

## Case Report

### Painless Foot Drop: An Atypical Etiology of a Common Presentation

Russel V. Gilchrist, DO\*, Sarjoo M. Bhagia, MD\*, David A. Lenrow, MD#, Larry H. Chou, MD#, David Chow, MD\* and Curtis W. Slipman, MD##

Weakness of the dorsiflexor muscles of the foot is a relatively common presentation. In most cases, the etiology involves a peripheral injury to the common peroneal nerve. These patients usually present with lower motor neuron findings on evaluation. In contrast, if upper motor neuron findings were present a central lesion should be suspected and appropriate imaging studies are performed.

We describe a patient with painless foot drop and lower

motor findings on examination that was diagnosed with multiple sclerosis. This case demonstrates that multiple sclerosis can masquerade as a peripheral process in some patients.

**Keywords:** Foot drop, peripheral nerve injury, lower motor neuron lesion, upper motor neuron lesion, multiple sclerosis

Foot drop can develop from weakness of the dorsiflexor muscles of the ankle. This weakness is caused by an upper or lower motor neuron injury. Most commonly a lower motor lesion is responsible for these symptoms; typically a common peroneal mononeuropathy. Compression of the common peroneal nerve as it courses around the fibular head is the most common site of a peroneal nerve injury. Other lower motor neuron causes of foot drop include sciatic mononeuropathy, lumbosacral plexopathy, polyneuropathy, or a severe L5 radiculopathy (1).

Less commonly, an upper motor neuron lesion can present

as weakness localized to the ankle dorsiflexors. History and physical examination findings sometimes allow for an increased suspicion and prompt diagnosis of a central lesion. Anecdotal case reports have noted lesions located in the parasagittal region of the brain to cause foot drop (2). Somatotopic ankle and toe motor function have previously been established in these parasagittal regions by electro-stimulating localization techniques (3, 4). We present a case report of a painless foot drop in a patient with an inflammatory upper motor lesion.

#### CASE DESCRIPTION

From Penn Spine Research Group, University of Pennsylvania Health System, Department of Rehabilitation Medicine, Philadelphia, Pennsylvania. \*Dr. Gilchrist and Dr. Bhagia are fellows at the Penn Spine Center, #Dr. Lenrow and Dr. Chou are assistant professors of the Department of Rehabilitation Medicine, ♦Dr. Chow is the Director of Interventional Spine in the Department of Physical Medicine and Rehabilitation at the University of Cincinnati and #Dr. Slipman is the director of the Penn Spine Center, associate professor of the Department of Rehabilitation Medicine and Chief of the Clinical Musculoskeletal Program. Address correspondence: Curtis W. Slipman, MD, 3400 Spruce Street, Philadelphia, PA 19104. E-mail: slipman@mail.med.upenn.edu

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A 43-year-old white female presented to our office with a one-week history of a right foot drop. The patient denied any recent trauma to the right lower limb. Pain had been experienced in the right inguinal and groin region during the preceding two weeks with concomitant numbness in the left foot and calf. Review of systems was negative for any visual changes, bowel or bladder dysfunction, fever, chills, headache, dizziness, lightheadedness, or paresthesias. The past medical history consisted Scheuermann's disease, traumatic right tibia/fibula fracture at age sixteen, traumatic right meniscus tear at age twenty-five, and irritable bowel syndrome. She denied any past history of diabetes, hypothyroidism, arteritis, cancer, or hepatitis. The patient denied any medication use or allergies. The family history was positive for multiple sclerosis in the mother and maternal uncle. There was no history of alcohol or drug abuse. The patient admitted to smoking cigarettes in the past, but had quit approximately fifteen years ago.

General inspection revealed a well-developed, well-nourished female in no apparent distress. Girth was symmetric bilaterally in the extremities. Distal pulses were intact. Capillary refill was normal. Passive range of motion was normal throughout all peripheral joints. No increased tone was noted in the extremities. Sitting, straight leg raising, and reverse straight raising tests were negative. Sensation to light touch and pinprick were decreased in the bilateral forefoot plantar surface and posterolateral left calf. Deep tendon reflexes were normal except for a hypoactive response at the right Achilles. Clonus and Hoffman's signs were absent. Babinski test was downgoing bilaterally. Muscle strength testing was 0/5 in the right tibialis anterior and extensor digitorum. There was trace motion noted in the right extensor hallucis longus. Right hamstring and iliopsoas muscle strength-measured 4+/5, gluteus medius were 4/5 bilaterally, and right quadriceps were 4+/5. Cranial nerves two through twelve were intact.

