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A retrospective review of cranial CT scans obtained over a 4 year period in patients with acquired immunodeficiency syndrome (AIDS) and documented central nervous system (CNS) pathology is presented. The spectrum of diseases and the value of CT in detecting new, recurrent, and superimposed disease processes were determined. Fifty-one AIDS patients with confirmed CNS pathology were identified. Six of them had two coexistent diseases. Opportunistic infections predominated, especially *Toxoplasma* encephalitis and cryptococcal meningitis, while tumor was seen infrequently. Initial CT was positive in 76% of cases. In contrast to meningeal processes, where it was not very effective, CT was very sensitive in detecting most parenchymal disease processes. Characteristic although not pathognomonic CT patterns were found for certain diseases. Improvement or resolution of CT abnormalities in patients on medical therapy for *Toxoplasma* encephalitis correlated well with clinical improvement. Recurrence of CT abnormalities correlated well with medical noncompliance. The optimal contrast enhancement technique for detecting CNS pathology and for monitoring the effectiveness of medical therapy was also evaluated by a prospective study in which both immediate (IDD) and 1 hr delayed (DDD) double-dose contrast CT scans were compared. The examination found to be diagnostically superior in 30 of the 41 IDD/DDD studies was the delayed scan. It is recommended that CT be used routinely and with the 1 hr DDD scan to evaluate and follow AIDS patients with neurologic symptoms and/or signs.

Recent reports in the literature have drawn attention to the fact that neurologic symptoms and signs occur often in patients with acquired immunodeficiency syndrome (AIDS) [1–6]. The causes of these neurologic abnormalities have been attributed to a variety of central nervous system (CNS) infections and tumors typical for the immunocompromised host [1–13]. Due to the severe nature of these CNS diseases the need for establishing early diagnosis has been recognized [3, 4, 6–10]. The crucial role that computed tomography (CT) plays in diagnosis has been acknowledged. However, the sensitivity and specificity of CT remains unknown as does the optimal contrast technique for establishing diagnosis. The use of delayed double-dose (DDD) scans in neurologically compromised patients with AIDS has been suggested, but a prospective study has not been performed [6, 7]. Studies of high-dose-iodine technique and/or of time-delay serial CT scans have mainly been described for neoplasms, demyelinating diseases, and for infection in non-AIDS patients [14–18]. We undertook our investigation to determine the sensitivity, specificity, and optimal contrast enhancement technique of cranial CT in AIDS patients.

Materials, Subjects, and Methods

The first part of this study was a retrospective analysis of CNS disease in AIDS patients at the University of Miami/Jackson Memorial Hospital complex. Those patients were identified who fit the criteria for AIDS established by the Centers for Disease Control and who had experienced neurologic symptoms and/or signs. Selected from this group were those patients

who had been evaluated by CT and had documented CNS pathology related to AIDS. CT scans obtained on the GE 8800 or 9800 CT/T scanners between April 1980 and July 1984 and the pathologic material were then reviewed to determine disease spectrum and the diagnostic capabilities of CT.

The second part of this research involved a prospective study in which AIDS patients with neurologic complaints were evaluated with both an immediate double-dose (IDD) and a 1 hr DDD scan of the brain. These scans were later compared for diagnostic quality. Excluded were those patients with a contraindication to this high-dose-iodine technique [14]. The double-dose technique involved the intravenous injection of 100 ml of Renografin 76 (36 g I) via a bolus followed by a continuous drip infusion of 150 ml of iothalamate meglumine 60% (42.3 g I).

Results

We found 51 AIDS patients who had been evaluated for intracranial symptoms and who had documented CNS pathology. The diagnoses (table 1) were confirmed by biopsy and/or autopsy in 43, by cultural methods in six, and by an appropriate response to therapy for *Toxoplasma* encephalitis in two. CNS infection was found to be the most common neurologic disease in the AIDS patient. Of the infections, *Toxoplasma* encephalitis and cryptococcal meningitis were the most common. Six patients had two CNS coexisting diseases (table 2). All were adults; 40 were men and 11 women. Thirty-one were Haitian, 17 were homosexuals, two were intravenous drug abusers, and one was married to a Haitian and had a child who had died from AIDS. Thirty-seven have died; 13 are known to be living, and one has been lost to follow-up.

Initial CT scans were positive in 39 (76%) of 51 patients and negative in 12 (24%) (the latter are summarized in table 3). CT was helpful in detecting the development of a second disease process in two of the six patients with coexistent CNS diseases (figs. 1 and 2). In the other four patients, all with *Toxoplasma* encephalitis, CT did not detect the second CNS disease (cryptococcal meningitis in one and cytomegalic inclusion virus [CMV] encephalitis in three). In two patients, however, the second organism was not contributing to the patient's neurologic symptoms and was found only microscopically at autopsy.

Initial CT scans were always abnormal when *Toxoplasma* encephalitis was the primary CNS disease. The CT features on initial presentation in the 31 patients with this infection are summarized in table 4. Most often seen were multiple deep and superficial enhancing lesions located in both cerebral hemispheres and associated with edema and mass effect. At presentation there was a single lesion in six patients, two to four lesions in 15 patients, five to 10 lesions in two patients, and more than 11 lesions in eight patients. The corticomedullary junction and the basal ganglia were the most common sites of involvement, being affected in 24 and 17 patients, respectively. Focal uptake of contrast material was almost always present in a ring and/or nodular configuration. The rings were thick and irregular in 11 patients with large lesions.

Serial scans were obtained in 28 of 31 patients with *Toxoplasma* encephalitis. Of these, 20 patients had CT scans while undergoing medical therapy for toxoplasmosis (table 5). The first follow-up scan in 18 of the treated patients was 4–35

TABLE 1: Spectrum of Central Nervous System Pathology in Acquired Immunodeficiency Syndrome

Disease Process	No. of Patients
Toxoplasma encephalitis	31
Cryptococcal meningitis	10
Cytomegalic inclusion virus encephalitis	5
Progressive multifocal leukoencephalopathy	3
Lymphoma	2
Tuberculous abscess	1
Fungal brain abscess	1
Nonspecific encephalitis	1
E. coli meningitis	1
Acanthamoeba castellani sinusitis and trigeminal ganglion involvement	1
Hemorrhage	1

TABLE 2: Types of Coexistent Disease Processes in Acquired Immunodeficiency Syndrome

Disease Process	No. of Patients
Toxoplasma encephalitis + cytomegalic inclusion virus	3
Toxoplasma encephalitis + cryptococcal meningitis	1
Toxoplasma encephalitis + tuberculous abscess	1
Cryptococcal meningitis + lymphoma	1

TABLE 3: Types of Disease Processes and Their Frequency in Acquired Immunodeficiency Syndrome Patients with Negative CT Scans

Disease Process	No. of Patients
Meningitis	9
Parenchymal infection (all microscopic):	
Fungal abscess	1
Progressive multifocal leukoencephalopathy	1
Encephalitis	1
Total	3

days (median, 11 days) after initiation of medical therapy. Evidence of improvement was noted in 16 of these scans and it corresponded to clinical improvement. CT demonstrated decreases in the areas of enhancement, amount of edema, and mass effect. These changes were usually apparent within 2 weeks of institution of therapy. The other two patients also improved clinically, but scans showed apparent worsening judged by a greater number of enhancing lesions. However, the increased number of lesions was probably related to the use of a higher dose of contrast material for the follow-up scan rather than to the development of new lesions.

