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Central nervous system tuberculosis in HIV-infected patients: clinical and radiographic findings.

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Central Nervous System Tuberculosis in HIV-Infected Patients: Clinical and Radiographic Findings

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PURPOSE: To characterize the radiographic findings on neuroimaging of 25 human immunodeficiency virus (HIV)-seropositive patients with proved central nervous system tuberculosis and to correlate those findings with clinical data. **METHODS:** Twenty-five HIV-seropositive patients with central nervous system tuberculosis were identified, and their imaging studies (CT and, in some cases, MR) and medical records were reviewed. The diagnosis of central nervous system tuberculosis was based on cerebrospinal fluid culture (n = 20), biopsy (n = 4), and/or autopsy (n = 5), with a clinical diagnosis of central nervous system tuberculosis in one additional patient. Results also were correlated with CD4 counts and chest x-ray findings. **RESULTS:** Nine (36%) of 25 patients demonstrated meningeal enhancement. Eleven (44%) of 25 demonstrated enhancing parenchymal lesions; 6 patients had tuberculomata, and 5 had tuberculous abscesses. Communicating hydrocephalus was present in 8 (32%) of 25, and infarction was seen in 9 (36%) of 25. Fifteen of 23 chest x-rays were suggestive of pulmonary tuberculosis. Mean CD4 count was 162. Nine (38%) of 24 patients had a history of pulmonary tuberculosis, and 5 (21%) of 24 had no history of tuberculosis or any other opportunistic infection. Overall mortality was 79%. **CONCLUSION:** Central nervous system tuberculosis has a very high mortality among HIV-infected patients. Because cerebrospinal fluid cultures can take 6 to 8 weeks, the neuroradiologist can play a critical role in patient treatment by suggesting the correct diagnosis based on characteristic imaging findings. Radiographic clues include multiloculated abscess, cisternal enhancement, basal ganglia infarction, and communicating hydrocephalus, which are not findings associated with the more commonly encountered central nervous system lymphoma or toxoplasma encephalitis. Central nervous system tuberculosis may be the initial presentation of acquired immunodeficiency syndrome. In patients with suspected central nervous system tuberculosis, chest x-ray may provide additional support for the diagnosis of tuberculosis.

Index terms: Acquired immunodeficiency syndrome (AIDS); Tuberculosis

The year 1993 was the first in which both pulmonary and extrapulmonary tuberculosis were included as acquired immunodeficiency syndrome (AIDS) indicator conditions for AIDS case definition. This underscores the importance of tuberculosis as a significant infection in the human immunodeficiency virus (HIV)-positive population. In the United States, tuberculosis has been on the rise since 1986 (1–3), with an increase in extrapulmonary manifestations as well (4). Approximately 30% of patients with tuberculosis are HIV seropositive (5, 6). Conversely, 5% to 9% of all AIDS patients have tuberculosis (all sites included) (2, 7, 8). Central nervous system (CNS) tuberculosis occurs in 2% to 5% of all patients with tuberculosis (9, 10) and in 10% of those with AIDS-related tuberculosis (9, 11).

Although toxoplasma encephalitis and CNS lymphoma are more commonly encountered in HIV-infected patients, CNS tuberculosis is clearly a significant cause of cerebral infection. Because prompt diagnosis may result in earlier

treatment, recognition of this disorder by the radiologist can play a critical role in patient treatment. Thus, the purpose of our study was to examine the radiographic findings on neuro-imaging of HIV-infected patients with proved CNS tuberculosis and to correlate those findings with clinical data.

Materials and Methods

We identified 25 HIV-seropositive patients with documented CNS tuberculosis seen at our institution since 1989. Patients were identified via pathology records and/or positive cerebrospinal fluid (CSF) culture results. Patients who were not HIV seropositive were excluded from the study. Neuroimaging studies were available for review from 24 HIV-seropositive patients with proved CNS tuberculosis, and from one additional pediatric patient who had a clinical diagnosis of tuberculosis. Imaging studies and medical records of these 25 patients were reviewed. The imaging studies were examined by two neuroradiologists in an open retrospective evaluation. There were 20 male and 5 female subjects. Age ranged from 3 to 67 years, with a mean age of 35.5 years. All patients (N = 25) had noncontrast cranial computed tomography (CT) scans, and 21 patients had contrast CT scans. Two patients had noncontrast magnetic resonance (MR) scans, and 3 patients had MR scans with gadolinium. MR exams were obtained on 1.0-T or 1.5-T units. Noncontrast MR studies included T1 weighted (500–900/20–28/1 [repetition time/echo time/excitations]) coronal or sagittal spin-echo images and dual-echo (2000–2500/20–40/60–100) axial spin-echo images. Contrast MR scans were obtained with spin-echo T1-weighted studies in three planes using gadolinium (0.1 mmol/kg) intravenously. Chest x-rays were available on 23 of the patients. CD4 counts were available on 22 patients.

