Clinical Research on Foot & Ankle

Bouysset et al., Clin Res Foot Ankle 2019, 7:1
DOI: 10.4172/2329-910X.1000286

Research Article Open Access

Features and Treatment Outcomes of Feet with Medial Branch Lesion of the Deep Fibular Nerve

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Received date: February 06, 2019; **Accepted date**: March 08, 2019; **Published date**: March 15, 2019

Abstract

Objective: To determine the clinical and ultrasound features of medial branch lesion of the deep fibular nerve and the improvement in symptoms after conservative treatment.

Patients and methods: Patients with sensory symptoms suggestive of deep fibular nerve lesion and at least a trigger zone radiating along the course of the nerve at physical examination were included in the study if the lesion was confirmed by electroneuromyography. Each foot underwent hypoesthesia testing of the first intermetatarsal webspace using light touch with finger and a monofilament; profile weight-bearing x-rays, as well as ultrasonography.

Results: Seventy-eight feet displaying a medial branch lesion of the deep fibular nerve in electroneuromyography were included in the study. Fifty-nine (76%) of these 78 feet had hypoesthesia of the first dorsal intermetatarsal webspace and 34 (44%) a pes cavus foot. Ultrasonography revealed at least one nerve impingement in 40 of these feet (51%). Conservative treatment was effective in 68 feet (87%), including 31/40 feet (78%) with nerve impingement. Ten feet out 78 (13%) had surgical nerve release.

Discussion: The association of trigger point on the nerve pathway and hypoesthesia of the first dorsal intermetatarsal webspace allowed diagnosis of medial branch lesion of the deep fibular nerve in three-quarters of all cases. Imaging should be performed to seek direct impingement on the nerve. However, impingement is often absent and the lesion of the nerve can be caused in this case by extrinsic compression at the foot as well as stretching of the nerve. Remarkably, we showed here that feet with medial branch lesion of the deep fibular nerve most often respond to conservative care, even in the cases of nerve impingement.

Conclusion: These results should encourage physicians in charge of patients experiencing pain of the dorsum of the foot to carefully seek clinical features of the medial branch lesion of the deep fibular nerve.

Keywords: Foot; Medial branch of the deep fibular nerve; Anterior tarsal tunnel syndrome

Introduction

In 1968, Marinacci called "anterior tarsal tunnel syndrome" the suffering of the deep fibular nerve (DFN) at the foot and ankle [1]. Since then, several reports have been published on the subject and Dellon has studied in detail 20 cases of medial branch lesions of the DFN (MDFN) [2].

The deep fibular nerve is one of the two terminal branches of the common fibular nerve. It arises at the head of the fibula and then runs between the tibialis anterior muscle and the extensor hallucis longus muscle. The tendon of this latter muscle overlaps the DFN at the ankle, then the DFN courses under the inferior extensor retinaculum and enters a flat space known as the anterior tarsal tunnel [1]. Within this tunnel course the dorsal artery of the foot and its vein, the DFN, and the tendons of the tibialis anterior, extensor hallucis longus, extensor digitorum longus and fibularis tertius muscles. The DFN divides into lateral and medial branches proximal to the head of the talus, more rarely distal to the head of this last bone [3-6]. The lateral branch provides motor innervation to the extensor digitorum brevis muscle. The MDFN continues onto the medial side of the dorsum of the foot and then passes under the medial bundle of the extensor digitorum brevis tendon. This MDFN is purely sensory and innervates the skin of the first intermetatarsal webspace.

It is important to be aware of MDFN entrapment because it can mimic the symptoms of tarsal osteoarthritis, entrapment of the common fibular nerve at the head of the fibula or L5 radiculopathy. Here, we investigated in patients with electroneuromyographic (ENMG) confirmation of MDFN lesion the importance of certain clinical symptoms and signs in this diagnosis, such as sensory symptoms, testing for trigger points, and ultrasound signs. Weight-bearing lateral radiography of the feet allowed determining static disorders. Functional signs improvement after treatment was also assessed.

Patients and Methods

Patients

Patients with foot pain and symptoms suggestive of MDFN injury, such as burning sensations, paresthesia, electrical sensations on the dorsomedial surface of the foot, were consecutively observed in the consultations of 3 expert practitioners in the management of foot and ankle pathology (MB, JYC, DB). Oral treatments, especially analgesics and anti-inflammatory drugs, sometimes venous tonics, and/or plantar orthoses had not significantly improved the symptoms. The medical history sought local traumatic or microtraumatic triggering factor. Detection of MDFN involvement was based on Mann and Baxter's recommendations for the study of tibial nerve lesions [7]. A first search for trigger points reproducing symptoms spontaneously experienced was performed by each recruiting practitioner. If at least one trigger point was found, an ENMG was performed to confirm the diagnosis of MDFN lesion. Patients were retrospectively included in our study if they presented at least one trigger point and an ENMG lesion of MDFN or the trunk of the DFN. Patients with symptoms suggestive of other foot or ankle neuropathy, general polyneuropathy context, or a context of insurance claims were excluded from the study. The study began in December 2007 and the last assessment of therapeutic outcomes was conducted in November 2012. The ethics committee of our institution has approved the study.