Negative scans were seen in four patients after 2¼–4½ months of continuous therapy. These scans remained normal over a 5–7½ month follow-up as patients were maintained on anti-*Toxoplasma* therapy. Scans turned negative in another three patients, but did not remain normal because of interruption of medical therapy.

Eleven episodes of recurrent *Toxoplasma* encephalitis were seen in eight patients, usually because of medical noncompliance but also because of cessation of therapy in two patients
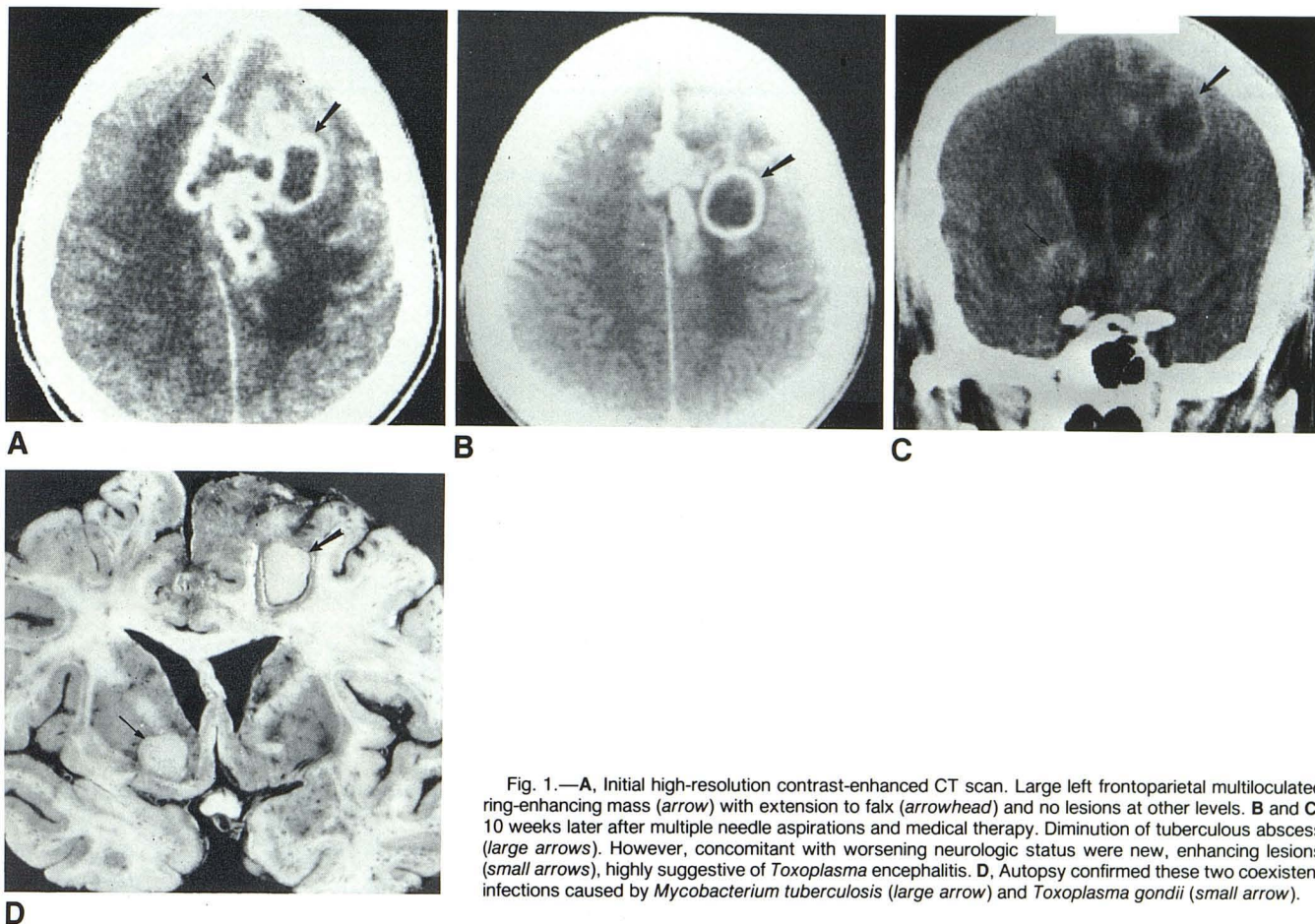
Fig. 1.—**A**, Initial high-resolution contrast-enhanced CT scan. Large left frontoparietal multiloculated ring-enhancing mass (*arrow*) with extension to falx (*arrowhead*) and no lesions at other levels. **B** and **C**, 10 weeks later after multiple needle aspirations and medical therapy. Diminution of tuberculous abscess (*large arrows*). However, concomitant with worsening neurologic status were new, enhancing lesions (*small arrows*), highly suggestive of *Toxoplasma* encephalitis. **D**, Autopsy confirmed these two coexistent infections caused by *Mycobacterium tuberculosis* (*large arrow*) and *Toxoplasma gondii* (*small arrow*).
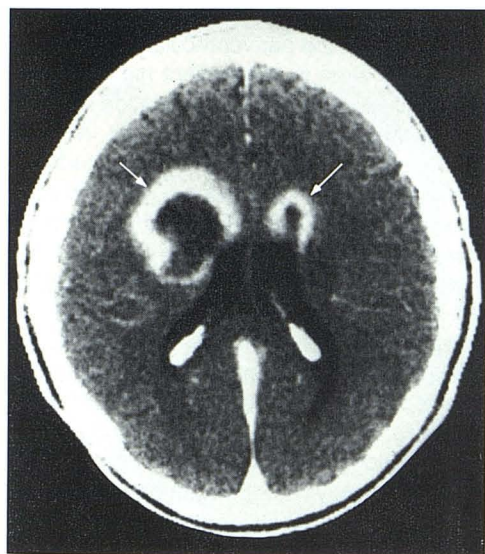

Fig. 2.—DDD scan in Haitian with cryptococcal meningitis. Large inhomogeneously enhancing periventricular lesions (*arrows*) prompted biopsy, which revealed primary CNS lymphoma.

after severe drug reactions. Recurrences were recognized on CT in all cases by an increased amount of edema and mass effect associated with the reappearance of some of the previously seen areas of enhancement (fig. 3). In 10 of the 11 cases new areas of abnormal enhancement were also seen. After reinstitution of medical therapy nine of the 11 episodes of recurrent disease were reevaluated by CT at intervals of 10 days to 3½ months (median, 24 days). In all nine episodes CT demonstrated decreased size and number of enhancing lesions with less edema and mass effect.