The pathologic diagnosis of central nervous system tuberculosis was determined for 24 of 25 patients in the following manner:

Method	No. of Patients
CSF culture	16
Culture and autopsy	2
Culture and biopsy	2
Biopsy	1
Autopsy	2
Culture/autopsy/biopsy	1

In all 24 cases, CNS infection was attributable to mycobacterium tuberculosis. The remaining patient, a 3-year-old, was diagnosed on the basis of clinical grounds and radiographic findings. CSF studies in this child revealed a glucose of 38.5 mg/dL, a protein of 188 mg/dL, and a CSF white blood cell count of 143/mm^3 with 31% polymorphonuclear leukocytes and 69% monocytes. Chest x-ray revealed a miliary pattern. This patient demonstrated rapid clinical and laboratory improvement after treatment with antituberculosis medication.

Results

Imaging

The following neuroimaging findings occurred with the frequency shown in these 25 HIV-infected patients with CNS tuberculosis:

Radiographic Finding	No. of Patients
Cisternal/meningeal enhancement	9 (36%)
Hydrocephalus	8 (32%)
Enhancing parenchymal lesions	11 (44%)
Infarction	9 (36%)

The basal cisterns were a favored site of meningeal enhancement; of the 9 patients with meningeal enhancement, 6 (24% of the total study population) demonstrated enhancement of the basal cisterns. Five of the 8 cases of hydrocephalus were associated with meningeal enhancement (Fig 1). In 2 patients, hydrocephalus was the sole finding. The 11 cases with focal enhancing parenchymal lesions included 5 cases of cerebral tuberculous abscess and 6 cases of granulomata (tuberculomas). The enhancing lesions were defined as granulomata versus abscess based on their radiographic appearance. All five abscesses were solitary lesions with ring enhancement, mass effect, and edema. Three of the five abscess lesions were more than 3 cm in size, three were multiloculated, and three were located in the posterior fossa. Four of the five abscesses were confirmed at surgery.

Tuberculomas had a different radiographic appearance. Tuberculomas were noted in 6 (24%) of 25 patients and were 1 cm or less in size. Both nodular (n = 4) and ring-enhancing (n = 2) patterns were observed (Fig 2). The tuberculomas demonstrated little or no mass effect and little edema (Fig 3). These lesions were either multiple (n = 4) or solitary (n = 2). Three of the six patients with tuberculomas had only supratentorial lesions, one patient had both supratentorial and infratentorial granulomata, and two patients had only infratentorial tuberculomas. Of the three patients with infratentorial lesions, two patients had lesions of the vermis, and two patients had lesions at the periphery of the brainstem, near an adjacent cistern. Of the four patients with supratentorial lesions, three had lesions that were adjacent to CSF spaces, either periventricular or perisylvian in location. Two of the four patients with supratentorial lesions had involvement of the corticomedullary junction, and one of the four pa-
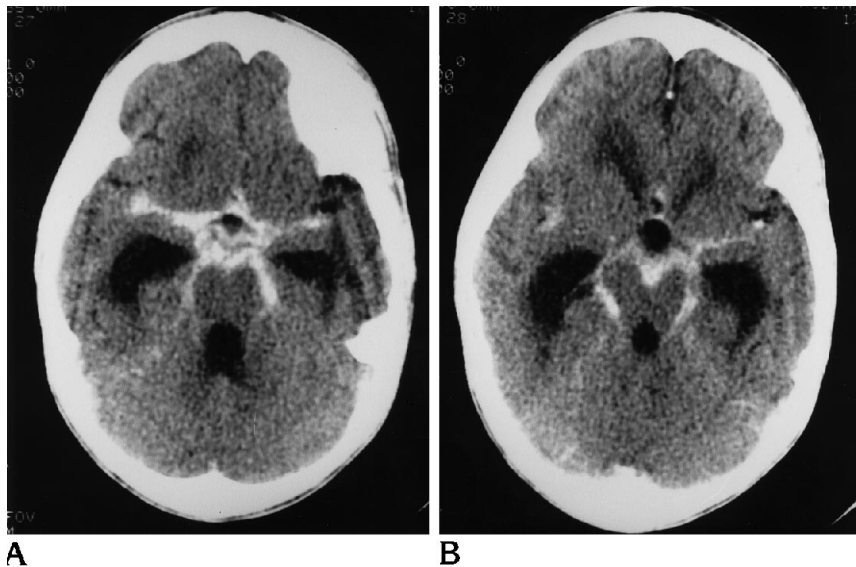
Fig 1. Meningeal enhancement/hydrocephalus.

