Unusual association of diseases/symptoms

Bilateral ptosis: an atypical presentation of neurocysticercosis

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Summary

Isolated ptosis is a rare presentation of neurocysticercosis (NCC). Here we report the case of a 45-year-old woman who presented with acute onset bilateral symmetrical ptosis with no other neurological deficit. Her imaging revealed a single cystic ring-enhancing lesion with perilesional oedema in the midbrain suggestive of NCC. She responded well to prednisolone.

BACKGROUND

Neurocysticercosis (NCC) is the most common parasitic infection of the central nervous system (CNS) in developing countries like India. Seizures are the commonest presentation of NCC though it has many rare presentations too. NCC presenting as bilateral ptosis has been rarely reported and sometimes can be confused with myasthenia gravis. NCC needs to be considered as a differential diagnosis of bilateral ptosis especially in endemic regions.

CASE PRESENTATION

A 45-year-old woman presented to us with acute onset bilateral ptosis of 4 days duration (figure 1A). She noticed drooping of both eyelids which progressed over 1–2 days to cover most part of her eyes. Owing to this, she had difficulty seeing for which she had to keep her head in extended position and elevate her eyelids with her hands most of the time. Drooping of eyelids was not fluctuating. There was no history of seizures, headache, vomiting or decreased vision. There were no complaints of double vision, fatigability, weakness, tingling, numbness or ataxia and no history suggestive of other cranial nerves involvement either. On examination she had bilateral symmetrical ptosis with intact extra ocular movements. Pupils were equally reacting to light and fundus was normal. Other cranial nerves, motor, sensory, reflexes and cerebellar examinations were normal. There was no neck rigidity or Kernig's sign. Suspecting myasthenia gravis, neostigmine test (1.5 mg) was done which turned out to be negative. Then, the patient was subjected to MRI of brain to rule out any structural lesion.

INVESTIGATIONS

Her routine biochemical parameters, complete haemogram, renal and liver function tests were normal. Cerebrospinal fluid analysis did not reveal any abnormality other than ELISA for immunoglobulin M antibodies for cysticercus antigen which was positive.

MRI with T1W, T2W, fluid attenuated inversion recovery (FLAIR) and 3D spoiled gradient recalled echo sequence (SPGR) sequences was done. It showed a cystic lesion with ring enhancement on gadolinium contrast in the dorsal midbrain (figure 2). Perilesional oedema was also present.

DIFFERENTIAL DIAGNOSIS

On the basis of history and examination, the following differential diagnosis was considered: (1) Myasthenia gravis—but, duration of illness was short and history and examination did not reveal fatigability. (2) Midbrain lesion—acute onset bilateral ptosis could be due to midbrain structural lesion such as inflammatory granuloma, haematoma or glioma. But the patient had no other neurological deficit such as long-tract signs or diplopia which is expected from a lesion in such a compact structure. (3) Miller Fisher syndrome—It is characterised by a triad of areflexia, ataxia and ophthalmoplegia. Patient had ptosis, but did not have double vision (which is usually the earliest symptom) or sensory ataxia.

After investigation (especially MRI) 1) Tuberculoma—it is another common central nervous system (CNS) infection in this part of the world. Tuberculomas are usually irregular, solid or disk shaped, and >2 cm in size. It is usually associated with perifocal oedema, mass effect and focal neurological deficits. But cysticerci are usually round in shape, <2 cm in size, with ring enhancement or visible scolex, and minimal cerebral oedema, but not severe enough to cause midline shift.

In this patient, single ring-enhancing lesion of <2 cm with minimal perifocal oedema is seen, which is suggestive of NCC; and ELISA was positive for cysticercal antigen.^{1 2} So a final diagnosis of NCC was made.

TREATMENT

Oral steroid, prednisolone 40 mg was given for 1 week. Prednisolone was tapered off over the next 3 weeks. Albendazole 400 mg was given for 3 days during the second week.

OUTCOME AND FOLLOW-UP

Patient improved after starting steroids. In 5 days, her ptosis improved completely (figure 1B). She was asymptomatic till the follow-up of 3 months. Repeat imaging was not done.

DISCUSSION

NCC is the commonest parasitic infection of CNS especially in developing countries like India. It is caused by the larval stage of *Taenia solium*.³ Clinical manifestations

Figure 1 Bilateral symmetric ptosis at the time of admission (A), improvement in ptosis after treatment (B).

of NCC are many depending on location, number and size. It can be intraventricular, intraparenchymal, sub-arachnoid and intraspinal in location. It usually presents with seizures, but many non-epileptic uncommon manifestations of NCC such as cranial neuropathy, meningitis, hydrocephalus and dementia also occur. Cranial nerve involvement in NCC may be due to various causes. Ramina *et al*⁴ reported a large cyst at the cerebellopontine angle causing sensorineural hearing loss, facial palsy, trigeminal sensory symptoms accompanied by long-tract dysfunction. Keane⁵ found that NCC causes raised intracranial pressure due to hydrocephalus, subacute meningitis and mass effect due to cysticerci and their surrounding oedema presents with papilledema as the most common sign with frequent pupillary abnormalities and nystagmus. Cysticercal arachnoiditis causes entrapment of cranial nerves. The oculomotor nerves have a long course along the basal meninges which predisposes them to entrapment.⁵ Lacunar stroke involving cranial nerve nuclei and other long tracts occurs due to involvement of smaller vessels in cysticercus arachnoiditis.⁶ Such cranial nerve or nucleus involvement usually presents with diplopia and blurring of vision due to extraocular muscle paralysis. Cranial nerve dysfunction due to parenchymal cysts is uncommon.

In our patient symmetric, simultaneous, bilateral ptosis occurred due to oculomotor subnucleus involvement in the dorsal midbrain due to NCC. Various other causes of

similar presentations were reported such as midbrain glioma, hematoma⁷ and infarct.⁸ Oculomotor involvement due to NCC is unusual and bilateral ptosis due to brainstem parenchymal involvement has rarely been reported. Singhi *et al*⁹ reported two children who presented with sudden onset ptosis due to midbrain NCC. A case of fluctuating ptosis presenting like myasthenia was seen by Netravathi *et al*.¹⁰ Third cranial nerve palsy due to midbrain NCC which rapidly deteriorated on tapering steroids was also reported by Kim *et al*.¹¹

Ptosis is caused by weakness of elevator muscles of eyelids, either levator palpebrae superioris (LPS) or muller muscle. Levator dysfunction is caused by muscle, nerve or nucleus involvement. Oculomotor nucleus is a complex of subnuclei lying in the caudal midbrain. A small single central subnucleus supplying both LPS lies in the caudal part of oculomotor nucleus complex, involvement of which leads to ptosis. In our patient MRI with gadolinium contrast revealed a single cystic ring-enhancing lesion in the dorsal midbrain with perilesional oedema which would have caused dysfunction of LPS subnucleus resulting in bilateral symmetrical ptosis. It responded well to prednisolone in terms of reduction of oedema.

Bilateral symmetric ptosis without any other neurological symptoms or signs is a rare presentation of NCC and it should be considered as a differential diagnosis of ptosis especially in endemic regions.

Figure 2 Contrast SPGR study showing dorsal midbrain ring-enhancing cystic lesion with perilesional oedema in axial (A), sagittal (B) and coronal (C) sections.

Learning points

- Neurocysticercosis (NCC) has protean manifestations.
- Ptosis because of midbrain NCC is one of the rare presentations.
- It should be considered in differential diagnosis of bilateral symmetric ptosis especially in the endemic region.

Competing interests None.

Patient consent Obtained.

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