

EMG Case No. 88, June 2007

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Presenting Symptom(s): knee pain, numbness/paresthesias from the knee down the posterolateral leg to the lateral malleolus/lateral calcaneus

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Disclosure: J Levin, None; R Werner, None

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Appropriate Audience: Residents and practicing physicians

Learning Objectives: After completing this educational activity, participant will be able to: (1) evaluate a patient with atypical lower extremity sensory abnormalities; (2) outline the peripheral nervous innervation of the lower extremities, and apply this to anatomic structures that may cause symptoms; and (3) explain a rare cause of lower extremity paresthesias.

Level of Difficulty: Advanced

History

A 64 year-old woman with a history of a two lumbar surgeries (laminectomy and fusion) presented with a 16 month history of left knee pain and numbness down the posterolateral distal left leg. Her symptoms began after undergoing an arthroscopic knee surgery to remove a Baker's cyst. Five days later, her symptoms were exacerbated when she was involved in a motor vehicle collision in which her knees hit the dashboard. X-rays of the knee demonstrated mild-to-moderate tricompartmental osteoarthritis. She consulted an orthopedic surgeon who recommended physical therapy, including closed-chain exercises, bicycle riding, knee strengthening, and weight loss. Her symptoms persisted and she was referred for an electrodiagnostic evaluation.

1. Prior to continuing, please develop a differential diagnosis and list each possible diagnosis in order of likelihood.
2. 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

At this time, the differential diagnosis is broad. It can be divided into processes affecting the nervous system, and processes affecting the knee joint.



When considering neuropathic processes, recall the anatomy of the popliteal fossa, which is the site of Baker's cysts. The popliteal fossa is bounded laterally by the biceps femoris, inferiorly by the gastrocnemius, and medially by the semimembranosus/semitendinosus. The floor of the popliteal fossa is the femur/knee joint/popliteus, and the roof is the popliteal fascia. The sciatic nerve typically enters the superior aspect of the popliteal fossa and bifurcates into the tibial and common peroneal nerves in the upper part of the fossa. Classically, the tibial nerve gives off one sensory branch, the medial sural cutaneous nerve, and continues distally with the motor branches. The common peroneal nerve gives off two sensory branches in the fossa, the lateral sural cutaneous nerve and the sural communicating branch. It then continues distally as the common peroneal nerve, passing across the fibular head before bifurcating into the deep and superficial peroneal nerves. The sural communicating branch joins the medial sural cutaneous nerve distal to the popliteal fossa to form the sural nerve.¹

Anatomic variations of the anatomic contribution of the sural nerve have been reported. These include absence of the lateral cutaneous sural nerve (16.7%-18.9% of specimens), absence of the medial cutaneous sural nerve (2.7%-6.7% of specimens), and no sural communicating branch, usually with the medial cutaneous sural nerve following the classic course of the sural nerve (6.7%-32.4% of specimens).^{2,3}

Given the anatomic position in the popliteal fossa of the sciatic nerve, tibial nerve, common peroneal nerve, medial sural cutaneous nerve, and lateral sural cutaneous nerve, any one, or a combination of several of these peripheral nerves, could have been affected by the Baker's cyst, or by a complication of the surgery. Therefore, the differential diagnosis includes sciatic, tibial, common peroneal, medial sural cutaneous, and lateral sural cutaneous mononeuropathies. Additionally, given the distribution of her symptoms, a sural mononeuropathy and a superficial sensory peroneal mononeuropathy should be included in the differential diagnosis. Also, one should consider the patient's history of multiple lumbar spine surgeries, and include radiculopathies in the differential diagnosis. Lastly, although unlikely, a lumbosacral plexopathy is included in the differential diagnosis.

In considering processes affecting the knee joint, one should consider knee osteoarthritis and referred pain as a possible cause of her symptoms. Other sources of knee joint pathology, including meniscal and ligamentous injury, should be included. Considering the mechanism of her motor vehicle collision (her knees hit the dashboard), a posterior cruciate ligament injury is added to the differential diagnosis.

