Central nervous system mass lesions in the acquired immunodeficiency syndrome (AIDS)

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The authors present the cases of nine patients with acquired immunodeficiency syndrome (AIDS) and intracerebral mass lesions, who were evaluated at the University of California, San Francisco, between April, 1979, and July, 1983. Eight patients were confirmed homosexual males, and none was Haitian. Their average age was 38.8 years. Tissue diagnosis was made in all patients from brain biopsy or autopsy material. Three patients initially presented for evaluation of their neurological deficits, while the other six already carried the diagnosis of AIDS at admission. Seven patients presented with multiple intracranial lesions and two had polymicrobial infection. In this series, three patients had *Toxoplasma gondii* brain abscesses, two had primary lymphoma, two had metastatic Kaposi's sarcoma of the central nervous system (CNS), two had focal cytomegalovirus encephalitis and one each had cryptococcal and *Candida albicans* brain abscesses. The clinical presentation, radiological evaluation, and serodiagnostic study of these patients were not helpful in establishing the nature of the CNS lesions. Brain biopsy is considered by the authors to be critical for the evaluation and appropriate treatment of mass lesions in patients with AIDS.

KEY WORDS • acquired immunodeficiency syndrome • abscess • Kaposi's sarcoma • toxoplasmosis • lymphoma • brain tumor • encephalitis

Several investigators have recently identified a clinical syndrome affecting primarily homosexual males and Haitians. With no known cause of diminished resistance, these patients present with diseases suggestive of a defect in cell-mediated immunity, including Kaposi's sarcoma, *Pneumocystis carinii* pneumonia, and other serious opportunistic infections. The syndrome has been designated the "acquired immunodeficiency syndrome" (AIDS) because of the presumed etiological immune deficiency. Patient groups at high risk of contracting AIDS include homosexual and bisexual males, intravenous drug abusers, Haitians, the hemophiliac population, and rare patients receiving multiple blood transfusions. Central nervous system (CNS) involvement has been demonstrated in approximately 10% of all reported AIDS patients. The majority of patients with CNS disease present with meningoencephalitis, although many patients have presented with intracerebral mass lesions. Twenty-one such cases of space-occupying lesions have appeared in the literature, including 16 patients with *Toxoplasma gondii* brain abscesses, three with primary CNS lymphoma, one with presumed metastatic Kaposi's sarcoma, and one with a cryptococcoma.

We report a series of nine homosexual AIDS patients with CNS mass lesions. These patients were evaluated at the University of California, San Francisco, between April, 1979, and July, 1983. These cases demonstrate the large variety of pathological processes involving the CNS in AIDS patients, and document the necessity for operative biopsies in all such cases.

Illustrative Case Reports

Case 1

This 30-year-old white homosexual man with AIDS presented with alterations in mental status in December, 1982. He had prior episodes of pancytopenia, *Pneumocystis carinii* pneumonia, and cytomegalovirus (CMV) pneumonitis. The patient's medical history was significant for documented episodes of rectal herpes, anal condylomata, syphilis, and hepatitis A.

Examination. Work-up upon admission included routine skin tests, which demonstrated complete anergy. *Toxoplasma* titers obtained by the Sabin-Feldman dye test were 1:32, while the immunoglobulin (Ig) M enzyme-linked immunosorbent assay (IgM-Elisa) toxoplasma test was nondiagnostic. *Toxoplasma* IgM
indirect fluorescent antibody (IgM-IFA) titers were less than 2 and Toxoplasma IgG-IFA titers were 1:256 (both nondiagnostic). The patient rapidly developed a left hemiparesis and marked aphasia. Shortly after a lumbar puncture was performed the patient became comatose. A computerized tomography (CT) brain scan was obtained on an emergency basis. This revealed a large contrast-enhancing lesion in the right internal capsule, as well as multiple bilateral contrast-enhancing lesions (Fig. 1A and B). There was a marked midline shift and a suggestion of uncal herniation.

Operation. The patient was transferred to the neurosurgical service and immediately taken to the operating room. Under ultrasound guidance, a large granulomatous lesion was removed from the region of the right internal capsule. Peroxidase-labeled antibody stains were positive for toxoplasmosis.

