



EMG Case No. 71, September 2004

Presenting Symptom(s):

Right foot drop after sustaining a right tibia and fibula fracture

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Disclosures: V.S. Bodeau, None; M.F. Daut, None; S. Kishner, None.

Appropriate Audience: Residents and practicing physicians

Learning Objectives: After completing this educational activity, participant will be able to (1) formulate a differential diagnosis for chronic foot drop, (2) evaluate the electrodiagnostic findings associated with compartment syndrome, and (3) summarize the etiology and treatment options for foot drop.

This case is no longer available for CME credit.

History

A 52-year-old white male presents for electrodiagnostic evaluation of a chronic right foot drop. He is being followed by the orthopedic team for a tibial malunion secondary to a complex right tibia and fibula fracture 2½ years ago. He states he wears cowboy boots at all times to avoid tripping over his toes.

- 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 for foot drop is extensive and includes common peroneal neuropathy at the fibular head, a L5 radiculopathy, deep peroneal neuropathy, compartment syndrome, sciatic neuropathy, lumbosacral plexopathy, or a CNS process such as a CVA. Of the above, a deep peroneal neuropathy is a high suspect as it commonly presents with foot drop. Given the extensive trauma to the right leg, a common peroneal neuropathy at the fibular head is also likely. Compartment syndrome is considered as it can occur in up to 30% of patients with tibia shaft fractures. An L5 radiculopathy or sciatic neuropathy could be possible depending on other injuries sustained in the accident or pre-existing medical history.

History, continued

Further questioning of the patient reveals that the injury was caused by a large metal drainage pipe falling onto his leg. He had no injuries to his right foot or thigh, back, hip, or left leg. Prior to the injury he had no history of back pain, difficulty in ambulating or symptoms of radicular pain. The foot drop started shortly after the injury. He has experienced both chronic pain and numbness in the right lower leg and foot since that time. He has undergone multiple surgical procedures including hardware placement, fibular osteotomy, and placement and removal of an external fixator device. Compartment syndrome and fasciotomy procedures were described to the patient in the EMG lab, but he



denies ever hearing this diagnosis or such a procedure. He denies surgeries other than those related to his injury, and his past medical history is significant only for alcoholic liver cirrhosis and a remote history of asthma. He has no known drug allergies and is not on any medications at this time.

- 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 additional history allows us to narrow our differential diagnosis. Trauma to the right leg and multiple surgical procedures suggest peroneal neuropathy at the fibular head and/or deep peroneal neuropathy as the likely sources of the foot drop. Although the patient denies a previous diagnosis of compartment syndrome, it cannot be excluded at this time. A foot drop from a CVA, L5 radiculopathy or sciatic neuropathy seem less likely given that the patient had no other injuries from the accident or suggestive symptoms. Adding peripheral neuropathy to the differential is appropriate given the patient's history of heavy alcohol use.

Physical Examination

On visual inspection of the right foot and leg, there is marked atrophy of the extensor digitorum brevis, and mild to moderate atrophy of the muscles in the posterior and lateral leg compartments. Multiple surgical scars are present over the right lower limb including the anterior tibial tubercle, lateral malleolus, and along the length of the leg at the previous external fixator sites. There are no scars indicative of a fasciotomy. Manual motor testing of the right lower limb show hip flexors and knee extensors 5/5, plantar flexion and dorsiflexion 4/5, great toe extension 0/5 and flexion 1/5. Foot inversion is 3/5 and eversion 2/5. These muscles all tested in left lower extremity are of normal strength. Sensory testing to light touch is also normal on the left. On the right, sensory testing is decreased to light touch in a nondermatomal distribution. Area delineated is most consistent with superficial and deep peroneal cutaneous and distal sural cutaneous distribution patterns. Light touch is intact over planter surface of the foot. Muscle stretch reflexes are 2/4 and symmetric at the left and right patella tendon and Achilles tendons. Right leg soft tissue is not tight or tender and capillary refill is normal. No pain with range of motion or compression noted.

- 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

Atrophy of the extensor digitorum brevis, decreased ankle plantarflexion and dorsiflexion, weak foot eversion, and lack of great toe extension (extensor hallucis longus) all point strongly to involvement of the superficial and deep peroneal nerves individually or the common peroneal nerve at the fibular head. Mild atrophy of the posterior compartment muscles indicates tibial nerve involvement, which had not previously been considered. Peripheral neuropathy secondary to alcohol use is still possible, but unlikely as the motor and sensory deficits were found only on the right side. Compartment syndrome was either previously considered and ruled out, in absence of fasciotomy scar, or was not diagnosed. However, given the extensive trauma history and multiple surgical interventions, compartment syndrome cannot be ruled out as a contributor to this patient's symptoms.



