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A Rare Case of Cortical Blindness Following Cysticercal Encephalitis in a Child

Koonwar S[1], Verma S[2*] and Singh J[3]

[1]Department of Pediatrics, King George's Medical University, Chowk, India

[2]Department of Pediatrics, T.S. Misra Medical College & Hospital, Amausi, India

[3]Department of Psychiatry, Era's Lucknow Medical College & Hospital, Sarfarazganj, India

[*]Corresponding author:

Shalini Verma,
Department of Pediatrics, T.S. Misra Medical College and Hospital, Lucknow, Uttar Pradesh, 226008, India, Tel: +91-9161207547;
E-mail: drshalinikgmu@gmail.com
ORCID Id: orcid.org/0000-0003-0704-069X

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1. Abstract

Neuro-cysticercosis, a parasitic infection of the brain caused by T. solium, is the most common central nervous system parasitosis globally. Seizures, headaches, hydrocephalus and varying degrees of loss of consciousness are common manifestations. Most patients have few intracranial lesions but few present with encephalitic features due to widespread multiple intracranial lesions. We report a one-year-old boy, who presented with low-grade fever, altered sensorium, seizures and features of raised intracranial tension. CT scan showed multiple calcifications in the cerebral cortex and an MRI brain revealed multiple cysts involving the whole brain including the occipital lobe as well as the optic chiasma. On the basis of clinical and radiological grounds, a diagnosis of cysticercal encephalitis was made and he was managed with intracranial pressure-lowering agents, anti-epileptics and steroids. Though his sensorium improved with treatment but visual acuity of only projection of rays (PR+) and perception of light (PL+) was reported.

2. Introduction

Neuro-Cysticercosis (NCC), is caused by cysticercus cellulosae, the larval stage in the multistage infection with Taenia solium which is the most common parasitic infection of the central nervous system worldwide [1]. Patients usually present with seizures but sometimes have headaches, focal deficit, hydrocephalus and raised intracranial pressure. Rarely a few patients present with acute encephalitis and raised intracranial pressure with multiple cysts and diffuse cerebral edema on neuroimaging giving it a starry sky appearance [2, 3]. Cortical blindness is an extremely rare complication of Cysticercal encephalitis with very few reported cases in the literature [4]. This case report describes a rare and interesting case of cysticercal encephalitis with an extremely rare complication of cortical blindness. Very limited literature is available in form of a few case reports on this topic.

3. Case History

A one-year-old boy presented to our emergency with fever, generalized tonic-clonic seizures and altered sensorium for the last one week with no history of trauma or focal neurological deficit. History revealed that the patient was prescribed phenytoin for similar complaints of on-and-off seizure-like movements involving all four limbs for six months of age. There was no associated fever, refusal to feed or altered sensorium when the patient started having the above-mentioned complaint. The patient had a non-contrast-enhanced tomography done at six months of age, suggesting a few hypodense lesions in the frontal and temporal regions.

At presentation patient was hemodynamically stable with a GCS of 9/15, his neurological examination revealed bilateral extensor planters and brisk deep tendon reflexes with no cranial nerve involvement. The rest of the systemic examination, fundus, tubercular workup and metabolic parameters including electrolytes and sugar were within normal limits. A contrast-enhanced magnetic resonance imaging (MRI) of the brain revealed innumerable diffuse scattered ring-enhancing lesions with eccentric scoleces with perilesional edema and calcification in the cerebral and cer-

ebellar hemispheres, pons (Figure 1a & 1b). Most of the lesions were showing blooming on SW1, suggestive of calcification. EEG showed diffuse encephalopathy in form of diffuse delta wave slowing. Routine microscopy and culture of cerebrospinal fluid (CSF) were inconclusive but both anti-IgM and IgG cysticercal antibodies were positive.