Postoperative Course. The patient was treated with pyrimethamine and sulfadiazine, and serial CT scans showed a progressive decrease in the size of all lesions as well as a decrease in their ring enhancement. This resolution continued following the withdrawal of steroids. The CT scans performed 1 month after surgery demonstrated complete resolution of the lesions (Fig. 1C and D). Postoperatively, the patient rapidly regained consciousness and within 1 month had only a minimal left hemiparesis.

During his prolonged hospitalization, the patient developed recurrent Pneumocystis carinii pneumonia which was unresponsive to antibiotic therapy. The patient died approximately 3 months after craniotomy. Postmortem examination of the brain was not permitted by the patient’s family.

Case 3

This 30-year-old white homosexual man presented with a generalized seizure, headaches, and a left-sided apraxia in August, 1982. His medical history was significant for multiple episodes of hepatitis, syphilis, shigellosis, and giardiasis.

Examination and Course. Tests for venereal disease were reactive on admission. The patient was immediately treated with phenytoin. A CT scan revealed a large ring-enhancing lesion with a surrounding low-density area and mass effect in the right occipital region, and a smaller ring-enhancing lesion in the left parietal region. Combined fluorescent antibody titers for Toxoplasma (sensitive to both IgG and IgM) were nondiagnostic at 1:128. The patient began a course of empiric antibiotic therapy, and biopsy of the right-sided lesion was performed via burr hole in September, 1982. All cultures were negative and histological evaluation was consistent only with chronic infection.

Shortly thereafter, the patient exhibited bilateral infiltrates on chest x-ray films, but bronchoscopy was nondiagnostic. He was treated with trimethoprim/sulfadiazine (TMS) for a presumed Pneumocystis carinii pneumonia. Additional empiric antibiotic therapy included erythromycin, ketoconazole, and metronidazole. A CT scan performed 1 month later demonstrated the presence of multiple new cerebellar lesions (Fig. 2 right). Subsequent monthly CT scans revealed a decrease in both the size and the enhancement of all lesions.

The patient developed multiple skin lesions during December, 1982, and biopsies of both the skin lesions and bone marrow were diagnostic of Kaposi’s sarcoma. Despite therapy, the patient’s condition slowly worsened. He became comatose and died in March, 1983.

Postmortem Examination. Postmortem evaluation revealed Kaposi’s sarcoma at multiple sites in the skin and lungs, as well as both Legionnaire’s disease and CMV in the lungs. Examination of the brain revealed multiple necrotizing foci in the cerebral cortex and brain stem, with considerable edema. Tissue cultures and histopathological sections revealed toxoplasmosis.
Central nervous system mass lesions in AIDS

**Case 3**

This 37-year-old white homosexual man presented with complaints of headache and lethargy in November, 1982. He had previously been treated for *Pneumocystis carinii* pneumonia, and had been diagnosed as having AIDS.

**Examination and Course.** Cultures from all secretions were positive for CMV in September, 1982. His medical history was significant for multiple episodes of gonorrhea, syphilis, herpes genitalis, amebiasis, giardiasis, moniliasis, and hepatitis A and B. The diagnosis of cryptococcal meningitis with disseminated cryptocooccosis was made by positive lumbar puncture and positive supraclavicular node biopsy, respectively. The patient was treated with 5-fluorouracil and amphotericin B. Serial CT scans during November, 1982, revealed diffuse mild enlargement of all cerebrospinal fluid (CSF) spaces without evidence of a mass lesion. The patient underwent biopsy of a forearm lesion in December, 1982, which was diagnostic of Kaposi’s sarcoma. At this time he was also found to have cryptosporidiosis in his stool for which he was treated with intravenous TMS.

The patient presented again in January, 1983, with headache and lethargy. Lumbar CSF contained cryptococcus as determined by India ink testing, cryptococcal antigen was 1:32, and serum antigen was positive at 1:10,000. The patient did not have a follow-up CT scan. The patient was treated with amphotericin B but continued to complain of headaches and lethargy. The patient was treated with hyperalimentation and antibiotic therapy, but failed to improve. He became febrile with positive blood cultures for *Klebsiella pneumoniae* and positive urine cultures for *Escherichia coli*. He was placed on appropriate antibiotic therapy but failed to respond and died in septic shock in March, 1983.

