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Tuberculous Meningoencephalitis: Intracranial Tumoral Presentation

A 37-year-old Iranian woman presented acutely with decreased level of consciousness and bilateral mydriasis to the emergency ward. CT scan and MRI showed a large intracranial mass in the left frontotemporoparietal region of the cerebrum. The clinical and imaging findings were suggestive of an intracranial tumor. Frontotemporal lobectomy was performed and the resected specimen contained white caseating material.

Grossly, the resected specimen was white and firm. Microscopically multiple granulomatous lesions with epithelioid histiocytes and Langerhans giant cells and central caseation were seen, surrounded by inflammatory infiltration of plasma cells and neutrophils as well as meningeal congestion. These findings were compatible with necrotizing granulomatous encephalitis of tuberculosis.

Parenchymal CNS tuberculosis with or without extra-cerebral manifestations may present as a space-occupying lesion. Because a tuberculoma is rarely suspected especially if there is atypical morphology, biopsy is required to establish the diagnosis and specific treatment.

Keywords: tuberculosis, intracranial mass lesion, imaging

Introduction

Common causes of granulomatous inflammation of the central nervous system include tuberculosis, fungal infections (especially cryptococcosis), syphilis and sarcoidosis.^{1,2}

Tuberculosis of the central nervous system as brain and spinal cord meningitis or meningo-encephalitis becomes symptomatic in 10-20% of patients with extrapulmonary tuberculosis. Only 1% of tuberculosis patients develop intracranial tuberculoma, usually in the form of miliary tuberculosis. In developing countries, tuberculomas still constitute 30% of the intracranial space occupying lesions, whereas in other countries intracranial tuberculomas have become rare and may represent as little as 0.1-0.2% of space occupying brain lesions. A tuberculoma is typically a solitary lesion, but in 15-34%, it may be multiple, arising from hematogenous spread from extracerebral foci, and in 10%, it may be associated with tuberculous meningitis. *Mycobacterium tuberculosis* is the most common organism cultured from intracranial tuberculomas; other mycobacteria are rarely found. Most intracranial tuberculomas manifest as an intracerebral lesion and a dura-based mass is rare.

Case report

A 37-year-old Iranian woman presented with decreased level of consciousness (GCS 4), bilateral mydriasis, and left anisocoria. She had a pulse rate of 60/min, irregular respiration, and a blood pressure of 100/60 mmHg. Her basilar reflexes were normal. She assumed a decorticated position to painful stimuli. There was areflexia of the four limbs, a neutral plantar reflex and no obvious signs of lateralization.

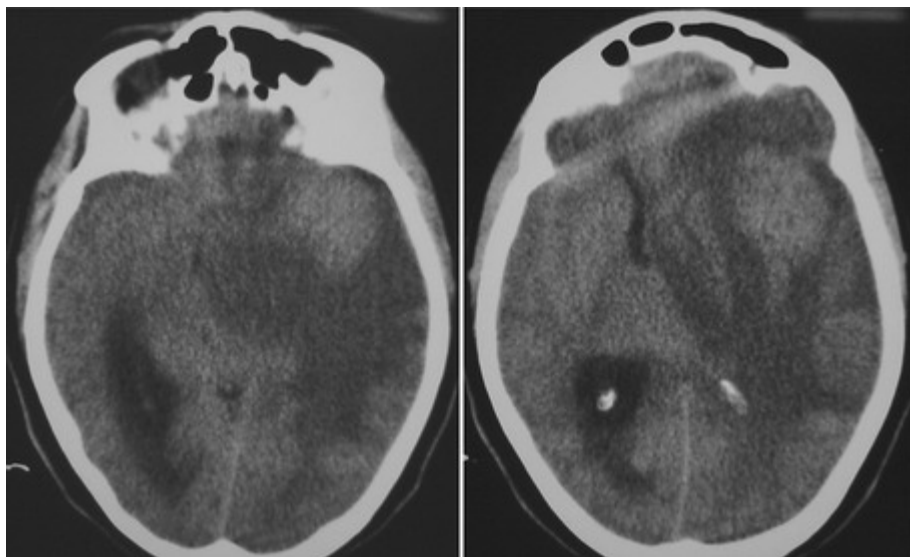


Fig 1. Axial brain CT scan without contrast shows a heterogeneous hyperdense mass lesion in the left frontotemporal lobe with widespread surrounding vasogenic edema. Subfalcian and descending transtentorial herniation are present. Pressure effect and obliteration of the left lateral ventricle were seen.

The patient's problem had started 2 months before, with morning headache and nausea and vomiting. She had no visual, sensory or motor problems until the morning of the day she was admitted. In the morning, she had a bout of generalized seizure followed by loss of consciousness and was admitted to our service.

