American Academy of Physical Medicine and Rehabilitation

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February 1999 EMG Case-of-the-Month

This case is no longer available for CME credit.

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Presenting Symptoms: Isolated foot drop

Learning Objectives: After completing this educational activity, participants will be able to (1) formulate a comprehensive differential diagnosis for isolated foot drop and (2) identify differences in electrodiagnostic studies in order to establish a diagnosis.

HISTORY

A 66-year-old previously healthy woman presents with a painless, isolated right foot-drop. Three months prior she had a decompressive laminectomy with lateral recess exploration at L4/L5 and L5/S1 for what was thought to be an L5 radiculopathy. Needle electromyography (EMG) at that time demonstrated 1+ fibrillationpotentials in the right tibialis anterior, peroneus longus, and right lumbar paraspinal muscles. There was normal conduction velocity in the right peroneal nerve, but reduced amplitude of the compound muscle action potential, without drop in the amplitudeacross the head of the fibula. The laminectomy provided temporary relief of her symptoms, but the patient began developing further painless weakness in the right lower limb.

- **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 presentation of progressive, isolated foot drop following lumbar spine decompression brings up the differential diagnoses of a cerebral hemispheric lesion, a spinal cord lesion, motor neuron disease (amyotrophic lateral sclerosis), sciatic compression (pelvic mass), or possible re-stenosis in the lumbar spine.

She did not have any bowel or bladder incontinence, cognitive/speech, swallowing, or behavioral problems, which makes a CNS problem or ALS less likely but does not completely exclude them. Absence of pain mitigates sciatic compression and spinal stenosis.

HISTORY, CONTINUED

The patient denies diabetes and other metabolic problems. There is no history of smoking or excessive alcohol use. The family history is negative. Magnetic resonance imaging (MRI) of the lumbar spine shows multi-level degenerative disc disease with significant stenosis of the spinal canal at L3/L4 and L4/L5.

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- **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

At this point, progressive nerve-root compression secondary to spinal deformation seems likely. There is no past history that identifies co-morbid risk factors, MRI findings of spinal stenosis at L4/L5 are compatible, and thesurgical procedure carries a known risk for progressive, postoperative, neurologic deterioration.

PHYSICAL EXAMINATION

On examination of the right lower limb, motor tone is markedly diminished. Muscle strength is graded on the MRC scale as follows: 2/5 strength in ankle dorsi- and plantarflexion and eversion, and 4/5 strength in knee and hip flexion and extension. Muscle stretch reflexes are absent. There is no apparent sensory loss.

In the remaining three limbs, no weakness is detected and muscle stretch reflexes are 2+. Responses to plantar stimulation are normal bilaterally.

- **At this point, review your differential diagnosis and revise as appropriate.**
- **Are there additional observations on physical examination that might be helpful in narrowing your differential list?**

PHYSICAL EXAMINATION, CONTINUED

No additional physical findings are elicited.

COMMENTARY III

The physical examination, now three months post surgery, is consistent with a neuropathic process that is more diffuse than an isolated nerve root, and which appears to be confined to the right lower limb. Because there is no pain or sensory loss clinically, amyotrophic lateral sclerosis (ALS) becomes a significant possibility. There are no physical signs of central nervous system pathology or underlying peripheral polyneuropathy. The differential diagnosis now includes motor neuron disease and lumbar polyradiculopathy.

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ELECTROPHYSIOLOGIC DATA

ELECTROMYOGRAPHY

n = normal incr = increased decr = decreased 0 = absent
1+ = minimal 4+ = maximal
crd = complex repetitive discharge fasc = fasciculation potential
myk = myokymic discharge myt = myotonic discharge
nmt = neuromyotonic discharge
p wave = positive sharp waves fib = fibrillation potentials
recr = recruitment
amp = amplitude dur = duration poly = polyphasic potential