History, continued

Her knee pain was mostly located at the medial aspect of the joint, and was worse with weight bearing. Although this knee pain was limiting her mobility, the numbness and paresthesias were more concerning and bothersome to her. She described these symptoms as pain, numbness, and tingling located along the posterolateral left leg from the knee to the lateral ankle and foot. The pain and paresthesias were worsened with prolonged sitting and improved with standing. There was no weakness, no back pain, no symptoms proximal to the knee, and no bowel or bladder abnormalities. Review of systems demonstrated no fevers or night sweats, no cardiac or pulmonary abnormalities, and no depression. Past medical history was negative for diabetes, thyroid disease, blood clots, and coagulopathy.

1. If necessary, revise your differential diagnosis based on the additional clinical history.
2. On which details of the physical examination should you focus at this point?

Commentary II

The additional history suggests a neuropathic process as the etiology of her primary complaint. The distribution of the numbness along the posterolateral distal leg and lateral foot and ankle suggests involvement in the distribution of the sural nerve, the lower lumbosacral plexus, and the L5/S1 nerve roots. Although involvement of the superficial sensory peroneal nerve cannot be ruled out at this time, it appears less likely to be involved. The absence of motor involvement suggests that a lesion of the sciatic, tibial, or common peroneal nerve is less likely, although still a diagnostic consideration. The exacerbation of her paresthesias with prolonged sitting raises two possibilities. One is compression of the popliteal fossa in the seated position with irritation of the involved peripheral nerve. A second possibility is a radiculopathy, as intradiscal pressures have been shown to be elevated in the seated position⁴⁻¹⁰, thereby potentially flaring radicular pain.

Physical Examination

Her height was 64 inches and weight was 221 pounds, for a body mass index of 38 kg/m². Vital signs were stable. Motor examination of the lower extremities demonstrated 5/5 strength in all major muscle groups. Sensory examination demonstrated impaired sensation in the left lower extremity at the lateral foot/ankle. Reflexes were 1+ bilaterally at the knees, ankles, and internal hamstrings. Examination of the left knee demonstrated good stability to anterior, posterior, valgus, and varus stresses. Knee range of motion was full. There was no effusion. There was mild fullness in the popliteal fossa consistent with a Baker's cyst. There was crepitus with full extension of the knee. Seated straight leg raise was negative.

1. At this point, review your differential diagnosis and revise as appropriate.
2. Are there additional observations on physical examination that might be helpful in narrowing your differential list?

Commentary III

The physical examination confirmed involvement in the distribution of the sural nerve. Normal strength and reflexes suggest involvement of a sensory nerve only. The negative straight leg raise lowers radiculopathy on the differential diagnosis list.

When investigating peripheral nerve lesions, Tinel sign can be helpful in localizing the lesion.

Physical Examination, continued

Tinel sign over the left common peroneal nerve at the fibular head was negative. Compression of the popliteal fossa (similar to a Tinel sign) reproduced her paresthesias down the posterolateral leg.

1. If necessary, revise your differential diagnosis based on the additional physical findings.
2. Design your approach to the electrophysiologic examination based on the existing data.

Commentary IV

Reproduction of the patient's typical paresthesias with compression of the popliteal fossa suggests that this is the site of a peripheral nerve lesion. Combining this information with the distribution of her symptoms and the lack of motor involvement places the lesion at the medial sural cutaneous nerve, the lateral sural cutaneous nerve, the sural nerve, or a combination of these nerves. Given the predominance of symptoms in the posterolateral leg and the fact that the sural nerve typically is not formed until distal to the popliteal fossa, involvement of the medial sural cutaneous nerve is most likely.