All of the above findings along with clinical examination pointed towards the diagnosis of cysticercal encephalitis. He was managed with iv fluids, 20% mannitol, acetazolamide, iv dexamethasone (two days) followed by oral prednisolone (2 mg/kg/day), antiepileptics and supportive care. His sensorium improved gradually over a few days. During their hospital stay, the mother complained of the child's blunted social gaze and lack of fixation. His pupillary reflex was normal, Fundus and B-scan did not reveal anything significant. A diagnosis of cortical blindness was considered. Gradually his GCS improved with no further seizures but his visual acuity was only projection of rays (PR+) and perception of light (PL+). He was discharged with a plan to taper oral steroids in follow-up with neuroimaging and VEP and ophthalmologic check-ups at regular intervals on an outpatient basis. On follow-up, the patient was given oral albendazole and prednisolone was continued. The steroid was tapered gradually after 4 weeks. After 3 months of follow-up, the patient was doing well but cortical blindness was still persisting.

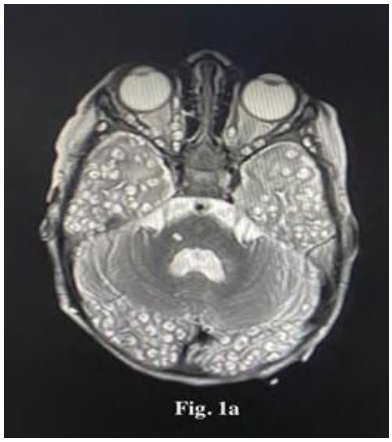

Figure 1a: T2 weighted Axial view MRI showing innumerable inflammatory granulomas involving orbit and brain parenchyma

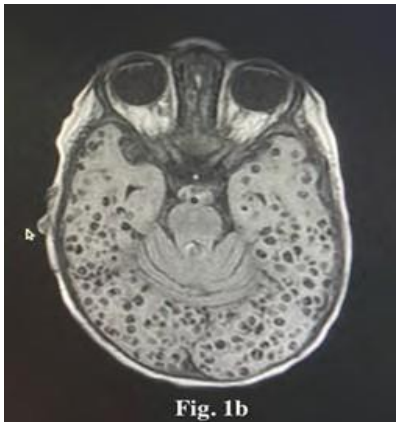

Figure 1b: T1 weighted Axial view MRI showing innumerable inflammatory granulomas involving both of the brain parenchymas

4. Discussion

NCC mostly presents with few intracranial lesions However, few of them develop multiple lesions leading to encephalitic and non-encephalitic presentations [5]. Our patient presented at the young age of one year with features of raised intracranial tension and encephalitis. During the hospital stay, our patient complained of diminished visual acuity with normal ophthalmic workup pointing towards cerebral visual impairment. Prasad et al have also reported a case of a six-year-old boy with cysticercal encephalitis who presented with cortical blindness in the form of diminished visual acuity of just projection of rays (PR+) and perception of light (PL+) (4). Ocular cysticercosis and Neuro-retinitis have also been reported in the past but in our patient fundus was normal on many occasions and B- scan also did not reveal anything which further strengthens the diagnosis of cortical blindness [4, 6]. The diagnostic criteria of NCC suggested by García et al et al were fulfilled by our patient [7]. MRI is the neuroimaging of choice for assessing the extent of infection, location, various stages and degenerative changes of the parasites, and visualisation of perilesional oedema. CT, however, remains more sensitive for the detection of calcifications [8].

Symptomatic management is the mainstay of treatment in these cases. Seizures generally respond to the first line of anti-epileptics which should be continued for seizure prophylaxis for a varied period of time. Raised ICP should be managed on a priority basis by using iv and oral steroids in minimal doses and for the shortest possible duration and hypertonic saline and cerebral dehydrants if not contraindicated [9]. Anti–helminthic drugs are contraindicated in cysticercal encephalitis due to the risk of exacerbating inflammatory response leading to worsening of intracranial oedema and eventually trans-tentorial herniation [7]. CT and MRI should be repeated after three months to monitor the treatment efficacy [4].

This case report emphasizes the possibility of NCC as one of the differential diagnoses in cases presenting with features of encephalitis, especially in developing countries. The blindness may be sometimes cortical instead of due to local lesions. Early accurate diagnosis and cautious prognostication are solicited for optimal management.

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