Transient superficial peroneal nerve palsy after anterior cruciate ligament reconstruction

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Abstract

A 19-year-old male subject was diagnosed with medial meniscal, lateral meniscal and anterior cruciate ligament (ACL) tear. The symptoms did not subside after 4 months of physical therapy, and he underwent arthroscopic partial medial and lateral meniscectomy and ACL reconstruction. Immediately after the patient woke up from general anesthesia, he started experience loss of sensation in the area of superficial peroneal nerve with inverted dorsiflexion of foot and ankle. Instantly, the bandage and knee brace was removed and a diagnosis of compartment syndrome was ruled out. After eight hours, post-operatively, the patient started receiving physiotherapy. He complained of numbness and tingling in the same area. After 24 h, post-operatively, the patient started to regain dorsiflexion and eversion gradually. Two days after the surgery, the patient exhibited complete recovery of neurological status.

Introduction

Anterior cruciate ligament (ACL) reconstruction is a surgical tissue graft replacement of the ACL, located in the knee, to restore its function after ACL injury. The torn ligament is removed from the knee before the graft is inserted. The surgery is performed arthroscopically (Figure 1).

The superficial peroneal nerve (SPN) supplies the muscles of the lateral compartment of the leg namely: peroneus longus and peroneus brevis. These two muscles assist with eversion and plantar flexion of the foot. Peroneal nerve palsy is a paralysis that affects patient's ability to lift the foot at the ankle. SPN palsy is relatively rare[1-8] and only a few bilateral cases have been reported in the literature.[9,10] SPN involvement in ACL reconstruction alone has not been described in the literature. Direct injury of the SPN in the lower leg has also been reported.[11] In the lower extremity, peroneal neuropathy is the most common isolated mononeuropathy. It is the third most common mononeuropathy overall.

Case Report

A 19-year-old male subject with an unremarkable medical history was treated in the Orthopedic Clinic of the local hospital (Arar, Saudi Arabia) for a painful right knee with severity 2/10 and instability after recreational sports activity. He was diagnosed with a tear of medial meniscus, lateral meniscus and ACLs, which was later confirmed by magnetic resonance imaging. The symptoms did not subside after 4 months of physical therapy. He later underwent arthroscopic partial medial and lateral meniscectomy and ACL reconstruction.

Immediately after the patient woke up from general anesthesia, he noticed a loss of sensation over the lateral calf and dorsum of the right foot except the 1st web space with inverted dorsiflexion of foot and ankle. Physical examination revealed sensory deficits on the lateral calf and dorsum of the right foot except the 1st web space and motor deficit of the peroneus longus and brevis muscle with normal reflexes. Examination of the lumbar spine and lower limbs revealed no clinical abnormalities in the joints and there was neither suspicion of nerve root compression at the level of the lumbar spine nor nerve entrapment at the neck of the fibula. Radiographic examination of the lumbar spine, knee, legs and feet were normal. Instantly the clean non-bloody bandage and knee brace were removed and a diagnosis of compartment syndrome was ruled out. Eight hours post-operatively, the patient started physiotherapy when he experienced numbness and tingling in the same area. Twenty-four hours post-operatively, he started to regain his dorsiflexion and gradually the eversion. Two days after the surgery, he completely recovered his neurological status.

Discussion

The SPN arises from the common peroneal nerve at the neck of the fibula and runs down the lateral compartment of the leg. It travels between the peroneus *longus* and *brevis*, the primary ankle evertors and innervates both muscles (Figure 2).[12] The peroneus *tertius* and extensor *digitorum longus* also contribute to ankle eversion. Segmental innervation is the same as for the deep peroneal nerve. Near to the midpoint of the lateral lower leg, the nerve becomes subcutaneous. It supplies sensation to the skin of the lateral leg and the dorsum of the foot and toes, sparing the small area between the first two toes and a variable lateral part of the foot.[13] Fifteen to twenty-eight percent of patients have an accessory peroneal nerve that branches off at the superficial peroneal to supply the extensor *digitorum brevis* (usually innervated by the deep peroneal).[14-16]

The most common complications following ACL ligamentoplasty recorded in recent literature[17,18] include re-admission (1.36-2.3%), complications of the surgical wound (0.75%), deep venous thrombosis (0.44%), septic arthritis (0.14-1.7%), and re-intervention (0.25%), although no data relating to emergency re-evaluations is available.

