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A Case of Rheumatoid Arthritis Presenting with A Brain Mass

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Abstract

Among patients with tuberculosis (TB), approximately 1 to 5 percent develop central nervous system (CNS) complications. A tuberculoma is a conglomerate granulomatous focus that develops from coalescing tubercles acquired during disseminated bacillemia. Radiographically, tuberculomas are discrete, ring- enhancing lesions of the brain surrounded by perilesional edema and may present as single or multiple lesions. The diagnosis of tuberculoma should be suspected in patients with mass lesion of the brain and relevant epidemiologic factors. A definitive diagnosis of tuberculoma is established via needle biopsy of the CNS lesion for histopathology and acid-fast bacilli (AFB) staining and culture.

In this case report, we present a 43-year-old woman with a past medical history of rheumatoid arthritis (RA) managed with steroids and hydroxychloroquine, who attended to us with a two- month history of numbness and paresthesia of her left extremities and recurrent episodic jerks involving the same side. Brain magnetic resonance imaging (MRI) revealed a large hemorrhagic mass (55 * 40 mm) in right temporo-parietal lobe surrounded by edema and mass effect with small nodule in left perirolandic cortex. Following clinical and laboratory clues, MR spectroscopy and brain biopsy, we started with corticosteorids and the following 4 anti-TB drugs: Isoniazid, Rifampin, Pyrazinamide, Ethambutol (HRZE). After 2 months, her symptoms completely resolved and a subsequent MRI scan revealed considerable shrinkage of the right-sided mass and left-sided lesion.

Keywords: Tuberculoma, Brain, Granuloma, Treatment

List of Abbreviations:

TB: Tuberculosis
CNS: Central Nervous System
RA: Rheumatoid Arthritis
MRI: Magnetic Resonance Imaging

ICP: Intra Cranial Pressure
CSF: Cerebro Spinal Fluid
AFB: Acid-Fast Bacilli
MTB: Mycobacterium Tuberculosis
EEG: Electro Encephalo Gram
LP: Lumbar Puncture
WBC: White Blood Cell
CT: Computerized Tomography
HSV: Herpes Simplex Virus
EBV: Epstein-Barr Virus
HIV: Human Immunodeficiency Virus
CPS: Cycle Per Second
NAA: N-Acetyl Aspartate
Cr: Creatinine

Introduction

Central nervous system (CNS) infection should always be considered in immunocompromised patients presenting with new neurological symptoms. These infections have a poor prognosis, especially if there are seizures, severe changes in mental status, or an increase in intracranial pressure (ICP) Diagnosis of CNS infection should be based on a systematic approach, including patient history (clinical signs, temporal evolution), features of brain imaging, and cerebrospinal fluid (CSF) analysis. [1]

One of the infections that should be considered in immunocompromised patients is Mycobacterium tuberculosis (MTB) infections, especially in areas that are endemic for MTB. Among patients with tuberculosis (TB), approximately 1 to 5 percent develop CNS TB [2]. CNS involvement in the form of meningitis, encephalitis, tuberculous arteriopathy, tuberculoma, abscess, infarct or miliary parenchymal lesions can be seen in patients with TB [3]. A tuberculoma is a granulomatous focus that results from coalescing tubercles obtained during disseminated bacillemia. These manifestations are more common in endemic areas and usually involve children or young adults presenting with headaches, seizures, progressive hemiplegia, and symptoms of increased intracranial pressure.

Radiographically, tuberculomas are single or multiple ring-enhancing lesions of the brain surrounded by edema. [4, 5, 6] The differential diagnoses include malignant lesions, sarcoidosis, pyogenic abscess, toxoplasmosis and cysticercosis. Therefore, this is a challenging clinical diagnosis [7].

