



October 1998 EMG Case-of-the-Month

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HISTORY

The patient is a 51-year-old right handed gentleman who presents with weakness of the left upper limb of approximately 3 weeks duration. His symptoms began with pain in the limb and cramping in his hand a day after he completed trimming a tree. He does not give a history of antecedent trauma.

- **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 differential diagnosis based on this information alone is quite broad. Possibilities include cervical radiculopathy, brachial plexopathy, nerve entrapment in the limb, and less likely, motor neuron disease or a central nervous system problem. Myopathy and peripheral polyneuropathy are unlikely given that the patient presents with symptoms only in one limb. It is also possible that the patient has a musculoskeletal problem.

It would be helpful to know the distribution of weakness and whether there are any sensory deficits. It would also be desirable to obtain a history of the patient's other medical problems, e.g. diabetes, whether he has had cancer, etc.

HISTORY, continued

The pain and cramping resolved in 2-3 days and it was then that the patient started noticing weakness in the limb, primarily at the shoulder. He complains of numbness over the lateral aspect of the forearm. He denies any bowel, bladder, or gait problems. His past medical history is significant for hypertension, mild chronic obstructive pulmonary disease and smoking a half pack of cigarettes a day for the past 30 years.

- **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 differential now points more toward a neuropathic process such as a radiculopathy at C5 or C6, or upper trunk/posterior cord plexopathy given both motor and sensory symptoms. A central process is less likely given symptomatology in one limb only unless



there is a focal lesion of the spinal cord at C5 and C6. Myopathy or monomelic amyotrophy is unlikely given that the patient has sensory changes.

PHYSICAL EXAMINATION

Cranial nerves II-XII are intact. There is minimal atrophy of the left deltoid muscle and no scapular winging. Strength in the left upper limb is: 3/5 shoulder abduction; 4/5 elbow extension; 4/5 wrist extension; 5/5 hand grip; 5/5 wrist flexion; 5/5 thumb opposition; 5/5 thumb abduction; 5/5 index finger and little finger abduction. No weakness is detected in the right upper limb or in the lower limbs. The left triceps and brachioradialis reflexes are less active than the right. The remainder of the muscle stretch reflexes are 2/4 and symmetrical bilaterally in the upper and lower limbs. There is no Babinski response with plantar stimulation and no Hoffmann response. In the left upper limb, decreased sensation to pin prick over the deltoid region and lateral forearm is noted. Sensation in the right upper limb is intact to light touch, pin prick, and proprioception in the right upper limb and both lower limbs.

- **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

Without long tract signs, the physical exam points away from an upper motor neuron lesion. Weakness of the left shoulder abductors, wrist extensors and elbow extensors points towards involvement of the upper trunk or posterior cord and possibly the suprascapular nerve (suprascapular). The sensory findings suggest involvement of the axillary and lateral antebrachial cutaneous nerves. Cervical radiculopathy is still a possibility with roots C5, C6, and/or C7 involved.

- **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 further data are available.

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

COMMENTARY IV

The clinical picture points towards a left cervical polyradiculopathy (since the findings are multisegmental), a brachial plexopathy or a multiple mononeuropathy. A logical place to start would be with the needle EMG examination of the left upper limb and corresponding cervical paraspinal muscles to better assess the extent of involvement. A sampling of muscles should also be tested in the right upper limb.

With this information, nerve conduction studies should be chosen to further localize or clarify the lesion(s). Sensory studies can help differentiate between pre- and post-ganglionic



lesions. In this patient, sensory evaluations should be performed on the left lateral antebrachial cutaneous and radial nerves. Contralateral nerves should be tested also for purposes of comparison. Motor studies of the left upper limb should be performed to assess axonal and demyelinating processes.

