Central Causes of Foot Drop: Rare and Underappreciated Differential Diagnoses

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Abstract

Background/Objective: Peripheral causes of foot drop are well recognized. However, causes stemming from the central nervous system represent rare, important, and underappreciated differential etiologies.

Methods: Two cases of foot drop stemming from central causes are described.

Patients: The first patient, a 46-year-old man with a remote history of lumbar spine fracture and L4-L5 instrumentation/fusion, presented with progressive weakness and numbness of the left foot, followed within 3 months by similar symptoms in the right foot. Lumbar spine imaging failed to reveal compressive nerve root pathology. Electromyography, nerve conduction studies, and muscle and nerve biopsy suggested a preganglionic lesion and ruled out a peripheral cause. Upper spine magnetic resonance imaging (MRI) revealed significant spinal stenosis at C4-C7 and T11-T12. Patient 2 was a 66-year-old man with a known left parasagittal convex meningioma diagnosed 2 years prior presented with a progressive right foot drop over 2 months. Spine imaging was normal, and serial brain MRI confirmed a slowly enlarging parasagittal meningioma.

Results: Following decompressive laminectomies at C4-C7 and T11-T12, patient 1's gait improved, with marked resolution of his right foot drop and significant improvement on the left. Patient 2 underwent craniotomy for microsurgical tumor resection. At the 2-week follow-up examination, he was taking daily walks.

Conclusions: Central causes, although rare, need to be considered in the differential diagnosis of foot drop. Central causative lesions usually occur at locations where pyramidal tract connections are condensed and specific and the function is somatotopically organized. These cases confirm that good results can be achieved when correctable central causes of foot drop are recognized.

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INTRODUCTION

Foot drop is defined as a weak anterior tibialis muscle (on foot extension) and is usually caused by lower motor neuron (LMN) disease. Common causes are L4-L5 radiculopathy, caused by either a herniated nucleus pulposus or foraminal stenosis, and peroneal peripheral neuropathy. Other causes include any axonal or demyelinating damage along the whole peripheral nervous system: conus medullaris, cauda equina, nerve plexus,

and peripheral mixed nerve. Central nervous system pathology can also cause foot drop. Foot drop has been reported to occur in 52% to 67% of patients with spinal upper motor neuron (UMN) pathology (1,2). Central causes tend to occur where nerve fibers are highly condensed along the UMN tracts: interhemispheric motor cortex homunculus (mass lesion, anterior cerebral artery stroke), corona radiata, internal capsule (lacune), cerebral peduncle (lacune), medulla, and spinal cord pyramidal tract (myelopathy). We present 2 illustrative patients with foot drop and negative lumbar spine imaging.

CASE REPORTS/FINDINGS

Patient 1

A 46-year-old white man presented with progressive symptoms of bilateral foot drop and numbness. He had

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undergone previous L4-L5 instrumented posterolateral fusion following a lumbar spine fracture 20 years prior. The patient did well for approximately 18 years; he then started to notice weakness on dorsiflexion in the left foot, followed by left foot numbness. Five months later, he developed right dorsiflexion weakness and numbness. Sensory examination revealed significant reduction of light touch and pinprick in the right L5 distribution and in the left L5 and S1 distributions. Motor examination revealed 3/5 strength in the right tibialis anterior and extensor hallucis longus and 0/5 in the left tibialis anterior and left extensor hallucis longus. Deep tendon reflexes of both lower extremities were 2+ in the right patella and bilateral Achilles and 3+ in the left patella. Plantar reflexes were downward bilaterally.

A left peroneal motor nerve conduction study revealed absent conduction response. Electromyography done 4 months prior to the patient's initial visit demonstrated left peroneal motor neuropathy and incomplete right peroneal neuropathy. There were 3+ fibrillations and positive sharp waves recorded in the left tibialis anterior, left peroneus longus, and right tibialis anterior. To rule out mononeuritis multiplex, a muscle and nerve biopsy was done. A biopsy of the left peroneus muscle showed severely atrophied muscle fibers consistent with neurogenic atrophy, whereas the left sural nerve biopsy was normal. These results were consistent with a preganglionic lesion.

