Case report

Brainstem tuberculoma in an immunocompetent patient

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INTRODUCTION

In the absence of other intracranial tuberculomas or systemic manifestation of tuberculosis isolated brainstem tuberculoma is a rare entity[1], that accounts for 6% of all brain stem masses in endemic areas[2]. The occurrence of these lesions is further rarer in the immunocompetent adults[3,4]. We discuss the findings of ring enhancing lesion of the brain stem where based on imaging findings we treated empirically with anti-tuberculous therapy with excellent outcome.

CASE REPORT

A 49 year female presented with tingling sensation in left half of the body of 3 months duration, gradual onset and progressive left sided weakness of 2 $\frac{1}{2}$ months, inability to see the objects on right side and diplopia of 1 month duration associated with blurring of vision, headache and vomiting. There was no history of neck pain, bowel or bladder disturbances. There was no history of previous tuberculosis either in the patient or in her family. Her general and systemic examination was normal. Higher mental functions were normal. There was internuclear ophthalmoplegia. There was grade 4/5 weakness of left upper and lower limbs including facial nerve. She was able to walk with support. Left plantar was extensor. There were left sided cerebellar signs including positive finger nose test, dysdiadochokinesia, intention tremor, and impaired knee heel test. Right side examination was normal. Blood investigations were normal except for an ESR of 28 mm in the first hour. X-ray chest was normal. Mantoux test was negative. Lumbar CSF examination was normal. CT scan of the brain showed a slightly hyperdense conglomerate lesion in the midbrain and pons more on right side with peripheral ring enhancement, irregular margins and significant perilesional edema leading to mild expansion of the contours of the brainstem. There was effacement of the ambient and quadrigeminal plate cisterns and the anterior wall of the fourth ventricle (Figure 1). With all these findings a neoplastic lesion was unlikely and a diagnosis of inflammatory granuloma was suspected. Considering the epidemiological setting, the possibility of a tuberculoma was considered. She was started on antituberculous therapy (isoniazid 5 mg/kg/day, rifampicin 10 mg/kg/day, pyrazinamide 25 mg/kg/day, and ethambutol 20 mg/kg/day along with pyridoxine 20 mg/day for 3 months followed by continuation of isoniazid 5 mg/kg/day, rifampicin 10 mg/kg/day for 9 more months) with a 4-week course of dexamethasone. She was followed up in the outpatient clinic with significant neurological improvement as shw was able to walk without support, cerebllar signs were disappeared and extraocular movmenets became full. Follow-up CT scan done after 6 weeks showed significant reduction in the size of the lesion and peri-lesional oedema (Figure 2).

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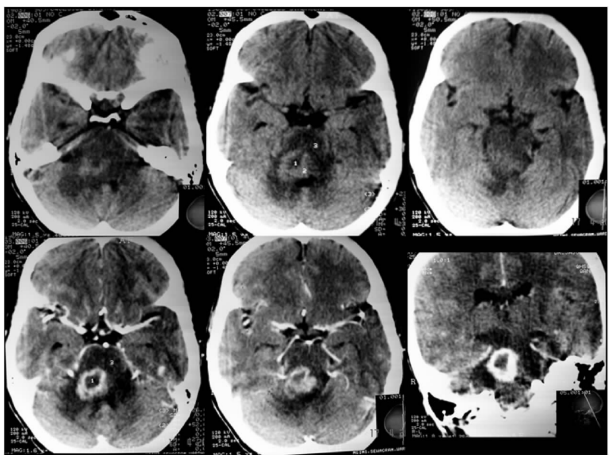

Figure-1: CT scan showing mildly hyperdense lesion on plain in the midbrain and pons with ring enhancement after contrast administration with significant peri-lesional oedema

