Thalamic Tuberculoma Mimicking a Malignant Brain Tumor

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Tuberculomas can occur at any site in the brain. In endemic areas of tuberculosis, intracranial tuberculomas account for 5% to 30% of all space-occupying brain lesions. A patient with frequent seizures presented to the neurosurgical department with a one-week history of progressive right upper limb weakness and numbness. CT scan and MRI findings revealed a malignant tumor in the left thalamus. The lesion was surgically excised because the patient's consciousness was rapidly deteriorating. Pathological examinations revealed a tuberculoma with mycobacterial infection. Sputum cultures revealed acid-fast stain positive bacilli infection two weeks after surgery despite normal chest X-ray findings. The patient took combined antituberculous drugs and recovered well without further neurological deficits during the two years of follow-up. Although rare, thalamic tuberculomas need to be considered in the differential diagnosis of intracranial lesions. **(Mid Taiwan J Med 2006;11:122-7)**

Key words

brain tumor, intracranial tuberculoma, thalamus

INTRODUCTION

Brain tumors arising from the deep brain are rare. Thalamic tumors occur predominantly in children and young adults and account for approximately 1% of all intracranial neoplasms [1]; in many cases, they are low-grade astrocytomas. Symptoms and signs of thalamic tumors include raised intracranial pressure (65%), motor deficits (40%), and seizures (35%) [1,2]. In endemic areas of tuberculosis, the incidence of intracranial tuberculomas ranges from 5% to 30% of all space-occupying brain lesions [3]. Thalamus is a rare location for an intracranial tuberculoma. However, no image findings are definitively diagnostic of intracranial tuberculomas. The diagnosis is therefore presumptive and based on supportive clinical data [4,5].

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CASE REPORT

A 37 year-old man with a history of hepatitis C, alcoholic liver cirrhosis, and recurrent pancreatitis, had 3 episodes of generalized tonic-clonic seizures within the previous year. CT scan, MRI of the brain, and EEG were performed following the first seizure one year ago; however, no specific findings were recorded except for diffuse brain atrophy. Alcohol withdrawal syndrome was the preliminary diagnosis. Visual and auditory hallucinations accompanied the second seizure 3 months prior to surgery, and repeated CT scan of the brain revealed a small slightly high-dense lesion with perifocal edema in left thalamus (Fig. 1A). The patient was admitted to the neurosurgical department because of a one-

