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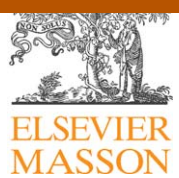

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CLINICAL REPORT

Tarsal tunnel syndrome and flexor hallucis longus tendon hypertrophy☆

D. Rodriguez[b], B. Devos Bevernage[a], P. Maldague[a], P.-A. Deleu[a,*], T. Leemrijse[a]

[a] *Clinique du Parc-Léopold, Foot & Ankle Institute, 38, rue Froissart, 1040 Bruxelles, Belgium*
[b] *Andres Caceres Avenue, 96B, Chiclayo City, Peru*

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KEYWORDS
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Summary Tarsal tunnel syndrome (TTS) defines an entrapment neuropathy of the posterior tibial nerve or one of its branches, within the tarsal tunnel. Numerous etiologies have been described explaining this entrapment, including trauma, space-occupying lesions, foot deformities, etc. We present an unreported cause of a space-occupying lesion in the etiology of TTS, namely the combination of a hypertrophic long distally extended muscle belly of the flexor hallucis longus and repetitive ankle motion. Surgical debulking of the muscle belly in the posterior ankle compartment resolved all symptoms.

Introduction

Tarsal tunnel syndrome (TTS) is an entrapment neuropathy of the posterior tibial nerve or one of its branches, within the tarsal tunnel for which many etiologies have been described, including trauma, space-occupying lesions, foot deformities, etc. [1]. The tarsal tunnel is an area of anatomic narrowing caused by tight ligamentous structures. It is a fibro-osseous tunnel bordered superficially by the flexor retinaculum, which passes obliquely from proximal to distal to anterior. The flexor retinaculum forms the roof of the tarsal tunnel, as well as the superior and inferior margins. The floor of the tunnel is formed by the medial wall of the talus and calcaneus and the distal medial wall of the tibia. The anatomic structures that run within the tarsal tunnel are, from medial to lateral, the posterior tibial tendon, flexor digitorum longus tendon, posterior tibial artery and its accompanying veins, posterior tibial nerve, and the flexor hallucis longus tendon [2,3]. In a literature review of TTS, it was estimated that in 60—80% of cases, the specific etiology can be identified [1]. We report a case of TTS caused by a hypertrophic long distally extended muscle belly of the flexor hallucis longus (FHL) that has been resolved with partial resection of its muscle.

Case presentation

A 32-year-old male, truck driver, presented at the outpatient clinic, complaining about bilateral foot pain, especially on the left side. The pain was described as a burning sensation

☆ Investigation performed at St-Luc Teaching Hospitals, Brussels, Belgium.

* Corresponding author. Tel.: +32 2 787 57 67.
E-mail address: pa.deleu@gmail.com (P.-A. Deleu).

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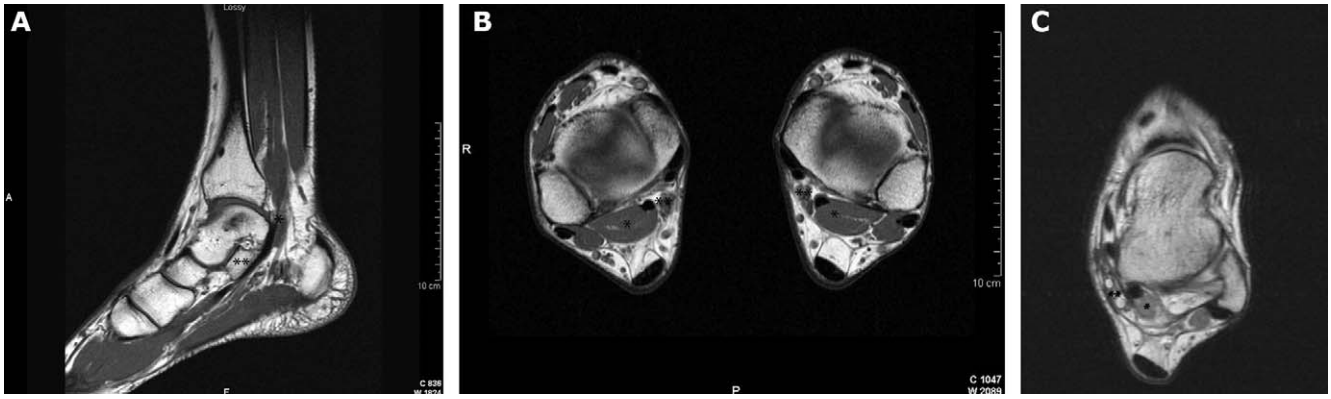

