Entrapment syndromes of the foot and ankle

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ABSTRACT

Neuropathies are under-diagnosed conditions, although their diagnosis and treatment could improve tremendously the quality of life of a patient. The compression syndromes reviewed in this article are the 6 most common neuropathies of the foot and the ankle, namely, the posterior and anterior tarsal tunnel syndromes, Baxter's nerve entrapment, the medial plantar nerve and the superficial peroneal nerve entrapment syndromes and Morton's neuroma. We reviewed published medical articles in order to gather information and obtain a broader understanding of these syndromes, in the interest of an adequate and successful management.

In this review paper, we will discuss elemental information on the anatomy of the nerve subject to compression, the possible aetiologies of the syndrome and the symptoms that would result from it. The diagnostic methods and treatments options available to us nowadays would, afterwards, be discussed and interpreted.

From this paper, we would highlight that the early diagnosis of a compression syndrome, by virtue of imagery or newly designed clinical tests, would significantly improve the outcome of a surgical treatment. This paper allows an integrative and comparative update on these syndromes.

KANALIKULARNI SINDROMI U PODRUČJU STOPALA I GLEŽNJA

SAŽETAK

Neuropatije su nedovoljno prepoznata stanja, iako njihova dijagnoza i liječenje mogu značajno poboljšati kvalitetu života pacijenta. Sindromi kompresije za koje je pružen osvrt u ovom članku su šest najčešćih neuropatija stopala i gležnja, tj., stražnji i prednji tarsalni sindrom tunela, Baxterova kompresija živaca, srednji planarni živac i površinski sindromi za uklještenje peronealnog živca te Mortonov neurom. Napravljen je osvrt na objavljene medicinske članke kako bismo prikupili informacije i dobili šire razumijevanje ovih sindroma, u interesu primjerenog i uspješnog liječenja.

U ovom radu raspravljat ćemo o osnovnim informacijama o anatomiji živca koji je podložan kompresiji, mogućim etiologijama sindroma i simptomima koji bi mogli proizaći iz njega. U kasnijem dijelu ovoga rada razmotriti ćemo i protumačiti dijagnostičke metode i mogućnosti liječenja koje su nam dostupne danas.

U ovom radu ističemo da bi rana dijagnoza kompresijskog sindroma, na temelju slika ili novo dizajniranih kliničkih ispitivanja, značajno poboljšala ishod kirurškog liječenja. Ovaj rad pruža intergravitne i najnovije informacije za usporedbu ovih sindroma.

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ABBREVIATIONS

PTTS	posterior tarsal tunnel syndrome
PTN	posterior tibial nerve
FHL	flexor hallucis longus muscle
FDL	flexor digitorum longus muscle
PT	posterior tibialis muscle
FDAL	flexor digitorum accessorius longus muscle
TCST	triple compression stress test
MTP	metatarso-phalangeal joint
MRI	magnetic resonance imaging
ADM	abductor digiti minimi muscle of the foot
EMG	electromyography
NCS	nerve conduction study
ATTS	anterior tarsal tunnel syndrome
DPN	deep peroneal nerve
EDB	extensor digitorum brevis muscle
EDL	extensor digitorum longus muscle
LPN	lateral plantar nerve
QP	quadratus plantae muscle
MPN	medial plantar nerve
SPN	superficial peroneal nerve
IDN	inter-digital nerve

1. INTRODUCTION

Because the peripheral nerves of the lower limb pass through fibro-osseous tunnels, they are at high risk for entrapment or compression, especially in the case of local obstruction, as with ganglions or osteophytic spurs. The general complaint of patients with entrapment syndromes would be an unpleasant tingling, pain or numbness. These would be intermittent and related to specific postures, which compromise the nerve. Because of overuse, the athletic population is more susceptible to compression than the general population, particularly in the case of an inadequate or a poor mechanism of running.

In this article, we will review 6 different entrapment syndromes of the foot and the ankle: the posterior and anterior tarsal tunnel syndromes, Baxter's nerve entrapment, the medial plantar nerve and the superficial peroneal nerve entrapment syndromes and Morton's neuroma. The paper will, firstly, give some important anatomy points and describes the signs and symptoms related to these conditions. Then, will follow the different diagnostics methods used for each syndrome. We will end each section with the treatment options and their respective outcomes.

