Case Report

Tuberculosis: a great masquerader

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INTRODUCTION

Central nervous system tuberculosis is a serious form of extra pulmonary tuberculosis which develops due to haematogenous spread of Mycobacterium tuberculosis. Intracranial tuberculosis can present as Tuberculous meningitis, Tuberculous encephalopathy, Tuberculous vasculitis, CNS tuberculomas and Tuberculous brain abscess. Here authors present a case of a 10-year-old girl who presented with insidious onset of early morning vomiting, excessive sleepiness with classical neuroimaging findings of intracranial tuberculosis. Authors emphasise that intracranial tuberculoma should be considered in the differential diagnosis of any intracranial space-occupying lesion with or without pulmonary involvement.

Keywords: ATT with steroids, Intracranial mass, Ring enhancing lesions, Tuberculoma

ABSTRACT

Central nervous system tuberculosis caused by Mycobacterium tuberculosis is the most severe form of tuberculosis, accounting for 1% of all TB cases. Intracranial tuberculosis can present as Tuberculous meningitis, Tuberculous encephalopathy, Tuberculous vasculitis, CNS tuberculomas and Tuberculous brain abscess. Here authors present a case of a 10-year-old girl who presented with insidious onset of early morning vomiting, excessive sleepiness with classical neuroimaging findings of intracranial tuberculosis. Authors emphasise that intracranial tuberculoma should be considered in the differential diagnosis of any intracranial space-occupying lesion with or without pulmonary involvement.

Keywords: ATT with steroids, Intracranial mass, Ring enhancing lesions, Tuberculoma

INTRODUCTION

Central nervous system tuberculosis is a serious form of extra pulmonary tuberculosis which develops due to haematogenous spread of Mycobacterium tuberculosis. Intracranial tuberculomas occur in approximately 1% of patients with active tuberculosis and 4.5 to 28% of cases with tubercular meningitis. It carries a high mortality and distressing level of neurological morbidity.1

Tuberculomas are rare forms of intracranial space occupying lesions in developed countries, however they constitute a significant proportion of intracranial lesions in tuberculosis endemic areas.

CASE REPORT

A 9 years old girl presented with 5 months history of early morning vomiting on and off, gradually increasing in frequency past 1 month, one episode of LOC and fall with spontaneous recovery. She also had recurrent giddiness, evening rise of temperature excessive tiredness and sleepiness for past one month. She had weight loss past 3 months and could not attend school for 1 month.

She had 3-day history of worsening vomiting and giddiness. There was no history of seizures or significant contact with case of tuberculosis. Her past medical history was unremarkable. She was vaccinated appropriately for her age and had received BCG immunisation at birth leaving a scar.

On examination she was conscious, alert, oriented, afebrile, thin built, her weight in the 25th centile for weight for age, pale, dehydrated, tired, bilateral pupil were equal and reactive to light. No signs of meningeal irritation observed. Cardiovascular, respiratory and abdominal system examinations were normal. There was no focal neurological deficit or cranial nerve palsy. Fundoscopy was normal.
The child was evaluated in view of suspected intracranial space occupying lesion. Baseline blood work up was normal. HIV and HBsAg were negative. CT Brain on admission (Figure 1) showed multiple ring enhancing lesions with perilesional oedema in cerebral hemispheres, cerebellum, thalamus and interpeduncular cistern with obstructive hydrocephalus. There was no spinal involvement. Paediatric neurologist and neurosurgical consultation were sought.

**Figure 1: Multiple ring enhancing lesions with perilesional edema.**

The child was started on IV dexamethasone, followed by intensive phase regimen of ATT (2HRZE). This child did not have any symptoms suggestive of pulmonary tuberculosis. Chest x-ray (Figure 2) which revealed mild right pleural effusion, with multiple ill-defined opacities in both upper lobe of lungs. USG chest revealed right pleural effusion. USG abdomen was normal. Induced sputum AFB for TRUENAT-MTB was positive and rifampicin sensitive.

