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Pictorial review

CNS involvement in AIDS: spectrum of CT and MR findings

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Abstract. The brain may be affected by a variety of abnormalities in association with human immunodeficiency virus (HIV) infection. Knowledge of their existence and characteristic imaging features are important to radiologists for detection, diagnosis, and initiation of an appropriate treatment. Although there is a considerable overlap in the imaging characteristics of different entities, some findings are found to be very suggestive of a particular disease. The CT and MR imaging techniques are commonly used in the diagnosis of neurological disorders in acquired immunodeficiency syndrome (AIDS) patients, to verify treatment response and to guide brain biopsy. This review attempts to describe CT and MR features of infectious and malignant brain disorders in HIV-seropositive patients.

Key words: Human immunodeficiency virus – Acquired immunodeficiency syndrome (AIDS) – Brain – CT – MR

Introduction

Currently, patients with acquired immunodeficiency syndrome (AIDS) are a considerable part of routine neuroradiological work. Usually, 10 % of all AIDS patients present with neurological signs and symptoms, and an additional 30–60 % of patients will develop neurological abnormalities during the course of the disease [1–3]. However, 70–90 % of all AIDS patients show neuropathological changes in the central nervous system (CNS) at brain autopsy.

The spectrum of CNS abnormalities can be divided into three main categories: (a) human immunodeficiency virus (HIV)-associated lesions; (b) opportunistic infections; and (c) neoplasms. Although these entities rarely have pathognomonic appearance on CT or MR imaging, certain radiological patterns are suggestive of specific entities. Therefore, imaging modalities play an important role in the diagnosis and therapeutic follow-up of AIDS patients with neurological disorders.

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HIV-associated CNS abnormalities

HIV encephalitis results from direct brain infection with neurotropic HIV, which occurs in 11–38 % of AIDS patients. The neurotropism of HIV includes neuroinvasiveness, neurovirulence, and replication in neural cells. Pathologically, hallmarks of the HIV-infected brain are

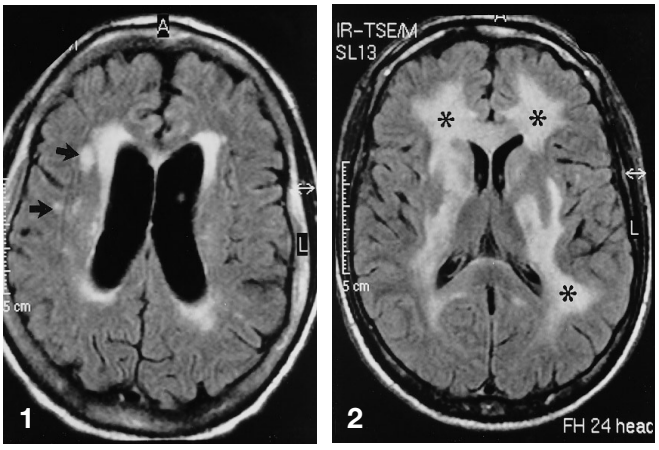


Fig. 1. Human immunodeficiency virus (HIV) encephalitis (focal white matter lesions). A 30-year-old HIV-seropositive man presented with dementia. Fluid attenuated inversion recovery (FLAIR TR/TE/TI 7000/150/2100) MR image showed multiple, small, periventricular white matter lesions. Note sulcal and ventricular enlargement due to cerebral atrophy

Fig. 2. HIV encephalitis (diffuse abnormalities). a FLAIR (7000/150/2100) MR image in a 35-year-old acquired immunodeficiency syndrome (AIDS) patient shows extensive symmetric regions of abnormally increased signal intensity *(asterisks)* without mass effect in white matter bilaterally

