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Sport related injuries of the nerves in the knee region

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Abstract. Although peripheral nerve lesions are quite rare in sport injuries, they can be a career ending cause and have a serious impact on the general quality of life of the former sportsman. Therefore, it is imperative that nerve lesions be diagnosed promptly, allowing early intervention prior to irreversible loss of nerve function. That's why sports medicine physicians must have a deep knowledge of peripheral nerve anatomy and physiology in order to diagnose the level of injury accurately and provide appropriate treatment and prognostic information to the patient.
Key words: *knee injuries, nerve lesions, compression, traction, laceration.*

Introduction

Nerves can be injured through a variety of mechanisms. The lesions can be acute or chronic. The main mechanism involved in sport injuries of the nerves is compression followed by ischemia (1). Pressure seems to be the responsible agent of injury with chronic entrapment of nerves, although ischemia may also play a small role.

Acute nerve injury may cause damage when applying high pressure for a short period of time (e.g., nerve injury from a fracture). In addition, a rapid reversible physiologic nerve impairment can occur when a high pression is applied to the nerve for a small period of time (e g. cross-legged sitting, causing numbness and paresthesia). This condition is mostly caused by ischemia. In the 6 hours interval, the restoration of nerve function is complete. After more than 8 hours of ischemia, the nerve lesion is irreversible, due to the axonal infarction. An acute compression may be caused by an ill-fitting equipment (2).

Chronic nerve injuries cause lesions by small or moderate compression for a long period of time or moderate pressure applied constantly on the same region, the last one being a condition which defines a professional sportsman.

Chronic compression may be done by an abnormal muscular band, or tendon or by a bony prominence which dimensions or development were exaggerated by sport practice (2--4).

Another nerve injury mechanism, quite frequent in sports, is traction. The elasticity of the nerves is due to the collagenous endoneurium, which allows a nerve to stretch 10-20% without significant structural or functional damage. When the nerve is stretched beyond his stretching capacity (elongation between 30 and 70%), injuries occur (1, 5, 6).

Not only the value of the tensile force applied is important, but also the rate of application which affects the degree of injury. Specialists report that a slow stretch applied for several years can elongate a nerve without functional impairment. But if a strong stretching force is applied acutely, complete transection may occur (5, 7, 8).

A rare mechanism of nerve lesions involved in sport injuries is laceration, in which the nerve continuity may be preserved or, more serious, the transection is complete and the two nerve fragments are apart from each other.

Transection is followed by apoptosis and cell death. The sensitive neurons are more susceptible to death than the motor ones. Cellular loss in the sensitive ganglia from the dorsal root of the spinal nerves may reach up to 50%.

Factors that influence this process are age, distance from the lesion to the spinal cord and the time passed between the occurrence of the injury and the surgical repair (1, 9, 10).

Nerves that can be injured in sport accidents involving the knee are the saphenous nerve, the common peroneal nerve and the tibial nerve.

The saphenous nerve is a cutaneous branch of the femoral nerve, the only branch that accompanies the femoral artery in the femoral triangle of Scarpa and the adductor canal (Hunter's canal). In the femoral triangle the nerve is placed laterally from the femoral vessels and, after crossing the femoral artery, in the adductor canal it is placed medially. The nerve exits the canal and emerges between the tendons of gracilis and sartorius muscles, then gives off the infrapatellar branch for the skin which covers the patella, then descends on the medial side of the tibia, along with the saphenous vein and ends in two terminal branches, the medial cutaneous branches of the calf.

The most susceptible place for the saphenous nerve to be injured is where it pierces the vastoadductor membrane along with the descendant genicular branch of the femoral artery. The causes for this are the sharp angle of its trajectory, the relations with the pes anserinus muscles and the repetitive shearing forces which act at the knee joint in sportsmen (11).

The saphenous nerve can also be injured iatrogenically during a knee arthroscopy, aspirations or infiltrations (12, 13). Saphenous lesions typically present with deep, aching pain and occasionally paresthesia in the distribution territory of the nerve. Pain can be localized anywhere along the course of the nerve, most often in the medial region of the knee or calf. Consequently, saphenous neuropathy can mimic various musculoskeletal complaints, such as a medial meniscal tear in the knee, pes anserine bursitis, medial tibial stress syndrome, or stress fracture.

