Mycobacterial Brain Abscess of the Cerebellum

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ABSTRACT
Central nervous system (CNS) tuberculosis is a serious form of extrapulmonary tuberculosis. CNS tuberculosis can present as meningitis, arachnoiditis, tuberculomas or the uncommon form of tuberculous subdural empyema and brain abscess. We present the clinical, radiological and pathological findings, and report on the successful treatment of an immunocompetent patient diagnosed with probable tuberculous brain abscess of the cerebellum. We also review the literature for the radiological studies, pathological criteria and treatment options of tuberculous brain abscess. (J Infect Dis Antimicrob Agents 2005; 22:71-6.)

INTRODUCTION
Tuberculosis is a major global public health issue. Central nervous system (CNS) tuberculosis is a serious form of extrapulmonary tuberculosis, occurring approximately 10 percent of all patients with tuberculosis.1,2 CNS tuberculosis can present as meningitis, arachnoiditis, tuberculomas or the uncommon forms of tuberculous subdural empyema3,4,8 and brain abscess. Almost all case reports of tuberculous abscess are described in HIV-infected or immunocompromised patients.1,6 We report on an immunocompetent patient who was diagnosed with probable tuberculous brain abscess of the cerebellum.

CASE REPORT
A 67-year-old Thai man had a 4-week history of vertigo, urinary incontinence, weakness of both legs and progressive confusion. He had neither fever nor any complaint of headache.

In his past medical history, he had been diagnosed with and treated for pulmonary tuberculosis 10 years ago from regional hospital, but he discontinued the antituberculous therapy by his own decision. Because of no available medical record, we had no any detail of previous medication.

On admission, the patient was found to have a temperature of 37.4 °C, pulse rate of 76 beats/min, respiratory rate of 20 breaths/min, blood pressure...
of 110/80 mmHg and fine crepitation of both lungs. Lymph node, liver and spleen cannot be palpated. Both tympanic membranes were normal. The neurological examination revealed alert but mild confused speech, truncal ataxia, impaired finger-to-nose test with dysdiadokinesia of the left side and paraparesis. He had neither nuchal rigidity nor abnormality on cranial nerve examination.

Laboratory examinations showed normal serum electrolyte levels, renal and liver function tests, and complete blood cell count showed 14.0 g/100 mL of hemoglobin, 6,600 white blood cells/mm$^3$ (66% neutrophils, 18% lymphocytes, 12% monocytes, 4% eosinophils) and 275,000 platelets/mm$^3$. A chest roentgenogram showed reticulonodular infiltration of both lungs (Figure 1). The sputum examination yielded negative result for acid-fast bacillus (AFB) stain, and the anti-HIV antibody was also negative. Cranial axial computed tomograms (CTs) with and without contrast enhancement revealed multiple isodensity nodules with rim enhancement and surrounding edema at the left cerebellar hemisphere, compressing the fourth ventricle. The lateral and third ventricles showed marked dilatation (Figure 2).

Because of the cerebellar lesion and obstructive hydrocephalus, he underwent an urgent suboccipital craniotomy to excise the nodular lesion, and ventriculostomy was applied to relieve the hydrocephalus. Cerebrospinal fluid (CSF) from ventriculostomy was clear. The mixed solid nodules and white, odorless, fluid-containing cysts were excised entirely. An intraoperative diagnosis of a cerebellar tumor was made, and thus CSF and the cystic content were not collected for examination. On the first postoperative day he regained full consciousness, and the ventriculostomy was removed on the fifth postoperative day.

The microscopic sections of the excised cysts showed the necrotic debris mixed with polymorphonuclear cell infiltration. The surrounding brain tissues showed edema, gliosis and granulation tissue formation.

![Figure 1. Chest roentgenogram showing reticulonodular infiltration of both lungs.](image-url)
Figure 2. Cranial axial computed tomograms without (A) and with (B) contrast enhancement showing the multiple isodensity nodules with rim enhancement and surrounding edema at the left cerebellar hemisphere, compressing the fourth ventricle. The lateral ventricles showed marked dilatation.

Figure 3. Microscopic sections showing A. necrotic debris with polymorphonuclear cell infiltration. The surrounding brain tissues showed edema, gliosis and granulation tissue formation. B. The AFB stain showing many acid-fast bacilli.

Neither epithelioid histiocyte nor Langhans type giant cell was seen (Figure 3A). The AFB stains showed many acid-fast bacilli (Figure 3B).

The patient was then diagnosed with pulmonary and cerebellar tuberculosis, and received rifampin, isoniazid, pyrazinamide and ethambutol. He was discharged home 11 days after admission with a course of these four antituberculous drugs for 2 months, and continued on isoniazid and rifampin for 12 months. The first-month follow-up examination revealed marked improvement of his neurological deficits. On the 6-month follow-up, he could walk using the orthosis, with clinically mild ataxia. The follow-up CT revealed no recurrence of cerebellar abscess, and the fourth ventricle was decompressed (Figure 4).

