Learning from errors

Why MRI of brain is superior to CT in multiple neurocysticercosis?

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Summary

A 6-year-old girl presented with history of fever, headache and partial seizures with secondary generalisation. The cerebrospinal fluid analysis revealed pleocytosis and elevated proteins. The clinical evaluation and cerebrospinal fluid analysis was suggestive of chronic meningitis. The patient was started antituberculous therapy considering tuberculous meningitis. The authors report this case because MRI of brain showed multiple cysticerci throughout the brain parenchyma which was completely missed on CT brain. Antituberculosis treatment was withdrawn and the patient was advised albendazole, antiepileptic drug and steroids. The patient showed remarkable improvement at follow-up after 3 months.

BACKGROUND

Neurocysticercosis (NCC) is a common parasitic infection of brain parenchyma. It is one of the common causes of symptomatic epilepsy in the developing world, especially in rural areas.¹ CT is a common screening investigation for diagnosing brain pathology. However, CT brain may fail to visualise vesicular neurocysticercal stage as well as meningeal NCC. On other hand, MRI may not demonstrate calcific NCC. NCC may be missed or confused for various central nervous disorders (central nervous system (CNS)) like CNS tuberculosis. In our patient, CT brain did not reveal any lesion. Her clinical history and cerebrospinal fluid (CSF) analysis were suggestive of chronic meningitis (tuberculous meningitis). Subsequently, MRI brain revealed multiple cysticerci in vesicular and colloidal stage throughout the brain parenchyma. In this case report, we highlight that MRI should be preferred over CT to rule out granuloma in the brain in patients with symptomatic epilepsy in NCC endemic regions.

CASE PRESENTATION

A 6-year-old girl child, who belongs to North India, admitted with history of fever, headache and vomiting since 5 months and seizures for last 1 month. Child's mother gave history of low to moderate grade of fever occurring almost daily and partly responsive to medication but recur once medication was stopped. It was accompanied by headache which started a month later. The headache was of moderate to severe intensity, holocranial, interfering in the child's daily activity and was accompanied by vomiting which was projectile and occurred immediately after eating anything. Since last month, she had four to five episodes of partial seizures involving right upper limb and right half of face with secondary generalisation with up rolling of eyes and followed by loss of consciousness. On examination, the patient was drowsy (Glasgow coma scale 10/15), terminal neck rigidity was present. There was no restriction of ocular movements and papilloedema was not found on fundus evaluation. The focal neurological deficit was

not present. The deep tendon jerks were 2+ and plantars showed flexor response.

INVESTIGATIONS

Her haemogram, liver function tests and renal function tests were within normal limits. CT brain with contrast revealed sulcal effacement suggestive of cerebral oedema (figure 1). Cerebrospinal fluid examination showed total cell count 20/mm³, all lymphocytes, protein 78.2 mg/dl, CSF sugar 90.3 mg/dl and corresponding blood sugar 143 mg/dl. Subsequent MRI of brain showed multiple cystic

Figure 1 CT of brain revealed, effacement of sulci, suggestive of cerebral oedema.

lesions throughout the cerebral hemispheres, periventricular region, cerebellar lobes and brainstem (figure 2). Spoiled gradient recalled acquisition with contrast study demonstrated multiple ring enhancing lesions (figure 3).

The ELISA showed positive results for cysticercal antibodies. The viral study in serum as well as in CSF was negative for herpes virus, Japanese encephalitis, cytomegalovirus and Epstein–Barr virus. The polymerase chain reactivity in CSF, depicted negative result for *Mycobacterium tuberculosis*.

TREATMENT

She was administered intravenous dexamethasone 0.4 mg/kg body weight/day, mannitol 0.25 g/kg body weight every 6 hourly, tab. acetazolamide 125 mg three times a day, tab. oxcabamezapine 150 mg twice daily and tab.

Figure 2 MRI, T2 fluid attenuated inversion recovery, demonstrated multiple hypointense cystic lesions with intense perifocal oedema suggestive of cysticercal encephalitis.

clobazam 5 mg HS. She was started on antituberculosis treatment (ATT) comprising of rifampicin 10 mg/kg, isoniazid 5 mg/kg, pyrazinamide 25 mg/kg and intramuscular streptomycin 150 mg daily after her CSF examination reports. Initially MRI could not be done due to financial limitations of patient. However, ATT was stopped once MRI was done for the patient after 5 days of starting the treatment. The albendazole therapy was initiated after taking precautions for anaphylactic reactions.

