



September 1998 EMG Case-of-the-Month

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HISTORY

A 16-year-old female track athlete noticed the sudden onset of right anterolateral thigh pain while cooling down after a work out. Her trainers thought it was a cramp and started an aggressive stretching program, along with local heat. She continued to compete but was plagued with persistent anterolateral thigh pain after strenuous running. After two months of continued post-exercise pain without relief from stretching and local heat she sought the advice of an orthopedist, who documented right thigh atrophy and a diminished right quadriceps reflex.

- **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 significant thigh atrophy and diminished patellar reflex imply either a severe muscle strain or, more likely, a neurogenic lesion. A midlumbar radiculopathy and femoral neuropathy should be part of the differential diagnosis.

HISTORY, continued

Past medical history was significant for intermittent light-headedness and tachycardia, however she had been asymptomatic for over one year. She was on no medications. There was no family history of diabetes or neuromuscular disorders. There was no history of trauma and plain radiographs of the pelvis, hip, and femur were normal. She was referred for electrodiagnostic studies, MRI of the lumbar spine and pelvis, and serological studies.

- **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 past history does not identify any obvious hereditary risk factors. There is nothing noted that would put her at higher risk for traumatic or entrapment neuropathy. The differential diagnosis remains unchanged.



PHYSICAL EXAMINATION

The patient had well developed musculature and overall physical examination was unremarkable except for right anterolateral thigh atrophy and a markedly diminished right quadriceps reflex. She did not have high arched feet or any other evidence of muscle atrophy. The right quadriceps reflex was graded as 1+, while the remainder of the muscle stretch reflexes, including left quadriceps, were graded as 2+. Strength examination (MRC scale) was graded 5/5 throughout, including knee extension bilaterally. She was able to do unilateral deep knee bends using the left lower limb, but not the right. Peripheral sensory exam was intact to light touch, pin prick, vibration, and proprioception throughout the upper and lower limbs. Distal pulses were normal.

- **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

There is no evidence of anything to suggest an underlying peripheral neuropathy. The differential diagnosis continues to include femoral mononeuropathy, lumbar radiculopathy, and severe muscle tear or strain.

PHYSICAL EXAMINATION, continued

There was a 3 cm circumferential difference between the right and left thighs measured at 15 cm proximal to the suprapatellar margin.

- **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

There is no new evidence to alter the differential diagnosis previously determined. Since the only abnormalities detected seem to lead to focal pathology involving the peripheral nerve distribution to the right quadriceps, perhaps limited to the vastus lateralis, the focal nature of the problem needs to be addressed in the electrodiagnostic study. At the same time, the possibility of a diffuse pathologic process must not be ignored.



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	recr	amp	dur	poly	effort
R	lumbar paraspinal	N	0	0	-	-	-	-	-	-
R	gluteus maximus	N	0	0	-	N	N	N	N	-
R	gluteus medius	N	0	0	-	N	N	N	N	-
R	adductor longus	N	0	0	-	N	N	N	N	-
R	rectus femoris	N	0	0	-	N	N	N	N	-
R	vastus lateralis	incr	3+	3+	-	2+decr	N	1+incr	N	-
R	vastus medialis	N	0	0	-	N	N	N	N	-
R	biceps femoris (short head)	N	0	0	-	N	N	N	N	-
R	anterior tibialis	N	0	0	-	N	N	N	N	-
R	peroneus longus	N	0	0	-	N	N	N	N	-
R	extensor hallucis longus	N	0	0	-	N	N	N	N	-
R	soleus	N	0	0	-	N	N	N	N	-
R	gastrocnemius (lateral)	N	0	0	-	N	N	N	N	-
R	gastrocnemius (medial)	N	0	0	-	N	N	N	N	-
L	lumbar paraspinal	N	0	0	-	N	N	N	N	-
L	lower extremity screening EMG	N	0	0	-	N	N	N	N	-



SENSORY NERVE CONDUCTION									
nr = no response									
NERVE	LATENCY (ms)			AMPLITUDE (µV)			CONDUCT VEL (m/s)		
	R	L	Norm	R	L	Norm	R	L	Norm
sural	-	-	-	-	-	-	-	-	-
calf to ankle (14cm)	3.1	-	<4.0	18.0	-	>10.0	-	-	-
saphenous	-	-	-	-	-	-	-	-	-
above knee to above ankle	2.5	-	<2.8	10.0	-	>7.0	-	-	-

MOTOR NERVE CONDUCTION									
nr = no response									
NERVE	LATENCY (ms)			AMPLITUDE (mV)			CONDUCT VEL (m/s)		
	R	L	Norm	R	L	Norm	R	L	Norm
femoral	-	-	-	-	-	-	-	-	-
above ing lig to vast med	6.7	6.8	<8.0	5.0	5.0	>4.0	-	-	-
below ing lig to vast med (latmedialis)	6.2	6.3	<7.5	5.0	5.0	>4.0	-	-	-
below ing lig to vast lat	5.9	6.1	<7.5	2.3	4.8	>4.0	-	-	-
peroneal	-	-	-	-	-	-	-	-	-
below fib head to EDB	12.2	12.5	-	4.3	4.3	-	-	-	-
ankle to EDB	4.1	4.2	<5.0	4.9	4.8	>4.0	45.0	45.0	>40.0
tibial	-	-	-	-	-	-	-	-	-
popliteal fossa to med foot	13.1	-	-	10.0	-	-	-	-	-
ankle to med foot	4.2	-	<4.5	11.0	-	>6.0	45.0	-	>38.0
peroneal F wave (at ankle)	48.0	-	<45.0	-	-	-	-	-	-



F-WAVE								
# = number of stimuli P = persistence CD = chronodispersion F:M = ratio of average F-wave amplitude to M-wave amplitude								
R/L	NERVE	#	LATENCY (ms)			CD (ms)	P (%)	F:M(%)
			min	mean	max			
R	peroneal	10	48.0	-	-	-	-	-

- **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.)**

ELECTRODIAGNOSTIC DATA, continued

No additional electrodiagnostic data are offered.

