American Academy of Physical Medicine and Rehabilitation

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EMG CASE No. 64, September 2003

Presenting Symptom(s): Right leg pain, weakness and numbness

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Presenting Symptom: Neck pain, arm pain, and right hand numbness

Appropriate Audience: Residents and practicing physicians

Learning Objectives: After completing this educational activity, participant will be able to: 1) summarize the neurological findings that may be encountered after hip or pelvic fracture, 2) identify electrodiagnostic findings consistent with lumbosacral plexopathy, and 3) discuss the mechanism of action of lumbosacral plexus injuries associated with hip and pelvic fractures.

This case is no longer available for CME credit.

History

A 42-year-old man sustained a right acetabular fracture with posterior and lateral dislocation after a motor vehicle accident. Open reduction and internal fixation were performed on the first day after the accident. He had noticed some weakness and numbness in the leg pre-operatively.

- 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

Differential diagnosis includes lumbosacral plexopathy, seen after acetabular fractures at an incidence of 0.38-24% (1, 2). The discrepancy in these numbers depends on whether the patients identified were those seen in the EMG lab upon referral (lower number) or those seen concurrently and all evaluated by EMG. The lesion likely is in the lumbosacral plexus rather than the lumbar plexus, the latter being more likely with a gunshot wound, direct injury after superior ramus fracture, or after retroperitoneal hemorrhage. Traction injuries do occur there but are less likely (3). Lumbosacral radiculopathy must be considered in the differential diagnosis, although no back pain is reported. Root avulsion is less common and seen with more severe pelvic and sacral fractures (4, 5, 6). Cauda equina injury should be considered in a trauma case such as this, although no bladder dysfunction was reported. Sciatic neuropathy with stretch at the piriformis muscle is a possibility, with the peroneal branch of the sciatic nerve most susceptible to damage. Peroneal neuropathy at the fibular head should be considered in a patient with hip external rotation due to lateral dislocation, with increased likelihood of compression due to bed rest.

History, continued

He presented to the rehabilitation service four days later for mobility training due to his non-weight bearing status.

- 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?

Physical Examination

On initial examination, left lower extremity strength was 5/5 with intact pin and light touch sensation and 2+ reflexes at the Achilles, patella, and medial and lateral hamstrings. In the right lower extremity, reflexes were 2+ at the patella, 1+ at the lateral hamstring, and absent at the medial hamstring and Achilles. Pin sensation revealed dysesthesias and hypoesthesias in portions of the L5 and S1 distributions on the calf and ventral foot. Sacral sensation was intact. Strength testing using isometric contraction was: hip flexors 3/5, hip adductors 5/5, hip extensors 2/5, hip abductors 2/5, knee extensors 5/5, knee flexors 2/5, ankle dorsiflexors 2/5, ankle plantarflexors 2/5, long toe flexors 4/5, and long toe extensors 1/5. Rectal exam shows normal tone and normal bilateral voluntary contraction with Valsalva maneuver.

- 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 II

Peroneal neuropathy or sciatic neuropathy is less likely due to weakness proximal to their distributions. Careful examination would include isometric muscle strength testing of proximal muscles. Pain inhibition needs to be considered although can be accounted for with careful strength testing.

Physical Examination, continued

- 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 III

Go to Electrophysiologic Data.

Electrophysiologic Data

The EMG study was completed exactly three weeks after the date of injury.

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SENSORY NERVE CONDUCTION STUDIES							
NERVE	**SIDE**	**STIM SITE**	**RECORD**	**cm**	**AMPL**	**LAT**	**CV**
Saphenous	R	Calf	Ankle	14	4.0	1.9	53
	L	Calf	Ankle	14	4.5	2.3	50
Sural	R	Calf	Ankle	14	10	3.5	48
	L	Calf	Ankle	14	4.0	3.2	56

MOTOR NERVE CONDUCTION STUDIES							
NERVE	**SIDE**	**STIM SITE**	**RECORD**	**cm**	**AMPL**	**LAT**	**CV**
Peroneal	R	Ankle	EDB	9	4.5	4.0	
	L	Ankle	EDB	9	0.6	4.7	
	L	Below Fibular Head	EDB	28	0.5		40
	L	Above Fibular Head	EDB	10	0.4		39
Tibial		Ankle	AH	8	10.0	5.1	
Tibial F Response		Ankle	AH			51.7	

- 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.)

  Nerve conduction studies reveal symmetric saphenous sensory nerve evoked amplitudes but a decreased right sural sensory evoked amplitude. Distal motor

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conduction studies reveal a decrease in the right peroneal motor evoked amplitude. There is no conduction slowing across the knee. Tibial F-response in the right lower extremity is normal.

