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Meningeal Tuberculosis Masquerading as Meningioma: A Case Report

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Abstract

A 61-year-old patient was suspected of having meningioma, and surgery was subsequently performed on the patient. It was found that the diseased tissue was closely adhered to the normal brain tissue, and extracted to determine the pathological type. Brain biopsy suggested a diagnosis of tuberculous meningitis. The patient subsequently received regular anti-tuberculosis treatment, ultimately, the myodynamia of the patient's left limbs gradually improved, and her mental symptoms also improved. When a patient's history, clinical manifestations, neuroimaging and other physiological changes indicate a diagnosis of intracranial infection, there is no doubt that cerebrospinal fluid examination must be performed first, and an unnecessary craniotomy should be avoided if possible.

Introduction

Tuberculous Meningitis (TBM) remains a global health problem and is the most lethal and disabling form of tuberculosis [1]. TBM is the most devastating form of extrapulmonary tuberculosis and the most common form that affects the central nervous system and is caused by seeding Mycobacterium Tuberculosis (MTB) to the meninges. The primary infection location of MTB within the host is the alveoli in the lung. In infection leading to TBM can cause an intense inflammatory response that leads to pathological processes, which can cause the symptoms of TBM and cranial nerve palsies. The most significant neurologic sequelae of TBM patients are considered to be the excessive inflammatory responses, which cause tissue injury and brain oedema.

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Due to the nonspecific clinical presentation and lack of efficient diagnostic methods, the diagnosis of TBM remains difficult. We report a case of atypical TBM who presented with a large intracranial lesion in the right frontal lobe, and the patient's clinical, radiological, and histopathological features were described.

Case report

The patient was a 61-year-old right-handed female. Before being hospitalized, she had exhibited slurred speech and had lost most function in her left limbs for more than 1 month, without fever or headache. She also had a long history of mental illness and was diagnosed with schizophrenia. At admission, the patient had clear consciousness, with severe dysarthria with slurred speech and normal right-limb muscle strength but decreased muscle strength in the left limbs. Physical examination showed no obvious positive signs. Examination of the patient's blood at admission showed no abnormalities.

Cranial Computed Tomography (CT) showed extensive compression with oedema of the frontal and parietal lobes in the right hemisphere but no hydrocephalus. Head Magnetic Resonance Imaging (MRI) showed that the midline structure shifted to the left, and the right side of the cerebral cortex was compressed and flattened (Figure 1A & Figure 1B). Head-enhanced MRI suggested extensive meningeal enhancement in the right frontotemporal area (Figure 1D). Chest CT showed multiple lymph node calcifications in the chest, including calcifications in the left lung (Figure 1E).

After excluding surgical contraindications, we performed intracranial surgery to remove all the lesion tissue. During the operation, it was discovered that the lesion was located under the dura mater and was adhered tightly to the cerebral cortex. The tissue was hard in texture, grey-yellow in colour and rich in blood supply and was difficult to separate from the unaffected tissue (Figure 1F). Therefore, the surgeon performed a biopsy. The report revealed that the lesions were granulomatous inflammation with a multinuclear giant cell reaction, and the acid-fast staining was positive (Figure 1C). Ultimately, the patient was diagnosed with TBM, and the follow-up treatment was to kill the MTB with oral drugs, including isoniazid, rifampicin, pyrazinamide and streptomycin. Dexamethasone was also used as an adjuvant therapy to control inflammation.

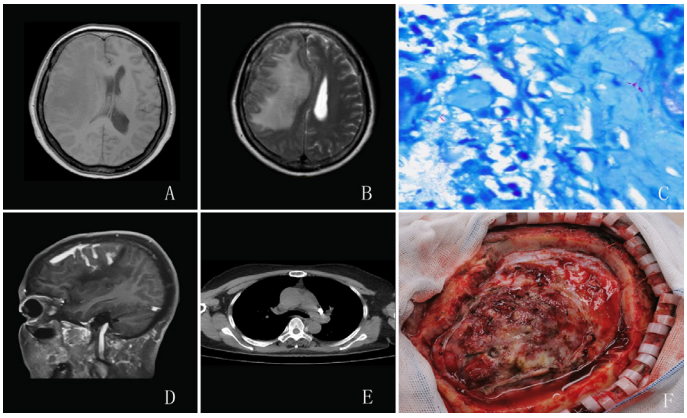

Figure 1: (A) T1-weighted MRI **(B):** T2-weighted MRI **(C):** Acid-fast staining, with mycobacteria indicated by red colour, while other bacteria and the background material are blue **(D):** Enhanced MRI **(E):** Lymph node calcification in the mediastinum **(F):** The lesion was grey-yellow in colour, hard in texture and rich in blood supply.