Transient acute neurapraxia usually involves a traumatic episode (*e.g.,* stretching or compression of the nerve), whilst less common mechanisms of injury may include percussion and vibration, thermal or electrical shock, inflammation and ischemia.[19] Entrapment of the SPN has traumatic and non-traumatic causes. Local trauma and compression are the most common causes of nerve entrapment. This may be due to recurrent stretch injuries or certain positions like prolonged kneeling and squatting, which cause perineural fibrosis.[19,20] Edema after trauma may result in a mini compartment syndrome which may occur when the tunnel was fibrotic, of low compliance and longer than 3 cm.[3] Chronic or exertional lateral compartment syndrome can also cause compression of the SPN, particularly in athletes.[20,21] Previous case reports have documented both unilateral and bilateral foot drop after childbirth due to direct external compression from grasping at the fibular head or from prolonged forceful knee flexion.[22,23] Fasciotomy of the anterior compartment for chronic anterior compartment syndrome may also cause compression of the SPN nerve.[20] Non-traumatic causes of SPN entrapment are commonly due to anatomical variations such as fascial defects, with or without

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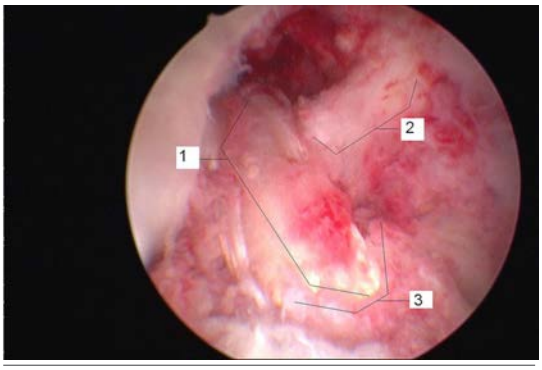

Figure 1. Arthroscopic anterior cruciate ligament reconstruction (right knee): (1) anterior cruciate ligament reconstruction by semitendinosus tendon autograft; (2) posterior cruciate ligament; and (3) tibial remnant of the original injured anterior cruciate ligament.

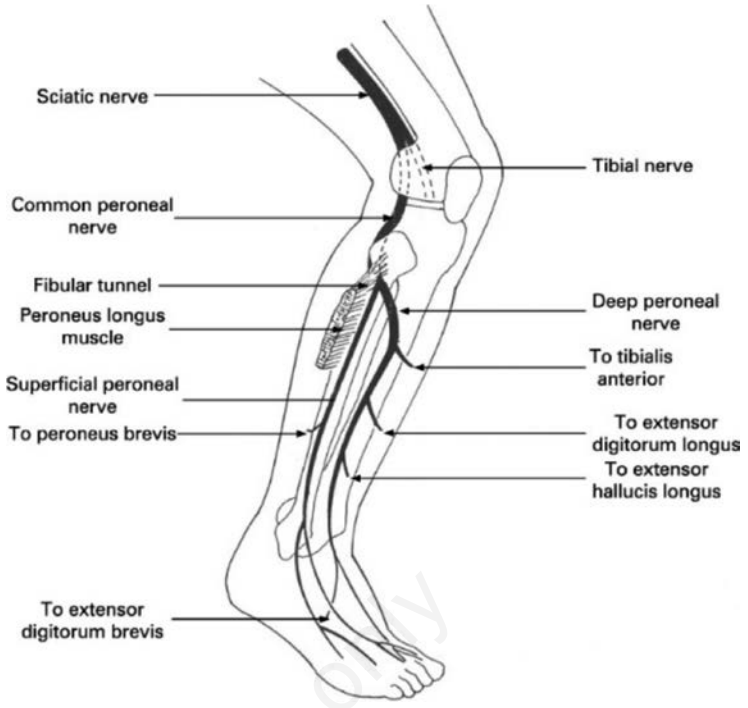

Figure 2. Nerve supply of the calf. Reproduced with permission from *Focal peripheral neuropathies* (3rd edition) by John D. Stewart, 2000.[12]

muscle herniation about the lateral lower leg, where the nerve is entrapped as it emerges into the subcutaneous tissue or a short peroneal tunnel proximally. Nerve compression in patients with fascial defects is explained by the normal increase in muscle relaxation pressure and intramuscular pressure at rest during and after exercise. This increase is sufficient to cause herniated muscle tissue and this can impinge upon or compresses the nerve.[21]

Conclusions

In the absence of any other causative factors, it was concluded that a temporary compression of the SPN at the level of the knee or below produced transient neurapraxia by direct external compression, which was performed by the grasping of the fibular head or mid-calf area or from prolonged forceful knee flexion during ACL reconstruction.

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