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Common Peroneal Nerve Entrapment At The Fibular Head: A Case Report

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Abstract: *Peroneal nerve palsy is the most common entrapment neuropathy of the lower extremity. Numerous etiologies have been identified; however, compression remains the most common cause. Although injury to the nerve may occur anywhere along its course from the sciatic origin to the terminal branches in the foot and ankle, the most common site of compression pathology is at the level of the fibular head. The most common presentation is acute complete or partial foot drop. Neurodiagnostic studies may be helpful for identifying the site of a lesion and determining the appropriate treatment and prognosis. Management varies based on the etiology or site of compression. Many patients benefit from nonsurgical measures, however surgical decompression should be considered for refractory cases and those with compressive masses, acute lacerations or severe conduction. Results of surgical decompression are typically favorable, tendon and nerve transfers can be used in the setting of failed decompression or for patients with a poor prognosis for nerve recovery*

Keywords : nerve entrapement syndrome, peroneal nerve, compression, neurolysis.

INTRODUCTION

Peroneal nerve palsy is associated with the onset of acute and progressive foot drop and is the most common compressive neuropathy of the lower extremity.[1-2] Below the knee, the peroneal nerve courses around the lateral aspect of the fibular neck, where it is highly vulnerable to injury [3-4]. Apart from laceration or stretching of the nerve during fractures or dislocations of the proximal fibula or ankle, idiopathic entrapment syndrome is the most common cause of loss of peroneal nerve sensory and motor function. As well as all compressive neuropathy, the key to a successful outcome is early identification and treatment. [4]

CASE REPORT

We report the case of 27 years old waiter man, who presented walk disorder installed 15 days before consultation. The story dates back to 6 months by atypical pain at the external face of right knee without improvement under symptomatic treatment. Evolution was marked by progressive installation of neurological signs such as paresthesia at the external face of the foot and leg; gradually the patient began to feel weakness in the right lower limb with difficulty walking which motivated his consultation.

The patient did not report notion of knee or ankle trauma, no notion of squatting for a long time before the symptoms occurred, no notion of taking any particular medication; however, it referred to had story of unprotected sex.

The clinical examination found a possible standing position, possible walking using a crutch with right steppage, amyotrophy of anterolateral compartment of the right leg; dorsiflexion deficit of right foot and decreased sensitivity on the anterolateral side of the leg and the external edge of the foot relative to the contralateral side. The ankle reflex was normal.

The diagnosis of injury of common peroneal nerve was evocated. An electromyographic study was requested, its came back in favor of a right common peroneal nerve sensitive-motor block at the knee (the neck of fibula) with signs of active muscle denervation. After confirmation of diagnosis, we have completed our investigations by standard knee radiography (figure) and MRI ; radiographs not revealed abnormality ; the MRI objectified suffering of the common peroneal nerve at fibular neck without extrinsic compression and muscle beginning suffer. Biological assessment did not reveal inflammatory syndrome and serology (HIV Hepatitis and Syphilis) came back negative.

Clinical and radiological arguments were in favour of advanced stage of common peroneal nerve idiopathic entrapment syndrome. Therefore, we decided to perform a surgical decompression.

We perform the surgery under regional anesthesia. In supine position, a tourniquet installed to the 1/3 middle of the thigh. The knee is flexed at 30 degrees using wedge blocking the foot, and the hip is kept in internal rotation using a lateral wedge placed at the level of tourniquet (Figure 1)

Figure 1: Patient installation and incision.

We used an oblique anterolateral approach at about 1 cm below the neck of fibula, directed forward and downward, it begins posterior to fibular neck and ends before reaching tibial crest (Figure 1). After opening the crural fascia, we identify and protect the lateral sural cutaneous nerve during the intervention; the common peroneal nerve is found just behind the fibular neck, it is put on lake before continuing the dissection. (Figure 2)

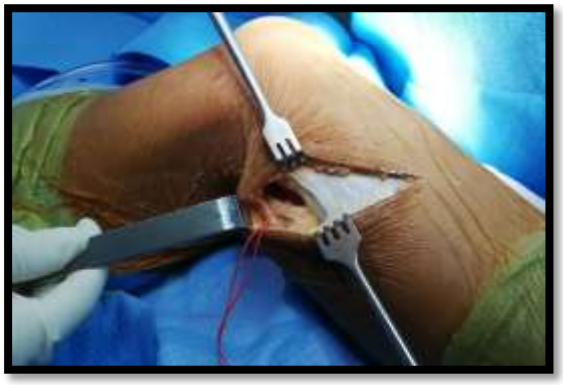

Figure 2: Common peroneal nerve put on lake after superficial fascia opening.