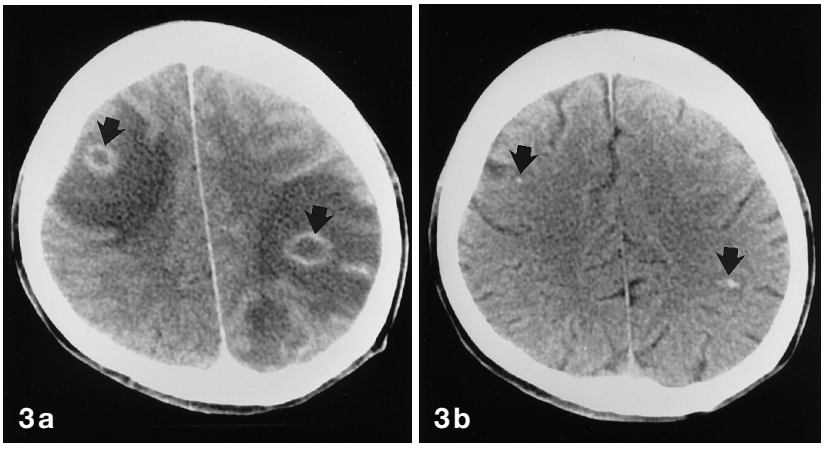
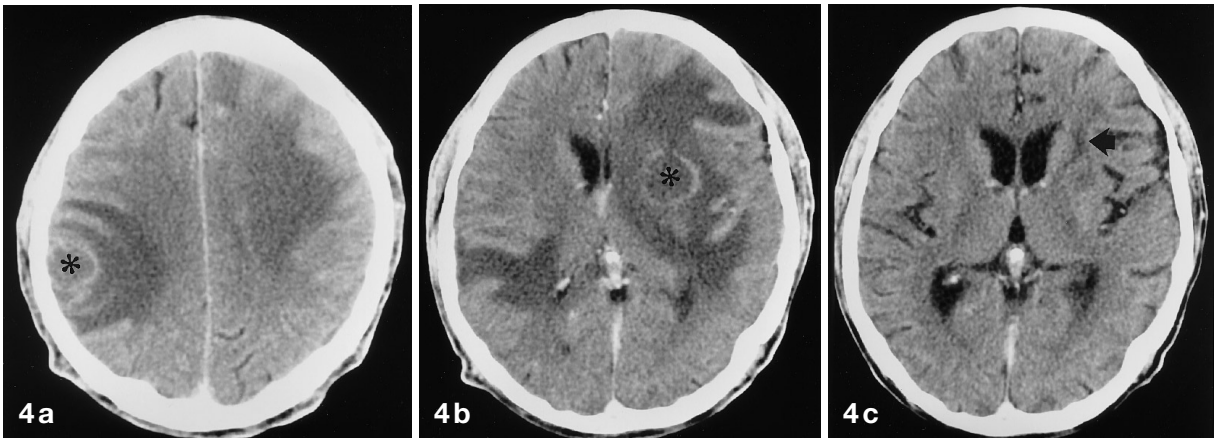


Fig. 3 a, b. Cerebral toxoplasmosis in a 35-year-old man with AIDS and recent development of motor weakness. **a** Contrast-enhanced CT scan shows multiple ring-like enhancing lesions *(arrows)* with marked surrounding edema in both hemispheres. **b** Complete resolution of lesions under medical therapy. Four months follow-up CT scan shows calcifications *(arrows)* at sites of original enhancement

Fig. 4 a–c. Toxoplasma encephalitis. **a** Initial contrast-enhanced CT scan shows poorly defined, incomplete ring-like enhancing lesions in the right parietal lobe *(asterisk)*, restricted to the cortex with surrounding edema, and in the left frontal lobe. Note marked vasogenic edema in contrast to relatively small size of lesions. **b** At a lower level, additional enhancing lesion *(asterisk)* with adjacent edema is found in the left basal ganglia. There is evidence of mass effect and compression of the left lateral ventricle. **c** Two months after specific treatment, contrast-enhanced CT scan reveals resolution of the lesions with residual hypodensity *(arrow)* in the left basal ganglia region



multifocal giant cell encephalitis (MGCE) and progressive diffuse leukoencephalopathy (PDL) [4].

At the time of diagnosis of AIDS, 25% of patients present with subclinical dementia and 33% with overt dementia. The AIDS-related dementia represents a subclinical type of dementia and is categorized into five clinical stages. Predominant symptoms are memory impairment, gait difficulty, and mental slowing. Cerebrospinal fluid (CSF) analysis shows nonspecific changes, which are useful to rule out other infection such as tuberculosis, syphilis or cryptococcosis. Electroencephalography (EEG) shows normal to diffuse slowing.