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
L	deltoid	incr	3+	3+	-	decr	N	N	N	N
L	biceps brachii	incr	1+	1+	-	N	N	N	N	N
L	triceps	incr	2+	2+	-	decr	N	N	N	N
L	ext carpi radialis	incr	2+	2+	-	decr	N	N	N	N
L	extensor indicis	incr	1+	1+	-	decr	N	N	N	N
L	flexor carpi radialis	N	0	0	-	N	N	N	N	N
L	pronator teres	N	0	0	-	N	N	N	N	N
L	abductor pollicis brevis	N	0	0	-	N	N	N	N	N
L	1 st dors interos	N	0	0	-	N	N	N	N	N
L	lower pectoralis major	N	0	0	-	N	N	N	N	N
L	supraspinatus	incr	1+	1+	-	N	N	N	N	N
L	serratus anterior	N	0	0	-	N	N	N	N	N
L	upper cervical paraspinal	N	0	0	-	N	N	N	N	N
L	middle cervical paraspinal	N	0	0	-	N	N	N	N	N
L	lower cervical paraspinal	N	0	0	-	N	N	N	N	N
R	deltoid	N	0	0	-	N	N	N	N	N
R	biceps	N	0	0	-	N	N	N	N	N
R	triceps	N	0	0	-	N	N	N	N	N
R	supraspinatus	N	0	0	-	N	N	N	N	N
R	pronator teres	N	0	0	-	N	N	N	N	N
R	1 st dors interos	N	0	0	-	N	N	N	N	N
R	ext carpi radialis	N	0	0	-	N	N	N	N	N



SENSORY NERVE CONDUCTION									
nr = no response									
NERVE	LATENCY			AMPLITUDE (µV)			CONDUCT VEL(m/s)		
	R	L	Norm	R	L	Norm	R	L	Norm
lateral antebrachial cutaneous	-	-	-	-	-	-	-	-	-
elbow to forearm	1.9	nr	<2.4	16	nr	>11	-	-	-
median	-	-	-	-	-	-	-	-	-
wrist to thumb	-	2.4	2.9	-	21	12	-	-	-
median	-	-	-	-	-	-	-	-	-
wrist to long finger	-	3.2	3.5	-	28	15	-	-	-
radial	-	-	-	-	-	-	-	-	-
wrist to thumb	-	2.5	2.9	-	3	7	-	-	-
ulnar	-	-	-	-	-	-	-	-	-
wrist to little finger	-	2.8	3.0	-	19	15	-	-	-

MOTOR NERVE CONDUCTION									
nr = no response									
NERVE	LATENCY (ms)			AMPLITUDE(mV)			CONDUCT VEL (m/s)		
	R	L	Norm	R	L	Norm	R	L	Norm
median	-	3.9	(4.1)	-	8.4	(6)	-	-	-
wrist to thenar	-	3.9	<4.1	-	8.4	>6	-	-	-
elbow to thenar	-	9.5	-	-	8.0	-	-	54	49
ulnar	-	3.2	(3.4)	-	10.2	(5)	-	-	-
wrist to hypothenar	-	3.2	<3.4	-	10.2	>5	-	-	-
abv. elbow to hypothenar	-	10.1	-	-	8.4	-	-	55	49
radial	1.9	2.2	-	4.0	1.5	-	-	-	-
forearm to ext indicis	1.9	2.2	-	4.0	1.5	-	-	-	-
abv. elbow to ext indicis	4.8	4.9	-	3.8	1.1	-	57	54	-
abv. sp. groove to ext. ind. indicis	7.1	6.9	-	3.5	0.8	-	55	50	-

- **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.)**



COMMENTARY V

Further testing could include axillary and musculocutaneous motor nerve conduction studies, F-waves, and dermatomal SEPs (DSEPs). However, these additional studies would add little to the localization of the lesion. The involvement of the axillary and musculocutaneous nerves are already assessed by testing the appropriate muscles by needle EMG. F-waves would not necessarily demonstrate abnormalities in any case, and in this case, it is the C5 and C6 segmental levels that need to be evaluated which our F-wave techniques do not permit. DSEPs may show abnormalities but would not help localize the site of the lesion.

ELECTROPHYSIOLOGIC DATA, continued

No further electrophysiologic data were collected.

Summary of Electrodiagnostic Findings

- The left lateral antebrachial cutaneous sensory response is absent.
- The left radial sensory response has an abnormally small amplitude.
- The left radial motor responses have abnormally small amplitudes compared to the right.
- The needle EMG demonstrates abnormality in muscles innervated by the axillary, radial, musculocutaneous and suprascapular nerves. The cervical paraspinal muscles are within normal limits.
- **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.**

DIAGNOSTIC IMPRESSION

Neuralgic amyotrophy

COMMENTARY I

The patient's clinical presentation, supported and clarified by the electrophysiologic findings, are typical of neuralgic amyotrophy. Acute pain at the onset without associated trauma followed by weakness a few days later is characteristic. (The episode of tree-trimming that preceded the onset in this case was never clearly connected to a traumatic incident and functioned as a distracter in terms of determining a differential diagnosis.) Neuralgic amyotrophy may present as a mononeuropathy, but most often involves multiple nerves (multiple mononeuropathy), frequently around the shoulder girdle.