The diagnosis of tuberculoma should be considered in patients with mass lesion of the brain and related epidemiologic factors, such as history of previous TB infection or disease, known or possible TB exposure, and/or past or present residence in or travel to an area where TB is endemic. A definitive diagnosis of tuberculoma is determined by needle biopsy of the CNS lesion for histopathology and acid-fast bacilli (AFB) staining and culture. In addition, lumbar puncture (LP) is usually avoided due to concern for increased intracranial pressure and risk of brainstem herniation [8]. Anti-tuberculosis drugs are essential for the successful treatment of intracranial tuberculomas although there is no agreement on the duration of therapy. [9,10]

We report a case of an intraventricular tuberculoma in a 43-year-old woman and we describe the radiological investigations and laboratory findings.

Case Presentation

In May 2021, a 43-year-old woman was admitted to the Neurology Department at Mehr Hospital in Tehran, Iran. The patient was a housewife from Sari, acity located in North of Iran. She had a two-month history of numbness and paresthesia of her left extremities and recurrent episodic jerks involving the same side. A plain magnetic resonance imaging (MRI) showed a large hemorrhagic mass (55 * 40 mm) in the right temporo-parietal lobe surrounded by edema and mass effect with small nodule in the left perirolandic cortex (Figure 1), for which she was referred to our department for further work up.

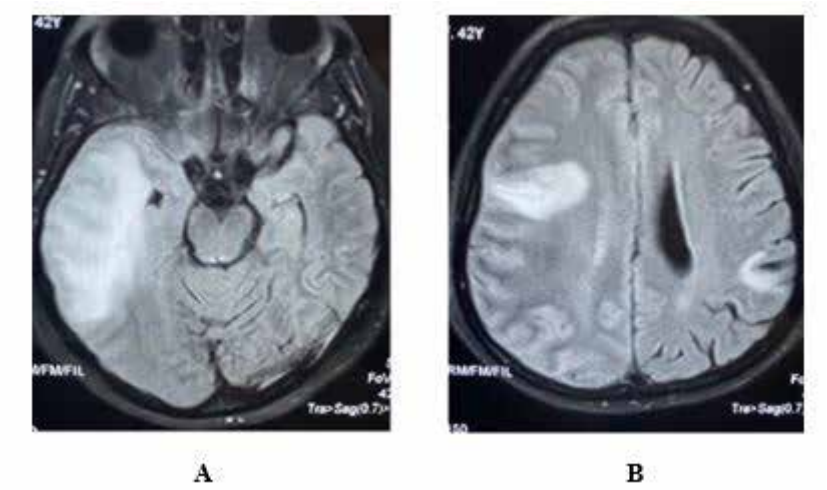

Figure 1. Image A: MRI showed a large hemorrhagic mass (55 * 40 mm) in the right temporal lobe surrounded by edema and mass effect. Image B: MRI showed a hemorrhagic mass in parietal lobessurrounded by edema and mass effect with a small lesion in the left perirolandic cortex

Upon admission, she denied any history of fever, weight loss, night sweats, headache, seizure, loss of consciousness, double or blurred vision, changes in her menstrual cycle or decreased power in extremities. She had a past medical history of rheumatoid arthritis (RA) for 2 years and her drug history included 5 mg prednisolone tablet daily and 200 mg hydroxychloroquine tablet twice a day. Her family history for cancer or tuberculosis was negative. Review of her systems did not reveal any abnormalities. Lungs were clear on auscultation and no organomegaly could be palpated. The neurological evaluation only revealed depressed mood, and the patient was well- oriented with a Glasgow Coma Scale of 15/15. Cranial nerves and optic disc examinations were normal. Force of her extremities were normal and deep tendon reflexes were 2+ but there was a positive left Babinski sign and dysesthetic sensation to touch and pinprick in left leg. Gait and coordination were normal.

Laboratory investigations were done following the initial presentation to our department. (Table 1) Paraneoplastic neurologic syndrome panel and metabolic panel were negative with positive rheumatoid factor. Lung, abdominal and pelvic computerized tomography (CT) scan were normal. LP results showed clear appearance and normal opening pressure, WBC count 16 cell/microL (90% lymphocyte with negative cytology for malignant cell), protein 45mg/dL, glucose 74 mg/dL (normal CSF/plasma glucose ratio) , and a negative CSF culture for bacterial and fungal pathogens. Polymerase chain reaction (PCR) was also negative for MTB, Herpes Simplex virus (HSV), Epstein-Barr virus (EBV), Toxoplasma gondii and Human Immunodeficiency virus (HIV).