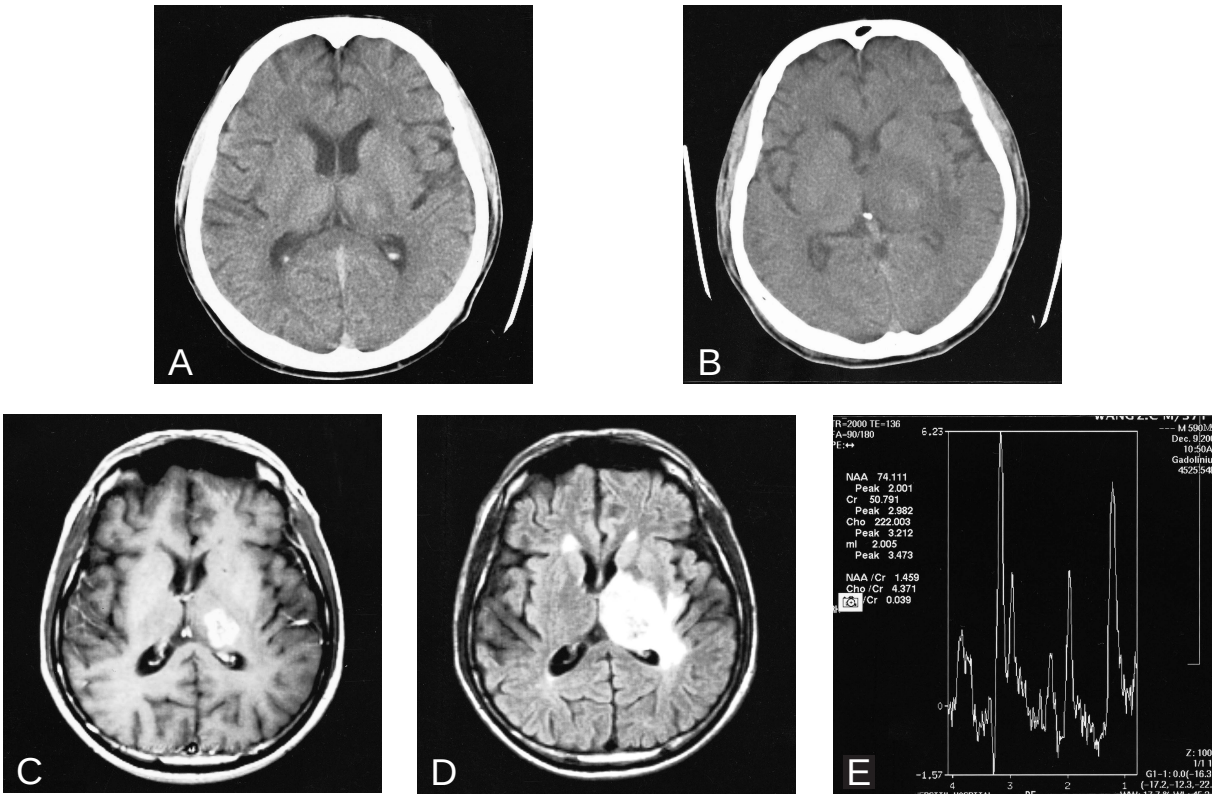

Fig. 1. A: Three months before surgery, non-contrast CT scan disclosed a slightly high-dense lesion at the left thalamus with perifocal edema and relatively early brain atrophy. B: Three days prior to surgery, non-contrast CT scan revealed that the central high dense lesion had enlarged and the perifocal edema had become more prominent. C: Axial T1WI with contrast disclosed a well-enhanced mass in the left thalamus with irregular margins and central necrosis. D: Axial FLAIR revealed a central hypointense lesion with severe perifocal white matter edema. E: Proton MRS shows increased choline/creatine ratio, increased lipid/creatine ratio, and decreased N-Acetylaspartate/creatine ratio. (The peak of N-acetylaspartate, × = 2.0; creatine, × = 3.0; choline, × = 3.2; and lipid, × = 1.2 ppm).

week history of progressive numbness and weakness in the right upper limb. Paresthesia over the right upper limb, right side of face, and dysmetria of the right hand were also noted. Muscle strength of the right upper limb was recorded as Brunnstorm stage 2-3. The patient had no fever, no body weight loss, and no history of mycobacterial infections. Serial chest X-ray studies revealed no specific findings. Follow-up CT scan (Fig. 1B) and MRI (Figs. 1C, 1D) of the brain disclosed a well-enhanced left thalamic mass with severe white matter edema. Proton magnetic resonance spectroscopy (MRS) showed increased choline/creatine ratio, decreased N-Acetylaspartate/creatine ratio, and increased lipid/creatine ratio (Fig. 1E). A malignant brain tumor was highly suspected initially. Steroid was given and stereotactic biopsy was scheduled. However, due to rapid deterioration of consciousness, the patient underwent surgical excision via a left transventricular (trigone) approach and superior parietal lobule cortical incision with the assistance of a navigation system to minimize optic radiation injury. Gross examination revealed a solid, whitish, and well-defined lesion measuring $2 \times 2 \times 2$ cm in the left thalamus. Pathological examinations revealed caseating granulomatous inflammation with Langhans giant cell formation. Many slender acid fast-positive bacilli were detected in the caseous necrotic and granulomatous regions by acid fast stain, confirming tuberculous mycobacterial infection (Fig. 2). Sputum cultures confirmed acid-fast positive bacilli infection two weeks after surgery.

Antituberculous drugs comprising

isoniazid, rifampicin, ethambutol and pyrazinamide were given for one year for both pulmonary tuberculosis and the intracranial tuberculoma. Follow-up MRI (Fig. 3) of the brain revealed no residual tuberculoma one year after surgery. Muscle strength of the right upper limbs returned to Brunnstrom stage 3-4 after rehabilitation. No further seizures, visual complications, or neurological deficits occurred during the two years of follow-up.