CT scan and magnetic resonance imaging showed a large intracranial mass in the left frontotemporoparietal region of the cerebrum. The clinical and radiological findings were suggestive of an intracranial tumor. The patient underwent emergency operation with a GCS 4.

The preoperative diagnosis was a tumoral mass lesion such as glioblastoma. Chest x-ray and urinalysis were normal. On CT scan there was a heterogeneous hyperdense mass lesion in the left frontotemporal lobe surrounded by vasogenic edema that extended to the frontotemporoparietal regions with subfalcian and descending transtentorial herniation. Pressure effect and obliteration of the left lateral ventricle were seen (Figure1). Following contrast media injection, a heterogeneous enhancement became visible at the above mentioned areas (Figure2).

Frontotemporal lobectomy was performed and the resected specimen was composed of white caseating material.

Two days after the surgery, there was a mass lesion in the frontotemporal region, which was isosignal on T1 and hypersignal on T2 weighted MRI. Heterogeneous enhancements were seen following gadolinium injection (Figure3).

On gross pathology, the resected specimen was firm and white. Microscopically, multiple granulomatous lesions with epithelioid histiocytes and Langerhans giant cells with central caseation necrosis were seen, surrounded by striated inflammatory infiltration of lymphoplasmic cells and neutrophils, and meningeal congestion. The findings were compatible with necrotizing granulomatous encephalitis due to tuberculosis (Figure 4). The patient was put on the standard four-drug anti-tuberculosis regimen (isoniazid, ethambutol, pyrazinamide and rifampin).

After 2 months in-hospital stay, the patient was discharged with normal neurologic assessment and at a satisfactory general condition.

Discussion

Tuberculous granulomas are often characterized by caseation with or without multinucleated giant cells of Langerhans type, surrounded by a rim of inflammatory cellular infiltration and fibrosis; however, caseation necrosis and multinucleated giant cells of Langerhans type are not always evident. This poses a major pathological diagnostic problem. Detecting acid-fast bacilli on histological sections, positive culture for mycobacteria, or detection of mycobacterial DNA by PCR analysis can help with the diagnosis in most cases. Tuberculous meningitis has a strong predilection for the base of the brain, especially Sylvian fissures; and preceding tuberculosis in other organs is often detected.

Cryptococcal infection, neurosyphilis and sarcoidosis can imitate a similar presentation. Cryptococcus is usually found by PAS and Grocott hexamine staining. Syphilitic meningitis is characterized by perivascular infiltration of plasma cells and lymphocytes, and is occasionally accompanied by proliferative obliterative endarteritis.³ Primary and/or secondary features of syphilis are mostly present before the manifestation of meningovascular syphilis. Sarcoidosis is a systemic granulomatous disease of unknown etiology. However, there are a few cases reported as primary neurosarcoidosis without systemic disease.⁴ Most of those cases have the characteristic sarcoid granulomas consisting of non-caseating epithelioid granuloma with occasional Schaumann and asteroid bodies. Some reported cases were described to show granulomatous vasculitis as a prominent feature of sarcoidosis.

None of the etiological causes mentioned above could exactly fit the present case.

CNS tuberculomas occur in the cerebrum, cerebellum, the subarachnoid, subdural, or epidural spaces. In children, infratentorial lesions dominate, whereas in adults, supratentorial lesions are more frequent and tend to be located at the grey- white matter junction and the periventricular regions.

Only half of the patients report a previous history of tuberculosis. The clinical course is subacute or chronic and presenting signs are typically those of raised intracranial pressure (headache, and seizure reviewed in 10). The tuberculin skin test is positive in 85% of patients⁴, and pulmonary tuberculosis is diagnosed on chest radiographs in 30-80%⁵. On MRI, a tuberculoma appears as isointense to grey matter on T1 weighted images with a nodular or rim enhancement. Caseating lesions may show a central hypointense signal on T2 weighted images.^{6,7}

Transformation of the caseous core may lead to the formation of a tuberculous abscess, which are less frequent and tend to have greater mass effect and perifocal edema as well as an accelerated clinical course with fever and focal neurologic deficits.⁸

These lesions contain abundant bacilli, appear thin walled, uniform with peripheral rim enhancement and a central area of hyperintensity on T2 weighted images.⁹ In vivo proton MR spectroscopy and magnetization transfer MR imaging have recently been