WBC count	$9.8*10^3$
Hemoglobin	12.4 g/dl
Hematocrit	39.7%
Platelet	$187*10^3$ µl
ASO	200
CRP	1+
ESR	30 mm/hour
PPD test	negative
Quantiferon test	negative
Liver function test	normal
Thyroid function test	normal
HIV Ab	negative

Table 1: Initial blood test findings

Electroencephalogram (EEG) showed synchronous and symmetrical 7 to 9 cycle per second (CPS) waves mixed with few brief runs of 2 to 3 CPS activities seen only during hyperventilation, in the right hemisphere (Figure 2). Cerebral MR angiography (MRA) was normal. In the next step, a 3D chemical shift imaging MR spectroscopy with color coded map was obtained. Measuring different metabolites including N-acetylaspartate/creatinine (NAA/Cr), Choline/creatinine (Choline/Cr), and choline/N-acetylaspartate (Choline/NAA) ratio, reporting an increase in that up to near 2.5-3- fold. It was concluded that there was a hemorrhagic mass in the right hemisphere, with mass effect without diffusion restriction. Metastasis or Glioma grade II-III reported as differential diagnoses (Figure 3).

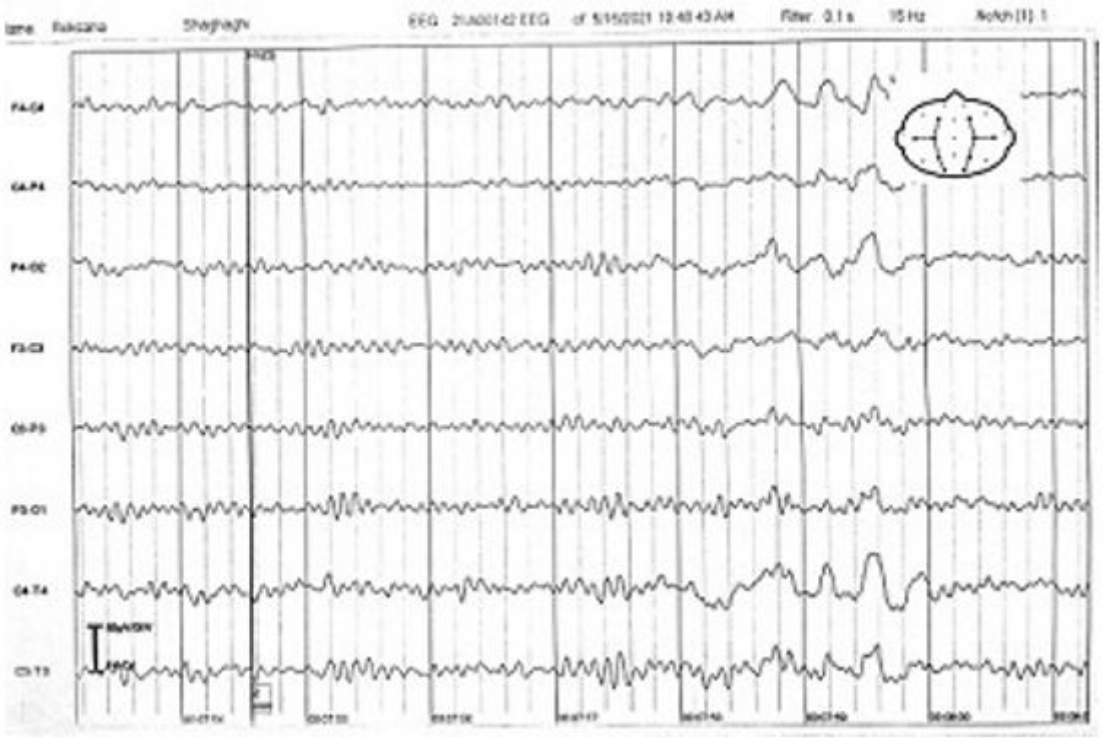

Figure 2: Electroencephalogram (EEG) showed synchronous and symmetrical 7 to 9 CPS waves mixed with few brief runs of 2 to 3 CPS activities seen only during hyperventilation, in the right hemisphere

