

A Challenging Diagnosis of Brain Tuberculoma in a Healthy GirlMuharliza M^{1,4}, Intan Shafinaz M.R¹, Naimah S², Ong B.H², Chia K.K³¹Department of Ophthalmology, Hospital Sultanah Bahiyah, Alor Setar, Kedah Malaysia²Department of Neuromedical, Hospital Sultanah Bahiyah, Alor Setar, Kedah Malaysia³Department of Radiology, Hospital Sultanah Bahiyah, Alor Setar, Kedah Malaysia⁴Department of Ophthalmology, Pusat Perubatan Universiti Kebangsaan Malaysia***Corresponding author**

Muharliza M

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Abstract: A 16-year-old healthy girl presented with headache, nausea and vomiting with double vision for three weeks. On ocular examination, there was right esotropia with bilateral papilloedema. Magnetic resonance imaging (MRI) brain showed multiple small ring enhancing nodules with perilesional edema in brain stem and cerebral and cerebellar hemisphere. All the works up for infection, inflammation and malignancies were negative. On the basis of MRI and endemicity of tuberculosis in Malaysia, she was diagnosed as a case of brain tuberculoma and treated with anti-tubercular drugs and had complete amelioration of symptoms at three months of follow up.

Keywords: Tuberculoma, CNS tuberculosis, brainstem, anti-tubercular treatment

INTRODUCTION

Tuberculosis remains the most devastating infectious diseases in developing countries. World Health Organization (WHO) reported an estimated 10.4 million new cases and 1.8 million deaths in 2016 globally [1]. Tuberculoma of the central nervous system (CNS) is rare as compared to other system involvement and is the most severe form of tuberculosis and children and immunocompromised patients are usually involved [2]. Unfortunately it remains a diagnostic challenge due to their insidious nature and nonspecific findings.

CASE REPORT

A 16-year-old right handed, healthy Malay girl was admitted to the neurology ward with sudden onset of diplopia, right eye squinting and persistent headache for three weeks duration associated with nausea and vomiting. She denied any cough, hemoptysis, fever, night sweats or weight loss and no known exposure to individuals with tuberculosis or recent travelling and no other neurological deficit. However, she had strong family history of malignancies.

Her cranial nerve examination was significant for horizontal diplopia on right gaze consistent with sixth nerve palsy and fundus showed papilloedema. There were no signs of meningeal irritation. Her chest examination was normal. Initial urgent CT brain revealed ring enhancing right frontal nodule with white matter oedema suggestive of cerebral abscess. She was then started on high dose of intravenous (IV) Ceftriaxone (2g BD).

Lumbar puncture was performed with entirely normal cerebrospinal fluid (CSF) examination and was negative for tuberculosis, other bacteria, virus, fungal and malignant cells. The rest of her blood works including infective, inflammatory and tumour markers were normal. Patient's chest X-ray and other TB work

up were negative with an ESR of 10mm/hour. CT scan thorax, abdomen and pelvis were all normal.

In ward, she had progressive left sided body weakness and partial ptosis over the left eye. Subsequent urgent MRI brain revealed multiple white matter lesions with involvement of brainstem and corpus callosum with contrast enhancement suggestive of cerebral tuberculomas. The brain lesion was not feasible for brain biopsy due to small in size. In view of the rapidly progressing brain lesion, MRI brain findings and also endemicity of tuberculosis, TB treatment with oral EHRZ regime daily dose was started, Ethambutol, Isoniazid, Rifampicin and Pyrazinamide together with IV Dexamethasone 8mg BD.

After five days of treatment, she showed marked improvement of the left limbs weakness (full recovery of the motor power). She was discharged home at day eight of anti-tuberculosis treatment and after completed a two weeks course of IV Ceftriaxone and continued with anti-TB as an outpatient. Ocular examination prior discharged was entirely normal.

Unfortunately, during subsequent follow-up at day fourteen of anti-TB, she had paradoxical reaction whereby she developed bilateral internuclear

ophthalmoplegia with right facial nerve palsy which resolved completely 2weeks later. Repeated MRI brain

4months later showed almost complete resolution of the tuberculoma with no new brain lesion.

