Common peroneal nerve palsy

see also Peroneal Nerve Entrapment.

A peroneal nerve injury (also called foot drop or drop foot), is a peripheral nerve injury that affects a patient's ability to lift the foot at the ankle.

The peroneal nerve is the most common nerve to develop acute compression palsy.

Functional anatomy

The sciatic nerve (L4-S3) consists of 2 separate nerves within a common sheath that separate at a variable location in the thigh (the peroneal division of the sciatic nerve is more vulnerable to injury than the tibial division)

1. posterior tibial nerve, or just tibial nerve (AKA medial popliteal nerve) which provides for foot inversion among other motor functions

2. common peroneal nerve (CPN), or just peroneal nerve (AKA lateral popliteal nerve):high injuries may involve the lateral hamstring (short head of the biceps femoris) in addition to the following.

The CPN passes behind the fibular head where it is superficial and fixed, making it vulnerable to pressure or trauma (e.g. from crossing the legs at the knee). Just distal to this, the CPN divides into:

a) deep peroneal nerve (AKA anterior tibial nerve): primarily motor

● motor: foot and toe extension (extensor hallicus longus (EHL), anterior tibialis (AT), exten- sor digitorum longus (EDL))

● sensory: very small area between great toe and second toe b) superficial peroneal nerve (AKAmusculocutaneous nerve)

● motor: foot eversion (peroneus longus and brevis)

● sensory: lateral distal leg and dorsum of foot

Etiology

While foot drop injury is a neuromuscular disorder, it can also be a symptom of a more serious injury, such as a nerve compression or herniated disc.

The most frequent cause of serious peroneal nerve injury is knee injury ± fracture.

1. entrapment as it crosses the fibular neck or as it penetrates the peroneus longus

2. diabetes mellitus and other metabolic peripheral neuropathies

3. inflammatory neuropathy: including Hansen's disease (leprosy)

4. traumatic: e.g. clipping injury in football players, stretch injury due to dislocating force applied to the knee, fibular fracture, injury during hip or knee replacement surgery

5. penetrating injury

6. masses in the area of the fibular head/proximal lower leg: popliteal fossa cysts (Baker cyst), anterior tibial artery aneurysm (rare)

7. pressure at fibular head: e.g. from crossing the legs at the knee, casts, obstetrical stirrups…

8. traction injuries: severe inversion sprains of the ankle

9. intraneural tumors: neurofibroma, schwannoma, neurogenic sarcoma, ganglion cysts

10. vascular: venous thrombosis

11. weight loss

Foot drop injury can be caused by an injury to the spinal cord or from other underlying diseases, such as amyotrophic lateral sclerosis (ALS), multiple sclerosis (MS), or Parkinson's disease. Sometimes, drop foot is a complication from hip replacement surgery, or other injuries (e.g., knee or joint dislocation or fracture, herniated disc).

A common peroneal nerve (CPN) palsy has been reported to complicate knee dislocations in 5-40 % of patients. Patients who suffer from a persistent foot drop have significantly worse functional outcomes.

Clinical features

1. sensory changes (uncommon): involves lateral aspect of lower half of leg

2. muscle involvement:

Common peroneal nerve palsy (most common) produces weak ankle dorsiflexion (foot drop) due to anterior tibialis palsy, weak foot eversion, and sensory impairment in areas innervated by deep and superficial peroneal nerve (lateral calf and dorsum of foot). There may be a Tinel’s sign with percussion over the nerve near the fibular neck. Occasionally, only the deep peroneal nerve is involved, resulting in foot drop with minimal sensory loss. Must differentiate from other causes of foot drop.

Examination/clinical correlation

● Buttock level injury: unless the injury is one that permits spontaneous regeneration, prognosis is poor for return of peroneal nerve function even with surgery

● thigh level injury: also di cult to get improvement with surgical repair. Some peroneus function may occur at ≥6 mos, early contraction of AT may take ≥1 yr

● knee level injury: with successful regeneration, peroneus contraction may begin by 3–5 months. First signs: quivering of muscle lateral to the proximal fibula on attempted foot eversion, or tightening of tendon posterior and behind the lateral malleolus on attempted ankle dorsiflexion.

Evaluation EMG

EMG takes 2–4 weeks from the onset of symptoms to become positive. Stimulate above and below fibular head for prognostic information: if absent in both sites, the prognosis is poor (indicates retrograde degeneration has occurred). Wallerian degeneration takes ≈ 5 days to cause deterioration. In addition to the expected findings ofdenervation – PSWs & fibs – in the anterior tibialis, evaluate:

1. L5 innervated muscles outside the distribution of the common peroneal nerve: a) posterior tibialis b) flexor digitorum longus

2. L5 muscles whose nerve originates above the knee (these muscles are spared in cases of compression of the peroneal nerve at the fibular head due to the fact that the nerve take off is proximal to the popliteal fossa):

a) biceps femoris (short or long head)

b) tensor fascia lata

3. paraspinal muscles: signs of denervation solidifies the location of the lesion as nerve root; not helpful if negative

MRI

May demonstrate causes such as tumor or a ganglion cyst arising from the superior tibiofibular articulation.

