



EMG Case No. 53, January 2002

Presenting Symptom: Left Foot Drop

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Appropriate Audience: Residents and practicing physicians

Learning Objectives: After completing this educational activity, participants will be able to (1) formulate the differential diagnosis of a foot drop, (2) utilize the physical exam to help narrow down the differential diagnosis of foot drop, and (3) review the principles of electrodiagnosis that can establish diagnosis of foot drop.

This case is no longer available for CME credit.

History

A 39 year-old male karate instructor with no significant past medical history complained of sub-acute onset of pain at the left lateral knee starting approximately 1 year ago. This pain was intermittent but was worse with activity, especially walking long distances. He recalls no specific trauma or injury. There also was no weakness or sensory symptoms until approximately 2 months ago. At that time he noticed severe exacerbation of his knee pain, which then radiated down his left leg. This more severe pain has persisted for several weeks, and he subsequently developed weakness of left ankle dorsiflexion. He then noticed atrophy of his left lower leg along with persistent ankle weakness. He has had some sensory loss of his left foot, mostly over the dorsum, but it has never gone completely numb. He denies any other weakness, pain, or sensory symptoms.

- *Prior to continuing, please develop a differential diagnosis and list each possible diagnosis in order of likelihood.*
- *Is there any additional information regarding the clinical history that might be helpful in clarifying your differential list or changing its order of priority?*

Commentary I

The patient presents with an acute exacerbation of left leg pain and subsequent foot drop. Differential diagnosis is:

1. Peroneal mononeuropathy at the fibular head
2. L5 radiculopathy
3. Deep peroneal mononeuropathy
4. Sciatic mononeuropathy
5. Lumbar plexopathy
6. Early motor neuron disease
7. Central nervous system process

Peroneal mononeuropathy at the fibular head and L5 radiculopathy are very common disorders that may present with foot drop and are therefore high on our differential diagnosis list. A deep peroneal mononeuropathy, as may occur in a compartment syndrome, is also quite possible, given his pain with activity and other symptoms. A left sciatic mononeuropathy or lumbar plexopathy are less likely as there is no injury to support these.

Motor neuron disease usually does not present with pain. The focal nature of the problem and lack of generalized neurological symptoms make a central process unlikely as well.

History, continued

Further questioning shows that he has no back or proximal left leg pain. The left leg does not currently hurt. He also denies any change in his bowel or bladder function. He has been evaluated by his primary care physician and was fit with an AFO to aid with his gait and a lumbar MRI was done. Medical history is otherwise unremarkable and preliminary laboratory work was within normal limits.

- *If necessary, revise your differential diagnosis based on the additional clinical history.*
- *On which details of the physical examination should you focus at this point?*

Commentary II

The absence of any lower back pain throughout the entire course makes a radiculopathy less likely but does not eliminate this diagnosis. The most likely etiologies of the foot drop are still a peroneal mononeuropathy at the fibular head and a deep peroneal mononeuropathy. Physical examination will help to better define the clinical cause of the foot drop.

Certain aspects of the physical examination will yield most useful information. Motor examination of the lower limb should be comprehensive and attempt to isolate all nerve roots in multiple peripheral nerve distributions, especially the L5 nerve root distribution in this case. For example, hip abduction will assess the gluteus medius (superior gluteal nerve), ankle dorsiflexion will assess the anterior tibialis (deep peroneal nerve), ankle eversion will assess the peroneus muscles (superficial peroneal nerve), and ankle inversion will assess the posterior tibialis (tibial nerve). It is important to note that ankle inversion/eversion strength is best measured with the foot placed passively in dorsiflexion/plantarflexion neutral. One may thus be able to localize the lesion to a specific root level or peripheral nerve distribution.

Knowing the dermatomal and peripheral sensory nerve distributions may also be useful to further localize the lesion. For example, the deep peroneal nerve has a very limited sensory distribution in the dorsal first web space.

Side to side comparison of the reflexes at the patella (L4), medial hamstring (L5), and Achilles (S1) may provide other clinical clues to narrow the differential diagnosis.

Physical Examination

Prominent atrophy was noted in the anterior compartment of the left lower extremity. Motor strength was normal in the lower limbs with the exception of absent ankle dorsiflexion and great toe extension on the left. Voluntary inversion and eversion was present but slightly diminished compared to the right side. Reflexes were 2+ and symmetric at the patella, medial hamstring, and Achilles. Although slightly diminished, there is intact pinprick sensation over the dorsum of the left foot. Straight leg raise is negative bilaterally for any radiating symptoms.

Lumbar spine MRI shows normal disc height without any disc herniation or nerve root impingement.



Commentary III

Physical examination shows deficits that appear to be isolated below the left knee. Intact reflexes, normal proximal L5 muscle strength, and a negative MRI put radiculopathy much lower on our list. The main deficits involve the anterior tibialis and great toe extensors. There is some weakness noted in left ankle inversion and eversion, however these are relatively intact compared to ankle dorsiflexion and great toe extension and may be related to prolonged immobility. Thus a deep peroneal mononeuropathy appears to be most likely.