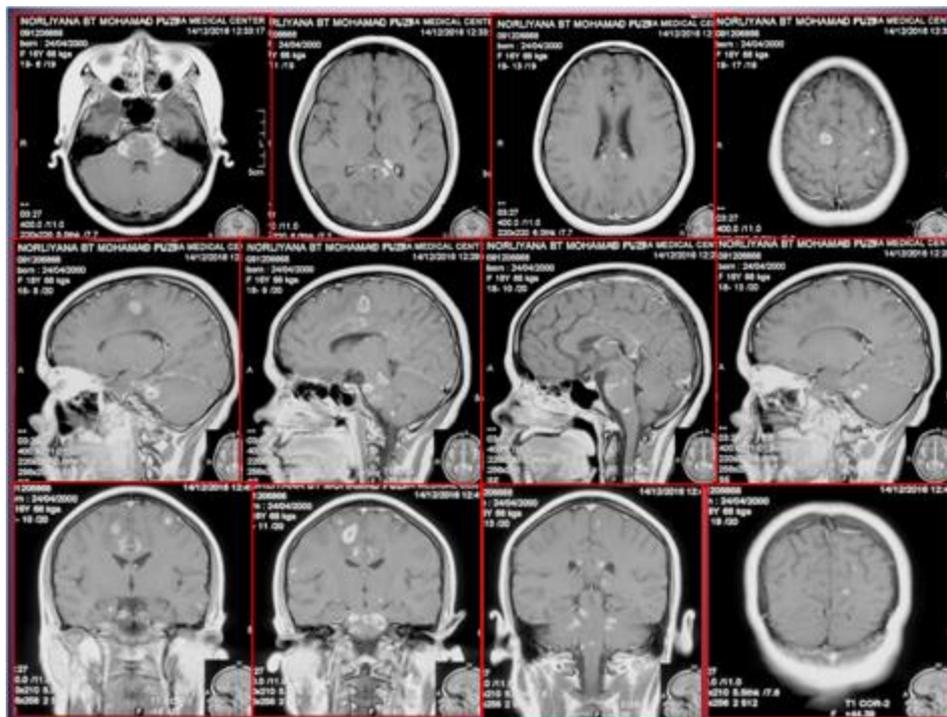


Fig-1: Magnetic resonance imaging brain T1 contrast sequence showing multiple well defined lesions seen at the left cerebellar peduncle, pons, midbrain, splenium of corpus callosum, white matter of the bilateral frontal and bilateral high parietal region with contrast enhancement.