On my 6th year of medical school, I followed an elective course on nerve compressions of the upper limb and was impressed by how an efficient diagnostic and a relatively simple surgery could ameliorate the quality of life of a patient. I was motivated to write this review since entrapment neuropathies of the foot and ankle are often overlooked and misdiagnosed. Their successful diagnostic and treatment are highly valuable, as it can improve the quality of life of the patients, notably in an active population.

2. METHODOLOGY

An online search for sources using advanced search options was performed using PubMed database in January 2018.

The MeSH terms used were:

“Posterior tarsal tunnel syndrome AND Treatment AND Outcome”

“Anterior tarsal tunnel syndrome AND Treatment AND Outcome”

“Baxter’s nerve entrapment AND Treatment AND Outcome”

“First branch of the lateral plantar nerve compression AND Treatment AND Outcome”

“Jogger’s Foot neuropathy”

“Medial plantar nerve entrapment AND Treatment AND Outcome”

“Superficial peroneal nerve entrapment AND Treatment AND Outcome”

“Morton’s neuroma AND Treatment AND Outcome”

After a more detailed examination of these articles 21 final articles will be used for this review. Only article published after 2000 and in English language have been selected.

3. RESULTS

A. POSTERIOR TARSAL TUNNEL SYNDROME

a. Anatomy

The PTTS is the compressive neuropathy of the PTN or one of its branches, the medial and lateral plantar nerves. The tarsal tunnel a busy passage for the PTN, the posterior tibial artery and vein, and the tendon for FHL, FDL and PT muscles. The roof of the tarsal tunnel is made by the flexor retinaculum, which also forms its superior and inferior margins. The floor of the tunnel is made from the medial wall of the talus, calcaneus and the distal medial aspect of the tibia.

The PTN originates from the sciatic nerve, which itself comes from the spinal cord segments L4 through S2. The sciatic nerve divides in the popliteal fossa into the tibial and common peroneal nerves. The largest one, the tibial nerve, goes in between the two heads of the gastrocnemius muscle, and then, continues to the medial aspect of the leg, on the way to the ankle. From cadaveric autopsies, it has been shown that in 93 to 95% of cases the tibial nerve divides into lateral and medial nerves while still in the tarsal tunnel and in only 5 to 7%, it bifurcates proximally to the tarsal tunnel (1). The two PTN branches exit the tarsal tunnel, then penetrate a 1cm length of fatty tissue and then enter their own tunnels. The increased cross-sectional area of the two branches passing through the tarsal tunnel may be a reason for increased risk of PTTS (1).

b. Aetiologies

An estimation of 60 to 80% of the cases of PTTS has a specific cause identified (1). The factor causing the TTS are classified as: intrinsic, extrinsic or both. The intrinsic causes, which are factors found within the tunnel itself, range from: space-occupying lesions such as varicosities (most commonly), lipoma, ganglia, tumour,

neuroma, osteophytes, hypertrophic retinaculum, fibrosis, synovitis, adhesions, medial talocalcaneal bar and tendinopathies (2,3). Rodriguez et al. (4) reported the case of another type of space-occupying lesions, the combination of a hypertrophic long distally extended belly of the FHL muscle and a repetitive ankle motion. The debulking of the muscle resolved the symptoms of TTS in their patient.

The extrinsic factors, or factors from outside the tarsal tunnel, include: direct trauma, compression from footwear, hindfoot valgus or varus, generalized limb oedema, post-traumatic perineural fibrosis or post-surgical scarring and systemic inflammatory arthropathies, as well as diabetes.

Supernumerary or accessory muscles present in the posteromedial aspect of the leg is a well-known occurrence, but the FDAL muscle as being a cause of PTTS is often disregarded. Deleu et al. (5) reported that the prevalence of the FDAL muscle is in up to 12% of patients.

c. Signs and symptoms

The patients with TTS present with neuritic symptoms on the plantar aspect of the foot such as: sensory disturbances and localized or radiating pain. The pain is commonly confined to the plantar aspect of the longitudinal arch and the heel. It may present as: a burning sensation, paresthesias, tingling or numbness. Disturbances in the perception of coldness and tightness around the foot might also be present. The most predominant symptom of PTTS would be pain directed over the tarsal tunnel, behind the malleolus.