Figure 1 A sagital (A: *: flexor hallucis longus and **: sustentaculum tali) and two axial (B + C: *: Flexor hallucis longus and **: posterior tibial neurovascular muscle) MRI illustrations of the bilateral hypertrophy of the long distally muscle belly of the flexor hallucis longus, up to the sustentaculum tali.

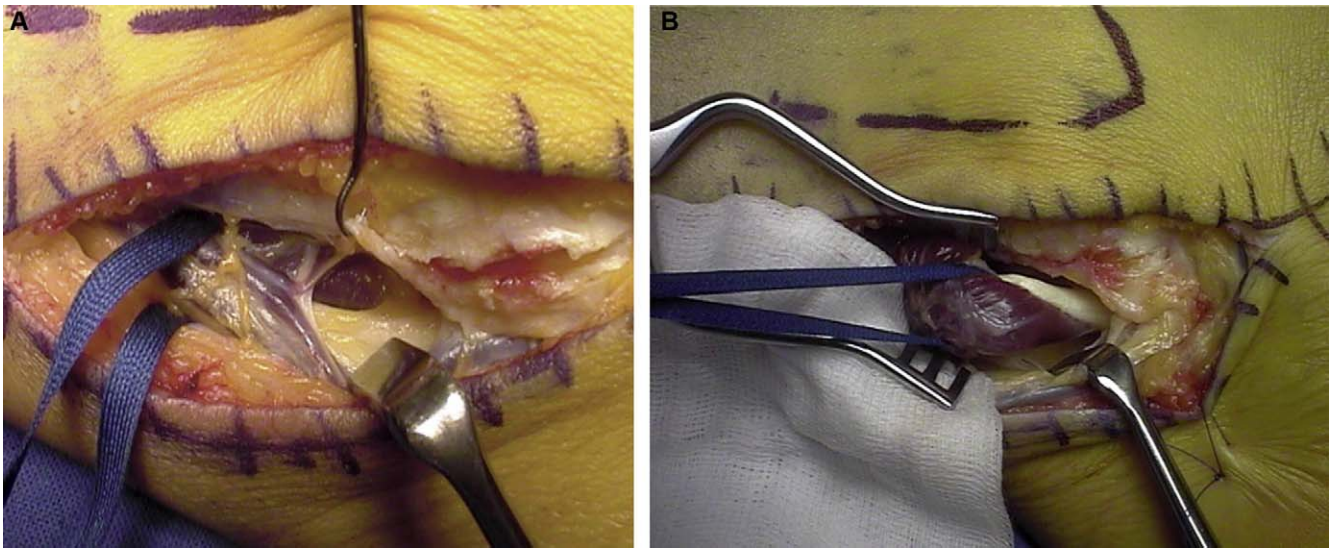

Figure 2 Image A shows the posterior tibial artery, its accompanying veins, and the posterior tibial nerve; image B shows the identification of muscle and tendon of FHL, thicker than normal.

in the internal retromalleolar zone, irradiating to the heel region without paresthesia. This tended to aggravate with plantar flexion, while driving his truck. The symptoms were present for at least 2 years, with worsening over the last several months.