Long-term follow-up scans in most patients on continuous medical therapy revealed no residual areas of enhancement, and occasionally areas of encephalomalacia manifested on CT as areas of low density with hydrocephalus ex vacuo and cortical atrophy (fig. 4). Less often, slight residual enhancement persisted unchanged on long-term follow-up studies. The original enhancing lesions partly or totally calcified in four treated patients (fig. 5). Calcifications were detected on plain CT scans within 2½–5½ months of the onset of symptoms. The presence of calcification on CT was not always an indicator of inactivity of the *Toxoplasma* lesions, since in some cases enhancing nodules were still seen in association with the calcifications.

TABLE 4: **CT Features of *Toxoplasma* Encephalitis before Surgical Intervention or Definitive Medical Therapy in Acquired Immunodeficiency Syndrome**

	No. of Patients
Nos. and locations of lesions:	
Single intraparenchymal:	
Deep (thalamus, hypothalamus, basal ganglia)	4
Superficial (corticomedullary junction)	2
Total	6
Multiple intraparenchymal:	
Cerebral hemispheres (deep and/or superficial)	25
Thalamus	8
Midbrain, pons, and/or medulla	6
Cerebellum	9
Total	25
Appearance on contrast-enhanced studies:	
Focal contrast uptake:	
Nodular	11
Ring	10
Nodular and ring	8
Total	29
No focal contrast uptake:	
Multiple low-density lesions only	1
Multiple low-density lesions associated with diffuse gyral enhancement	1
Total	2
Edema:	
Mild	9
Moderate	16
Marked	5
Total	30
Mass effect:	
Mild	13
Moderate	10
Marked	3
Total	26
Hydrocephalus (2° partial outlet obstruction):	
Mild	7
Moderate	7
Total	14

TABLE 5: **CT Scan Data and Clinical Outcomes in Acquired Immunodeficiency Syndrome Patients Treated for *Toxoplasma* Encephalitis and Having Follow-up CT Scans**

	No. of Patients (n = 20)
Time intervals from first to last follow-up scan after institution of medical therapy:	
1–14 days	3
15–59 days	2
2–6 months	9
7–12 months	4
13–15 months	2
Scan appearance over time:	
Developed negative scans with no later CT recurrences .	4
Developed negative scans with later single or multiple recurrences with eventual improvement (one) or death (two)	3
Initial scan improvement followed later by single or multiple recurrences, with long-term improvement (two) or eventual death (two)	4
Showed improvement on short-term follow-up scans (no long-term follow-up available yet)	5
Showed long-term stable improvement on follow-up scans	2
Apparently worsened on early scans, but clinical improvement (one later showing CT improvement also, then recurrence, then improvement, and the other later dying of unrelated causes)	2

The effectiveness of medical therapy for *Toxoplasma* encephalitis was evident from clinical and CT evaluations and from autopsy correlation. Eight of the 20 treated patients are alive, clinically well, and radiographically improved over a follow-up period of 3–13 months. Another patient was lost to follow-up. Of the 11 patients who died, eight died from unrelated causes without evidence of active *Toxoplasma* encephalitis. Proof of inactivity came from autopsy examination in five and from CT scans in three. The five autopsied patients had been maintained on medical therapy for time periods of 1½–9¼ months; healed *Toxoplasma* lesions were found on examination of the brain, which correlated with the CT appearance of inactive lesions. Autopsies were not performed in three other patients who were also maintained on medical therapy for periods of 8–14¼ months, but serial CT scans showed no evidence of activity of *Toxoplasma* encephalitis. Autopsies in the other three patients revealed that they had died from *Toxoplasma* encephalitis; all were severely neuro-

logically compromised before initiation or reinstitution of medical therapy.

CT scans were negative in eight of the 10 patients who had meningitis secondary to *Cryptococcus neoformans*. In the two with positive scans, the abnormalities were caused by a superimposed disease process: *Toxoplasma* encephalitis in one and lymphoma in the other. The eight negative CT scans included the two patients in whom cryptococcal perivascular infiltration of the brain was found at autopsy.

Abnormalities secondary to CMV encephalitis were found on CT in two of five patients. Diffuse bilateral low densities in the centrum semiovale and in the periventricular white matter without mass effect were seen in one patient (fig. 6). These hypodense areas correlated well with the location of CMV found at autopsy. In the second patient a small, nodular, enhancing lesion was evident at the parietal corticomedullary junction. Autopsy revealed that the infection was much more widespread than indicated on CT. In another two individuals CT findings were restricted to those caused by healed *Toxoplasma* lesions. The microscopic foci of CMV found at autopsy were clinically and radiographically silent. In the fifth patient CMV encephalitis was found throughout the brain at autopsy. However, the infection was not suspected from serial CT scans, which showed changes compatible with treated *Toxoplasma* encephalitis.

