



## September 1997 EMG Case-of-the-Month

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### **HISTORY**

A 37-year-old woman, 29 weeks into her first pregnancy, began to experience constant pain and numbness along the lateral aspect of her left foot three weeks ago. During this period she has also been experiencing intermittent pain in her calf and posterior thigh. She has had chronic low back and left buttock pain since she was involved in a motor vehicle accident seven years ago.

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

### **COMMENTARY I**

The most obvious diagnosis in the differential list at this point is left S1 radiculopathy. The pain and numbness are in the proper distribution, and there is a history that suggests low back injury in the past. Also, S1 radiculopathy is a common problem. Other possible diagnoses include sacral plexus neuropathy (possibly due to compression related to the pregnancy), sciatic neuropathy, focal sural nerve pathology, and tarsal tunnel syndrome. Nothing in the history thus far points to any of these other diagnoses, all of which are less common than radiculopathy.

### **HISTORY, continued**

The patient has been in good health. She has not been experiencing any pain, numbness, or tingling, except as described above. She denies weakness, muscle cramps, and muscle twitching. She has a family history of hypertension, but no neuromuscular disease.

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

### **COMMENTARY II**

The additional details of the history do not change or add to the differential diagnosis. Therefore, the physical examination should focus on the left lower limb with emphasis on the nerve fibers that cross the S1 segmental level. As always, at least a cursory generalized neurologic examination should be performed.



## PHYSICAL EXAMINATION

Straight leg raising on the left at 40° reproduces the pain in the patient's left foot, though the Valsalva maneuver does not. No muscle atrophy is observed in the lower limbs, and no muscle weakness is detected. Quadriceps, medial hamstring, and ankle reflexes are easily elicited and are symmetric. There is a cutaneous sensory deficit along the lateral aspect of the left foot, including the third, fourth, and fifth toes as well as an elongated area extending proximally 15-20cm onto the posterolateral calf. The lateral aspect of the heel is almost anesthetic, while the lateral border of the foot is dysesthetic. There is no detectable abnormality of proprioception.

- **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?**

## COMMENTARY III

The positive straight leg raising maneuver is a very strong sign in support of radiculopathy. The sensory deficit is confined to the distribution of the S1 dermatome. Combining the history and physical findings, it appears most likely that the problem is S1 radiculopathy, although the case would be stronger if weakness limited to the S1 myotome had been detected.

There are, however, two findings in this case that are not typical of radiculopathy. The first is absence of motor involvement. It is quite possible to have compromise of the dorsal root while the ventral root is spared, but it is not typical to have a severe sensory deficit without at least mild motor involvement.

The second and more important atypical finding is a normal ankle reflex on the left in the presence of clear sensory loss. Muscle stretch reflexes are more affected by impairment on the sensory side of the reflex arc than on the motor side. This, presumably, is due to dyssynchrony of afferent impulses reaching the synapses in the spinal cord. In this case, with a severe sensory deficit, one would expect a diminished or absent reflex. If the problem is S1 radiculopathy, consideration should be given to what sort of lesion might permit a bolus of afferent impulses to cross the dorsal root and arrive at the spinal cord in a synchronous manner. Such a lesion would have to damage some afferent fibers severely while sparing totally a substantial number of others. (The findings can be explained by damage resulting either in demyelination or axonal degeneration.) This is not likely to happen as a result of a compressive lesion.

The data collected so far do not suggest additional involvement in other areas, but a general screening neurologic examination is needed.

## PHYSICAL EXAMINATION, continued

Gait and spinal alignment appear normal. Cranial nerves are intact. In the upper limbs, muscle bulk and tone are normal, and there is no weakness. Muscle stretch reflexes are present and symmetric bilaterally. No sensory abnormality is noted.

- **If necessary, revise your differential diagnosis based on the additional physical findings.**



- Design your approach to the electrophysiologic examination based on the existing data.

**COMMENTARY IV**

The problem continues to present as an S1 radiculopathy. The electrodiagnostic examination should focus on the left lower limb. A needle electrode examination should be done to look for subclinical motor involvement. Sensory nerve conduction studies in the distribution of the sensory deficit should assess possible loss of sensory axons. H-reflex studies should be done to supplement the observation of normal ankle reflexes.

**ELECTROPHYSIOLOGIC DATA**

<b>ELECTROMYOGRAPHY</b>										
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 potentials										
R/L	MUSCLE	INSERTION		SPONTAN		VOLUNTARY				
		activ	p wave	fib	other	recrt	amp	dur	poly	effort
L	parasp - lumboscral	N	1+	1+	0	N	N	N	N	-
L	vastus lateralis	N	0	0	0	N	N	N	N	-
L	anterior tibialis	N	0	0	0	N	N	N	N	-
L	internal hamstrings	N	0	0	0	N	N	N	N	-
L	gluteus medius	N	0	0	0	N	N	N	N	-
L	peroneus longus	N	0	0	0	N	N	N	N	-
L	biceps femoris	N	0	0	0	N	N	N	N	-
L	medial gastroc	N	0	0	0	N	N	N	N	-
R	parasp - lumboscral	N	0	0	0	N	N	N	N	-
R	anterior tibialis	N	0	0	0	N	N	N	N	-
R	peroneus longus	N	0	0	0	N	N	N	N	-
R	biceps femoris	N	0	0	0	N	N	N	N	-
R	medial gastroc	N	0	0	0	N	N	N	N	-



