OMCR

Oxford Medical Case Reports, 2016;1, 12–14

doi: 10.1093/omcr/omv074
Case Report

CASE REPORT

Frontal lobe tuberculoma

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Abstract

Tuberculomas are usually infratentorial in children, and supratentorial lesions predominate in adults. We present a 4-year-old girl with multiple seizures, papilloedema and brisk reflexes. On investigation, she was found to have a large left parafalcine tuberculoma. She was treated with antitubercular treatment (ATT) and steroids. The child improved, seizures stopped and the papilloedema gradually disappeared. Follow-up magnetic resonance imaging brain after 8 months showed a mild reduction in the size of the lesion. Child is on regular follow-up.

INTRODUCTION

Tuberculomas are frequently infratentorial and multiple in children [1]. Here, we report a case of supratentorial tuberculoma in a child, successfully treated with antitubercular treatment (ATT) and steroids.

CASE REPORT

A 4-year-old girl presented with a history of multiple episodes of seizures since the last one and half years. The seizures were varying in semiology—initially multifocal with secondary generalization and later tonic seizures with frequency gradually increasing from once every 6 months to once every 2 weeks. There was no fever, vomiting or headache. On examination, she had papilloedema and brisk deep tendon reflexes. Weight was 13 kg (<15th centile) and blood pressure 110/70. Other systems were normal. There was no focal neurological deficit.

On investigation, haemogram, chest X-ray was normal. Computed tomography (CT) scan brain showed evidence of a large conglomerate ring enhancing focal lesion in the parafalcine frontal cortices mainly on the left side with extensive white matter hypodensity in both frontal lobes (Fig. 1). Small tuberculomas were also seen in the left occipital lobe and left lateral inferior cerebellum, tegmentum of pons and right temporal lobe.

Magnetic resonance imaging (MRI) brain showed a 5.7 × 3.9 × 4.8 cm-sized heterogeneously hyperintense peripherally enhancing conglomerate lesion involving the left frontal parafalcine region crossing onto the midline and right parafalcine frontal lobe, extending into the genu of the corpus callosum and showing patchy restricted diffusion. The lesion showed extensive perilesional vasogenic oedema (Fig. 2) with multiple ring and disc enhancing lesions seen in the bilateral cerebral and cerebellar parenchyma and in the right hemipons. Magnetic resonance spectroscopy revealed lactate double peak and diffuse reduction in all the metabolite peaks suggestive of tuberculoma. The Mantoux test was negative.

Patient was started on i.v. dexamethasone, which was given for 5 days, and subsequently lumbar puncture was done. Cerebrospinal fluid (CSF) examination was normal. CSF Gene Xpert revealed no *Mycobacterium tuberculosis* complex, and TB MGIT was negative for Acid Fast Bacilli. Antitubercular treatment (ATT) with isoniazid (10 mg/kg/day), rifampicin (10 mg/kg/day), ethambutol (20 mg/kg/day) and pyrazinamide (35 mg/kg/day) along with oral prednisolone at 2 mg/kg/day was started on Day 5 of

Received: October 23, 2015. **Revised:** December 5, 2015. **Accepted:** December 10, 2015

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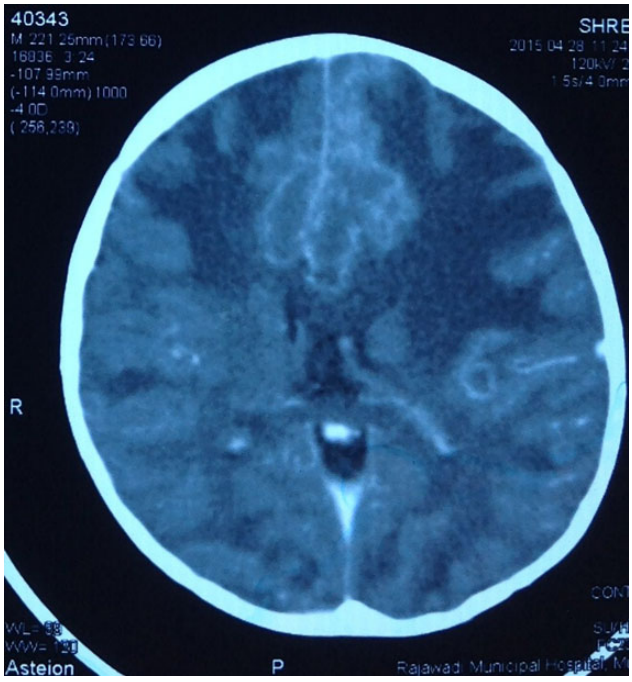

Figure 1: CT brain (axial view) with contrast showing ring enhancing focal lesion in parafalcine frontal cortex with perilesional oedema.

hospitalization. The child improved, seizures stopped and the papilloedema gradually disappeared.

A neurosurgical opinion for biopsy of lesion was taken, but in view of classical MRI findings, it was deferred.

