Neurosyphilis in HIV-Positive and HIV-Negative Patients: Neuroimaging Findings

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PURPOSE: To evaluate and describe the neuroimaging findings of patients with neurosyphilis. **METHODS:** The neuroimaging studies of 35 patients with documented neurosyphilis were reviewed. Diagnosis was established in 34 patients with cerebrospinal fluid for a Venereal Disease Research Laboratory test, complemented by autopsy in 1 and brain biopsy in 1. All patients had reactive fluorescent treponemal antibody tests with absorption in their sera. Imaging studies included plain and contrast-enhanced CT of the brain, plain and gadolinium-enhanced MR, MR angiography, and conventional angiography. Imaging findings were also correlated with the relevant pathologic findings at autopsy in three additional patients with neurosyphilis who did not have brain imaging studies. **RESULTS:** Of the 35 patients with imaging studies, 32 tested human immunodeficiency virus (HIV)-seropositive, and 3 were HIV-seronegative. Eleven (31%) of 35 patients had normal radiographic findings. Cerebral infarctions were seen in 8 (23%) of 35 patients, and nonspecific white matter lesions in 7 (20%) of 35. Cerebral gummas and extraaxial enhancement indicating meningitis were noted in 2 (6%) of 35 patients, respectively. Arteritis was demonstrated in 2 (50%) of 4 patients who underwent either MR angiography or conventional angiography. The 3 subjects who had autopsy but not imaging studies were found to have manifestations of meningovascular syphilis, including syphilitic leptomeningitis and an obliterative endarteritis. **CONCLUSION:** We conclude that findings of vascular occlusive disease manifested as infarction or arteritis, enhancing cortical lesions with or without adjacent meningeal enhancement, focal or diffuse extraaxial enhancement, and white matter disease, although nonspecific, in the proper clinical setting should prompt appropriate testing for neurosyphilis, a treatable disease, in patients with and without HIV infection.

Index terms: Syphilis; Acquired immunodeficiency syndrome (AIDS)

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Syphilis is caused by the spirochete *Treponema pallidum* and remains an important and frequently encountered sexually transmitted disease. During the era of acquired immunodeficiency syndrome (AIDS), there has been a dramatic rise in the number of cases of syphilis and a corresponding increase in the incidence of neurosyphilis. Invasion of the central nervous system by the organism can occur at any stage of syphilitic infection, and occurs in about 5% to 10% of untreated patients (1). However, neurosyphilis has been reported to develop in one third of patients who progress to late stages of the disease (2).

Syphilis is clearly recognized as one of the infectious complications of infection with the human immunodeficiency virus (HIV) (3). Evidence of an increased occurrence of syphilis with HIV infection has been previously reported (4). The association is not unexpected because AIDS and neurosyphilis are both sexually transmitted diseases. Early diagnosis is critical in the management of the disease in that it is easily treatable with appropriate antibiotics, but establishing the diagnosis is often difficult because most patients are asymptomatic or present with

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nonspecific symptoms (1). Some classic forms of the disease (general paresis of the insane and tabes dorsalis) are seen rarely in the era of antibiotics, and it appears that the expected progression of disease is altered by HIV infection (3). Identification of the more common radiologic appearances of neurosyphilis are important in order that appropriate clinical testing and treatment can be initiated. The purpose of this study is to characterize and define the neuroimaging findings of patients with neurosyphilis in a group of patients with and without HIV infection.

Subjects and Methods

The neuroimaging studies of 35 patients with documented neurosyphilis who were hospitalized during the period 1984 to 1994 were reviewed. Diagnosis was established in 34 by the presence of a reactive Venereal Disease Research Laboratory test in the cerebrospinal fluid in the absence of gross blood contamination. All patients had reactive fluorescent treponemal antibody tests with absorption in their sera. In 2 patients the diagnosis was complemented by autopsy (1 patient) and brain biopsy (1 patient). Nineteen patients underwent computed tomography (CT). CT studies were obtained on high-resolution CT scanners before and after the administration of intravenous contrast material (100 mL diatrizoate meglumine [reno-m-60], 282 mg iodine/mL). All studies were done with axial 5-mm- or 10-mm-thick sections. MR studies were performed on middle- or high-field units. Spin-echo MR sequences included T1-weighted (500-750/16-30/1-2 [repetition time/echo time/excitations]) images in the axial, coronal, or sagittal planes, and T2-weighted (2550-2650/20-80/1) images in the axial and coronal planes. Section thickness was 4.5 mm to 7.0 mm with an intersection gap of 1.0 to 1.5 mm and a 356 × 200 or 256 × 192 matrix. Twenty-two patients underwent plain MR studies, 11 of whom also had intravenous contrast-enhanced exams (gadopentate dimeglumine, 0.1 mmol/kg).