**Postmortem Examination.** Neuropathological evaluation revealed generalized meningitis and ventriculitis. The patient was noted to have multiple bilateral parenchymal cryptococcal abscesses, as well as parenchymal CMV microabscesses in different sites.

**Case 8**

This 43-year-old white homosexual man was first admitted in January, 1983, with complaints of chronic diarrhea and weight loss, as well as severe headaches and slurred speech. The patient was noted at this time to have a T helper:suppressor ratio of 0.02:1 (1.8 to 2.4:1 is normal). Medical history was significant for multiple venereal diseases including several episodes of gonorrhea, syphilis, and amebiasis. The patient had suffered documented episodes of hepatitis, mononucleosis, and herpes simplex. A close friend had died recently of *Pneumocystis carinii* pneumonia associated with AIDS.

**Examination and Course.** Admission work-up included multiple serological studies, all of which were negative. Serum *Toxoplasma* IgG-IFA titers were less than 16 and serum *Toxoplasma* IgM-IFA titers were less than 2 (both nondiagnostic). A CT brain scan was performed which revealed bilateral contrast-enhancing lesions in the deep parietal regions (Fig. 3). An ultrasound-guided needle brain biopsy was performed which revealed focal encephalitis, but all cultures were negative. The patient was treated presumptively for toxoplasmosis, initially with intravenous TMS and then with sulfadiazine and pyrimethamine. During the patient’s hospitalization, other antimicrobial agents were administered, including ketoconazole and nystatin for oral and gastrointestinal candidiasis, acyclovir (Zovirax) for localized perianal herpes simplex, and metronidazole for amebiasis.
Reported cases of CNS mass lesions in patients with AIDS*  

<table>
<thead>
<tr>
<th>Authors, Year</th>
<th>No. of Cases</th>
<th>Risk Category</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rutsaert, et al., 1980</td>
<td>1</td>
<td>unknown</td>
<td>toxoplasmosis</td>
</tr>
<tr>
<td>Hymes, et al., 1981</td>
<td>1</td>
<td>homosexual</td>
<td>presumed Kaposi’s sarcoma</td>
</tr>
<tr>
<td>Vilaseca, et al., 1982</td>
<td>1</td>
<td>homosexual</td>
<td>toxoplasmosis</td>
</tr>
<tr>
<td>Lehrich, et al., 1983</td>
<td>1</td>
<td>homosexual</td>
<td>primary lymphoma</td>
</tr>
<tr>
<td>Pitchenik, et al., 1983, &amp; Post, et al., 1983</td>
<td>15</td>
<td>12 Haitian, 3 homosexual</td>
<td>13 toxoplasmosis, 1 toxoplasmosis + tuberculous abscess, 1 cryptococcal abscess</td>
</tr>
<tr>
<td>Snider, et al., 1983</td>
<td>2</td>
<td>2 homosexual</td>
<td>2 primary lymphoma</td>
</tr>
<tr>
<td>Levy, et al., 1984</td>
<td>9</td>
<td>8 homosexual, 1 unknown</td>
<td>2 toxoplasmosis, 1 toxoplasmosis + CMV, 1 cryptococcal abscess + CMV, 1 Candida albicans abscess, 2 Kaposi’s sarcoma, 2 primary lymphoma</td>
</tr>
</tbody>
</table>

* CNS = central nervous system; AIDS = acquired immunodeficiency syndrome; CMV = cytomegalovirus.

Computerized tomography scans were obtained bi-weekly. The first scan showed no change in the size of the lesions. A CT scan performed 11 days after surgery revealed, however, a definite increase in the size of the contrast-enhancing lesions with increasing surrounding low-density areas and mass effect. Multiple small enhancing periventricular lesions were also noted. A repeat biopsy was performed which revealed brain with chronic inflammation, focal necrosis, and gliosis. All cultures and histological studies again failed to document the pathological process; repeat serological work-up was nondiagnostic. The patient was continued on therapy for presumed toxoplasmosis. Follow-up CT scans revealed increasing mass effect from the lesions already noted, as well as new diffuse enhancement of all cerebral sulci. The patient’s condition progressively deteriorated and he died 2 months after the second surgical procedure.

