



Case Report

Tuberculoma masked by glioma: a case report

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ABSTRACT. Tuberculous meningitis (TM), a common infectious disease of the central nervous system that is also seen in other types of tuberculosis infections, has higher mortality rates in young and middle-aged patients. TM is difficult to diagnose and treat owing to its non-specific clinical features and often atypical cerebrospinal fluid changes. Patients who present with focal neurologic signs, cough, low-grade fever and illness duration of more than 5 days, have intracalvarial abnormalities, and do not meet Thwaites' criterion findings should be diagnosed using computed tomography or magnetic resonance imaging. *Mycobacterium* infections can also be diagnosed by acid-fast staining of smears, cerebrospinal fluid culture, diagnostic polymerase chain reaction for *Mycobacterium tuberculosis*, and purified protein derivative test. To prevent TM misdiagnosis, clinicians must have sufficient knowledge of the clinical manifestations of tuberculosis. Appropriate application of tuberculosis chemotherapy drug principles, including early diagnosis and treatment, combination therapies, and consistent administration of treatment at appropriate dosages, can

greatly reduce TM mortality rates and improve satisfactory treatment outcomes.

Key words: Tuberculous meningitis; Diagnostic errors; Tuberculoma

INTRODUCTION

Tuberculous meningitis (TM), a common infectious disease of the central nervous system, is difficult to diagnose and treat due to its non-specific clinical features and often atypical cerebrospinal fluid (CSF) changes. Thus, TM has higher mortality rates in young and middle-aged patients. Herein, we described the case of a woman who was diagnosed glioma turned out to be TM, and analyzed the lack in our diagnosis and treatment.

CASE REPORT

A 37-year-old-woman presented to our Neurology Department on March 1, 2011 for left limb weakness and sensory disturbance. She reported a fever and cough that had started on February 19. She received anti-inflammatory drugs from her local clinic, but the fever and cough did not improve, and her left limb weakness and sensory disturbance developed after treatment. The left limb weakness increased and she was unable to execute fine functions with her left hand and she was unable to walk. The patient had no headache, nausea, vomiting, commissure distortion, or other neurologic defects. She had no specific personal or family history of past illness. Initial blood laboratory examinations were normal except for blood sedimentation (32 mm/h); chest radiographs also showed normal findings.

The patient's body temperature was 36°C. Her blood pressure and general state of health were normal. Her breath sounds were coarse but without rales. Cardiac auscultation and abdominal touch examinations were both normal. The patient was conscious and could speak. Myodynamic testing showed left upper grades of IV⁺, left lower grades of IV, and right extremity grades of V, respectively. Alternate motion of her left upper extremity was awkward and both sides had positive Hoffmann and negative Babinski signs. Additional physical examination of her nervous system yielded normal results. Based on these clinical observations, we suspected 1) non-specific encephalitis, 2) cerebral infarction caused by cerebral vasculitis, or 3) brain abscess. The patient was administered piperacillin and sulbactam upon hospital admission.

On March 1 and 2, 2011 her temperature increased to about 38°C. Her left limb myodynamia decreased. She had no headache, nausea, or muscular spasms, but talked nonsense. Laboratory examination showed the following results: hemoglobin, 102 g/L; white blood cell (WBC), $4.7 \times 10^9/L$ (neutrophils, 56.18%); prothrombin time, 12.06 s; fibrinogen, 3.6 g/L; creatine kinase-MB, 13.4 U/L, as well as normal liver and renal functions and antinuclear antibody levels. Electrocardiograph findings were normal. On March 3, head magnetic resonance imaging (MRI) revealed isointensity and a hypointense lesion in the right frontal lobe on T1-weighted images and an asymmetrical hyperintense region on T2-weighted images. A head computed tomography (CT) examination showed an irregular low-density lesion (CT value: 28 HU) in the right frontal lobe. Neurosurgeons consulting on this case suspected the lesion to be a glioma and recommended the patient be transferred to the Department of Neurosurgery for appropriate treatment. She was admitted to the neurosurgery department on March 4 with

