1	1	1	1	1	1	1	3	3	2	2
Week 12	2	2	2	2	1	1	1	1	1	1	3	3
Patient 9												
Week 0	2	2	1	1	2	2	1	1	3	3	1	1
Week 12	2	2	2	2	2	2	3	3	3	3	2	2

*Nonresponder.
0 = severe hypoperfusion; 1 = moderate hypoperfusion; 2 = mild hypoperfusion; 3 = normal.

DISCUSSION

The results of our study show that the cerebral perfusion abnormalities in early ADC characteristically involve the cortices and periventricular regions bilaterally. The involvement is usually diffusely patchy and symmetrical. In addition, the abnormalities are usually mild (occasionally moderate). Of particular interest is that, despite the well-documented neuropathological changes that occur dominantly in the basal ganglia of patients with ADC, the basal ganglia were infrequently involved in our study. However, deterioration in perfusion was more marked in the basal ganglia than in the cerebral cortex in the one patient that did not respond to therapy and who was able to complete the protocol. The reasons are unclear but may indicate late but overwhelming involvement of the basal ganglia in the evolution of ADC.

A quantitative approach is theoretically more objective. There are, however, limitations to the method of quantitation.

TABLE 5
Semiquantitative SPECT Results at Week 12

Patient no.	Cortex/cerebellum ratios						Basal ganglia/cerebellum ratio	
	Anterior		Middle		Posterior			
	R	L	R	L	R	L	R	L
Patient 4								
Week 0	0.91	0.92	0.90	0.90	1.06	1.01	1.07	0.92
Week 12	0.94	0.94	0.99	0.89	1.06	0.97	0.99	1.02
Patient 5*								
Week 0	0.99	0.91	1.01	0.92	0.99	1.00	1.03	1.02
Week 12	0.77	0.73	0.80	0.76	0.87	0.80	0.90	0.97
Patient 8								
Week 0	0.80	0.83	0.80	0.82	0.87	0.75	0.91	0.95
Week 12	0.90	0.87	0.97	0.92	0.89	0.87	1.03	1.11
Patient 9								
Week 0	0.86	0.86	0.97	0.94	0.95	0.88	1.02	1.07
Week 12	0.95	1.03	1.05	1.00	1.06	1.97	1.13	1.21

*Nonresponder.

