



An Unusual Case of Bilateral Femoral Neuropathy: a case report.

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ABSTRACT

Case: A 49-year-old female presented to the electrodiagnostic laboratory with complaints of weakness and stabbing pain of the lower limbs. The patient was admitted to acute care after being found in a semi-prone position with one leg held in full extension and the other in a kneeling posture. She had apparently overdosed on Xanax, Soma and Vicodin. She developed pressure ulcers on her knees and the left medial hand and right elbow secondary to positioning on the floor during loss of consciousness. At presentation to the electrodiagnostic lab, the patellar reflexes were not obtained secondary to knee ulcerations. Light touch sensation was decreased in bilateral saphenous distributions and right lateral thigh. Hip flexion was 4/5 bilaterally. Hip extension, abduction and adduction graded 5/5 bilaterally. Knee extension was 2/5 on the right and 1/5 on the left side. Dorsiflexion was 4/5 on the right and 5/5 on the left side. Plantarflexion was 5/5 bilaterally. Electrodiagnostic findings were indicative of a severe bilateral femoral neuropathy with almost total axon loss and severe demyelination. Loss of the saphenous sensory nerve action potential amplitude confirmed this impression. This was likely due to a stretch injury superimposed on compression of the femoral nerve under the inguinal ligament based on the positioning of her body during prolonged loss of consciousness. There was no electrodiagnostic evidence for lumbosacral plexopathy or radiculopathy as the proximal muscles innervated by other nerves were normal. Treatment included knee ankle foot orthosis' with free ankle joints that enabled the patient to ambulate and the addition of pregabalin for neuropathic pain management. **Discussion:** This is an unusual case of bilateral femoral nerve compression caused by positioning during a prolonged period of unconsciousness. Bilateral femoral entrapment neuropathies at the inguinal ligament are rare and the literature search is bereft of similar positional neuropathy cases not associated with surgery. Usually the prognosis is favorable and full recovery is observed, but based on compound muscle action potential amplitudes of the femoral nerve it is likely that recovery may be incomplete and prolonged up to two years.

CASE DESCRIPTION

A 49 year-old-female with history of depression and bipolar disorder presented to Moss Rehab following a presentation at an acute care hospital with rhabdomyolysis and multiple decubitus wounds. Admission examination revealed light touch sensation decreased in bilateral saphenous distributions and right lateral thigh. Hip flexion was 4/5 on both sides. Hip extension, abduction and adduction graded 5/5 bilaterally. Knee extension was 0/5 bilaterally. Dorsiflexion was 4/5 on the right and 5/5 on the left side. Plantarflexion was 5/5 bilaterally. Bilateral ulcerations were present distal to the knees, right elbow and left hand. **Imaging:** MRI of the knees revealed joint effusion with subcutaneous edema without evidence of patellar tendon tears. EMG and NCS findings were indicative of a severe bilateral femoral neuropathy with almost total axon loss and severe demyelination (Figure 1 and 2). Loss of saphenous sensory nerve action potential amplitude confirms this impression. There was evidence for mild right peroneal and tibial neuropathy with minimal axon loss proximally and significant axon loss in the distal area especially in the peroneal nerve. **Course:** The patient failed to make significant recovery in the femoral innervated muscles during her acute inpatient rehabilitation stay. Treatment included KAFO's with free ankle joints that enabled the patient to ambulate and the addition of pregabalin for neuropathic pain management. Goals achieved at discharge include independent with activities of daily living, contact guard to minimum assistance for with transfers, independent with wheelchair mobility and ambulation 80 feet with a rolling walker and bilateral knee ankle foot orthosis and minimal assistance.