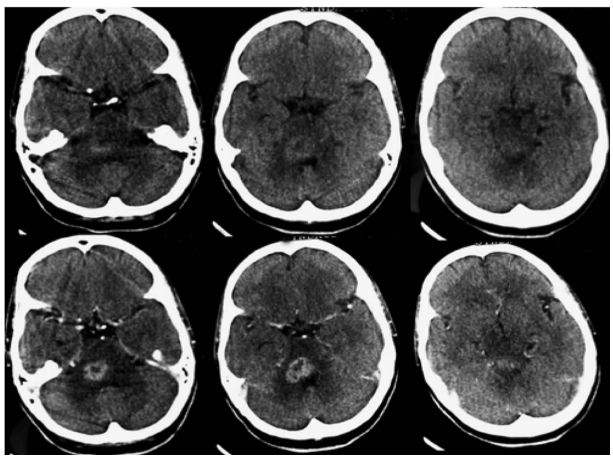

Figure-2: Follow up CT scan after six weeks showing significant decrease in the size of lesion and resolution of peri-lesional oedema

DISCUSSION

Clinically it is impossible to differentiate tuberculomas from any other brainstem masses as these lesions present with focal neurological signs similar to any other lesions of brainstem[3,5]. Brainstem tuberculoma needs to be differentiated from other ring-enhancing lesions in the brainstem including pyogenic abscess, metastatic disease, cysticercus granuloma and a high-grade glioma[1,6]. As in present case laboratory studies, including erythrocyte sedimentation rate, Mantoux test and CSF studies can be normal[3,7]. In expert hands CT-guided stereotactic biopsy is a safe method to diagnose brainstem tuberculoma[1], however there is a risk of fatal complications including bleeding or meningeal spread of infection (inappropriate to diagnose potentially a benign disease)[8]. Magnetic resonance imaging (MRI) helps in the diagnosis of and can objectively determine other accompanying abnormalities[9] however MRI findings may not be crucial in the diagnosis of central nervous system tuberculoma[6]. A good quality contrast enhanced CT scan is helpful for defining the characteristics of the tuberculoma, the presence of multiple lesions, and also to exclude meningitis[10]. Radiologically tuberculomas generally have an irregular margin with significant perilesional edema[1]. The most frequent fidings on CT scan include a small isodense or slightly hyperdense nodules with peripheral edema, showing homogeneous or ring like enhancement after contrast . Larger ring-enhancing lesions or lobulated masses represent maturer forms. The "target sign" has been described as characteristic of tuberculomas, consisting of a ring-enhancing lesion with an additional central area of enhancement or calcification[6,7,10,11]. The above CT appearance of tuberculomas has been correlated well with the microscopic picture[10]. Histopatholgically the tuberculoma consists of a central zone of caseous material surrounded by reactive epitheloid cells, Langerhans giant cells, and varying numbers of lymphocytes, polymorphs, and plasma cells[12]. The central lucent zone on CT scan represents the caseous necrosis. This is surrounded by a more slowly perfusing tissue compartment of granulation tissue (reactive epitheloid cells, Langerhans giant cells, and varying numbers of lymphocytes, polymorphs, and plasma cells). Multiple small tubercles, some with caseating centers, can be seen dispersed within edematous brain and later on these tubercles coalesce to form a larger lesion[10]. The incidence of calcification on initial CT scan in tuberculomas varies from none 10 to 6%[5]. Small residual punctate calcifications can be seen at follow up scan at the tuberculoma site[10]. Based on these typical signs in present case we started a course of anti-tubercular therapy and steroids with excellent results. The issue of therapeutic ATT is debated in literature. Because of unnecessary exposure to antituberculous therapy several authors recommend histological confirmation of the lesion prior to starting antituberculous therapy[4,13], and some authors have described the use of antituberculous therapy a useful diagnostic tool rather than radiological studies[3,4,6,7,14]. The suggested duration of treat-

ment is at least 12 months regardless of resolution of the lesion in such a case[14]. Concomitant corticosteroid therapy is recommended as paradoxical expansion under treatment can cause neurological deterioration[15]. In an appropriate setting contrast enhanced CT-scan with anti-tubercular therapy can be a helpful, objective and non-invasive method for diagnosis and follow-up of the outcome of drug treatment.

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