A, Postcontrast axial computed tomography of a 6-year-old HIV-positive child with culture-proved CNS tuberculosis. There is intense enhancement of the basal cisterns. Enlargement of the fourth ventricle and temporal horns is compatible with communicating hydrocephalus.

B, Axial CT of the same patient at the level of the sylvian fissures. Again noted is enhancement of basal cisterns as well as ventricular enlargement and meningeal enhancement within the sylvian fissures, right greater than left.

tients had a lesion of the basal ganglia. One case of tuberculomata was confirmed at autopsy. The other five cases did not undergo surgery or autopsy. Although coexistent infection could not be completely excluded, these lesions were presumed to be tuberculous in origin, because CSF culture results were positive for tuberculosis at the time these patients had abnormal imaging studies. Three of the six tuberculoma cases were associated with meningeal enhancement, and three were associated with hydrocephalus.

Cerebral infarctions were present in 9 (36%) of 25 patients. These infarcts were usually small, affecting primarily the small perforating vessels arising from the middle cerebral artery (Fig 4). These infarcts were often multiple (6 of 9). Seven of the nine patients with infarcts had involvement of the basal ganglia (Fig 5).

Chest x-rays were available for 23 patients and findings were abnormal in 20 (87%) of these 23 patients. However, five of those abnormal chest x-rays demonstrated interstitial disease, suggestive of pneumocystis carinii pneumonia rather than of tuberculosis. Excluding those five cases compatible with pneumocystis carinii pneumonia, 15 (65%) of the 23 chest x-rays were abnormal. Findings included infiltrates (n = 11), adenopathy (n = 4), and pleural effusions (n = 4) (Fig 6).
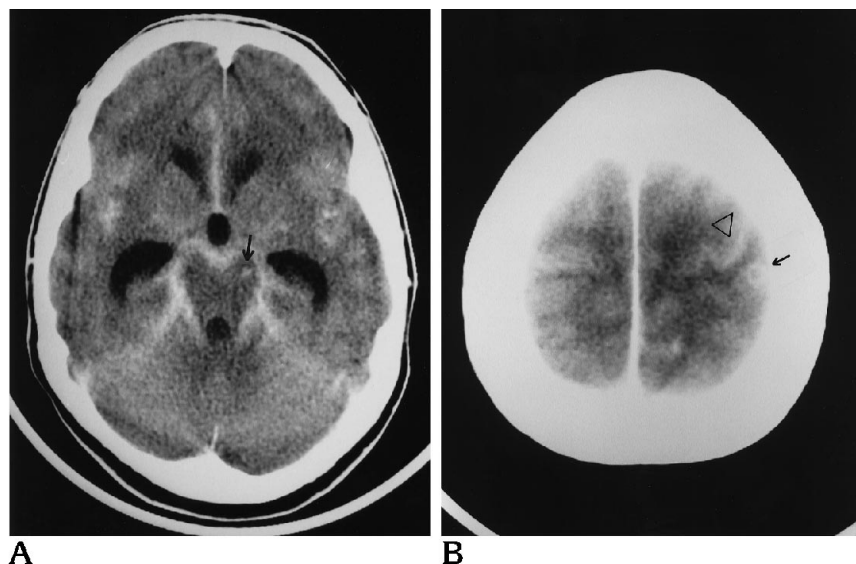

Fig 2. CNS tuberculoma: nodular versus ring enhancement.

A, Postcontrast axial CT of a 42-year-old HIV-positive woman with culture-proved CNS tuberculosis. Striking enhancement of the basal cisterns is noted as is enhancement within the sylvian fissures bilaterally. Enlargement of the lateral, third, and fourth ventricles is noted, compatible with communicating hydrocephalus. A focus of nodular enhancement is seen in the left cerebral peduncle, without mass effect or edema, consistent with a tuberculoma (arrow).