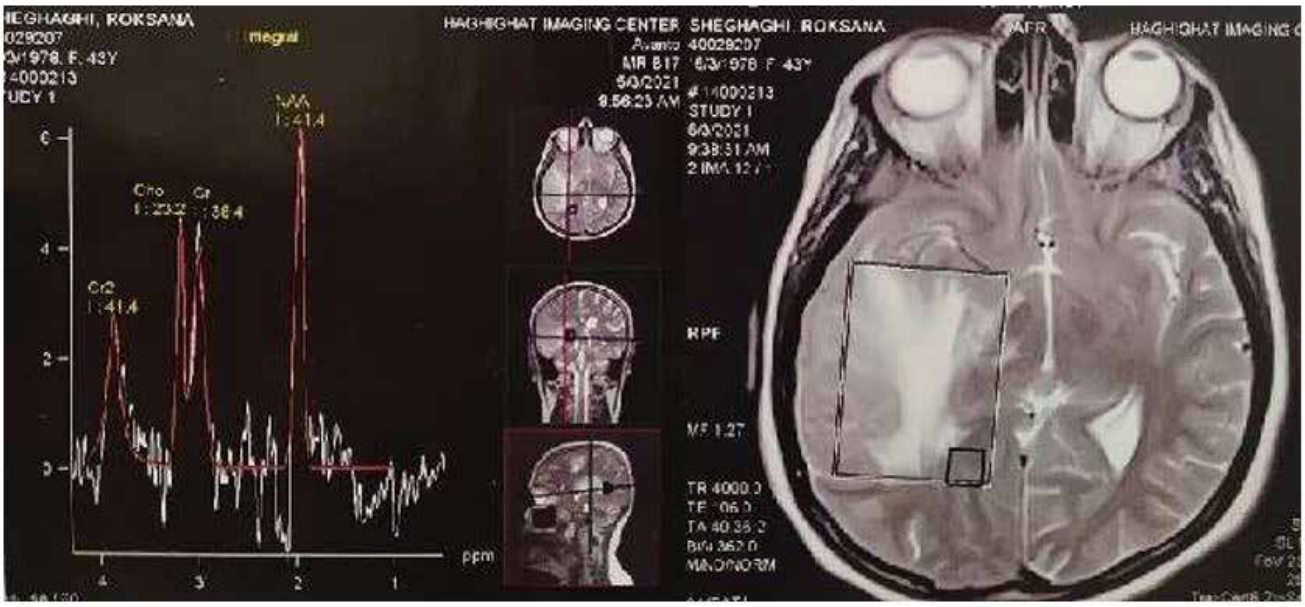

Figure 3: MR spectroscopy with color coded map was obtained. Measuring different metabolites including NAA/Cr, Choline/Cr, and Choline/NAA ratio, reporting a rise in that up to near 2.5-3- fold. It was concluded that there was a hemorrhagic mass in the right hemisphere, with mass effectwithout diffusion restriction. Metastasis or Glioma grade II-III reported as differential diagnosis

At this stage, despite the extensive work up, we had not yet reached a definite diagnosis for this patient. A stereotaxic biopsy sample of the patient's brain lesion was sent for smear, culture and histopathology. The pathology result was dense perivascular lymphocytic infiltration and granulomatous lesion. The differential diagnosis were lymphoma, RA, lupus and TB (Figure 4). However, attention to some pieces of positive and negative information obtained from the patient, history, laboratory and radiological aspects, as mentioned below, suggested that tuberculoma was highly suspicious as the offending etiologic factor.