SENSORY NERVE CONDUCTION									
nr = no response									
NERVE	LATENCY (ms)			AMPLITUDE (µV)			CONDUCE VEL (m/s)		
	R	L	Norm	R	L	Norm	R	L	Norm
sural	-	3.2	<3.8	-	22	-	-	44	>35
lat. plantar	-	2.4	<3.0	-	14	-	-	41	>32

H-REFLEX					
NERVE	STIMULATE	RECORD	LATENCY		
			R	L	Norm
tibial	popliteal fossa	triceps sural	27.2	27.0	<31.5

The muscles studied on needle electrode examination encompass all the segmental levels of the lower limb with special attention to the S1 level. The only abnormalities detected were positive waves and fibrillation potentials in the left lumbosacral paraspinal muscles.

Sensory nerve conduction studies were performed in the distribution of the patient's sensory deficit. All values were within normal limits.

H-reflex studies across the S1 segmental level were within normal limits.

- **On the basis of both the clinical and electrophysiologic evaluations, formulate your final 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.**
- **What other diagnostic procedures are needed?**

**DIAGNOSTIC IMPRESSION**

There is an acute left S1 radiculopathy involving (a) the dorsal root proximal to the dorsal root ganglion and (b) the posterior primary ramus. Since the findings, both clinical and electrophysiologic, are not characteristic of most radiculopathies, a cause other than osteoarthritis or herniated nucleus pulposus should be considered.

**COMMENTARY**

The electrophysiologic data support the diagnosis of radiculopathy already predicted by the clinical examination. It is the clinical examination that defines the level at S1.



Abnormal findings on the needle examination are limited to the paraspinal muscles. This places the lesion at or proximal to the posterior primary ramus and provides the only indication of motor nerve fiber involvement.

Normal amplitudes for the sensory nerve action potentials make it most likely that the clinical sensory loss is due to a lesion of the ventral root proximal to the dorsal root ganglion. Normal amplitudes can be seen with demyelination (and axonal sparing) as distal as the site of stimulation, but the needle examination has already placed the lesion in a very proximal location.

The normal H-reflex reveals that afferent impulses are traversing the ventral root in a normal or near-normal fashion and in sufficient quantity to produce a motor response of normal proportions.

At this point, the location is rather clear. The pathologic nature of the lesion is uncertain, but, to speculate, one explanation of the clinical and electrophysiologic data might be focal necrosis of a few nerve fascicles, such as might happen with disruption of the vasa nervorum. Focal demyelination might also explain the abnormal findings.

## **FOLLOW-UP REPORT**

Imaging studies of the lumbosacral spine were normal. The patient received medication for relief of pain and was treated with physical therapy. Improvement was mild and gradual. Four months later, a few weeks after a normal delivery and postpartum recovery, she developed numbness over the dorsum of the right foot and a sensation of tightness in both legs up to the knees. She denied symptoms in the upper limbs or trunk.

On examination, the straight-leg-raising sign noted previously had resolved. No atrophy or muscle weakness were apparent. The left medial hamstring reflex was less active than the right, and right ankle reflex was less active than the left. The cutaneous sensory deficit noted previously on the left foot was still present, but there was less dysesthesia. Sensory loss over the dorsum of the right foot was confirmed, and diminished proprioception distally in both lower limbs was detected.

A needle electrode examination of both lower limbs was within normal limits. No sensory nerve action potentials could be recorded from either the right or left superficial peroneal nerve or from the right sural nerve. The amplitude of the left sural nerve was abnormally small. The left H-reflex across the S1 segmental level was abnormally slow as compared to the right side. Motor conduction studies were within normal limits.

At this point, multifocal axonal mononeuropathy was apparent, and a tentative diagnosis of nonsystemic vasculitic neuropathy was made. This diagnosis was supported by subsequent blood studies. Because the course of the disease was mild, it was elected to avoid an aggressive treatment program, specifically immunosuppressive therapy. The patient's condition stabilized, but a year later she developed new symptoms in all four limbs. Electrodiagnostic reevaluation showed mild, multifocal loss of sensory axons, this time involving the upper limbs as well as the lower. Motor abnormalities were limited to abnormal chronodispersion found both in the left tibial compound muscle action potential and the right peroneal F-wave distribution.

Beyond the technical analysis of this case, there are broader concepts to remember. First, it should always be kept in mind that radiculopathy is not always due to a herniated disc or an



osteophyte. Next, when clinical or electrophysiologic findings deviate from the typical or expected, one should be prompted to consider a wider range of possibilities.

## **BIBLIOGRAPHY**

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