The worsening of symptoms occurs after a day of prolonged walking or even standing but may also be present at night, especially after weight bearing activities throughout the daytime. The patients may find relief with rest and elevation of the lower limb.

The “dorsiflexion-eversion test”, made by Kinoshita et al. (6), is performed by maximally dorsiflexing the ankle and fully extending the toes, in order to stretch and compressed the PTN beneath the flexor retinaculum. This position should be held for 5 to 10 seconds, while the physician simultaneously palpates for tenderness on the PTN. The test is positive if the patient complains of an increase in tenderness on the PTN. This study showed that, numbness and pain should not be considered in this test, as their sensitivities were too low. Contrarily, the sensitivity for tenderness is 93% and specificity of the test is 100%, giving it a strong clinical value.

Abouelela et al. (7) designed a new “triple compression stress test” in order to elicit a positive sign in PTTS. The foot is kept maximally inverted and in full plantar flexion while the physician applies a continuous pressure posteriorly to the medial malleolus. The test is positive if the patient complains about numbness or pain around the heel. Their results expressed that the TCST was positive in 61 out of 65 (93.5%) symptomatic feet for PTTS. It also provoked symptoms in 6 new asymptomatic feet. The clinical TCST sensitivity is 85.9% and its specificity is 100%, making it highly reliable.

d. Diagnostic methods

As the symptoms mimic other lower extremities conditions, such as plantar fasciitis and stress fractures, PTTS often go under-diagnosed. The false negative electro-diagnostic tests contribute to this, although it is the most common chronic compression neuropathy of the lower leg.

The electrodiagnostic studies have been the gold standard in the diagnosis of PTTS. In PTTS, the motor fibres of the medial and lateral plantar nerves reveal prolonged latencies in AH muscle and reduced muscle action potential in ADM muscle (3). Nerve conduction studies and electromyography may be assessed for the confirmation and the evaluation of the severity of the condition. But because it is not

uncommon for them to be false negative, such a result should not exclude the diagnosis of PTTS. In their clinical report Rodriguez et al. (4) explained that it might actually be more appropriate to perform EMG studies after an exhausting exercise. The EMG and NCS are useful in helping distinguish patients with PTTS and patients with a compression of the first sacral nerve root (3).

Ultrasound may be used to detect a mass occupying space in the tarsal tunnel, such as: ganglia, lipoma or varicose veins. It can also reveal talocalcaneal coalition and tenosynovitis (3). A negative ultrasound should not be sole tool in the diagnosis of PTTS, as Samarawickrama et al. (8), reported an abnormal nerve ultrasound in all 9 cases of electrophysiologically confirmed PTTS.

Plain X-rays of the ankle are useful in demonstrating structural abnormalities such as hindfoot varus or valgus, tarsal coalitions, osteophytes and evidence of previous trauma. The MRI adds further detail and is highly accurate (83%) when investigating space-occupying lesions (4).

A relatively newer procedure, called the pressure-specified sensory device (PSSD), allows for an earlier diagnosis on the progression of the PTTS (1).

e. Management and outcomes

The conservative treatments options range from taping, bracing, icing, massage to ultrasound and aspiration of the ganglia. A pressure decrease on the nerve may be performed by shoe modifications and the use of orthotics. It may also involve anti-inflammatory medication, such as corticosteroids injections for the reversal of the intra-neural oedema. Immobilisation or night splint and boot walker may be supplemented, and some activity modifications with progressive mobilisation exercises promoted. Physiotherapy may be recommended but the evidence of its effectiveness is lacking (3).

The surgical procedure is indicated after a failed course of conservative treatments. Reichert et al. (9) recalled that the operative treatment of the PTTS has shown sub-optimal results, from 44% to 96% of satisfaction. The reason for such a wide range of satisfaction may come from the selection of patients, the time between diagnosis and surgery and the type of operative technique used. The best results are achieved if the patient is having surgery within 6 months after onset of symptoms and the surgery would mostly be ineffective if the surgery were to be delayed for more than 10 months (9).