B, Axial postcontrast CT reveals a small, ring-enhancing lesion in the left parietal region (arrow). A similar lesion also was present in the right temporal lobe. Note also meningeal enhancement (arrowhead).
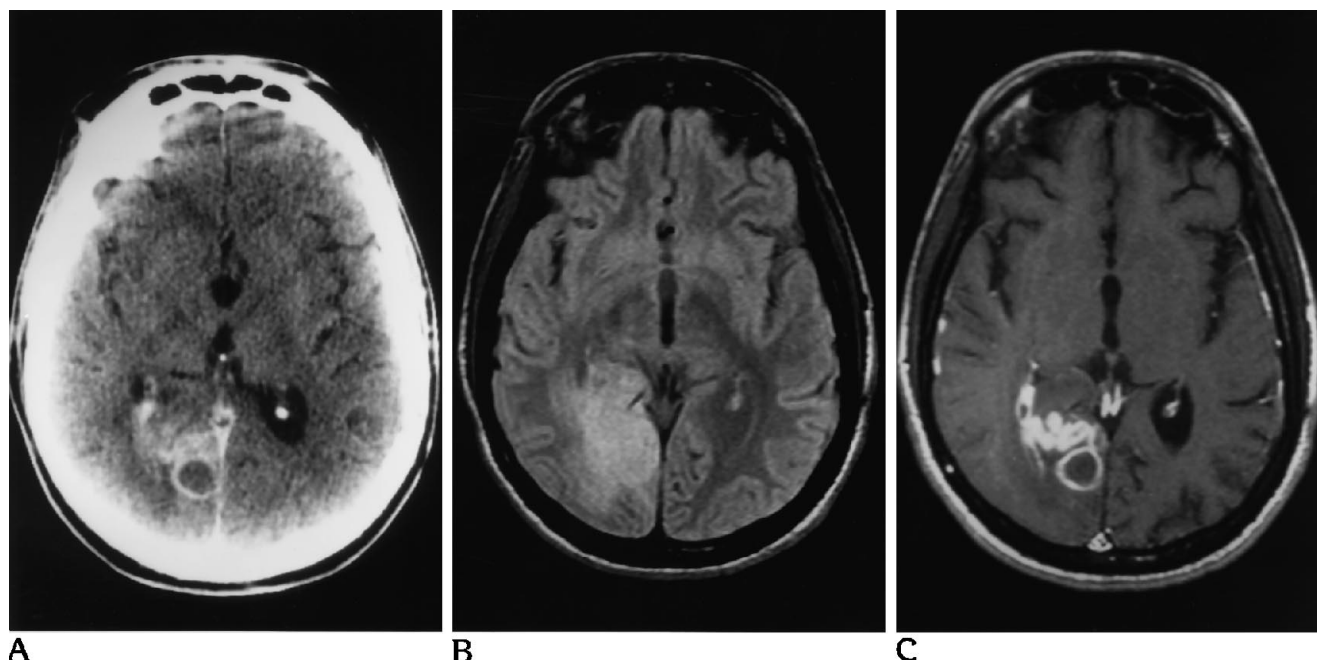
Fig 3. Tuberculomata versus tuberculous abscess.

A, Axial postcontrast CT of a 31-year-old patient with AIDS and a biopsy-proved tuberculous abscess. CSF cultures were also positive for tuberculosis. A multiloculated mass lesion is seen in the right occipital lobe with compression of the right atrium and enhancement extending to the subependymal region of the right atrium.

B, Proton density–weighted(2400/20) axial image of the same patient reveals a large area of increased signal in the right occipital region with local mass effect on the right atrium.

C, Contrast-enhanced (0.1 mmol/kg gadopentate dimeglumine) T1-weighted (600/20) axial MR at a similar level again demonstrates the multiloculated appearance of the lesion with extension to the ependyma of the right lateral ventricle. Note surrounding edema and sulcal effacement.

D, T2-weighted (730/20) axial MR with gadolinium (0.1 mmol/kg) in a 36-year-old HIV-positive man reveals two areas of nodular enhancement (*arrows*) compatible with tuberculous granulomata. This patient had both supratentorial and infratentorial lesions. Note the paucity of mass effect/edema. These granulomata were confirmed at autopsy.