This case, early on, is multisegmental in its distribution. It then sorts into a multiple mononeuropathy. A normal cervical paraspinal exam begins immediately to exclude a diagnosis of cervical radiculopathy, especially polyradiculopathy. The radial sensory amplitude recording from the thumb is diminished, but the median is not, a further indication of a lesion distal to the spinal nerve. Also, sensory nerve recordings at the C7 and



C8 levels are normal, a location where there is motor abnormality (extensor indicis). These sensory studies increasingly negate the possibility of cervical radiculopathy.

The distribution of some of the findings on the needle exam suggest either involvement of the upper trunk or multiple nerves that originate from the upper trunk (supraspinatus, deltoid, biceps), but an upper trunk lesion tends to be excluded by a normal needle exam of the upper pectoralis major and the pronator teres. Note that a normal exam of the infraspinatus muscle suggests that the nerve to the supraspinatus may be affected independently of the suprascapular nerve. Other findings on needle exam implicate involvement of the posterior cord and/or the radial nerve.

The nerve conduction studies show abnormalities in amplitude without evidence of conduction block, thus suggesting that the process is an axonopathy. This is further corroborated by the findings on the needle exam. Since neuralgic amyotrophy is an axonopathy, this is another item of supportive evidence.

Neuralgic amyotrophy has been and continues to be called by many names. Parsonage and Turner described a large series of cases in 1948 and the condition continues to be called Parsonage–Turner Syndrome. Other names include acute brachial neuropathy, acute brachial radiculitis, acute brachial plexitis, acute mononeuropathy of the shoulder girdle, acute multiple neuropathy of the shoulder girdle, acute shoulder neuritis, amyotrophic paralysis of the periscapular muscles, brachial neuritis, brachial plexus neuropathy, cryptogenic brachial neuropathy, idiopathic brachial neuritis, idiopathic brachial plexitis, idiopathic brachial plexopathy, idiopathic brachial plexus neuropathy, localized neuritis of the shoulder girdle, localized nontraumatic neuropathy, multiple neuritis of the shoulder girdle, paralytic brachial neuritis, serratus magnus palsy, shoulder girdle neuritis, and shoulder girdle syndrome. As the many names suggest, this problem has been and continues to be, poorly understood and inconsistently recognized by physicians.

The name neuralgic amyotrophy has been proposed because it does not convey any assumptions about etiology or location of the lesion. Leaving the name "brachial" out of the name is also anatomically correct because there are cases in which there is involvement of nerves that do not pass through the brachial plexus (e.g. spinal accessory, phrenic, cervical roots). This condition can also occur in the lower limbs. The etiology remains unclear. About half the cases have been associated with antecedent illnesses or events such as a vaccination. There is also a familial form of the disease.

Most patients experience acute onset of constant and rather intense sharp pain about the shoulder and in the upper limb. The pain usually decreases within 2-3 weeks. Weakness may be present from the onset, but typically is noted as the pain subsides. Most patients recover functionally, sometimes in a few months, but it can take up to 3 years. The cause of this condition is not known and there are no specific laboratory studies other than electrodiagnosis to help with the diagnosis. Up to one third of patients have bilateral symptoms that are asymmetric.

Most commonly affected are the suprascapular, long thoracic, and axillary nerves. The nerve conduction abnormalities are usually less pronounced than the needle electromyographic examination.

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



Laboratory studies are not abnormal in neuralgic amyotrophy. Treatment is individualized to the person, his activities, and the specific neurologic deficits. General principles are: a) avoid surgery; b) allow the body to heal itself; c) gentle active and passive range of motion when the pain subsides to avoid contractures; d) exercise as the neurologic function allows; and, e) pain management. The long-term prognosis is good with 80% of patients reporting recovery at two years and 90% at four years. (Dyck)

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