



January 1998 EMG Case-of-the-Month

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HISTORY

A 66-year-old woman complains of severe pain and weakness in her left foot and leg. Four weeks prior to this electrodiagnostic consultation she had a three vessel coronary artery bypass graft, with postoperative pain in her left foot and lower leg. Her perioperative course was complicated by congestive heart failure, sepsis, hypoxic and hypotensive episodes, use of an intraaortic balloon pump for three days, and cardiac arrhythmias. She was on the ventilator for 3 weeks postoperatively and was confused before and after extubation. When she started to talk, she complained of leg and foot pain and weakness.

- **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 differential at this stage, lacking further details, is broad. The primary symptoms of acute pain and weakness in the leg below the knee may be due to a) peroneal neuropathy; b) tibial neuropathy; c) single or multiple level radiculopathy (L4 to S1); d) sciatic neuropathy; or e) lumbosacral plexopathy; and there is no information at this stage to separate them. Possible perioperative problems that may or may not be associated with peripheral nerve injuries and cause these symptoms include a) compression; b) compartment syndrome; c) ischemic monomelic neuropathy; d) asymmetric (atypical) presentation of a diffuse polyneuropathy (critical illness neuropathy); e) focal CNS lesion (stroke or spinal lesion); or f) reflex sympathetic dystrophy (complex regional pain syndrome). Other diagnoses seem less likely.

HISTORY, continued

Prior to this operation the patient had a history of mild hypertension and anginal symptoms. She is not diabetic. She has no history of prior extremity weakness or pain. She did not develop renal failure during this acute illness. She did receive aminoglycosides and neuromuscular blocking agents. The intraaortic balloon pump was placed in the left femoral artery. She currently does not have pain in the left groin, buttock, back, or right leg. She does not have bowel and bladder incontinence. She does not have sensory symptoms or weakness in the upper limbs or right lower limb.

- **If necessary, 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

Due to the additional history that this patient was treated with an intraaortic balloon pump in the left femoral artery perioperatively, the diagnosis of ischemic monomelic neuropathy becomes more likely. There is nothing in the additional information that helps with localization of the lesion.

PHYSICAL EXAMINATION

The patient was alert and oriented, with intact memory. She was able to follow three step commands.

No weakness is detected in the upper limbs or the right lower limb. In the left lower limb, it is difficult for the patient to give full effort with hip flexion or knee extension, but there does not appear to be any weakness in these muscles. There is no detectable contraction of the muscles in the anterior compartment (anterior tibialis or extensor hallucis longus) or lateral compartment (peroneal longus) of the leg. Testing of hamstring strength is limited by pain, but a grade of no less than 4/5 is present. Weakness of plantar flexion is detectable manually with the patient supine.

Muscle stretch reflexes in the upper limbs are present (2-3/4) and symmetric. The quadriceps reflexes are 2/4 bilaterally. The left ankle reflex appears to be absent though pain and poor relaxation are influencing factors. The right ankle reflex is easily elicited (2/4).

In left leg there was circumferential numbness in a stocking distribution below the left knee but constant complaints of pain with non-painful stimuli limited detailed localization.

There was trace pitting edema, and the left foot felt colder than the right. Pulses in the dorsalis pedis and posterior tibialis were 2/4 and equal on the left and right.

- **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 physical examination at this point reveals distal lower limb weakness definitely in the distribution of the peroneal nerve and probably the tibial nerve. There is more information from manual muscle testing that may help more accurately to localize the problem. The function of the gastrocnemius and soleus muscles is difficult to assess in a patient who cannot stand; therefore, muscle testing the toe flexors further assesses the tibial nerve component. Additionally, hip abduction strength gives information on L5-S1 function outside the distribution of the sciatic nerve. It would help to determine the exact location of the sensory problems to identify if the tibial and/or saphenous nerves are involved in addition to the peroneal.



PHYSICAL EXAMINATION, continued

Further exam of the left foot shows toe flexion is at least grade 3/5, but testing is limited by pain. Hip abduction strength is at least 4/5 but pain also limits testing. Pinprick testing reveals hyperalgesia in tibial, saphenous, and peroneal distributions but apparent anesthesia in the distal half of the foot. Proprioception in the left great toe is absent. Vibratory sensation to a tuning fork at the first metatarsal head is diminished. These tests are normal in the right foot.