CT was abnormal in two of the three cases of progressive multifocal leukoencephalopathy (PML). In the patient with no CT abnormalities, only microscopic lesions were found at autopsy. In the two positive cases, high-resolution CT with double-dose technique showed low-density lesions without mass effect with parietooccipital involvement in both cases and additional frontal involvement in another. In one case, time-interval scans over a 3½ week period were negative,
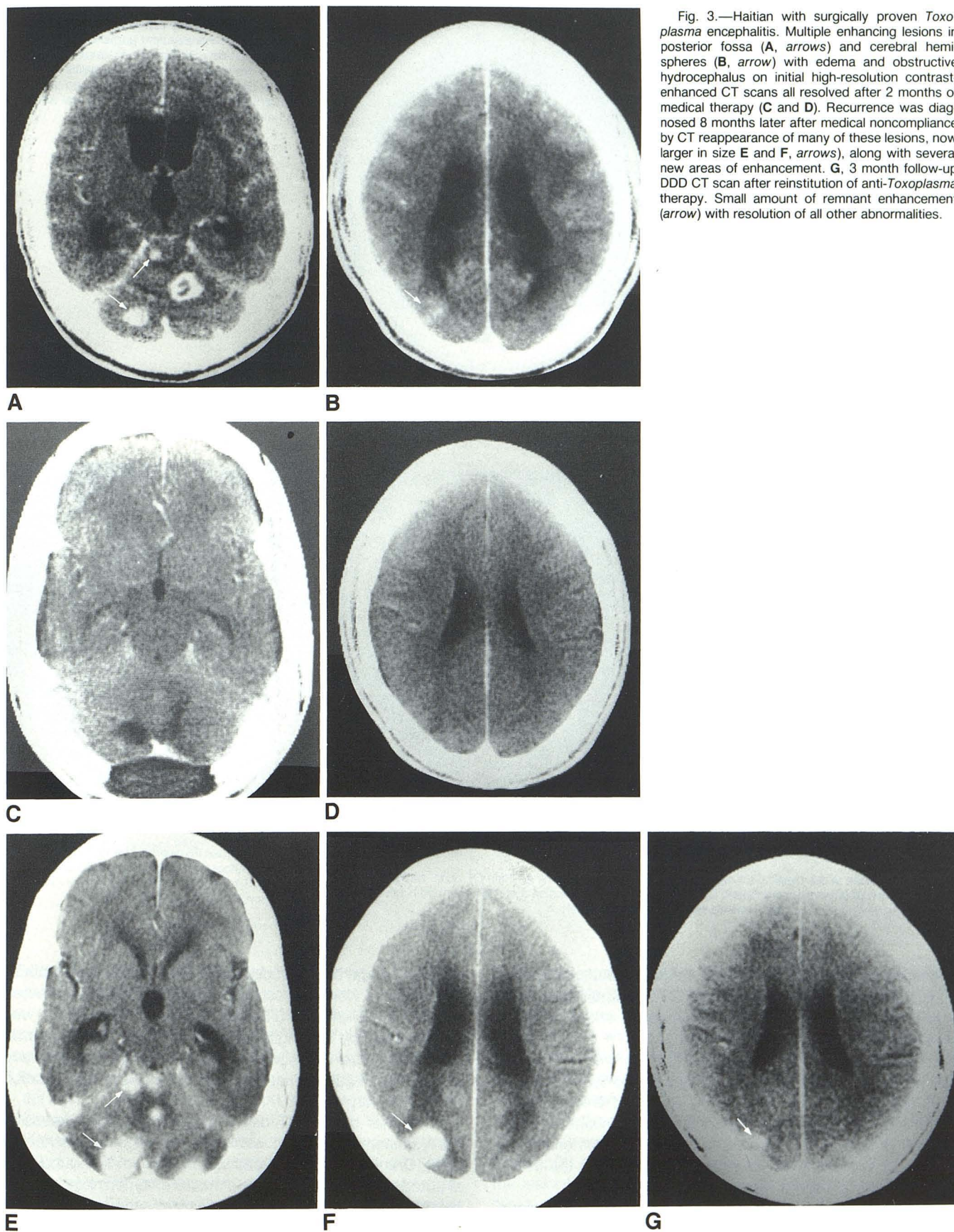
Fig. 3.—Haitian with surgically proven *Toxoplasma* encephalitis. Multiple enhancing lesions in posterior fossa (**A**, *arrows*) and cerebral hemispheres (**B**, *arrow*) with edema and obstructive hydrocephalus on initial high-resolution contrast-enhanced CT scans all resolved after 2 months of medical therapy (**C** and **D**). Recurrence was diagnosed 8 months later after medical noncompliance by CT reappearance of many of these lesions, now larger in size **E** and **F**, *arrows*), along with several new areas of enhancement. **G**, 3 month follow-up DDD CT scan after reinstitution of anti-*Toxoplasma* therapy. Small amount of remnant enhancement (*arrow*) with resolution of all other abnormalities.
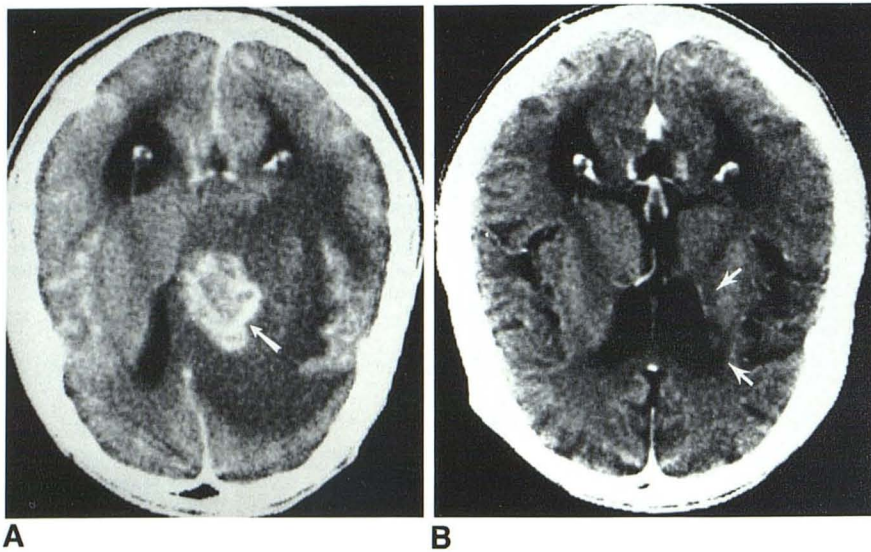
Fig. 4.—**A**, Initial high-resolution contrast-enhanced CT scan. Densely enhancing basal ganglionic mass (*arrow*) with edema and partial outlet obstructive hydrocephalus. **B**, 9 months later after therapy for biopsy-proven *Toxoplasma* encephalitis. Only residual low densities (*arrows*) with ex vacuo dilatation of adjacent frontal horn and diffuse cortical atrophy. (**A** reprinted from [7].)
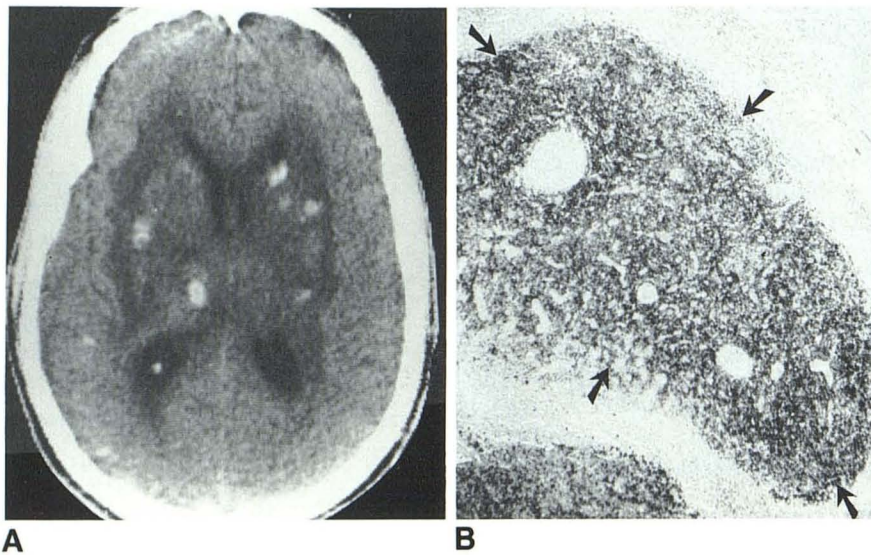

Fig. 5.—Haitian under medical therapy for *Toxoplasma* encephalitis. **A**, Plain CT scan. Multiple calcifications at sites of original enhancement. **B**, von Kossa stain of autopsied material confirmed black calcium phosphate granules (*arrows*) and showed vestiges of blood vessels.
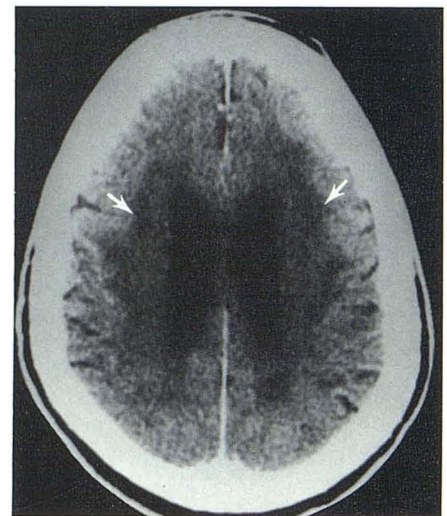
Fig. 6.—Diffuse involvement of white matter secondary to autopsy-proven CMV encephalitis manifested on CT by marked hypodensity in periventricular regions (*arrows*) and centrum semiovale.

becoming positive only 2 days before the patient's death (fig. 7). In the other case progression of CT abnormalities over a 3 week interval correlated with deterioration of the patient's neurologic status (fig. 8).