Four patients were studied angiographically, two by MR angiography and three by conventional angiography. One of these underwent both MR angiography and conventional angiography. MR angiography protocols included two-dimensional time-of-flight studies as follows: 39/9/1, 50° flip angle; 38 sections, each 1.8 mm thick with an intersection gap of 0.3 mm; 22-cm field of view; 256 × 256 matrix. Three-dimensional time-of-flight studies were also obtained; 45/710/1; 20° flip angle; 38 to 46 sections 1.0 mm thick; 25-cm field of view; 256 × 256 matrix. Source images were acquired in the axial and coronal planes with presaturation slabs adjacent to the area of interest and were supplemented with maximum intensity protection "angiograms." No intravenous gadolinium was used.

The imaging findings of the 35 patients in our series were correlated with autopsy material in three additional patients with neurosyphilis who had died without having brain imaging studies. The information obtained from these autopsied subjects was used to demonstrate several pathologic manifestations of neurosyphilis that are responsible for the radiologic appearances described. Clinical data from these three autopsied patients were not included in the clinical analysis of the 35 patients.

Results

Clinical and Imaging Data in 35 Patients

Of the 35 patients with imaging studies, 30 were men and 5 were women. Their ages ranged from 21 to 60 years, with a mean age of 38. Thirty-two patients tested HIV-seropositive and 3 were HIV-seronegative. Clinical findings that prompted the radiographic studies included altered mental status (7 [20%] of 35), hemiparesis (6 [17%] of 35), fever or elevated white blood cell count (5 [14%], of 35), seizures (3 [9%] of 35), and visual or gait disturbances (2 [6%] of 35). Ten patients were asymptomatic, and 9 of these were followed clinically and radiologically for neurologic complications of HIV infection as part of an ongoing longitudinal multidisciplinary study. In 8 patients clinical histories were unavailable. Five patients had clinical and/or radiologic evidence of coexistent diseases, and these included presumed progressive multifocal leukoencephalopathy, HIV encephalitis, and tuberculous meningitis.

CT, MR, and angiography results are summarized in the Table. Thirty-one percent (11 of 35) of the patients had normal radiographic findings. Ninety-one percent (10 of 11) of the pa-

CT, MR, and angiography findings in 35 patients with neurosyphilis

Findings	No. (%) of Patients
Normal	11 (31)
Atrophy	13 (37)
Mild	7 (54*)
Moderate	5 (38*)
Severe	1 (8*)
Cerebral infarction	8 (23)
Cortical/subcortical	4 (50†)
Brain stem	2 (25†)
Basal ganglia/thalamus	5 (63†)
White matter lesions	7 (20)
Gummas	2 (6)
Extraaxial enhancement	2 (6)
Arteritis	2 (50‡)

* Percentage of total number of patients with atrophy.
† Percentage of total number of patients with infarctions.
‡ Percentage of total number of patients who had angiography.

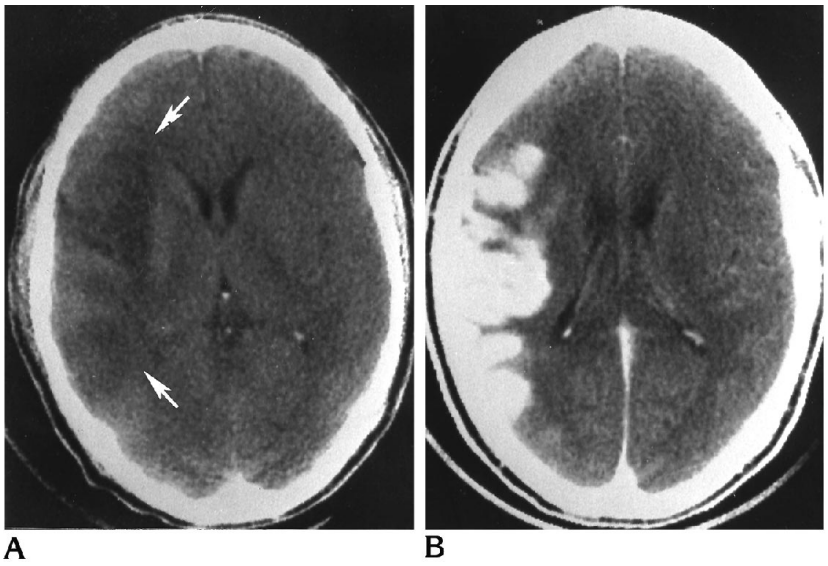


Fig 1. A 33-year-old HIV-positive woman with seizures and left hemiparesis.