A computerized tomographic myelogram revealed arachnoiditis and mild spinal stenosis at L2-L3 and L3-L4, which were thought to be inconsistent with the degree of clinical presentation. Magnetic resonance imaging (MRI) of his cervical and thoracic spine revealed severe spinal stenosis at C4-C5 (Figures 1C and D), moderate stenosis at C5-C6, and mild stenosis at C6-C7. In addition, there was severe stenosis in the thoracic spine at T11-T12 (Figures 1A and B). Bilateral laminectomies of C4-C7 with left C4-C5 foraminotomy and simultaneous laminectomy of T11-T12 were performed. Six months after surgery, his right tibialis anterior had improved from 3/5 to 4/5. The left side appeared not to improve, with strength remaining at 1/5.

Patient 2

A 69-year-old right-handed Chinese American man, with a known left parasagittal convex meningioma found to be slowly enlarging with serial MRIs, presented in the months prior to surgery with progressively worsening right foot drop. The patient's initial MRI 20 months prior to surgery demonstrated the tumor overlying his motor strip. Follow-up MRI 3 months later indicated stable tumor size. One surgeon suggested expectant observation, and another recommended surgical resection. More than 20 years prior, the patient had suffered a left internal capsule lacunar infarction with residual right-sided arm and leg weakness, which disappeared except for some residual weakness in the right hand. Six months

preceding the surgery, the patient started to notice right lower extremity weakness. He also felt intermittent cold sensation in his left arm, yet denied any recent illness or flulike syndrome.

On physical examination prior to surgery, he had a tendency to drag his right foot on gait with a foot drop on that side. Sensation (all dermatomes) and joint position sense were equal bilaterally. Motor examination revealed 5/5 in proximal muscle groups bilaterally, distal upper extremity 4+/5 (wrist extensors and hand grip), and lower extremity weakness, predominantly in the right tibialis anterior and extensor hallucis longus. He had hyperreflexia on the right compared to the left, respectively: 3+ biceps vs 2+, 3+ brachioradialis vs 2+, and 3+ Achilles vs 2+. There were bilateral flexor and plantar responses.

Preoperatively, his MRIs and magnetic resonance venography suggested a stenotic yet patent sagittal sinus with possible wall involvement on the left side by the tumor (Figure 2A and B). A judicious microsurgical resection was carried out, with possible second-stage gamma knife radiosurgery for any residual attachment along the sagittal sinus. Somatosensory-evoked potential cortical mapping of the motor strip was indicated because of the lesion's epicenter overlying the medial homunculus primary motor strip. At 6-week follow-up, the patient had motor strength 5/5 in the lower extremities and 4+/5 right upper extremity. He had improved significantly, to the point that he was able to take daily walks.

DISCUSSION

Foot drop is defined as weakness of the anterior tibialis and is frequently accompanied by weakness of the extensor hallucis longus and extensor digitorum longus (3). It is usually caused by LMN pathology, commonly disruption of conduction from the deep peroneal nerve (L4-L5). L4-L5 radiculopathy is the most common recognized cause of foot drop, usually caused by herniated nucleus pulposus or foraminal stenosis (4). Peripheral peroneal neuropathy is the next most common and is brought on by neurogenic and nonneurogenic causes such as nerve entrapment, diabetes mellitus, inflammatory neuropathy, trauma, masses or pressure near the fibular head, intraneural tumors, and vascular pathology (3). Central or UMN causes are extremely rare but must be considered.

The UMN of the leg are organized somatotopically from the medial motor cortex down the internal capsule to the ventral gray matter of the spinal cord, where they are grouped in longitudinally oriented columns (5). Compression of these fibers along the path from the cortex, the (posterior part of posterior limb) internal capsule (lacune), the cerebral peduncles, and the ventral spinal cord (6), can potentially result in disruption of signal to the lumbar nerve roots. It is estimated that 20% of stroke survivors (7) have a spastic drop foot (7,8).

Lacunar (internal capsule, cerebral peduncle) infarctions are perhaps the most common causes of stroke in patients with a focal deficit or partial recovery. Other common locations include the interhemispheric motor cortex homunculus (mass lesion, anterior cerebral artery stroke), and the spinal cord pyramidal tract (myelopathy).