Postmortem Examination. Neuropathological examination revealed multiple sites of necrotic immunoblastic lymphoma which corresponded to the lesions evident on CT scanning. No infectious agents were identified.

Discussion

Of the more than 2000 cases of patients with AIDS reported to the Centers for Disease Control (CDC), approximately 10% have been noted to have CNS involvement. These neurological diseases include infections, primarily Toxoplasma gondii, Cryptococcus, and viruses; malignancies, primarily lymphoma; progressive multifocal leukoencephalopathy, and (as we now report) Kaposi’s sarcoma. Twenty-one case reports of CNS mass lesions in AIDS patients exist in the literature, including 16 Toxoplasma gondii infections. Three cases of primary lymphoma, one case of presumed metastatic Kaposi’s sarcoma, and one case of cryptococcal abscesses have been described. One case of polymicrobial infection, involving both a Toxoplasma and a mycobacterial tuberculous abscess, has also been reported (Table 1). It is of note that 11 of the 12 reported cases of Haitians with AIDS and CNS mass lesions have had Toxoplasma gondii brain abscesses. Only nine of the 21 patients reported with AIDS and intracerebral mass lesions have been homosexuals, and five of these nine patients had Toxoplasma gondii CNS abscesses. The higher association of Toxoplasma brain infection in Haitian patients with AIDS is not yet understood.

We report a series of nine non-Haitian homosexual male patients presenting with CNS mass lesions (Table 2). Eight of the nine patients were confirmed homosexuals, and seven of nine had AIDS by the criteria established by the CDC. While the other two cases do not fulfill all CDC requirements for AIDS, their membership in patient categories at high risk for contracting AIDS, their opportunistic infections, and both their clinical presentation and hospital course suggested the diagnosis of AIDS. Their subsequent therapies were predicated upon the assumption that they were AIDS patients. The inclusion of these two patients adds only Candida albicans as a possible cause of CNS mass lesions in AIDS patients, and does not alter either the conclusions or the recommendations of this review.

The average age of these patients was 38.8 years. Three of the nine patients presented initially for evaluation of their neurological complaints; two of these had seizures and the third had headaches and aphasia. The other six patients already carried the diagnosis of AIDS and most had had previous opportunistic infections, usually Pneumocystis pneumonia.

The current series of nine AIDS patients includes three patients with Toxoplasma gondii abscesses, two with primary CNS lymphoma, two with metastatic CNS Kaposi’s sarcoma, two with focal CMV encephalitis, and one each with cryptococcal and Candida albicans abscesses. These include two cases of polymicrobial infection, one consisting of Toxoplasma gondii brain abscess with CMV microabscesses and the other of focal cryptococcoma and CMV microabscesses. Of particular note is that in these polymicrobial infections the different organisms were identified in separate lesions. Seven of the nine patients presented with multiple intracerebral lesions.
### TABLE 2

