Unusual presentation of more common disease/injury

Spastic foot-drop as an isolated manifestation of neurocysticercosis

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Summary

Foot-drop is a rare but important manifestation of intracranial pathologies ranging from space-occupying lesions to cerebrovascular accidents. Being most commonly associated with peripheral nerve lesions or radicular compressions, it remains an underappreciated feature of central-structural abnormalities. We describe an interesting case of a 14-year-old boy who had presented with acute onset right-sided foot-drop due to a left-sided parasagittal neurocysticercus lesion, without seizures and discuss the location of the lesion in the precentral area in reference to Penfield's motor homunculus.

BACKGROUND

Foot-drop is defined as weakness of dorsiflexors of foot in varying combinations with weakness of foot everters and toe extensors. It is commonly caused by peripheral lesions like L5 radiculopathy or peroneal nerve palsy.¹ Foot-drop due to a central lesion is better known as a spastic foot-drop, owing to the presence of upper motor neurone signs, and is caused by lesions affecting the parasagittal precentral (motor) gyrus representing the leg area.²⁻³ A review of literature shows that head trauma, cortical dysplasia, abscess, stroke, metastatic and primary brain tumours are the various central lesions which can manifest as foot-drop.⁴⁻⁹ Neurocysticercosis is an important cause of localisation-related epilepsy in India and its presentation as foot-drop in isolation is yet to be documented. Such a presentation leads to diagnostic uncertainties that need to be dissected to prevent unnecessary investigations and delay in diagnosis.

CASE PRESENTATION

A 14-year-old boy presented to our institute, a tertiary care neurology centre, with complaints of acute onset weakness of the right leg for 4 days. The weakness was appreciated while the boy was playing, when he was not able to hold his footwear properly in his right foot. Difficulty in walking followed over the next few hours in the form of inability to clear the ground without tripping. There was no difficulty in getting up from the squatting position or suggestion of involvement of the upper limbs or any cranial nerve. He denied any history of backache, radicular pain or trauma in the recent or remote past. There was no history of seizures, sensory abnormalities and bladder or bowel complaints.

On examination, he was conscious, orientated and cooperative; higher mental functions were intact. There was no cranial nerve deficit. At the right ankle, dorsiflexion was grade 0/5 while plantar-flexion was grade 2/5 on Medical Research Council (MRC) scale; extensor hallucis longus contraction was grade 1/5. Power at other joints

and in rest of the muscle groups was normal. Deep tendon reflexes were normal with an extensor plantar response on the right side. Primary modalities of sensation as well as cortical sensations were normal. There was no cerebellar dysfunction. He had a high steppage gait as evidenced by an abnormal lift of the right lower limb. Examination of the peripheral nerves, and search for any cutaneous marker, did not reveal any abnormality. The nomenclatures used in this manuscript are in accordance with the *Terminologia Anatomica*.

INVESTIGATIONS

The patient was evaluated in compliance with the *Declaration of Helsinki*. Haemogram and serum biochemistry were normal. The serology was non-reactive to HIV-I and II. MRI of the brain revealed two cystic lesions, one in the left parasagittal precentral (motor) strip and the other one in the right temporo-occipital region, with perilesional oedema (figure 1A,B,D,E). The spoiled gradient recalled acquisition images with gadolinium contrast showed ring-enhancement of the lesions as well as visualisation of the scolex in the right temporo-occipital lesion (figure 2C,F). T2-weighted coronal image rendering to demonstrate the Penfield's motor homunculus was done to show the area of involvement (figure 2A,B). Electroencephalography and nerve conduction studies did not reveal any abnormality. Direct and indirect ophthalmoscopy was normal.

TREATMENT

The patient was initiated on oxcarbazepine (15 mg/kg body weight in two divided doses), in view of the lesion involving the motor strip, to prevent triggering of any epileptic activity. The patient was primed with oral prednisolone (0.75 mg/kg body weight) for 3 days before starting albendazole (15 mg/kg body weight in two divided doses) which were administered for 3 and 2 weeks, respectively.

Figure 1 MRI of the brain depicts a cystic lesion (arrow) with scolex-associated signal variation in the left parasagittal precentral cortex on axial T2-weighted (A) and fluid-attenuated inversion recovery (B) sequences, with perilesional oedema; a lesion with similar characteristics (arrow) is visualised in the temporo-occipital region on axial T2-weighted (D) and fluid-attenuated inversion recovery (E) sequences. Axial-spoiled gradient recalled acquisition images with gadolinium contrast (C and F) show ring-enhancement of the lesions (arrow) as well as visualisation of the scolex in the right temporo-occipital lesion.

OUTCOME AND FOLLOW-UP

The patient started showing improvement from the third day onwards and had significantly improved by the end of second week. At the time of discharge on the 24th day, mild residual weakness (grade 4+/5 by MRC scale) in the extensor hallucis longus was the only deficit.

DISCUSSION

Foot-drop is defined as weakness of dorsiflexors of foot in varying combinations with weakness of foot everters and toe extensors.¹ Tibialis anterior, extensor hallucis longus and extensor digitorum longus act as dorsiflexors of foot and ankle; these muscles derive their major innervations

Figure 2 Coronal section of the T2-weighted (A) MRI of the brain depicts the parasagittal lesion. A rendered image (B) at the same level demonstrates the Penfield's motor homunculus and shows the area of involvement (toes and ankle).

from the fourth and fifth lumbar, and partially from the first sacral nerve. An involvement of the peripheral nervous system starting from the level of radicals to the innervating nerves may lead to weakness of these muscles leading to foot-drop. The commonly observed causes of foot-drop are common peroneal nerve entrapment at the fibular head and lower lumbar disc prolapse with nerve root compression; other causes include trauma to the leg, leg compartment syndromes, peripheral polyneuropathies and systemic diseases such as connective tissue disorders, vasculitis and diabetes mellitus.¹ Hansen's disease should be suspected especially in a tropical country like India.

Guthrie *et al* coined the term 'spastic foot-drop' to differentiate the same from the commonly observed lower-motor-neurone phenotype. It was defined as 'spastic' owing to the presence of upper motor neurone signs, and was observed to be caused by lesions affecting the parasagittal precentral (motor) gyrus representing the leg area.² The Penfield's motor homunculus provides a visual somatotopic localisation of ankle and toe in the parasagittal region which has also been confirmed by brain functional localisation studies.¹⁰

In our patient, the common peripheral causes of foot-drop were excluded by clinical examination, laboratory findings and normal nerve conduction studies. Findings incompatible with the diagnosis of a peripheral cause of foot-drop included motor deficit inconsistent with nerve distribution, lack of sensory deficit or paraesthesias, preserved deep tendon reflexes and an extensor plantar response; these details did not favour a cord lesion either. Technically, our patient presented with a flail foot which prompted us to perform an MRI of the brain, which revealed cystic cortical lesions.

Various central lesions reported to cause foot-drop are stroke, brain tumour, head injury, cortical dysplasia and abscess;^{2–9} neurocysticercosis, however, has not been reported. Neurocysticercosis is a parasitic disease of the central nervous system caused by the larval stage of *Taenia solium*. Neurocysticercosis is widely endemic in most of the tropical areas of the world, especially the poor ones, and is a major cause of seizures.¹¹ Importantly, seizure is the most common manifestation of a cortical neurocysticercal lesion and its presentation otherwise, as in our case, is very atypical. MRI of the brain showing a cystic lesion with demonstration of scolex is an absolute criterion for diagnosing neurocysticercosis.¹² In our patient, signal intensity variation consistent with scolex could be seen on T2-weighted and fluid attenuated inversion recovery sequences; gadolinium contrast study depicted the ring enhancing nature of the lesions and scolex in the temporo-occipital lesion.

Image rendering of coronal T2-weighted sequence depicted the clinico-radiological association in our case, complementing the structural imaging of the cortical lesion.

Learning points

- ▶ Parasagittal precentral (motor) gyrus represents the leg area and its involvement may cause a contralateral foot-drop.
- ▶ Presence of upper motor neurone signs, defined as spastic foot-drop, should prompt a search for a central cause of the same.
- ▶ Although seizure is the most common presenting feature of neurocysticercosis, it may atypically present with focal neurological deficit posing diagnostic dilemma.
- ▶ MRI with gadolinium contrast should be used to investigate a central cause of the foot-drop.

Competing interests None.

Patient consent Obtained.

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