The needle EMG exam shows evidence supporting acute denervation in the right quadriceps confined to the vastus lateralis muscle.

The nerve conduction data are normal with the exception of diminished amplitude of the motor response to the right vastus lateralis.

There is no evidence of a diffuse neuromuscular pathology.

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

DIAGNOSTIC IMPRESSION

Denervation of the vastus lateralis probably due to compression or stretch of the branch of the femoral nerve to that muscle.

COMMENTARY I

The combination of atrophy of the entire vastus lateralis muscle plus electrophysiologic data supporting diffuse denervation of the muscle strongly favors a neurogenic process and excludes muscle rupture or strain. The focal nature of the problem, that is the limitation of clinical and electrophysiological findings to one muscle, excludes the possibilities of radiculopathy and neuropathy of the main trunk of the femoral nerve.

Anatomically, the lesion in this patient was likely very distal, involving only the branch(es) that directly innervate the vastus lateralis, thus sparing the sensory and other motor branches of the femoral nerve. A less plausible possibility would be a more proximal lesion involving only the fascicles that terminate in the vastus lateralis.

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



Serum chemistries, including Mg^{++} and ionized Ca^{++} , were within normal limits. Serum aldolase was 4.6 U/L, creatine kinase was 100 IU/L, both within normal range. Serial fasting blood sugars and glycosylated hemoglobin (HbA_{1c}) levels were normal. Anti-nuclear antibody (ANA) titer was 1:40 and erythrocyte sedimentation rate (ESR/Westergren) was 7mm/hr. Complete blood count (CBC) and urinalysis were normal.

Given this unique presentation, the patient was referred for MRI studies of the quadriceps muscle groups bilaterally. Utilizing a 1.0 Tesla magnetom impact scanner, the patient's thighs were imaged beginning with a T1-weighted axial sequence through the area of interest. This was followed by a dual echo axial sequence and a T1-weighted coronal sequence. A dual echo coronal sequence was then obtained, followed by an short T1 inversion recovery (STIR) coronal sequence. No abnormality was noted in the left thigh. On the right, there was focal decreased muscle mass localized only to the vastus lateralis. The overall volume of the vastus lateralis was decreased and there was increased signal throughout the entire muscle belly on both the T2-weighted as well as the inversion recovery images. The vascular structures showed no asymmetry between right and left and there were no bony abnormalities or masses noted. The study was felt to be compatible with denervation of the right vastus lateralis.

This case demonstrates how the soft tissue contrast and high spatial resolution of MRI can confirm the anatomical distribution of muscle pathophysiology documented by EMG. Recent literature establishes that denervated skeletal muscle shows increased signal intensity on T2 weighted and STIR MRI sequences. In one study using traumatic nerve injury as a model, MRI of skeletal musculature demonstrated reduced sensitivity to acute denervation (less than one month after onset), but excellent sensitivity in detecting subacute nerve lesions. McDonald and colleagues recently demonstrated a close correspondence between increased signal intensity of denervated musculature on MRI and evidence of denervation by needle electromyography.

The cause for signal intensity changes in denervated muscles has not been fully elucidated. Denervation of rat skeletal muscle has been documented to result in longer T1 and T2 relaxation times by MR spectroscopy. These changes were felt to be due to increased size of extracellular fluid relative to intracellular fluid spaces in denervated muscle. A shift in fluid from the intracellular to the extracellular compartment has been felt to be the most likely explanation for the signal changes of denervated muscle on MRI. Extracellular water has a much longer T2 than intracellular water. The STIR sequence may be even more sensitive than the T2 weighted sequence given its fat suppression characteristics and its ability to enhance differences between water content of tissues.

- **What treatment would you recommend?**

The patient was given a short course of oral prednisone which did not produce a clinical response. She was placed on a functional electrical stimulation program to prevent atrophy.

FOLLOW-UP

Repeat needle EMG at five months showed no abnormal rest activity. There was an increased number of abnormally large amplitude, long duration, polyphasic MUAPs, consistent with reinnervation.

The precise etiology of the EMG and MRI findings in this patient were not confirmed. Compression (possibly entrapment) or stretch of the branch to the vastus lateralis remain



the most likely diagnoses. Other possibilities that may have occurred in this patient include focal nerve infarction from minor trauma or focal vasculitis. It is very unlikely that an infectious process such as pyomyositis or post-viral myositis would be isolated to the vastus lateralis. The creatine kinase was normal, making idiopathic inflammatory myopathy unlikely. Exercise and muscle strain may produce signal intensity changes in skeletal muscle on MRI, however, the localized atrophy of the muscle, confirmed in our case by physical examination and MRI would not be expected. In addition, exercise and muscle strain should not produce fibrillation on EMG.

In conclusion, the EMG and MRI findings in this young woman are most compatible with an incomplete axonal lesion isolated to the distal femoral motor branch innervating the vastus lateralis, with subsequent neurogenic atrophy. The highly unusual anatomical presentation of this case illustrates the emerging complementary usefulness of EMG and MRI in delineating neuromuscular pathology.

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