Data on nine male patients with AIDS and intracranial mass lesions

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs)</th>
<th>Risk Factors</th>
<th>Concomitant Non-CNS Disease</th>
<th>Diagnosis</th>
<th>Autopsy</th>
<th>Biopsy</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>30</td>
<td>homosexual, multiple</td>
<td>Pneumocystis pneumonia</td>
<td>Toxoplasma, none</td>
<td>pyrimethamine, sulfadiazine</td>
<td>+ complete resolution</td>
</tr>
<tr>
<td>2</td>
<td>25</td>
<td>homosexual</td>
<td>Pneumocystis pneumonia</td>
<td>Toxoplasma, non-diagnostic</td>
<td>clindamycin, TMS</td>
<td>- progression</td>
</tr>
<tr>
<td>3</td>
<td>30</td>
<td>homosexual, multiple</td>
<td>Pneumocystis pneumonia, CMV pneumonia, Legionnaire's disease, Kaposi's sarcoma</td>
<td>Toxoplasma, gondii, CMV</td>
<td>erythromycin, ketoconazole, Flagyl (metronidazole), TMS</td>
<td>+ partial resolution</td>
</tr>
<tr>
<td>4</td>
<td>37</td>
<td>homosexual, multiple</td>
<td>Pneumocystis pneumonia, disseminated CMV &amp; cryptococciosis, E. coli, UTI</td>
<td>Cryptococcus, CMV</td>
<td>5-flucytosine, + no change</td>
<td>N/A</td>
</tr>
<tr>
<td>5</td>
<td>37</td>
<td>homosexual, IV drug abuse</td>
<td>Pneumocystis pneumonia, disseminated CMV &amp; cryptococciosis, Kaposi's sarcoma</td>
<td>None</td>
<td>5-flucytosine, + no change</td>
<td>N/A</td>
</tr>
<tr>
<td>6</td>
<td>60</td>
<td>?, sexual preference</td>
<td>Pneumocystis pneumonia</td>
<td>Candida albicans</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>7</td>
<td>47</td>
<td>homosexual</td>
<td>Kaposi's sarcoma</td>
<td>None</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>8</td>
<td>43</td>
<td>homosexual, multiple, VI drug abuse, associate of AIDS patients</td>
<td>Kaposi's sarcoma</td>
<td>None</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>9</td>
<td>40</td>
<td>homosexual</td>
<td>Disseminated CMV, cryptococcosis, Kaposi's sarcoma</td>
<td>None</td>
<td>primary CNS</td>
<td>none</td>
</tr>
</tbody>
</table>

**Response to Treatment:**
- +: Complete resolution
- -: Progression
- ?: Partial resolution
- NA: Not available

**Survival Time:**
- 3 mos: 3 months
- 9 mos: 9 months
- 7 mos: 7 months
- 4 mos: 4 months
- 2 mos: 2 months
- 1 mos: 1 month
- NA: Not available

**Diagnosis and Treatment of CNS Lesions:**
- TMS: Trimethoprim/sulfamethoxazole
- CT: Computerized tomography
- CMV: Cytomegalovirus
- VD: Venereal disease
- NA: Not applicable
- GI: Gastrointestinal
- IV: Intravenous
- +: Positive
- -: Negative

**Cause of Death:**
- Respiratory (recurrent Pneumocystis pneumonia)
- CNS (toxoplasmosis)
- CNS (toxoplasmosis + CMV)
- Sepsis
- Respiratory (Kaposi's sarcoma)
- GI bleeding (disseminated intravascular coagulation)
- CNS (primary lymphoma)

* AIDS = acquired immunodeficiency syndrome; CNS = central nervous system; CT = computerized tomography; VD = venereal disease; CMV = cytomegalovirus; UTI = urinary tract infection; TMS = trimethoprim/sulfamethoxazole; NA = not applicable; GI = gastrointestinal; IV = intravenous; + = positive; - = negative.

† See text.
Eight of the nine patients died, and the ninth was lost to follow-up review. Mean survival of these eight patients was 9.0 months after first presentation and 4.4 months after neurological presentation. While all cases with follow-up review have died, only three of these patients died of their CNS disease. Of these three cases, one had primary CNS lymphoma which was not diagnosed until autopsy. This patient received therapy for presumptive toxoplasmosis. The second patient died of CNS toxoplasmosis despite appropriate therapy. The third patient was also treated for CNS toxoplasmosis, and was noted at autopsy to have both CNS toxoplasmosis and CMV infection.

