FDG-PET in Differentiating Lymphoma from Nonmalignant Central Nervous System Lesions in Patients with AIDS

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Structural imaging studies such as CT or MRI are not able to accurately differentiate infectious from malignant cerebral lesions in patients with AIDS. We studied 11 individuals with AIDS and central nervous system (CNS) lesions with ^{18}F-fluoro-2-deoxyglucose (FDG) and positron emission tomography (PET). FDG-PET was able to accurately differentiate between a malignant (lymphoma) and nonmalignant etiology for the CNS lesions. Both qualitative visual inspection of the images as well as semiquantitative analysis using count ratios was performed and revealed similar results. FDG-PET may be useful in the management of AIDS patients with CNS lesions since high FDG uptake most likely represents a malignant process which should be biopsied for confirmation rather than treated presumptively as infectious.

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The acquired immunodeficiency syndrome (AIDS) continues to be a significant global public health concern (*1,2*) and estimates are that 750,000 to 1.5 million individuals in the United States are currently infected with the human immunodeficiency virus (HIV). Over the past decade, there has been increasing emphasis on the recognition and treatment of the neurologic complications of AIDS (*3,4*). The virus has been cultured from brain tissue (*5*) and studies have shown that the virus has a cytopathic effect on cerebral tissue (*6,7*). HIV is neurotrophic and is involved in the pathogenesis of several of the neurologic syndromes seen with HIV infection, including HIV encephalopathy and progressive dementia. The central nervous system (CNS) may also be involved with opportunistic infections or malignancies associated with progressive immunosuppression. As many as 10% of all AIDS patients will initially present with neurologic symptoms (*8–10*) and several reports indicate that 40%–60% of patients with AIDS will ultimately develop neurologic sequelae at some point in the course of their illness (*8,11–14*).

The opportunistic infection which most commonly involves the CNS in patients with AIDS is *Toxoplasma gondii* (*15–20*). This infection may produce a diffuse meningoencephalitis or cause focal lesions. Focal neurologic findings include hemiparesis, aphasia and ataxia (*17*), while lethargy and altered consciousness are less specific but a more common presentation (*17,19*). Imaging studies such as computed tomography (CT) or magnetic resonance imaging (MRI) are used to detect treatable complications of HIV infection such as toxoplasmosis or lymphoma and may often reveal focal or multifocal ring-enhancing lesions. However, without histopathologic confirmation, a specific diagnosis may be difficult (*17,21*). It is not possible to differentiate CNS lymphoma from toxoplasmosis in the HIV infected individual on the basis of CT or MRI findings due to the similarity in appearance of the lesions (*22–26*). Recently, in non-HIV infected patients, primary CNS lymphoma was demonstrated to have increased accumulation of ^{18}F-fluoro-2-deoxyglucose (FDG) on positron emission tomography (PET) imaging (*27,28*). The present study evaluates the potential role of FDG-PET in differentiating malignant from nonmalignant CNS lesions in patients with AIDS.

METHODS

Patients

A total of 11 HIV-positive individuals (10 males, 1 female) with a mean age of 39.2 yr (range = 25–65 yr) were studied (Table 1). Patients were selected because the majority were presumptively treated for toxoplasmosis and failed therapy or had known lymphoma elsewhere (Table 1). Two individuals were studied shortly after institution of anti-toxoplasmosis therapy and prior to resolution of lesions on CT scan. One individual with progressive multifocal leukoencephalopathy (PML) was

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TABLE 1
Patient Characteristics

Patient no.	Age	Sex	Selection criteria	Diagnosis	Method of diagnosis	Therapy at time of PET	Treatment results	FDG uptake	Count ratio
1	29	M	A	Toxo	Bx inflammation Necrosis, + toxo serology	Decadron 4 mg bid BW 566 Clindamycin	Resolved with treatment	2	0.3
2	35	M	A	Lymphoma	Bx lymphoma	Decadron 2 mg qid	Decadron, rad Tx lesions resolved	4	1.1
3	44	M	B	Toxo	+ toxo serology	Pryimethamine Clindamycin	Lesion resolved	2	0.9
4	38	M	C	Lymphoma	Bx lymphoma	Clindamycin Pyrimethamine	rad Tx resolving lesions	5 4 4	1.8 1.2 1.7
5	25	M	A	Syphillis (Gumma)	Bx reactive lymphocytosis + CSF VDRL	Penicillin	Resolving with penicillin Tx	2	0.7
6	43	M	A	Lymphoma	Bx lymphoma	Decadron 10 mg q 6° Pyrimethamine Sulfadiazene	Died prior to reassessment	5	1.9
7	38	F	A	Toxo	Bx nonspecific inflammation	BW 566	Resolving lesions	1	0.5
8	40	M	D	PML	– toxo serology		Died	3	1.3
9	32	M	B	Toxo	+ toxo serology	Pyrimethamine Sulfadiazene	Resolved	2	0.9
10	65	M	C	Lymphoma	Presumed metastatic lymphoma	Pyrimethamine Sulfadiazene	Died prior to reassessment	5 5	2.7 2.8
11	42	M	A	Lymphoma	Bx lymphoma	Decadron 4 mg qid	Resolving	5 5	1.9 2.8