Follow-up MRI brain after 8 months of treatment showed a mild reduction in the size of the lesion to 4 × 4 × 2.3 cm with mild perilesional vasogenic oedema. The ring enhancing lesions in the bilateral cerebral hemispheres were still present. However, the cerebellar and pontine lesions had disappeared (Fig. 3). Child is on regular follow-up.

DISCUSSION

A tuberculoma is a conglomerate mass of tissue made up of small tubercles which consist of a central core of epitheloid cells derived from altered mononuclear phagocytes and surrounded by lymphocytes. Tubercles originate during initial bacteraemia, but the extent and rate of progression into tuberculoma are extremely variable and depend upon complex and incompletely understood mechanisms [2].

Tuberculomas are usually located at the corticomedullary junction and periventricular region, as expected for haematogenous dissemination. They are mostly infratentorial in children and supratentorial in adults [3, 4]. They may present months to years after infection [5]. Our patient presented with a large supratentorial tuberculoma without any other manifestations except seizures, which is a rare presentation.

The incidence of intracranial tuberculomas in tuberculous meningitis is 17% [6]. Intracranial tuberculomas are mostly diagnosed based on clinical features, cerebrospinal fluid changes and imaging characteristics; therefore, ATT is usually administered on a presumptive basis. Bacteriological confirmation is not possible for the majority of patients as serological tests do not have sufficient sensitivity and specificity [7]. Similarly, in our patient,

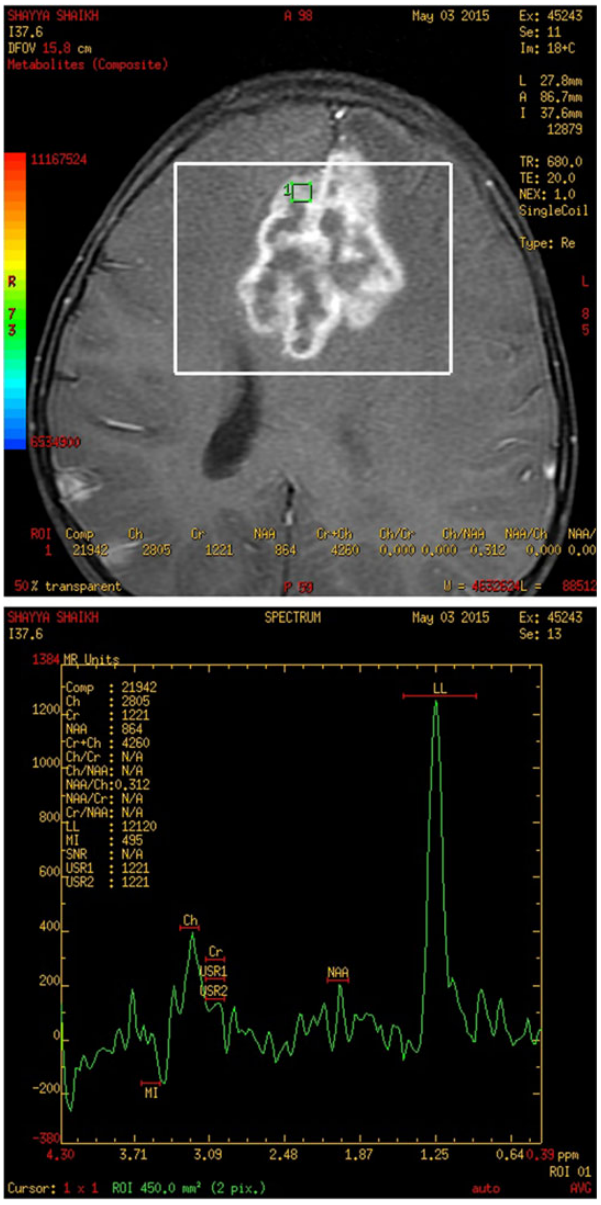

Figure 2: MRI brain spectroscopy showing left frontal parafalcine tuberculoma with lipid lactate peak.

diagnosis was based on imaging findings, and CSF examination was normal.

Initial management of intracranial tuberculomas aims to reduce intracranial pressure and other mass effects and to confirm the diagnosis. Open surgical treatment is associated with excessive surgical trauma [8]. Our patient was also treated first with anti-oedema measures to decrease the mass effect and then started on ATT. Duration of ATT for tuberculous meningitis is 12 months. However, the duration of therapy may be prolonged depending on response [7].