Four other patients received appropriate therapy for their CNS disease and were either lost to follow-up review or died of non-CNS disease. One patient, treated with pyrimethamine and sulfadiazine for Toxoplasma brain abscesses, demonstrated both a complete disappearance of his brain lesions on follow-up CT scans and a marked neurological improvement. A second patient, treated with radiation therapy for metastatic Kaposi’s sarcoma, demonstrated both a marked clinical improvement and a significant decrease in the size of the CNS lesion. The third patient, treated for cryptococcal meningitis, had a marked clinical improvement but subsequently developed a cryptococcal brain abscess and focal CMV encephalitis, despite continued therapy. These patients died of recurrent Pneumocystis carinii pneumonia, disseminated Kaposi’s sarcoma, and sepsis, respectively. The fourth patient, treated with amphotericin B and 5-flucytosine for Candida albicans abscesses, had resolution of his headaches following the surgical removal of a large brain lesion but failed to demonstrate any decrease in the size of the remaining lesions over 4 months before being lost to follow-up review. Two patients were not treated for their CNS disease and succumbed from non-CNS disease. Thus, recognition and appropriate treatment of the CNS lesion may have some bearing on the patient’s immediate clinical course, although long-term follow-up at this time indicates that the patients will ultimately succumb to some other opportunistic disease process.

Pitchenik, et al.,!* suggested that, in a subgroup of patients with AIDS, mass lesions on CT brain scan, and high Toxoplasma gondii titers, particularly those in whom the lesions lie in critical brain regions, a trial of antibiotic therapy for presumptive Toxoplasma infection is an acceptable alternative to establishing the diagnosis by brain biopsy. While in their group of predominantly Haitian AIDS patients the nonsurgical treatment of CNS mass lesions may be reasonable, a potential danger arises if this approach is generalized to all patients with AIDS and CNS mass lesions. In fact, in our population of non-Haitian homosexual patients with AIDS, this does not appear to be an acceptable option for several reasons. First, Toxoplasma gondii infections accounted for only 30% of the CNS mass lesions in our series. Other causes with similar clinical presentations and CT appearance, including viruses, lymphoma, Candida, and Kaposi’s sarcoma, were more frequently encountered. Second, the ability of serodiagnostic tests to differentiate these Toxoplasma abscesses from other disease processes is poor. In all three of our patients with Toxoplasma gondii infections, acute serological titers were nondiagnostic (Table 3). Making a reliable diagnosis of toxoplasmosis by serodiagnosis is fraught with difficulties. As the incidence of chronic positive serological titers for toxoplasmosis in the normal population has been reported to be as high as 70%,¹ the diagnostic use of a single serological value in predicting or excluding Toxoplasma gondii CNS abscesses is often problematical. There exist nonspecific cross-reactive antibodies for certain of the serological techniques. In addition, it has also been difficult to distinguish between acute infection and chronic (latent) infection. Recent development and use of the IgM-IFA and the IgM-Elisa tests for detection of early antibodies to Toxoplasma in acute infections has been more promising. In general, the presence of a single IgG titer in any of the serological tests available establishes the presence of prior Toxoplasma infection but not necessarily acute illness. In order to diagnose acute, active Toxoplasma infection by serology, one must usually show a significant rise in titer (either by dye test, citrovorum factor, or IFA). Thus, although suggestive of active acute infection, a single high IgG titer is not diagnostic. Moreover, in our patients with acute CNS toxoplasmosis, none of the single titers (dye test, IgG-IFA, IgM-IFA, or IgM-Elisa) performed were diagnostic.

Unlike classical precepts with respect to pyogenic brain abscess in immunocompetent hosts, the nature of the CNS lesions in this unique population of patients bears little relation to other underlying infections or

<table>
<thead>
<tr>
<th>Case No.</th>
<th>CNS Diagnosis</th>
<th>Test</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Toxoplasma gondii</td>
<td>IgG-IFA</td>
<td>1:32</td>
</tr>
<tr>
<td></td>
<td></td>
<td>IgM-IFA</td>
<td>1:256</td>
</tr>
<tr>
<td></td>
<td></td>
<td>IgM-Elisa</td>
<td>less than 1:2</td>
</tr>
<tr>
<td>2</td>
<td>Toxoplasma gondii</td>
<td>Toxoplasma antigen</td>
<td>negative</td>
</tr>
<tr>
<td></td>
<td></td>
<td>IgG-IFA</td>
<td>1:1024†</td>
</tr>
<tr>
<td></td>
<td></td>
<td>IgM-Elisa</td>
<td>less than 1:4†</td>
</tr>
<tr>
<td>3</td>
<td>Toxoplasma gondii + CMV</td>
<td>IgG &amp; IgM-combined</td>
<td>less than 1:128</td>
</tr>
<tr>
<td>4</td>
<td>primary CNS lymphoma</td>
<td>IgG-IFA</td>
<td>less than 1:16</td>
</tr>
<tr>
<td></td>
<td></td>
<td>IgM-IFA</td>
<td>less than 1:2</td>
</tr>
</tbody>
</table>