- **If necessary, revise your differential diagnosis based on the additional physical examination results.**
- **Are there laboratory or other tests that could help you in your differential diagnosis?**

COMMENTARY IV

At this stage electrodiagnostic testing will be helpful in localizing the pathology and estimating its severity. Differentiation of root, plexus, or a more peripheral process can be done by examining areas where physical findings are sparse. Evaluating the physical findings in the tibial, peroneal, and saphenous distribution will provide information on severity and will be a major goal of this study. The saphenous nerve (sensory extension of the femoral nerve) is likely to be affected in ischemic monomelic neuropathy and is normal with a sciatic neuropathy. Evaluating for a generalized polyneuropathy should also be done. If all the electrophysiologic data are normal, this will support a CNS lesion.

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 potentials										
R/L	MUSCLE	INSERTION		SPONTAN		VOLUNTARY				
		activ	p wave	fib	other	recrt	amp	dur	poly	effort
R	rectus femoris	n	0	0	-	n	n	n	n	n
R	anterior tibialis	n	0	0	-	n	n	n	n	n
R	tensor fascia lata	n	0	0	-	n	n	n	n	n
R	gastrocnemius	n	0	0	-	n	n	n	n	n
R	lumbar paraspinals	n	0	0	-	-	-	-	-	-
L	psoas	n	0	0	-	n	n	n	n	n
L	rectus femoris	n	0	0	-	n	n	n	n	n



L	adductor longus	n	0	0	-	n	n	n	n	n
L	anterior tibialis	n	3+	2+	-	0	0	0	0	0
L	extensor hallucis longus	n	3+	2+	-	0	0	0	0	0
L	tensor fascia lata	n	0	0	-	n	n	n	n	n
L	short head biceps femoris	n	1+	0	-	n	n	n	n	n
L	medial hamstrings	n	0	0	-	n	n	n	n	n
L	medial gastrocnemius	n	2+	2+	-	decr	n	n	incr	n
L	lateral gastrocnemius	n	3+	2+	-	decr	n	n	incr	n
L	abductor hallucis	n	3+	3+	-	0	0	0	0	0
L	lumbar paraspinals (upper)	n	0	0	-	-	-	-	-	-
L	lumbar paraspinals (lower)	n	0	0	-	-	-	-	-	-

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	3.9	nr	<4.2	25	nr	>8			-
saphenous	3.8	nr	<4.2	15	nr	>5			-
superficial peroneal	4.0	nr	<4.2	12	nr	>5			-
ulnar	-	3.5	<3.9	-	25	>10			-

MOTOR NERVE CONDUCTION									
nr = no response									
NERVE	LATENCY (ms)			AMPLITUDE (mv)			CONDUCT VEL (m/s)		
	R	L	Norm	R	L	Norm	R	L	Norm
-	-			proximal/distal			-		
peroneal to EDB	4.8	nr	<6.0	6/6	nr	>2	45	nr	>40
tibial to abductor hallucis	5.0	nr	<6.0	10/10	nr	>5	43	nr	>40
ulnar	3.0	-	<3.8	8/8	-	>4	55	-	>50
peroneal to anterior tibialis	-	nr	-	-	nr	-	-	nr	-

Significant Findings:

1. Abnormal EMG in all distal left lower limb muscles, with peroneal distribution more affected than tibial.
2. Normal EMG in all proximal left lower limb muscles including muscles innervated by posterior primary rami (L2-S1); sciatic; inferior gluteal; superior gluteal and femoral nerves and also covering roots L2-S1. The only exception is the short head of the biceps femoris.
3. Absent left saphenous, superficial peroneal and left sural sensory responses.