The nerve was sandwiched between posterior crural intermuscular septum superficially and deep tendinous fascia profoundly, we incised this two structure to release the nerve before its passage under peroneous longus. We then incise the superficial muscle fascia in the direction of the nerve to expose the peroneus longus and compressive intermuscular septa (Figure 3 and 4).

Figure 3: Incision of superficial muscle fascia.

Figure 4: Black star: articular and muscular branches of CPN; White arrow: Septum between peroneus longus and extensor digitorium longus; Black arrow: Septum between extensor digitorium longus and tbialis anterior.

The joint and muscle branches rising from CPN are identified and protected (Figure 4). The muscles appearance was normal; the intermuscular septa were completely visualized (Figure 4), the first between peroneus longus and extensor digitorum longus, the second between extensor digitorum longus and tibialis anterior. These intermuscular septa are divided (Figure 5).

Figure 5: section of septa and deep release.

The release then continues proximally, at the same time we release the lateral sural cutaneous nerve (Figure 6).

Figure 6: proximal release of CPN and lateral sural cutaneous nerve.

At the end of the the tourniquet is released, and a careful hemostasis is performed before closing. The ankle is immobilized by posterior splint at 90° of flexion.

The patient received analgesic and vitamin therapy (Vit B6), Passive mobilization of the ankle and isometric contraction were started as soon as pain decreased at D4 after surgery.

This decompression technique described by Makinonn et al [25] is an extensive technique that allows a complete release of the CPN, the associated release of the lateral sural cutaneous nerve also improves the symptoms (pain due to the external face of the knee).

DISCUSSION:

The peroneal nerve arises as the lateral branch of the sciatic nerve in the distal posterior thigh, the medial branch being the larger posterior tibial nerve. The peroneal nerve receives innervation from the L4, L5, S1 and S2 nerve roots. The peroneal nerve runs along the lateral margin of the popliteal fossa in between the lateral head of gastrocnemius muscle medially and the biceps femoris tendon laterally. It descends posterior to the head of the fibula, giving off the lateral sural cutaneous nerve and passes laterally to wrap around the lateral aspect of the fibular neck. This segment of the nerve is superficial and vulnerable to injury, as it is covered by only skin and connective tissue. The peroneal nerve then pierces the tendinous arch that runs between the two heads of the peroneus longus muscle. It then divides into the superficial and deep peroneal nerves.

Although compressive etiology remains the most common cause, many other factors contribute to injury. Traumatic causes include knee dislocation, severe ankle inversion injuries, lacerations, and direct blunt trauma. These traumatic injuries are typically associated with poorer outcomes. [5-6]The link between diabetes mellitus and lower extremity neuropathies (eg, polyneuropathy, mononeuropathy) has been well established. [7] Iatrogenic injury is common as well, with acute foot drop often seen as a result of surgery about the hip, knee, and ankle; positioning during anesthesia; prolonged bed rest; casting; bracing; compression wrapping; and the use of pneumatic compression devices. [8]

In patients with injury to the peroneal nerve, clinical presentation varies based on the location and severity of the injury and the presence of anatomic variations. Most commonly, patients report the classic symptoms of foot drop or catching the toes while ambulating. Foot drop can develop acutely or over a period of days to weeks, depending on the etiologies, and can be complete or partial in severity. Numbness or dysesthesia may also be present along the lateral leg, dorsal foot, and/or the first toe web space; pain may be present in some cases (eg, traumatic wounds, compressive lesions), but it is not a common complaint. [2]

Neurological examination can reveal weakness in the following muscle groups: ankle everters, ankle dorsiflexors and toe dorsiflexors. Weakness will not be present in the hip extensors, knee flexors and ankle inverters, all muscle groups also supplied by the L4 to S2 nerve roots, the same roots that supply the peroneal nerve. This is a useful distinguishing feature when trying to determine whether a foot drop is because of a lumbosacral root or a peripheral nerve lesion. In advanced lesions, wasting of the tibialis anterior and peroneal muscles may be observed. The ankle reflex is normal, as it is mediated through the posterior tibial nerve. Sensory loss can be present in anterolateral leg and dorsum of the foot. There may be tenderness over the course of the peroneal nerve at the fibular neck, as well as a positive Tinel's sign. Straight leg raise is uninhibited, except in the presence of concurrent lumbar disc disease.