At the follow-up nearly two months after surgery, the patient's myodynamia of the left limbs had gradually improved, and the right frontal lobe and parietal lobe presented reduced inflammation on MRI (Figure 2). The patient was no longer taking anti-psychotic drugs was no longer taking since there had been no obvious further deterioration in her mental problems.

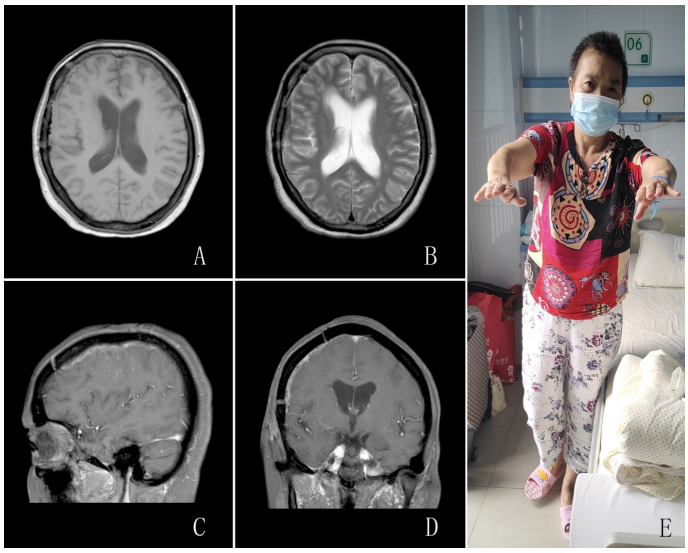

Figure 2: (A) T1-weighted MRI **(B):** T2-weighted MRI **(C&D):** Enhanced MRI. At the two-month follow-up, the patient's myodynamia of the left limbs had gradually improved **(E)**.

Discussion

TBM has complicated clinical manifestations, and the confirmation of the diagnosis is often difficult. Bacteriological confirmation of the diagnosis is ideal but is often difficult because of its paucibacillary nature and the low sensitivity and specificity of the diagnostic tests [2]. Early diagnosis is difficult but essential to avoid neurologic disability and death. However, on chest CT, we only found multiple calcifications.

For much of the world, the diagnosis of TBM involves staining to search for and scrutinize MTB by microscopy in the CSF and bacterial culture. TBM is a paucibacillary disease; in approximately 50% of cases, a definitive microbiologic diagnosis is not achieved, and it is often difficult to isolate MTB in CSF by conventional methods [3]. For many years, the accuracy of nucleic acid amplification tests has not been considered completely satisfactory.

In neuroimaging, patients with TBM frequently demonstrate basal meningeal enhancement, infarcts, hydrocephalus, and tuberculomas [4]. TBMs are considered suggestive when combining these features together. Research has clearly indicated that basal meningeal enhancement is the most specific imaging feature among prominent CT features of tuberculous meningitis [5]. The characteristic feature of TBM is the presence of a gelatinous, thick inflammatory exudate in the basal cisterns and subarachnoid spaces of the brain, which may block the circulation of CSF, leading to hydrocephalus [6].

Early diagnosis and prompt treatment of TBM are essential to reduce morbidity and mortality. The most important treatment objectives are to reduce the intracranial pressure, optimize the brain perfusion, kill the bacteria and control the inflammation in the brain. Related studies have shown that patients receiving adjuvant steroid therapy during treatment have significantly reduced risks of death and disabling neurological deficits [7].

Conclusion

Standardized anti-tuberculosis treatment can treat MTB, but the real question is whether the disease can be correctly diagnosed. The clinical manifestations of meningeal tuberculosis are diverse, but mental symptoms are very rare. It is difficult to diagnose TBM by imaging examination because it has a similar imaging appearance to many other intracranial lesions.

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Conflicts of Interest

We declare that we do not have any commercial or associative interest that represents a conflict of interest in connection with the work submitted.

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