BW 566 = experimental protocol therapy for toxoplasmosis; BX = biopsy; PML = progressive multifocal leukoencephalopathy; A = treatment failure for presumed toxoplasmosis; B = FDG-PET shortly after anti-toxoplasmosis therapy was instituted; rad tx = radiation therapy; toxo = toxoplasmosis; C = presumed metastatic lymphoma; D = presumed PML.

studied (Table 1). All subjects were studied with the approval of the Duke University Medical Center Investigational Review Board. Four individuals had biopsy proven lymphoma. Six individuals had infectious or inflammatory lesions, including four with toxoplasmosis, one with syphilis and one with progressive PML. The infectious or inflammatory lesions were confirmed by biopsy (three patients), serum titers (two patients) or response to therapy.

Structural Imaging

A CT or MR scan was used for initial assessment of intracranial pathology, correlation with the FDG-PET study and for follow-up evaluation in these particular individuals. The CT scans were typically obtained on a GE 9800 scanner (GE Medical Systems, Milwaukee, WI) and the MR scans on a GE Signa 1.5T Unit (GE Medical Systems, Milwaukee, WI). Intravenous contrast was used in the majority of CT scans (Isoview 200, Squibb, Princeton, NJ) and MRI studies (Magnevist, Berlex, Wayne, NJ).

PET Studies

All PET scans were obtained on an ECAT III tomograph (CTI Inc., Knoxville, TN) with a FWHM of 8.6 mm or GE 4096 tomograph (GE Medical Systems, Milwaukee, WI) with a FWHM of 6.8 mm. Approximately 10 mCi of ^{18}F-FDG was administered intravenously following a 4-hr fast. All subjects were kept quiet in a dimly lit room with eyes and ears unoccluded for 30 min prior to scanning. The subjects were positioned in the tomograph gantry with the imaging plane parallel to the cantho-meatal line. Four or five 10-min image sets (three image planes per set) were then acquired from the base of the skull to the vertex for those studies obtained on the ECAT III. Fifteen image planes were obtained from the patients studied with the GE 4096 tomograph. All images were corrected using calculated or geometric attenuation correction (*29*) and a Hann (0.5 cm^{-1}) filter.

Image Analysis

The lesions were graded qualitatively by consensus for FDG uptake by three reviewers blinded to clinical information using the following scale: 1, less than contralateral white matter; 2,

equal to contralateral white matter; 3, between contralateral white and gray matter; 4, equal to contralateral gray matter and 5, greater than contralateral gray matter. Semiquantitative analysis was also performed. A circular region of interest (ROI) was selected by consensus of three reviewers and was drawn on the FDG-PET image which most closely correlated with the structural imaging study where stereotatic biopsy was performed or the lesion was most notable. The circular ROI was placed on the lesion and contralaterally in the corresponding homologous brain region. A count ratio of lesion-to-contralateral homologous brain was then obtained. In the individuals with more than one lesion, each was visually and semiquantitatively evaluated.

Statistical Analysis

The qualitative gradings were correlated with lesion type (lymphoma, toxoplasmosis, PML, syphilis) using a Kruskal-Wallis analysis of variance by ranks. Analysis of variance was similarly used to determine significant differences using the count ratio. The data were then classified as malignant (lymphoma) or nonmalignant and a Student's two-tailed t-test or Mann-Whitney test was used to see if a significant difference existed between the nonmalignant and malignant lesions. Significance was defined as a p value less than or equal to 0.05.

RESULTS

A summary of patient data is provided in Table 1. Of the 11 subjects in this study, 6 have died. Seven subjects had biopsy confirmation of lesions. Four of the biopsies revealed lymphoma and three revealed nonspecific reactive lymphocytosis. Three of the six subjects with nonmalignant lesions had positive serology for toxoplasmosis. The individual with PML was diagnosed by his clinical course and the characteristic abnormalities on CT and MR scans. The single case of CNS syphilis had histologic diagnosis of nonspecific reactive lymphocytosis. This patient had a positive CSF-VDRL and was treated with high dose penicillin with resolution of the majority of his symptoms as well as lesion resolution on the CT scan.

The results of both the qualitative and semiquantitative analyses are shown in Table 2. In every case of lymphoma, FDG uptake was significantly higher both by qualitative visual inspection as well as semiquantitative analysis when compared to the nonmalignant lesions (Table 2).