Both CT and MR imaging have low sensitivity in identification of early disease due to the microscopic size of lesions [5, 6]. The most frequent radiological finding is cerebral atrophy. Atrophy is commonly seen in the HIV population, but may be attributable to other causes, such as cytomegaloviral (CMV) infection or neurosyphilis.

Foci of demyelination in the white matter may be detected only on MR imaging. At the early stage of disease, hyperintense lesions (on T2-weighted MR imaging) are usually unilateral (frequently in the frontal lobe), and will progress to bilateral involvement (Fig. 1) [5–10]. Extensive symmetric, deep white matter disease can easely be detected on MR imaging. Fluid attenuated inversion recovery (FLAIR) MR technique was found to be very useful in detection of the lesions in HIV encephalitis, especially in the regions of the brain where CSF partial volume effect interferes with high signal intensity lesions (Fig. 2) [11]. The abnormalities show no enhancement after application of contrast medium, and lack of mass effect. In one study diffuse white matter lesions were the most common MR imaging finding in patients with AIDS dementia complex (ADC) [6].

Nevertheless, the diagnosis of HIV encephalitis may be suggested in a patient presenting clinically with ADC and the combination of cerebral atrophy and periventricular or diffuse nonenhancing white matter lesions on CT and MR imaging.

Opportunistic infections

Toxoplasmosis

Toxoplasmosis is the most common CNS infection in the AIDS population, occurring in approximately 15–50% of patients [2, 3] The disease is caused by obligate intracellular protozoan *Toxoplasma gondii*. Pathologically, there are three morphological types of toxoplasmic lesions: (a) necrotizing abscess; (b) organizing abscess; and (c) chronic abscess.

Neurological signs and symptoms, as well as CSF findings, are nonspecific. Positive serology is not diagnostic and a negative serum antitoxoplasmosis IgG antibody titer does not exclude toxoplasmosis.

Typical CT findings related to toxoplasmosis are multiple hypodense or isodense lesions on unenhanced images, which show ring-like or solid nodular enhancement with perifocal edema and mass effect. The lesions are localized in the basal ganglia (in up to 75%), corticomedullar junction, and posterior fossa (Figs. 3, 4) [12, 13] On T2-weighted MR images the majority of the lesions are hyperintense; however, iso- or hypointense lesions as well as mixed pattern may be found (Fig. 5) [12–14]. Both imaging techniques have proven to be useful diagnostic and therapeutic follow-up modalities. Ten to fourteen days after beginning of a specific treatment, the lesions show regression in size, enhancement, and perifocal edema. If the lesions remain stable or increase in size, immediate brain biopsy is indicated. Improvement due to therapy cannot be monitored on the basis of radiological studies in patients receiving steroids. Complete resolution may be prolonged up to 6 months and a life-long maintenance therapy is necessary to prevent relapse. On follow-up CT scans, previous toxoplasma lesions may completely resolve or remain as residual lucencies or hyperdense foci of calcifications (Fig. 3) [15].

Recently, thallium-201(TI-201) brain spectral emission computed tomography (SPECT) has been introduced in differentiating toxoplasmosis from lymphoma. In patients with toxoplasmosis, no uptake of TI-201 was found in the CNS lesions. This is in contrast to lymphoma, where an abnormal increased uptake is usually seen [16]. Another adjunctive method for the differential diagnosis of focal brain lesions in AIDS is MR spectroscopy. Preliminary results indicate that toxoplasmosis and lymphoma have highly distinctive chemical features. In toxoplasmosis lesions lactate and lipids are elevated, with absence of all normal brain metabolits, whereas an increase in choline could be observed in lymphoma [17].

Cryptococcosis

In addition to HIV and Toxoplasma gondii, saprophytic fungus *Cryptococcus neoformans*is the third most common infectious organism causing CNS disease in AIDS (6–7%) [2, 18]. Cryptococcal infection of the CNS manifests as a subacute basilar granulomatous meningitis or parenchymal disease.

The most common clinical symptoms are: headache, fever, meningismus, and seizures.

