Case Report

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A Rare Case of Pediatric Intraventricular Neurocysticercosis

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Abstract

Neurocysticercosis is caused by the infestation of human central nervous system with larval stage of *Taenia solium*. Many previous studies have reported poor outcomes in cases of intraventricular form of the disease. We present the case of an eleven-year-old male child who presented with complaints of fever, headache, vomiting and seizures. He was misdiagnosed elsewhere as tuberculoma two years back. A solitary, non-migratory cyst was visualized in the fourth ventricle on the magnetic resonance imaging scan done this time (intraventricular neurocysticercosis). Patient was treated with anti-helminthics, steroids and anticonvulsants and is asymptomatic on follow-up. A repeat magnetic resonance imaging scan done 18 months later revealed complete resolution of the intraventricular neurocysticercosis. Points of interest in the present case (intraventricular neurocysticercosis) are the initial misdiagnosis, absence of severe hydrocephalus and presentation as seizures.

Keywords: Albendazole; Brain; Cyst; Cysticercus; Intraparenchymal; Intraventricular; Seizures; Taenia; Magnetic Resonance Imaging.

Introduction

Neurocy sticercosis (NCC) is one of the common parasitic infestation of the central nervous system world wide [1]. The presence of tapeworm cysts inside the cerebral ventricular system is seen in 20–30% cases [2,3]. In India, the intra parenchymal disease is more common than intra ventricular NCC [1]. The prognosis for intra ventricular neurocysticercosis is relatively poorer than intra parenchymal NCC [2].

Case Presentation

An eleven-year-old boy, presented with intermittent fever for five days, headache and vomiting for three days and three episodes of seizures 1 day back. The child had past history of headache and vomiting, for which he was admitted elsewhere and investigated two years previously. Computerized tomography of the head then had revealed a ring-enhancing lesion (not available for review).

Presence of a tuberculoma was suspected then and he was treated with anti-tubercular therapy for nine months. Neurological examination now revealed absence of meningeal signs, focal neurological deficits, or cerebellar signs. The cerebrospinal fluid examination (CSF) showed neutrophils – 18 cells/cumm, lymphocytes – 52 cells/cumm and red blood cells–one cell/cumm. The CSF protein level was 69 mg/dL and glucose was 64 mg/dL (blood glucose 112mg/dL). Magnetic resonance imaging (MRI) scan showed slightly dilated ventricular system with an intraventricular neurocy sticercosis in the fourth ventricle (Figures 1a and 1b).

He received oral prednisolone for 3 days followed by oral albendazole for 7 days. Sodium valproate was given for convulsions. Repeat MRI scan (after 15 days) did not show any change in the position of the cyst or size of the ventricles (Figure 2). The patient did not have any complaints later and a MRI scan done 18 months later showed absence of NCC (Figure 3).

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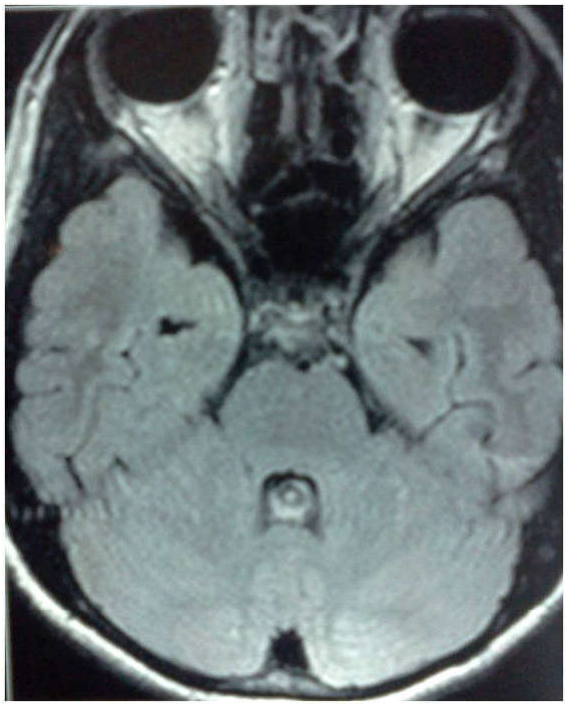

Fig. 1a: Magnetic resonance imaging: Note the intraventricular cystic lesion (neurocysticercus) in the fourth ventricle.

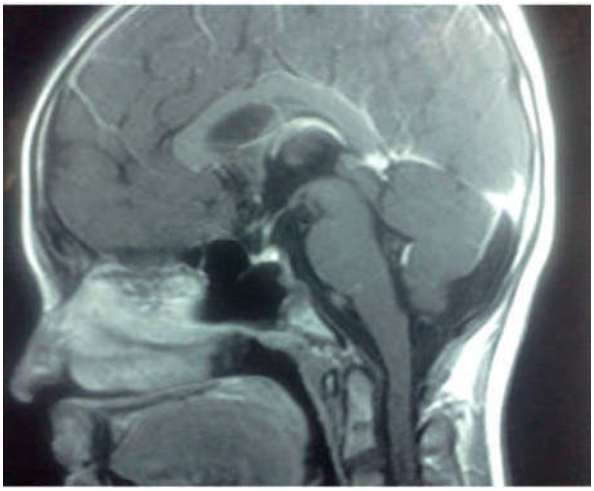

Fig. 1b: Magnetic resonance imaging (sagittal view): Note the intraventricular cystic lesion (neurocysticercus) in the fourth ventricle.