Reichert et al. (9) reported that two of the most important factors relevant to the outcome of the surgery are: the cause of the PTTS and the presence of a positive Tinel's sign (tingling elicited by tapping over the nerve). They also presented that the results of treatment in patients with idiopathic PTTS were the weakest. In patients with external causes they reported 82% of improvement and with internal causes the results were positive in 89% (9). Low level of satisfaction of PTTS surgical release may be due to the poor understanding of the detailed anatomy of the tarsal tunnel. For instance, the three defined though fascial septa in the sole of the foot may be compression sites for the PTN and its branches, in addition to the flexor retinaculum (15). Preoperative localization of the site of the nerve compression may lead to better outcomes.

B. ANTERIOR TARSAL TUNNEL SYNDROME

a. Anatomy

The ATTS is a chronic entrapment neuropathy involving the DPN beneath the thin Y-shaped inferior extensor retinaculum (or cruciate ligament) on the top of the ankle. The inferior extensor retinaculum forms the roof of the route covered by the DPN at the ankle, before dividing in its lateral (motor) and medial (sensory) branches. The

floor of the tunnel is the fascia overlying the talus and navicular bones. In the tunnel there are four tendons, the dorsal pedis artery, the anterior tibial veins and the DPN. Therefore, the anterior tarsal tunnel is a flattened and a relatively unprotected space in the ankle (14).

b. Aetiologies

The most common cause of compression is trauma to the dorsum of the foot (fractures, subluxation or sprains), which could cause local fibrosis or modify the configuration of the tunnel. The ATTS may also be related to a talanavicular osteophytosis, a fibrous band or the wear of high-heeled and tight fitting shoes.

It may also be caused by: localized oedema, long-standing abnormal posture, non-neurogenic pes cavus and ganglion in various combinations.

c. Signs and symptoms

The DPN compression results in dull pain in the dorsum of the foot, with numbness and paresthesias extending to the first dorsal web space. The symptoms seem to worsen with plantar flexion or inactivity. Symptoms may progress proximally and exacerbate at night. In extremely serious cases, weakness of toe extension and atrophy of the EDB muscle may appear (10).

d. Diagnostic methods

Although it is a rare condition, the DPN entrapment remains poorly diagnosed. A careful history and physical examination should be performed. The EDB muscle may appear atrophied and weaken. A positive Tinel's sign may be elicited by taping over the superior and inferior retinaculum along the course of the DPN.

The final diagnosis can be confirmed by carrying out an EMG and NCS.

A radiographic evaluation is critical in the work-up, as the most common causes of ATTS are trauma and impingement of the nerve by osteophytes around the talonavicular joint. A MRI may be used if a space-occupying lesion is suspected (11).

e. Management and outcomes

Conservative measures to treat ATTS are: shoe-wear modifications and custom made orthotics or padding, and corticosteroids/lidocaine injections at the site of entrapment (14).

The surgical release of the nerve has been performed by open surgery, although recently Lui (11) reported successful surgical treatment of the ATT by endoscopic technique. His method preserved the inferior extensor retinaculum and seemed less traumatic then a traditional surgical release. Nevertheless, Lui (11) stated a possible complication, the compression injury of the DPN as the endoscopic instruments are inadequate for this narrow tunnel and may result in a temporary increase of paresthesia.

C. BAXTERS'S NERVE ENTRAPMENT

a. Anatomy

The PTN divides into medial and lateral plantar nerves in the tarsal tunnel, after exiting the tunnel they pass along to the plantar aspect of the foot. The Baxter's nerve or the first branch of the lateral plantar nerve (also known as the inferior calcaneal nerve) originates from the LPN at various levels beneath the deep fascia of the AH muscle. It courses between the AH and the QP muscles, then makes a sharp turn and runs to the lateral underneath side of the calcaneus to supply the ADM muscle.

b. Aetiologies

The Baxter's nerve entrapment is fairly common in gymnasts, runners and ballerina (14). Multiple etiologic factors have been proposed, including stretching of the nerve in running athletes, muscle hypertrophy, bone spurs and bursitis. The entrapment of the Baxter's nerve is due to hypermobility in pronated position of the foot, hypertrophic AH and QP muscles, or in the presence of an accessory muscle (12).