Patients underwent a comprehensive and comparative examination of the feet performed by one practitioner (MB) [2,8,9]. A weight-bearing lateral foot radiograph and an ultrasonography (TT) were performed. Haddad has recommended that "trigger points" should be elicited by direct pressure over the nerve [8]. Pressure was applied with the thumb until the appearance of discoloration under the thumbnail and was maintained for 30 seconds [8]. Proximal or distal radiation of pain along the nerve path reproduced the spontaneous symptoms [8]. Each examined foot displayed at least one trigger point [2,3,10]. These trigger points included the distal neck of the talus corresponding to the anterior tarsal tunnel itself, the navicular bone and the area at the junction of the bases of the 1st and 2nd metatarsals with the cuneiform bones (Figure 1) [2,3,5,10]. Here were considered sensory signs of DFN lesion within the anterior tarsal tunnel itself and of its medial branch. Careful testing for hypoesthesia or anesthesia of the first dorsal intermetatarsal webspace was carried out [2,3,9,11]. The importance of this testing has been underlined by Dellon [2]. Superficially located fibers (including light touch fibers) exhibit an increased level of compression in case of entrapment. It is therefore important to investigate this tactile sensory for diagnosis [5,12]. Light touch perception was assessed using a monofilament 10 gram level 5.07 [6] and finger [2,3,5] in the first dorsal intermetatarsal webspace. The monofilament was in light touch contact with the skin of the area studied. The first search for sensory disorders is often poorly informative. Therefore to be more specific, each foot was tested with three series of three contacts with a delay of about one minute between each series. The light touch of the first intermetatarsal webspace was performed under the same conditions with the pulp of the middle finger. Hypoesthesia was confirmed if a decrease in perception of light touch with monofilament and finger was observed 3 times in succession. Hypoesthesia was sought in comparison with the ipsilateral superior side, which is supplied by the superficial fibular nerve, and with the contralateral foot when possible in the case of unilateral damage [2].

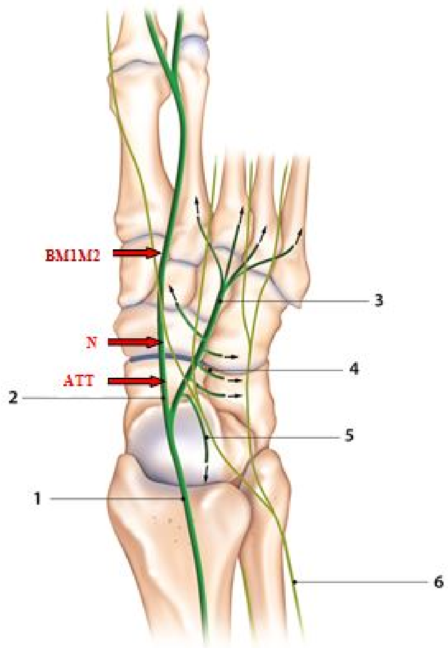

Figure 1: Trigger points on the medial branch of the deep fibular nerve. ATT: Anterior Tarsal Tunnel; N: Navicular Bone; BM1M2, bases of the first and second metatarsal bones. 1. Deep fibular nerve. 2. Medial branch of the deep fibular nerve (MDFN). 3. Lateral branch of the deep fibular nerve (DFN). 4. Branches arising from the lateral branch, one of which penetrates into the tarsal sinus. 5. Recurrent branch of the DFN which supplies the anterolateral capsule of the ankle. 6. Superficial fibular nerve (from « Pathologie ostéo-articulaire du pied et de la cheville » de la clinique aux examens complémentaires, Sauramps Médical Ed. Bouysset M, Delmi M, Morvan G 2014.).