The imaging findings are nonspecific, and most often CT and MR images appear normal [18–20]. Four distinct patterns, however, may be observed on CT or MR imaging:

1. Meningitis with mild dilatation of the ventricular system or (rarely) nodular meningeal enhancement on postcontrast images.
2. Dilated Virchow-Robin spaces filled with fungi, resulting in the formation of nonenhancing cystic lesions of low density on CT scan, or low signal intensity on T1-weighted MR images, and high signal intensity on T2-weighted MR images. The cystic lesions are found predominantly symmetrically in the basal ganglia and thalamus (Fig. 6) [20]. Macroscopic examination shows no inflammatory cells, and organisms are found only in perivascular region without invading the surrounding brain [20].
3. A solid or ring-like contrast-enhancing, parenchymal mass (so-called cryptococcoma), which is extremely rare and found preferentially in the ependyma of the choroid plexus. Cryptococcomas represent collections of organism, mucoid material, and inflammatory cells.
4. "Gelatinous pseudocysts," nonenhancing intraparenchymal cystic lesions usally also located bilaterally in basal ganglia. These lesion have also been termed "soap bubles" [21, 22].

These findings, although nonspecific, are very suggestive of CNS cryptococcosis in their clinical setting.

Cytomegalovirus infection

Cytomegalovirus (CMV) ventriculoencephalitis has been found in 33% of autopsies performed in AIDS patients [23]. The cytomegalovirus belongs to the herpes virus family, and the underlying pathology is characterized by microglial nodules, microinfarctions, and giant cells with CMV inclusions. Patients with CMV retinitis have a tenfold increased risk of having CMV encephalitis [24].

Radiographic studies of CMV infection in the CNS are nonspecific and, in almost all cases, CT and MR imaging do not reveal any abnormality. Necrotic ventriculitis may cause periventricular subependymal enhancement around the lateral ventricles, septum pellucidum, corpus callosum, and fornics, or demyelination may result in diffuse white matter abnormalities [23, 25]. On T2-weighted MR images periventricular hyperintensity could be observed (Fig. 7). Because of nonspecific neuroradiological features, the diagnosis of CMV infection must be confirmed by histological identification of typical intranuclear inclusions (owl's eyes).

Tuberculosis

Central nervous system tuberculosis has a high mortality (79%) among AIDS patients, underlining the necessity for an early diagnosis. Five to nine percent of all AIDS patients have tuberculous infection, and 10% have CNS involvement [26, 27].

The following neuroimaging findings can be seen on CT and/or MR [28, 29]:

1. Basal meningitis usually presents with meningeal enhancement in the basal cisterns and/or hydrocephalus. In the series of Villoria et al. of 35 patients with AIDS and proven intracranial tuberculosis, hydrocephalus was the most frequent finding (51%), followed by meningeal enhancement (41%) [29].
2. Tuberculomas are solitary or multiple ring/nodular enhancing lesions, which are reported to be small lesions with little mass effect and/or edema. They are re-

