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Peroneal nerve palsy due to rare reasons: a report of three cases

Üç olguda nadir nedenlerle oluşan peroneal sinir felci

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Peroneal sinir yaralanması görülen üç hastada (2 erkek, 1 kadın) klinik bulgular ve elektrofizyolojik çalışmalar ile sinir sıkışması tanısı kondu. Bir olguda uzun süre çömelmeye bağlı iki taraflı postural tipte sıkışma, diğer iki olguda ise tek taraflı mekanik tipte sıkışma vardı. İlk olarak, düşük ayak bileği splinti ile birlikte B vitamini verilerek konservatif tedavi uygulandı. Bir olgu konservatif tedaviyle iyileşti. İki taraflı bir olguda sağ taraf iyileşirken, sol taraf tedaviye yanıt vermedi. Üç ay sonra klinik ve elektrofizyolojik olarak düzelme saptanmayan iki hastanın iki ekstremitesine cerrahi tedavi ile dekompresyon uygulandı. İki taraflı tutulumu olan olguda cerrahi tedaviyle başarılı sonuç alındı; diyabeti olan bir hastada ise düzelme görülmedi.

Anahtar sözcükler: Ayak bileği yaralanmaları; dekompresyon, cerrahi; elektromiyografi; sinir sıkışma sendromu/etyoloji/cerrahi; paralizi/etyoloji/cerrahi; peroneal sinir/yaralanma/cerrahi; peroneal nöropati/cerrahi.

Peroneal nerve entrapment was diagnosed in three patients (2 males, 1 female) by clinical and electrophysiological studies. Of these, one patient had postural bilateral involvement due to prolonged squatting, while two patients had mechanically-induced entrapment. Initially, all the patients were treated conservatively with a drop-foot splint and vitamin B. One patient responded to treatment; in one patient with bilateral involvement, right-sided peroneal nerve palsy improved. Upon detection of no clinical and electrophysiological improvement after three months of conservative treatment, surgical decompression was performed in two patients, which resulted in a successful outcome in the patient with bilateral palsy. Incomplete recovery was obtained in the other patient with diabetic polyneuropathy.

Key words: Ankle injuries; decompression, surgical; electromyography; nerve compression syndromes/etiology/surgery; paralysis/ etiology/surgery; peroneal nerve/injuries/surgery; peroneal neuropathies/surgery.

Acute injury to the peroneal nerve is a frequent occurrence due to trauma, surgery or postural entrapment of the nerve at the fibular head.[1-4] Nontraumatic causes are rare and commonly involve tumours, intraneural ganglia, hematoma or cysts.[1-6]

The peroneal nerve branching from the sciatic nerve at the popliteal groove, passes over the lateral head of the gastrocnemius muscle lateral to the groove.[3,7,8] Having a very superficial route in the 4 cm long area below the knee and around the fibular head and neck, the nerve is only protected by the skin and the superficial fascia.[3,7,9] It passes through a fascial fibrous arch surrounded by the long peroneal muscle and the intermuscular septum.[7,8] In the peroneal nerve mononeuropathy frequently encountered in the lower extremity, the nerve is injured commonly in this 4 cm long area where it shows a superficial location or is entrapped when the fibrous arch is thickened, narrowing the tunnel the nerve passes through.[1-4,7] This fibrous arch is prone to dynamic entrapment during sports activities and postural entrapment during squatting or leg crossing

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due to positional changes.[1]

During squatting, the nerve is compressed between the biceps tendon, lateral head of the gastrocnemius muscle and the fibular head, due to compression forces on the muscles with the weight of the body.[7,8] Peroneal neuropathy may also rarely occur with forced inversion during ankle distortion, when the nerve is stretched by the long peroneal muscle or when it becomes compressed by the hematoma formation due to rupture of the vasa nervosum.[2,10,11]

This report presents treatment methods and results obtained in three patients with peroneal nerve injury due to mechanical reasons after ankle distortion and postural reasons due to prolonged squatting.

Case report

Three patients were treated in our clinic for drop foot after peroneal nerve injury. Electromyography (EMG) and nerve conduction tests were performed in all three patients in order to finalize the diagnosis, to detect the extent of injury and evaluate the results of treatment. Findings were compared with the normal side during motor function analysis. Toe and ankle muscle tests were performed. Sensory evaluation consisted of a search for hypoesthesia and numbness on the anterolateral aspect of the calf, dorsal surface of the foot and between the great and second toes. Palpation was performed in the popliteal fossa and around the fibular head, looking for mass lesions, while the Tinel test was executed with percussion over the fibular head.