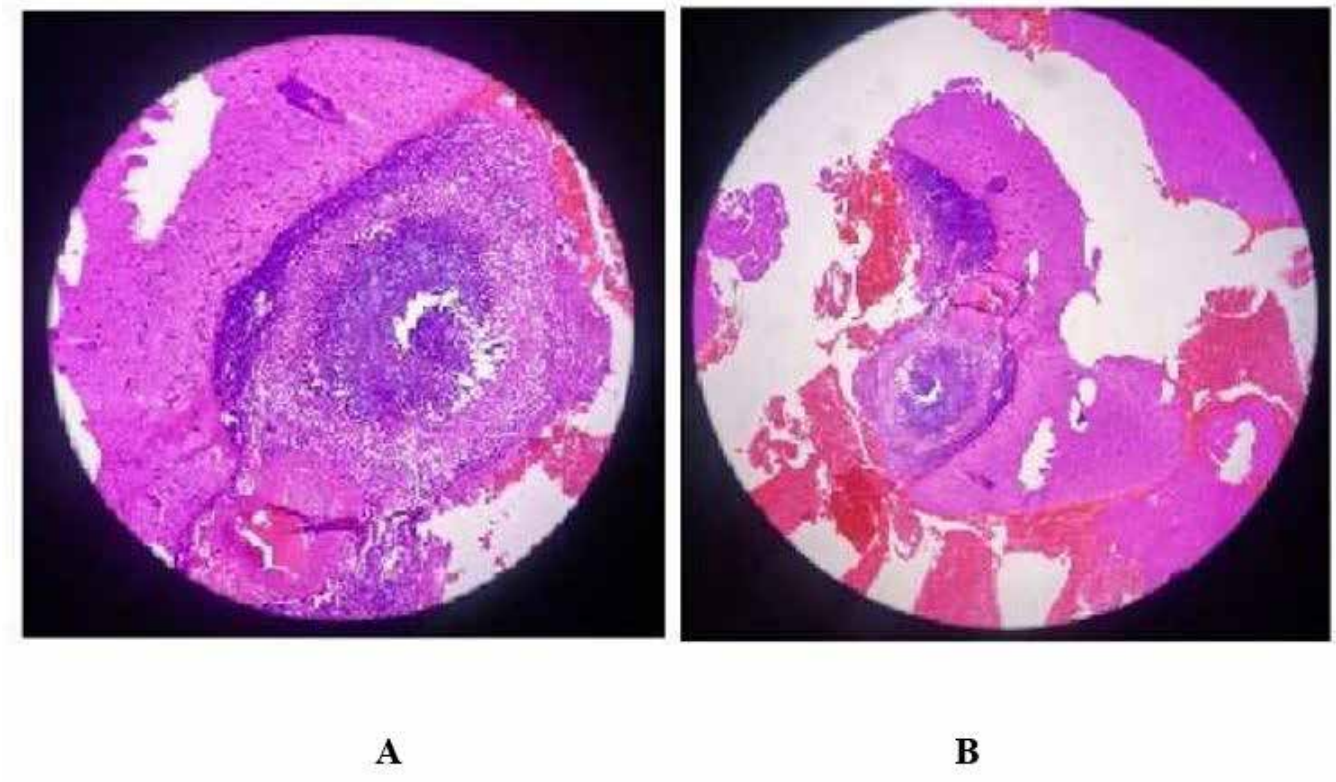

Figure 4. Images A and B: The pathology result showed dense perivascular lymphocytic infiltration and a granulomatous lesion

The course of disease was a chronic or subacute one, indicating the presence of slow growing lesion with minor clinical symptoms.

1. No headaches or papilledema with such a large mass.
2. LP with WBC of 16 cells/microL and other parameters being within normal range denotes the possibility of a chronic infectious process.
3. With such a large mass, brain MR Angiography was normal without abnormal vasculature or tumor blush.
4. It is unusual for such a large mass to have minor EEG abnormalities (Figure 2).
5. Patient was on corticosteroids for RA.