The ENMG was performed by an experienced physician (JD). As it has been previously accepted for detection of tibial nerve lesions [7], ENMG was considered as the theoretical reference to establish a positive diagnosis of a DFN nerve lesion. Motor nerve conduction velocities (MCVs) were measured with a 3 Hz-3 kHz bandwidth. Stimulations were successively performed on the anterior aspect of the bimalleolar line, under and above the head of the fibula, with the reception surface electrode located on the short toe extensor muscle. Sensory nerve conduction velocities (SCVs) were measured orthodromically with a 20 Hz-3 kHz bandwidth. The reception surface electrode was placed on the anterior and medial aspect of the ankle and bipolar stimulation was applied between the first and second metatarsal bones. The distance between stimulation and reception electrodes varied between 8 and 12 centimeters. The weight-bearing lateral radiograph of the feet allows calculation of the medial arch angle formed by the intersection of two lines. The first one joins the inferior pole of the medial sesamoid bone and the lowest point of the talonavicular joint, while the second is formed by joining the latter point and the lowest point of the calcaneus [13]. Static disorders such as flat foot (>130°) and pes cavus foot (<118°) were assessed. Clinical assessment and reading of X-rays were carried out by a single physician (MB). Ultrasonography with foot mobilization during the examination was used to assess nerve impingements. Ultrasonography was performed with high-frequency linear array transducers (13 MHz and 18 MHz) and a small 15 MHz hockey-stick-shaped transducer. Combined B mode and power Doppler images were obtained using the lift technique (dynamic ultrasonography) by rapid movement of the probe from top to bottom and bottom to top. Ultrasonography was used to assess synovitis, tenosynovitis, and subcutaneous tissue inflammation and determine one or several impingements on the nerve. In addition, power Doppler helped distinguish between vascular structures and nerves. The ultrasounds were performed by a single physician (TT).

Treatment

All patients underwent a 6-month conservative treatment to avoid compression and tension of the involved nerve: rest or lower physical activity levels, non-compressive footwear for pressure reduction on the dorsum of the foot, no high heels [2,3,4,14], hyperpronation correction by plantar orthoses [3], use of cushioning protectors that can help to protect the fragile area around the nerve, application of topical non-steroidal anti-inflammatory drugs, anti-edematous therapies sometimes useful in cases of subcutaneous edema [9], wearing of night rest splints to avoid significant plantar flexion of the ankle, which causes compression and stretching of the nerve on the navicular bone [3,5,8]. Trigger point injections with corticosteroids were used as a last resort [5,8,9]. If a patient had significant residual symptoms or functional impairment after 6 months of conservative treatment, surgical indication was considered based on the frequent beneficial outcomes described in published cases [3,4,15]. The patient was then referred to one of the orthopaedic surgeons involved in the study (JYC and DB).

The results of surgical release were estimated between 6 and 12 months postoperatively using a visual analogic scale. Similarly to surgery of tarsal tunnel syndrome by Pfeiffer et al., results were rated using patient assessment of functional discomfort related to pain [16]. These results were classified into 5 grades: 0, no improvement; grade 1, poor result, slight improvement (from 0 to 30%) but important functional discomfort persists in everyday life and analgesic drugs are often used; grade 2, average result, from 31 to 59% improvement but the patient must still be careful in certain movements and with some positions of foot and ankle and analgesic drugs are sometimes used; grade 3, good result, marked improvement (60 to 79%) but some minor and intermittent symptoms persist that do not modify everyday life and do not require medication; grade 4, from 80 to 100% improvement, very good result, the pain has completely resolved.

Results

Seventy-eight feet from 72 patients with a sensory abnormality of MDFN on ENMG were included. Patient characteristics are detailed in Table 1. Fifty-nine of the 78 feet (76%) had hypoesthesia or anesthesia of the first webspace. Radiographs revealed static disorders for 47 feet (60%) with 34/78 pes cavus feet (44%) and 13/78 (17%) flat feet. Ultrasonography showed nerve impingement in 40/78 feet (51%). These impingements were due to osteophytosis (32 cases), an arthrosynovial cyst at the talonavicular joint (4 cases), a talonavicular cyst and an osteophytosis at the same time (3 cases), or an osteophytosis associated with tenosynovitis of the extensor digitorum longus tendon (1 case) (Table 2). Among the 59 feet with hypoesthesia, 30 (51%) had an impingement.

Sex	Male	18
	Female	54
Age (years)	Mean [min-max]	57 [20-78]
Number of feet	Total	78
	Right foot	28
	Left foot	50
Symptom duration (months)	Mean [min-max]	21 [3-84]

Table 1: Medial branch of the deep fibular nerve: 72 patients (78 feet) with positive ENMG and sensory deficit.

Sensory deficit of the 1st webspace	59
Static Disorders	
Pes cavus feet	34
Flat feet	13
Causes of impingements (ultrasonography)	
Total	40
Osteophytes	32
Synovial Cysts	4
Osteophytes + synovial cysts	3
Osteophytes and EDL	1
EDL : Tenosynovitis of Extensor Digitorum Longus Tendon	

Table 2: 78 feet with medial branch lesion of the deep fibular nerve at ENMG.