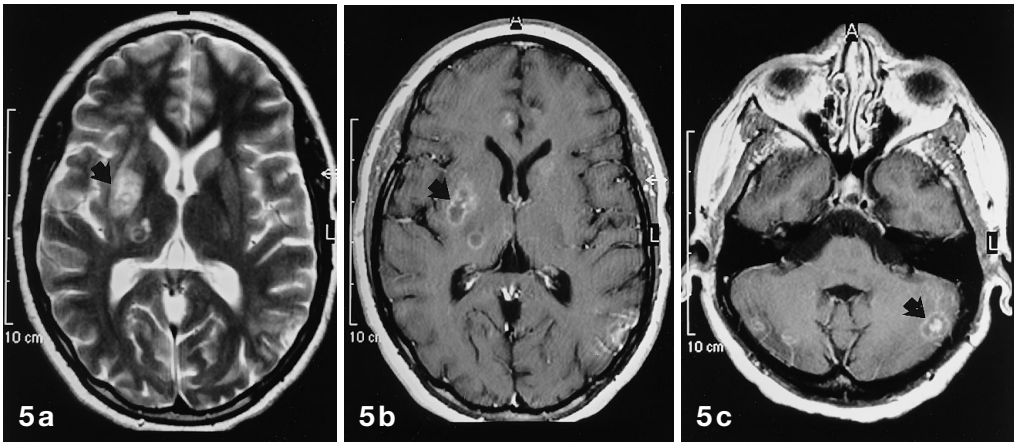
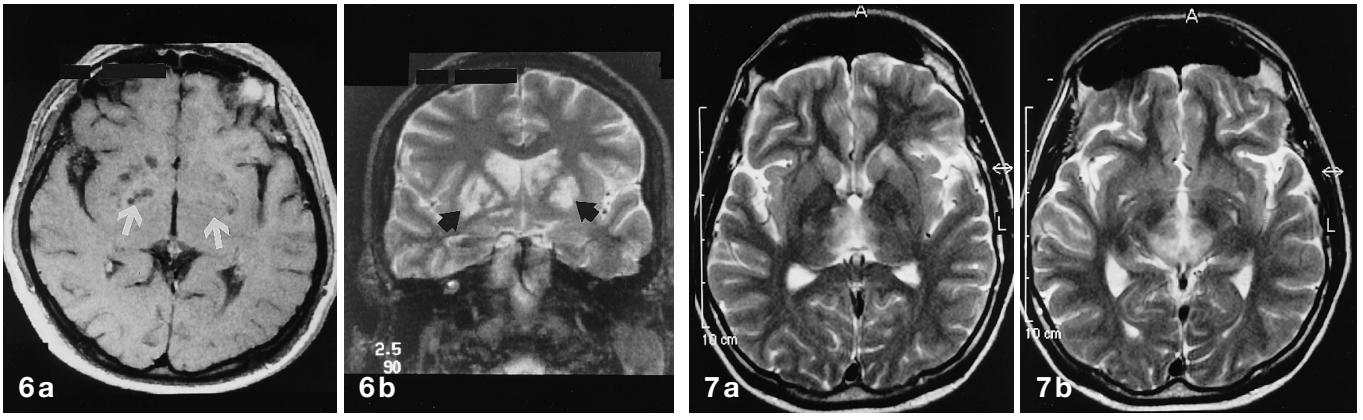


Fig. 5 a–c. Toxoplasma encephalitis. **a** Axial T2-weighted (TSE 2870/120) MR image in a 32-year-old black woman with AIDS shows two lesions of high signal intensity *(arrow)* in the basal ganglia, caput nuclei caudati, and frontal and occipital lobes. **b** Axial contrast-enhanced T1-weighted (SE 639/15) MR image at corresponding level shows the lesions with ring-like enhancement and irregular margins. **c** Axial contrast-enhanced T1-weighted (SE 639/15) MR image at a lowel level shows multiple additional lesions with a "target" appearance *(arrow)*



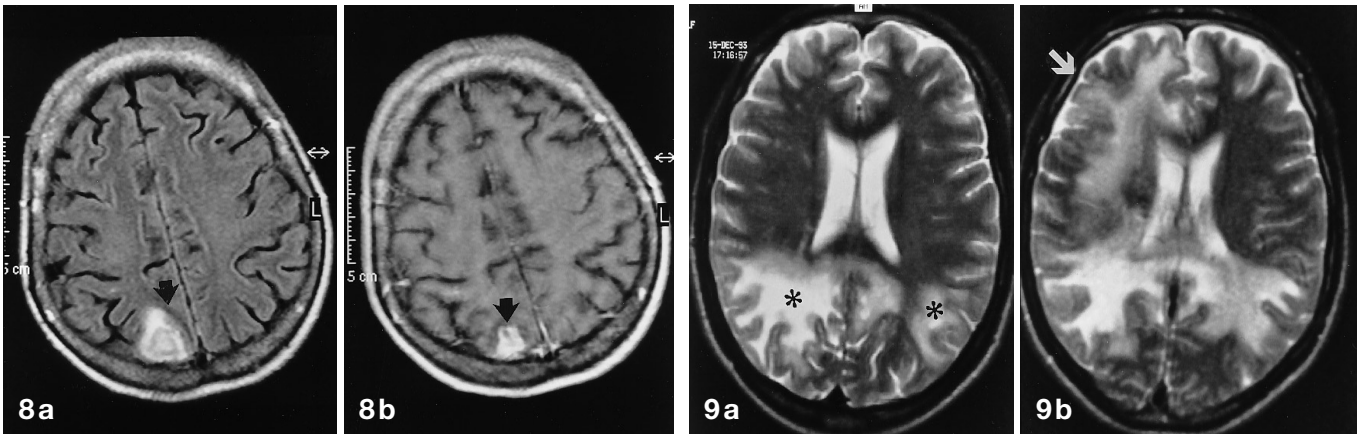


Fig. 6 a, b. Cryptococcosis. **a** Axial T1-weighted (SE 700/19) MR image shows multiple cystic-like hypointense lesions *(arrows)* in both basal ganglia. **b** On corresponding coronal T2-weighted (SE 2500/90) MR image, clusters of punctate hyperintense foci bilaterally in the basal ganglia regions *(arrows)* were seen. These represent multiple dilated Virchow-Robin spaces filled with numerous fungi (proven by autopsy)

