

A 'brain tumor' in an intravenous drug abuser

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Abstract: A male intravenous drug abuser who was infected with hepatitis B and C, presented with a slowly progressive hemiplegia. Contrast enhanced computerized tomography of the head showed a solitary ring-enhanced mass with surrounding edema. Clinically brain tumor was suspected but a brain biopsy confirmed cerebral toxoplasmosis. An HIV test was not considered until the result of brain biopsy. He also had lymphopenia and positive serum toxoplasma antibody. His subsequent HIV test was positive. He deteriorated after a brain biopsy. Empirical antitoxoplasma treatment is recommended in HIV-positive patients with ring-enhanced lesions with surrounding edema and with positive toxoplasma serology. Cerebral toxoplasmosis is still the commonest cerebral opportunistic infection in HIV-infected patients even though the incidence has declined with the use of antiretroviral therapy. It is often diagnosed in those patients as an initial presentation of HIV infection or in those who failed to attend for disease monitoring. Clinical features and differential diagnosis of cerebral toxoplasmosis in immunocompromised patients are discussed.

Keywords: brain tumor, cerebral toxoplasmosis, intravenous drug abuser, primary brain lymphoma, HIV/AIDS

Case report

In May 2005, a 45-year-old ex-intravenous drug abuser with a suspected brain tumor was transferred by a consultant physician at a District General Hospital to a neurosurgical unit at a teaching hospital. His main complaints were fever, headache, speech impediment, and blurring of vision. On examination the patient was conscious and had left sided 3rd nerve palsy. Fundi were normal. He subsequently slowly developed a dense right hemiplegia. Investigations revealed hemoglobin 11.8 gm/l, white cell count $3.9 \times 10^9/l$ with lymphocyte count of 0.6, platelets 174 and positive hepatitis B surface antigen and hepatitis C PCR. Chest radiograph was normal. Contrast-enhanced computerized tomography (CT) of the head showed a solitary ring enhanced mass in the mesencephalon with surrounding edema (Figure 1). Clinically a brain tumor was suspected, but stereotactic brain biopsy revealed numerous tachyzoites confirming cerebral toxoplasmosis (CTx) (Figure 2).

An HIV antibody test was positive with CD4 count of less than 10 cells/mm³ and HIV RNA (viral load) level of 2.2 millions copies/ml. Toxoplasma antibody dye test was positive at 64 iu/ml. Toxoplasma IgM antibody was negative. After brain biopsy he deteriorated but a repeat CT scan did not reveal any intra cerebral bleeding. He was treated with antitoxoplasma and antiretroviral therapy and was able to walk with an aid of stick in July 2005. In December 2005 his HIV RNA level became undetectable and CD4 count was 78. No further follow up was possible as the patient voluntarily returned to Portugal in February 2006.

Discussion

Cerebral toxoplasmosis is almost always due to reactivation of latent infection and typically occurs in patients with CD4 count of less than 100 cells/mm³. The major

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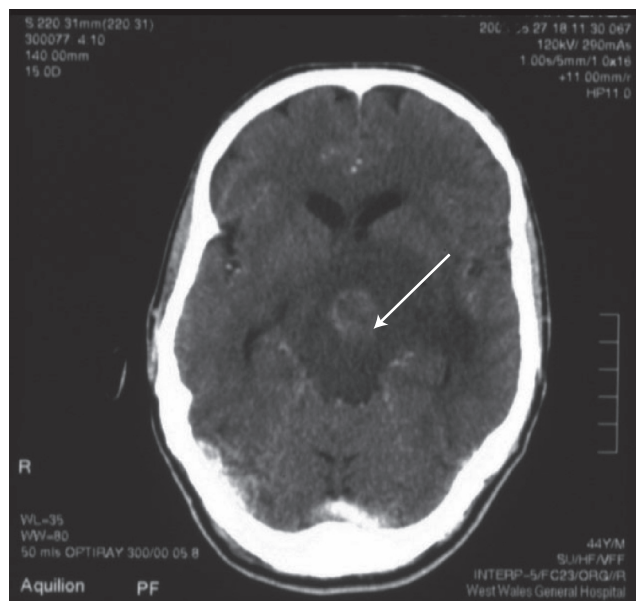


Figure 1 Computed tomography scan showing ring-enhanced mass with surrounding edema.

clinical features of CTx include headache, hemiparesis, speech disturbances, cerebellar dysfunction, cranial nerve palsies, and sensory loss.¹ It can rarely present with movement disorders.² Our patient presented with headache, dysphasia and subacute onset of hemiplegia, with isolated cranial nerve palsy. Ocular and pulmonary infections are the most common extracerebral toxoplasmosis seen in HIV-infected patients.³ A study by Ammassari and colleagues looked at 281 HIV-positive patients with focal neurological deficits. Toxoplasmic encephalitis was present in 36.4%, primary brain lymphoma in 26.7%, progressive multifocal leucoencephalopathy in 18.2%, and focal HIV encephalopathy in 5.0%.⁴