Patient no 1 – Twenty-six year-old female patient was admitted with pain and swelling over the lateral side of her right ankle after an inversion type of ankle sprain while walking on rough ground one month previously. She had difficulty in walking and moving her ankle, after bed rest for a few days and her right ankle was waddling with her toes drooping and dragging on the ground. She referred to our clinic since she could not benefit from the drugs prescribed by another physician three weeks prior to her admission and her symptoms were still prominent. Physical examination revealed mild edema and hypersensitivity on the lateral aspect of her right ankle. Extension and eversion of the right ankle and extension of the toes were not possible. Plantar flexion and invertion was normal. There was sensory loss on the anterolateral side of the calf and the dorsal surfaces of the foot and toes. Tinel test was negative over the fibular head. No mass lesions were identified around the knee joint with palpation. Nerve conduction studies revealed a lower combined muscular action potential of the peroneal nerve on the right side as compared to the left (5.6 and 6.9 respectively). Conduction velocity of the right peroneal nerve was 33 M/sec at the popliteal fossa-fibular head segment and 55 M/sec at the infrapopliteal-ankle segment. She was diagnosed to have acute partial lesion with axonal degeneration of the peroneal nerve close to the fibular head and vitamin B treatment was initiated together with an ankle foot orthosis.

Control EMG study performed after one month of treatment revealed findings indicative of healing and the presence of regeneration was considered. Three months later, EMG detected denervation potentials on the right tibial and peroneal muscles, while the conduction velocity of the right peroneal nerve at the popliteal fossa-fibular head level increased from 33 M/sec to 45 M/sec. Motor function of the anterior tibial muscle was grade 2, and of extensor hallucis and digitorum longus muscles were grade 3 at physical examination. Two years later, motor function was complete in all muscles innervated by the peroneal nerve, the patient had normal gait and no sensory loss was noted in the leg or foot.

Patient no 2 – A slender, twenty year-old male patient (height 1.85 m, weight 65 kg) presented with bilateral foot dropwithout any history of trauma. He had to squat for 6-7 hours about 10 days previously and his complaints followed this episode, and he indicated that he could not flex his ankle and toes. He had taken medication during this period, however he referred to our clinic when his gait disturbance persisted with frequent tripping episodes. EMG demonstrated severe injury to peroneal nerves on both sides at the fibular head level. No orthopaedic abnormalities or mass lesions could be identified around the knee joint and generalized peripheral neuropathy was not present. He was prescribed vitamin B and recommended not to squat for long periods. Control EMG study three months later demonstrated some healing in the muscles innervated by the peroneal nerve; however, the left side was less

responsive to therapy. The injury to the left peroneal nerve was determined as axonal loss rather than segmental demyelination. For this reason surgery was considered and the left peroneal nerve was decompressed over the fibular head.

Significant improvement was noted after surgical intervention. Fifteen days postoperatively, his gait had improved and extension strength of the left ankle and toes had increased. Two months later, his gait was normal, with only a mild loss of strength in extending the great toe as compared to the normal foot. He is still under follow-up for three years and final examination revealed no sensory loss in the lower extremities and motor function was complete and equal on both sides.

Patient no 3 – Twenty-two year-old male patient with diabetes referred with difficulty in walking and foot drooping after an inversion type of ankle distortion. He coumd not perform flexion on his right ankle and toes. Tinel test was positive over the fibular head. Motor evaluation demonstrated that function was only grade 1 or 2 as compared to the normal side for the anterior tibial, extensor hallucis longus and extensor digitorum longus muscles. However, eversion, inversion and plantar flexion were normal. Electromyography showed significant sensorimotor polineuropathy on the extremities. Complete denervation was present in all muscles innervated by the deep branch of the peroneal nerve at the right lower extremity. He was treated conservatively; however, surgery was planned after a control EMG four months later failed to demonstrate regenerating motor unit potentials (MUPs).

During surgery, the peroneal nerve was noted to be entrapped by the proximal part of the long peroneal muscle at the level of the fibular head. The edematous fascial band compressing the nerve was dissected and surrounding soft tissues released. Postoperatively Robert Jones bandage was applied for three days and the patient was immobilized. Exercises wer initiated afterwards. Control EMG evaluation six months later demonstrated regenerating MUPs only in the right anterior tibial muscle and an incomplete recovery was present. Tendon transfer was proposed since his diabetes prolonged tissue repair; however, the patient refused this option. Twenty months postoperatively, right ankle and toe extension were limited compared to the left side, sensory loss was prominent at the lower extremity due to diabetic polyneuropathy, motor function of the anterior tibial muscle was grade 3 and those of extensor hallucis longus and extensor digitorum longus muscles grade 2.