a temperature of 38.8°C. Myodynamia grades of her left upper and lower extremities had decreased to III and II, respectively. On March 5, CT examination showed slightly coarse lung markings and a small quantity of hydrops in both thoracic cavities. B-ultrasound revealed a hepatic cyst and increased spleen thickness. The neurosurgeons believed the lesion in her head to be a glioma, but also considered the possibility of an intracranial infection. Combined with treatment for a potential pulmonary infection, the patient continued piperacillin and sulbactam therapy. CSF examination showed a CSF pressure of 100 mmH₂O, red blood cell (RBC) concentration of 1000 × 10⁶/L, WBC count of 10 × 10⁶/L, glucose >50 mg/dL, albumin 22.5 mg/dL, chlorine 118 mM, negative Pandy test. Globulin, purified protein derivative, sputum, and tumor marker tests were all negative. One week after starting anti-inflammatory treatment, the patient still had a low-grade fever and left limb myodynamia grades of 0. Babinski signs were negative for both lower limbs. On March 12, CT examination showed cerebral edema in the right part of the lobus parietalis and forehead; enhancement scanning showed that the lesion might be a cerebral infarction. On March 21, another head CT revealed a low-density lesion along the sulcus of the right parietal lobe. On March 23, MRI indicated possible multiple metastatic tumors, and biopsy of the right parietal lobe was performed. Parts of the right parietal lobe tissue were isabelline, with punctiform pus. Examination of frozen sections revealed inflammatory necrotic tissue without allotype cells, no parasites, or tubercular nodules. Examination of the frozen sections by the Department of Pathology of the Second Affiliated Hospital of Zhejiang University reported chronic granulomatous inflammation and positive acid-fast staining for bacillus. The patient was finally diagnosed with tuberculoma and received antituberculosis chemotherapy. On April 3, she presented to the Hospital for Infectious Diseases for antituberculosis chemotherapy treatment, and her temperature decreased. However, the patient continued to have constant psychiatric symptoms. Postdischarge, she received irregular antituberculosis chemotherapy until she was readmitted on May 15 for worse symptom. On May 18, she was discharged from the hospital in a coma, with right-sided mydriasis and cephalocele from a skull defect. The patient died 2 days later.

DISCUSSION

Clinical criteria for TM diagnosis reported by Thwaites et al. (2004) include a “definite” TM diagnosis when acid-fast bacilli are present in the CSF. TM is “probable” in patients with one or more of the following: suspected active pulmonary tuberculosis on chest radiography, acid-fast bacilli found in any specimen other than CSF, and clinical evidence of other extrapulmonary tuberculosis. TM is “possible” in patients with at least four of the following: history of tuberculosis, predominance of lymphocytes in CSF, illness duration of more than 5 days, a CSF to plasma glucose ratio less than 0.5, altered consciousness, yellow CSF, and focal neurologic signs.

Thwaites’ criteria have been verified in our country by Li et al. (2007). They evaluated 68 TM cases using Thwaites’ criterion and found clinical indices and higher masculine rates associated with altered consciousness (100%), an illness duration of more than 5 days (99%), predominance of lymphocytes in the CSF (80%), and a CSF to plasma glucose ratio of less than 0.5 (70%) as well as CT or MRI abnormalities (59%) and eyeground changes (49%).

We analyzed this case using Thwaites’ criteria owing to the patient’s altered consciousness, illness duration of more than 5 days, and focal neurologic signs combined with head CT and MRI results; however, diagnostic errors such as this, which lasted for more than 1

month, should be avoided. Although the patient received antituberculosis chemotherapy, drug administration was irregular after she was discharged from the hospital. On May 15, she died shortly after being readmitted for intracranial hypertension.

This case emphasized several important lessons. First, the importance of detailed case history: the patient had a fever and cough as well as focal neurological signs. Second, abnormal CT findings were ignored. Third, the count of CSF's RBC and WBC were not considered since the CSF pressure was normal. Fourth, we should have performed additional tests such as smears for *Mycobacterium* and acid-fast staining, CSF culture, diagnostic polymerase chain reaction for *Mycobacterium tuberculosis*, and purified protein derivative tests. Finally, irregular administration of antituberculosis drugs aggravated this case.

In June, 2009, the British Infection Society released guidelines for treatment of tuberculosis of the central nervous system that included isoniazid, rifampicin, and pyrazinamide as a core group of antituberculosis drugs, and suggested selecting one of three drugs-ethambutol, streptomycin, or fluoroquinolones as the fourth drug as a combination treatment for tuberculosis (Thwaites et al., 2009). In this case, the patient improved with a regimen comprising isoniazid, rifampicin, pyrazinamide, and streptomycin. However, irregular drug administration led to patient relapse and death. Therefore, prompt diagnosis and regular treatment with a combination of four antituberculosis drugs are essential for patients with TM.

Conflicts of interest

The authors declare they have no conflict of interest.

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