Fig. 7 a, b. Cytomegalovirus (CMV) encephalitis in a 33-year-old AIDS patient who presented with disorientation. T2-weighted MR images at the level of **a** frontal horns and **b** third ventricle show periventricular, subependymal hyperintensity adjacent to the third ventricle. On T1-weighted MR images, the abnormality showed low signal intensity (not shown)

Fig. 8 a, b. Intracranial tuberculosis in a 44-year-old female AIDS patient with a history of pulmonary tuberculosis. **a** Axial FLAIR (TR/TE/TI 7000/150/2100) MR sequence reveals an inhomogenous ring-like lesion with surrounding edema located in the cortex of the right parietal lobe *(arrow)*. **b** On contrast-enhanced T1-weighted (SE 550/20) MR image, the lesion shows intense enhancement *(arrow)*. Brain biopsy revealed a granuloma due to tuberculosis (tuberculoma)

Fig. 9 a, b. Progressive multifocal leukoencephalopathy (PML). **a** Axial T2-weighted (TSE 3600/93) MR image obtained in a 31-year-old male AIDS patient shows areas of high signal intensity in both parietal lobes *(asterisks)*. There is no evidence of mass effect. **b** Follow-up axial T2-weighted (TSE 3600/93) MR image 2 months later shows rapid progression of disease with involvement of white matter in the complete right hemisphere and transcallosal extension. Note clearly visible involvement of subcortical fibers *(arrow)*

