Isolated toxoplasmosis of the thoracic spinal cord in a patient with acquired immunodeficiency syndrome

Case report

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Toxoplasmosis and primary CNS lymphoma are the two most common causes of intraparenchymal cerebral mass lesions in patients with acquired immunodeficiency syndrome (AIDS). The clinical and radiographic features of the intracranial lesions have been well described. Because of the high frequency of toxoplasmosis in the AIDS population, common therapy for patients presenting with intracranial mass lesions consists of an empirical trial of anti-Toxoplasma chemotherapy, with biopsy reserved for cases demonstrating features considered to be more consistent with lymphoma, or for lesions that do not improve despite adequate anti-Toxoplasma treatment. A similar treatment algorithm does not exist for intramedullary lesions of the spinal cord.

The authors describe a patient who presented with paraparesis resulting from an isolated thoracic intramedullary lesion. An open biopsy of the lesion revealed characteristic structures containing Toxoplasma tachyzoites. The clinical and radiographic presentation of the lesion is discussed, the available literature is reviewed, and a treatment strategy for spinal cord lesions in AIDS patients is proposed.

KEY WORDS • acquired immunodeficiency syndrome • toxoplasmosis • lymphoma • spinal cord lesion

Approximately 10% of patients with acquired immunodeficiency syndrome (AIDS) present with neurological complaints as the first manifestation of their disease and up to 80% will have central nervous system (CNS) involvement during the course of their disease.6,11 Toxoplasmosis and primary CNS lymphoma are the two most frequent intracranial lesions responsible for focal neurological deficits in patients with AIDS. Differentiation between these two disorders may be difficult given the similarity of clinical symptomatology and radiographic findings. Although certain imaging characteristics of these lesions are thought to be more consistent with lymphoma, such as single lesions or periventricular location, attempts to differentiate CNS toxoplasmosis from lymphoma based on the imaging characteristics of intracerebral lesions have been largely unsuccessful.4,5

Because differentiation between Toxoplasma infection and CNS lymphoma is difficult based solely on radiographic criteria, empirical anti-Toxoplasma chemotherapy is commonly used, with biopsy reserved for those cases that either show atypical features or do not respond to treatment.11 Toxoplasmosis of the spinal cord is rare, however, and has not been previously described ante mortem without the presence of widespread supratentorial CNS or disseminated disease.8,9,12 We describe an unusual case of toxoplasmosis presenting as an isolated, intrinsic spinal cord mass.

Case Report

This 45-year-old right-handed bisexual white man was found to be human immunodeficiency virus–positive in 1987. The patient had no previous opportunistic infection or neoplasm. Approximately 6 months prior to presentation, he noted the gradual onset of lower-extremity weakness and uncoordination. This was defined by the patient as being easily fatigued by ambulation and losing sexual function. One week prior to presentation, the patient was...
able to stand and walk only with great difficulty. One day prior to admission, he developed urinary retention. He denied any history of back pain, fever, chills, or other constitutional symptoms.

**Examination.** Evaluation in the emergency department revealed a markedly distended bladder that required catheterization. He had 3+/5 strength in both lower extremities with intact sensation including position sense. Reflexes were hyperactive in the lower extremities with bilateral positive Babinski signs. A computerized tomography (CT) scan of the head, with and without contrast, was obtained and proved to be normal. Further diagnostic workup included magnetic resonance imaging of the thoracic spine, with and without gadolinium enhancement, which revealed a 1-cm wide by 2-cm long intramedullary lesion at the T-4 level. The lesion enhanced homogeneously after administration of gadolinium and displayed a moderate amount of surrounding signal abnormality consistent with spinal cord edema (Fig. 1). A lumbar puncture was performed, and evaluation of the cerebrospinal fluid (CSF) revealed a white blood cell count of one, a red blood cell count of four, a glucose level of 23 mg/dl, and a protein level of 359 mg/dl. Cerebrospinal fluid was sent for cytology, bacterial, and fungal cultures; viral cultures; and VDRL (syphilis check), Lyme titer, and cryptococcal antigen testing. All were negative. Anti-Toxoplasma immunoglobulin (Ig) G immune titers were negative in a ratio of 1:16, and an IgM assay was also negative. At the time of evaluation, the patient’s CD4 cell count was 29 cells/mm². After these initial studies were completed, he was started on a course of decadron, and radiation therapy was administered (200 rad/day) for presumed lymphoma. Over the next 48 hours, the patient progressed to complete paraplegia with a clear T-4 sensory level.

**Biopsy and Histological Examination.** Because the patient’s neurological status did not improve, a spinal cord biopsy was performed through a T3–4 laminectomy. The spinal cord had a distinct fusiform enlargement; the involved section had a firm consistency. No other focal abnormality was apparent. Another biopsy was obtained through a midline myelotomy. The biopsy tissue was soft and off white in color. Histological examination revealed profuse acute and chronic inflammation. Structures resembling *Toxoplasma* tachyzoites were found throughout the biopsy specimen (Fig. 2), which showed an intense reaction with immunohistochemical stains using anti-*Toxoplasma* antibody (Fig. 3). Electron microscopy revealed typical *Toxoplasma* tachyzoites (Fig. 4).