Physical Examination, continued

Patient’s medical records had been requested and only radiologic studies were available for review. An MRI of the right leg, done 6 months ago, revealed healing fractures of the proximal and distal third of the tibia, mild periosteal reaction and edematous signal in the bone marrow, non-displaced fractures of the fibular shaft at the level of the tibial fracture. Also mild fatty replacement of muscles in the leg, particularly in the posterior compartment was 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 imaging findings confirm the high possibility of direct nerve injury to the common peroneal nerve or its divisions, and the sural or tibial nerves due to the significant extent of the injuries. These injuries also lend credibility to possible ischemic injury to nerves and muscles from an untreated acute or chronic subacute compartment syndrome. The fatty infiltration in the muscles is especially interesting as it is a known late sequelae of compartment syndrome. An EMG/NCS is performed to help delineate which muscles and nerves were affected.

Electrophysiologic Data

SENSORY NERVE CONDUCTION STUDIES							
NERVE	SIDE	STIM SITE	RECORD	cm	AMPL	LAT (peak)	CV
Sural	R	Calf	Ankle	14	NR	NR	
Superficial Peroneal	R	Ankle	Foot	14	NR	NR	
Medial Plantar	R	Sole	Ankle	14	3.1	3.9	50
Sural	L	Calf	Ankle	14	17	4.0	51
Superficial Peroneal	L	Ankle	Foot	14	8	4.0	53
Medial Plantar	L	Sole	Ankle	14	3.8	3.4	58



MOTOR NERVE CONDUCTION STUDIES							
NERVE	SIDE	STIM SITE	RECORD	cm	AMPL	LAT	CV
Peroneal	R	Ankle	EDB	8	NR	NR	NR
Peroneal	R	Fib Head	EDB	8	NR	NR	NR
Peroneal	R	Across Fib Head	EDB	10	NR	NR	NR
Peroneal	R	Fib Head	Tib Ant	8	7.2	4.7	49
Peroneal	R	Across Fib Head	Tib Ant	10	7.0	6.7	49
Tibial	R	Ankle	Abd Hal	8	3.0	6.2	37 (pop fossa to ankle)
Tibial	R	Popliteal Fossa	Abd Hal	41	2.9	11.1	37
Peroneal	L	Ankle	EDB	8	3.2	5.9	45 (below fib head to ankle)
Peroneal	L	Fib Head	EDB	33	3.1	13.2	45
Tibial	L	Ankle	Abd Hal	8	6.5	5.5	44 (pop fossa to ankle)
Tibial	L	Popliteal Fossa	Abd Hal	42	6.3	15.0	44



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	Lumbar paraspinal	N	0	0	N	N	N	N	N
R	Gluteus maximus	N	0	0	N	N	N	N	N
R	Gluteus medius	N	0	0	N	N	N	N	N
R	Vastus med	N	0	0	N	N	N	N	N
R	Tib ant	N	0	0	N	N	N	N	N
R	Med gastroc	Dec	0	0	N	Dec	N	N	N
R	Ext Hall Longus	Very decr	0	0	N	Very dec	N	N	N
R	Flex hall longus	Very dec	0	0	N	Very dec	N	N	N
R	Peroneus longus	Dec	0	0	N	Dec	N	N	N
R	Biceps fem sh	N	0	0	N	N	N	N	N
R	Peroneus brevis	Dec	0	0	N	Dec	N	N	N
R	Tibialis posterior	Dec	0	0	N	Dec	N	N	N
R	Abd digit mini	Dec	0	0	N	Dec	N	N	N

- 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.)

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

Diagnostic Impression

Nerve conduction studies of the right leg show absence of sural or superficial peroneal sensory nerves, while normal in the left. The right medial plantar sensory nerve is present however latency is prolonged and amplitude decreased especially when compared to the unaffected side. Motor studies reveal absent peroneal activity above and below fibular head. The peroneal nerve on left was within normal limits. The right tibial nerve is present but with lower amplitude when compared to the left. Also, the latency is prolonged and conduction velocity below normal limits. The needle EMG studies show decreased recruitment and decreased insertional activity of motor unit action potentials especially in the extensor hallucis longus, but also in peroneus longus and peroneus brevis. These findings indicate branches of both deep and superficial peroneal nerves are affected but with sparing of more proximal deep peroneal muscles such as tibialis anterior. (although this could also be evidence of completed sprouting and recovery) These same chronic ischemic findings of decreased activity are seen in tibial innervated muscles especially the flexor hallucis longus, but also in medial gastrocnemius, tibialis posterior and adductor digiti minimi. All muscle abnormalities generally show decreased insertional activity with normal appearing but decreased numbers of motor unit action potentials. There is no abnormal spontaneous activity present in any muscles tested. This constellation suggests an old injury to the right leg complicated by an untreated compartment syndrome. There is no evidence of ongoing reinnervation.