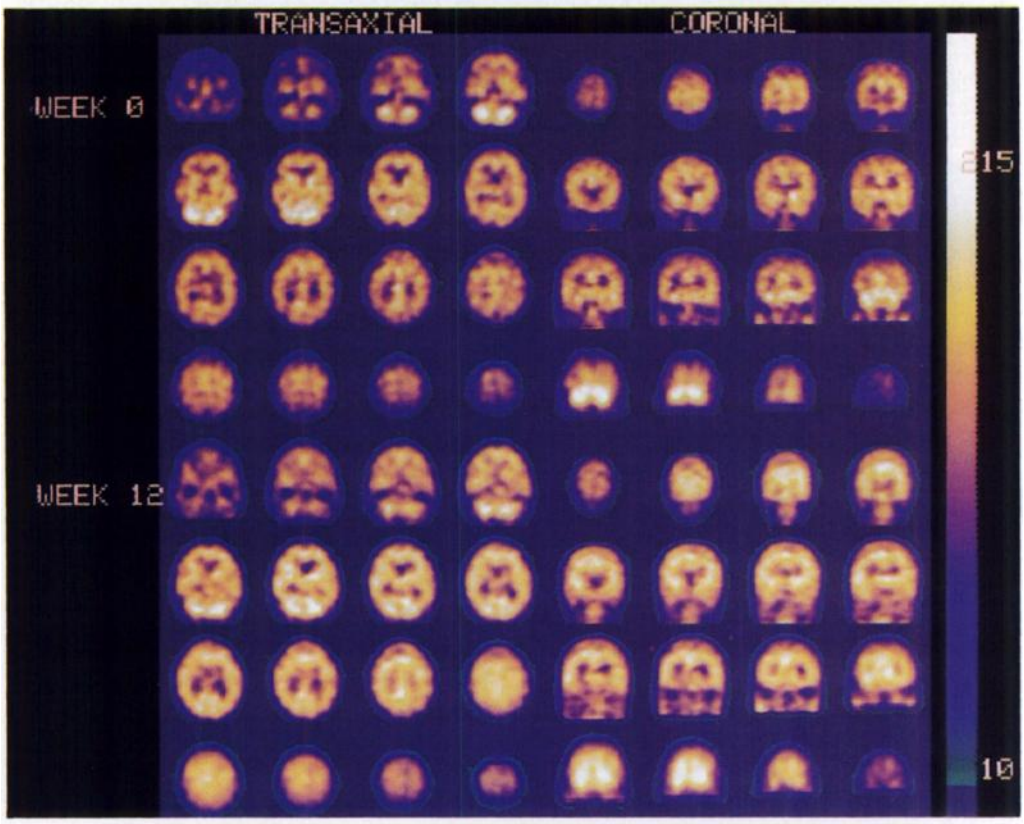

FIGURE 1. SPECT scans of Patient 8 (responder to therapy) at Weeks 0 and 12.

First, the type of quantitation necessary is not clear. Second, most investigators, including ourselves, compare regional uptake with cerebellar uptake. However, the effect of HIV involvement of the cerebellum is uncertain. We found that the trends observed in our semiquantitative data in the four patients able to be followed-up always reflected the direction of change seen in the corresponding qualitative data. Thus, although our quantitative analyses provided useful confirmatory results, they yielded no additional information over our qualitative analyses and we found the qualitative approach more useful practically.

The comparison of regional cerebral perfusion to cerebellar perfusion is suboptimal when cerebellar involvement is uncer-

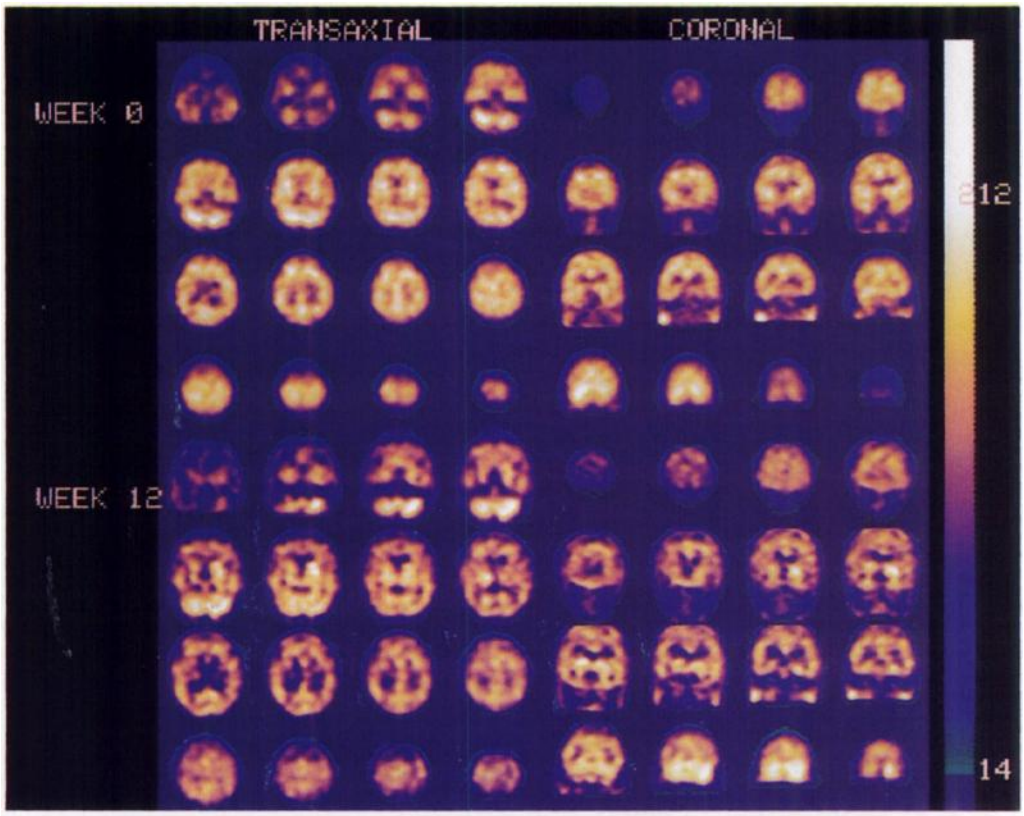

FIGURE 2. SPECT scans of Patient 5 (nonresponder to therapy) at Weeks 0 and 12.