**Postoperative Course.** Postoperatively, the patient showed no improvement in neurological status. He was treated with anti-*Toxoplasma* chemotherapy and was discharged to a rehabilitation center 1 month after surgery.
Spinal cord toxoplasmosis in an AIDS patient

Fig. 4. Electron micrograph of a thoracic spinal cord biopsy specimen in a 45-year-old man with acquired immunodeficiency syndrome. *Toxoplasma gondii* tachyzoites demonstrate conoidlike structures (arrow). Original magnification × 5000.

**Discussion**

Toxoplasmosis and lymphoma are the two most common intracranial lesions responsible for focal neurological deficits in AIDS patients. Although certain radiographic features such as a single lesion or periventricular location may support the diagnosis of lymphoma, no definitive diagnosis can be made from imaging criteria alone. Serum and CSF cytological and immunological examination may provide important information, such as the presence of antibodies to toxoplasmosis or atypical lymphocytes; however, the lack of such findings does not exclude either diagnosis.

In a recent series from the University of California in San Francisco, 22% of patients with histologically diagnosed cerebral toxoplasmosis had undetectable anti-*Toxoplasma* IgG antibodies. Similarly, malignant lymphoma of the central nervous system has been reported without the presence of malignant cells in the CSF. The treatment of intracranial lesions now includes empirical anti-*Toxoplasma* chemotherapy followed by biopsy, stereotactic or otherwise, if no definite improvement is seen clinically or in imaging studies obtained 7 to 14 days after treatment is initiated. This practice may be modified to include biopsy at an earlier stage if the lesion is solitary or atypical. The literature concerning intramedullary spinal cord lesions in AIDS patients fails to provide a similar management protocol.

**Toxoplasmosis in Patients With AIDS**

There have been three reported cases of *Toxoplasma* causing intramedullary spinal cord lesions in AIDS patients. The first case involved a patient with a weak left upper extremity who was noted to have diffuse thickening of the spinal cord in the medcervical region. He underwent radiological therapy for a presumed lymphoma and died 11 days later from respiratory insufficiency. The diagnosis of toxoplasmosis was made at autopsy. The second case involved a patient with an asymmetric paraparesis who was found to have diffuse spinal cord thickening in the T3–5 region, bilateral basal ganglia lesions, and a positive serum anti-*Toxoplasma* antibody titer of 1:2048. This patient underwent open biopsy of the spinal cord lesion. He was paraplegic postoperatively and failed to improve despite anti-*Toxoplasma* chemotherapy. The final case involved a patient with widely disseminated *Toxoplasma* infection who had evidence of disease involving the anterior horn of the spinal cord at autopsy.

Although still relatively rare, primary involvement of the spinal cord by lymphoma has been reported with much greater frequency. Hénin, et al. reported four cases of primary CNS lymphoma involving the spinal cord in his autopsy series of 138 consecutive AIDS patients. Hautzer and colleagues described six cases of primary lymphoma localized to the spinal cord, and many authors have reported cases of lymphoma localized to the spinal cord in both AIDS and non-AIDS patients.

**Management of AIDS Patients With Spinal Cord Lesions**

The management of an AIDS patient with an enhancing intramedullary spinal cord tumor poses problems. The initial evaluation should include serum and CSF cytology and antibody studies as well as complete radiographic imaging of the entire neuraxis. If immunological or cytological studies are diagnostic, focused treatment strategies can be implemented. Although magnetic resonance imaging has been reported by some authors to be more sensitive in its ability to detect CNS lymphoma and toxoplasmosis, others have reported equivalent sensitivity using delayed-contrast CT scanning. If multiple lesions, particularly lesions located in the basal ganglia, are detected, the diagnosis of *Toxoplasma* is more likely. Further diagnostic precision can be obtained using stereotactic biopsy of the intracranial lesions with much less risk of neurological morbidity than via open spinal cord biopsy. In the present case, immunological and cytological tests were not helpful and imaging studies revealed no intracranial lesions. Open biopsy was performed because the patient had shown no sign of improvement despite treatment for presumed spinal cord lymphoma.

The differential diagnosis of a solitary intramedullary spinal cord lesion should include toxoplasmosis. If a diagnosis cannot be made by means of noninvasive methods, open biopsy may be necessary in the patient who is experiencing rapidly declining function or in whom function fails to improve despite treatment. In a patient in whom the substantial risks of spinal cord biopsy are deemed inappropriate, empirical anti-*Toxoplasma* chemotherapy with radiological therapy may be used as part of the management strategy.

**References**


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