

Brain tuberculoma: Report of a case presented with prolonged nonspecific symptoms and multiple brain tuberculoma

Received: 10 Sep 2011
Accepted: 08 Dec 2011

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Keywords

Brain Tuberculoma, Diagnosis, Delayed

Five decades of tuberculosis control programs using potentially effective medications have been unsuccessful in diminishing the prevalence of the infection in most parts of the world and tuberculosis continues to kill all age groups.¹ Tuberculosis may manifest with atypical clinical manifestations and delayed diagnosis may give rise to unexpected grave outcomes. As a result, high degree of clinical suspicion is required to prevent late diagnosis.

A 18-year-old girl was admitted to the department of neurology at Sina Hospital (a teaching hospital of Tehran University of Medical Science, Tehran, Iran) with chief complaints of fever and headache. The patient had been well till three months before admission, when she developed malaise and anorexia. After two months, she got paresthesia in lower extremities and two weeks later was admitted at a local hospital. The physicians noted mild spastic paraparesis without any other abnormality on physical examination, chest radiography, spinal neuroimaging and routine laboratory examinations. Therefore, she received high doses of methylprednisolone, with the impression of possible

multiple sclerosis, but headache and projectile vomiting were added to her previous problems and she was referred to our hospital. On admission, the patient also complained of weight loss and night sweating.

On physical examination, she was generally wasted and drowsy. She had oral temperature of 38°C, pulse rate of 90 per minute, respiratory rate of 16 per minute and blood pressure of 110/80 mmHg and neck stiffness was present. No obvious abnormalities were detected in lung, heart, and abdomen and extremities examination. The patient was oriented but sleepy. Cranial nerves were intact. Right sided hemiparesis, with the proximal and distal strength of 4 out of 5 was detected and bilateral Babinski sign was present. Sensory and cerebellar exams were normal. She had only a hemiparetic gait.

Brain computerized tomographic scan (CT-scan) showed multiple small enhancing lesions in supra- and infra-tentorial structures, but there was no hydrocephalus. Brain magnetic resonance imaging (MRI) revealed similar lesions with no dural enhancement (Figure 1). These lesions were highly intense on T2 weighted MRI. The differential diagnoses included multiple brain abscess, multiple



Figure 1. Contrast-enhanced T1 weighted magnetic resonance imaging (MRI) showed multiple enhancing lesions with central hypointensity

tuberculomas and brain lymphoma. Cerebral toxoplasmosis was another possibility. The first chest X-ray was reported normal. The second chest X-ray with under penetrated films showed miliary tuberculosis. On chest CT-scan, tree-in-bud appearance was present which was in favor of miliary tuberculosis.

Lumbar puncture (LP) was performed for the patient. Cerebrospinal fluid (CSF) pressure was 280 mm H₂O. Findings of CSF analysis were as follows: glucose, 50 mg/dl; protein, 57 mg/dl; red blood cells (RBC), 2/ mm³; white blood cells (WBC), 0/ mm³; CSF Wright, VDRL and cytology were negative. CSF smear and culture for fungal infection and tuberculosis were negative. Complete blood count (CBC) was normal (WBC: 5600 /mm³, hemoglobin: 14 mg/dl). A purified protein derivative (tuberculin/PPD) had 5 millimeter induration. Erythrocyte sedimentation rate was elevated as high as 60 mm in 1 hour. Serum Wright and VDRL were negative. Serum glucose, creatinine, urea, sodium, potassium, calcium and liver enzymes

were within normal limits. Smear from gastric lavage was positive for acidfast bacilli.

As the patient was drowsy on arrival, anti-TB drugs were started immediately because of clinical and radiological suggestion of tuberculosis. The regimen consisted of isoniazid, rifampin, ethambutol and pyrazinamide with appropriate dosages and vitamin B6 was also added to the above drugs. Drowsiness got better after several days and her general condition improved after two weeks, but she had fever occasionally. After one month she became afebrile. The patient was discharged from hospital after one month with a good general condition. She was visited after one month at the clinic without any neurological deficits. Ethambutol and pyrazinamide were discontinued and treatment was continued by INH and rifampin. She completed the one year course of treatment without any complications. At the moment, after about two years, the patient is in a good general condition, without any symptoms or signs. On the last brain MRI, brain tuberculomas still are present but with smaller sizes and without enhancement.

In conclusion, the patients with brain tuberculoma may have nonspecific symptoms or show different neurological deficits in respect to the site of the lesions. CNS tuberculoma is a non-threatening condition with a good prognosis and effective therapy options. Enhanced brain and spine MRI should be considered to confirm that the diagnosis is not overlooked.² In our reported case, the patient have had malaise, low grade fever and nausea since several weeks before admission and on examination, we found only mild neurological deficit without obvious systemic signs. The brain CT and MRI showed nonspecific multiple lesions and the first routine chest X-ray did not show definite abnormality. The PPD test was negative. But we continued our work up and found other evidences that suggested tuberculosis as the best impression. Therefore, it seems that physicians should consider tuberculosis, as an important differential diagnosis of CNS diseases, especially in developing countries.

References

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