A, Noncontrast brain CT image reveals a region of low attenuation in the distribution of the right middle cerebral artery that involves cortex and adjacent white matter, consistent with infarction (*arrows*).

B, On the contrast-enhanced image there is striking parenchymal enhancement indicating a subacute stage of the infarction.

tients with normal findings had asymptomatic neurosyphilis. The only asymptomatic patient with abnormal imaging findings had five to seven punctate (2 to 3 mm), bilateral deep white matter hyperintensities on T2-weighted images that were nonspecific.

Atrophy, either central or cortical (subjectively assessed by visual inspection, not volumetric analysis), was the most frequent abnormal finding, occurring in 37% (13 of 35) of the patients. Seven patients had mild cortical atrophy, 5 moderate central and/or cortical atrophy, and 1 severe central and cortical atrophy. This atrophy could not be considered specific for neurosyphilis because it could have been caused by the HIV infection itself.

Eight (23%) patients had cerebral infarctions. Four of these had moderate-to-large well-defined lesions involving both white matter and adjacent cortex, conforming to a vascular distribution. Mild mass effect was observed. These lesions were hypodense on CT and hyperintense on T2-weighted MR images. Three of these patients showed parenchymal gyriform enhancement correlating with the subacute stage of the infarction (Fig 1). Two patients had pontine lesions that were large, hyperintense on proton-density and T2-weighted images, and exhibited mild mass effect and minimal enhancement (Figs 2 and 3). One patient had a corresponding pontine hypodensity on CT. The remaining patients with infarctions had lesions localized to the basal ganglia and thalamus, that varied in size from small to large and were well demarcated. One patient

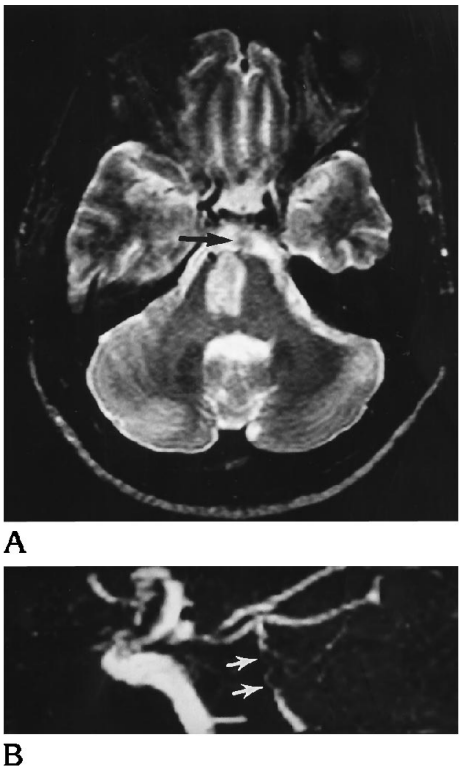


Fig 2. A 43-year-old HIV-positive man with left hemiparesis and seizures who had a brain stem infarction with basilar arteritis.

A, T2-weighted (2550/80/1) axial MR image shows a large area of increased signal in the pons. No normal flow void is present in the basilar artery. The area of low intensity anterior to the pons (*arrow*) represents pulsatile cerebrospinal fluid flow in the basilar cisterns in this patient with meningovascular syphilis. The contrast enhanced T1-weighted images (not shown) revealed mild pontine enhancement.

B, Sagittal projection image from 3-D time-of-flight MR angiography shows high-grade narrowing of the basilar artery (*arrows*).