NEEDLE ELECTROMYOGRAPHY
INSERtional activity: N, sust, unsust
FIB: 0, 1+, 2+, 3+, 4+
OTHer: 0 or fascic, myotonia, myokymia
EFFort: N, decr
RECruitment: N, inc or dec 1+, 2+, 3+, 4+
AMPlitude: N, inc or dec 1+, 2+, 3+, 4+
DURation: N, inc or dec 1+, 2+, 3+, 4+
POLyphasia: N, inc or dec 1+, 2+, 3+, 4+

R/L	MUSCLE	INSER	FIB	OTH	EFF	REC	AMP	DUR	POL
R	Anterior Tibialis	Inc.	4+	0	N	0			
R	Peroneus Longus	Inc.	4+	0	N	Dec 3+	N	N	Inc. +/-
R	Medial Gastroc.	Inc.	2+	0	N	N	N	N	N
R	Vastus Medialis	Inc.	unsust	0	N	N	N	N	N
R	Adductor Longus	Inc.	unsust	0	N	N	N	N	N
R	Gluteus Medius	Inc.	4+	0	N	0			
R	Iliopsoas	Inc.	4+	0	N	0			

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

Needle examination reveals denervation and neuropathic motor unit changes in the right L5 and S1 nerve root distributions. This includes proximal gluteal nerve supplied muscles. Similar findings are seen in the iliopsoas but not the vastus muscles.

Disgnostic Impression

There is evidence of a right lumbosacral plexopathy proximal to the take off of the gluteal nerves. There is also evidence of a right lumbar plexopathy affecting only the proximal fibers going to the iliopsoas. The fibers that make up the femoral and obturator nerves are relatively spared.

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

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Commentary IV

Lumbosacral plexus injuries are common in patients with sacral and pelvic fractures. SI disruption leads to a high incidence of plexopathy as the proximal plexus arising from the L5-S2 nerve roots run in front of the SI joint (7, 8). Concomitant L5 transverse process fracture (far out fracture) is sure to result in an L5 nerve root injury. Sacral fractures result in nerve root injury if the fracture passes through a nerve foramen or if the fracture is transverse across the spinal canal (9). Isolated pelvic fractures lead to a relatively lower incidence of plexus injury. However, nerve stretch, direct trauma from bone fragments, or retroperitoneal hemorrhage can result in nerve damage. In the first, the lumbosacral plexus is more vulnerable, accounting for 80% of the injuries (3), while in the latter two, it is the lumbar plexus that is most affected. The lumbar plexus proximal branches to the iliopsoas are the most susceptible fibers after traction injury (10). Nerve traction sites for the lumbosacral plexus include the dura attachments, connective tissue attachments to the sacral ala and anterior SI joint, and connective tissue attachments to the piriformis and sacrotuberous ligament (7). Though lumbosacral plexus injuries are most common after open book fracture with SI disruption and are commonly seen in acetabular fractures with dislocation, these injuries are also seen in simple acetabular fractures, femur fractures, and after total hip replacement.

EMG and nerve conduction studies, completed at least 10-14 days after injury, are very sensitive in evaluating this lesion. Sensory nerve conduction studies of the saphenous, peroneal, and sural nerves bilaterally allow for the confirmation of damage distal to the dorsal root ganglia. Amplitude drops of 50% or more on the affected side are consistent with an injury to the plexus, with 72% showing abnormalities on sural nerve testing and 41% showing abnormality on other sensory nerve testing. Motor studies confirm the distribution of motor axons involved, with 55% showing abnormalities in motor evoked amplitude (2). Needle examination of proximal and distal muscles clarifies the distribution of the nerves involved. Needle electrode study is the gold standard by which the diagnosis of plexopathy is made in most cases. Evaluation should include muscles of the anterior (obturator) and posterior (femoral) divisions of the lumbar plexus as well as anterior (tibial) and posterior (peroneal, gluteal) divisions of the lumbosacral plexus. In the lumbosacral plexus, evaluation of the gluteal muscles helps to clarify traction at the piriformis to a lesion more cephalad in the plexus. The role of evaluation of sacral plexus muscles (piriformis, quadratus femoris, obturator internus) in this disorder has not been clarified. Paraspinal examination is useful for evaluating the presence or absence of root level pathology. If present, root involvement should be further evaluated by MRI scan of the lumbar spine and CT scan for the sacral roots.

Outcome data on this patient population is lacking in the literature. Case studies on the rare lumbosacral nerve root avulsion showed a generally poor prognosis with less than antigravity motor return in the affected nerve root distribution. This gentleman improved to greater than 4/5 strength in all muscles within three months. He continued to have sensory loss in an L5 and S1 distribution. Bone healing has allowed him to progress to weight bearing as tolerated.

Based on this limited information, management recommendations would include that all patients with high risk injuries be tested by EMG. This would include all patients with acetabular fractures, posterior hip dislocation, SI fracture or dislocation, unstable pelvic ring

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fractures, or sacral fractures across a neuroforamen or across the spinal canal. Clinical management is supportive with stretching to prevent contracture formation and strengthening to minimize atrophy in cases with nerve continuity. Correct positioning in bed is required to prevent superimposed peroneal neuropathy at the fibular head. Patients are usually non-weight bearing for a significant period of time after these fractures, though decisions on ankle orthoses for ambulation to control foot drop should be made in anticipation of advancing the patient's weight bearing status. If the injury involves the gluteal nerves, an ambulation assistive device such as a cane or walker may be necessary. Functional electrical stimulation to affected muscles is recommended only if nerve continuity is present. For severe nerve injury, the location of the plexus injury usually precludes surgical exploration.

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