GO TO ELECTROPHYSIOLOGIC DATA



MOTOR NERVE CONDUCTION STUDIES							
NERVE	SIDE	STIM SITE	RECORD	cm	AMPL	LAT	CV
Peroneal	Left	Ankle	Extensor digitorum brevis (EDB)	9	4.6mV	4.0ms	
Peroneal	Left	Below knee	EDB	25	4.3mV	9.2ms	47.9m/s
Tibial	Left	Ankle	Abductor hallucis	8	7.5mV	7.5ms	
Tibial	Right	Ankle	Abductor hallucis	8	5.1mV	4.0ms	
Tibial F response	Left	Ankle	Abductor hallucis			47.1ms	

SENSORY NERVE CONDUCTION STUDIES							
NERVE	SIDE	STIM SITE	RECORD	cm	AMPL	LAT	CV
Sural	Left	Calf	Lateral malleolus	14	5.8µV	3.8ms	42.4m/s
Sural	Right	Calf	Lateral malleolus	14	15.2µV	3.6ms	48.3m/s
Superficial peroneal	Left	Anterior shin	Anterior ankle	12	5.9µV	3.6ms	40.0m/s
Superficial peroneal	Right	Anterior shin	Anterior ankle	12	4.1µV	3.3ms	44.4m/s

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
L	Anterior Tibialis	N	0	0	N	N	N	N	N
L	Medial Gastrocnemius	N	0	0	N	N	N	N	N
L	Abductor Hallucis	N	0	0	N	N	N	N	N

1. 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.
2. 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.)

Electrophysiologic Data, continued

No further electrophysiologic data was obtained.

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

Diagnostic Impression

There is electrodiagnostic evidence of a left lower extremity sural mononeuropathy. The low amplitude left sural sensory response suggests significant axon loss. Given the insignificant difference in the latencies and conduction velocities when compared to the unaffected side, the site of the lesion is proximal to the sural nerve stimulation site. Based on the clinical findings, the location of the lesion is at the popliteal fossa, most likely involving the medial sural cutaneous nerve. The residual sural response is likely due to intact contribution from the lateral sural cutaneous nerve.

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

Commentary V

Imaging of the popliteal fossa, including ultrasound and/or MRI, is indicated to evaluate for a correctable cause of the patient's symptoms. An MRI was subsequently obtained and demonstrated a mildly complex 2.5cm Baker's cyst. Additionally, the MRI demonstrated severe medial compartment-predominant osteoarthritis with complete cartilage loss, subchondral edema, and sclerosis. There was also a severely macerated medial meniscus and a torn lateral meniscus. Musculoskeletal ultrasound, which was not obtained, may have been appropriate for greater resolution of superficial structures.

An injection of 1mL of 0.5%mg bupivacaine and 40mg of Depo-Medrol was performed into the area around the Baker's cyst and she was started on Lyrica 50mg po bid. This provided complete resolution of her pain and paresthesias for 1 month. She returned for a follow-up visit 2 months later, and the injection was repeated in addition to an intra-articular left knee injection. She returned for a follow-up visit 3 months later and reported no ongoing symptoms.

Complications from Baker's cysts are rare,¹¹ however, several cases of peripheral nerve involvement due to Baker's cysts, including the tibial nerve,¹²⁻¹⁷ the peroneal nerve,¹⁷⁻¹⁹ and the sciatic nerve,²⁰ have been reported. The authors are aware of one prior report of a sural mononeuropathy secondary to a Baker's cyst.¹⁷

Sural nerve injury from any cause is rare. In the largest series of 20 sural nerve mononeuropathies over 12 years, the majority (12/20) were found to be iatrogenic (external malleolus ORIF [5], small saphenous vein stripping [4], nerve graft [1], plaster cast for an ankle sprain [1], and synovial cyst removal from the dorsum of the foot [1]). Other etiologies included poorly adapted shoes (3), trauma (2), calcaneus fracture (1), small saphenous vein phlebitis (1), and possible mononeuritis (1).²¹ Although rare, it is important to consider sural mononeuropathy as the etiology of pain and paresthesia of the distal lower extremity.

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