Nerve Entrapment Syndromes of the Lower Limbs

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1 Introduction

Compressive pathologies of peripheral nerves are important causes of lower limb pain in the general population. Specifically, in athletes, the incidence is up to 15%. This syndrome should always be on the list of differential diagnoses, since the delay in diagnosis can cause worsening on pain, difficulties to maintain activities, and irreversible lesions [18].

The clinical complaint is usually nonspecific, and deep burning pain may occur, eventually associated with paresthesia, dysesthesias, shocks, numbness, and muscle weakness. The Tinel sign (pain and shock sensation to the digital percussion at the site of peripheral nerve compression) is individually the most specific sign [18]. Due to this irregularity of clinical presentation and variations in physical examination, these pathologies have often a delay in the diagnosis [8].

The electromyographic study assists in the diagnosis but may be negative due to intermittent compression. Imaging tests can help in the location of compression, in the diagnosis of extrinsic compressions, and through indirect signs such as muscle atrophies secondary to denervation.

Differential diagnoses affecting peripheral nerves, but not related to peripheral compressions, should be remembered. Myopathic diseases include inflammatory myopathies (polymyositis, dermatomyositis), endocrine myopathies (toxic or drug-induced), muscular dystrophies, myotonic dystrophy, acid maltase deficiency,

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benign congenital myopathies (myotubular, nemaline), neuromuscular blockage pathologies such as myasthenia gravis, Lambert Eaton syndrome, Guillain Barré syndrome, and chronic demyelinating polyneuropathy. Neurogenic pathologies include diabetic neuropathy, lumbar root lesions, motor neuron diseases, and spinal muscle atrophy. Other causes such as muscle atrophies secondary to joint pathologies and upper motor neuron lesions should also be remembered [17].

Other pathologies that often cause pain in the lower limbs, especially in athletes, should also be searched and ruled out. Such pathologies include medial tibial stress, stress bone injuries, popliteal functional artery compression syndrome, and exercise-related compartment syndromes [15].

2 Compressive Pathologies of the Lower Limbs

2.1 Common Peroneal Nerve

The common peroneal nerve is a sensory and motor nerve with contributions from L4 to S2 roots. It is a branch of the sciatic nerve, just above the knee, supplying the short head of the biceps femoral muscle. It curves laterally and distally, passing through an osteofibrous tunnel at the fibular neck, deep to the origin of the peroneus longus muscle (fibular tunnel), where it is divided into superficial and deep peroneal nerve and lateral sural skin branch.

The most common site of irritation of this nerve is in the neck of the fibula, in its subcutaneous trajectory. It may be affected by internal compressions (intra and extra-neural masses), external compressions (pneumatic cuffs), traction (associated to ankle inversion traumas), and local direct trauma, the most common cause of this neuropathy.

The prognosis, in the great majority of the cases, is satisfactory, showing no signs of severity in most patients [19]. The clinical presentation includes alteration of sensitivity in the lateral aspect of the leg and dorsum of the foot, in addition to weakness of the extensor and evertor musculature of the foot. It may evolve to the classic drop foot, and consequent alteration of the gait pattern. Tinel at the neck of the fibula may be present in up to 97% of patients [6].

When compressive pathologies of the common peroneal nerve are suspected, the imaging evaluation includes radiographs to assess the presence of bone alterations that may cause compression, ultrasonography, and magnetic resonance imaging to assess local soft tissue masses. Electroneuromyography can be also helpful in identifying the lesion, its severity and location [16]. About 38% of cases are considered idiopathic, that is, without known cause [24].

Clinical treatment includes mainly removal of extrinsic irritant factors (position of crossing the legs or stockings with high elastic at the level of the fibula neck). Full or partial recovery is expected depending on the nerve injury extent. The prognosis in the case of neuropraxia is excellent with nonsurgical treatment [31].

In the case of severe motor and sensory deterioration (>50% conduction delay) or when there is no recovery within 4–6 months from the injury, surgical treatment should be indicated.

In open lesions, wound should be clean and nerve repaired within 72 hours, to improve chances of recovery.

Masses that cause extrinsic compression should be removed in the usual way, and intra-nervous masses (schwannomas and neurofibromas) should be approached by specialists so as not to aggravate the nerve lesion. In cases of idiopathic compressive neuropathy, early decompression shows, in a series of cases, superior results with better motor recovery [29]. On the other hand, open lesions should be surgically addressed and corrected in the acute phase of the lesion [22].

Decompression is performed through a curvilinear incision that follows common peroneal nerve route, starting at the fibular neck with an anteromedial extension. Release of the common peroneal nerve starts at the fibular neck (easily identified through palpation), following the nerve into the fibular tunnel (peroneal longus fascia or posterior intermuscular septum). Then release should also include the anterior intermuscular septum (septum between anterior and lateral compartments). This septum is exposed through retraction of the peroneus muscle belly [22].

2.2 Solear Arch Syndrome

It refers to the compression of the tibial nerve in the proximal portion of the leg, when it passes under a fibrous arch at the origin of the soleus muscle [32]. Patients complain of pain and discomfort in the calf. In cases of failure of surgical treatment of a patient diagnosed with tarsal tunnel syndrome, this compressive pathology should be remembered as a differential diagnosis.

Clinically, pain is noted on palpation of the proximal portion of the leg, 9 cm from the popliteal fold. There may be alteration of sensitivity in the sensory innervation territory of tibial nerve and weakness of the flexor hallucis longus. Electroneuromyography is frequently not useful. High-resolution magnetic resonance imaging may show suggestive changes at the site of compression.

Conservative treatment modalities include removal of extrinsic causes of compression, changes in exercise pattern, anti-inflammatory drugs, and infiltrations. Surgical treatment with release of compression tissue and adhesions shows satisfactory results without major associated complications.

2.3 Deep Peroneal Nerve

After the common peroneal nerve division, the deep peroneal nerve turns around the fibular neck and passes to the anterior leg compartment through a septum between the lateral and anterior compartments, located 3–4 cm distally to the fibular neck (place of possible compression). It innervates the anterior tibial muscle (AT), extensor digitorum longus (ELD), extensor hallucis longus (ELH), and peroneus tertius. The EHL crosses this nerve approximately 3 cm above the anterior ankle joint line. The deep peroneal nerve runs its course between the EHL and the EDL, under the upper extensor retinaculum, until it is divided into medial and lateral branches. The medial branch follows the dorsal pedis artery and is responsible for the sensitivity of the first interdigital web space. The lateral branch innervates the extensor digitorum brevis muscle. This branch passes under the two bands of the lower extensor retinaculum.

The most common site of deep peroneal nerve compressions is under the upper extensor retinaculum, known as anterior tarsal tunnel syndrome. This space contains the dorsal pedis artery and vein, the deep peroneal nerve, besides the peroneus tertius, anterior tibialis, EDL, and EHL. Among the causes of compression are space-occupying lesions in the anterior ankle (osteophytes, ganglions, synovitis, or low myotendinous insertion), local trauma, and the use of compressive shoes.

Clinically, the patient presents with uncharacteristic pain in the anterior ankle and foot dorsum, and there may be a sensitivity change on the first interdigital space or weakness of toes extension. The discomfort may be aggravated by forced plantar flexion of the ankle, which stretches the deep peroneal nerve. Tinel's sign may be present on the nerve pathway. The most common differential diagnosis is the chronic compartment syndrome of the anterior leg compartment.

Imaging investigation and anesthetic blocks may help in identifying the cause and location of compression. Electroneuromyography should be performed to evaluate the location of the compression. Ultrasonography and magnetic resonance imaging can also be useful in diagnosis and location of extrinsic compression factors (Figs. 1 and 2).

Conservative treatment is initiated with ankle repositioning, use of footwear that reduces extrinsic compression, topical or systemic anti-inflammatory drugs and analgesics, and infiltration with lidocaine or corticoids [10].

If conservative treatment fails, surgical release should be indicated, with care being taken to remove all possible causes of compression, including retinaculum release. The prognosis of this procedure is very good, with good and excellent results in up to 80% of patients, once the proper diagnosis has been made [2].

2.4 Superficial Peroneal Nerve

After the division of the common peroneal nerve, the superficial peroneal nerve enters the lateral compartment, and crosses the crural fascia on the anterolateral side of the leg 9–14 cm above the lateral malleolus, the crural tunnel. It innervates the peroneal (brevis and longus) muscles. As it approaches the anterior ankle, it divides into the medial and intermediate dorsal cutaneous branch, sensory branches of the foot dorsum.

The most common cause of neuropathy of this nerve are repetitive traumas and ankle sprains [21], related to the practice of physical activities (hockey, tennis,

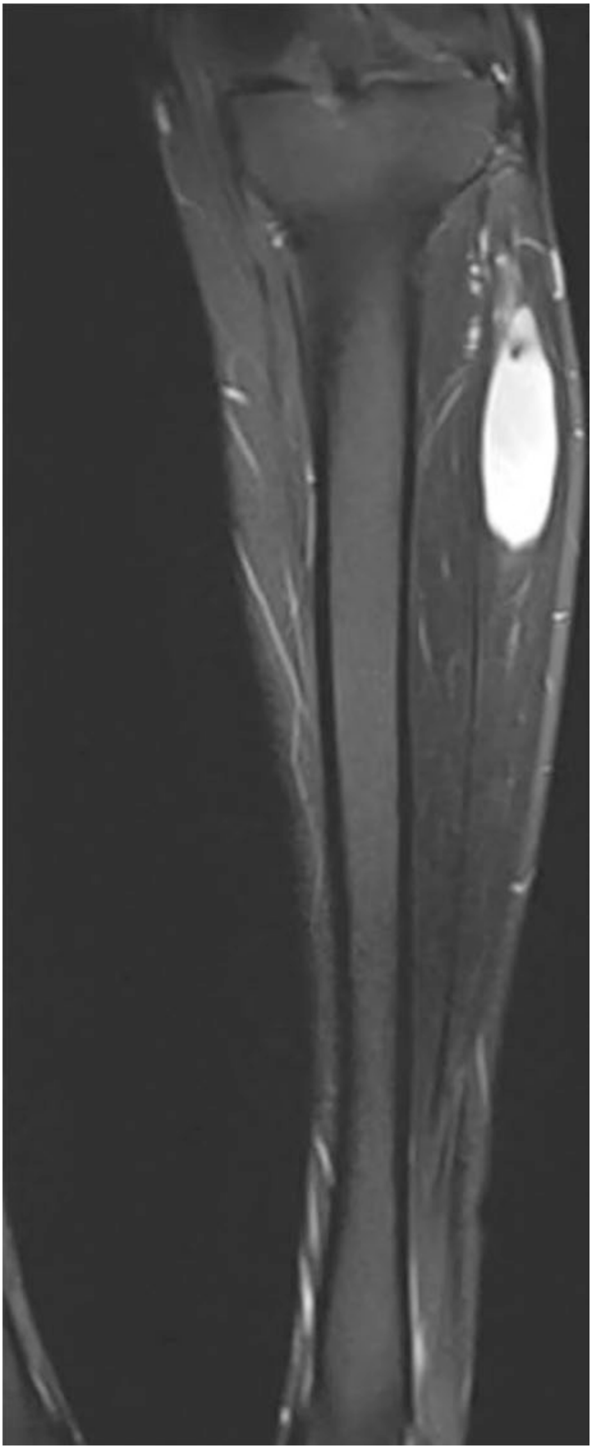

Fig. 1 Axial T2-weighted magnetic resonance imaging section showing a large synovial cyst in the path of the deep peroneal nerve just after the common peroneal nerve division, causing neuropathic pain and weakness of the ankle and toe extensors

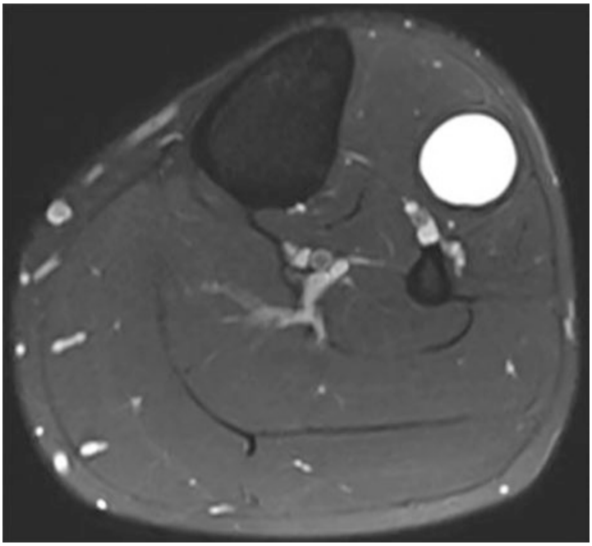

Fig. 2 T2-weighted MRI axial view, from the proximal portion of the leg, showing the same synovial cyst shown in Fig. 1

soccer). Compression at the crural tunnel by muscle hernias, local tumors, or just local changes of the fascia can cause irritation of this nerve. The chronic lateral compartment syndrome may simulate the symptoms of superficial peroneal nerve compression, as well as compressive pathologies of L5. Iatrogenic lesions should be remembered by frequency (lateral portal of anterior ankle arthroscopy or surgical incisions for ankle fractures).

The most common complaint is related to pain, burning, and tingling on the lateral distal leg and foot dorsum. The typical pain pattern is located in the distal lateral leg portion, with or without a burning sensation. It is usually aggravated by activity.

Diagnostic investigation includes imaging examinations such as ultrasonography and magnetic resonance imaging. The dynamic character of ultrasonography can be useful, since the muscle hernias can disappear at rest. The electroneuromyography can also be useful in identifying the cause and location of neuropathy.

The treatment of superficial peroneal nerve neuropathy will depend on its cause. Conservative treatment consists of shoe adjustment, physiotherapy to strengthen the peroneal muscles and insoles. Infiltrations can bring symptoms relief. If conservative treatment fails, surgical treatment should be employed. Decompression through localized fasciotomy and neurolysis present success rates of up to 80% [28]. If chronic ankle instability and chronic lateral compartmental syndrome are associated, these should be treated as well.

2.5 *Saphenous Nerve*

The saphenous nerve is a cutaneous branch of the femoral nerve, with fibers of L3 and L4, following distally through the inner face of the thigh through the adductor canal together with the artery and femoral vein. In the lower portion of the canal, it crosses the sartorius muscle fascia, dividing at this level into an infrapatellar branch and a descending sartorius branch. The infrapatellar branch innervates the skin on the anteromedial side of the knee. The sartorius branch follows up to the foot as the saphenous nerve, following the great saphenous vein, up to the anteromedial surface of the ankle, innervating the skin of the medial surface of the leg, region over the medial malleolus and the medial surface of the foot.

The saphenous nerve is most commonly affected in the region of the adductor canal, due to direct trauma (dislocation of the patella), extrinsic compressions, scars, irritation related to cycling or rowing, and iatrogenically when the saphenous vein is dissected for revascularization procedures.

The most common symptom is vague pain on the medial surface of the knee and/or the medial surface of the foot and leg. Diagnostic imaging investigation can help in situations related to trauma or extrinsic compression, but the electrophysiological study in most cases is negative.

Conservative treatment with local protection, and infiltrations can be performed. In the failure of conservative treatment, decompression, neurolysis, or even neurectomies may be indicated. The resection of neuromas can cause changes of sensitivity in the territory of this nerve.

2.6 Sural Nerve

The sural nerve receives contribution from the tibial nerve (medial sural cutaneous branch) and common peroneal nerve (lateral sural cutaneous branch) in 80% of the population [23]. The nerve begins in the distal portion of the popliteal fossa, passes between the gastrocnemius heads and through the deep fascia in the middle portion of the leg, to its subcutaneous trajectory. At the ankle, its path is posterior to the lateral malleolus (between the Achilles tendon and fibula), where it innervates the skin of the lateral aspect of the calcaneus and foot. Distal to the lateral malleolus, it emits calcaneal branches, responsible for the sensation on the lateral side of the heel.

The compression of the sural nerve is more common in athletes, related to extrinsic compression (shoes), due to trauma such as ankle sprains, and iatrogenic lesions after foot and ankle surgery. Iatrogenic causes are relatively common, especially when incisions are made on the lateral side of the hindfoot. Symptoms include diffuse burning or pain and changes in sensitivity in this nerve territory. Tinel's sign may be present.

Investigation with electroneuromyography can assist in site diagnosis and cause of compression. Conservative treatment should be instituted initially, and in its failure neurolysis or neurectomy can be performed.

2.7 Tibial Nerve

It is the terminal branch of the sciatic nerve, with roots from L4 to S3. It begins in the popliteal fossa and passes deeply into the gastrocnemius and soleus, entering the deep posterior compartment of the leg. It traverses in the posterior and medial ankle, through the tarsal tunnel, giving then origin to the calcaneal branch. It divides then into medial and lateral plantar nerve, which are responsible for the plantar cutaneous innervation of the foot. It innervates the muscles of the posterior compartments of the leg (superficial and deep compartments) and the intrinsic plantar musculature of the foot.

The most common site of compression of this nerve is the tarsal tunnel, a space limited medially by the tibia, talus, and calcaneus, and laterally by the flexor retinaculum (lancinate ligament) as its roof. The tarsal tunnel contains the tibial nerve, the posterior tibial tendon, flexor digitorum longus (FDL) tendon, flexor hallucis longus (FHL), and the posterior tibial artery and vein. Compressions occur mainly in athletes, and between the causes are valgus alignment of the hindfoot, direct compression by space-occupying lesions inside the tarsal tunnel (tumors, cysts, varicose veins), extrinsic compressions (shoes), plantar fasciitis, and direct trauma. However, in more than 50% of cases the compression cause in not identified, being classified as idiopathic [14, 32].

The patients present plantar and medial pain in the foot and ankle, with burning sensation, shocks, and alteration on sole of the foot sensibility (Fig. 3). The symptoms worsen with the activity and when the patient remains in orthostatic position for a prolonged period of time. Tinel's sign may be present, as well as motor alterations of the foot intrinsic musculature.

The evaluation of the hindfoot alignment should be performed. Ultrasonography can be very useful in the diagnosis and identification for most lower limb compressive pathologies [3]. Magnetic resonance imaging is another option, and is capable of demonstrating a compressive cause in up to 80% of patients with this syndrome (Fig. 4) [9]. On the other hand, the electromyographic study has a high rate of false positive results, and it should be interpreted using caution [20].

Fig. 3 Illustration showing the tarsal tunnel area in red, the most common location of tibial nerve compression

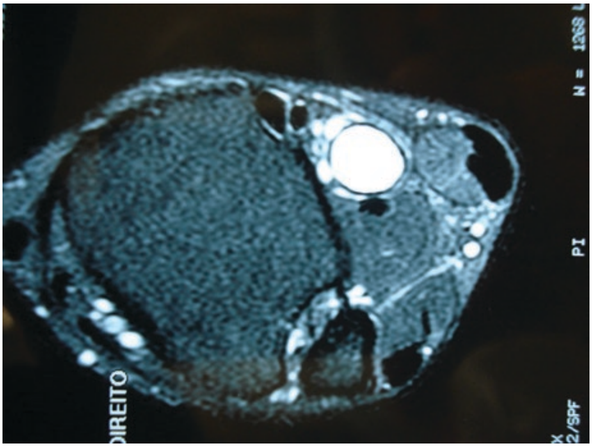

Fig. 4 Posterior tibial nerve cyst in the tarsal tunnel causing a tarsal tunnel syndrome

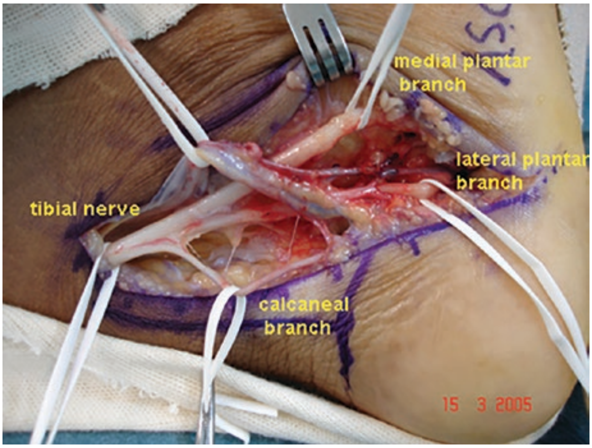

Fig. 5 Intraoperative photo demonstrating the tibial nerve and its branches (calcaneal, lateral plantar, and medial plantar nerves)

Conservative treatment includes immobilization, use of analgesics and anti-inflammatory drugs, insoles to partially correct a hindfoot valgus, and muscle rebalancing exercises. Corticoid infiltrations guided by ultrasonography can help as well.

Surgical treatment with tarsal tunnel decompression is reserved for cases of failure of conservative treatment.

The decompression is performed through a posteromedial incision, following the nerve trajectory, and includes release of the retinaculum (lancinate ligament – proximal tunnel) and the tibial nerve and its branches (distal tunnel), taking care to release even the nerve to the abductor digiti minimi, which can be compressed at the medial edge of the plantar fascia (Fig. 5). The results are good, especially when a cause is identified and treated (extrinsic compression). In so-called idiopathic cases, the prognosis is less favorable [7].

2.8 First Branch of the Lateral Plantar Nerve: Baxter Nerve

It is the motor nerve for the abductor digiti minimi muscle or calcaneal inferior nerve. It originates in most cases from the lateral plantar nerve, but it can be a direct branch of the tibial nerve as well, in the lower portion of the tarsal tunnel. It traverses into the space between the quadratus plantae muscle and the abductor hallucis muscle. It innervates the quadratus plantae, flexor digitorum brevis (FDB), and the abductor digiti minimi muscles.

It can be a source of pain in athletes, especially runners, dancers, and gymnasts. It should be considered in the differential diagnosis of sub-calcaneal pain in athletes, along with plantar fasciitis, calcaneal fat pad atrophy, apophysitis, and calcaneal stress fractures [4]. The nerve is most commonly compressed between the abductor hallucis fascia and the quadratus plantae muscle, but compression may also occur between the FDB and the calcaneus.

Clinically, the patient typically presents with medial plantar pain in proximal portion of the plantar fascia. Tinel's sign may be present, along with burning pain irradiated to the lateral and central portion of the hindfoot. Characteristically, the patient complains of tenderness at a point located 5 cm distal from the posterior edge of the calcaneus at the transition of the dorsal and plantar skin on the medial, central, and/or lateral surface of the heel. Causes of compression include hypermobile foot, abductor hallucis, and quadratus plantae hypertrophy, presence of accessory muscles, and thickening of the proximal portion of the plantar fascia (Fig. 6) [12]. Electroneuromyography is often able to detect focal neurologic abnormalities, but the exact sensitivity of the examination is uncertain [27]. Magnetic resonance imaging may show signs of atrophy of the abductor digiti minimi.

Conservative treatment with orthoses, arch-supported insoles, and strengthening of the plantar musculature has satisfactory results in up to 90% of patients, and

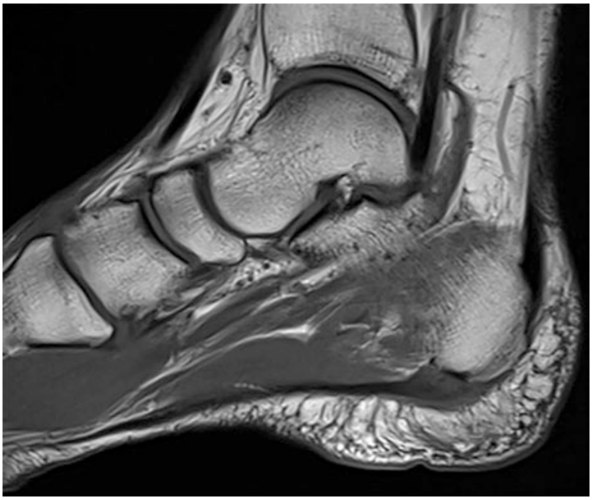

Fig. 6 T1-weighted sagittal resonance imaging, showing thickening of the proximal portion of the plantar fascia, causing symptoms related to Baxter nerve compression

Fig. 7 Intraoperative photograph demonstrating the release of Baxter's nerve

should be attempted for a period of 12–20 months [30]. In case of failure of conservative treatment, the release of the nerve associated with plantar fasciotomy has a satisfaction rate higher than 85% [11]. The procedure can be performed openly (Fig. 7) or arthroscopically.

2.9 Medial Plantar Nerve

After passing through a single osteofibrous tunnel in the tarsal canal, the medial plantar nerve follows distally along the medial surface of the foot, between the abductor hallux muscle and the FDL. At the height of Henri's knot, it is divided into digital branches for the three medial toes. It is responsible for the motor innervation of the abductor hallucis (AbH), flexor hallucis brevis (FHB), FDB, and first lumbrical. It is responsible for the sensation in the medial plantar portion of the forefoot, the two medial toes, and the medial aspect of the third toe.

The compressive pathology that affects this nerve is known as the runner foot. It occurs most commonly at the level of Henri's knot. It may occur by extrinsic compression (raised arch insole), or intrinsic (between the AbH fascia and its origin in the calcaneus, by AbH hypertrophy, running, hyperpronation, or hindfoot valgus) [25]. Iatrogenic lesions of this nerve related to previous surgical procedures in the medial plantar region of the foot should be remembered as well.

Compression causes neuropathic pain in the arch, usually related to exercises, with irradiation to the medial toes and changes in sensitivity in its territory. The symptom worsens with the forefoot support, which tensions or compresses the AbH. Investigation with magnetic resonance imaging is mandatory and can identify atrophy of the AbH.

Conservative treatment should be attempted initially, with use of anti-inflammatory drugs, infiltrations, physiotherapy, changes in training pattern, and

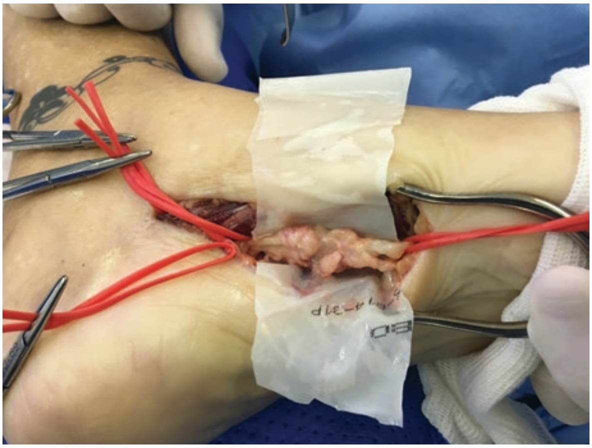

Fig. 8 Image of surgical dissection of large neuroma of the medial plantar nerve caused by iatrogenic lesion in previous surgery to obtain the flexor hallucis longus tendon, which was used to reconstruct a calcaneal tendon lesion

correction of the hindfoot alignment with shoes or insoles. If conservative treatment fails, surgical decompression is indicated through a medial incision in the foot, with release of the medial plantar nerve up to Henri’s knot (Fig. 8). The literature is scarce in the description of the results of this treatment.

2.10 Medial Digital Nerve of the Hallux

The so-called Joplin neuritis occurs by irritation of the medial digital branch of the hallux at the level of the medial sesamoid or hallux metatarsophalangeal joint, where it exits the fascia of the AbH. On physical examination, local compression causes pain. Often this pathology can be confused with medial sesamoiditis. Conservative treatment is usually indicated, with the use of silicone cushioning in footwear to relieve the overload. When it fails, surgical release by medial approach is recommended [13]. This neuritis can occur in hallux valgus patients as well given the medial prominence.

2.11 Interdigital Nerves

Interdigital nerves are branches of the lateral and medial plantar nerves and are located below the transverse intermetatarsal ligament. The nerve that occupies the third interdigital space is formed by the union of terminal branches of the medial and lateral plantar nerves, this being the most common site of involvement of these nerves.

Compressive neuritis causes the formation of a nervous thickening, known as Morton’s neuroma, which corresponds to a neuralgia caused by perineural fibrosis [5].

Clinically, it manifests as pain related to the use of narrow shoes, associated with a burning sensation or paresthesia in the toes. Pain on palpation of the affected space is present, and the compression of the metatarsal heads causes worsening of the pain and clicking sensation in the space occupied by the neuroma (Mulder positive test).

The conservative treatment is based on the correction of footwear, lidocaine infiltrations, corticoids, or sclerosing agents with or without ultrasonography guidance. The results are satisfactory in the early stages of the pathology. Surgical treatment with neuroma resection is indicated in cases refractory to conservative treatment. The procedure is usually performed through the dorsal route (Fig. 9). The plantar route is recommended for revision cases. The nerve section should be performed as proximal as possible to prevent recurrence of symptoms, typically caused by plantar branches that were not resected (a minimum of 4 cm nerve resection is recommended). The resection prognosis is very good, with up to 85% satisfactory results (for more information, go to Morton's neuroma chapter).

2.12 Diabetic Neuropathy

Neuropathy caused by diabetes causes symptoms of neuropathic pain, which must be differentiated from peripheral compressive pathologies. In the literature, there are indications that early decompression of nerves affected by diabetic neuropathy diminishes the consequences in the medium and long term, decreasing the incidence of plantar ulcerations, a situation of difficult control that can lead patients to chronic infection and amputations [1].

There is evidence in the literature that young patients, with positive Tinel sign, adequate metabolic control, and no major sensitivity changes, are those who respond best to surgical decompression [26].

Fig. 9 Intraoperative photograph demonstrating the presence of interdigital neuroma in the third intermetatarsal space (Morton's neuroma)

3 Summary

Compressive neuropathies are common and can have varied clinical presentations, which make clinical diagnosis difficult.

Small incidence of these compressive peripheral nerve pathologies must be related to lack of diagnosis, or late diagnosis, and it may affect negatively treatment prognosis.

The knowledge of these pathologies, its symptoms and presentations, the importance of the adequate evaluation through physical examination, imaging, and electrodiagnosis facilitate its identification, and as a consequence, its adequate treatment.

The patient should be well oriented regarding the pathology and prognosis of the various forms of treatment, since the results are not homogeneous, and sequelae can remain.

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