Clinical

Clinical findings were as follows in these 25 patients:

Sign/Symptom	Patients, %
Fever	54
Headache	50
Altered mental status	42
Cough	33.3
Nausea/vomiting	21
Meningismus	17
Shortness of breath	13
Seizures	4

Information on drug susceptibility was available for 19 patients. Seven (37%) of 19 patients had resistance to at least one of the medications tested. The drugs tested included isoniazid, rifampin, ethambutol, and streptomycin. The mean CD4 count was 162. Information concerning prior opportunistic infection was available for 24 of 25 patients. Nine (38%) of the 24 patients had a history of pulmonary tuberculosis. Three (13%) of the 24 had a history of extrapulmonary (non-CNS) tuberculosis. In 5 patients, CNS and pulmonary tuberculosis were simultaneous in onset. Six patients had prior pneumocystis carinii pneumonia infection, and one patient had a history of Kaposi sarcoma. Five (21%) patients had *no* prior opportunistic
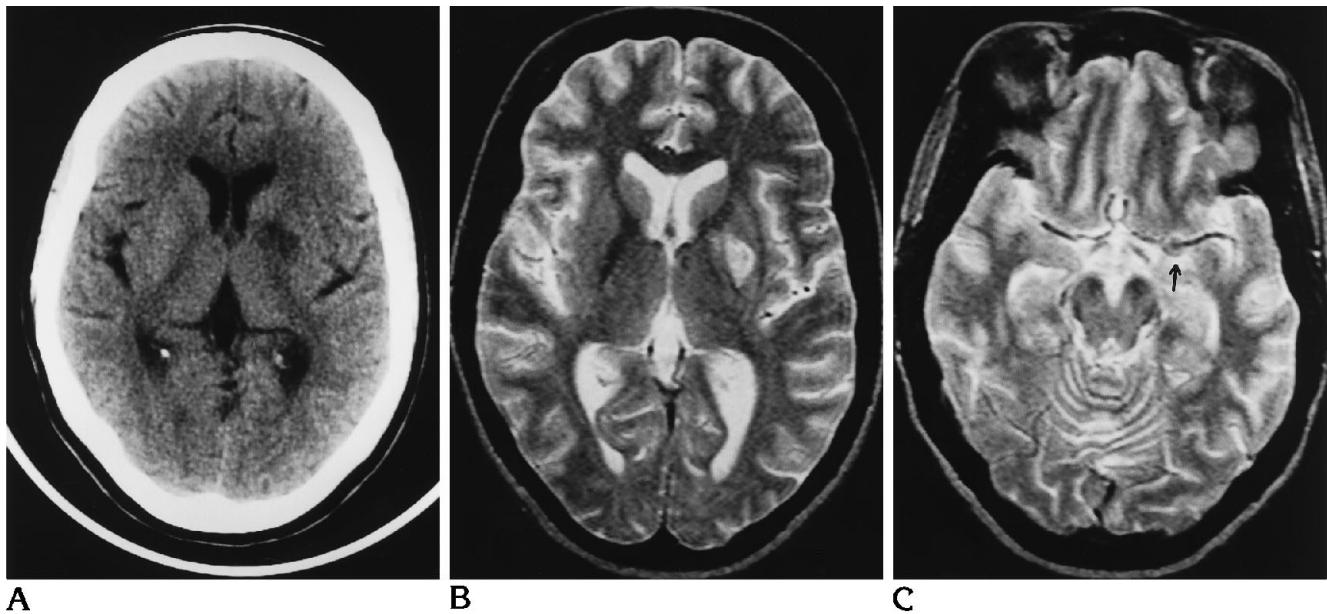
A   B   C

Fig 4. Basal ganglia infarction.

A, Noncontrast axial CT of this 43-year-old woman with CSF cultures positive for mycobacterium tuberculosis. Hypodensity is seen within the left globus pallidus and putamen.

B, T2-weighted (2400/80) axial MR reveals increased signal in the left basal ganglia.

C, T2-weighted (2400/80) MR demonstrates a basilar exudate encircling the proximal left middle cerebral artery (*arrow*).

infections and no evidence of pulmonary tuberculosis, and thus CNS tuberculosis was the first manifestation of AIDS in those patients.

Overall mortality was high, at 79%. A 67% mortality was seen within 2 months of diagnosis, and a 71% mortality was noted by 1 year after diagnosis. The mortality was directly attributable to tuberculosis in 90% of those patients who died.

Discussion

There has been a resurgence of tuberculosis in the United States, particularly among HIV-infected patients (1–3). CNS involvement is present in approximately 10% of those with AIDS-related tuberculosis (9, 11), but in only 2% to 5% of those with non–AIDS-related tuberculosis (9, 10). It is therefore important for the radiologist to recognize this disease so that appropriate therapy may be promptly instituted.

Meningeal enhancement was observed in 36% of patients in this study. The cause of this enhancement is a basal leptomeningitis. Two different mechanisms have been proposed for the pathogenesis of tuberculosis meningitis (12–14). The first theory suggests rupture of subependymal or subpial granulomata into the CSF. The other proposed mechanism involves penetration of the walls of meningeal vessels by hematogenous spread, most often from a pulmonary or gastrointestinal source. A thick gelatinous exudate is found in the basal cisterns.