tain and there are no normal controls, and this is a potential weakness of this study. However, the main objective of this study was to observe any change in regional cerebral blood flow of patients with ADC before and after therapeutic intervention with clinical correlation, and we believe our observations to be valid. This study demonstrates preliminary evidence of the efficacy of atevirdine in the treatment of ADC.

CONCLUSION

It is important to develop therapies for the treatment of ADC because, until now, only one drug, AZT, has shown efficacy in adults. The promising results of atevirdine in this study needs further investigation. This study adds to the existing data on the usefulness of cerebral SPECT in the detection and assessment of therapeutic responses in ADC.

REFERENCES

1. Navia BA, Jordan BD, Price RW. The AIDS dementia complex: 1. Clinical features. *Ann Neurol* 1986;19:517–524.
2. Brew BJ, Sidtis J, Petito CK, Price RW. The neurological complications of AIDS and human immunodeficiency virus infection. In: Plum F, ed. *Advances in contemporary neurology*. Philadelphia: FA Davis Co.; 1988:1–49.
3. Price RW, Brew BJ, Sidtis J, Rosenblum M, Scheck AC, Cleary P. The brain in AIDS: central nervous system HIV-1 infection and the AIDS dementia complex. *Science* 1988;239:586–592.
4. Patiti CK, Navia BA, Cho ES, et al. Vacuolar myelopathy pathologically resembling subacute combined degeneration in patients with the acquired immunodeficiency syndrome. *N Engl J Med* 1985;312:874–879.
5. Yarchoan R, Thomas RV, Grafman J, et al. Long-term administration of 3′-azido-2′,3′-dideoxythymidine to patients with AIDS related neurological disease. *Ann Neurol* 1988;23(suppl):582–587.
6. Price RW, Koch MA, Sidtis J, et al. Zidovudine (AZT) treatment of the AIDS dementia complex (ADC): results of a placebo-controlled multicentred therapeutic trial [Abstract]. Montreal, Canada: Fifth International Conference on AIDS Programme; 1989:331.
7. Schmitt FA, Bigley JW, McKinnis R, et al. Neuropsychological outcome of zidovudine (AZT) treatment of patients with AIDS and AIDS related complex. *N Engl J Med* 1988;319:1573–1578.
8. Sidtis J, Gatsonis C, Price RW, et al. Zidovudine treatment of the AIDS dementia complex: results of a placebo controlled trial. *Ann Neurol* 1993;33:343–349.
9. Richman DD, Fischl MA, Grieco M, et al. The toxicity of azidothymidine (AZT) in the treatment of patients with AIDS and AIDS related complex. *N Engl J Med* 1987;317:192–197.
10. Butler KM, Husson RN, Balis FM, et al. Dideoxyinosine in children with symptomatic human immunodeficiency virus infection. *N Engl J Med* 1991;324:137–144.
11. Portegies P, et al. AIDS dementia complex and peripheral neuropathy in zidovudine-intolerant HIV-infected patients treated with didanosine (DDI) [Abstract]. *Clin Neuropathol* 1993;12(suppl 1):S18.
12. Lambert JS, Seidlin M, Reichman RC, et al. 2′,3′-dideoxyinosine (ddI) in patients with the acquired immunodeficiency syndrome or AIDS-related complex. *N Engl J Med* 1990;322:1333–1340.
13. Romero DL, Busso M, Tan C-K, et al. Novel non-nucleoside reverse transcriptase inhibitors which potently and specifically block HIV-1 replication. *Proc Nat Acad Sci* 1991;88:8806–8810.
14. Maio SM, Voorman RL. Distribution of U87201E to brain following oral administration in rats (Protocol 91–004). Upjohn Technical Report 7256–91-049; 1991.
15. Masdeu JC, Yudd A, Van Heertum RL, et al. Single photon emission computed tomography in human immunodeficiency virus encephalopathy: a preliminary report. *J Nucl Med* 1991;32:1471–1475.
16. Rosci MA, Pigorini F, Bernabei A, et al. Methods for detecting early signs of AIDS dementia complex in asymptomatic HIV-1- infected subjects. *AIDS* 1992;6:1309–1316.
17. Schielke E, Tatsch K, Pfister HW, et al. Reduced cerebral blood flow in early stages of human immunodeficiency virus infection. *Arch Neurol* 1990;47:1342–1345.
18. Maini CL, Pigorini F, Pau FM, et al. Cortical cerebral blood flow in HIV-1-related dementia complex. *Nuc Med Commun* 1990;11:639–648.
19. Ajmani A, Habte-Gabr E, Zarr M, et al. Cerebral blood flow SPECT with Tc-99m exametazine. Correlates in AIDS dementia complex stages: a preliminary report. *Clin Nucl Med* 1991;16:656–659.
20. Ell PJ, Costa DC, Harrison M. Imaging cerebral damage in HIV infection. *Lancet* 1987;i:569–570.
21. Pohl P, Vogl G, Fill H, et al. Single photon emission computed tomography in AIDS dementia complex. *J Nucl Med* 1988;29:1382–1386.
22. Costa DG, Ell PJ, Burns A, et al. CBF tomograms with ^{99m}Tc-HMPAO in patients with dementia (Alzheimer type and HIV) and Parkinson's disease: initial result. *J Cereb Blood Flow Metab* 1988;8:5109–5115.
23. Kramer EL, Sanger JJ. Brain imaging in acquired immunodeficiency syndrome dementia complex. *Semin Nucl Med* 1990;20:353–363.
24. Brunetti A, Berg G, Di Chiro G, et al. Reversal of brain metabolic abnormalities following treatment of AIDS dementia complex with 3′-azido-2′,3′-dideoxythymidine (AZT, Zidovudine): a PET-FDG study. *J Nucl Med* 1989;30:581–590.
25. Price RW, Brew BJ. The AIDS dementia complex. *J Infect Dis* 1988;158:1079–1083.
26. Price RW, Sidtis JJ. Evaluation of the AIDS dementia complex in clinical trials [Abstract]. *J AIDS* 1990;3(suppl 2):S51–S60.