Physical examination revealed bilateral pain on palpation over the posterior tibial nerve directly beneath the flexor retinaculum, with a predominance on the left side. A positive pseudo-Tinel's sign was found. Electromyographic analysis (EMG) was not conclusive; Bonescan was normal; laboratory tests were also normal. MRI showed a bilateral hypertrophic long distally extended muscle belly of the flexor hallucis longus (FHL), descending all the way down to the sustentaculum tali (Fig. 1).

The diagnosis of bilateral TTS was made, based on the compression of the nerve by the hypertrophic FHL muscle belly. Conservative treatment with modifications to his truck (changing from standard to automatic transmission), orthopedic shoes, and avoiding forced plantar flexion seemed effective. He came back only after 3 years, with the same kind of pain but this time, associated to paresthesia. Symptoms were still more intense at the left side. We then proposed a surgical release of the left TTS.

By a conventional medial approach, we explored the tarsal tunnel. No anomalies were found in the posterior tibial tendon, flexor digitorum longus tendon, posterior tibial artery or its accompanying veins. The posterior tibial nerve showed a zone of compression in the proximity of a low, thickened FHL, occupying almost 50% of the tarsal tunnel. The FHL was dissected and we removed the muscle belly so that only the tendon entered the posterior compartment of the ankle. Anatomic pathology confirmed the normality of the resected muscle fibers. The FHL was further inspected to be sure that further no soft tissue impingement was present (Fig. 2).

The early and medium term follow-up period was excellent. At two years, he is still pain-free and returned to his daily activities with some restrictions for sport activities.

Discussion

TTS can be attributed to etiologies that lie within the anatomic confines of the tarsal tunnel, often caused by ''space-occupying lesions'', or to forces or factors external to the tarsal tunnel. The most common space-occupying lesions are varicosities or venous congestion, ganglia, perineural fibrosis, lipoma, neurilemoma, hypertrophic flexor retinaculum, hypertrophic or accessory abductor hallucis muscle, flexor digitorum accessory longus muscle, partial rupture of the flexor hallucis longus tendon, fluid retention and chronic phlebitis. Other etiologies are osseous prominences, tendon pathology, postsurgical scarring [4—13]. Extrinsic factors can be due to trauma, generalized lower extremity edema, heel varus or valgus or the compensation for these hind foot deformities, diabetes. Inflammatory disorders, such as rheumatoid arthritis, ankylosing spondylitis and synovial osteochondromatosis all can produce a proliferate synovitis that exerts a mass effect in the tarsal tunnel [4—13]. TTS can also occur during repetitive motions, such as sprinting, jumping, and others. When sprinting and jumping, dorsiflexion and plantar flexion motions of the ankle joints are repeated quickly and forcibly. During these motions, the tibial nerve in the tarsal tunnel undergoes compression, as the pressure in the tarsal tunnel rises when the ankle is in a dorsiflexed position [14]. In this case, the pain worsened with repetitive plantar flexion, while performing his professional activities. Hence two factors — hypertrophy of the long distally extended of FHL and repetitive motions — contributed to the development of TTS.

The fact that the EMG was not conclusive, can be explained by the dynamic aspect (repetitive motions of the ankle and activity of the FHL) of our case. Takakura et al. reported that 16% of their 31 TTS cases did not present any EMG abnormalities [15]. The authors believe that EMG cannot always be conclusive at rest. EMG studies after an exhausting exercise could be more conclusive.

The outcome of our conservative initial treatment and the surgical management of our patient are very similar to others reports [3,4,6,7]. The short-term follow-up was good; in the literature, the result depends mostly if the etiologic factors have been addressed properly [4]. Moreover, better results are obtained in patients with space-occupying lesions in comparison to those with an idiopathic or post-traumatic etiology or those with foot deformities [16].

Conclusion

We present an unreported cause of a space-occupying lesion in the etiology of TTS, namely the combination of a hypertrophic long distally extended muscle belly of the flexor hallucis longus and repetitive ankle motion. Surgical debulking of the muscle belly in the posterior ankle compartment resolved all symptoms.

Conflict of interest statement

No conflict of interest.

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