Outcome

A vastly different prognosis can be expected for patients who suffer an incomplete versus a complete CPN palsy. The majority of patients with an incomplete palsy will achieve a full motor recovery while <40 % of patients with a complete motor palsy will regain the ability to dorsiflex at the ankle. While neurologic interventions show promise for the future, the outcomes in knee dislocation patients remain poor. The most predictable means of reestablishing antigravity dorsiflexion in a persistent CPN palsy is a posterior tibial tendon transfer [1)]

Treatment

Patients in whom conventional peroneal nerve repair surgery failed to reconstitute useful foot lift need to be evaluated for their suitability to undergo a concomitant tendon transfer procedure or nerve transfers.

When treatment can eliminate a reversible cause, the outcome is usually good. Surgical exploration and decompression may be considered when there is no reversible cause or when improvement does not occur.

Nonsurgical management

Bracing: ankle-foot-orthosis (AFO) compensates for loss of ankle dorsiflexion which inserts unobtrusively into a shoe. If this is inadequate, or to stabilize the ankle, a spring-loaded kick-up foot brace built into a shoe may be used. The patient should be instructed in techniques to avoid contracture of the Achilles tendon (heel cord) which would impair ankle dorsiflexion if nerve function returns.

Surgical management

At the level of the popliteal fossa the skin incision is made just medial to the tendon of the short head of the biceps femoris (lateral hamstring) as the peroneal nerve is best located deep to or slightly medial to this tendon. The incision is carried distally slightly laterally along the surgical neck of the fibula. The biceps femoris is retracted laterally and the nerve is isolated and tagged with a Penrose drain. The sensory sural nerve branches o the peroneal nerve at variable sites ranging from the sciatic portion of the nerve (proximal to the flexor crease) or distal to this.

In cases of compression, the fascia from the lateral gastrocnemius and soleus overlying the nerve distal to the fibular head is lysed and the nerve is exposed in 360°. As the nerve crosses the fibular neck it divides into superficial and deep branches. The superficial branch travels directly distally to supply the peroneus longus and brevis (foot evertors). The deep branches curve anteriorly to the anterior tibialis, EHL, and toe extensors.

If a graft is needed, the contralateral sural nerve is usually used, which may be supplemented with the ipsilateral sural nerve if needed.

Case series

Between 2007 and 2013, 8 patients were operated on for foot drop after unsuccessful nerve surgery. Six patients without fatty degeneration of the anterior tibial muscle and proximal lesion of the peroneal nerve were oriented for tibial to peroneal nerve transfer. In the other 2 cases where the anterior and lateral compartments were destructed, the anterior tibial muscle function was reconstructed with a neurotized lateral gastrocnemius transfer. For each patient, we graded postoperative results using the British Medical Research Council scheme and the Ninkovic assessment scale.

Of the 6 patients who underwent nerve transfer of the anterior tibial muscle, 2 patients had excellent results, 1 patient had good results, 1 patient had fair results, and 2 patients had poor results. Of the 2 patients that underwent neurotized lateral gastrocnemius transfer, 1 patient achieved excellent results after tenolysis, whereas 1 patient achieved poor results. After the nerve transfer, 5 patients did not wear an ankle-foot orthosis. Four patients did not limp. Four patients were able to walk barefoot, navigate stairs, and participate in activities.

Early clinical results after tibial to peroneal nerve transfer and neurotized lateral gastrocnemius

transfer appear mixed. The results of nerve transfer seem, on the whole, less reliable than the literature reports on tendon transfer [2].

Common peroneal nerve (CPN) palsy following knee dislocations in a twelve-year surgical series of 26 patients presenting with a traumatic injury of the lateral sciatic nerve and no spontaneous recovery were reviewed from 1988 to 1991, and performed nerve surgery alone on 3 patients. Their results were highly disappointing and in none did we observe muscle recovery. Since 1991 nerve surgery was associated with a palliative procedure for 23 patients. Although at surgical exploration, severe nerve damage was found in 87% of these patients (thereby indicating the need for graft repair), the overall outcome was good, with a score of M3 on the BMRC scale in about 75% of the cases. These results suggest that the one-stage association of microsurgical nerve repair and tibialis posterior tendon transfer changed the destiny of these injuries [3].

Case reports

High-Resolution Ultrasound as a Powerful Diagnostic Tool in Peripheral Nerve Lesions: Detection of an Intraneural Ganglion Cyst in a Patient with Painful Subacute Peroneal Nerve Palsy [4].

A 25-year-old white man, right after bilateral rhytidoplasty, presented with agitation, necessitating use of haloperidol. Some hours after, he developed severe pain in his legs and a diagnosis of neuroleptic malignant syndrome (NMS) was considered. Even with treatment for NMS he still complained of pain. A diagnosis of lower limb compartment syndrome (CS) was done only 12 hours after the initial event, being submitted to fasciotomy in both legs, disclosing very pale muscles, due to previous ischemia. This syndrome was not explained only by facial surgery, his position and duration of the procedure. It can be explained by a sequence of events. He had a history of pain in his legs during physical exercises, usually seen in chronic compartment syndrome. He used to take anabolizant and venlafaxine, not previously related, and the agitation could be related to serotoninergic syndrome caused by interaction between venlafaxine and haloperidol. Rhabdomyolysis could lead to oedema and ischemia in both anterior leg compartment. This report highlights the importance of early diagnosis of compartment syndrome, otherwise, even after fasciotomy, a permanent disability secondary to peripheral nerve compression could occur [5].

References

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