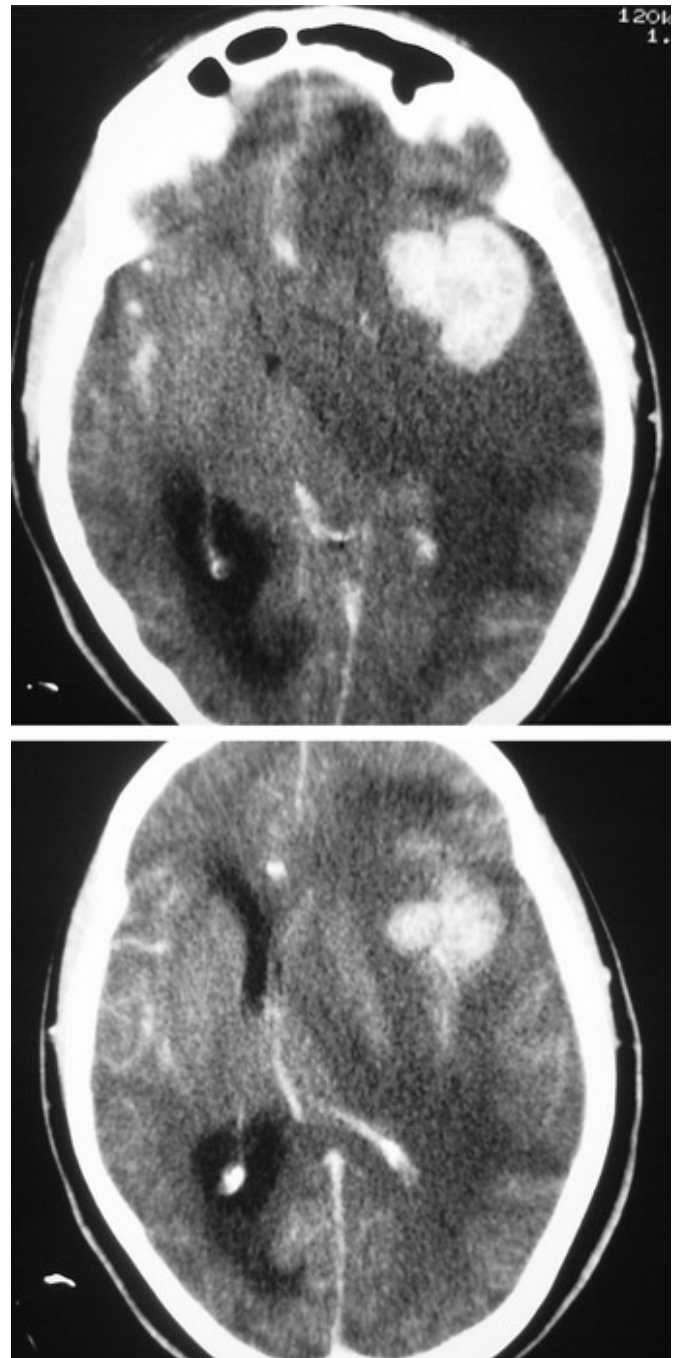


Fig 2. Axial brain CT scan with contrast administration shows a heterogeneous enhancement in the left frontotemporal part of brain. This findings are suggestive for brain masses like tuberculous granulomas.

demonstrated to differentiate tuberculous from other pyogenic brain abscesses.¹⁰

Less frequently, enhancement of the adjacent brain tissue indicates an area of cerebritis.¹¹ Generally, parenchymal CNS tuberculosis is more common in immunocompromised patients and has been reported in 15-40% patients affected by AIDS and CNS tuberculosis.¹²