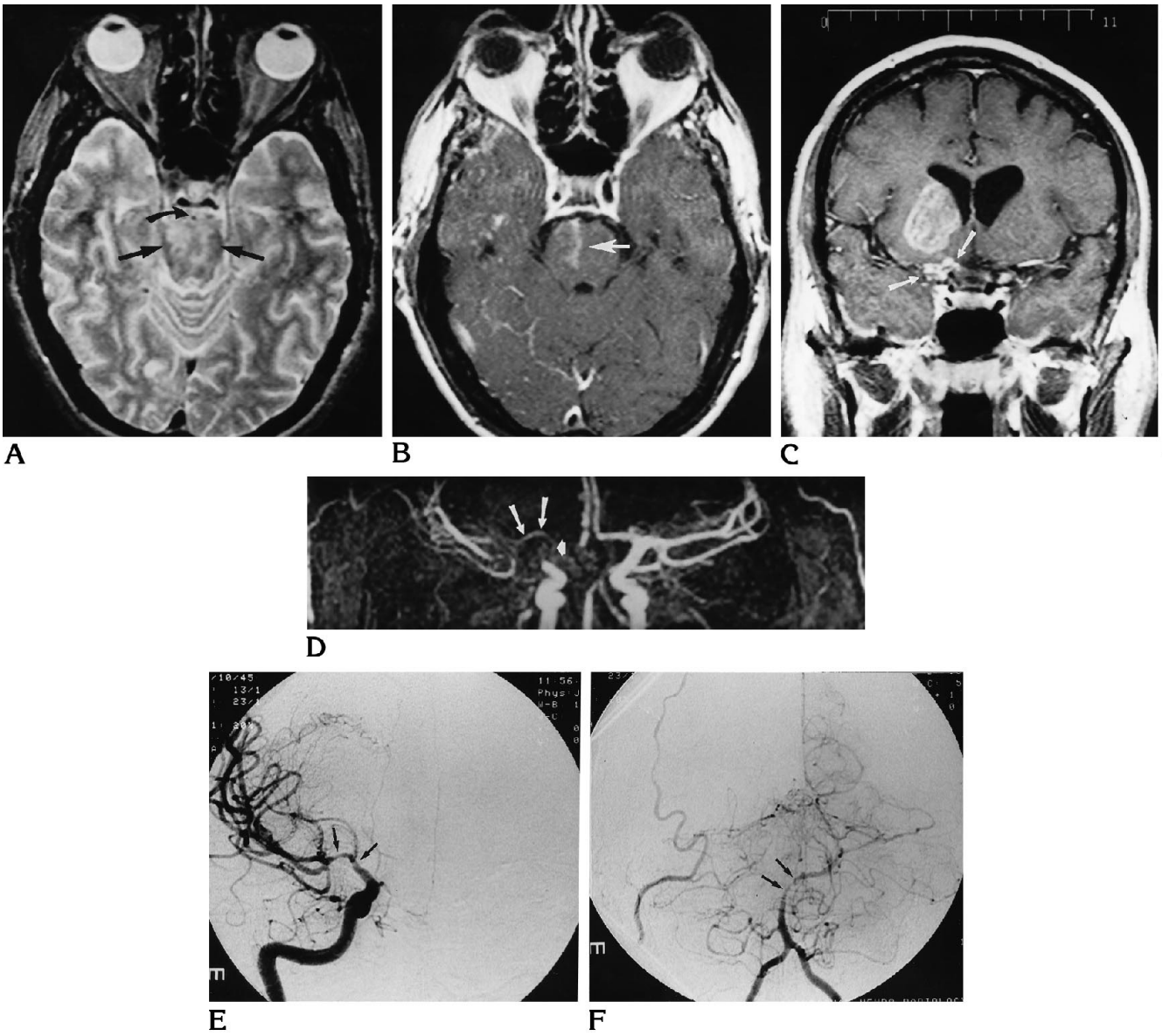


Fig 3. A 48-year-old HIV-negative man with fever, elevated serum white blood cell count, and left hemiparesis, who had multiple manifestations of neurosyphilis.

A, Axial T2-weighted (2650/80/1) image reveals poorly defined regions of increased signal in the pons (*straight arrows*) corresponding to areas of brain stem infarction. A flow void in the basilar artery is present, unlike in Figure 2, but it is small (*curved arrow*).

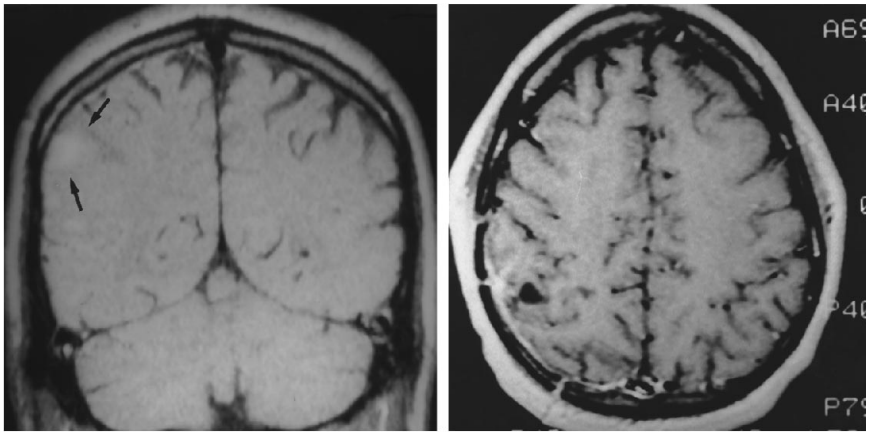
B, Axial enhanced T1-weighted (600/30/1) image demonstrates a linear band of enhancement within the area of pontine infarction (*arrow*).