Electrophysiologic Data

MOTOR NERVE CONDUCTION STUDIES							
NERVE	SIDE	STIM SITE	RECORD	Cm	AMPL	LAT (Peak)	CV
Peroneal	L	Ankle	EDB	9.0	NR	-	-
Peroneal	L	Below Knee	Ant Tib	6.0	0.6	1.7	-
Peroneal	L	Above Knee	Ant Tib	10.0	0.4	4.2	40.0
Tibial	L	Ankle	Abd Hal	8.0	10.7	3.9	-
Peroneal	R	Ankle	EDB	9.0	10.7	3.8	-
Tibial F-Response	L	Ankle	Abd Hal	-	-	49.0	-

SENSORY NERVE CONDUCTION STUDIES							
NERVE	SIDE	STIM SITE	RECORD	Cm	AMPL	LAT	CV
Sural	L	Calf	Ankle	14	27.0	3.4	51.9
Superficial Peroneal	L	Ankle	Foot	12	15.2	3.0	50.2



<p align="center">NEEDLE ELECTROMYOGRAPHY Insertional activity: N, sust, unsust FIB: 0, 1+, 2+, 3+, 4+ OTHER: 0 or fascic, myotonia, myokymia EFFort: N, decr RECRuitment: N, inc or dec 1+, 2+, 3+, 4+ AMPLitude: N, inc or dec 1+, 2+, 3+, 4+ DURation: N, inc or dec 1+, 2+, 3+, 4+ POLyphasia: N, inc or dec 1+, 2+, 3+, 4+</p>									
R/L	MUSCLE	INSER	FIB	OTH	EFF	REC	AMP	DUR	POL
L	Vastus Medialis	N	0	0	N	N	N	N	N
L	Medial Gastrocnemius	N	0	0	N	N	N	N	N
L	Anterior Tibialis	Sust	3+	Crd	N	0	-	-	-
L	Extensor Hall Longus	Sust	3+	Crd	N	0	-	-	-
L	Peroneus Longus	N	0	0	N	N	N	N	N
L	Gluteus Medius	N	0	0	N	N	N	N	N
L	Biceps Short Head	N	0	0	N	N	N	N	N
L	Low Lumbar Paras	N	0	0	-	-	-	-	-

Nerve conduction studies show an absent or severely diminished left peroneal compound muscle action potential amplitudes. The remainder of the nerve conduction studies were within normal limits. Needle electromyography shows abnormal insertional activity and no observable motor units in the in the left deep peroneal nerve distribution.

- *On the basis of both the clinical and electrophysiologic evaluations, formulate your diagnostic impression. List the most likely diagnosis first and follow in order with the other possibilities that are not excluded by the data. Eliminate those diagnoses not supported by the data.*
- *Are there additional electrophysiologic data that you feel would further delineate the diagnosis? (Remember, collecting data that are not needed for the diagnosis is costly and uncomfortable for the patient.)*

Diagnostic Impression

Based upon the clinical and electrodiagnostic findings the diagnosis of a severe left deep peroneal mononeuropathy was made. There were no active motor units observed to confirm nerve continuity or reinnervation. The normality of the peroneus longus and short head of the biceps muscles as well as the normal superficial peroneal sensory evoked response do not support involvement of the superficial peroneal or common peroneal nerves, and thus neither a more proximal peroneal mononeuropathy. A left lower extremity radiculopathy or plexopathy was also ruled out by the needle examination. With the patient's clinical



presentation, this left deep peroneal mononeuropathy is most likely a result of acute anterior compartment syndrome.

Commentary IV

Compartment syndrome is defined as increased pressure within an osteofascial compartment that compromises circulation to muscles, nerves, and other tissues. Anterior compartment syndrome (ACS) is the most common in the lower limb and will often result in a foot drop. Characteristically, the anterior compartment contains the deep peroneal nerve, so foot inversion (tibial nerve) and eversion (superficial peroneal nerve) will be spared. The cutaneous sensory distribution of the deep peroneal nerve is limited to the first dorsal web space and wide spread sensory deficits do not occur with ACS.

A crush injury, tibial fracture, or any trauma may cause excessive bleeding or swelling within the compartment and cause vascular and neurologic compromise if not treated promptly. The five "P's" - Pain, Paralysis, Pulselessness, Pallor, and Paraesthesias represent the hallmark presentation. A chronic compartment syndrome (CCS) can arise, related to a ramp up in exercise causing a transient increase in intra-compartmental pressure. Symptoms will usually subside with rest, although there may be an intercurrent acute compartment syndrome with permanent deficits, as most likely occurred here.

ACS is a surgical emergency; treatment consists of fasciotomy to reduce the intra-compartmental pressure. Prolonged increased pressure > 30mm Hg can cause permanent neurologic deficits after just eight hours. People with CCS may also choose to have an elective fasciotomy to help with their exercise tolerance or simply stop exercising at the onset of pain. If they opt for the latter, they must seek medical attention at once if the symptoms persist despite rest.

Early diagnosis is crucial. Our patient, for example, presented after the neurologic damage was severe and he will likely never fully recover. Management for him will be supportive with appropriate range of motion exercises and orthotics. Tendon transfers and nerve grafting procedures are available but have limited success.

Bibliography

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