TABLE 2
Qualitative and Semiquantitative Analyses

Lesion type	Qualitative* mean	Semiquantitative† mean ± 1 s.d.
Lymphoma (n = 5)	4.8(4,5,5,5,5)	1.8 ± 0.60
Toxoplasmosis (n = 4)	1.75(2,2,1,2)	0.65 ± 0.3
Syphilis (n = 1)	2	0.70
PML (n = 1)	3	1.3

*Mann-Whitney test revealed a significant difference in qualitative FDG uptake between malignant and nonmalignant lesions (p = 0.006).

†Students' two-tailed t-test revealed a significant difference between count ratios in malignant and nonmalignant lesions (p = 0.006).

Representative Cases

Patient 1. The patient was a 29-yr-old man with a history of HIV infection since 1986. In April 1991, he developed right body weakness and discoordination. A CT scan revealed a contrast-enhancing lesion in the left parietal region felt to be consistent with toxoplasmosis. He was treated with Pyrimethamine and Sulfadiazine with initial improvement but developed a rash and was then treated with Pyrimethamine and Clindamycin. The patient was eventually discharged; however, there was a progression of his neurologic symptoms. In June 1991, he underwent stereotactic brain biopsy. Histopathology showed reactive lymphocytosis suggestive but not diagnostic of toxoplasmosis. Dexamethasone was added to the patient's drug regimen to decrease intracerebral swelling. He again showed clinical improvement, but in July 1991 the patient experienced progressive neurologic deterioration. A CT scan (Fig. 1A) revealed a left temporal parietal enhancing lesion (arrow). Since the patient failed conventional anti-toxoplasmosis therapy, he was begun on a trial of protocol therapy with an investigational oral nitroimadozole (BW566) for toxoplasmosis. An FDG-PET study (Fig. 1B) was performed when the patient was receiving 4 mg of Dexamethasone twice daily; it revealed grade 2 FDG accumulation (arrow). There was also diminished FDG uptake throughout the left hemisphere consistent with edema and previous stroke. He responded well to the experimental BW566 compound and the CNS lesions showed greater than 50% reduction in size on follow up CT scan. The patient eventually developed an anaerobic pneumonia and died in October 1991.

Patient 5. The patient is a 25-yr-old man with a history of HIV infection since 1986. In August 1990, the patient developed a severe frontal headache. Approximately 1 wk later, he had a tonic-clonic generalized seizure and a CT scan revealed a right parietal occipital lesion. Evaluation revealed a positive VDRL (1:64), positive FTA-ABS, negative spinal fluid VDRL and negative serologies for CMV and toxoplasmosis. Lumbar puncture showed an elevated CSF protein of 71 and a white blood cell count of 14 cells/mm^3 (98% lymphocytes). All CSF cultures were negative. An open brain biopsy was performed with histology revealing polyclonal plasmacytic immunoblastic reaction. The patient was begun on empiric therapy for toxoplasmosis, which was initially Sulfadiazine and Pyrimethamine and then Pyrimethamine and Clindamycin. In September 1990, a CT scan showed no change in the contrast-enhancing parietal occipital lesion, and in October 1990 the patient developed a severe headache and experienced a second generalized tonic-clonic

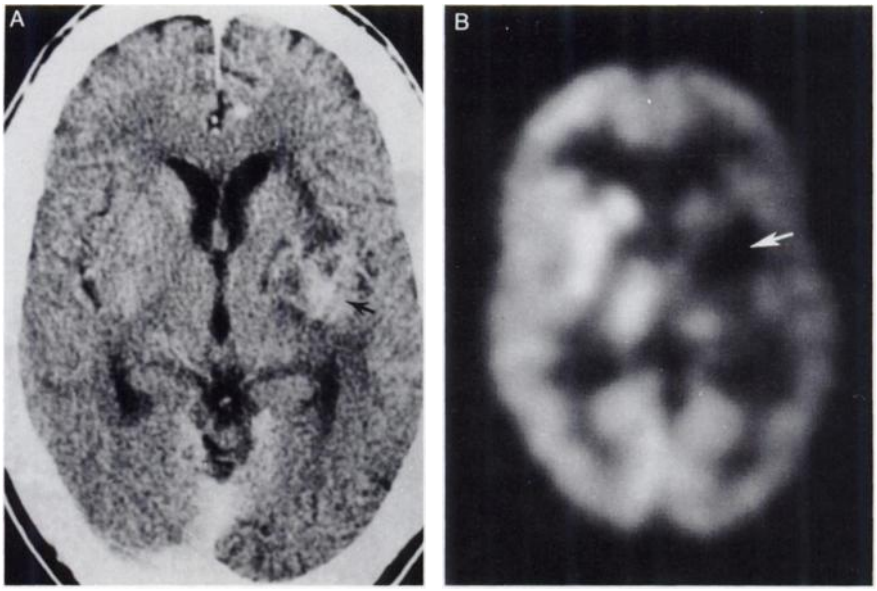

FIGURE 1. Patient 1 (Table 1). Enhanced CT scan (A) shows a left subcortical enhancing lesion (arrow). FDG-PET study (B) shows a hypometabolic abnormality in the left subcortical region (arrow). This patient had toxoplasmosis.