C, Coronal enhanced T1-weighted (600/30/1) image reveals extraaxial enhancement around the supraclinoid segment of the right internal carotid artery (*arrows*) corresponding to localized meningitis. There is also a large enhancing infarction in the right ganglio-capsular structures producing mass effect on the ventricular system.

D, Coronal 3-D time-of-flight MR angiography projection image shows severe stenosis of the right supraclinoid internal carotid artery (*short, thick arrow*), with decreased caliber of the ipsilateral proximal middle cerebral artery (*long, thin arrows*).

E, The sites of arteritis identified in D are confirmed on conventional angiography of the right internal carotid artery (*arrows*).

F, Vertebrobasilar angiogram also demonstrates vasculitis involving the distal basilar artery, with marked irregularity (*arrows*).



A B

Fig 4. Cerebral gumma discovered in a 22-year-old HIV-positive man with headache and ataxia (courtesy of H. Waskin, MD).

A, Coronal T1-weighted (750/25/2) image after contrast administration reveals an ill-defined enhancing mass in the right parietal cortex with surrounding edema (*arrows*). Slightly more posteriorly, there was a faint tag of meningeal enhancement (not shown).

B, Axial enhanced T1-weighted (500/20/1) image at the level of the lesion after approximately 3 months of high-dose penicillin therapy shows resolution of the mass and edema.

had MR findings of subacute hemorrhage in the area of infarction.

Nonspecific white matter lesions were seen in 7 (20%) of 35 patients and appeared as multiple bilateral, discrete, well-demarcated high- intensity lesions on proton-density and T2-weighted MR images, ranging from 2 to 8 mm in size within the deep periventricular and subcortical white matter, lacking mass effect and contrast enhancement.

Two patients, or 6% of the 35, were discovered to have a cerebral gumma that appeared as a single enhancing nodule with surrounding edema. In both cases, these masses were less than 2 cm and located in the cortex of the cerebral hemispheres. One lesion was evaluated by MR and was isointense on T1-weighted images and hyperintense on T2-weighted images, showed homogeneous contrast enhancement, and had associated focal meningeal enhancement adjacent to the nodule (Fig 4). The second case was isodense on noncontrast CT, had dense enhancement with contrast administration, and did not have visible meningeal enhancement, but the mass was located subjacent to the inner table of the skull, which could potentially obscure extraaxial enhancement. Follow-up studies in both patients, one with biopsy confirmation, showed resolution of the lesions after high-dose intravenous penicillin therapy.

Meningitis was present in 2 (6%) of 35 patients, those found to have extraaxial enhancement. The first patient had diffuse extraaxial enhancement, but this patient's clinical course was complicated by superimposed tuberculous meningitis. The second patient had more focal leptomeningeal enhancement around the distal basilar artery, proximal middle and posterior cerebral arteries, and ipsilateral supraclinoid internal carotid artery (Fig 3).

Two patients, or 50% of the 4 patients with angiographic studies, demonstrated arteritis as evidenced by segmental arterial narrowing and irregularity, favoring large and medium-size vessels around the circle of Willis. Both patients had associated cerebral infarctions (Figs 2 and 3).

Pathologic Findings in Three Autopsied Patients

Pathologic material obtained in the three autopsied patients with neurosyphilis who did not have neuroimaging studies served to demonstrate the spectrum of pathologic manifestations of meningovascular syphilis. The first patient, a 60-year-old HIV-negative man with dementia, who died of neurosyphilis, was discovered to have a thick, yellowish-white exudate at the base of the brain on gross inspection (Fig 5). The patient had microscopic findings consistent with syphilitic meningitis. This case vividly depicts the severity of the inflammatory process that can affect the meninges. The second patient was a 33-year-old HIV-positive man who died of AIDS complicated by lymphocytic pericarditis and epididymitis. Microscopic findings of mild focal meningovascular syphilis were also present, and these included focal meningeal thickening and a perivascular infiltrate by lymphocytes and plasma cells, classic findings of syphilitic infection (Fig 6). There was no brain parenchymal involvement. This patient was neurologically asymptomatic, and this mild pathologic picture paralleled the lack of symptoms. Our third patient, a 32-year-old HIV-neg-

Fig 5. A 60-year-old demented HIV-negative man with syphilitic meningitis. There is a thick exudate coating the ventral surface of the cerebellar hemispheres (*arrows*).

ative woman with large-cell lymphoma of the chest and abdomen, was also neurologically asymptomatic, but had several cerebral cortical microinfarctions, subacute meningitis, and findings of Nissl-Alzheimer arteritis, with extensive adventitial cellular infiltrate (Fig 7). This patient's findings illustrate the obliterative endarteritis typical of the disease. The cause of death was systemic lymphoma.