As in our case, up to 97% of patients with CTx have toxoplasma IgG antibodies and the levels are usually over 1:256 in patients with CTx.⁵ However the level of antibodies is not always predictive of the diagnosis as in our patient,⁶ but a negative test makes the diagnosis unlikely. The sensitivity and specificity of *Toxoplasma gondii* polymerase chain reaction (PCR) in blood for the diagnosis of CTx were 80% and 98%, respectively.⁷ Eight out of 19 (42.1%) patients who were histologically confirmed with CTx had positive PCR in the cerebrospinal fluid (CSF).⁸ A negative PCR in the CSF will therefore not exclude CTx.⁹

CT scan typically reveals bilateral, multiple, hypodense ring-enhanced lesions with surrounding edema in 70%–80% of patients.¹⁰ These lesions tend to involve the basal ganglia and corticomedullary junctions in either or both hemispheres.

As in our patient lesions can be solitary in 27% of patients¹¹ and clinically mistaken for a neoplasm. Magnetic resonance imaging scan is more sensitive than CT scan. However radiological findings are not pathognomonic for CTx hence stereotactic brain biopsy remains the main stay of diagnosis of CTx. Findings range from granulomatous reaction with gliosis to necrotising encephalitis. Both free tachyzoites and encysted bradyzoites may be found at the periphery of the necrotic foci. A study by Gildenberg and colleagues of stereotactic brain biopsy in 250 patients revealed pathologies in 246 patients (98%). Multiple pathologies were found in 16 (6%) biopsies. Four out of the first 32 (12%) had intracranial bleeding with 3% fatality. After correction of coagulopathy, seven out of 218 (3%) developed intracerebral bleeding with 2% fatality.¹²

Increased uptake on thallium-201 single-photon emission CT is suggestive of central nervous system (CNS) lymphoma. The sensitivity and specificity of these findings for the diagnosis of CNS lymphoma range from 86%–100% and from 76%–100%, respectively.⁵ Among primary brain tumors, anaplastic astrocytoma and glioblastomas account for 38%, and meningiomas account for 27%. Other less common primary brain tumors include, in decreasing order of frequency, pituitary tumors, schwannomas, CNS lymphomas, oligodendrogliomas, ependymomas, low-grade astrocytomas, and medulloblastomas.¹³ Brain metastases outnumber primary brain tumors and occur in 20%–40% of patients with cancer.¹⁴ The most common cancers causing metastases to the brain are lung cancer (50%), breast cancer (15%–20%), unknown primary cancer (10%–15%), melanoma (10%), and colon (5%). The common sites of metastases occur in cerebral hemispheres (80%), cerebellum (15%), and brain stem (5%).¹⁴ Metastases to the brain are often multiple in more than 70% of cases, but solitary metastases also occur. Clinical features of brain tumors include headache, nausea, vomiting, and personality changes. Another clinical symptom is seizures, which occur in 20% of patients with supratentorial brain tumors and may antedate many months with slow growing brain tumors.¹⁵

Our patient was infected with blood-borne viruses (Hepatitis B and C) and had a low absolute lymphocyte count. A high index of suspicion in those patients with focal neurological signs would have prevented unnecessary neurosurgical intervention. Empirical antitoxoplasma treatment is recommended in HIV-positive patients with ring-enhanced lesions with surrounding edema and with positive toxoplasma serology. By avoiding the need for brain biopsy, hospital stay

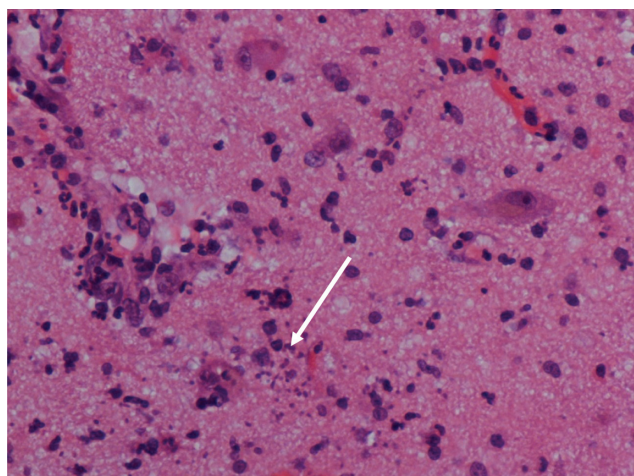


Figure 2 Brain biopsy showing numerous tachyzoites.

is shortened and subsequently costs are saved.¹⁶ Brain biopsy should only be considered in patients with negative toxoplasma serology and who do not respond to antitoxoplasma treatment.¹⁷

Disclosure

The author reports no conflicts of interest in this work.

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