

Do Not Overlook Alternative Causes of Foot Drop

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► Most cases of foot drop are an orthopaedic no-brainer and are caused by nerve compression in the lumbar spinal canal, the neuro-foramen, or, occasionally, deep to the piriformis muscle. Still, alternative causes of foot drop are frequently overlooked, leading to delayed diagnosis. This article explores some of those often-missed causes of foot drop and their importance.

The clinical implications of neglected foot drop cannot be overestimated. With foot drop, inability to fully dorsiflex the foot during ambulation results in a steppage gait. In steppage gait, increased flexion of the knee and hip in the swing phase of gait helps toes clear the floor. Inadequate compensatory hip and knee flexion can result in a fall as the toes catch on any raised edge while attempting to clear the floor. As such, fall evaluations should always include looking for a foot drop and its underlying cause.

Differential diagnoses

An acute presentation of foot drop in the emergency department (ED) could result from compartment syndrome secondary to injury or repetitive exertion. If compartment pressures are high, surgical intervention to include a fasciotomy is indicated. Of course, fracture and tendon rupture need to be ruled out in the ED as well. Other compression-type syndromes include compression at the fibular head as the fascia and muscles cross the nerve in a tight space adjacent to the fibula. This particular compression has multiple etiologies, including cyst formation secondary to a lateral meniscus tear, a scar from a previous injury, and instability or arthritic changes in the proximal tibiofibular joint. Although pathologic nerve compression at the fibular head is taught in medical school, clinicians are often guilty of prematurely attributing foot drop to the far more common pathology of lumbar disk herniation.

Chronic conditions should not be ignored during evaluation of foot drop. A known history of diabetes is important to factor into any lower-extremity neurologic concern. Distal symmetric polyneuropathy, a form of diabetic neuropathy, can cause foot drop in its later stages. Early presentation typically includes constant pain and sensory abnormalities. Symptoms typically begin distally and spread proximally. A deficit in the Achilles tendon reflex may also be observed.

Neurologic disorders are also contained in the differential for foot drop.

Symptoms of multiple sclerosis, such as localized lower-extremity weakness, may present as foot drop. Likewise, amyotrophic lateral sclerosis can present with foot drop in its early stages. Understanding that there is a strong genetic component of neurodegenerative pathology, a detailed family history is an important clinical tool in such patients.

Nerve entrapment at the fibular head

Nerve entrapment at the head of the fibula can be caused by bony exostosis formation, scar tissue formation secondary to a proximal fibula fracture or knee surgery, ganglion cyst formation, and discrete thickening of fascia overlying the nerve. The fibular nerve is especially vulnerable to direct injury given its superficial location. Nerve entrapment at the fibular head should also be included in the differential of more complex cases where “double crush syndrome” (DCS) may be a factor. DCS involves injury to a peripheral nerve at more than one location, such as the lumbar region, hip, and/or knee near the fibular head.

Clinical diagnosis of fibular nerve entrapment at the knee includes a positive Tinel’s sign over the path of the fibular nerve. MRI is a useful diagnostic tool in terms of being able to appreciate minor muscle atrophy secondary to denervation, pathology at the fibular head, cysts, fractures, tumors, and osteoarthritis. However, the most useful diagnostic tools in this situation are electromyography and nerve conduction studies (NCS). Related alternatives may have other symptoms. For example, lumbar spine findings include dermatomal pain, positive straight leg raise test, or pain with a Valsalva maneuver. Pain radiating above the knee is not common in fibular nerve compression but may be seen in sciatica and other nerve compression syndromes that originate from higher anatomic locations.

The pathology is direct compression of the nerve with findings of hyperemia above the area of compression and narrowing of the nerve at the sight of the compression. A typical NCS finding is reduction in fibular nerve conduction velocity when recorded at the tibialis anterior, which is the primary dorsiflexor of the ankle. Conduction velocity may also be reduced at the extensor digitorum brevis. Electromyographic reduction of innervation and fasciculations at the tibialis anterior and fibularis longus muscles may also be present.