In the two cases of lymphoma, CT showed enhancing lesions in a periventricular location (figs. 2 and 9). The type of contrast uptake included both ring and solid enhancement. When ring enhancement was present, DDD scans showed filling-in of hypodense centers with contrast material. The lesions were multiple in one patient and single in the other. Mass effect and edema were negligible.

CT was strikingly abnormal in the patient with a tuberculous abscess (fig. 1). In contrast, CT scans were negative in the cases of fungal brain abscess and viral encephalitis. Both of these lesions were microscopic, however, and only detected at autopsy. Likewise, CT was normal in the patient with *Escherichia coli* meningitis. In the individual with *Acanthamoeba castellani* infection involving the maxillary sinuses and the trigeminal ganglion at autopsy, CT showed opacification of the maxillary sinuses.

In another patient CT demonstrated hypodense areas in the brainstem and thalami with some periventricular enhancement. One week later this patient suddenly deteriorated, and at autopsy was found to have a massive hemorrhage in these areas. A marked proliferation of vessels suggested the possibility of Kaposi sarcoma with secondary hemorrhage.

CT OF AIDS

Fig. 7.—Haitian with autopsy-proven PML. **A**, Initial high-resolution contrast-enhanced CT scan is normal. **B**, 1 month later. Hypodense lesions bilaterally in white matter of centrum semiovale (*arrows*).
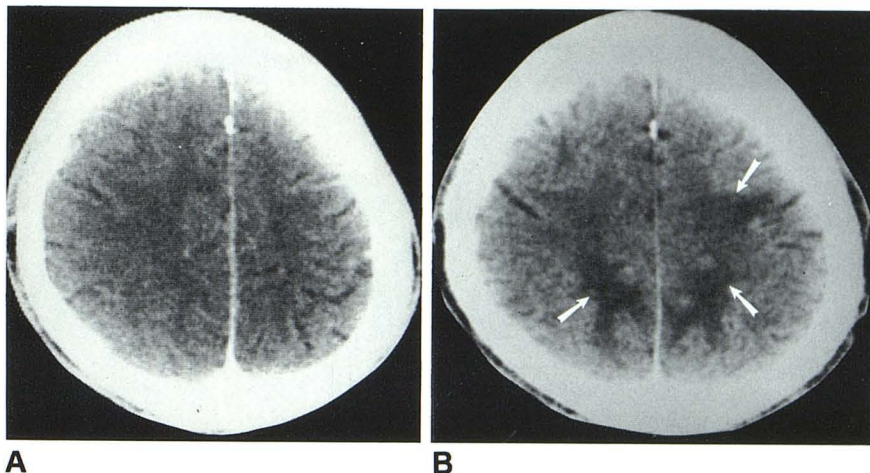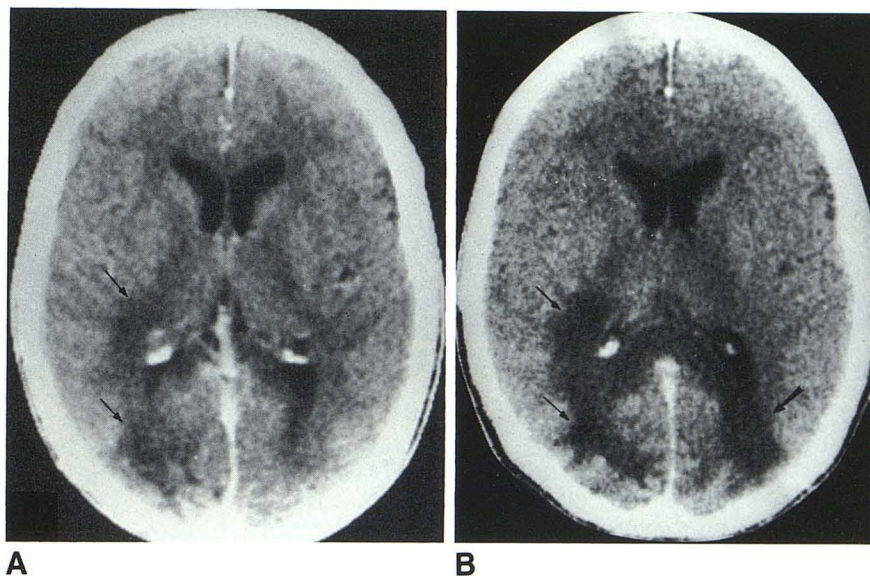

A **B**

Fig. 8.—**A**, Contrast-enhanced CT scan. Low density in right periventricular white matter (*arrows*) without significant mass effect. **B**, 3 weeks later. Bilateral hypodense periventricular lesions (*arrows*) were found at biopsy to represent PML.

A **B**

Evidence of atrophy was present on CT scans in 28 of the 51 patients. Diffuse cortical sulcal enlargement was seen; it was of mild degree in 25 and moderate in three. Fifteen patients also had mild ex vacuo dilatation of the lateral ventricles and enlargement of the cerebellar cisterns. The cause of these abnormalities is not known. In 14 they were noted on the initial scans and in 14 only on follow-up studies after treatment.

CT was used in our series to detect central nervous system pathology, localize lesions for biopsy, monitor therapy, and discern recurrent and superimposed diseases. It was also used to reevaluate those few patients with nondiagnostic biopsies; the results prompted rebiopsies. CT also aided in

the detection of postoperative hemorrhagic complications in seven patients, two of which were sizeable.

The technique that was found optimal for CT evaluation of AIDS patients with neurologic symptoms and signs was the DDD study. This determination was made by an analysis of 41 IDD/DDD examinations performed in 25 patients. Comparison of these contrast studies (table 6) revealed that the DDD CT scan was diagnostically superior in 30 IDD/DDD studies, showing either (1) a greater number of enhancing lesions; (2) a greater degree of contrast accumulation within the lesions; and/or (3) a larger size to the lesions (figs. 10–12).