Symptoms get worse with knee flexion or with compression from tight equipment or braces. The differential diagnosis should include L4 radiculopathy, lumbar plexopathy, or a more proximal femoral neuropathy.

Physical examination may show a well demarcated area of altered sensation within the distribution of the saphenous nerve, symptom provocation with passive thigh hyperextension, and pain or paresthesia with deep palpation proximal to the medial femoral condyle.

Because the saphenous nerve is purely sensory, there should be no associated weakness. Any weakness, atrophy, or loss of the quadriceps deep tendon reflex requires further evaluation for a more proximal nerve lesion.

Once the diagnosis has been established, the treatment is symptomatic. If the pain is the main complaint, a local anesthetic and steroid injection may be both diagnostic and therapeutic.

In athletes, elimination of any provocative factors such as tight clothing or equipment along the course of the saphenous nerve is important. Surgical release is rarely indicated, but occasionally excision of a neuroma is necessary.

The common peroneal nerve is the lateral terminal branch of the sciatic nerve, which emerges at the superior angle of the popliteal fossa. Then it descends parallel to the biceps femoris tendon, then passes beneath this tendon and directs anteriorly and laterally passing posterior and then encircling the fibular neck. It then runs deep to the proximal part of the peroneus longus muscle, between the muscle and the peroneal bone, then it divides into two terminal branches: the superficial peroneal nerve and the deep peroneal nerve.

The common peroneal nerve is the most frequently involved nerve in lower limb sport-related injuries. Because of its intimate relations with the fibula, the common peroneal nerve is susceptible to both compression and tension/stretch mechanisms. Compressions occur in case of a direct lesion near the fibular head or neck, usually encountered in soccer, football or hockey (14).

In athletics, due to an overdevelopment of the peroneus longus, repetitive and chronic compression of the nerve may occur. At the level of the knee joint, chronic nerve injury can be caused by hypermobility of the fibular head (15-17).

Stretch injuries of the common peroneal nerve can be seen in a large variety of knee injuries, such as dislocations, fractures, and ligamentous disruptions. Typically, this mechanism of injury acts when a varus stress is applied across the knee joint with the foot planted.

Occasionally, the nerve is injured in this manner in the absence of any ligamentous or bony lesions. Knee dislocations, which are typical for high-velocity trauma or contact sports, have a high incidence of peroneal nerve injuries (18- 20).

Peroneal neuropathy can occur after minor trauma, typically in the same region of the fibular head and neck, because in that place, the nerve is