c. Signs and symptoms

The compression of Baxter's nerve presents as a chronic medial plantar heel pain, frequently similar in location to that of plantar fasciitis. However, in contrast to it, symptoms are more proximal and medial, tend to worsen with activity and may be exacerbated with eversion and abduction of the foot. Night pain is rare but 25% of patients complain of severe pain in the morning. The pathognomonic sign of Baxter's nerve entrapment is a maximal pain on the medial heel, superior to the plantar fascia origin and parallel to the posterior tibia (12).

d. Diagnostic methods

Plain radiographs may reveal underlying bony and structural abnormalities, and electro-diagnostic studies may assist with confirming the diagnosis and determining the exact compression site.

The chronic compression of Baxter's nerve may results in atrophy of the ADQ, but it is primarily a clinical diagnosis. Plantar fasciitis is commonly co-existing with Baxter's nerve entrapment syndrome as it is present in 52.5% of cases with MRI finding of ADQ atrophy, compared to in only 6.3% of the general population (13).

e. Management and outcomes

The non-operative options for Baxter's nerve entrapment syndrome are: steroid injections, physical therapy or orthotics, in order to correct the hyper-pronation of the feet. An approximate of 90% of patients improved with conservative treatment alone (14). For Ferkel et al. (14) non-surgical measures are recommended for at least a period of 12 months. For Lareau et al. (13), if symptoms persist for more than 3 months and all conservative approaches have been unsuccessful, a surgical decompression is recommended.

The surgical decompression consists of the release of the superficial and deep fascia of the AH muscle and present with excellent to good results in up to 93% of the cases (14).

Lareau et al. (13) believe that there might be a need for an extensive decompression with the release of the FDB and QP muscles, if they appear having a direct effect on the course of the LPN.

D. MEDIAL PLANTAR NERVE ENTRAPMENT

a. Anatomy

The MPN is the larger of the two terminal divisions of the PTN. It enters the foot midways between the medial malleolus and the medial tubercle of the calcaneus under the flexor retinaculum. The MPN then passes deep to the AH and FDB muscles.

The MPN may be compressed between the AH fascia and its origin at the navicular and calcaneus bones, between the AH muscle belly and the knot of Henry, or while it passes through the medial intermuscular septum.

b. Aetiologies

The MPN entrapment is also known as the “jogger’s foot syndrome” as it presents, typically, in long distance runners. Athletes with valgus hind feet may be more susceptible to this syndrome (14).

The internal factor that could cause the MPN entrapment is the direct compression from adjacent muscles such as the FDL and FHL and from the knot of Henry. The medial arch compression from orthotic footwear is a potential external factor for the MPN entrapment (14).

c. Signs and symptoms

Patients report exercise-induced pain on the medial plantar surface of the foot. The pain often radiates distally to the plantar surface of the first, second and third toes and may extend proximally into the medial heel and the ankle (14). The MPN entrapment causes the loss of sensation over the anteromedial sole of the foot, with some radiation towards the medial arch and dysesthesia over the heel (12). The symptoms worsen with physical activity and with eversion of the foot, as it tightens the AH muscle (14).

d. Diagnostic methods

The MPN entrapment should be considered in a patient with a burning medial heel pain, a longitudinal arch aching, and medial sole paresthesias (12). The physical examination findings include: a positive Tinel’s sign on the plantar border of the navicular tuberosity and some dysesthesia along the heel, the medial arch and the first through third toes (15).

Radiographs are necessary to rule out bony abnormalities and to assist in diagnosing causative deformities of the foot in order to make sure that the foot and heel are appropriately aligned.

The MRI findings may include: muscle denervation, oedema or atrophy of the AH, FDB and FHB muscles, and some space occupying masses between the AH and the FDB muscles. A nerve block can confirm the diagnosis (14,15).

e. Management and outcomes

The conservative measures to treat "jogger's foot" are: rest, NSAIDs, corticosteroids injections, training modification, physical therapy and removal or modifying of rigid orthotics. For Peck et al. (12) non-operative treatment is often indicated and successful.