* AIDS = acquired immunodeficiency syndrome; CNS = central nervous system; Ig = immunoglobulin; IFA = indirect fluorescent antibody.
† Tests run simultaneously on two samples obtained 10 days apart without change.
Central nervous system mass lesions in AIDS

TABLE 4
CT findings in AIDS patients with intracranial mass lesions*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Lesion Site Enhancement</th>
<th>Location of Lesions</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Single &amp; Bilat</td>
<td>Multiple</td>
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<tr>
<td>1</td>
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<tr>
<td>2</td>
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<td>7</td>
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<td>+</td>
</tr>
<tr>
<td>8</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>

* CT = computerized tomography; AIDS = acquired immunodeficiency syndrome. Cases 4 and 7: CT scans early in course did not demonstrate CNS mass lesion; no later scans were obtained. Case 9: CT scans not performed.

systemic diseases. We report, for example, a patient with widely metastatic Kaposi’s sarcoma with autopsy-proven primary CNS lymphoma, as well as a patient with Kaposi’s sarcoma and Pneumocystis carinii pneumonia with biopsy proven Toxoplasma brain abscesses.

The two patients with polymicrobial infection also tend to support the argument that empiric therapy without brain biopsy may be an inadequate approach. Although present antiviral agents are not effective in treating CMV encephalitis, promising new antiviral agents are being developed for this illness and adenine arabinoside and acyclovir have been already proven useful for therapy of herpes simplex infections. Especially disconcerting is the observation that the different organisms in these polymicrobial infections were isolated from separate lesions. The treatment of AIDS patients with CNS mass lesions must take into account this possibility. Thus, following diagnosis and institution of specific therapy, frequent CT follow-up review must be conducted to detect a differential response to therapy of one or more masses as compared to the mass from which the biopsy was obtained. Should such a differential response occur, a biopsy of the other mass(es) should be considered to determine whether other intracranial disease processes are present.

In conclusion, our experience with AIDS patients with intracerebral mass lesions indicates that the clinical presentation and CT appearance of these lesions are not useful in establishing a diagnosis (Table 4). The various types of disease processes discovered at biopsy or autopsy, the unreliability of serodiagnostic techniques for toxoplasmosis, and the lack of correlation between the patients’ CNS lesions and non-CNS infections or systemic diseases all emphasize the need to perform a brain biopsy in such cases. As the therapies for the different lesions encountered are widely divergent, ranging from radiation therapy and chemotherapy to amphotericin B and sulfa drugs, tissue diagnosis is critical for directing appropriate therapy. The morbidity and mortality rates associated with biopsy procedures, particularly those using stereotaxic techniques and ultrasound guidance, have decreased so markedly over the past several years that medical treatment of these patients without a tissue diagnosis seems unwise.

Addendum

In the interval between the submission of this manuscript and March, 1984, we have evaluated five additional patients with AIDS and mass lesions of the CNS. All five were homosexual men ranging in age from 26 to 45 years. Three presented with neurological symptoms as the first indications of AIDS. Computerized tomography scans revealed bilateral ring-enhancing lesions in three patients and solitary unilateral low-density lesions in two patients. Two patients had biopsy-proven Toxoplasma gondii abscesses; one patient had a biopsy-proven herpes simplex Type II focal infection. One patient was demonstrated to have a massive hemispheric infarction on CT, and at autopsy was proven to have nontuberculous meningitis. The fifth patient, with a low-density unilateral basal ganglia lesion, revealed as a low-density area on CT scanning, has undergone two nondiagnostic biopsy procedures. These findings confirm the broad spectrum of CNS mass lesions in the patient with AIDS, and the need for biopsy in cases where the diagnosis is in question.

References


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