Discussion

The clinical spectrum of neurosyphilis has been divided in one schema into four syndromes based on neurologic symptoms and the time interval between primary infection and the appearance of symptoms (5). Acute syphilitic meningitis usually occurs in the first 2 years of infection and manifests itself as headache, meningeal irritation, and confusion. Cranial nerve palsies are common. Cerebrovascular

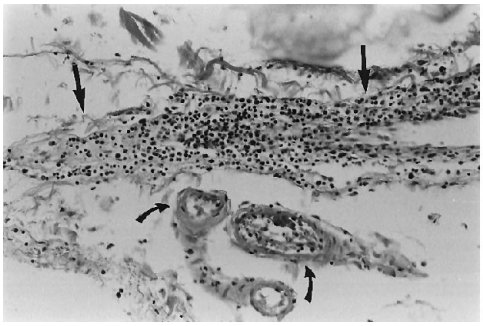


Fig 6. A 33-year-old HIV-positive man with typical microscopic findings of syphilitic leptomeningitis. There is a dense lymphoplasmacytic infiltrate thickening the meninges (*straight arrows*), which also has a perivascular predilection (*curved arrows*) (hematoxylin and eosin, × 130).

neurosyphilis usually occurs around 5 to 7 years after the primary infection, and these patients will often have prodromal symptoms of headache, vertigo, or personality disturbances weeks to months before an acute vascular event. The encephalitis or general paretic form typically has a 10- to 20-year latent period and presents as an insidiously progressive dementia associated with a delusional state, fatigability, intention tremors, and loss of facial muscle tone. A latent period of 15 to 20 years is usually seen with clinical tabes dorsalis, characterized classically by lightning pains, dysuria, ataxia, Argyll Robertson pupil, areflexia, and loss of proprioception. Of the symptomatic patients in our series with available histories, altered mental status (7 [41%] of 17), hemiparesis (6 [35%] of 17) and fever or elevated white blood cell count (5 [29%] of 17) were the clinical findings observed with greatest frequency.

Unlike in the prepenicillin era, the clinical features of neurosyphilis are now less well defined, particularly with the advent of AIDS. It has been suggested that the natural history of syphilis is altered by HIV infection, such that HIV-infected patients are more likely to progress to clinical neurosyphilis and that neurologic symptoms develop after shorter latent periods than in non-HIV-infected patients (3). Katz et al, for example, demonstrated that distinct differences in clinical features exist in patients with neurosyphilis depending on the presence or absence of HIV infection. HIV-infected patients were younger, more commonly developed syphilitic meningitis, and more frequently had features of secondary syphilis, whereas non-HIV patients presented with a greater variety of types of neurosyphilis (6). Also, a more fulminant form, referred to as *necrotizing neurosyphilis*, has been reported more often in HIV-positive patients (7). Although we did not see a significant difference in the radiographic manifestations of neurosyphilis in the HIV-positive and HIV-negative patients, the number of HIV-negative patients in our series was too small for statistical analysis.

Nearly one third (31%) of the patients in our series had normal radiographic findings; the reason for this high percentage is likely that 91% of these patients with normal imaging findings were asymptomatic, being followed clinically and radiographically as part of a multidisciplinary study of HIV infection.

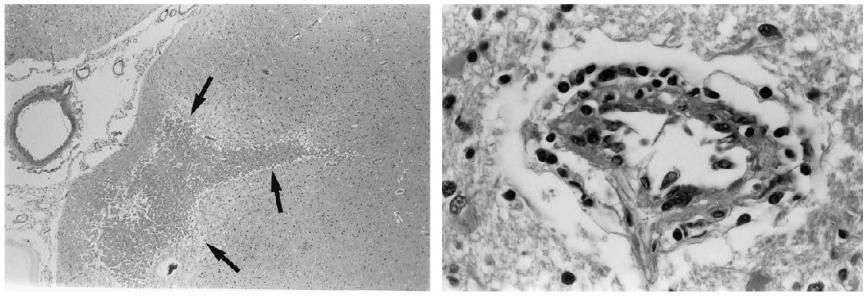


Fig 7. A 32-year-old HIV-negative woman with systemic lymphoma and syphilis-induced intracranial arteritis.