Communicating hydrocephalus is a common sequela of tuberculous meningitis. Five of our eight cases of hydrocephalus also were associated with meningeal enhancement. The hydrocephalus is primarily a result of obstruction of the basal cisterns by the dense inflammatory exudate. Occasionally, the hydrocephalus may be of the obstructive type, attributable to a focal parenchymal lesion with mass effect or attributable to entrapment of a ventricle by granulomatous ependymitis (13–15).

Tuberculomas were seen in 24% of our patients. These granulomata may result from extension of CSF infection into adjacent parenchyma via cortical veins or small penetrating arteries or may result from hematogenous spread of systemic disease (15). In our study, lesions were seen both at the corticomedullary junction as well as in periventricular and pericisternal locations. Pathologically, these tuberculomas are composed of a central zone of solid caseation necrosis, surrounded by a capsule of collagenous tissue, epitheloid cells, multinucleated giant cells, and mononuclear inflammatory cells. Few tubercle bacilli are seen on smears
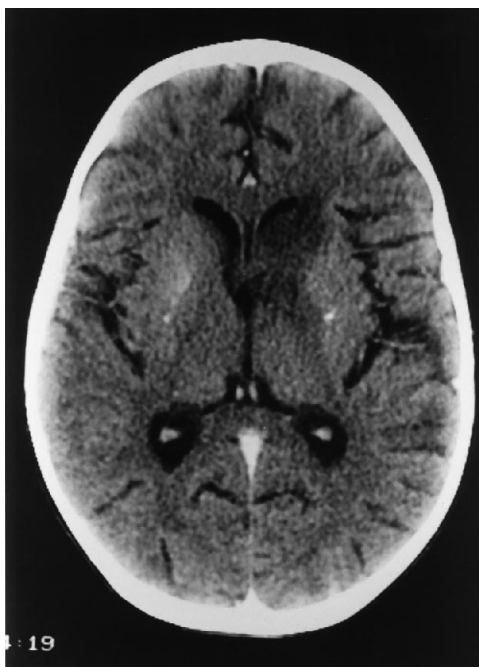
Fig 5. Infarction in a pediatric AIDS patient. Contrast-enhanced CT scan of a 3-year-old AIDS patient with CNS tuberculosis. Calcification is seen in the basal ganglia bilaterally, which is often present in HIV-infected children. The calcification is thought to be attributable to a calcific vasopathy and is a direct result of HIV infection. In addition, there is hypodensity involving the left globus pallidus, the caudate head, and the anterior limb of the left internal capsule. These infarctions were presumed to be a result of CNS tuberculosis. Rapid clinical improvement was evident after treatment with antituberculous therapy.

(16–18). Tuberculomas may be found in the cerebrum, cerebellum, subarachnoid space, subdural space, or epidural space. The majority are said to be supratentorial (19, 20). In our study, three of six patients had supratentorial tuberculomas, one patient had both supratentorial and infratentorial tuberculomas, and two patients had only infratentorial tuberculomas. Parenchymal disease may occur with or without coexistent meningitis (21), as was seen in our study.

Tuberculomas may be solitary or multiple. Four of our six cases demonstrated multiplicity. These lesions may show nodular enhancement, seen in four of our six cases, or may reveal ring enhancement, seen in two of the six cases. All our tuberculomas were 1 cm or smaller and displayed little mass effect and/or edema.

The MR appearance of a granuloma appears to differ significantly from that of a tuberculous abscess. Early tuberculomas appear hypointense on T2-weighted images, and mature tuberculomas contain a hypointense center surrounded by an isointense capsule on T2-weighted sequences (26). This is attributable to the solid caseation necrosis present in tuberculomata. Central liquefaction with pus formation appears hyperintense on T2-weighted images (26), suggesting abscess formation. However, some tuberculomas (27) may show a bright central core on T2-weighted images, associated with a low-intensity, well-defined rim. Thus signal characteristics alone may not distinguish tuberculoma from abscess; however, the presence of significant edema and mass effect favors abscess. The tuberculomas may be indistinguishable from some lesions of toxoplasmosis or CNS lymphoma. However, the associated findings of cisternal enhancement, basal ganglia infarction, and/or communicating hydrocephalus should alert the radiologist to the possibility of tuberculous infection, because these associated findings are not usually encountered in toxoplasma encephalitis or primary CNS lymphoma.