When clinical examination indicates a potential injury to the peroneal nerve, plain radiography should be considered as part of the initial workup. The close proximity of the peroneal nerve to the fibular neck as well as its superficial location makes it susceptible to injury secondary to direct trauma and impingement from both soft-tissue and bony sources. CT may be used to further evaluate bony abnormalities. MRI and ultrasonography should be considered to evaluate for potential soft-tissue sources of impingement or masses [1-9]

Nerve conduction velocity (NCV) studies and electromyography (EMG) are valuable tools for diagnosing suspected peroneal nerve palsy. These studies help the clinician evaluate the motor and sensory axons of the peroneal nerve and its branches. They also are useful for localizing the site of injury, determining the severity of a lesion, and monitoring recovery after a nerve injury has been identified. [10] An electrophysiology study should be performed to obtain a baseline in all patients who present

with new-onset foot drop; the study may be repeated every 3 months to monitor for improvement or deterioration. In the setting of traumatic injury or postoperative palsy, immediate neurodiagnostic tests are not warranted and should not be performed for 2 to 6 weeks.

Entrapment neuropathy of the peroneal nerve is a common cause of peripheral nerve lesions in the lower limb. [11] Historically, patients with this condition who have a foot drop have been treated with orthotic support devices. These reduce some of the morbidity associated with a foot drop while allowing spontaneous recovery. [12] Many patients do recover spontaneously from perineal nerve entrapment neuropathy. [13] Surgically, tendon transfers have also been used to correct a foot drop. [14] The sensory symptoms have frequently gone untreated or managed symptomatically with analgesic agents.

There are no randomized controlled trials comparing conservative and surgical managements of peroneal nerve entrapment neuropathy. However, there are case series published in the literature that assess outcomes of patients managed with either conservative or surgical management.

Neurolysis of the CPN at the fibular head provides a fasterrecovery than does rehabilitation therapy. If needed, the source of the compression can be removed Aspiration of an articular cyst may be required. An intraneural cyst shouldbe removed by intra-fascicular neurolysis combined with ligation of the articular branch [15-16]. Superficial fibular nerve entrapment requires release of the tunnel [17-18]

Baron [19] has advocated non-operative treatment combining the elimination of risk factors, rehabilitation therapy, and the use of a brace in patients with severe foot drop. A protective cushion can be placed at the neck of the fibula. Local glucocorticoid injections have also been suggested.

Uzenot et al. [20] have stated that surgery can be considered in the absence of recovery after 6 months. Dallari et al. [21], in contrast, have recommended early surgery as soon as the diagnosis is confirmed, regardless of the cause, in order to obtain a full and complete recovery. Ismael et al. [22] found that the extent of functional recovery increased as time to surgery decreased.

Both patients with CPN entrapment due to prolonged squatting achieved a full recovery after neurolysis, in keeping with a report by Fabre et al. [23].

Excision of a synovial cyst responsible for compression of the CPN has been reported in two patients [15-22] Rapid pain relief was obtained. Anterolateral leg muscle function recovered gradually and partially after 2 months, a full clinical recovery of sensory and motor function was achieved within 8 months after surgery in one patient [15] and after nearly a year in the other patient [22]. According to Bahri et al. [24]early treatment is more likely to produce good outcomes in patients with CPN entrapment at the fibular head due to fibrous and inflammatory changes induced by forced inversion of the foot (equinus varus).

CONCLUSION :

The CPN can be compressed in the fibular tunnel resulting in associated motor deficits and sensory symptoms, most importantly foot drop. The investigation of choice is electrophysiological testing to demonstrate delayed conduction and reduced motor unit recruitment. MRI can also be helpful in the workup of patients with foot drop in order to exclude proximal lesions. Many patients will experience spontaneous recovery early in the course, and hence observation and symptomatic treatment is the first line of management. However, spontaneous recovery has been described as delayed and incomplete. We prefer early surgery within the first few months if the symptoms do not start to resolve after the first month.

DISCLAIMER: The authors, their immediate families, and any research foundations with which they are affiliated have not received any financial payments or other benefits

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