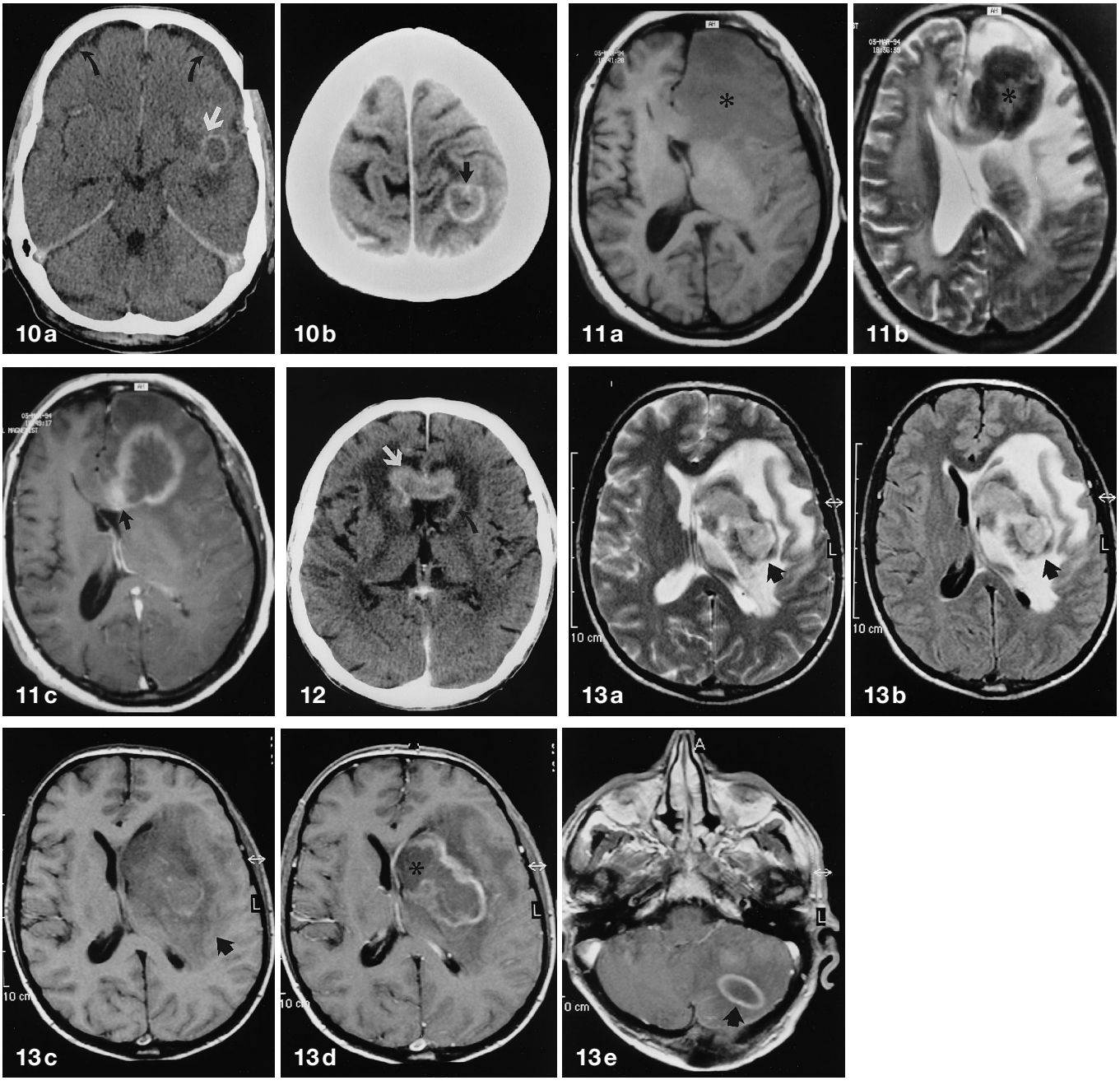


Fig. 10 a, b. Invasive aspergillosis. **a** Contrast-enhanced CT scan in a 44-year-old AIDS patient who presented with cognitive impairment and hemiparesis shows ring-like enhancing lesion *(white arrow)* with moderate adjacent edema in the left insular region, and bilateral frontal subdural hygroma *(black arrows)*. **b** At a higher level, additional cortical ring-enhancing lesion *(arrow)* is shown in the left parietal lobe. Brain biopsy revealed diagnosis of disseminated necrotic encephalitis due to aspergillosis

Fig. 11 a–c. Primary central nervous system (CNS) lymphoma. **a** T1-weighted (SE 627/15) MR image shows large hypointense lesion *(asterisk)* in the white matter of left frontal lobe with severe mass effect. **b** On axial T2-weighted (TSE 3500/93) MR image, the lesion is typically hypointense *(asterisk)* with clear demarcation from high signal intensity perifocal edema. **c** Contrast-enhanced T1-weighted (SE 627/15) MR image shows heterogenous peripheral enhancement and irregular, sinous margins of the lesions. The mass involves the corpus callosum *(arrow)*

Fig. 12. Primary CNS lymphoma. Contrast-enhanced axial CT scan shows a peripherally enhancing symmetric lesion *(white arrow)* in the genu corporis callosi extending along the walls of the lateral ventricles *(black arrow)* and the caput of nucleus caudatus. Note moderate mass effect of lesion. At autopsy, multiple disseminated necrotic lymphomas were found, resulting from tumor spread through the ventricular system

Fig. 13 a–e. Primary CNS lymphoma in a 44-year-old male with AIDS. **a** T2-weighted (TSE 3452/120) MR image shows an inhomogenous lesion in the left basal ganglia with perifocal edema and mass effect (arrow). **b** FLAIR sequence shows improved delineation of the lesion from edema as compared with T2-weighted images (arrow). **c** Large hypointense mass lesion is shown on T1-weighted MR image (SE 550/20) in the left hemisphere with midline shift (arrow). **d** Contrast-enhanced MR image (SE 550/20) shows hypointense lesion with peripheral enhancement (asterisk). **e** At a lower level, an additional oval ring-enhancing lesion is shown in the left cerebellar hemisphere (arrow). At autopsy, a large necrotic lymphoma was found

sult of hematogenous spread of infection or of direct extension of infection from CSF. The signal intensity on T2-weighted MR images depends on the stage of the granuloma. In the early stages they are hypointense (solid caseation necrosis), and tend to become isointense capsule (Fig. 8) [28].
3. Tuberculous abscesses are a rare form of infection.They are larger than tuberculomas and mostly multiloculated. The ring-like enhancing lesions cannot be differentiated radiologically from abscesses of other etiologies.
4. Cerebral infarction is a common form of CNS tuberculosis. In a study by Whiteman et al. [28] infarction was seen in 36% of patients, Villoria et al. have observed vascular complications only in 23% of the cases [29]. The most common locations are the regions of basal ganglia.