Discussion

Peripheral nerves may be entrapped along their course either acutely or as part of a chronic process. Internal or external compression of nerves are termed compressive or entrapment neuropathy. Examples of external types of entrapment neuropathy include radial nerve injury due to alcoholism or inappropriate use of crutches, ulnar nerve injury due to prolonged leaning on elbow and peroneal nerve compression by a short leg cast.[12] Nerves may also be internally entrapped by bony spurs, around bone calluses, by sinovial thickening or due to tumours, ganglions, fibrous bands or muscles.[4,5,8,12] In patients with extensive polyneuropathy and diabetic patients, the nerves are more prone to injury and less responsive to treatment.[8,12]

The peroneal nerve is usually compressed around the fibular head or neck or may be injured by direct trauma. It has a rather superficial course in this area and is only covered by the skin and subcutaneous tissue.[3,9] Moreover, the fibular head is excessively mobile and causes a continuous mechanical irritation for the nerve.[13] Compared to the tibial nerve, the peroneal nerve has a smaller amount of nerve fibers and supportive tissue, is fixed to the fibular neck and thus, is both more prone to stretching and is unable to absorb axial forces.[9,10,14] all three patients had peroneal nerve injuries at the level of the fibular head.

Prolonged squatting, leg crossing and yoga may cause postural peroneal nerve palsy.[1,7,8,12] Only a few reports are present concerning prolonged squatting and bilateral peroneal nerve injury. Toğrol et al[12] reported bilateral peroneal nerve injuries in three patients aged 13, 20 and 47 years after squatting for more than 5-6 hours. They indicated that the first two young patients were thin and slender and that the nerve became more sensitive to mechanical irritation or direct pressure in the presence of undernourishment, metabolic factors or thinning of the protective subcutaneous tissue. In their twenty year-old patient who was tall and slender (case no 2) the appearance of bilateral peroneal nerve injury after prolonged squatting and the identification of lesions

at the level of the fibular head by EMG correlate with the mechanisms of injury previously identified in the literature.

The nerve may be injured following total knee arthroplasty or proximal tibial osteotomy, knee dislocations, long-term hospitalization and prolonged pelvic surgery in the lithotomy position, due to short leg casts or splints or leg orthoses.[2,12,15,16] Iatrogenic nerve injury may be minimized during knee surgery by a meticulous approach and good knowledge of anatomy. We have not encountered with nerve lesions due to casts, total knee arthroplasty or patient position in our clinic.

Inversion type anke sprain is a frequent injury of the lower extremity. They most commonly affect the lateral collateral ligaments of the ankle. Rarely, peroneal nerve paralysis may occur.[11] In two of our patients, the peroneal nerve injury was due to an inversion type of ankle sprain.

Conservative treatment may be effective when a certain type of activity of posture is related to the entrapment.[8] According to a generally accepted opinion, 3-4 months of conservative treatment should be considered initially, after which, in case of failure and in the presence of significant axonal injury surgical neurolysis or release of the arch located between the two heads of the peroneal muscle are recommended.[1,8,10,12,16] The success rate is reported to be lower when surgery is delayed for more than six months.[15] Conservative treatment was considered in the two patients who had unilateral nerve injury related to ankle sprain. Healing was observed in one patient after three months; in the other patient with diabetes, surgical release at the level of the fibular head was performed in the fourth month when no sign of healing could be detected. Regeneration potentials in the anterior tibial muscle were noted in the sixth month after surgery and an incomplete resolution could be achieved. We believe that diabetes was detrimental to the healing process in this patient. Again, conservative treatment was initially considered for the patient who had bilateral nerve injury after prolonged squatting. After three months, significant healing was present on the right side despite absence of healing on the left side. Control EMG in this patient demonstrated serious axonal injury and surgical decompression was therefore performed. Following surgery, prominent clinical and electrophysiological healing was observed.

In conclusion, peroneal nerve function should be evaluated in slender patients with a history of gait disturbance or foot drop after prolonged squatting or in patients with inversion type of ankle sprain. When clinical and electrophysiological evidence of nerve injury is present, conservative treatment under follow-up with EMG should be preferred for three months, and surgical decompression with a meticulous approach should be preserved for failed cases.

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