Tuberculous abscess is a rare complication of CNS tuberculosis in the general population (14), seen in 4% to 8% of patients with CNS tuberculosis without HIV infection (22, 23). Tuberculous abscess was present in 20% of patients in this study, and thus tuberculous abscess may be one form of CNS tuberculosis that occurs more commonly in HIV-infected patients. In contrast to the solid caseation observed in tuberculomas, with few tubercle bacilli, the center of the abscess is semiliquid pus teeming with tubercle bacilli (16, 17). The wall of a tuberculous abscess lacks the giant cell epitheloid reaction of a tuberculosis granuloma (24). Further distinguishing characteristics include size and multiplicity. Abscesses tend to be larger than tuberculomas and have a more accelerated clinical course (25). Three of our five cases of abscess were over 3 cm, and three of five were multiloculated. Our tuberculomas were all 1 cm or smaller, without multiloculation. Although tuberculomas are often multiple, all our abscess lesions were solitary. Ring enhancement was observed in all five cases of tuberculous abscess, similar to the appearance of bacterial abscess.

Cerebral infarction was seen in 36% of our 25 patients. Infarction is a common complication of CNS tuberculosis, a result of spasm and thrombosis as arterial vessels course through the gelatinous basal exudate. The small perforating branches supplying the basal ganglia are
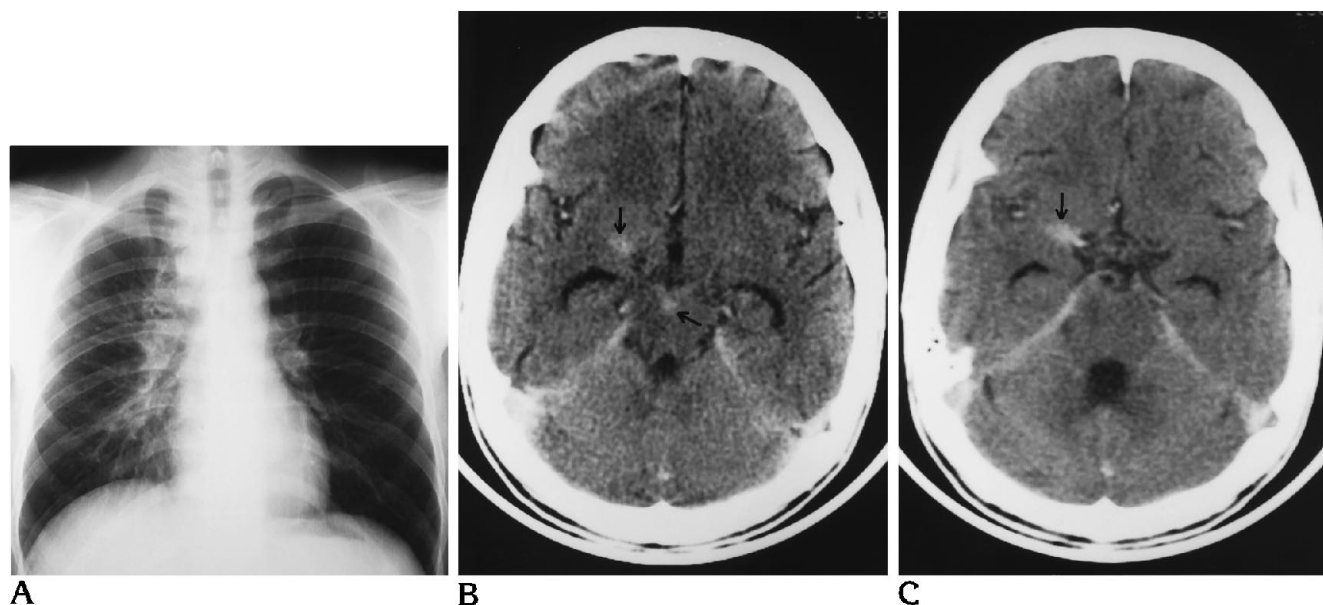
Fig 6. Hilar and mediastinal adenopathy in a patient with disseminated tuberculosis.

A, Chest x-ray reveals bilateral hilar adenopathy, right greater than left, as well as adenopathy of the right superior mediastinum.

B and *C*, Postcontrast axial CT images reveal meningeal enhancement involving the cistern of the right middle cerebral artery as well as the interpeduncular cistern (*arrows*). Cerebrospinal fluid cultures were positive for mycobacterium tuberculosis. Autopsy revealed disseminated tuberculosis with meningeal involvement.

those most often affected (14, 28), and were involved in seven of the nine patients with infarcts.