R/L	MUSCLE	INSERTION		SPONTAN		VOLUNTARY				
		activ	p wave	fib	other	recrt	amp	dur	poly	effort
R	lumbar paraspinal	n	0	1+	0	-	-	-	-	-
R	adductor longus	n	0	0	0	n	n	n	n	-
R	vastus lateralis	incr	1+	1+	0	decr	n	n	Incr	-
R	vastus medialis	n	0	0	0	n	n	n	Incr	-
R	rectus femoris	n	0	0	0	n	n	n	n	-
R	gluteus medius	n	0	0	0	n	n	n	n	-
R	biceps femoris, short head	incr	2+	2+	0	decr	n	n	Incr	-
R	tibialis anterior	incr	2+	2+	0	decr	n	n	Incr	-
R	peroneus longus	incr	2+	2+	0	decr	n	n	Incr	-
R	soleus	incr	2+	2+	0	n	n	n	Incr	-
R	gastrocnemius (lateral)	n	0	0	0	n	n	n	n	-
R	gluteus maximus	n	0	0	0	n	n	n	n	-
R	gastrocnemius (medial)	incr	2+	2+	0	decr	n	n	Incr	-
R	first dorsal interosseus	n	0	0	0	n	n	n	n	-
L	lumbar paraspinal	n	0	0	0	n	n	n	n	-
L	lower limb screening EMG	n	0	0	0	n	n	n	n	-

MOTOR NERVE CONDUCTION

nr = no response

NERVE	LATENCY (ms)			AMPLITUDE (MV)			CONDUC VEL(M/S)		
	R	L	Norm	R	L	Norm	R	L	Norm
				(pos. to neg. peak)					

Peroneal	-	-	-	-	-	-	-	-	-
ankle-to-EDB	4.1	4.2	<5	4.9	4.8	4-6	-	-	-
B fib head-to-EDB	12.2	12.5	-	4.3	4.3	-	40	40	>35
Peroneal F-wave	-	-	-	-	-	-	-	-	-
ankle-to-EDB	47.4	48.1	<50	-	-	-	-	-	-
Tibial	-	-	-	-	-	-	-	-	-
ankle-to-med foot	4.0	-	<4.5	1.9	-	>6	-	-	-
pop Fossaa-to-med foot	12.9	-	-	1.4	-	-	39	-	>38
Tibial F-wave	-	-	-	-	-	-	-	-	-
ankle-to-med foot	46.9	-	<50	-	-	-	-	-	-

SENSORY NERVE CONDUCTION									
nr = no response									
NERVE	**LATENCY (ms)**			**AMPLITUDE(mv)**			**CONDUC VEL (m/s)**		
	R	**L**	**Norm**	**R**	**L**	**Norm**	**R**	**L**	**Norm**
				(baseline to peak)					
Sural	-	-	-	-	-	-	-	-	-
calf-to-ankle	3.4	-	<4.0	16	-	>10	-	-	-
Saphenous	-	-	-	-	-	-	-	-	-
knee-to-calf	2.7	-	<2.8	11	-	>7	-	-	-

- **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.)**

ELECTROPHYSIOLOGIC DATA, CONTINUED

No additional electrophysiologic data were collected.

- **Make the final revisions of your diagnostic impression(s).**

DIAGNOSTIC IMPRESSION

The study is consistent with an acute neuropathic process occurring proximal to the level of the intervertebral foramina in the right lumbar spine, affecting the right lower limb with involvement not confined to a single segmental level or peripheral nerve distribution.

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- **What other diagnostic procedures (laboratory tests, etc.), if any, are needed?**
- **What treatment would you recommend?**

COMMENTARY V

Given this atypical presentation, the patient was referred for repeat lumbar and lower limb short TI inversion recovery (STIR) MRI studies.