seizure. An enhanced CT scan (Fig. 2A) showed increased cerebral edema around the previously diagnosed mass lesion (arrow). He was continued on Dexamethasone, Clindamycin and Pyrimethamine. An MR scan was obtained revealing a large mass in the right parietal occipital region with edema and minimal mass effect. Repeat brain biopsy was performed under stereotactic localization. The biopsy revealed perivascular lymphocytic infiltrate with numerous plasma cells admixed with lymphocytes. Immunoperoxidase stain showed predominate T cells without a monoclonal population. Due to the patient's history of syphilis, he was started on a high dose of penicillin (20,000,000 units per day intravenously for 10 days) and anti-toxoplasmosis therapy was discontinued. The patient experienced dramatic clinical improvement. An FDG-PET study (Fig. 2B) was performed and revealed grade 2 FDG uptake (arrow). A MR scan done in December 1991 showed almost complete resolution of the right parietal occipital mass. The patient continues to do well 18 mo following diagnosis.

Patient 6. The patient was a 43-yr-old man with a history of HIV infection since March 1987. In May 1988, the patient developed a right hemiparesis and intermittent tonic-clonic seizure activity. A CT scan revealed a left frontal lobe mass. Neurosurgical consultation at that time recommended treatment for toxoplasmosis. The patient was started on Pyrimethamine and Sulfadiazine. A subsequent CT scan showed increasing lesion size with a midline shift (Fig. 3A). The patient's right hemiparesis progressed and seizure frequency increased. A stereotactic biopsy was performed in August 1988 and histopathology demonstrated a B-cell lymphoma. The patient underwent an FDG-PET scan (Fig. 3B) which demonstrated grade 5 FDG uptake (arrow). He then began whole-brain

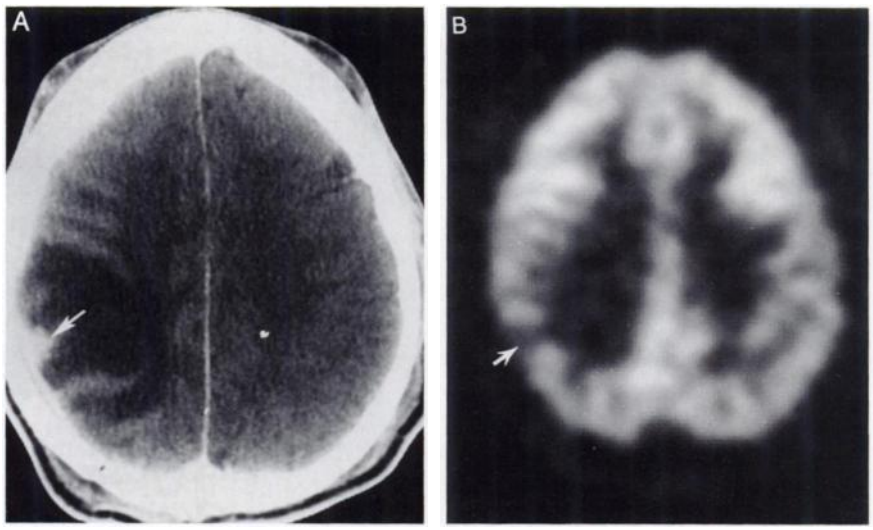

FIGURE 2. Patient 5 (Table 1). Enhanced CT scan (A) with cerebral edema around an enhancing mass lesion (arrow) in the right parietal region. FDG-PET study (B) shows a hypometabolic lesion with grade 2 FDG uptake (arrow) in the right parietal region. This lesion was a syphilitic gumma.

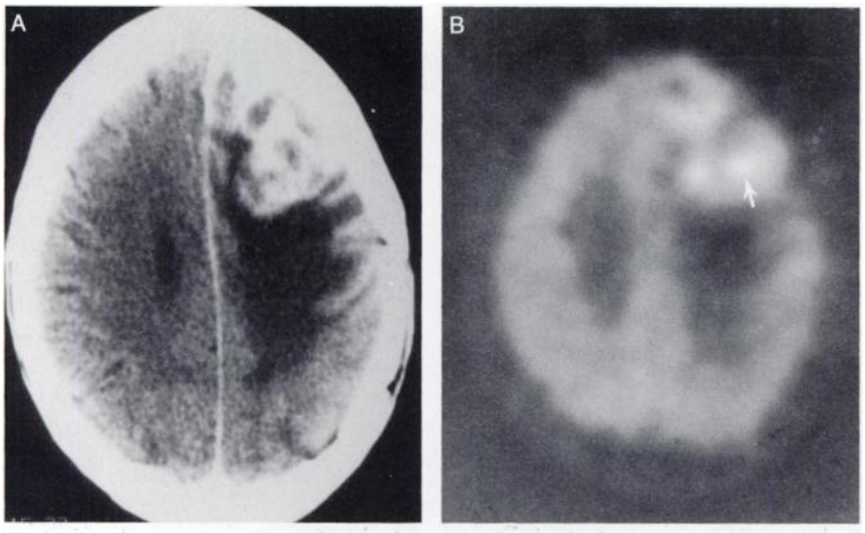

FIGURE 3. Patient 6 (Table 1). Contrast-enhanced CT scan (A) shows a large enhancing left frontal mass. FDG-PET scan (B) shows a hypermetabolic inhomogeneous lesion (arrow) localized to the left frontal lobe. This lesion was CNS lymphoma.