OUTCOME AND FOLLOW-UP

The patient seizures were well controlled and remained asymptomatic at follow-up after 3 months.

DISCUSSION

NCC caused by *Tinea solium*, is an endemic parasitic infection of brain in developing world. It is one of the common

Figure 3 Spoiled gradient recalled image with contrast study showed multiple ring enhancing lesions, suggestive of multiple neurocysticercosis.

Table 1 Neuroimaging of neurocysticercosis

Neuroimaging of neurocysticercosis	Computerised scanning (brain)	MRI (cranium)
Vesicular	Rounded hypodense lesions without contrast enhancement	Rounded lesions similar to CSF signals on T1 and T2 weighted imaging, hyperintense nodule (scolex)
Colloid	Ill-defined lesions with peri focal oedema with ring enhancement	T2 weighted image shows thick and hypointense wall with perilesional oedema
Granuloma	Nodular hyperdense lesion with peri focal oedema on contrast study	Areas of signal void on T1 and T2 weighted image with hyperintense rim and perilesional oedema with contrast administration
Calcified	Small hyperdense nodule, usually absence of perifocal oedema, no contrast enhancement	Calcified lesions usually not visualised on MRI
Meningeal (subarachnoid)	Hypodense lesions in subarachnoid spaces, may show hyperdense ependymal layer	Hydrocephalus, may show periventricular lucencies, sometimes large multi-lobulated appearance in cerebrospinal fluid spaces
Cysticercal encephalitis	Diffuse swelling of brain, intense cerebral oedema, small chinked ventricles. Contrast study reveals multiple small nodular lesions	Global brain oedema, ventricular effacement, contrast MRI shows multiple disseminated ring and nodular lesions

CSF, cerebrospinal fluid.

Table 2 Important radiological differential diagnosis of multiple lesions of brain

Infective
Multiple tuberculoma
Multiple neurocysticercosis
Multiple abscess
Histoplasmosis
Actinomycosis
Cryptococcosis
Toxoplasmosis
Neoplasm
Multiple metastasis
CNS lymphoma
Inflammatory-demyelinating disorders
Multiple sclerosis
Acute disseminated encephalo-myelitis
Neurosarcoidosis
Systemic lupus erythematosus

CNS, central nervous system.

causes of symptomatic epilepsy in the rural areas of India due to poor sanitation and hygiene.¹⁻³ The NCC has plethora of clinical manifestations depending upon site, size and number of lesions which include seizures, encephalopathy, pyramidal tract symptoms, cranial neuropathy and raised intracranial pressure.⁴ The imaging findings of NCC may depend upon the stages of evolution of the parasite (table 1).⁵ The live parasite or 'vesicular stage' appears as small non-enhancing, hypodense lesion without perilesional oedema. Vesicular cysticerci may be occasionally missed on CT brain.⁶ On MRI, it has characteristic 'hole with dot' sign due to visualisation of scolex. In a study from Central India, Patil *et al* showed that scolex could be visualised only in 37.5% cases of vesicular cysticerci.² The dying parasite incites a strong inflammatory response leading to an ill-defined ring enhancing lesion with perilesional oedema. This is known as 'colloidal stage' which appears hyperintense on T1 and T2 weighted images and fluid attenuated inversion recovery images with perilesional oedema. The granular nodular stage appears as solid or ring enhancing lesion with minimal oedema. Presence of calcified lesions

in the brain parenchyma may be an incidental finding on the CT scan or hypointensities on gradient echo imaging (table 2).^{5 6}

In this case, CT brain was suggestive of cerebral oedema but no ring lesion could be visualised. However, MRI brain showed presence of multiple vesicular and colloidal stage cysticerci with perilesional oedema. Although MRI brain is the imaging modality of choice for NCC, on many occasions it may be prohibitory due to financial constraints and presentation of calcific NCC may not be visualised. In our case, the diagnosis could have been easily missed if MRI brain was not done.

Learning points

- ▶ NCC can be missed on CT brain.
- ▶ CT should be screening investigation for NCC.
- ▶ MRI brain is a superior investigation in the diagnosis of NCC. It is of great help in intraventricular NCC, brainstem cysts and subarachnoid NCC.
- ▶ NCC may also cause CSF pleocytosis with elevation of proteins mimicking chronic meningitis like tuberculosis.

Competing interests None.

Patient consent Obtained.

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