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Primary Brain Tuberculoma Associated with Tuberculous Meningitis: A Case Report

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1. Introduction

Intracranial tuberculoma is usually the result of hematogenous infection dissemination. Primary intracranial tuberculoma is a relatively rare extrapulmonary tuberculous lesion, especially when associated with Tuberculous Meningitis (TBM). It is difficult to diagnose because it's signs and symptoms are not specific, so misdiagnosis may delay the treatment. Tuberculoma is neither the complications of TBM nor the result of its progression. Only less than 10% of TBM are associated with tuberculomas [1].

2. Case Report

A 20-year old female presented with a 10-day history of headache and vomiting, with fever of 38^0C. Symptoms were relieved after antibiotic therapy at a local hospital. After one day, she was transferred to our hospital because of recurrent headache and fever. No cough or expectoration was observed during the progression of the disease. The patient denied any history of Tuberculosis (TB) or any contact with TB [2].

Neurological examinations on admission revealed no abnormalities. The patient's body temperature was 38.2^0C. Magnetic Resonance Imaging (MRI) (Figure 1) revealed a lesion in the right frontal lobe. Initial peripheral blood counts showed WBC 7.31 × 109/L (percentage of neutrophils = 68.04%, lymphocytes = 13.11%, and monocytes = 18.2%). The chest film and blood culture were normal [3].

The initial diagnosis was brain abscess. Ceftriaxone and vancomycin were administered as combined antibiotic therapy. During the five-day treatment, the patient's temperature gradually increased. Purified Protein Derivative (PPD) test was negative reaction. Cerebrospinal Fluid (CSF) was colorless and the pressure was higher than 300 mmH2O. The CSF contained 100 × 106/L WBC, 1.62 g/L protein (0 to 0.41 g/L), and 1.48 mmol/L glucose. Lactic acid in the CSF was 7.1 mmol/L; CSF TB-DNA was detected by fluorogenic quantitative Polymerase Chain Reaction (PCR) was 3.27 × 103 copy/mL. The CSF immunoglobulin (IgA, IgM, and IgG) concentrations were 52.3 mg/L, 18.2 mg/L, and 52.3 mg/L respectively. Cytology of the CSF showed a mixed reaction; 79% of the cells were small lymphocytes; the tryptophan test was positive and the ink stain was negative. Serum Adenosine Deaminase (ADA) was 46 U/L and the anti-TB antibodies were positive. Then the patient was started on an anti-TB regimen consisting of rifampicin, isoniazid, and sodium aminosalicylate. However, the patient's condition aggravated rapidly [4]. On the 12th day of hospitalization, the patient suffered from cerebral hernia. Emergency surgery for resection of the right frontal lobe lesions and decompressive craniectomy were performed (Figure 2). Pathologic examination revealed brain tuberculoma and TBM. On the second postoperative day, the patient died of cardiorespiratory failure.

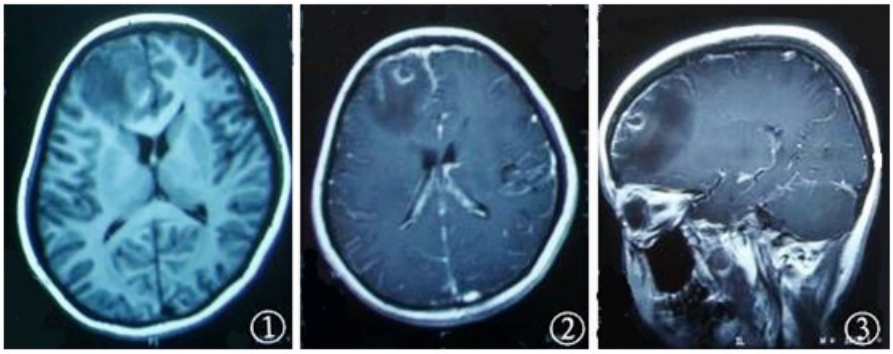

Figure 1: MRI shows a lesion in the right frontal lobe. Round reinforcement was observed after gadolinium injection. The lesion was surrounded by evident edema