Causes of Femoral Neuropathy

Compression within psoas muscle, aortic or iliac aneurysms/tumors	NCS should include sensory studies of the saphenous nerve and motor studies of the femoral nerve.
Diabetic patients have an unusual predilection for femoral/proximal neuropathies	On EMG, the quadriceps should show neuropathic changes
Direct trauma as a result of penetrating wounds or fractures of the hip or pelvis	Results on the symptomatic side should be compared to those on the asymptomatic side.
Lithotomy position during delivery or surgery	The iliopectus is not involved when the lesion is at the inguinal ligament
Iatrogenic causes include direct pressure or trauma to the nerve during pelvic or abdominal surgery. Damage at the femoral triangle due to a difficult femoral line placement.	The adductor magnus and brevis, which share lumbar innervation with quadriceps and iliopectus, are spared since they are innervated primarily by the obturator and sciatic nerves

EMG/NCS findings

Table 1

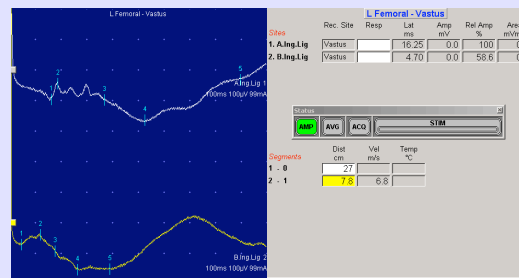


Figure 1

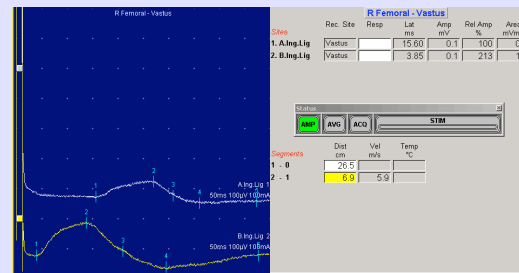


Figure 2

DISCUSSION

Femoral neuropathy was first described by Descartes in 1822 as "anterior crural neuritis." (3) Since that time, femoral nerve injury has been reported with various conditions and procedures, including hip replacement, femoral vessel catheterizations, obstetric and gynecologic procedures, general and urologic surgery explorations, ilioinguinal nerve blocks, and hematologic or neoplastic conditions. (4) The most common cause of femoral neuropathy is often said to be diabetes. Though the femoral nerve is predominately involved in diabetic amyotrophy, there is more widespread denervation process involving the roots and lumbosacral plexus.(5) The femoral nerve is the largest branch of the lumbar plexus and is comprised of the ventral rami of the second, third, and fourth lumbar nerves with occasional contribution from the first lumbar nerve. These branches converge to form the femoral nerve within the psoas muscle where they travel obliquely to emerge laterally between the psoas and the iliacus deep to the iliacus fascia approximately 4 cm superior to the inguinal ligament. The nerve continues into the thigh deep to the inguinal ligament and lateral to the femoral sheath, dividing approximately 4 cm distal to the inguinal ligament into anterior and posterior divisions. The motor branches of the femoral nerve innervate the psoas and iliacus muscles as well as the quadriceps, sartorius, and pectineus muscles. Femoral neuropathy causes weakness of the quadriceps, which results in knee instability during ambulation.(2) The sensory branch of the femoral nerve, the saphenous nerve, innervates skin of the medial thigh and the anterior and medial aspects of the calf. Patients with femoral neuropathy can be treated conservatively with physical therapy, avoidance of excessive hip abduction and external rotation, and knee bracing to prevent buckling of the knee with ambulation.(2) Although the prognosis is favorable and full recovery is usually observed, the disabling effect of the neuropathy may last for several months. With regard to electrodiagnosis, some prognostic value may be assigned. It has been shown in a series of 31 patients with various etiologies for femoral neuropathy that estimates of axonal loss are the only significant prognostic variable. This data demonstrated that in patients with axonal loss estimated at less than 50%, all patients improved within 1 year. In patients with axonal loss greater than 50%, fewer than half of the patients could be expected to improve with conservative management alone.(1)

CONCLUSION

We present an unusual case of bilateral femoral neuropathy attributed to the position of the patients' body during a prolonged loss of consciousness. As with other reported cases of femoral neuropathy the pathology is direct pressure to the nerve by the inguinal ligament when the body is placed in a compromised position. Although case reports exist in field of surgery secondary to lithotomy position no similar cases to the one presented here have been reported. In patients with femoral neuropathy associated with positional compression or retraction compression during surgery or delivery, recovery typically occurs over 3-4 months. The patient should be followed clinically to evaluate for signs of recovery. Electrodiagnostic evaluation should be performed beginning at 6 weeks and repeated at 3 and 6 months after the event to evaluate recovery.(2)

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