Unusual presentation of more common disease/injury

Cysticercal encephalitis with cortical blindness

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Summary

The authors report a 6-year-old boy, who had presented with low-grade fever, altered sensorium, headache and seizure for 5 days. On examination, he had features of raised intracranial pressure with left VI cranial-nerve palsy and bilateral extensor plantar response. CT scan showed multiple calcifications in cerebral cortex. MRI cranium showed multiple cysts involving whole of the brain. He was diagnosed as having cysticercal encephalitis, based on immunological and imaging study. He was managed with 20% mannitol, phenytoin and albendazole, and regained consciousness 7 days later, but had residual neurological deficit as left-lower-limb monoparesis and visual acuity of just projection of rays (PR+) and perception of light (PL+).

BACKGROUND

Neurocysticercosis is the most common parasitic infection of central nervous system and result from infection with the intermediate stage of *Taenia solium*. In India, cysticercosis is highly prevalent and CNS involvement is seen in 60–90% of infested patients.¹ Cerebrum and cerebellum are common sites but may involve brainstem, basal ganglion, thalamus and lateral sinus. Seizures are the commonest presenting feature in majority but can present with headache, focal deficits, hydrocephalus and raised intracranial pressure.² Cysticercal encephalitis with cortical blindness has been reported in only one patient in literature till now.³

CASE PRESENTATION

A 6-year-old boy was brought to emergency with complains of fever, altered sensorium, headache and generalised tonic–clonic seizure for 5 days. After care of airways and breathing, seizure was controlled with intravenous lorazepam (0.1 mg/kg) and then phenytoin (loading dose: 15 mg/kg followed 5 mg/kg twice daily). There was no past history of seizures, head trauma, ear discharge and tuberculosis. On examination, he was febrile (38°C) and unconscious (GCS: 8/15) with left VIth cranial palsy. His pupils were normal in size and reacting normally to light. There were no signs of meningeal irritation. On motor examination, all four limbs had power of grade III/V, exaggerated deep tendon reflexes and bilateral extensor plantar response. Funduscopy did not show papilledema or any retinal cyst. The macula was normal. The examination of chest, cardiovascular system and abdomen were unremarkable.

INVESTIGATIONS

On investigation, his CBC, x-ray's chest, blood glucose, electrolytes, liver and renal function tests were normal. The Mantoux test was negative. Gastric aspirate (three morning samples) stain for acid fast bacilli was negative. Cerebrospinal fluid study showed cells, 12/mm³ (Lymphocyte 80% and Neutrophil 20%); protein, 52 mg/dl; and sugar was 68 mg/dl. ELISA of cerebrospinal fluid for cysticercal antibody,

both IgM and IgG, was strongly positive. Cerebrospinal fluid culture for both bacteria and viruses was sterile.

CT scan showed multiple calcifications in cerebral parenchyma (figure 1). MRI cranium showed multiple hyperintense lesions with few hypointense lesions with surrounding edema in brain parenchyma (figure 2). A final diagnosis of cysticercal encephalitis was made.

TREATMENT

The patient was treated with intravenous fluid, 20% mannitol (1 g/kg/dose intravenously 8 hourly for 2 days), dexamethasone (2 days), and then prednisolone (1.5 mg/kg/day) and albendazole (15 mg/kg/day twice daily) were given for 4 weeks. On day 7, patient regained consciousness but had left-lower-limb monoparesis with grade III power. His visual acuity was just projection of rays (PR+) and perception of light (PL+). His pupillary reflex was normal.

OUTCOME AND FOLLOW-UP

The patient was put on regular physiotherapy. At 2 weeks, child was seizure free but had persisting monoparesis and cortical blindness. At 6 weeks, monoparesis was persisting, which got resolved after 6 months; however, cortical blindness continued to persist. Repeat CT and MRI of brain at 3 months of follow-up were normal.

DISCUSSION

Neurocysticercosis is a life-threatening illness. It has myriads of manifestations and includes seizures, headache and focal neurological deficits. Unusual manifestations are stroke, visual loss, ataxia, dystonia, dementia and hydrocephalus.^{2–4} Cysticercal granuloma as cause of Weber's syndrome and ptosis⁵ have also been reported. Cerebral infarction may result from obstruction of small terminal arteries or vasculitis. Intraventricular (5–10%) and meningeal cysticercosis are associated with hydrocephalus, signs of meningeal irritation and raised intracranial pressure. Our patient had cysticercal encephalitis with cortical blindness.

Figure 1 Tomography showing multiple calcifications in whole brain parenchyma.

Liberation of cytokines either due to natural degeneration of active cysts or death due to albendazole therapy may lead to increase in body temperature, raised intracranial pressure and intractable seizure. Neuro-retinitis and occipital lobe syndrome has been reported in four patients of neurocysticercosis.⁶ This is possibly the second case report of cysticercal encephalitis with cortical blindness.

CT scan and MRI are useful in anatomical localisation of cysts, but MRI cranium is more sensitive in picking up active lesions. Our case fulfilled the diagnostic criteria of neurocysticercosis.⁷ Patients with active cysts remain at risk of serious complication. Therefore, all patients with multiple cysts should receive treatment with steroid to reduce intracranial pressure and cysticidal drug, that is, albendazole. Efficacy of treatment should be monitored by repeat CT and MRI after 3 months.

This report highlights the importance of suspecting neurocysticercosis in cases presenting conditions like encephalitis in tropical countries and not to start albendazole without proper evaluation.

Learning points

- ▶ Cysticercosis is common in tropical countries, including India.
- ▶ Cysticercal encephalitis is rare as presentation of neurocysticercosis.
- ▶ Neurocysticercosis resulting in cortical blindness is extremely rare.

Competing interests None.

Figure 2 T-1 weighted image showing multiple hyperintense with few hypointense lesions with perilesional oedema.

Patient consent Obtained.

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