**Figure 2: Minimal right pleural effusion with multiple ill-defined opacities.**

Her oral intake, appetite improved and she bounced back to her routines and interests.

The child was advised 2 HRZE for 2 months and 2 HRE for 6 months as per the recent NTEP guidelines. Oral steroid was advised for 6 weeks. TB screening of contacts and siblings were negative.

**Figure 3: Multiple ring enhancing lesions and mrspectroscopy elevated choline, prominent lipid peak and severely depressed NAA.**

Repeat CT done after 3 months revealed reduction in the size of the lesions, perilesional oedema and mass effect, concurrent with findings of MRI Brain.

MR spectroscopy revealed elevated choline, severely depressed NAA, prominent lipid peak consistent with tuberculomas (Figure 3, Figure 4) The child is on 6th month of ATT, on regular follow up and doing well.

**Figure 4: MRI brain done after 3 months of ATT revealed reduction in the size of the lesions, perilesional edema and mass effect.**

**DISCUSSION**

Tuberculomas are the most common infectious cause of central nervous system space occupying lesions in
developing countries. Young adults and children are commonly affected in developing countries, while developed countries report predominantly older patients.⁴

Table 1: Classification of CNS tuberculosis.²³

<table>
<thead>
<tr>
<th>Intracranial</th>
<th>Spinal</th>
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<tbody>
<tr>
<td>Tuberculous meningitis</td>
<td>Pott’s spine and</td>
</tr>
<tr>
<td>Tuberculous encephalopathy</td>
<td>Pott’s paraplegia</td>
</tr>
<tr>
<td>Tuberculous vasculopathy</td>
<td>Non osseous spinal</td>
</tr>
<tr>
<td>CNS tuberculoma (single or multiple)</td>
<td>tuberculoma</td>
</tr>
<tr>
<td>Tuberculous brain abscess</td>
<td>Spinal meningitis</td>
</tr>
</tbody>
</table>

Tuberculomas are said to develop from “RICH focus” which consists of tubercular bacilli seeded into the meninges during primary tubercular infection. The focus does not rupture into meninges but expands locally forming a granuloma within the brain parenchyma.⁵

Tuberculomas are avascular, granulomatous intracranial space occupying lesions, with diameters ranging from 0.1-10 cm. They can be single or multiple and the number of lesions can vary from 1 - 100(or more). The central areas of tuberculomas may show caseous necrosis.⁶

Unlike the stormy presentation of tuberculous meningitis, tuberculoma of the brain shows an insidious course, though sometimes both may co-exist in the same patient.⁷

Clinical presentations are not due to tubercle- bacilli or its antigens but due to pressure effects of SOL.⁷ Patients present with single or repeated episode of focal seizures (60–100%), signs of raised intracranial tension (56-93%) and focal neurological deficits (33-68%).⁸ Tuberculomas are mostly supratentorial in adults and infratentorial in children.⁹

In developing world, tuberculomas accounts for 20-30% of SOL in brain, estimated to be higher in paediatric population. Intracranial tuberculomas are the least common presentation of CNS TB, found in 1% of these patients.¹⁰ They are multiple in 15-33% of cases.¹¹

Conventionally, intracranial tuberculosis is diagnosed with surrogates evidence of other foci of tuberculosis, peripheral lymphadenopathy, chest X-ray, sputum sampling for ZiehlNeelson staining and Ultrasonography of the abdomen for evidence of hepatosplenomegaly and intra-abdominal lymphadenopathy.¹²

An extra- neural focus of tuberculosis should be sought clinically and radiologically in all patients with CNS TB to identify safer and more accessible sites for diagnostic samplings.² Analysis of cerebrospinal fluid in patients with tuberculomas may not always be possible due to the presence of raised intracranial pressure which is a contraindication for lumbar puncture. The radiological features are also nonspecific and differential diagnosis includes malignant lesions, sarcoïdosis, pyogenic abscess, toxoplasmosis and cysticercosis.¹¹⁻¹³