more firmly tethered. The axon loss can result from a proper lesion of the nerve, or from a compression hematoma or from ischemia due to vasa nervorum rupture.
Because of the relative superficial location of the nerve at the fibular neck, an injury can be caused even by direct application of ice on that area (21).
As in saphenous nerve lesions, the common peroneal nerve can be injured iatrogenically, during arthroscopy or open knee surgery (22).
Owing to the topography of the nerve fibers at the level of the fibular neck, with the deep fibers lying more anterior, many of the same mechanisms of injury that produce a common peroneal neuropathy can result in an isolated proximal deep peroneal neuropathy (1, 15).
Foot drop is a common and important manifestation of peroneal nerve injury. This condition results from the loss of motor innervations of the tibialis anterior muscle and causes significant gait impairment and disability. The complaint of paresthesia over the anterolateral leg, foot, or first interosseous space is very common in athletes.
Injury to the common peroneal nerve or to both its superficial and deep branches causes weakness of the deep peroneal nerve dorsiflexors - the anterior tibialis and peroneus tertius and toe extensors - extensor digitorum brevis and longus and the extensor hallucis brevis and longus, which extend the second through fifth toes and the great toe, respectively.
With proximal involvement of the deep peroneal nerve, the athlete complains of foot slap or catching of the toes, which tends to be worse when walking barefoot (1).
There is also weakness of the peroneus longus and brevis muscles, innervated by the superficial peroneal nerve. Because the common peroneal nerve and its two terminal branches are mixed nerves, the motor impairment is associated with sensory symptoms. Deep peroneal injury results in decreased sensation in the first interosseous space of the foot, while superficial peroneal involvement results in paresthesia in the anterolateral calf and the dorsum of the foot.
The differential diagnosis in a patient with peroneal lesions should include more proximal nerve involvement (sciatic nerve, lumbosacral plexus), as well as compartment syndromes. With an isolated peroneal neuropathy in the absence of trauma or compression, nonathletic causes should be kept in mind, such as inherited neuropathy.
If there is no sign of sensory loss associated with foot drop, motor neuron disease should also be considered, particularly if the painless weakness progresses to involve other nerves or limbs.
Treatment of peroneal nerve injuries depends on the etiology and severity of the injury. If compression is present, it must be removed, including surgical release. In the case of a stretch injury, observation is needed, with surgical intervention following only in the case of a severe traction injury with complete or nearly complete axon discontinuity. While awaiting recovery, it is important to protect the nerve (by splinting or bracing) and to strengthen the involved muscles gradually. Proprioceptive retraining of the ankle muscle groups can be helpful for avoiding repetitive nerve injury related to weak ankle evertors and recurrent ankle sprain.
Nerve regeneration following common peroneal nerve repair is poor compared with other peripheral nerves and this can explain the uncertain attitude of many specialists toward exploration and repair of this nerve.
One factor explaining the poor outcome of reinnervation might be the imbalance between the functioning flexors and the weakly innervated extensors that can result in fixed varus equinum of the foot with associated Achilles tendon shortening (1).
Other surgical options besides direct nerve repairs include nerve transfers and tendon transfers. Nerve transfer involves taking a branch from a less important lower leg muscle in the tibial distribution and connecting it to nerve to the muscle that lifts the foot in the peroneal distribution.
A tendon transfer involves taking a tendon that moves the foot inward, and connecting it to the top of the foot so that it now lifts the foot upwards and outwards, thereby resolving the foot transfers to deep peroneal nerve have been reported in the literature.
The tibial nerve is the medial terminal branch of the sciatic nerve, which usually emerges at the same level as the common peroneal nerve, in the popliteal fossa. In this region it forms, together with the popliteal vessels, the neurovascular bundle. It then passes underneath the soleus muscle and continues between the two layers of posterior crural muscles, between the posterior tibial and flexor digitorum longus muscles. At the ankle, it divides into two terminal branches, the lateral and medial plantar nerves.

Because of its deeper location, the tibial nerve is less vulnerable to sport injuries than the common peroneal nerve. The most frequent injuries occur together with knee dislocation and severe injuries of the posterior part of the articular capsule. Often, this type of injury is associated with lesions of the popliteal vessels (23).

At the knee level, the nerve can be compressed by space-occupying lesions in the popliteal fossa, such as haematoma or Baker's cyst, or even a popliteus muscle rupture, as reported in literature (24). At the same level, the tibial nerve can be injured in a fracture of the tibial shaft with posterior displacement of the bone fragments (25). Symptoms related to tibial nerve injury vary depending on the level of the injury. If the nerve is injured at the knee level, the patient develops weakness of the plantar flexors, invertors, toe flexors, and intrinsic foot musculature (tibialis posterior, flexor hallucis and digitorum longus). This weakness, prolonged in time, can lead to clawing of the toes and foot deformity.

Pain and paresthesia may occur in the posterior calf (medial sural cutaneous nerve) or lateral aspect of the foot (sural nerve) if the injury occurs above the level at which these nerves branch from the tibial nerve and/or the plantar aspect of the foot (medial and lateral plantar and medial calcaneal nerves).

The differential diagnosis includes S_1 radiculopathy, sciatic neuropathy, and lumbo-sacral plexopathy. In the case of an isolated tibial neuropathy, it is important to consider nonathletic causes of nerve injury such as an inflammatory processes or an inherited neuropathy.

Treatment of tibial neuropathy depends on the cause of injury. In the case of nerve laceration, immediate exploration and repair may be indicated. In case of entrapment and compression, immediate relief, which may require surgical intervention, is recommended. Traction injuries are usually best managed conservatively with observation, protection of the ankle joint, maintenance of flexibility, ankle proprioceptive retraining, and calf and intrinsic foot muscle strengthening.

If there is significant weakness of the gastrocnemius and soleus muscles, a foot orthosis may be helpful in stabilizing the ankle (1). If the pain is significant, it can be treated with neuropathic pain medication, such as tricyclic antidepressants or membrane-stabilizing agents such as gabapentin. Topical neuropathic pain medications can also be helpful.