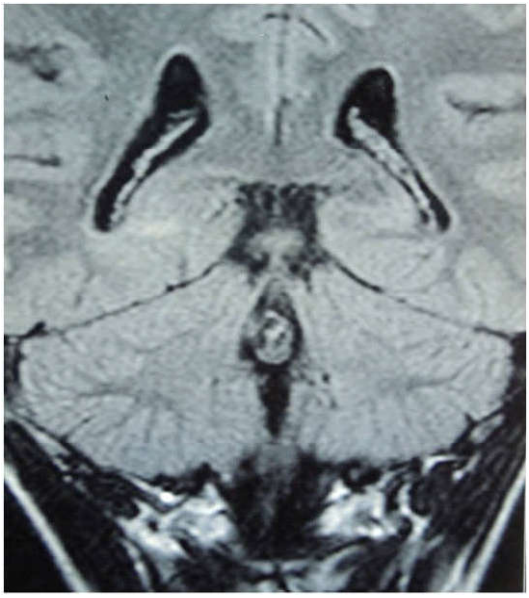

Fig. 2: A repeat magnetic resonance imaging scan (done after 15 days of the first scan), which does not show change in the position of the cyst or increase in the size of the ventricles.

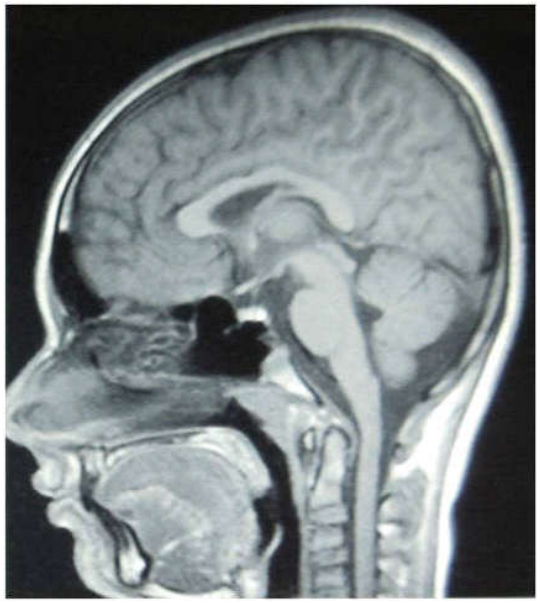

Fig. 3: A follow-up magnetic resonance imaging scan (sagittal view) done 18 months later with no evidence of residual neurocysticercous cyst.

Discussion

In developing world, neurocy sticercosis is a common cause of seizures and epilepsy [3,4]. Increased international travel, immigra-tion from developing countries and improved diagnostic techniques have led to emergence of neurocysticercosis as one of the common infections in developing as well as developed countries [5].

NCC has been classified into subarachnoid-cisternal, parenchymal, intraventricular and spinal forms [2,3,5-7]. Intraventricular NCC accounts for 0.3% to 20% of all cases of neurocysticercosis with fourth ventricle being the most common site (50%), followed by lateral ventricles, third ventricle and aqueduct [1-3,5,6]. Intraventricular cysts may be single or multiple and can coexist with parenchymal and subarachnoid cysts [2]. Ventricular NCC often leads to ob-structive hydrocephalus because of the

migrating cyst and ventriculitis due to ependymal inflammatory response [1,2,6-8]. Active viable intraventricular cysts do not (usually) produce a host reaction, but they can obstruct the cerebrospinal fluid flow [2,8]. Intraventricular NCC is a potentially fatal condition [2,8]. The clinical presentation of intraventricular NCC is variable (depends upon location, size, number and stage of parasite and host inflammatory response) [1-3,6,9]. Headache, vomiting, loss of consciousness may occur along with symptoms caused by cerebrospinal fluid obstruction [9]. Patients with neurocysticercosis cyst in the fourth ventricle may also present with episodic headache, papilledema, neck stiffness, positional vertigo, and unconsciousness, with rapid recovery (Brun's syndrome) [9].

On computerized tomography, ventricular NCC appears as iso-attenuating relative to CSF and are not well visualized [6,9]. Their presence can be concluded due to distortions of the ventricles with asymmetric or ob-structive hydrocephalus [9]. Most intraventri-cular cysts are well defined at MRI because their signal intensity may differ from that of cerebrospinal fluid on T1 or T2-weighted images [6,10,11]. MRI is able to detect perilesional edema and degenerative changes of the parasite and is suitable for small cysts or those located in the ventricles, brainstem, cerebellum, base of the brain, eye and spine [6,10-12].

Results of serology depend on the specific test used, cyst burden and phase of the infection [9,12]. Enzyme-linked immunoelectrotransfer blot assay is reported to be highly sensitive (90%) and specific for the diagnosis of human cysticercosis except in a patient with a single cyst or calcified lesion [9,12]. Presence of antibodies may indicate previous exposure to or infection with the parasite and not always active/ current viable infection, serology should always be reviewed along with clinical picture and neuroimaging [12].

Intraventricular NCC can cause hydrocephalus by blocking the CSF outflow tracts and surgical intervention may be required either for cyst removal or for CSF shunting [9]. The treatment of intraventricular neurocysticercosis depends on the location, stage of the cyst, clinical status, complications and includes administration of antihelmintic drugs with steroids, emergency ventriculostomy, resection of the cysts, ventriculo-peritoneal shunt placement, or a combination of these [8,9]. When both hydrocephalus and raised intracranial pressure occur, either a ventriculo-peritoneal shunt or ventriculostomy is required (to control intracranial pressure) while cyst removal can be done by endoscopy [8,9].

Points of interest in our case include- the initial misdiagnosis (with unnecessary antitubercular treatment), absence of severe hydrocephalus and presentation as seizures (without parenchymal neurocysticercal cysts).

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