The initial differential diagnosis was multilevel radiculopathy involving the right L4 and L5 nerve roots, common peroneal neuropathy, retroperitoneal process causing a lumbar plexopathy, and central nervous system lesion. An electromyogram/nerve conduction study (EMG/NCS) was performed and magnetic resonance imaging (MRI) of the lumbar spine was ordered. EMG/NCS demonstrated complete absence of active recruitment in the right tibialis anterior and peroneus longus muscles. Needle sampling of the right gastrocnemius, vastus medialis, tensor fascia lata, gluteus maximus, and lumbar paraspinal muscles were normal. No acute denervation potentials were present in all muscles tested. No significant decrease in conduction velocity or amplitude was seen in the right common peroneal nerve. An MRI of the lumbar spine revealed a mild developmental stenosis of the central canal at all lumbar levels. A right para-median focal protrusion at L5/S1 disc level and mild right foraminal stenosis at L5/S1 level were noted. The focal protrusion was mild and did not appear to be abutting the right L5 nerve root. Since the MRI of the lumbar spine and EMG/NCS were inconclusive an MRI of the pelvis was performed to exclude lumbar plexopathy as a possible diagnosis. No abnormalities were seen on MRI of the pelvis. An MRI of the brain was then performed to identify any central lesion as a cause for the patient's symptoms. Brain MRI identified multiple punctate foci of increased signal in the periventricular area.

A tentative diagnosis of multiple sclerosis (MS) was made and the patient was referred to neurology for completion

of the work-up. The patient was fitted with an ankle foot orthosis (AFO) to correct their steppage gait pattern and prevent ankle injury. At one month follow-up the patient had mild improvement in their right ankle strength. They continued to use the AFO for community ambulation, but did not wear it for household ambulation. Neurology confirmed the diagnosis by performing further work-up including lab work and MRI of the cervical and thoracic spine. All studies were reported to be normal by the patient.

## DISCUSSION

Multiple sclerosis is one of the most common neurologic diseases in the United States, affecting approximately 500,000 people at any one time (5). The average age of onset occurs between the ages of 20 and 40 years old (6). The etiology remains unknown, however, current evidence supports an autoimmune etiology (7). Pathologically, multiple sclerosis results in inflammatory focal demyelinating lesions occurring in both white and gray matter of the central nervous system (8). Central nervous system plaques form in place of the destroyed myelin. These plaques are found in perivenous areas and periventricular white matter of the cerebrum, brainstem, and spinal cord (9). The axons of the central nervous system are relatively preserved in this disease (10).

Magnetic Resonance Imaging (MRI) is the preferred imaging tool to aide in the diagnosis of multiple sclerosis. The identification of three or more lesions greater than 3mm in diameter is considered highly suspicious for multiple sclerosis (11). Typical areas of abnormalities occur in the periventricular regions of the supratentorial white matter. Less commonly, abnormalities may be found in the brainstem, cerebellum, and cervical spine (12), presence of lesions by MRI has not shown correlation with clinical findings, disability, or response to steroid therapy (13). Evoked potentials may aide in the diagnosis of multiple sclerosis via their ability to document slowing in conduction velocity of central nervous system myelinated pathways. Visual evoked potentials are abnormal in 75% of patients with definite multiple sclerosis, and in 15-60% of patients with possible multiple sclerosis. Electromyographic and Nerve Conduction studies offer little information toward the direct diagnosis of multiple sclerosis (14). However, it is useful to rule in or out a peripheral nerve etiology. This was the case in our patient. Weakness is the most common symptom at diagnosis of multiple sclerosis (15). This weakness generally occurs in the bilateral lower extremities, one lower extremity, or

one leg and ipsilateral arm. Rarely does weakness solely occur in one or both upper extremities (16). The weakness tends to involve the entire extremity and usually does not present in a myotomal or peripheral nerve distribution. The presence of upper motor findings on physical exam should cause increased suspicion of a central nervous system lesion. In contrast, our patient's initial presentation was weakness only in the ankle dorsiflexors with complete absence of upper motor findings on exam. This localized weakness led to our provisional diagnosis of peripheral nerve versus root lesion as the site of injury. The absence of findings on electromyography/nerve conduction testing or abnormalities on the pelvic MRI led us to seek a central nervous system lesion. Pain may occur in up to 50% of patients with multiple sclerosis (17, 18). This pain commonly manifests in the extremities or low back. It presents as a dysesthetic pain that becomes chronic in 90% of patients (18). Our patient denied any pain complaints on presentation to our office. Based upon this data, the presentation of painless weakness in our patient is atypical of multiple sclerosis. The absence of pain in our patient may only be temporally related, however, at follow-up visit, the patient continued to deny the presence of pain in the back or right lower limb.

In conclusion, the presence of painless footdrop as a presenting symptom should alert the clinician as multiple sclerosis as a potential cause. The positive evidence of upper motor neuron findings on physical examination should increase suspicion of a central lesion, and an immediate MRI of the brain would be justified. In the absence of upper motor neuron findings on examination one should first suspect peripheral nerve injury. A normal electromyogram/nerve conduction study would require investigation via MRI of the brain and spinal cord for a central cause. This case further demonstrates that multiple sclerosis might present in a manner that mimics a peripheral nerve or root injury, and that it should always be included in the differential diagnosis of a patient with painless weakness in a lower limb.

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