A, Microscopic section of occipital cortex shows an area of cortical microinfarction (*arrows*) (hematoxylin and eosin, × 26).

B, An obliterative endarteritis of the Nissl-Alzheimer type is seen involving a vessel at the periphery of the infarction (hematoxylin and eosin, × 390).

Atrophy was observed in 37% of patients. The atrophy could have been caused by neurosyphilis (8). However, inasmuch as atrophy is commonly seen in the HIV population, it could also have been attributable to other causes seen in that subgroup of patients, such as HIV encephalitis, cytomegaloviral encephalitis, and/or other associated central nervous system abnormalities.

Cerebral infarction caused by neurosyphilis was found in nearly 25% of the patients in our series (Figs 1–3). Six of these eight patients presented with hemiparesis. Interestingly, two patients had a more indolent progression of weakness rather than abrupt onset. The literature describes meningovascular syphilis as often presenting as a subacute syndrome of encephalitis combined with cerebrovascular events, differing from the more acute picture of embolic or thrombotic stroke (9). In the remaining patients with infarctions, one presented with a gait disturbance and cranial nerve palsies, and the history of another was not available. The findings of infarction in young adult patients with neurosyphilis have been reported previously (2, 10–13). Tien et al, for example, reported that five of six young adult patients with HIV and neurosyphilis had evidence of ischemic infarction on MR (2). Four different case reports describe stroke as the presenting symptom in young adult patients with neurosyphilis (10–13).

The meningovascular form of neurosyphilis characteristically affects arteries, and two of the four patients in our series who were studied angiographically had radiographic evidence of arteritis (Figs 2 and 3). Heubner arteritis, the most common form of syphilitic arteritis, affects large and medium-size arteries and is characterized by fibroblastic proliferation of the intima, thinning of the media, and adventitial fibrous and inflammatory changes. Nissl-Alzheimer arteritis typically affects small vessels and is manifested as endothelial and adventitial thickening, more so in the adventitia (Fig 7) (1, 11, 14). Both types of arteritis produce narrowing and focal dilatation of the vessel lumen, correlating with the segmental "beading" seen angiographically (Fig 3). Vascular occlusion results in ischemia and subsequent infarction. Therefore the clinical picture of cerebral infarction, particularly in a young adult patient, should generate investigation into causes of vasculitis. Although the causes of vasculitis are relatively extensive—including infectious agents (eg, tuberculosis, syphilis, fungal disease), collagen-vascular diseases, Takayasu arteritis, sarcoidosis, and drug-related and radiation-induced conditions—in the young adult immunocompromised host prone to opportunistic infection who presents with symptoms of stroke, meningovascular syphilis should be considered strongly.

Cerebral infarctions are relatively uncommon in HIV-positive patients. In a large autopsy series they were seen pathologically in 8% of AIDS patients (15). The infarctions have been reported as having a broad spectrum of causes including nonbacterial thrombotic endocarditis and herpes zoster arteritis (16–18). Likewise, additional causative factors of cerebrovascular disease have been postulated in HIV-positive patients such as a lupus anticoagulant factor, an IgG or IgM immunoglobulin that prolongs partial thromboplastin time but nevertheless predisposes to thrombotic events, as well as cerebral granulomatosis (19–21). As a result of the variety of causes of cerebral infarction in HIV-positive patients, we cannot totally exclude these additional causes as being contributory to infarctions in our neurosyphilis population.

White matter lesions were seen in 20% of patients in our series. Their cause is uncertain,

and their appearance is quite nonspecific in that there were no associated findings of mass effect or parenchymal enhancement. Conceivably, they too were related to underlying ischemia as a consequence of syphilitic involvement of the small feeding vessels to the deep and subcortical white matter, because many of the lesions were greater in size and number than typical unidentified bright objects (UBOs). Multifocal areas of demyelination from other causes must also be considered, but the appearance of the white matter lesions in our patient population was not as extensive as expected for progressive multifocal leukoencephalopathy or HIV encephalitis.