All patients were treated with non-compressive footwear, cushioning protectors with a layer of cotton around trigger points and night rest splints with around 10% of plantar flexion. Thin plantar orthoses were added in cases of valgus flat foot (17%). A corticosteroid injection was performed close to the trigger point in 26/78 feet. Conservative treatment was effective in a total of 68/78 feet (87%), and remarkably 31/40 feet (78%) with impingement on ultrasonography were improved by conservative treatment. Therefore, only 10/78 feet (13%) needed surgery for MDFN release. Nine of these operated feet had hypoesthesia and 9 had an impingement detected by imaging. The foot without hypoesthesia was from a patient with bilateral MDFN impairment on ENMG. Surgical treatment was indicated for both feet in this patient because of an impingement due to osteophytosis. Eight patients considered their final results very good or good (two of these patients had a second surgical procedure for fibrosis 3 and 6 months later respectively), while two patients had poor results (one had fibromyalgia and the other had incurred a major injury to the dorsum of the foot with possible anatomic lesion of the nerve).

Discussion

Any compressive nerve lesion may cause MDFN symptoms [2,4,5]. Some of these may be caused by extrinsic factors. At the dorsum of the foot, MDFN is especially prone to injury because of its proximity to the bone surface and its very thin protective tissue layer [4]. Nerve compressions occur as a result of either direct or indirect trauma such as fractures, subluxations, ankle sprains, or contusions [1,4,14] or microtrauma injuries on the dorsum of the foot due to tight shoes for example. Subcutaneous edema of the foot and ankle may cause compression in the shoe [14]. Imaging can reveal some causes of compression: Osteophytes on the anterior aspect of the tibia [17], more often dorsal osteophytes of the tarsus and arthrosynovial cysts at the talonavicular joint as found in our study [4,10,15,17]. We also observed in our sample a tenosynovitis of the extensor digitorum longus tendon in a large pes cavus foot in a marathon runner. Other causative lesions have been frequently reported: Os intermetatarseum [18-19], aneurysms [17], lymph nodes in the anterior tarsal tunnel [4], thrombosis of the dorsal artery of the foot [6,20]. The MDFN courses under the medial bundle of the extensor digitorum brevis tendon which can compress it [9,21]. The os intermetatarseum is a relatively uncommon accessory bone of the foot with an estimated frequency from 1.2 to 14% in the literature. Usually located between the bases of the first and second metatarsal bones and in most cases asymptomatic [19], it has not been observed in our cohort. The presence of the os intermetatarseum predisposes to traumatic or microtraumatic lesions of the MDFN. Similarly, pes cavus foot [2,3,5] and dorsal osteophyte of the tarsus, which usually precedes the onset of irritation symptoms, may be predisposing factors of MDFN lesion.

Ultrasonography showed the presence of an impingement in 51% of cases of MDFN lesion, highlighting the possibility of extrinsic nerve compression at the foot but also other causes in the remaining cases. The effects of nerve tension were already cited [2,3]: The nerve is placed under maximum stretch when foot hyperplantar flexed and the toes dorsiflexed, particularly if there is a cavus foot [2]. In effect, it has been observed that repeated stretching of the DFN on the dorsum of the tarsus may promote the development of anterior tarsal tunnel syndrome [22]. High heels cause both stretching and compression of the nerve against the navicular bone with the movements of plantar flexion and hyperextension of the toes [1,3,6,14,23]. These movements may explain for Liu et al. the symptoms of a ballet dancer [4]. Mechanical stresses related to foot static disorders are predisposing factors [2,3,5]. Pes cavus foot is at greater risk of lesion as the longitudinal arch with prominent talo-navicular and cuneo-navicular joints stretches the inferior extensor retinaculum and causes compression. Any element limiting the nerve mobility can lead to injury [4], including scarring fibrosis with disappearance of the protective tissue layer around the nerve [3].

Theoretically, the presence of an impingement on the nerve would have suggested a pejorative result of conservative treatment. Remarkably, conservative treatment achieved improvement in 78% of cases of impingement visualized by ultrasonography with the disappearance of the symptoms and the persistence of the cause. This result underscores the importance of an initial conservative treatment that was often effective in our sample.

The differential diagnosis of DFN neuropathy should exclude other pain on the dorsum of the foot due to osteoarthritis or tendinopathy of the extensor tendons, a superficial fibular nerve lesion that may be associated (sensory defect on the dorsum of the foot except in the first inter-toe webspace), a lesion of the common fibular nerve (tibialis anterior and extensor digitorum longus tendon muscles weakness). L5 radiculopathy and polyneuropathy must also be excluded. These last disorders highlight the importance of the ENMG in eliminating other neuropathies.

Only one foot with a surgical indication did not have hypoesthesia but the patient displayed bilateral MDFN impairment on ENMG. This case suggests that hypoesthesia appeared to be significant in the most affected MDFN, and that the contralateral foot with a less injured MDFN had less sensory disorders on physical examination.

Acknowledgment

We wish to thank Dr. Nadine Charles for her valuable advice.

Role of the Funding Source

There was no funding for this manuscript.

Competing Interests

The authors declare that they have no competing interests.

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