On CT, tuberculomas are characterised as hypo or hyperdense masses with round or lobulated margins. The lesions show intense homogenous or ring enhancement in contrast studies. Perilesional oedema is often seen. MRI helps in differentiating whether tuberculoma is non-caseating or caseating, with solid centre or liquid centre.¹⁴⁻¹⁶

MR Spectroscopy demonstrates a very high lipid peak, reduction in NAA and creatinine and a choline/creatinine ratio of >1. Lipid peak in MRS in context of ring enhancing lesion is specific for tuberculoma and is absent in cases of NCC.¹⁷⁻¹⁹ The distinction between NCC and tuberculomas (Table 2) is important because parenchymal cysticercosis is a benign and self-limiting condition whereas tuberculoma is an active infection requiring prolonged therapy and involve potentially toxic drugs.

Table 2: Differences between neurocysticercosis and tuberculoma of the brain.

<table>
<thead>
<tr>
<th>Features</th>
<th>Neurocysticercosis</th>
<th>Tuberculoma of the brain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lesions</td>
<td>Smaller (&lt; 20 mm),</td>
<td>Larger (&gt; 20 mm),</td>
</tr>
<tr>
<td></td>
<td>single or multiple</td>
<td>often multiple,</td>
</tr>
<tr>
<td></td>
<td></td>
<td>conglomerated</td>
</tr>
<tr>
<td>Associated meningitis</td>
<td>Absent</td>
<td>Present</td>
</tr>
<tr>
<td>Location</td>
<td>Junction of grey-</td>
<td>Common in posterior</td>
</tr>
<tr>
<td></td>
<td>white matter</td>
<td>fossa</td>
</tr>
<tr>
<td>MR spectroscopy</td>
<td>Multiple amino</td>
<td>Lipid peaks</td>
</tr>
<tr>
<td></td>
<td>acid peaks</td>
<td></td>
</tr>
<tr>
<td>Focal neurological deficits</td>
<td>Absent</td>
<td>Present</td>
</tr>
<tr>
<td>Raised ICP</td>
<td>Transient</td>
<td>Present</td>
</tr>
<tr>
<td>Constitutional symptoms</td>
<td>Absent</td>
<td>Present</td>
</tr>
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</table>

Treatment aims to resolve neurological signs, constitutional symptoms and lesions on neuroimaging. The recommended duration of ATT is 9-12 months initially and neuroimaging at 3 months and at 9-12 months to monitor treatment and radiological response. The NTEP guidelines for the treatment of newly diagnosed CNS tuberculosis is ATT comprising, 8 weeks intensive phase treatment with Isoniazid (H), Rifampicin (R), Pyrazinamide (Z) and Ethambutol (E). Pyrazinamide is discontinued in the continuation phase which lasts for 4 months.

In extra-pulmonary tuberculosis, continuation phase can be extended for 3-6 months based on clinical decisions.
Systemic corticosteroids as adjuvant therapy are indicated when there is perilesional edema or paradoxical progression during treatment. Dosage of prednisolone is 1-2 mg/kg/day or dexamethasone 0.6 mg/kg/day or its equivalent is used for 4 weeks and then tapered over next 4 weeks. Majority of tuberculomas can be effectively treated without surgical intervention. Surgical intervention must be considered if there is progressive increase in the size of the hydrocephalus, increased mass effect due to progression of perilesional edema, progressive worsening of neurological deficit, raised intracranial pressure, paradoxical worsening to ATT, development of multidrug resistance and poor drug compliance.20-22

CONCLUSION

- Intracranial tuberculosis should be considered in the differential diagnosis of any intracranial mass.
- MRI brain with MR spectroscopy is the imaging modality of choice in the diagnosis of tuberculoma.
- MRS and DWI obviate the need for unnecessary biopsy and helps clinician in immediate management.
- Treatment with ATT and steroids is the mainstay of management.
- Neurosurgical intervention is rare and limited to treatment of hydrocephalus and brain stem compression due to large tuberculoma.

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REFERENCES