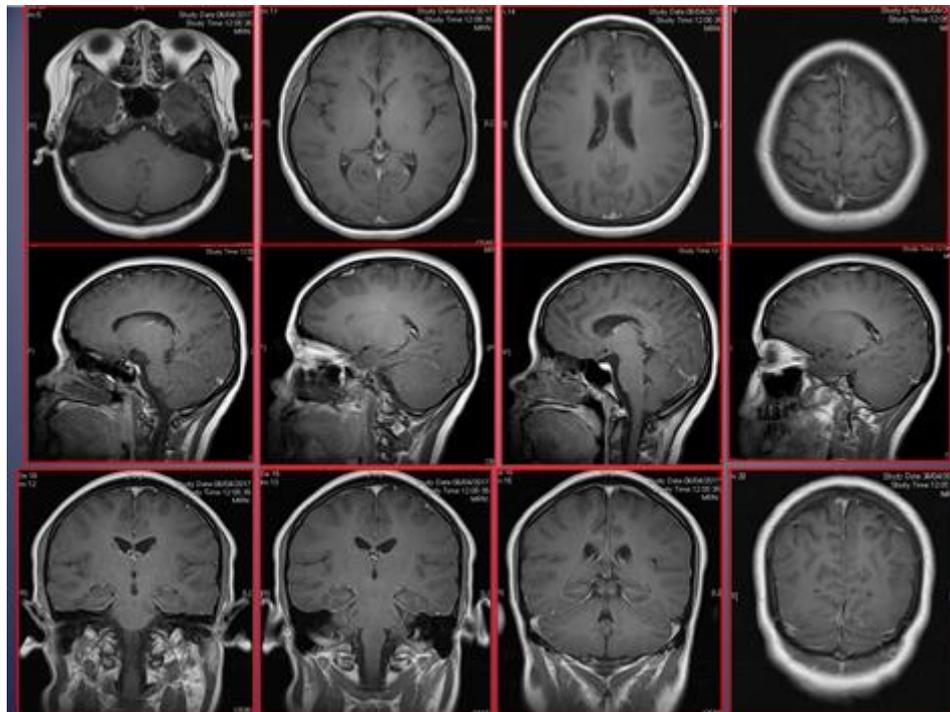


Fig-2: Magnetic resonance imaging brain T1 contrast sequence after 4months of TB treatment showing previously seen lesions have mostly reduced in size, numbers and enhancement.

DISCUSSION

Tuberculomas are organized clusters of inflammatory cells meant to limit the spread of

Mycobacterium bacilli. Intracranial tuberculomas are rare, usually involve the cerebral or cerebellar hemispheres due to the high blood supply to these areas

and uncommonly located in the brain stem [2-4]. CNS tuberculoma usually confused with intracranial neoplasm whenever presented without signs and symptoms of tuberculosis. Depending on their size and location, intracranial tuberculomas can have many signs mimicking primary CNS tumour such as high intracranial pressure, focal neurological deficits, and seizures. Many established methods exist to detect tuberculosis. However, diagnosis of CNS tuberculoma remains challenging as granulomatous encasement may preclude *Mycobacterium tuberculosis* (MTB) detection in serum or CSF samples [2], as in our patient all tuberculosis work up were negative.

Making the diagnosis of intracranial tuberculoma is essential for rapid treatment. Patients can present with single or multiple lesions with sizes varying from 1 mm to 8 cm, typically supratentorial [2,3]. Although often misdiagnosed as neoplasms, radiographic features of tuberculoma have been described. MRI has been found to be superior to CT of the brain for visualization of tuberculomas.

They are generally hypodense with ring enhancement on CT scans but it is non-specific; MRI with gadolinium enhancement offers the greatest detail in visualizing anatomic location, edema, and soft tissue involvement of suspected lesions [3]. The characteristic “target sign” visualized on CT scans pre-contrast may indicate tuberculoma and the radiographic characteristics of a tuberculoma can vary based on whether the lesion is solid, noncaseating, caseating with a solid center, or caseating with a liquefied center [2-4]. Noncaseating lesions are generally hypointense on T1-weighted MRI and hyperintense on T2-weighted MRI. Caseating lesions with a solid center are iso- to hypointense on T1- and T2-weighted MRI, while those with liquefied centers are hypointense on T1- and hyperintense on T2-weighted MRI; both demonstrate ring enhancement.

We also need to note that other differential diagnosis like cryptococcal meningitis, neurocysticercosis, viral encephalitis, sarcoidosis, meningeal metastasis and lymphoma which can have similar radiological findings of ring enhancing lesions, where both CT and MRI cannot accurately differentiate them from tuberculoma. As in our case, multiple well defined lesions seen at the left cerebellar peduncle, pons, midbrain, splenium of corpus callosum, white matter of the bilateral frontal and bilateral high parietal region which show isointense in T1, hyperintense in T2 and not suppressed in FLAIR with enhancement post contrast (Figure 1). In view of this radiological picture, a decision to treat as brain tuberculoma was made. However definitive diagnosis required histopathological examination. Treatment of tuberculoma mainly medical and surgery is rarely required where diagnosis in doubt [4, 5]. In most of cases anti-tubercular treatment started on radiological and laboratory basis as in our case.

Anti-tubercular drugs are usually given for a period of 12 to 18 months along with high dose steroid for 4 weeks and tapered over next 4 weeks [3-6]. Most of the patient responded well within 3 months as in our case.

CONCLUSION

We conclude that a high index of suspicion is essential to make an early diagnosis of CNS tuberculosis in an immunocompetent patient who presented with multiple intracranial mass in endemic country such as Malaysia as timely initiation of medical therapy is a key to a good neurologic outcome and reducing morbidity and mortality.

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