Parapharyngeal Meningioma Extending from the Intracranial Space Evaluated by FDG PET

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We report a rare case of parapharyngeal meningioma extending from the intracranial space evaluated by PET with [^{18}F]-2-fluorodeoxyglucose (FDG). Although the parapharyngeal meningioma had a high rate of glucose metabolism, it was proved to be pathohistologically benign. The high rate of glucose metabolism of the tumor reflected tumor aggressiveness well because the tumor grew in a relatively short time.

Key Words: parapharyngeal meningioma; PET; fluorine-18-fluorodeoxyglucose

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CASE REPORT

A 66-yr-old woman had a history of left-sided oculomotor paralysis that had been getting worse for 6 mo. CT and MRI showed a large parapharyngeal tumor. This tumor extended from the left parasellar region to the parapharyngeal space via the foramen ovale. After administration of gadopentetate dimeglumine, the tumor was moderately enhanced. The intracranial portion of the tumor invaded the cavernous sinus and surrounded the left internal carotid artery (Fig. 1A). A left carotid angiogram showed that the intracranial portion of the tumor was fed by small branches of the internal carotid artery. The extracranial portion of the tumor was fed by branches of the left external carotid artery.

We examined the glucose metabolism of the tumor using PET with ^{18}F-fluordeoxyglucose (FDG). PET was carried out with a Headtome V PET scanner (Shimadzu, Kyoto, Japan), which provides 47 tomographic slices and an in-plane spatial resolution of 4.0 mm FWHM and an axial spatial resolution of

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