DISCUSSION

Mycobacterium tuberculosis is usually a systemic infection, transmitted through respiratory contact or by the percutaneous route. This case is an example of cerebellar tuberculosis occurring without an overt pulmonary focus.
Tuberculoma; rarely is it an abscess. For this reason, tuberculous abscess should be considered in patients with preexisting extracranial tuberculosis and presented with brain abscess especially individuals from areas where tuberculosis is endemic.

Tuberculous brain abscess is a focal collection of pus containing abundant AFB, surrounded by a dense capsule consisting of vascular granulation tissue. This condition is more commonly seen in immunocompromised patients who are unable to mount a granulomatous inflammatory response.

Granuloma formation or tuberculoma is the most frequent form of parenchymal brain tuberculosis, consisting of the focal mass of dense granulomatous inflammatory tissue that contains epithelioid and giant cells, not a true abscess.

The pathogenesis of tuberculous brain abscess is similar to other forms of CNS tuberculosis. It is postulated to be a hematogenous dissemination from the lung. The tissue reaction to AFB depends on the state of the host immunity, the inoculum size of the bacteria, the specific tissue infected and whether chemotherapy has been instituted. The inoculation of a small number of bacteria in a hypersensitive individual leads to a tubercle formation or tuberculoma, whereas a large amount of bacterial inoculation with leads to an exaggerated exudative response with caseation. The softening of the caseous material with an influx of polymorphonuclear leukocytes can form true pus. In addition, tuberculous brain abscess may develop from the absence of an adequate cell-mediated immune response in immunocompromised patients.

The appearances of tuberculomas on CT usually reveal small rings or nodular-enhancing lesion with only mild edema and mass effect, and on magnetic resonance imaging (MRI), they often have an isointensity or hypointensity center on T-2 weighted images. A tuberculous brain abscess is generally larger, solitary, frequently multiloculated, associated with considerable edema and mass effect, and typically produces a hyperintensity signal on T-2 weighted images in MRI scans at the center of the abscess. Large ring-enhancing lesion of a tuberculous brain abscess cannot be distinguished from a pyogenic brain abscess. A relatively long clinical history of symptoms may indicate tuberculous brain abscess.

In 1978 Whitener proposed the following criteria for establishing a diagnosis of tuberculous brain abscess.
abscess:

1) Macroscopic evidence of abscess formation within the brain parenchyma, characterized by cavity formation with central pus.

2) Histological confirmation that the abscess wall was composed of vascular granulation tissue, containing acute and chronic inflammatory cells, particularly polymorphonuclear leukocytes.

3) Proof of tuberculous origin by either a positive culture for *Mycobacterium tuberculosis* or demonstration of AFB in the pus or abscess wall.

Following these criteria, our patient was diagnosed with tuberculous abscess of the cerebellum despite the culture of *Mycobacterium tuberculosis* was not performed.

Because of the high mortality rate of tuberculous brain abscess, abscess aspiration can be performed for diagnosis and drainage, and antibiotic treatment should be started without delay if the lesion appears encapsulated by CT scan criteria. Whitener found that combined surgical excision and antituberculous chemotherapy results in more survivors than medical treatment alone. The minimal invasive technique, stereotactic aspiration, may be a useful alternative modality for surgical treatment in patients with deep seated and inaccessible lesion, such as in the thalamic region. Some authors recommend early excision of the lesion because the thick fibrotic capsule of a fully developed tuberculous brain abscess hampers catheter drainage.

As tuberculous abscess of the posterior cranial fossa risks rapid deterioration from brainstem compression, obstructive hydrocephalus and tonsillar herniation, many authors decided to surgically excise the abscess, as occurred with our patient. When a tuberculous abscess was present in the critical area such as brainstem, a successful treatment was reported with antituberculous drug only. Other reports have suggested that treatment with prolonged antituberculous chemotherapy may be adequate especially in patients who have relatively early lesions with poorly formed capsules.

There was also a successful case of tuberculous brain abscess in Thailand, reported by Chotmongkol et al in 1991. The patient developed neurological symptoms on the third month of antituberculous therapy for pulmonary tuberculosis. During therapy his chest symptoms improved despite worsening of the neurological conditions. Cranial CT showed a brain abscess, and surgical excision of abscess was done, and revealed pale greenish odorless pus which was positive for AFB stain. The patient was then continued antituberculous drugs for 6 months when the neurological recovery was observed.

CONCLUSION

The tuberculous brain abscess is a very rare but serious condition. Almost all reports are described in immunocompromised patients, but it can present in immunocompetent patients, as our report. Prompt surgical procedure for diagnosis and drainage with prolonged antituberculous chemotherapy lead to the successful outcome.

References

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