If there is no improvement with conservative methods, surgery should be performed. It consists of the release of the superficial and deep fascia of the AH muscle, followed by exposing the knot of Henry to release the calcaneonavicular ligament (14). Peck et al. (12) reported successful surgical releases in refractory cases and stated that it is, generally, a distal extension of the surgery performed for the PTTS.

E. SUPERFICIAL PERONEAL NERVE ENTRAPMENT

a. Anatomy

The SPN courses in the lateral compartment of the lower leg between the peroneus longus and brevis muscles. It exits the fascia approximately 12.5 cm proximal to the tip of the lateral malleolus and then becomes subcutaneous.

e. Aetiologies

The SPN entrapment typically results from chronic ankle sprains or as the nerve tethers during a forceful inversion or plantar flexion, or following a trauma (14).

Others causes are space-occupying masses such as ganglion, cysts, tumours (for instance schwannomas or lipomas) (14). It can also result from the thickening or a defect of the deep fascia of the leg or by herniation of a muscle (15).

In addition, factors of SPN entrapment reported are: healing fractures sites, anorexia nervosa and wearing tight high boots (16).

f. Signs and symptoms

The entrapment of the SPN results in chronic pain over the dorsal surface of the ankle and at the lower quarter of the lateral side of the leg. The symptoms worsen with physical activities and are relieved by rest (15). Pain may be elicited by the passive inversion and the plantar flexion at the ankle. Sensory abnormalities such as numbness or tingling may appear over the dorsum of the foot (14).

A positive Tinel's sign may be present along the exit point of the nerve while the foot is in plantar flexion (14).

g. Diagnostic methods

The diagnosis of SPN entrapment is mainly clinical and made by the presence of three positive provocation tests (16). It is imperative to rule out any spinal involvement and to evaluate for a common peroneal nerve entrapment at the fibular head (14).

A MRI can identify any facial defects or nerve compression mass effect that may be present. The NCS can help, but a negative test should not rule out a SPN entrapment (14).

Searching for a positive Tinel's sign and the injection of lidocaine into this point followed by the relief of symptoms is a reliable diagnostic (17).

e. Management and outcomes

The initial management of the SPN entrapment is directed at removing any external factors that may be causing compression and stabilizing any instability that may put tension onto the nerve.

Physical therapy management with soft tissue mobilization, peroneal strengthening and neural mobilization may be considered (14). Reduction of pain with physical therapy was achieved and the complete pain resolution was maintained at the 6 months follow-up in a clinical case reported by Anandkumar (16). Also, avoiding excessive valgus and varus of the hindfoot may reduce the stretching of the nerve and so the accompanying pain.

A surgical procedure is rarely required and when it is, it is a simple decompression of the fascia around the nerve's exit point. Fasciotomies obtain 80% of relief but only 50% of patient's satisfaction (14). Malavolta et al. (17) reported that the surgical treatment relieves the pain completely, however few patients had further sensory discomfort.

F. MORTON'S NEUROMA

a. Anatomy

Morton's neuroma is an IDN pain resulting from a perineural fibrosis and the nerve degeneration of the common digital nerve leading to the toes (14). Morton's neuroma occurs as the nerve passes under the ligament connecting the metatarsals.

Most frequently it develops between the third and fourth toes, in response to irritation or pressure. Because of the anatomy of the IDN complex, the third web space is more prone to develop symptoms. The IDN is compressed by tight inter-metatarsal ligament and the surrounding metatarsals (18).

b. Aetiologies

Morton's neuroma results from a neuropathic compression and is not a true neuroma (14). It can be defined as neuralgia caused by the focal fibrous thickening of any of the IDN branches (19).

The IDN is over-stretched by forced pronation and MTP joint hyperextension as seen with patients wearing high-heeled shoes or narrow shoeboxes (18,20). Morton's neuroma affects ten times more commonly women than men (14).

c. Signs and symptoms

Morton's neuroma presents as a neuropathic dull or sharp pain radiating to the affected web space (18).