The patient was treated with a standard anti-TB drug regimen (Isoniazid, Rifampin, Pyrazinamide and Ethambutol) and corticosteroids. She was discharged to be followed in the outpatient clinic. After two months, the patient was completely asymptomatic, and treatment was reduced to maintenance therapy with 2 drugs (Isoniazid, Rifampin). Post treatment brain MRI with and without contrast media showed remarkable shrinkage of the right hemisphere mass (8 * 10mm) as well as the left-sided lesion (Figure 5).

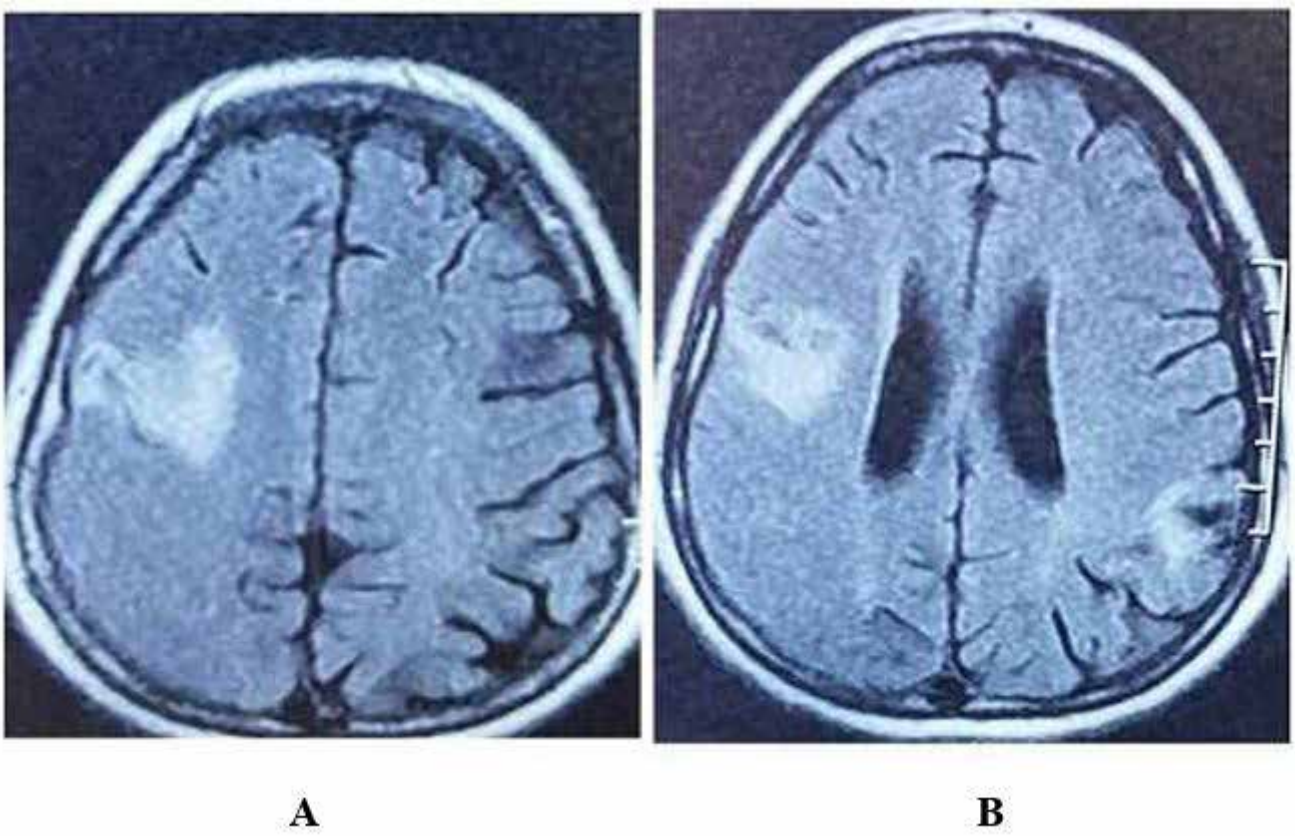

Figure 5. Image A: Post-treatment brain MRI with and without contrast media showed remarkableshrinkage of right hemisphere mass (8 * 10mm). Image B: Post-treatment brain MRI with and without contrast media showed the left-sided lesion