Treatment

If conservative treatment fails, surgical decompression of the fibular nerve should be considered. Some evidence suggests improvement and often complete resolution of symptoms with fibular nerve decompression (Figs. 1 and 2). A retrospective cohort analysis of surgical decompression performed by Anselmi et al., resulted in 14 of 17 patients achieving a good to excellent resolution in symptoms and 100 percent achieving some form of symptomatic improvement. A similar analysis demonstrated post-surgical resolution of foot drop in 69 percent of patients. Many patients have had symptoms for at least six months

before diagnosis and many for even longer. There are few data about how long you can wait to decompress the fibular nerve. One patient in our series had a cyst removed 20 years ago and had a recurrence caused by continued fibular head instability with nerve recovery after proximal tibial-fibular fusion, removal of the recurrent cyst, and nerve decompression. In comparison, Masud et al., reviewed the timing of carpal tunnel release and noted it is best before six months and less beneficial after a year. It can be inferred that decompression before six months of symptoms may have better outcomes for this peripheral nerve compression syndrome as well.



Fig. 1 Intraoperative image showing the shape of the initial incision to be made for a common fibular nerve decompression procedure
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Fig. 2 Intraoperative image showing the exposed common fibular nerve crossing the neck of the fibula
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Conclusion

When a patient presents with sensory loss in the first web space of the foot, extensor hallucis longus weakness, or a foot drop, workup should be considered for causes other than lumbar compression. In some cases, entrapment at the knee should be suspected. This is especially true when there is local trauma to the knee as part of the history. Examination should be performed to include added palpation of the nerve at the knee and testing for a Tinel's sign at the area where the nerve

crosses the fibular neck. The examiner should also test for proximal tibiofibular movement or instability, look for swelling that may indicate a ganglion cyst near the fibular neck, and test for a lateral meniscus tear that could be associated with a meniscal cyst causing local compression. Neurologic, metabolic, and systemic causes should also be considered.

If there are clinical findings of compression, electromyography is helpful but not always diagnostic. An MRI or ultrasound may be required to rule

out nerve compression due to a proximal cyst. Decompression of the nerve should be considered if the clinical findings indicate the diagnosis. Care should be taken to treat the concomitant aggravating pathology, such as a local cyst, including the cause of the local cyst, namely proximal tibiofibular joint instability or a meniscus tear.

References for the studies cited can be found in the online version of this article, available at www.aaosnow.org.

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Surgical technique for nerve decompression at the fibular head

Surgical decompression of the fibular nerve begins by arranging the patient in a supine position with the knee flexed between 40 degrees and 60 degrees. Placement of the incision first requires identification of the fibular head. The common fibular nerve traverses the fibular head in a lateral to medial direction before bifurcating into the deep and superficial fibular nerves. Mark an incision that crosses the fibula just below the nerve to avoid the risk of nerve injury and to leave a soft-tissue sleeve over the nerve once the decompression is complete (Fig. 3a). The mark should be extended proximally in the direction of the biceps femoris and distally over the anterior compartment of the leg in the direction of the muscle fibers, roughly achieving a final "Z" shape. The authors prefer sharp corners in the incision lines to create a more cosmetic closure, aid in aligning the skin after nerve release, and decrease scar widening as it is near the knee joint.

After opening the skin at the incision site, use standard soft-tissue dissection to find the adipose tissue around the nerve (Figs. 3b and 3c). More proximally,

tear a few centimeters above the biceps femoris insertion on the fibular head. The nerve is more mobile here, so there is less risk of accidentally injuring it. Next, continue the dissection following the nerve itself as it runs from posterior to anterior starting just below the biceps femoris tendon. Follow the path of the nerve, dissecting with a small,

blunt-tipped scissor, until it crosses the neck of the fibula (Figs. 3d and 3e). At this point, there are usually dense fibrous bands crossing the nerve that need to be released. Muscle fibers often also cross the nerve as it descends into the muscles of the anterior compartment of the leg. These too can be a cause of compression and need to be carefully

released. The nerve will split into multiple branches 1 cm to 2 cm past the end of its course over the fibular neck and dive as separate branches into the muscle. Expose these distal branches for only a short distance to avoid damaging them. Leave the fascia layer open and close only the fat and skin to complete the procedure. (Figs. 3f and 3g).



Fig. 3 Surgical decompression of the fibular nerve: (A) initial "Z" type incision, (B) exposure of the overlying fascia, (C) identification of the nerve just below the fibular head before it crosses the fibular neck, (D) following the nerve proximally when it is not tethered and the soft tissues are more forgiving, (E) releasing fascia over nerve, (F) exposing the area of compression, (G) fully decompressed fibular nerve from proximal to distal until it splits into smaller branches

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