radiation. In September 1988, the patient developed right upper quadrant abdominal pain, nausea and fever and was treated with intravenous antibiotics. Blood cultures revealed enterococcus. He continued to deteriorate and died in October 1988.

Patient 8. The patient was a 40-yr-old man with a diagnosis of HIV infection and *Pneumocystis carinii* pneumonia since 1988. In 1989, he was treated for cryptococcal meningitis. In October 1989, he had an onset of right-sided numbness, weakness and headache. A contrast-enhanced CT scan at that time revealed left posterior white matter abnormalities felt to be consistent with PML (Fig. 4A). No biopsy was obtained. An FDG-PET study (Fig. 4B) was performed in November 1989 and demonstrated grade 3 FDG uptake in the left posterior white matter. The patient continued to deteriorate and by January 1990 he was bedridden and noncommunicative. He had recurrent episodes of aspiration pneumonia and died in May 1990.

Patient 11. The patient is a 37-yr-old man who initially tested positive for HIV in 1985. In 1990, the patient was diagnosed with Kaposi's sarcoma. The patient continued to do well until May 1992. At that time, he began experiencing ataxia and difficulty with handwriting. A contrast-enhanced CT scan was obtained that showed two enhancing lesions. These were felt to be consistent with CNS toxoplasmosis and the patient was started on Pyrimethamine and Clindamycin. His condition continued to deteriorate with increasing weakness, ataxia, nausea and vomiting. A repeat contrast-enhanced CT scan was obtained on May 19, 1992. Two distinct enhancing lesions were again noted but increased in size, one in the superior head of the right caudate (Fig. 5A) and in the left cerebellum (Fig. 5B). An FDG-PET scan (Fig. 5C) was performed revealing grade 5 uptake in both the striatal and cerebellar lesions. Stereotactic biopsy of the caudate abnormality was performed revealing large-cell malignant lymphoma. The patient was begun on radiation therapy.

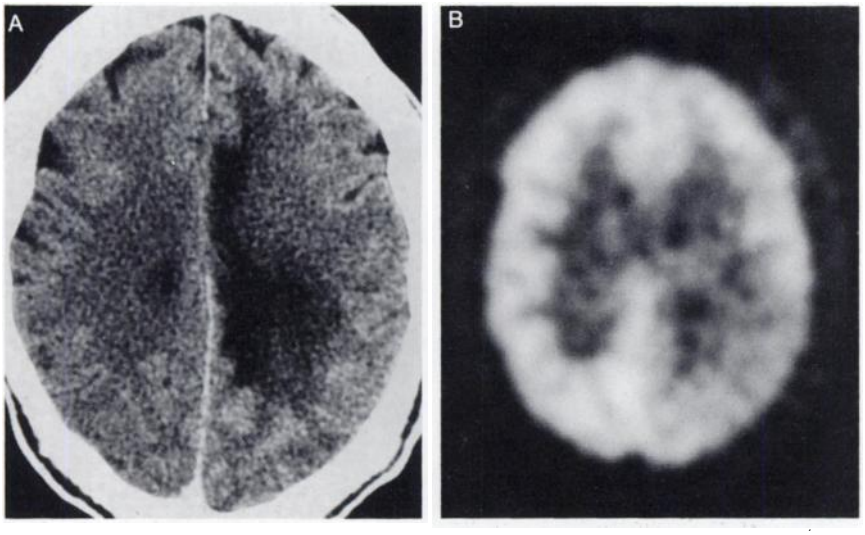

FIGURE 4. Patient 8 (Table 1). Contrast-enhanced CT scan (A) shows changes localized to the left frontal and parietal white matter. FDG-PET study (B) demonstrated grade 3 inhomogeneous FDG uptake in the left parietal white matter. This individual had presumed PML.