DISCUSSION

Epidemiology

The prevalence of tuberculomas varies in different countries and age groups. The incidence of tuberculosis also varies among different socio-economic classes. Tuberculomas can occur at any site in the brain. Arvind et al reported in a series of 1247 cases that the parietal hemisphere accounted for 47% of intracranial tuberculomas and that left-sided lesions were more common than right-sided ones [6]. Infratentorial tuberculomas are most commonly found in children, whereas a supratentorial locale is most common in older adults. In Taiwan, however, where CNS tuberculosis is common, tuberculomas have been reported to account for only 1% of all intracranial lesions [7]. According to a multicenter study in Taiwan, mycobacterial tuberculosis was the third common cause of encephalitis following herpes simplex virus and varicella zoster [8].

Diagnosis

Clinical manifestations of tuberculomas depend on their size and location. In supratentorial lesions, the most frequent symptoms and signs are raised intracranial pressure associated with focal or generalized seizures. Fever and evidence of systemic infection are rarely present, and patients are usually in good general condition [9]. The diagnosis of CNS tuberculoma is primarily based on clinical features, CSF changes, and imaging findings.

Thalamic tumors account for approximately 1% of all intracranial neoplasms. In many cases they are low-grade astrocytomas; however, primitive neuroectodermal tumors, ganglion cell tumors, oligodendrogliomas, lymphomas, and germinal neoplasms can also develop [2]. Clinical features of thalamic tumors reflect the pressure of the lesion on the CSF pathways, the pyramidal tracts, the thalamic nuclei, and on the optic radiations. An acute presentation is not rare, although most thalamic tumors have a subacute or slow evolution. Behavioral and mental changes are not uncommon and have been reported in 25% of patients with thalamic tumors [1]. Infants and young children with thalamic tumors may

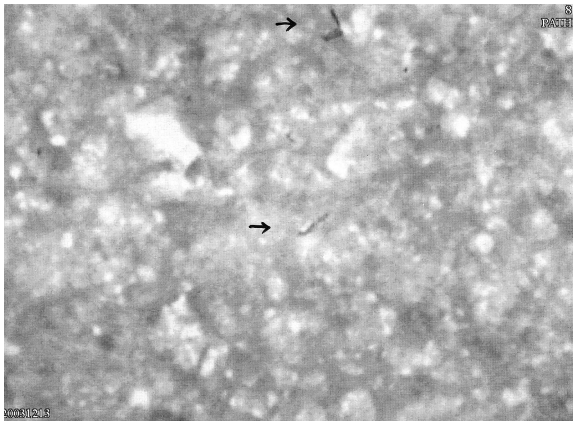

Fig. 2. Slender acid fast-positive bacilli are demonstrated by acid fast stain (×400) in the caseous necrotic and granumatous regions (arrows).

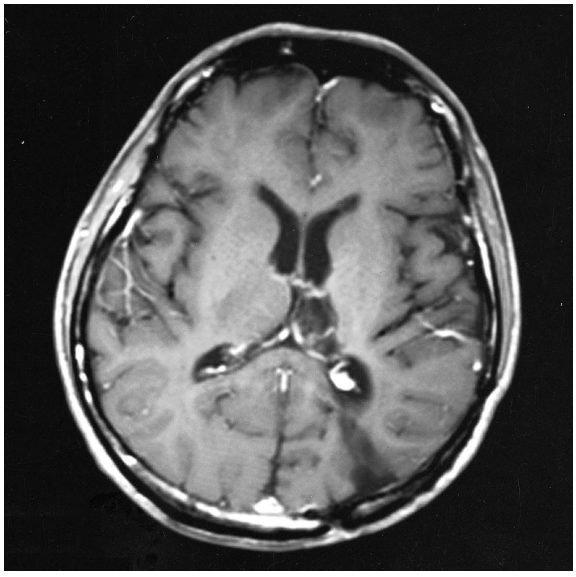

Fig. 3. One year after surgery, axial T1WI with contrast reveal no residual tuberculoma; however, brain tissue destruction is noted in the surgical trajectory and left thalamus.

present with macromegaly, psychomotor delay, visual impairment or ocular movement disorders. Our patient presented with all of the clinical features of thalamic tumors, although there was no evidence of extracranial mycobacterial infection before surgical intervention.