Discussion

The approach to the diagnosis of infection in patients with immunosuppression is different from the usual clinical approach. Clinical manifestations may be subtle or atypical due to an altered inflammatory response, and some investigations will be of limited use. Treatment should be based on identification of the organism and choosing the appropriate antibiotics [11]. The diagnosis and management of CNS infections in immunosuppressed patients creates a difficult challenge for neurologists. Effective care for these patients requires attention to patients' underlying disease, prophylactic and vaccination strategies, community and nosocomial epidemiologic trends, travel and zoonotic exposure histories, and changes in microbial susceptibilities. [12] In these situations, bacteria, viruses, fungi and parasitic infections. Should be considered and investigated appropriately [11].

CNS involvement is one of the most serious forms of MTB infections. Clinical CNS MTB involvement include meningitis, intracranial tuberculoma and spinal tuberculous arachnoiditis [13]. Among these, intracranial tuberculomas are the least common presentation of CNS TB, found in 1% of these patients [3]. They present as multiple lesions in only 15%-33% of the cases [9]. Large tuberculoma can mimic malignant brain tumors because of the clinical, laboratory and radiologic features of cerebral tuberculomas as nonspecific, which may lead to the wrong diagnosis and at times, unnecessary surgery as part of the treatment [14]. Other differential diagnoses of intracranial focal lesions are fungal granulomas, primary, and metastatic neoplasms. In asymptomatic patients with suspected brain mass of non-neoplastic lesions, stereotactic brain biopsy is preferred for diagnosis. When the stereotactic brain biopsy is followed by paraffin sectioning and histopathological examination, the diagnostic efficacy is 85%. In non-diagnostic results, open brain biopsy should be considered for a definitive diagnosis. Brain tuberculoma is treated with an anti-tuberculosis regimen starting with four medications and usually without surgical intervention. Surgical intervention in a patient with CNS tuberculoma is indicated in patients with acute symptoms due to increased ICP or in those with imaging that shows a persistent or enlarged mass after at least three months of anti-TB drugs. The treatment response is monitored by brain CT scan or MRI [15].

The presence of two brain lesions in our case suggested metastases in the first place. However, the patient underwent extensive laboratory and neuroimaging work up, but none led us to any definitive diagnosis. A sample of the patient's brain lesion was sent for smear, culture and histopathology with stereotaxic biopsy. The pathology result was dense perivascular lymphocytic infiltration and granulomatous lesion. The differential diagnoses were lymphoma, rheumatoid arthritis, lupus and TB. With respect to few clues obtained from disease course, patient, clinical finding and neuroimaging, we were convinced that the patient's symptoms and brain lesions are most probably due to tuberculoma. We decided first to treat patient with a short course of chemotherapy for tuberculosis. Although the treatment response is unpredictable, with lesions sometimes enlarging paradoxically or persisting for many years despite appropriate TB treatment and corticosteroid therapy [16], treatment for our patient resulted in fast improvement of clinical and neuroimaging findings.

Conclusion

The diagnosis of tuberculoma should be suspected in patients with mass lesion of the brain and relevant epidemiologic factors, such as history of prior TB infection or disease, known or possible TB exposure, and/or past or present residence in or travel to an area where TB is endemic. A definitive diagnosis of tuberculoma is established via needle biopsy of the CNS lesion for histopathology and AFB staining and culture. Our case report highlights the importance of consideration of intracranial tuberculoma in the differential diagnosis of CNS lesions in high-risk patients, such as those who are immunosuppressed.

Acknowledgments

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