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

Commentary V

Compartment syndrome, either initially acute and untreated, or subacute and chronic is the most likely diagnosis to explain the physical and electrophysiologic findings in this patient. The decreased recruitment pattern and decreased insertional activity seen on EMG studies confirms the diagnosis of distal nerve degeneration and chronic muscle fiber necrosis. The injury seen in chronic compartment syndrome involves degeneration of the distal nerve endings, which appear more susceptible to ischemic injury as well as segmental muscle necrosis. In this setting, the typical findings of a myopathy, ie, increased recruitment and small amplitude motor units, are not seen. Other causes for decreased insertional activity can be excluded by proper needle placement in muscle, to exclude technical error, and absent history of polio or active periodic paralysis syndrome. Aside from these causes, only compartment syndrome can explain the chronic changes seen in these muscles as well as the abnormalities seen on nerve conduction. Partial nerve injuries from traction, pressure, or trauma is also a possibility; but less likely because no neuropathic units, from reinnervation, were evident in the involved muscles tested by EMG. In classic compartment syndrome, the sural nerve is frequently spared but is absent in this case. The sural nerve is a part of the superficial deep compartment, however, and can occasionally be affected by external pressure from a cast, for example. As previously stated, compartment syndrome can be seen in up to 30% of tibial shaft fractures and risk is increased with further manipulation including surgery or pin/rod placement.

Compartment syndrome is caused when elevated pressure within a closed fascial compartment reduces capillary perfusion to below the level of tissue viability. It was first described by Volkmann in 1881. Compartment syndrome in the leg usually affects the



anterior compartment; while in the forearm, the flexor compartment is most susceptible. Compartment syndrome can be seen after femur fractures, but is less common secondary to the large thigh fascial compartments.

Clinically, compartment syndrome is characterized by pain, paresthesia, painful ROM, and a tense swollen fascial compartment. Loss of two-point discrimination is a sensitive indicator of increased pressure. If compartment syndrome is suspected, pressure within the fascial compartment must be checked quickly and repeated frequently. Fasciotomy is the definitive treatment. Normal resting compartment pressure is 0 – 8 mm Hg. Elevation of intra-compartment pressure to 30 mm Hg for 6 – 8 hours can cause substantial and irreversible damage. If undetected, these ischemic changes can occur at various nerve innervation levels and individual muscles can be randomly damaged. This is evident in the above electrodiagnostic findings of ischemic changes in some muscles with sparing of other muscles innervated by the same nerve. For example, the distal branch of the deep peroneal nerve to the extensor hallucis longus shows severely decreased insertional activity while the more proximal branch to the tibialis anterior is spared. While most cases of compartment syndrome affect the anterior compartment only, in this case, all compartments show evidence of ischemic changes. In the anterior compartment, as explained above, only the extensor hallucis longus shows decreased activity and the deep peroneal sensory branch is also affected based on clinical exam. In the lateral compartment, all contents are involved including decreased insertional activity in the peroneus longus and brevis and superficial peroneal sensory absence on nerve conduction study. In the deep posterior compartment, the flexor hallucis longus and tibialis posterior have decreased activity. The tibial nerve is also affected significantly with decreased amplitude, increased latency and decreased conduction velocity all indicating axonal injury. The extent of this injury extends beyond the ankle and is evident by decreased amplitude and increased latency of medial plantar study and decreased activity seen in abductor digiti minimi with needle testing. In the fourth and final compartment, the superficial deep compartment, there is decreased activity of the gastrocnemius and absent sural sensory activity on nerve conduction studies.

The presenting symptom of foot drop in this patient is more difficult to explain and more complicated than initially thought. His foot drop may be related to and complicated by other factors such as contractures and/or muscle fatigue because there is sparing of the peroneal nerve to the tibialis anterior. Of the abnormal nerves tested, the findings reveal absence, and therefore likely irreversible damage, of sensory nerves and motor nerves. Electrodiagnostic information suggests chronic changes without evidence of any reinnervation so prognosis for functional recovery is poor given 2-year length of symptoms. The patient may benefit from an ankle foot orthosis.

Bibliography

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