4. Absent left peroneal motor and reduced amplitude left tibial motor.
5. Normal NCS in both upper limbs and right lower limb.
- **On the basis of both the clinical and electrodiagnostic evaluations, formulate your final impression. List the most likely diagnosis followed by other possibilities that are not excluded by the data. Eliminate those diagnoses not supported by the data.**
- **What other diagnostic procedure are needed?**

DIAGNOSTIC IMPRESSION: ELECTRODIAGNOSTIC REPORT

1. There is electrodiagnostic evidence for severe axonal neuropathy in the left lower limb involving the a) peroneal (all three branches); b) tibial; c) sural; and d) saphenous nerves. These findings in this patient are most consistent with ischemic monomelic neuropathy (IMN).
2. There is no electrodiagnostic evidence for a) lumbosacral radiculopathy; b) lumbosacral plexopathy; or c) generalized polyneuropathy.
3. Clinically, these findings explain her left lower extremity symptoms.

The history of severe distal extremity weakness and pain following use of an intraaortic balloon pump is highly suggestive of IMN. This problem is due to interruption in blood flow to the vasonervorum, thus causing axon loss in the nerve distribution of each artery that has low or absent blood flow. In this case there are multiple mononeuropathies of the peroneal (all three branches), tibial, sural, and saphenous nerves. The low blood flow to the distal limb is probably due to multiple factors including low cardiac output, mechanical obstruction in femoral artery from the IABP cannula, vasoconstriction of arterioles and arteries, and increased distance from the aorta.

In IMN there is selective damage to the nerves with little or no evidence for severe muscle, skin, or other tissue necrosis. The reason for nerve susceptibility to ischemia is unclear. With prolonged, severe ischemia other tissues (muscle, skin, connective tissue, etc.) may be involved and develop necrosis, perhaps necessitating amputation.

This distribution of abnormalities cannot be explained by any single area of focal lesion or interruption of a nerve along its course and requires a different model to understand the distribution of nerve damage. There is severe, complete nerve damage in the deep peroneal distribution. These axons originate in the L4, L5, and S1 roots, go through the lumbosacral plexus, then through the sciatic nerve to the common peroneal, and finally to the deep peroneal nerve. Had there been a lesion in the roots or plexus, there would be abnormalities in the superior gluteal distribution affecting the tensor fascia lata and gluteus medius muscles. It is important to note that the blood flow to the gluteal area is primarily through the gluteal arteries, which would not be affected by the mechanical obstruction in the femoral artery. This is also much closer to the aorta than the anterior tibial area and thus more likely to receive adequate blood flow.

The concept of increased nerve damage more distal to the aorta can be demonstrated by several other findings which describe a proximal to distal gradient of involvement:



1. In the tibial nerve distribution, there is severe but incomplete axonal loss in the gastrocnemius, but there is complete axonal damage in the abductor hallucis (demonstrated by no recruitment on EMG and absent tibial motor NCS).
2. The proximal femoral nerve innervated muscles are normal on EMG, but the distal nerve (saphenous) has a completely absent response.
3. The proximal peroneal innervated (peroneal division of the sciatic) muscle, the short head of the biceps femoris, has a small amount of axon loss with normal recruitment while the distal portion has complete axonal loss, demonstrated by the absent peroneal motor response and the lack of recruitment on EMG.

The recovery from IMN is variable. Since the injury is axonal, the prognosis is guarded. This woman recovered grade 4/5 gastrocnemius strength and 2/5 anterior tibialis strength. Pain in the foot persisted.

Although this example of IMN involved the lower extremity, IMN can also occur (less commonly) in the upper extremity and was first reported in patients who had arteriovenous shunts placed for renal dialysis. As in the lower limbs, symptoms include pain and distal weakness reflecting involvement of multiple nerves.

SUGGESTED READING

1. Brown, WF, Bolton, CF (eds). Clinical Electromyography, 2nd ed. Stoneham, MA, Butterworth-Heinemann, 1993; pages 371-8.
2. Honet, JC, Wajszczuk WJ, Rubenfire, M, Kantrowitz A, Raikes JA. Neurological Abnormalities in the Legs After Use of Intraaortic Balloon Pump: Report of Six Cases. Archives of Physical Medicine and Rehabilitation, 56:346-352, 1975.
3. Levin, Kerry H., M.D. AAEM Case Report #19: Ischemic Monomelic Neuropathy. Muscle and Nerve, 12:791-5, 1989.
4. Willbourn, AJ, Furlan, AJ, Hulley, W, Ruschhaupt, W. Ischemic Monomelic Neuropathy. Neurology, 33:447-51, 1983.