A radiographic study of 35 AIDS patients with proved intracranial tuberculosis (20) showed findings similar to ours, although hydrocephalus was more common in that study (51% versus 32%). A number of other authors have investigated the radiographic findings associated with CNS tuberculosis in both HIV- and non-HIV–infected patients (see Table). It is difficult to draw substantive conclusions from these disparate results. It is possible that the disparities evident from the Table may be related to regional differences in patient population.

Chest x-ray findings were abnormal in over 65% of our cases. Pleural effusion, adenopathy, and cavitary infiltrates may suggest tuberculous

infection and thus provide supportive evidence in cases of suspected CNS tuberculosis. Thus, if neuroimaging findings are suggestive of CNS tuberculosis, a chest x-ray should be obtained, because the findings on chest x-ray may further support the diagnosis.

CSF cultures for tuberculosis can take 6 to 8 weeks, and thus early diagnosis by the radiologist can play a critical role in the treatment of these patients. CSF studies in tuberculous meningitis typically reveal a low glucose and elevated protein. If tuberculosis is suspected, adenosine deaminase levels also can be determined, which are elevated in patients with tuberculosis meningitis. Even more helpful as a confirmatory test is the polymerase chain reaction, which can speed the diagnosis of tuberculosis from several weeks to 2 days. Clues to the

Neuroimaging findings in central nervous system tuberculosis

Author	HIV+ Study Population?	No. of Study Patients	Meningeal Disease	Hydrocephalus	Infarcts	Enhancing Parenchymal Lesions
Berenguer et al (9)	no	16	6%	44%	6%	0%
Jinkins (22)	no	80	29%	40%	12.5%	83%
Chang et al (23)	no	26	39%	58%	42%	23+%
Bhargava et al (29)	no	60	82%	83%	28%	10%
Berenguer et al (9)	yes	26	23%	42%	27%	15%
Villoria et al (20)	yes	35	45%	51%	23%	37%
Present study	yes	25	36%	32%	36%	44%

radiographic diagnosis of CNS tuberculosis (in HIV-positive patients) established by our study include a multiloculated abscess, cisternal enhancement, infarction of the basal ganglia, and communicating hydrocephalus. Meningeal disease and infarction also are commonly seen in neurosyphilis; however, correlation with serum and CSF studies as well as chest x-ray findings should differentiate between these two entities. The above radiographic clues are not associated with toxoplasma encephalitis, although this infection is far more common than CNS tuberculosis in AIDS patients. Toxoplasma encephalitis does not present as a multiloculated mass, and thus the finding of such a lesion in an AIDS patient should suggest the diagnosis of CNS tuberculosis. Although a multiloculated mass could simulate a necrotic CNS lymphoma, and meningeal enhancement also may be associated with lymphoma, infarction of the basal ganglia and communicating hydrocephalus and radiographic clues are not usually associated with primary CNS lymphoma, a common neoplasm in these patients.

Latent tuberculosis often manifests early in HIV disease (30, 31). Thus, CNS tuberculosis may be the initial presentation of AIDS, as was seen in as many as 42% of our patients (five patients without any prior opportunistic infection, and five patients with synchronous CNS and pulmonary tuberculosis). Consequently, if CNS tuberculosis is documented, prompt evaluation of HIV status should be undertaken.

CNS tuberculosis has a very high mortality (79%) among HIV-infected patients. This is multifactorial and includes weakened immunity, general debilitation, delayed diagnosis, and morbidity/mortality from concommittant diseases. In addition, a significant percentage of patients demonstrate resistance to at least one of the drugs (isoniazid, ethambutol, rifampin, streptomycin) commonly used to treat tuberculosis (32). This is supported by our results, with 37% of patients tested revealing resistance to at least one drug. This resistance often results from transmission of resistant organisms from patients with tuberculosis who have received inadequate or inappropriate therapy (33). Inadequate therapy may be a result of noncompliance, whereas inappropriate therapy may result from treatment with less than the recommended four-drug regimen or early cessation of medical therapy. Current Centers for Disease Control recommendations for initial treatment of tuberculosis suggest a four-drug regimen of isoniazid, rifampin, pyrazinamide, and either streptomycin or ethambutol (34) for 2 months. Drug susceptibility testing should be performed on the first positive tuberculosis isolate. When drug susceptibility results are available, treatment should be altered as appropriate. In HIV-infected patients, this treatment regimen should continue for a total of 9 months (34).

Acknowledgment

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