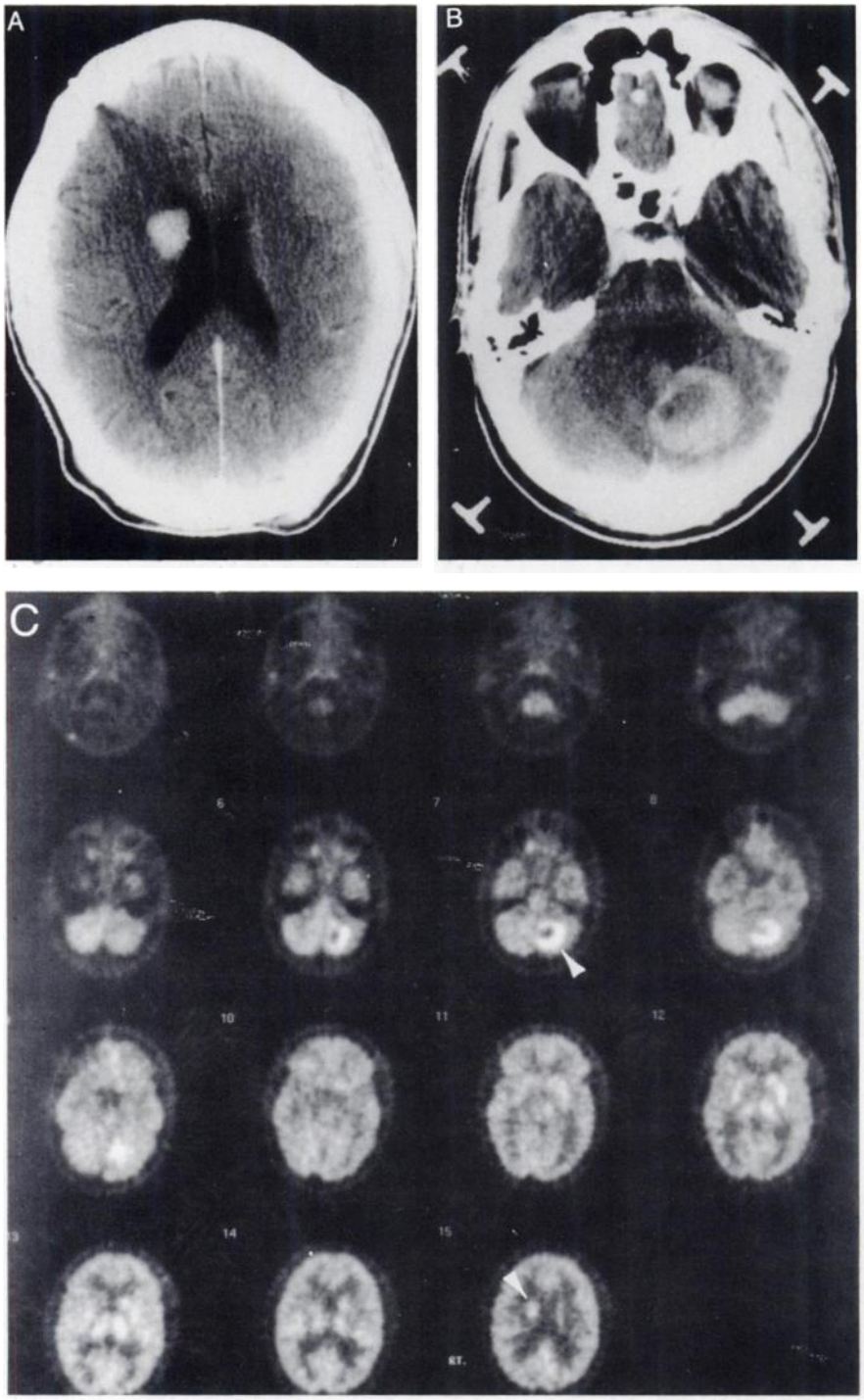

FIGURE 5. Patient 11 (Table 1). Contrast-enhanced CT at the level of the ventricles (A) shows an enhancing lesion in the superior head of the caudate region. On the same CT study, an enhancing ring lesion was noted in the left cerebellum (B). FDG-PET study (C) showed hypermetabolic lesions in the left cerebellum (arrow) as well as in the head of the right caudate (arrow). This patient had biopsy proven lymphoma.

DISCUSSION

During the past decade, a tremendous amount of information has been learned about the neurologic complications of HIV infection. Approximately 40% of individuals with HIV infection will have associated neurologic manifestations (*4,11–13*). Central nervous system complications of AIDS include encephalopathy, encephalitis, PML, opportunistic infection, CNS lymphoma and various cranial or peripheral nerve abnormalities. Neurologic manifestations may be the initial complaint in asymptomatic individuals with HIV infection (*9,10*). At autopsy, 90% of individuals with HIV infection have documented pathologic alterations in the CNS (*12*). The most common neurologic manifestation of AIDS is that of dementia or HIV encephalopathy (*30,31*). Patients with HIV encephalopathy or AIDS associated dementia have been studied with PET, SPECT, MRI and CT (*24–26,32–35*).