Primary brain tumors, which are commonly of parasagittal location, that lead to foot drop include high-grade osteogenic sarcoma (9), oligodendroglioma (10), falx and parasagittal masses (11), meningiomas (12), and metastatic tumors (13). Baysefer et al (11) reported 3 cases of brain tumors with parasagittal pathology in which foot drop was the initial complaint. Eskandary et al (14) reported good neurologic recovery following surgery in 5 cases of foot drop from parasagittal pathology, including a meningioma, a brain abscess, a grade 2 astrocytoma, periventricular demyelinating plaques, and a depressed parietal fracture.

Van der Salm et al (1) identified impaired foot contact in 52% of 21 patients with traumatic incomplete spinal cord injury. Tokuhashi et al (2) found that 2 of 3 subjects with T12-L1 disk herniation had symptoms that included foot drop. Cervical spondylosis, although rare, can lead to foot drop. Engsberg et al (15) described the case of a 65-year-old woman who, upon C2-C3 anterior discectomy and fusion, experienced expedient improvement. Spinal tumors, both primary (16) and metastatic (17), can present with foot drop. In one series, 78% of patients with spinal-dural arteriovenous fistulas presented with leg weakness at the time of diagnosis (18).

Ozdemir et al (19) ascribed the misdiagnosis of central causes to inefficiently performed physical examinations and added that, if UMN findings are detected on physical examination, such as positive Babinski, hyperreflexia, or clonus, the clinician should undoubtedly consider central causes.

Dissociated symptoms may indicate central pathology in the spinal cord or brain UMN as opposed to peripheral nerve or nerve root pathology. Patient 1 presented with bilateral foot drop; although it was significantly greater on the right, bilateral foot drop is

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Figure 1. Patient 1. (A) Axial T2 MRI of the thoracic spine demonstrating flattening of the cord at the intervertebral level of T11-T12.

(B) Sagittal T2 image of the thoracic spine demonstrating severe stenosis at vertebral levels T11-T12.

(C) Axial T2 MRI of the cervical spine, demonstrating flattening of the cord at the intervertebral level of C5-C6.

(D) Sagittal T2 image of the cervical spine demonstrating severe stenosis at vertebral levels C4-C5, with signal changes in the cord at that level, moderate stenosis at C5-C6, and mild stenosis at C6-C7.

Figure 2. Patient 2. (A) Axial post-gadolinium contrast-enhanced MRI of the brain showing a 3 × 3-cm enhancing mass abutting the left cortical motor strip. (B) Coronal T2 fluid-attenuated inversion recovery (FLAIR) MR image demonstrating the same parasagittal mass as in (A) compressing the vertex of the motor cortex and abutting the sagittal sinus.

less likely indicative of peripheral nerve or nerve root pathology. In addition, he had subjective loss of light touch and pinprick on the contralateral (the left) distal lower extremities, a clear indication of spinal cord UMN. Patient 2 complained of right foot drop and abnormal “cold” sensations in his left arm. This could have suggested spinal cord pathology, were it not for the known parasagittal meningioma. Both patients had hyperreflexia on examination (patient 1: patellar; patient 2: Achilles), and neither had a Babinski sign. These presentations emphasize the importance of a careful neurologic examination. If bilateral foot drop or dissociated findings are present, with or without hyperreflexia, Babinski, or clonus, electromyography should be done to rule out peripheral causes, followed by MRIs of the spine and brain. Perhaps a T1 (with and without gadolinium contrast) and T2 of the spine should be performed first, and if negative, followed by T1 (with and without gadolinium contrast), T2, fluid-attenuated inversion recovery, and diffusion-weighted images of the brain to rule out tumors and/or stroke. Subsequent management is pathology dependent.

CONCLUSION

Although rare and underappreciated, central causative lesions need to be considered in the differential diagnosis of foot drop. A thorough history and physical examination may reveal signs of UMN disease, including dissociated symptoms, hyperreflexia, and Babinski response (14). Electromyography and nerve conduction studies should be included in the workup and can assist in ruling out peripheral causes. Total central nervous system imaging, including MRI of the complete spine and head, may reveal pathology, with resultant peripheral neuropathy, that may otherwise be missed otherwise. Most central cases of foot drop can result in rather dramatic recovery following resection or decompression (10,14) as seen in these individuals with parasagittal meningioma and cervical spinal stenosis. Central causes of foot drop must be included in the workup once peripheral and radiculopathic causes have been ruled out.

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