CT scan is the primary radiological modality for visualizing intracranial tuberculomas. In the first stage of formation, tuberculomas may appear as a non-enhanced lesion of low attenuation on CT scans (Fig. 1A). On T1-weighted MR images, this early lesion with associated edema appears hypointense. On T2-weighted images, the lesion appears well-demarcated but shows scattered areas of hypointensity, surrounded by edema, hyperintensity on T2, and represents a longer T2 relaxation time [5]. Most patients with intracranial tuberculomas receive medical attention at the time when a fully formed granuloma and hydrocephalus have developed. A plain CT scan in such cases is not diagnostic and reveals an isodense or minimally hyperdense lesion surrounded by a zone of edema (Fig. 1B). On T2-weighted MR images, these later-stage lesions have a hypointense necrotic center surrounded by a capsular ring, which is isointense with respect to the brain parenchyma. Edema is also present at this stage and is identified by hyperintensity consistent with long T2 relaxation time (Fig. 1D). Later-stage and early-stage lesions, and associated edema appear hypointense on T1-weighted images. Contrast enhancement reveals 3 types of characteristic patterns of a tuberculomatous lesion: 1) a solid enhancing lesion; 2) a ring enhancing lesion (Fig. 1C); and 3) mixed or combined forms of lesions. These lesions may be single or multiple, calcified or non-calcified [10].

Furthermore, proton MRS may detect the high lipid content of tuberculomas, as shown in this patient (Fig. 1E), which contributes to the hypointensity on T2-weighted images, thereby providing an additional diagnostic tool [11,12].

The diagnosis of intracranial tuberculomas on the basis of CT and MR images, however, is entirely presumptive (although MR is slightly more accurate). As there are no features that are exclusively unique to tuberculomas, the diagnosis must be based on other clinical features of the patient.

Treatment

Although medical regimens are the mainstay of therapy, surgical intervention has its role in the management of intracranial tuberculosis and tuberculomas. Recent reviews have shown that stereotactic or open biopsy of the lesion may hasten diagnosis. Stereotactic biopsies have been shown to be 85% accurate in the diagnosis of intracranial tuberculomas and these procedures are considered to be safe when performed by experts with an overall complication rate ranging from 0.6% to 6.3% [13]. Mohanty et al suggested that all suspected tuberculomas, except for large tumors and those located in deep-seated eloquent regions, should be stereotactically biopsied [13]. Rajshekhar and Chandy [14] reported that a diagnosis of chronic inflammation obtained by CT-guided biopsy, in correlation with the clinical and radiological findings, often provides confirmatory evidence of a tuberculoma in a patient with an intracranial mass; it also rules out a neoplasm and avoids empiric therapy of brain masses. The indications for surgical removal of tuberculomas are either progressive neurological signs and symptoms of raised intracranial pressure or non-responsiveness to antituberculous medications.

Although rare, thalamic tuberculomas need to be considered in the differential diagnosis in patients who present with intracranial lesions.

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表現出惡性腦瘤象徵之視丘結核

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在結核病盛行地區，顱內結核約佔顱內腫瘤的5至30個百分比，可發生在腦內之任何部位。視丘腫瘤約佔顱內腫瘤的百分之一，而視丘是顱內結核罕見之位置，文獻上僅有極少之報告。本文所呈現的案例，有經常癲癇發作的過去病史，其住院之主訴爲一星期漸進性的右上肢麻木及無力。電腦斷層及核磁共振檢查高度懷疑是一左側視丘的惡性腫瘤。病人由於腦壓升高合併意識惡化而接受外科手術。病理切片證實爲一顱內結核。術後二個星期的痰液培養證實有Acid-fast stain陽性反應的桿菌。患者接受多種抗結核的藥物治療後，追蹤之核磁共振顯示視丘結核已消失，病人恢復的情況良好且無進一步的惡化。儘管少見,視丘核結仍需列入顱內腫瘤的鑑別診斷。（中台灣醫誌 2006;11:122-7）

關鍵詞

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