Medical therapy with a regimen of isoniazid,

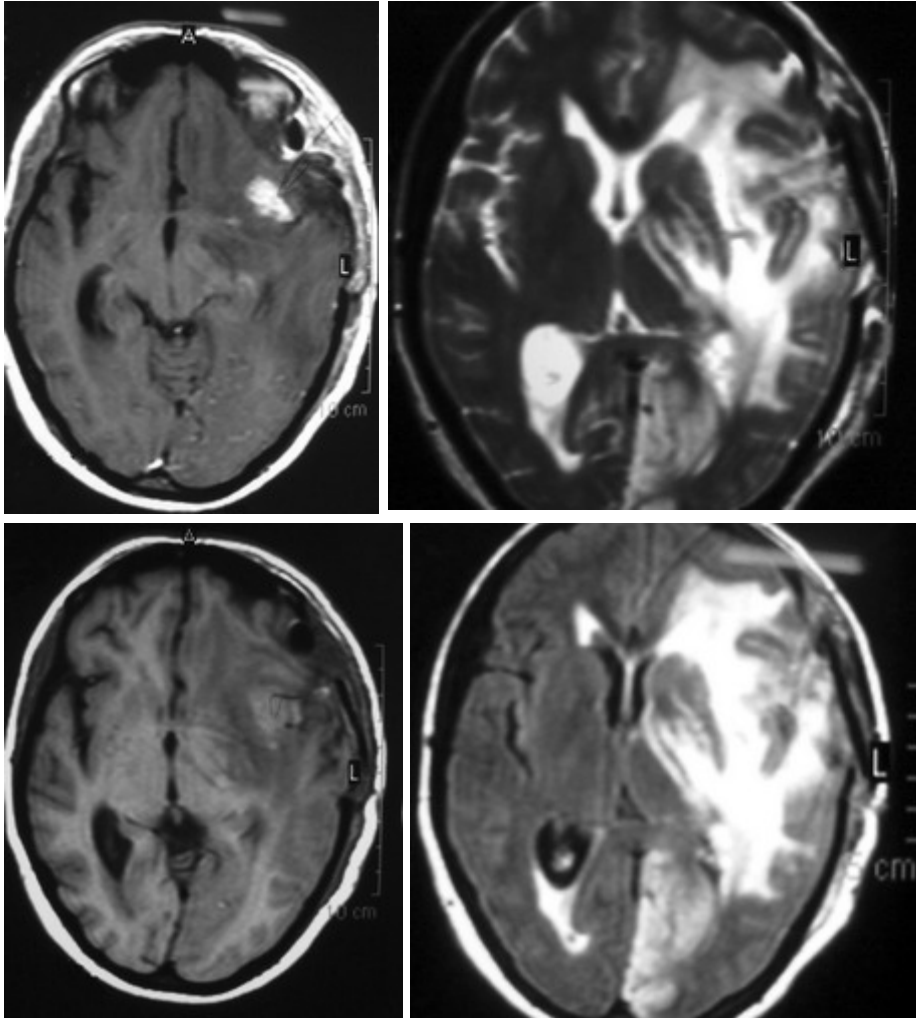


Fig 3. Two days after surgery; MRI images show a mass lesion isosignal on T1 weighted and hypersignal on T2 weighted images at the frontotemporal region. After gadolinium administration homogenous enhancements are seen.

ethambutol, pyrazinamide, rifampicin, and steroids usually results in a decrease in size and complete resolution of a tuberculoma within 3 months;¹³ nevertheless, much longer times, up to years, may be required.¹⁴ Second line drugs include streptomycin, kanamycin, capreomycin, ciprofloxacin, ofloxacin, ethionamide, and cycloserine (reviewed 15). When a tuberculoma is suspected, medical therapy alone is indicated in the majority of cases but because of the difficulties in differentiation of these lesions from other disorders on neuroimaging alone, a biopsy may be required to expedite treatment.¹⁵ Histology often fails to demonstrate acid-fast bacilli¹⁶ and, a caseating granulomatous lesion, with typical Langerhans type cells in combination with a positive tuberculin skin test may provide an adequate basis, on which to start chemotherapy. Growth of mycobacterium tuberculosis from CSF, if successful, may take up to six weeks.

Surgical resection is indicated for lesions that cause increased intracranial pressure and severe neurologi-

cal deficits. It may be required also when there is a failure to respond to drugs or even a paradoxical response despite a good response of the systemic disease.^{17, 18}

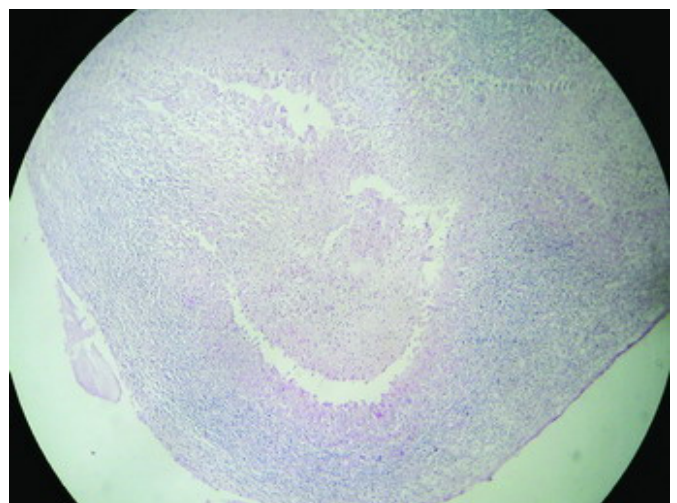


Fig 4. Pathology smear from brain tissue shows a necrotizing granuloma with central necrosis, peripheral epithelioid cells and infiltration of lymphocyte and monocyte/tissue macrophage cells.

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