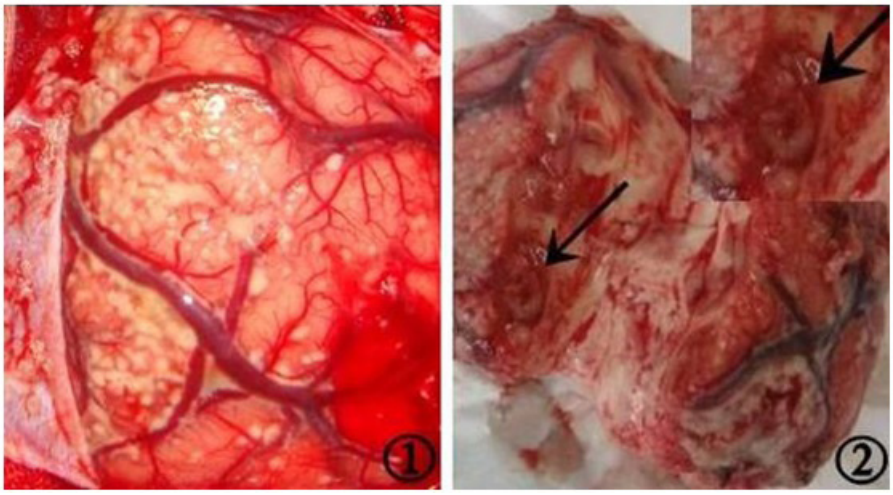

Figure 2: Spotty purulent secretion was found on the surface of the brain. There was focal necrosis (arrow) inside the lesion

3. Discussion

TB of the Central Nervous System (CNS) is usually results from hematogenous dissemination from other organs. Extraneurological TB can be found in many, but not all patients. Tuberculosis bacilli pass through the blood–brain barrier, so minute lesions can be found on the surface of the brain. Lesions in the brain may develop into tuberculomas or TB abscess, which are dependent on the cellular immunity of the host. TB in the CNS is the severe or acute type of the TB, with high morbidity and mortality. Some cases have atypical clinical course, without primary extraneurologic lesions, which may result in misdiagnosis and, consequently, poor prognosis. In 2009, Ku Bon reported a case suffering extensive meningeal and prenchymal calcified tuberculomas as long-term residual sequelae of TBM; however, there is no similar report as the present case [5].

The present case provides information on enhancing the diagnosis and the cure rate of TB in the CNS. The clinical manifestations of TB in the CNS vary. Special attention should be given to patients suspected of having CNS infection with no improvement after 3 to 5 days of antibiotic therapy to exclude the possibility of specific infections. The CSF should be tested as early as possible.

Several specific tests are helpful for early diagnosis of CNS TB. A PPD test should be performed for patients suspected with tuberculosis, although some have false negative results. In testing the CSF, Mycobacterium tuberculosis DNA testing is the gold standard, with 51% to 85% positive rate and 98% to 100% specificity rate respectively. The value of ADA in the blood and CSF can be an important index for identifying TBM or non- tuberculous meningitis, especially when the results of other tests are atypical. The presence of anti-TB antibodies in the serum also has certain specificity. For patients who cannot be definitively diagnosed for tuberculosis, experimental anti-TB therapy may be an alternative treatment modality.

4. Conclusion

Cerebral TB with atypical clinical manifestations and no extranervous tuberculous lesion is rarely seen. For patients clinically suspected of intracranial infection, whose fever cannot be controlled by antibiotics, we should conduct a comprehensive examination. Specific tests of Mycobacterium tuberculosis DNA in the CSF and ADA in both the blood and the CSF should be performed as early as possible to exclude TB lesions, because any hesitance may lead to delayed diagnosis and consequent poor outcome.

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