References

1. Akuthota V, Herring SA (2009). *Nerve and vascular injuries in sports medicine*. Springer.
2. Lundborg G, Rydevik B (1973). Effects of stretching the tibial nerve of the rabbit: a preliminary study of the intraneural circulation and the barrier function of the perineurium. *J Bone Joint Surg Br*; 55(2): 390–401.
3. Akuthota and E Maslowski (1973). Circulation and the barrier function of the perineurium. *J Bone Joint Surg Br*; 55(2):390–401.
4. Dawson DM, Hallett M, Wilbourn AJ, eds (1999). *Entrapment Neuropathies*. 3rd ed. Philadelphia: Lippincott Raven.
5. Lorei MP, Hershman EB (1993). Peripheral nerve injuries in athletes: treatment and prevention. *Sports Med*; 16(2): 130–47.
6. Sunderland S (1961). Stress-strain phenomena in human peripheral nerve trunks. *Brain*; 84:102–19.
7. Sunderland S (1990). The anatomy and physiology of nerve injury. *Muscle Nerve*; 13(9): 771–84.
8. Grant GA, Goodkin R, Kliot M (1999). Evaluation and surgical management of peripheral nerve problems. *Neurosurgery*; 44(4): 825–39; discussion 39–40.
9. Burnett MG, Zager EL (2004). Pathophysiology of peripheral nerve injury: a brief review. *Neurosurg Focus*; 16(5): E1.
10. Schon LC (1994). Nerve entrapment, neuropathy, and nerve dysfunction in athletes. *Orthop Clin North Am*; 25(1):47–59.
11. Hemler DE, Ward WK, Karstetter KW, et al (1991). Saphenous nerve entrapment caused by pes anserine bursitis mimicking stress fracture of the tibia. Arch Phys Med Rehabil; 72(5): 336–7.
12. Logue EJ, DrezDJr (1996). Dermatitis complicating saphenous nerve injury after arthroscopic debridement of a medial meniscal cyst. *Arthroscopy*; 12(2):228–31.
13. Iizuka M, Yao R, Wainapel S (2005). Saphenous nerve injury following medial knee joint injection: a case report. *Arch Phys Med Rehabil*; 86(10): 2062–5.
14. Leach RE, Purnell MB, Saito A (1989). Peroneal nerve entrapment in runners. *Am J Sports Med*; 17(2): 287–91.
15. Moller BN, Kadin S (1987). Entrapment of the common peroneal nerve. *Am J Sports Med*; 15(1): 90–1.

16. Seckler MM, DiStefano V (1996). Peroneal nerve palsy in the athlete: a result of indirect trauma. Orthopedics; 19(4):345–7; discussion 7–8.
17. Streib EW (1983). Traction injury of peroneal nerve caused by minor athletic trauma: electromyographic studies. *Arch Neurol*; 40(1):62–3.
18. Wascher DC, Dvirnak PC, DeCoster TA (1997). Knee dislocation: initial assessment and implications for treatment. *J Orthop Trauma*; 11(7):525–9.
19. Malizos KN, Xenakis T, Mavrodontidis AN, et al (1997). Knee dislocations and their management: a report of 16 cases. *Acta Orthop Scand Suppl*; 275:80–3.
20. Putukian M, McKeag DB, Nogle S (1995). Noncontact knee dislocation in a female basketball player: a case report. *Clin J Sport Med*; 5(4):258–61.
21. Peicha G, Pascher A, Schwarzl F, et al (1998). Transection of the peroneal nerve complicating knee arthroscopy: case report and cadaver study. *Arthroscopy*; 14(2): 221–3.
22. Moeller JL, Monroe J, McKeag DB (1997). Cryotherapyinduced common peroneal nerve palsy. Clin J Sport Med; 7(3): 212–6.
23. Dyck PJ, Thomas PK (2005). Peripheral Neuropathy. 4th ed. Philadelphia: Elsevier Saunders.
24. De Ruiter GC, Torchia ME, Amrami KK, et al (2005). Neurovascular compression following isolated popliteus muscle rupture: a case report. *J Surg Orthop Adv*; 14(3):129–32.
25. Krivickas LS (2003). Lower-extremity nerve injuries. In: Feinberg JH, Spielholz NI, eds. *Peripheral Nerve Injuries in the Athlete*. Champaign, IL: Human Kinetics; 106–41.

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