We discovered two patients with leptomeningeal enhancement indicative of focal or diffuse meningitis (Fig 3). The enhancement reflects the inflammatory process that can be extensive enough to produce a thick exudate about the brain surface on gross inspection (Fig 5). Pathologically, there is meningeal thickening with a perivascular infiltrate dominated by lymphocytes (Fig 6) (14). Unlike the pathologic findings of syphilitic meningitis, the radiologic appearance can be observed in many other conditions such as bacterial or fungal meningitis, sarcoidosis, other inflammatory conditions, and neoplastic disease such as lymphoma. In fact, in our series one patient's extraaxial enhancement could have been explained by coexistent tuberculous meningitis.

In our series, we encountered two patients with a syphilitic gumma presenting as a single enhancing nodule within the brain parenchyma (Fig 4). Cerebral gummas are a manifestation of tertiary syphilis that result from an exuberative cell-mediated response to *T pallidum*, a seemingly paradoxical response in the immunosuppressed host with HIV infection (22, 23). These lesions represent a circumscribed mass of granulation tissue characterized by infiltration of the brain and meninges by lymphocytes and plasma cells that is gradually replaced by fibrous tissue and necrosis (22, 23). Spirochetes are rarely found in these lesions. They typically arise from the dura and pia mater and produce symptoms similar to other intracranial tumors (22, 24). The meningeal origin of these lesions offers a unique localizing feature that can serve as a diagnostic clue. The presence of a cortical lesion in an HIV-positive patient, as was observed in our two cases, particularly if associated with adjacent meningeal enhancement, should raise suspicion for the presence of a cerebral gumma.

Syphilitic cerebral gummas are extraordinarily uncommon. Merritt and colleagues observed only 1 among 676 patients treated for neurosyphilis at Boston City Hospital over a 15-year period in the prepenicillin era (24). Its imaging features are varied. By CT, gummas have been shown to be hypodense with no contrast enhancement or mass effect (25), or as densely enhancing masses with surrounding edema (26, 27). MR descriptions have included lesions hypointense to isointense on T1-weighted images and hyperintense on T2-weighted images, associated with homogeneous contrast enhancement (2, 27, 28). Agrons et al described a gumma with a rim of hyperintensity on T1-weighted images and a rim of hypointensity on T2-weighted images (27).

Because of the rarity of cerebral gummas, however, alternative diagnoses should be considered that are more common in HIV-positive patients with parenchymal masses. *Toxoplasma* encephalitis, the most common central nervous system opportunistic infection, usually presents with multiple lesions, and it usually improves rapidly with specific antitoxoplasmatic treatment. Also, a purely cortical lesion is an unusual presentation for *Toxoplasma* encephalitis. Other diagnostic possibilities would include pyogenic, mycobacterial, or fungal abscesses, as well as neoplasms, usually lymphoma. However, lymphoma is more often deep in location (ie, corpus callosum, subependymal, and periventricular), unlike the expected location of gummas.

To optimize detection of the broad spectrum of findings in neurosyphilis, the results of our study indicate that several strategic factors should be considered in tailoring the radiologic work-up in these patients. First, after a noncontrast examination of the brain, we recommend an imaging study that includes an intravenous contrast agent. Because cerebral gummas are usually small and their imaging appearance is variable, it is conceivable that they could be missed on noncontrast examinations, and the presence of associated meningeal enhancement adds specificity to the diagnosis. In addition, diffuse extraaxial enhancement that suggests meningitis will go undetected without the use of a contrast agent. MR is preferred over CT, because MR has the added advantage of more accurately depicting white matter disease. How-

ever, CT may be more useful in evaluating potential complications of cerebrovascular disease such as intracranial hemorrhage. Second, when clinical findings and initial radiologic studies of the brain favor a diagnosis of cerebral infarction in a young adult patient, we suggest that MR angiography be the screening examination of choice in the diagnosis of syphilis-induced arteritis, because the arteritis more commonly affects large and medium-size vessels. In the future, it is possible that in the work-up of intracranial vasculitis, MR angiography may replace conventional angiography.

In conclusion, just as the clinical presentation of neurosyphilis is varied and the diagnosis is often a difficult one, the neuroimaging findings are also diverse and a high radiologic index of suspicion is paramount to early detection and diagnosis. Findings of vascular occlusive disease manifested as infarction or arteritis in a young adult patient who presents with symptoms of stroke, or an enhancing lesion discovered in the cerebral cortex associated with meningeal enhancement, should serve as strong indicators that a form of neurosyphilis may be present in either the HIV-positive or HIV-negative population. Additionally, diffuse extraaxial enhancement, and even atrophy or white matter disease, although nonspecific, in the proper clinical setting should prompt appropriate testing for neurosyphilis, a potentially treatable disease.

Acknowledgments

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