The most common CNS mass lesion seen in patients

with HIV infection is toxoplasmosis. Focal neurologic deficits with an associated altered level of consciousness and seizures are the typical presenting manifestations of CNS toxoplasmosis (*17*). Contrast-enhanced CT scans in HIV infected patients with toxoplasmosis reveal single or multiple ring-enhancing lesions (*12,18,23*). Nonenhancement and more homogenous patterns of contrast enhancement have also been noted (*4*). More recent studies have described the increased sensitivity of MRI in the detection of intracranial mass lesions in individuals with HIV infection (*20,22,32*). Various techniques have also been described for increasing the ability to visualize lesions using certain CT contrast enhancement schemes (*36*). However, as more individuals were studied, it became apparent that CT or MRI could not differentiate primary CNS lymphoma from toxoplasmosis.

Primary CNS lymphoma is a rare tumor (*37*) but occurs in approximately 2%–6% of patients with AIDS (*38,39*). In the nonimmuno-compromised host, primary CNS lymphoma typically shows homogeneously enhancing, well circumscribed, hyperdense lesions on contrast-enhanced CT (*40–43*). CNS lymphoma in the immuno-compromised individual typically presents as multiple ring-enhancing abnormalities on CT or MRI (*25,38,44*). Therefore, the appearance of CNS lymphoma is the same as toxoplasmosis. The two diseases, however, differ in treatment and prognosis. Differentiating between these two syndromes is imperative for appropriate therapy, particularly in the critically ill patient. A delay in diagnosing CNS lymphoma may have significant effects on morbidity and mortality. Due to the less frequent occurrence of CNS lymphoma, patients with the characteristic ring-enhancing lesions are typically treated empirically with anti-toxoplasmosis therapy. If there is no improvement in clinical or structural imaging studies, a diagnosis other than toxoplasmosis is contemplated. It has been recommended by some investigators (*3,12,21*) that brain biopsy be performed in patients with AIDS and CNS mass lesions since the reliability of diagnosis is problematic.

This particular study is an extension of previous work describing the utility of FDG-PET in assessing primary CNS lymphoma (*27,28*). We previously noted that CNS lymphoma is an extremely metabolically active tumor with markedly elevated FDG uptake even when the patient is receiving steroid therapy (*27*). Therefore, the present study is an extension of our previous study: the use of FDG-PET to differentiate malignant (typically CNS lymphoma) from nonmalignant (typically toxoplasmosis) in individuals with AIDS.

Our results indicate the potential utility of an FDG-PET scan in the evaluation of individuals with HIV infection and enhancing lesions on CT or MRI. The FDG-PET scan was able to accurately differentiate a nonmalignant from malignant etiology in the CNS lesions in all 11 individuals. In several instances, the FDG-PET scan was consistent with the diagnosis of lymphoma early in the course of illness. Since this was a research protocol, the patients were initially treated in the usual manner, typically with multi-drug toxoplasmosis therapy. One patient (#2, Table 1) with an FDG-PET scan consistent with lymphoma eventually underwent brain biopsy because of treatment failure for toxoplasmosis, and the biopsy confirmed the diagnosis of large-cell immunoblastic lymphoma. At our institution, all individuals with intracranial pathology suggestive of toxoplasmosis are followed clinically and with structural imaging studies and do not receive an FDG-PET scan. The cost-effectiveness as well as availability of FDG-PET for the widespread differentiation of these particular disorders is difficult to assess. Therefore, it is our feeling that an FDG-PET scan is helpful in the management of the AIDS patient with intracranial lesions if a poor response to anti-toxoplasmosis therapy is noted or in those individuals with negative toxoplasmosis serologies, a single CNS lesion or where disease elsewhere (i.e., lymphoma, nocardia or syphilis) suggests a diagnosis other than toxoplasmosis.

Several possible confounding issues could alter our results. All individuals with both malignant and nonmalignant CNS lesions were on some type of therapy at the time of the FDG-PET study. In those individuals with possible infectious processes, this included anti-toxoplasmosis therapy as well as other antibiotic regimens. We have not studied any individuals with an infectious cause of intracranial pathology who were not receiving therapy. An acute infectious process other than toxoplasmosis that causes a ring-like abnormality on FDG-PET is possible. Sasaki and colleagues have recently reported a patient with a bacterial abscess in whom ring enhancement and increased FDG uptake was noted (*45*). The FDG-PET pattern of untreated CNS toxoplasmosis is unknown. It is quite difficult, however, to study such patients prior to institution of therapy given the clinical need for rapid institution of therapy. As a result, all patients studied with FDG-PET at our institution have received at least some type of initial therapy prior to evaluation.

Obtaining the FDG-PET study after implementing therapy may have affected the results of our study. Both clinical and structural imaging techniques have demonstrated that anti-toxoplasmosis therapy has rapid effects (*12,23,24*). Therefore, one may wish to perform the FDG-PET scan approximately 1–2 wk after instituting anti-toxoplasmosis therapy if a poor response to therapy is noted. In those individuals with CNS lymphoma, the anti-toxoplasmosis therapy will be ineffective and the lesions will remain metabolically active.