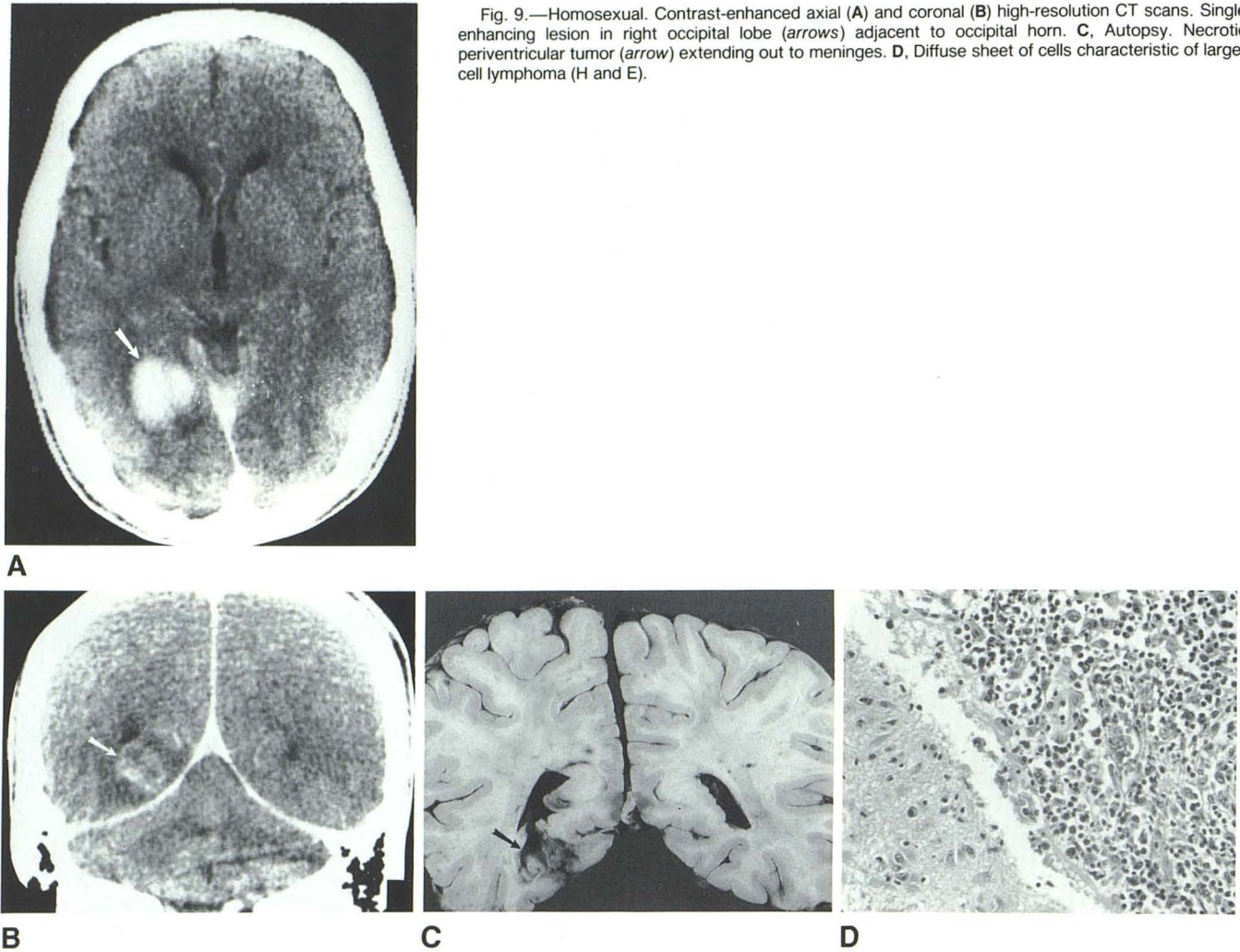On the IDD studies a distinction occasionally could not be

Fig. 9.—Homosexual. Contrast-enhanced axial (**A**) and coronal (**B**) high-resolution CT scans. Single enhancing lesion in right occipital lobe (*arrows*) adjacent to occipital horn. **C**, Autopsy. Necrotic periventricular tumor (*arrow*) extending out to meninges. **D**, Diffuse sheet of cells characteristic of large-cell lymphoma (H and E).

TABLE 6: **Comparison of Diagnostic Quality of Immediate (IDD) and Delayed (DDD) Double-Dose Examinations in 25 Patients with Acquired Immunodeficiency Syndrome**

Quality of Examination	No. of Examinations
DDD diagnostically superior	30
IDD diagnostically superior	1
No significant difference in enhancing abnormalities (all chronic cases)	5
IDD and DDD both negative	3
IDD and DDD both showed only low-density lesions (in progressive multifocal leukoencephalopathy)	2
Total	41

made between normal contrast-filled vessels and enhancing lesions. This distinction, however, could be made by a 1 hr delay. In a few cases the IDD studies on initial workup were either completely negative or showed only edema, while the DDD examinations demonstrated enhancing lesions that made the diagnosis of *Toxoplasma* encephalitis in these cases very likely and that localized the lesions for biopsy confirmation (fig. 13). In a few other cases the IDD scans showed no

residual enhancing lesions in patients undergoing medical therapy, whereas the DDD study demonstrated residual enhancing lesions that suggested the need for continuation of therapy.

The IDD studies did detect disease in most patients. In one case the IDD examination was superior to the DDD study, which had been performed after a 2 hr delay. Despite the attempt to perform the DDD examinations precisely after a 1 hr delay, the actual interval between the 41 IDD and DDD studies was 24–161 min (median time, 69 min). Prolonged time intervals between the IDD and DDD scans resulted in a less striking difference between the diagnostic quality of the two examinations. The margins of the lesions became less distinct as the contrast material began to fade in the lesions of the DDD scans obtained after 1½ hr.

Renal status was assessed after the IDD/DDD examinations in 40 cases. There was maintenance of a normal BUN and creatinine in 34 cases. In four cases deterioration of renal function occurred within 2–10 days of the examinations. Two were in patients with preexisting renal insufficiency; double-dose injections occurred when BUN and creatinine were abnormal. In the other two cases there was normal renal function and the subsequent development of mild renal insuf-

Fig. 10.—A, IDD scan. No definite lesions. B, 77 min later. Four enhancing lesions (arrows) found at autopsy to be Toxoplasma encephalitis.