A 1.0 Tesla magnetom impact scanner was used to obtain T_1-weighted axial and coronal images as well as STIR axial images of both lower limbs. Gadolinium-enhanced images of the lumbar spine were also performed. In the lumbar spine there were post-laminectomy changes at L4/L5 and L5/S1 with no central or foraminal stenosis and no root or conus compression. The cauda equina was normal in appearance. In the lower limbs, no abnormality was noted on the left. On the right, there was a diffuse decrease in muscle bulk and increase in signal intensity throughout the anterior and posterior compartment muscles of the leg on the STIR images, compatible with subacute denervation. The right psoas muscle showed moderate atrophy but the visualized lumbar paraspinal musculature appeared normal and symmetric. The vascular structures were symmetric bilaterally and there were no bony abnormalities or masses.

FOLLOW UP

Initially, electrodiagnostic abnormalities remained confined to the right lower limb. Then, a year after the postoperative evaluation, patchy findings consistent with denervation became apparent in the left lower limb and in both upper limbs. There was no significant reinnervation. Laboratory work-up was within normal limits including serum and urine protein electrophoresis, urine heavy metal screen, and anti-GM_1, anti-Yo (purkinje cell), and anti-Hu (neuronal nuclear) antibody titers. Cerebrospinal fluid showed only mildly elevated protein.

One month later, the patient presented with shortness of breath and midepigastric pain. Ventilation-perfusion scan showed large bilateral pulmonary emboli without evidence of thrombosis in the lower limbs. Computerized axial tomographic (CT) scan of her abdomen revealed a 4x4-cm pancreatic mass with multiple metastatic lesions in the liver. Biopsy revealed moderately differentiated duct cell adenocarcinoma of the pancreas. Bone scan showed no evidence of spinal metastasis. The patient underwent 4 months of chemotherapy with gemicatabine, but continued to lose strength, eventually expiring from pneumonia. At post mortem, the spinal cord appeared normal. There was no evidence of anterior horn cell necrosis or demyelination.

DISCUSSION/CONCLUSION

This patient initially had electrodiagnostic and imaging studies consistent with an acute, right L5 radiculopathy due, presumably, to degenerative disc disease and spinal stenosis, although she had no back or lower limb pain and numbness. She underwent surgical decompression with some improvement, suggesting that root compression contributed to at least part of the initial electrodiagnostic abnormalities. Three months postoperatively, her electrodiagnostic studies were consistent with amotor neuronopathy confined to the right lower limb. It was assumed that she was developing amyotrophic lateral sclerosis (ALS).

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This remained her working diagnosis until she developed pulmonary emboli in the absence of deep venous thrombosis in the lower limbs, a known paraneoplastic effect including pancreatic adenocarcinoma.[8] By the time of discovery the tumor was already quite large and had invaded the liver.

A painless, peripheral motor neuronopathy, usually involving lower limbs first, without bulbar or upper motor neuron involvement, has been reported in Hodgkin's disease and lymphoma, and referred to as a subacute motor neuronopathy (SMN). A sensory neuronopathy has been described with pancreatic adenocarcinoma and a sensory neuronopathy and atypical motor neuron disease (MND) have been reported with small cell carcinoma of the lung.

Paraneoplastic SMN may go into remission and improve with chemotherapy. Our patient's neuropathy progressed along with the malignancy, despite chemotherapy. The normal post mortem appearance of the spinal cord essentially ruled out ALS or neoplastic polyradiculopathy since there was no evidence of anterior horn cell or motor neuron necrosis or leptomeningeal tumor invasion. Normal repetitive stimulation studies ruled out a paraneoplastic neuromuscular junction disorder (Lambert-Eaton).

We conclude that this case represents a paraneoplastic SMN associated with a pancreatic adenocarcinoma, remaining confined to the nerve distribution of the right lower limb for one year. The neuropathy may have been responsible for some or all of the EMG abnormalities that were originally interpreted as a radiculopathy. This reaffirms the need for cautious interpretation of electrodiagnostic and imaging studies that may be consistent with a given diagnosis yet do not fit with the clinical symptoms expected with that diagnosis (e.g., lack of back and lower limb pain and numbness). Further, screening for malignancy should be done in the geriatric population with unexplained or atypical neuropathies.

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