Bhagwati *et al.* [9] report on 31 children, of whom 5 needed surgical intervention—in 4 of them because they were thought to harbour brain tumours and in the fifth one because of significant mass effect in spite of treatment. Similarly, our patient was initially suspected to have a brain tumour on CT scan based on the size and location of lesion; however, MRI brain spectroscopy

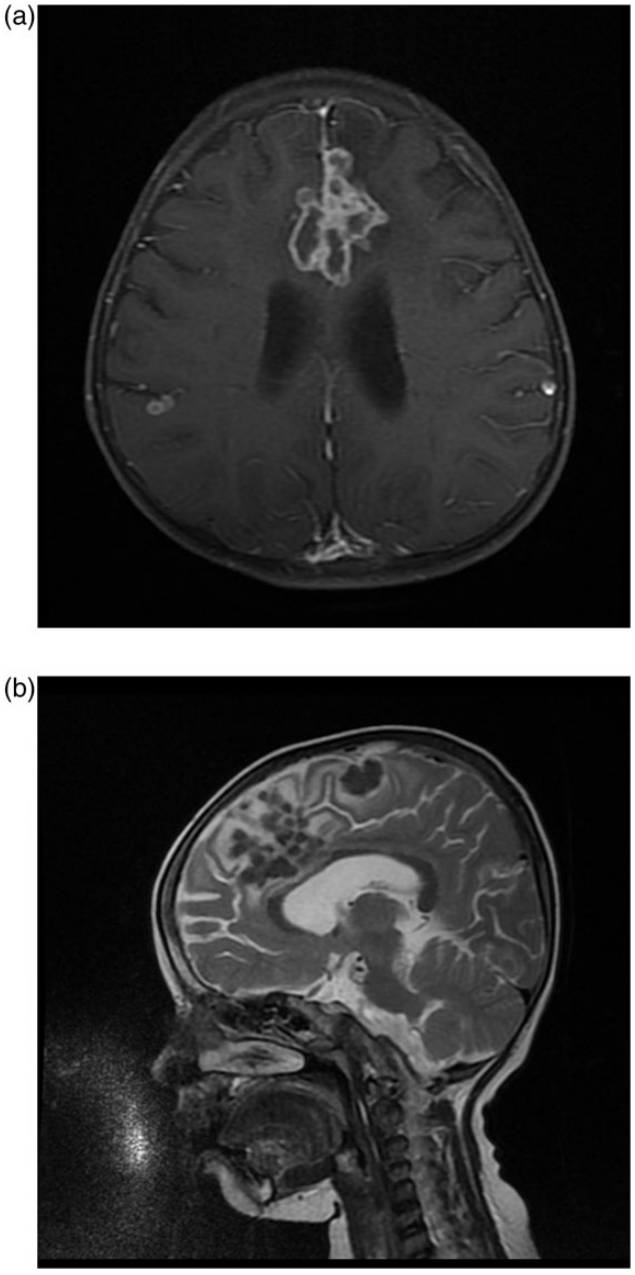

Figure 3: Follow-up MRI brain with contrast (**a**) axial and (**b**) sagittal views showing reduction in the size of lesion with mild perilesional oedema.

was classical of tuberculoma. Hence, surgical intervention was not done.

Most intracranial tuberculomas completely disappear with conservative treatment.

Large frontal lobe tuberculomas are not unusual in tuberculosis. Treatment duration may be prolonged based on response to therapy.

ACKNOWLEDGEMENTS

The authors would like to acknowledge Dr Y. K. Amdekar, Medical Director, Bai Jerbai Wadia Hospital for Children, Parel, Mumbai 400012, India.

CONFLICT OF INTEREST STATEMENT

None declared.

ETHICAL APPROVAL

Taken from Hospital Ethics Committee.

CONSENT

Patient's written consent was taken for publication.

GUARANTOR

N.M.D.P., DNB, DCH will act as guarantor.

REFERENCES

1. Shah I. Tests for diagnosis in tuberculosis. In: Shah I, ed. *Tuberculosis*, 1st ed. Mumbai: Pediatric Oncall, 2010, 18–30.
2. Udani PM. Neurotuberculosis. In: Seth V, Kabra SK, eds. *Essentials of Tuberculosis in Children*, 4th ed. New Delhi: Jaypee Brothers, 2011, 175–177.
3. Welchman JMM. Computerised tomography of intracranial tuberculomata. *Clin Radiol* 1979;**30**:567–573.
4. Bhargava S, Tandon PN. Intracranial tuberculomas: a CT study. *Br J Radiol* 1980;**53**:935–945.
5. Chatterjee S. Brain tuberculomas, tuberculous meningitis and post -tubercular hydrocephalus in children. *J Pediatr Neurosci.* 2011;**6**(Suppl. 1):S96–S100.
6. Ravenscroft A, Schoeman JF, Donald PR. Tuberculous granulomas in childhood tuberculous meningitis: radiological features and course. *J Trop Pediatr*. 2001;**47**:5–12.
7. Thwaites G, Fisher M, Heminway C, Scott G, Solomon T, Innes G; British Infection Society. British Infection Society guidelines for the diagnosis and treatment of tuberculosis of the central nervous system in adults and children. *J Infect* 2009;**59**:167–187.
8. Ersahin M, Hakan T, Ayan E, Berkman Z, Ekinci O, Ceran N, *et al*. Diagnostic and therapeutic role of CT-guided stereotactic surgery in the management of intracranial tuberculomas. *Turk Neurosurg*. 2010;**20**:295–302.
9. Bhagwati SN, Parulekar GD. Management of intracranial tuberculoma in children. *Childs Nerv Syst.* 1986;**2**:32–34.