Chest X-ray abnormalities may be additional help for the suspected diagnosis of cerebral tuberculosis.

Progressive multifocal leukoencephalopathy

Progressive multifocal leukoencephalopathy (PML) is a subacute demyelinating opportunistic infection of the CNS caused by *JC virus* (the initials of the patient in whom the virus was first isolated) with tropism for oligodendrocytes. During the era of AIDS, there has been a dramatic rise in the frequency of PML, with the range of 0.7–7.7% [2, 30, 31]. At macroscopic examination of the brain irregularly demarcated, patchy discolorations are found [32]. The subcortical fibers are always affected.

Clinically, PML may present with nonspecific headache, visual disturbance, dementia, hemiparesis, cognitive impairment, or seizure [31].

The CT and MR imaging techniques correlate well with the pathological findings. The lesions are typically multifocal, scalloping hypodensities on CT scans, and hyperintense patchy areas on T2-weighted MR images (Fig. 9). They demonstrate rapid progression in size, resulting in confluence of lesions [33, 34]. There is a lack of mass effect, and contrast enhancement. The lesions may show a central area of marked hypodensity (CT), or hyperintensity (T2-weighted MR images), resembling complete loss of the neuronal structures (necrosis).

The disease is progressive and prognosis remains poor. Even though some patients live up to 2 years after the onset of symptoms, the mean survival ranges from 6 to 9 months [35].

Candidiasis and aspergillosis

A CNS infection with opportunistic fungal organisms is a result of hematogenous dissemination. The radiological features are nonspecific. The enhancing lesions (nodular or ring-like) involving the gray matter are indistinguishable from abscesses of other etiologies (Fig. 10). Involvement of other organs (paranasal sinus, chest) may confirm the diagnosis [36].

Neoplasm

Primary CNS lymphoma

With the advent of AIDS, the prevalence of primary CNS lymphoma (PCL) has increased to 6% among AIDS patients [2, 37]. The disease is always indicative of a high-grade malignancy, with a predominance of large cell types. Lymphoma favors brain parenchyma rather than meninges.

There are distinct differences in radiological features in patients with PCL, depending on the presence or absence of AIDS [38]. In the AIDS population, PCL presents as heterogeneous or ring-like enhancing lesions (irregular nodular type of enhancement), typically isointense to gray matter on T2-weighted MR images, and usually with a moderate perifocal edema (Fig. 11). The iso/hypointensity of the central areas are the result of necrosis due to the coagulation necrosis and thrombosis of the vessels. Lymphoma predominantly involve the basal ganglia, corpus callosum, periventricular white matter, frontal lobes, and thalamus (Fig. 12) [39–41]. The autopsy studies have shown that PCL are multicentric in 80–100% in AIDS population (Fig. 13) [37]. A large (more than 3.5 cm in diameter) inhomogenous enhancing lesion, localized in the deep white matter, close to the midline, or a periventricular lesion, with a moderate degree of edema and mass effect, with rapid progression, should be suggestive of PCL.

The differential diagnosis of PCL to toxoplasmosis is difficult, and brain biopsy is still necessary to ensure rapid improvement of the lesions with specific antitoxoplasmotic treatment [42–44]. The prognosis of PCL is still poor, with mean survival of 5.5 months.

Conclusion

Because of the rising incidence of AIDS, and the large number of AIDS patients who develop neurological disease, it is increasingly important to recognize AIDS-related neuroradiological findings. The CT and MR techniques are both excellent means of detection of cerebral lesions in AIDS patients, useful in initial diagnosis and in therapeutic follow-up evaluation.

The MR technique has a higher sensitivity and may identify lesions that were not seen on CT; therefore, MR should be the method of choice. Thallium-SPECT and MR spectroscopy are additional help especially in differentiating the toxoplasmosis from lymphoma. Finally, brain biopsy is still necessary in unresolved cases.

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