Another potential limitation of this study is the absence of lesion histologic documentation by biopsy in all individuals. However, we used accepted methods for making a diagnosis, including effectiveness of therapy and positive serology. Thus, the absence of biopsy proof of diag-

nosis is not a major limitation with infectious or inflammatory lesions. One patient had very metabolically active lesions, which were presumed to be metastatic lymphoma. Although these lesions were not biopsied, the patient had biopsy proven lymphoma elsewhere and the clinical course and lack of response to anti-toxoplasmosis therapy make lymphoma the most likely diagnosis. In another individual (Patient 8, Table 1), PML was diagnosed from classic CT findings and a consistent progressive clinical course (*46–49*). This patient's lesion had the highest FDG uptake of all the nonmalignant lesions. The results of FDG-PET studies in assessing lesions such as PML have not been reported.

In summary, FDG-PET may be a useful adjunct in the management of AIDS patients with intracranial mass lesions. As more persons with HIV infection become clinically symptomatic, early and accurate diagnosis of CNS complication becomes essential. Therapies are improving to treat the complications of HIV infection and an FDG-PET scan may be helpful in differentiating an infectious from malignant etiology. Our preliminary results indicate that FDG-PET is able to make this important differentiation. Many factors concerning the role of FDG-PET are still unknown such as FDG uptake in the acute nontreated phase of toxoplasmosis. However, if further data support our results, a rational and sound basis for the incorporation of an FDG-PET scan in the evaluation of these complicated patients is possible.

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(continued from page 5A)

FIRST IMPRESSIONS

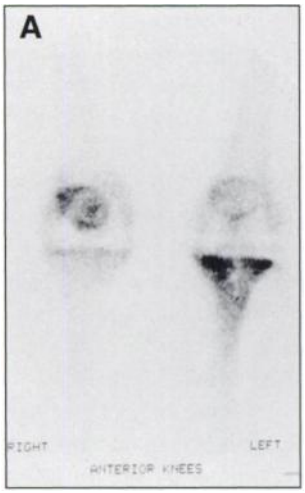

FIGURE 1A. Bone scan.

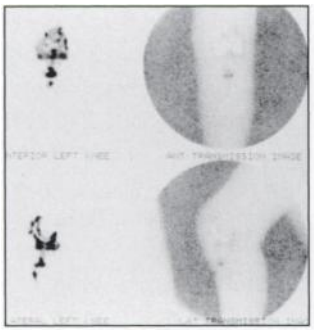

FIGURE 2. Arthrogram.

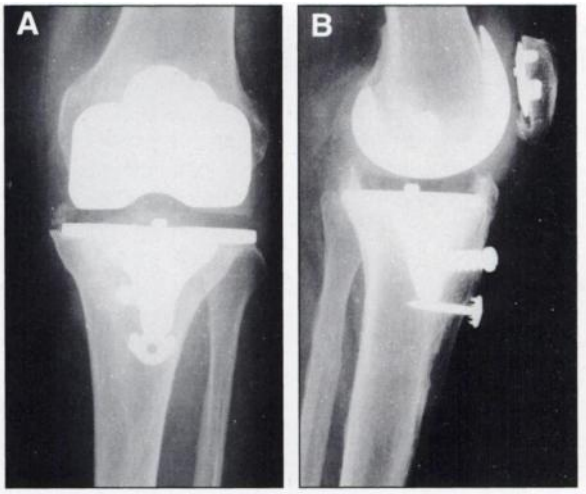

FIGURE 3A-B. Arthrogram shows no abnormality in the femoral component.

FIGURE 1B. Bone scan.

PURPOSE

A 72-yr-old woman was referred for investigation of left knee pain of sudden onset on weightbearing 1 mo previously. She had a past history of bilateral total knee joint replacements (TKR) 2 yr previously, with revision of the tibial component on the left 11 mo ago. Loosening of the left TKR was suspected and bone, gallium and radionuclide arthrograms, using a transmission image technique for orientation, were obtained. The bone scan (Fig. 1A-B) was inconclusive due to postsurgical changes, and the gallium scan was negative (not shown). The arthrogram (Fig. 2) was positive for loosening and demonstrated tracking of tracer around the stem of the tibial component of the left TKR. Tracer was also visualized anterior to the shaft of the upper left tibia at the site of a previously undiagnosed fracture. No abnormality was seen in relation to the femoral component (Fig. 3A-B).

TRACER

^{99m}Tc-MDP (20 mCi) and ^{99m}Tc-colloid (0.5 mCi).

ROUTE OF ADMINISTRATION

Intra-articular.

TIME AFTER INJECTION

3 hours ^{99m}Tc-MDP and 6 hours ^{99m}Tc-colloid. There was a 3-week delay between ^{67}Ga and ^{99m}Tc-colloid studies.

INSTRUMENTATION

LFOV gamma camera with a high-resolution collimator and ^{57}Co (10 mCi) flood for the transmission image.

CONTRIBUTORS

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