Introduction:
Extrapulmonary manifestations of tuberculosis involving central nervous system via haematogenous route are significant in developing countries; which includes tuberculous meningitis, tuberculoma or brain abscess. Intracranial tuberculomas are rather common lesions, accounting for 10-30% of all intracranial masses in developing countries.1 Common sites for these lesions in adults include cerebral hemispheres and basal ganglia as well as cerebellar hemispheres in children.2

Tuberculomas are granulomatous mass lesions composed of a central zone of caseation surrounded by a collag enous tissue capsule arising in the brain parenchyma or the spinal cord. Lack of specific clinical and imaging characteristics often makes confident diagnosis of tuberculoma difficult to establish, particularly in the absence of extracranial lesions and histological data.3,4 For this reason, especially in areas with high disease prevalence, an empirical trial of antituberculosis therapy containing potent drugs such as rifampicin (RMP) is advocated.5 Only a high index of suspicion or a presumptive diagnosis based on typical clinical and neuro-image findings suffices to warrant such treatment. For patients with neurological deficits, surgical intervention is recommended.6,7

Here, we are presenting 5 cases with tuberculoma we have managed in our hospital.

Patients
Case 1.
A 20 year old lady was admitted to our unit with headache & vomiting for 2 months with history of several episodes of convulsion for 2 weeks. No H/O fever.

On examination, she was drowsy, had bilateral papilaedema and right hemiparesis. Her temperature

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was 37° C. ESR was 60mm in 1st hour. Chest radiograph was normal.

MRI of brain showed multiple hypo intense coalescence lesions with peripheral hyper intense rim on left parietal region with significant midline shift.

Case 2.
An 18 year old boy was admitted to our unit with headache & vomiting for 7 months with history of several episodes of convulsion for 2 weeks. He had a history of recent contact with a PTB patient, but no H/O fever.

On examination, he was conscious, had bilateral papilaedema. His temperature was 37° C. ESR was 60mm in 1st hour. Chest radiograph was normal.

MRI of brain showed multiple hypo intense coalescence lesions with peripheral hyper intense rim on right occipital region.

Patient underwent right occipital craniotomy with removal of the lesion. Histopathology confirmed as tuberculoma. Patient was treated with WHO recommended fixed dose combination anti-tubercular regimens for 18 months, in addition to oral steroids with tapering doses and anti-convulsants for 3 months.

On follow up visit after 3 months of starting anti-tubercular drugs, patient was living a normal life with visual impairment.

Case 3.
A 15 year old girl was admitted to our unit with headache & vomiting for 4 months with right hemiparesis for 2 weeks.

On examination, she was conscious, alert & oriented; had bilateral papilaedema and muscle power 3/5 in right upper & lower limbs, jerks normal, planter reflex normal. Her temperature was 37° C. ESR was 60mm in 1st hour. Chest radiograph was normal.

MRI of brain showed multiple hypo intense coalescence lesions with peripheral hyper intense rim on left parietal region.

Patient underwent left parietal craniotomy with removal of the lesion. Histopathology confirmed as tuberculoma. Patient was treated with WHO recommended fixed dose combination anti-tubercular regimens for 18 months, in addition to oral steroids with tapering doses and anti-convulsants for 3 months.

On follow up visit after 3 months of starting anti-tubercular drugs, patient was completely symptomless living a normal life.
equivocal B/L. Her temperature was 37°C. ESR was 45mm in 1st hour. Chest radiograph was normal.

MRI of brain showed an irregular hypo intense lesion with peripheral hyper intense rim on left parietal region with mild perilesional oedema.

Patient underwent left parietal craniotomy with removal of the lesion. Histopathology confirmed as tuberculoma. Patient was treated with WHO recommended fixed dose combination anti-tubercular regimens for 18 months, in addition to oral steroids with tapering doses and anti-convulsants for 3 months.

On follow up visit after 3 months of starting anti-tubercular drugs, patient was completely symptomless living an apparently normal life.

Case 4.
A 40 year old diabetic female was admitted to our unit with headache & vomiting for 4 months with occasional focal convulsion for 3 weeks.

On examination, she was conscious, alert & oriented; had bilateral papilaedema. Her temperature was 37.5°C. ESR was 75mm in 1st hour. Chest radiograph was normal.

CT scan of brain showed an irregular hypo dense lesion with light peripheral hyper dense rim on left fronto-parietal region with mild perilesional oedema.

Our clinical diagnosis was tuberculoma. Patient was empirically treated with WHO recommended fixed dose combination anti-tubercular regimens for 18 months, in addition to oral steroids with tapering doses and anti-convulsants for 3 months.

Follow up CT scan of brain showed no persistence of old lesions and normal brain findings.

On follow up visit after 3 months of starting anti-tubercular drugs, patient was completely symptomless living an apparently normal life.

Case 5.
A 16 year old girl was admitted to our unit with headache & vomiting for 2 months with occasional focal convulsion for 1 week & occasional abnormal behavior.