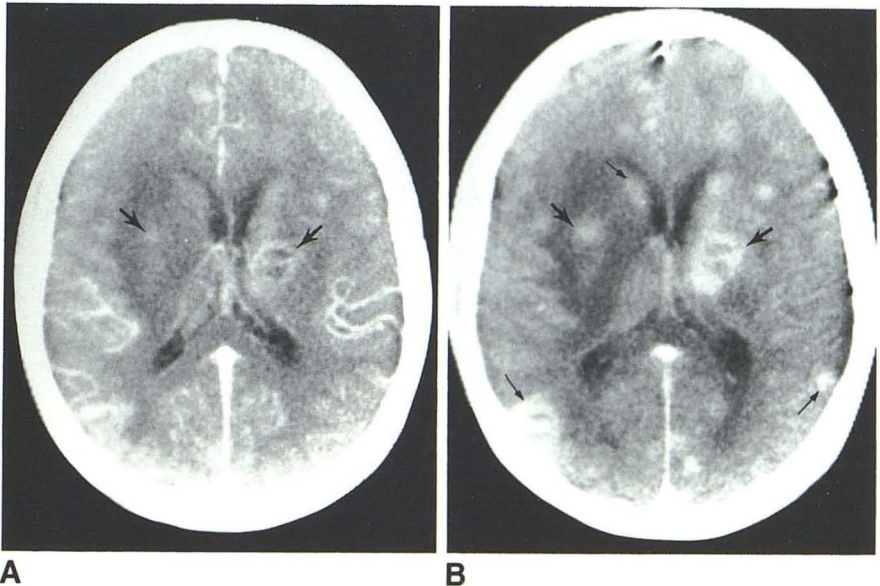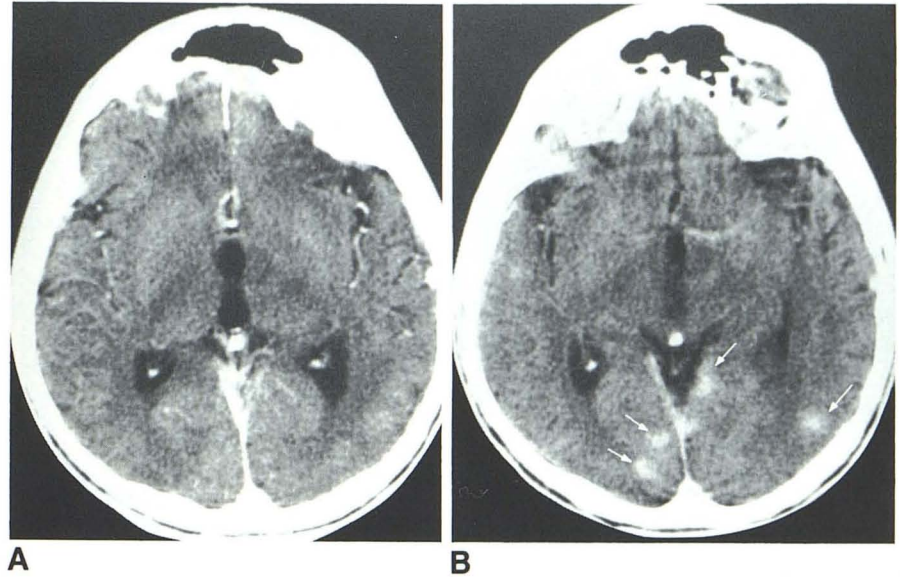A   B

Fig. 11.—Haitian with biopsy-proven Toxoplasma encephalitis. A, IDD scan. Multiple bilateral enhancing lesions (arrows) appeared larger and denser on subsequent delayed scan (B, large arrows). DDD scan also identified additional (small arrows) and more biopsy-accessible lesions.

A   B

ficiency after the contrast-enhanced studies. In one of these cases the patient was on nephrotoxic medication. In another two cases deterioration of renal function developed 2–4 weeks after injection and was due to non-contrast-related causes.

Discussion

CNS disease may be the first expression of AIDS, but it may also occur as a late manifestation [1]. A defect in cell-mediated immunity makes AIDS patients susceptible to op-portunistic infections and certain tumors. The organisms that have been reported in the literature to cause either encepha-litis, brain abscess, or meningitis in AIDS patients include Toxoplasma gondii, CMV, papova virus, C. albicans, Myco-bacterium tuberculosis, C. neoformans, and various pyogenic bacteria [1–13]. The tumors include either primary lymphoma or Kaposi sarcoma. Opportunistic infections predominate, with Toxoplasma encephalitis being the most frequent disease affecting the CNS, especially in the Haitian with AIDS [1, 3, 4, 6].

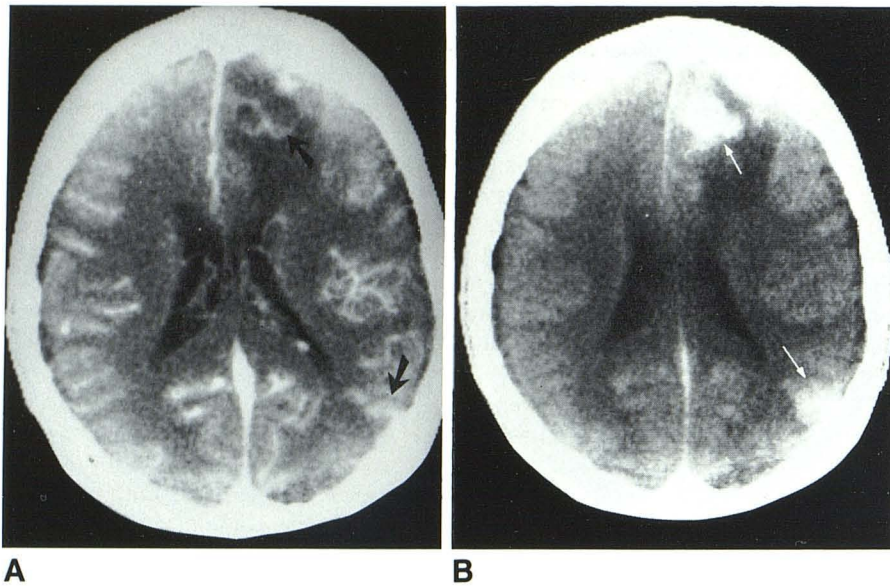CT is very sensitive in detecting the lesions in the brain

Fig. 12.—Homosexual with *Toxoplasma* encephalitis. **A**, High-resolution IDD CT scan. Two areas of abnormal enhancement (*arrows*). **B**, DDD scan. With time delay, more contrast material accumulated in these two lesions, making them denser and larger (*arrows*). Two other sites of involvement were identified in left temporal lobe on DDD study (not shown).
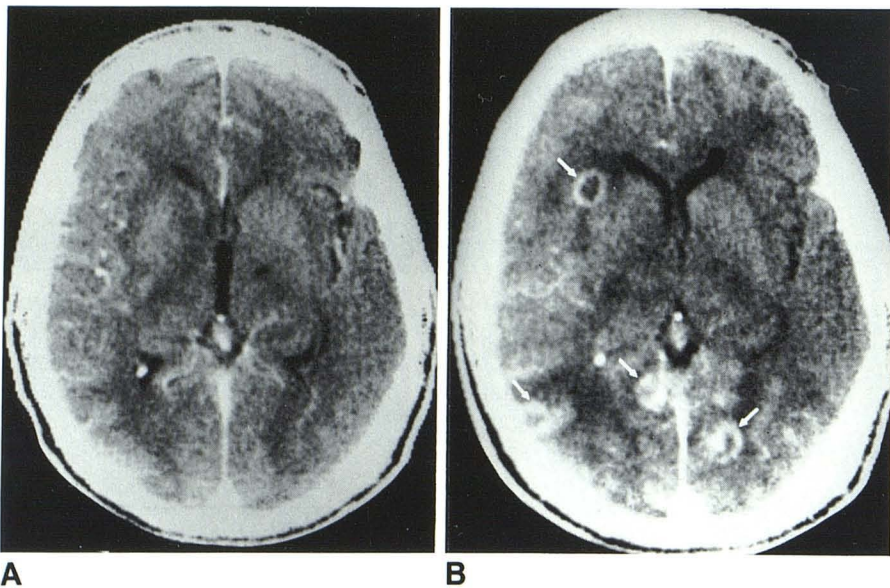

Fig. 13.—Homosexual with *Toxoplasma* encephalitis. Multiple ring-enhancing lesions seen only on DDD scan (*arrows*, **B**) and not on IDD (**A**) study.

caused by *Toxoplasma gondii*. Scans were abnormal in all the cases in our series and in most of the cases described in the literature [3, 4, 6, 7–10]. A characteristic pattern often seen in this disease consists of multiple ring and/or nodular enhancing lesions at the corticomedullary junction and the basal ganglia with edema and mass effect. This pattern is not pathognomonic. Single lesions are occasionally seen as well as lesions at less typical sites such as the cerebellum and brainstem. Uptake of contrast material is not always demonstrated. This lack of enhancement, however, is rare and is probably related to the use of small volumes of contrast material.

Our experience and that in the literature indicates that CT is effective in monitoring patients being treated for *Toxoplasma* encephalitis [3, 4, 6, 8, 9]. Serial CT scans show progressive diminution of enhancing lesions, edema, and mass effect, beginning within 2–4 weeks after institution of therapy. With continuous and long-term medical therapy, healing does occur, as evidenced by the return to normal of some scans and the appearance on other scans of areas of encephalomalacia. Due to the persistent cellular immune defect in AIDS patients, however, this healing process may be reversed when medical therapy is interrupted. When this occurs, CT is a reliable indicator of recurrence. CT changes

in recurrent disease include the reappearance of contrast uptake at sites of prior enhancement and the development of edema, mass effect, and often new lesions. These CT findings are virtually diagnostic and would appear to make rebiopsy unnecessary.

As for other infections that involve the brain parenchyma in AIDS patients, they are usually detected by CT if they are seen as focal masses. A case in point was our patient with a tuberculous abscess as shown by characteristic pathology [19]. This lesion was seen on CT as a large ring-enhancing mass. Although this abnormality is not specific for a tuberculous abscess, it localized the lesion for aspiration biopsy. Brain abscess caused by *C. albicans* is another infection that CT has been reported to detect [3, 4]. Its appearance on CT was that of a ring-enhancing lesion [3].

CT seems to be less sensitive in the early detection of infections that are diffuse and infiltrative in nature such as CMV encephalitis and PML. It has been our experience in autopsied AIDS patients with CMV encephalitis that the lesions are more numerous and widely disseminated than in the iatrogenically immunosuppressed patient. Pathologically, most of the lesions are below the threshold of unaided eye resolution. These microscopic foci of multifocal nodules and intranuclear viral inclusions may be found in the cerebrum, brainstem, and cerebellum as well as the subependymal regions and may affect both the white and gray matter [20]. CT scans may be negative, especially early in this disease. When positive, the CT picture may be quite variable because this virus can affect so many different parts of the CNS. In our series we saw both white-matter disease manifested on CT by diffuse hypodensities and gray-matter disease manifested by an enhancing cortical nodule. More recently, we have also seen subependymal involvement evident on CT by diffuse enhancement of the borders of the lateral ventricles and confirmed at autopsy. In the literature a small ring-enhancing lesion has been demonstrated on CT in the cerebellum [4]. Like others, we sometimes have found CMV as a second disease process in AIDS patients [4].

While PML, caused by the papova virus, has a more typical CT pattern, it also may be difficult to detect early in its course. Initial CT scans may be negative. When positive, CT characteristically shows central and/or convolutional white-matter low densities often in a parietooccipital location and with negligible mass effect [6, 21]. It has also been reported in the cerebellum in AIDS patients where it appeared as a large low-density lesion [11]. Diseases such as CMV and other viral encephalitides and noninfectious lesions may, of course, simulate the CT appearance of PML.

In contradistinction to parenchymal infection, meningitis infrequently appears as an abnormality on CT. When positive, findings such as communicating hydrocephalus, dilated cerebrospinal fluid (CSF) spaces, and meningeal enhancement are nonspecific; parenchymal involvement is seen infrequently [3, 4, 6]. CSF analysis is, however, usually diagnostic. The value of CT then lies in excluding any superimposed parenchymal diseases that might make lumbar puncture dangerous and in detecting communicating hydrocephalus, which might require shunting.

In regards to CNS tumors that affect AIDS patients, CT seems to be quite sensitive in detecting them since they usually are seen as focal parenchymal mass lesions. Both large-cell and Burkittlike lymphomas have been seen [1, 4, 6, 11]. On CT these lymphomas have usually been described as ring-enhancing periventricular lesions and have been contrasted against the lymphomas occurring in non-AIDS patients, which have been noted to enhance homogeneously in a periventricular and/or parenchymal location [6, 22]. We believe, however, that the types of enhancement are influenced largely by the amount of contrast material injected and the length of delays in scanning time. Filling-in of ring-enhancing lesions occurred on DDD scans in both of our lymphoma cases.

Kaposi sarcoma is the other CNS tumor reported in AIDS patients; it has been seen in the frontal lobe where it appeared on CT as an enhancing lesion [3]. A tendency for this tumor to hemorrhage has been found at autopsy [3]. Obviously the paucity of reports makes it impossible to describe any characteristic pathologic and CT findings in this tumor.

Although CT is sensitive to tumor detection, it is not specific. It cannot differentiate neoplasm from a variety of CNS infections. Since treatment techniques are so different in tumor and in infection, early biopsy would seem important to establish a specific diagnosis.

Early biopsy would also seem important in cases with a high clinical suspicion of infection because of the lack of specificity of CT findings in the various types of infection and because of the possibility of coexistent diseases. Even infections having more characteristic CT patterns cannot be diagnosed pathognomonically by CT. The critical role of CT then in AIDS patients presenting for the first time with neurologic symptoms is to detect lesions and to localize them for possible biopsy.

The procedure of choice for detecting and localizing CNS lesions in AIDS patients is the 1 hr DDD contrast scan if there are no contraindications to its use [4, 23]. After an unenhanced CT study, the DDD study should be performed because it increases the sensitivity of CT. It detects lesions that may not be seen on either a single-dose or an IDD study and that may be more biopsy-accessible. It also enhances the demonstration of lesions, making them appear denser and larger.

Should an initial DDD study be normal and neurologic symptoms persist or worsen, follow-up plain and DDD scans should be obtained. Also, a second imaging method, magnetic resonance imaging, should be used since it has the potential for being even more sensitive than CT in detecting parenchymal lesions.

Follow-up CT scans in patients under treatment should be obtained to monitor the effectiveness of medical therapy, to detect medical noncompliance, and to discern superimposed disease. The studies should be performed with similar DDD techniques so that accurate comparisons can be made with earlier studies.

In conclusion, we recommend that CT with the DDD technique be used to evaluate and follow all AIDS patients with neurologic symptoms and/or signs because of its sensitivity in disease detection.

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