The symptoms worsen with: running, standing, walking, toe dorsiflexion and wearing shoes in general. It causes a forefoot pain and metatarsalgia with a burning sensation, numbness, discomfort and the feeling of having a pebble under the metatarsal area (21).

d. Diagnostic methods

On physical examination, the symptoms are reproduced with direct pressure placed plantarly between the metatarsal heads. A clinical diagnosis can be made by provocative tests such as the metatarsal "squeeze test", in which the physician presses the metatarsals together during palpation. A distally radiating pain suggests the diagnosis. The "squeeze test" may also result in a Mulder click, as the neuroma subluxes between the metatarsals. Although, this sign is a rare and unreliable in patients with neuroma (18).

Another method of confirming the diagnosis is to verify whether the patient reports pain relief following an isolated lidocaine injection proximal to the metatarsal head and plantar to the inter-metatarsal ligament or to the web space (14,18).

Ultrasonography and MRI can both reliably identify the neuroma. However, identification of a neuroma with these modalities does not correlate with symptomatology; many asymptomatic patients may also have positive MRI or ultrasonography results. These tests should be used only to confirm the diagnosis after clinical suspicion (14).

Radiographs are used to rule out differential diagnosis, such as osteonecrosis, stress fracture, or arthritis at the MTP joint.

A new electroneurographic procedure performed by Pardal-Fernandez et al. (19) showed high sensitivity and specificity, it was also able to identify symptomatic patients that remained undetected by MRI.

e. Management and outcomes

Non-surgical management options include: custom-made orthoses, metatarsal padding, accommodative footwear, NSAIDs, and steroid injections.

Saygi et al. (21) reported that steroid injections result in 82% of complete or partial pain relief in patients, compared to 63 % of pain relief in alteration of footwear only. Steroids injections as a primary treatment and shoe modifications, with steroid injection at 6 months, appear to give better results in Morton neuromas than shoes modifications alone (21). Furthermore, Saygi et al. (21) concluded that this level of satisfaction could be reached after steroid injections alone. Otherwise, at the 1 year follow up there was no significant benefit found compared to the shoewear modifications. Steroid injections are effective for both the diagnosis and the treatment of Morton's neuromas, as they may act on the etiologic factors (increased fibrosis, inter-metatarsophalangeal bursitis and nerve entrapment). Ferkel et al. (14) reported that multiple steroid injections may lead to an increase in the joint instability and may cause wound healing difficulties. Kennedy et al. (18) warned that the overuse of corticosteroids injections might lead to atrophy of the plantar fat pad and the

degeneration of the collateral ligament. Also, they suggested ultrasound guidance in order to decrease the risk of complications.

A surgical treatment is indicated when non-surgical management fails to provide relief. Historically, the most common surgical intervention is excision of the neuroma and dorsal or plantar approaches show no difference (14). Surgical excision of the neuroma has been proven effective, 70% to 85% of patients have shown improvement after the surgery (14). The IDN and its neuroma should be sent for further histological investigations. The most common complication is the recurrence of pain as a result of inadequate nerve resection or removal of the incorrect tissue (ie, commonly the lumbrical tendon or the digital artery).

REFERENCES

(1) Franson J. Baravarian B. Tarsal tunnel syndrome: a compression neuropathy involving four distinct tunnels. Clin Podiatr Med Surg. 2006;23(3):597-609

(2) Lui TH. Acute Posterior Tarsal Tunnel Syndrome Caused by Gouty Tophus. Foot Ankle Spec. 2015;8(4):320-3

(3) Ahmad M. Tsang K. Mackenney PJ. Adedapo AO. Tarsal tunnel syndrome: A literature review. Foot Ankle Surg. 2012;18(3):149-52

(4) Rodriguez D. Devos Bevernage B. Maldague P. Deleu PA. Leemrijse T. Tarsal tunnel syndrome and flexor hallucis longus tendon hypertrophy. Orthop Traumatol Surg Res. 2010;96(7):829-31

(5) Deleu PA Devos Bevernage B. Birch I. Maldague P. Gombault V. Leemrijse T. Anatomical Characteristics of the Flexor Digitorum Accessorius Longus Muscle and Their Relevance to Tarsal Tunnel Syndrome, a systemic review. J Am Podiatr Med Assoc. 2015;105(4):344-55

(6) Kinoshita M. Okuda R. Morikawa J. Jotoku T. Abe M. The dorsiflexion-eversion test for diagnosis of tarsal tunnel syndrome. J Bone Joint Surg Am. 2001;83-A(12):1835-9

(7) Abouelela AA. Zohiery AK. The triple compression stress test for diagnosis of tarsal tunnel syndrome. Foot (Edinb). 2012;22(3):146-9

(8) Samarawickrama D. Therimadasamy AK. Chan YC. Vijayan J. Wilder-Smith EP. Nerve ultrasound in electrophysiologically verified tarsal tunnel syndrome. Muscle Nerve 2016;53(6):906-12

(9) Reichert P. Zimmer K. Wnukiewicz W. Kulinski S. Mazurek P. Gosk J. Results of surgical treatment of tarsal tunnel syndrome. Foot Ankle Surg. 2015;21(1):26-9

(10) Logullo F. Lupidi F. Di Bella P. Anterior tarsal tunnel syndrome: a misunderstood and misleading entrapment neuropathy. Neurol Sci. 2014;35(5):773-5

(11) Lui TH. Endoscopic Anterior Tarsal Tunnel Release: A case report. J Foot Ankle

Surg. 2014;53(2):186-8

(12) Peck E. Finnoff JT. Smith J. Neuropathies in runners. Clin Sports Med. 2010; 29(3):437-57

(13) Lareau CR. Sawyer GA. Wang JH. DiGiovanni CW. Plantar and medial heel pain: diagnosis and management. J Am Acad Orthop Surg. 2014;22(4):372-80

(14) Ferkel E. Davis WH. Ellington JK. Entrapment Neuropathies of the Foot and Ankle. Clin Sports Med. 2015;34(4):791-801

(15) Beltran LS. Bencardino J. Ghazikhanian V. Beltran J. Entrapment neuropathies III: lower limb. Semin Musculoskelet Radiol. 2010;14(5):501-11

(16) Anandkumar S. Physical therapy management of entrapment of the superficial peroneal nerve in the lower leg: a case report. Physiother Theory Pract. 2012;28(7):552-61

(17) Malavolta M. Malavolta L. Surgery for superficial peroneal nerve entrapment. Oper Orthop Traumatol. 2007;19(5-6):502-10

(18) Kennedy JG. Baxter DE. Nerve disorders in dancers. Clin Sports Med. 2008;27(2):329-34

(19) Pardal-Fernández JM. Palazón-García E. Hernández-Fernández F. de Cabo C. Contribution of a new electrophysiologic test to Morton's neuroma diagnosis. Foot Ankle Surg. 2014;20(2):109-14.

(20) Ormeci T. Güler O. Malkoc M. Keskinbora M. Güngören FZ. Mahirogulları M. Diagnostic Value of Elastrography in Diagnosis of Intermetatarsal Neuroma. J Foot Ankle Surg. 2016;55(4):720-6

(21) Saygi B. Yildirim Y. Saygi EK. Kara H. Esemenli T. Morton neuroma: comparative results of two conservative methods. Foot Ankle Int. 2005;26(7):556-9

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BIOGRAPHY

I was born in Paris, France where I took my primary education and chose the scientific field in high school. I graduated from a Baccalauréat in Sciences, with a major in Physics and Chemistry. I then entered the international program of the Medical School at the University of Zagreb.

I loved studying anatomy and physiology, my favourite aspect being the physiology of sports and the locomotor system. Since early on in my studies I realized that Orthopaedics was the field I was really interested in.

In the summer 2017 I joined the Foundation Team 5, a group of veteran doctors from the U.S army, which travels in remote areas worldwide to give medical care to isolated population. I left for Peru and the Amazon forest, where we set up day clinics in remote villages with the help of the Peruvian Navy. We treated 800 patients and 20 cleft palate surgeries over the course of 9 days. This is so far my most beloved work experience. I hope in the future I will be able to join them again as an orthopaedic surgeon.

As part of the clinical rotations of my final year, I joined the Orthopaedics Department KBC-Salata and the department of Traumatology KBC-Rebro, this experience comforted me in my wish to complete a residency in Orthopaedics and Traumatology.