Follow up CT scan of brain showed no persistence of old lesions and normal brain CT findings.
On examination, she was conscious, alert & oriented; had bilateral papiliaedema. Her temperature was 37.5°C. ESR was 80mm in 1st hour. Chest radiograph was normal. Follow up MRI of brain showed no persistence of old lesions as well as normal brain findings.

**Discussion:**
Tuberculosis is still a serious health problem in developing countries. There is also resurgence in developed countries due to human immunodeficiency virus (HIV), immigration and development of multi-drug resistant strains. Central nervous system involvement of tuberculosis is 10%, and it may appear as tuberculous meningitis, tuberculoma, abscess or Pott's disease. Tuberculomas account for 10-30% of all intracranial masses in developing countries, and 0.5-2% in developed countries. They are commonly located in cerebral hemispheres and basal ganglia in adults, and in cerebellar hemispheres in children, due to the large blood supply to these areas. Rare locations, such as brainstem, cerebellopontine angle, and pituitary gland have also been reported.

The clinical manifestations of brain tuberculoma are pleomorphic. Mainly related to individual differences in size and topography of lesion. Symptoms and signs of raised intra cranial pressure (ICP) are usual features and constitute headache, vomiting, blurring of vision, and papilloedema. Lateralising signs when the lesion is in cerebrum. Focal or generalized convulsions also. Signs and symptoms of meningitis i.e. neck stiffness and fever.

The presenting symptoms & signs are summarized in Table 1.

<table>
<thead>
<tr>
<th>Symptoms &amp; Signs</th>
<th>No of Patients (n)</th>
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<tbody>
<tr>
<td>Headache</td>
<td>05(100%)</td>
</tr>
<tr>
<td>Vomiting</td>
<td>05(100%)</td>
</tr>
<tr>
<td>Convulsion</td>
<td>04(80%)</td>
</tr>
<tr>
<td>Hemiparesis</td>
<td>03(60%)</td>
</tr>
<tr>
<td>Papilloedema</td>
<td>05(100%)</td>
</tr>
<tr>
<td>Fever</td>
<td>01(20%)</td>
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Duration of symptoms at presentation varies from 2 to 7 months. None of the patients had extracranial TB. None had previous H/O PTB. But one patient had H/O contact with PTB patient & another patient was diabetic. Therewere no other systemic diseases among the patients of this series. In all cases HIV 1 & 2 was negative, though sylhet is a known zone for a number of HIV infected patients.

In our cases, CT findings of the lesions presented as irregular hypo dense lesion with hyper dense rim.
enhancement (case 4). MRI findings revealed in rest of the cases as hypo intense lesion in T1WI & hyper intense lesion in T2WI.

CT scans of a tuberculoma reveal an iso – to – hypodense lesion with varying contrast enhancement pattern. T1 weighted MRI demonstrates an iso – to – hypointense lesion. On T2-weighted images, it can appear as a hyperintense lesion or a hyperintense center surrounded by a hypointense rim. Therefore there are no pathognomonic radiological findings for a tuberculoma. There is also a recent paper regarding diffusion-weighted MRI and MRI spectroscopy in the diagnosis of tuberculoma, but it is concluded that these techniques are also unable to provide a specific characterization.

Surgery was performed in 3 of the 5 cases, in order to relief the mass effect and also to establish a diagnosis. In all 3 cases; the lesions were completely removed. Histopathological examination of a tuberculoma reveals a necrotic caseous center surrounded by a capsule composed of fibroblasts, epitheloid cells, Langhans giant cells and lymphocytes. Biopsy of the lesion is essential for establishing a diagnosis. Recent papers also suggest stereotactic biopsy as an alternative to craniotomy, and diagnostic yields up to 85% have been reported.

Mainstay of treatment in intracranial tuberculomas is medical. Surgery is reserved for large, solitary lesions with significant mass effect and unresponsive to medical treatment. Medical treatment consists of isoniazid, rifampin, and pyrazinamide for the initial 2 months, followed by only isoniazid and rifampin for the remaining time. Although the recommended duration of treatment is 12 months, shorter and longer courses are proposed.

In our cases we advised our patients 18 months supervised regimens. We have follow up the patients monthly, as all the cases were common at 3rd month follow up, we presented our follow up at 3rd month in this literature. Addition of steroids to the regimen is advised, in order to prevent paradoxical expansion of the lesion during medical treatment. Overall mortality is 10%.

Conclusion:
In conclusion, intracranial tuberculomas can be situated in every location and can mimic any lesion. Pulmonary involvement is not always present and radiological studies are not conclusive. Biopsy is diagnostic and treatment is medical. Especially in the presence of risk factors, a high index of suspicion should be maintained. But in case of large, solitary lesions with significant mass effect, patients with neurological deficits & unresponsive to medical management need surgical interventions.

References: