Foot Drop Caused by A Focal Brain Injury  
- Two Case Report -

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Foot drop is caused by peripheral and spinal motor neuron lesions and muscular dystrophy. Foot drop secondary to brain lesions is rarely seen in practice. We present two cases with foot drop caused by focal brain injury. A 49-year-old male patient was admitted with right foot drop for 4m falling down injury. Neurologic examination revealed normal except for weakness at dorsiflexion of the right ankle and big toe. L-spine MRI, L-spine CT and both legs EMG showed normal findings. A 69-year-old male patient was referred with left foot drop and headache after bicycle accident. Neurological examinations showed normal except for weakness in dorsiflexion of the left ankle and big toe without sensory loss. L-spine MRI, L-spine CT and both legs EMG showed normal findings. In first case, there are important focal brain injury lesions in premotor area and SMA. In second case, premotor area, SMA and primary motor area may be damaged. In two cases, hemorrhagic cortical contusions in parasagittal area were the causes of the foot drop. And the foot drop was the initial clinical presentation in both cases. Clinicians, if they detect upper motor type neurologic deficits such as positive Babinski’ sign, hyper-reflexia or clonus in a patient with a foot drop, should take into account the possibility of central lesion as reason for the foot drop.

Key Words: Foot drop · Focal brain injury

INTRODUCTION

Foot drop is caused by peripheral and spinal motor neuron lesions and muscular dystrophy. Foot drop secondary to brain lesions is rarely seen in practice. Failure in giving proper consideration to this possibility may result in an unnecessary delay in diagnosis through preoccupation with much investigation not needed otherwise. In this report, we present two cases of foot drop caused by focal brain injury.

CASE REPORT

CASE 1

A 49-year-old man was referred to the emergency room for head injury due to fall down. He showed right foot drop and his neurologic examination was normal except for weakness at dorsiflexion of the foot. The power of right ankle dorsiflexion and right toe was grade 1/5 and positive Babinski’s sign, hyper-reflexia in right deep tendon reflex were noted. There was 4cm scalp laceration in left frontoparietal area. Brain CT showed diastatic fracture of sagittal suture and comminuted fracture of left frontoparietal vertex of the skull vault(Fig 1-A.) and hemorrhagic cortical contusion in left premotor area. Axial, Coronal T2 weighted magnetic resonance image (MRI) showed a hemorrhagic cortical contusion in left premotor area(Containing SMA) as hypointense center with hyperintense in the periphery(Fig 1-B, C). Lumbar computed tomography, lumbar magnetic resonance
Fig. 1-A. Diastatic fracture of sagittal suture and comminuted fracture of left frontoparietal vertex of the skull vault and hemorrhagic cortical contusion in left premotor area.

Fig. 1-B. Axial T1WI, T2WI MRI showing hemorrhagic cortical contusion in left premotor area (containing SMA) as hypointense center with hyperintense periphery.

Fig. 1-C. Coronal and Sagittal T2WI showing a hemorrhagic contusion in left premotor area (containing SMA) as hypointense center with hyperintense in the periphery.

Fig. 2. Axial T1WI and T2WI showing resolved hemorrhagic cortical contusion left premotor area (containing SMA).

Fig. 3-A. Axial T1WI and T2WI showing hemorrhagic contusion in the right precentral gyrus, postcentral gyrus and premotor area (containing SMA).

Fig. 3-B. Sagittal and Coronal T1WI showing hemorrhagic contusion in the right precentral gyrus, postcentral gyrus and premotor area (containing SMA).

Imaging (MRI) and electromyography studies were performed and all of these studies demonstrated no pathologic finding. On discharge after 3 weeks, he was improved in all respects except for some residual weakness (4/5) (Fig 2).

**CASE 2**

A 69-year old man suffering from left foot drop and headache after bicycle accident was referred to a neurologist. Neurological examination revealed weakness of left knee flexion and left ankle and toe dorsiflexion without sensory loss. The power of left ankle dorsiflexion and left toe was grade 1/5 and positive Babinski's sign, hyper-reflexia in the left deep tendon reflex were noted. Brain CT and MRI showed epidural hematoma in right frontal convexity and hemorrhagic brain contusion in the right precentral gyrus, postcentral gyrus and premotor area (containing SMA) (Fig. 3). There was no spinal lesions on lumbar magnetic
Table 1. Reported cases with foot drop caused by focal brain injury

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Pathologic Findings</th>
<th>Symptoms</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lt. Superior frontal gyrus (SMA)</td>
<td>Cranial gunshot wound</td>
<td>Rt. Foot drop</td>
<td>N. OZDEMIR et. al. Zeki Gokcil</td>
</tr>
</tbody>
</table>

DISCUSSION

An upper motor neuron lesion affecting the pyramidal tract, a cord lesion affecting the L5 motor neuron pool, a spinal lesion interfering with L5 outflow, or peripheral lesions affecting the L5 nerve root, lumbosacral trunk, the sciatic nerve peroneal division, or the peroneal nerve in exclusion may all result in foot drop [7,9,13,14]. The most common causes of foot drop are fourth, fifth lumbar lesion (disc herniation, metastatic lesion, neurofibroma, meningioma etc.) and peroneal nerve lesion (diabetic neuropathy, fracture of the head of the fibula etc.) [5,11,16]. First case, important focal brain injury lesions were noted in premotor area and SMA. In case of second case, premotor area, SMA and primary motor area may be damaged. There are reported cases with foot drop caused by focal brain injury(Table 1) [5,11,16]. Three principle motor areas are recognized in the cerebral cortex: ① the primary motor area, ② the premotor area, and ③ the supplementary motor area [9]. The primary motor area, commonly called area 4 of Brodmann, is located on the anterior wall of the central sulcus and adjacent portions of the precentral gyrus. The region designated as 6aα on the lateral convexity of the hemisphere is considered the premotor area; area 6aα on the medial aspect of the hemisphere is the supplementary motor area [9,11,12]. Primary motor area contains a group of networked cells in mammalian brains that controls movements of specific body parts associated with cell groups in that area of the brain. The area is closely linked by neural networks to corresponding areas in the primary somatosensory cortex. There is a precise somatotopic representation of the different body parts in the primary motor cortex, with the leg area located medially (close to the midline), and the head and face area located laterally on the convex side of the cerebral hemisphere (motor homunculus) [4,7]. The arm and hand motor area is the largest and occupies the part of precentral gyrus, located in between the leg and face area. It receives input from several areas involved in higher-order motor control, including the supplementary motor area, premotor area, and posterior parietal cortex [11,15]. The primary motor cortex (also known as M1) works in association with pre-motor areas to plan and execute movements. M1 contains large neurons known as Betz cells which send long axons down the spinal cord to synapse directly onto alpha motor neurons which connect to the muscles [9]. Pre-motor areas are involved in planning actions (in concert with the basal ganglia) and refining movements based upon sensory input (this requires the connection with the cerebellum). The premotor area has a specific role in sensorially guided movements. Units of the premotor area are activated in the response to visual, auditory, and somatosensory stimuli. Damage to the premotor area leads to the release of certain reflexes. For example, the grasp response is greatly enhanced (in which grasping movements of the fingers are elicited by tactile stimulation of the palm). The supplementary motor area (SMA, medial area 6) plays an important role in complex motor planning [16]. SMA contains a topographic representation of the body with the head located in the anterior portion of SMA and the legs in the posterior part, adjacent to area 4. Mild motor deficits after a hemorrhagic cortical contusion may be explained by this location, because SMA drives upper motor neurons in the primary motor cortex, but the corticospinal projections form SMA terminate principally on spinal interneurons and not directly on lower motor neurons [1,2,3,16].
CONCLUSION

In our two cases, hemorrhagic cortical contusions in primary motor and supplementary motor area were the causes of foot drop. By this point of view, foot drop may also occur with brain lesion, so central causes should be in the differential diagnosis list. Clinicians, if they detect upper motor neuron findings such as positive Babinski’s sign, hyper reflexia or clonus in a patient with a foot drop, should take into account the possibility of central of lesion as the reason for the foot drop.

REFERENCES