



# Tarsal tunnel syndrome—A narrative literature review



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## HIGHLIGHTS

- Aetiology dependent analysis.
- Conservative therapy precedes nerve conduction tests.
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## ABSTRACT

**Background:** Tarsal tunnel syndrome is classified as a focal compressive neuropathy of the posterior tibial nerve or one of its associated branches individually or collectively. The tunnel courses deep to fascia, the flexor retinaculum and within the abductor hallucis muscle of the foot/ankle. The condition is rare and regularly under-diagnosed leading to a range of symptoms affecting the plantar margins of the foot. There are many intervention strategies for treating tarsal tunnel syndrome with limited robust evidence to guide the clinical management of this condition. The role of conservative versus surgical interventions at various stages of the disease process remains unclear, and there is a need for a structured, step-wise approach in treating patients with this syndrome based on derived empirical evidence. This narrative review attempts to scrutinize the literature to date by clarifying initial presentation, investigations and definitive treatment for the purpose of assisting future informed clinical decision and prospective research endeavours.

**Process:** The literature searches that have been incorporated in compiling a rigorous review of this condition have included: the Cochrane Neuromuscular Group's Specialized Register (Cochrane Library 2013), the databases of EMBASE, AMED, MEDLINE, CINAHL, Physiotherapy evidence database (PEDRO), Biomed Central, Science Direct and Trip Database (1972 to the present). Reference listings of located articles were also searched and scrutinized. Authors and experts within the field of lower-limb orthopaedics were contacted to discuss applicable data. Subject-specific criteria searches utilizing the following key terms were performed across all databases: tarsal tunnel syndrome, tibial neuralgia, compression neuropathy syndromes, tibial nerve impingement, tarsal tunnel neuropathy, entrapment tibial nerve, posterior tibial neuropathy. These search strategies were modified with differing databases, adopting specific sensitivity-searching tools and functions unique to each. This search strategy identified 88 journal articles of relevance for this narrative literature review.

**Findings:** This literature review has appraised the clinical significance of tarsal tunnel syndrome, whilst assessing varied management interventions (non-surgical and surgical) for the treatment of this condition in both adults and children. According to our review, there is limited high-level robust evidence to guide and refine the clinical management of tarsal tunnel syndrome. Requirements for small-scaled randomized controlled trials in groups with homogenous aetiology are needed to analyse the effectiveness of specific treatment modalities.

**Conclusions:** It is necessary that further research endeavours be pursued for the clinical understanding, assessment and treatment of tarsal tunnel syndrome. Accordingly, a structured approach to managing patients who have been correctly diagnosed with this condition should be formulated on the basis of empirical evidence where possible.

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## 1. Introduction

Tarsal tunnel syndrome is classified as a focal compressive neuropathy of the posterior tibial nerve or one of its associated branches individually or collectively [1]. The tunnel courses deep to fascia, the flexor retinaculum and within the abductor hallucis muscle of the foot/ankle [2–4]. This rare condition is regularly under-diagnosed leading to a range of symptoms affecting the plantar margins of the foot [5]. There are many intervention strategies for treating tarsal tunnel syndrome with limited robust evidence to guide the clinical management of this condition [4,6]. The role of conservative versus surgical interventions at various stages of the disease process remains unclear, and there is a need for a structured, step-wise approach in treating patients with this syndrome based on derived empirical evidence. This narrative review critically evaluates the literature to date by clarifying the initial presentation, diagnostic investigations and definitive treatment of tarsal tunnel syndrome, for the purpose of assisting future informed clinical decision and prospective research endeavours.

## 2. Literature appraisal

### 2.1. Search strategy

The review is a holistic, comprehensive and definitive analysis of this poorly understood condition and has furthermore considered all levels of evidence pertaining to Tarsal tunnel syndrome to date. Non-English language reports were excluded. The literature searches that have been incorporated in compiling a rigorous review of this condition have included: the Cochrane Neuromuscular Group's Specialized Register (*Cochrane Library* 2013), the databases of EMBASE, AMED, MEDLINE, CINAHL, Physiotherapy evidence database (PEDRO), Biomed Central, Science Direct and Trip Database (1972 to the present). Reference listings of located articles were also searched and scrutinized. Authors and experts within the field of lower-limb orthopaedics were contacted to discuss applicable data. Subject-specific criteria searches utilizing the following key terms were performed across all databases: *tarsal tunnel syndrome, tibial neuralgia, compression neuropathy syndromes, tibial nerve impingement, tarsal tunnel neuropathy, entrapment tibial nerve, posterior tibial neuropathy*. These search strategies were modified with differing databases, adopting specific sensitivity-searching tools and functions unique to each. This search strategy identified 88 journal articles of relevance for this narrative literature review.

### 2.2. Description of the condition

The first clinical description of tarsal tunnel syndrome was believed to be recorded in 1918 by Von Malaise. However, it wasn't until 1962 that Keck and Lam officially described and named the condition in medical literature [7,8]. The condition is typically characterized by increased pressure at the tarsal tunnel confines, often precipitated by any variable that reduces the available space of its boundaries [9–11]. External pressure may impede blood flow in the vessels supplying the tibial nerve causing local ischemia. This phenomenon has an immediate impact on the ability of the nerve axons to transmit action potentials [1,12,13].

Often referred to as posterior tibial neuralgia, this condition may lead to a broad range of symptoms affecting the postero-medial ankle and plantar aspects of the foot [5]. Classic symptoms may include dysesthesia and paraesthesia at all or varied sites of the tarsal tunnel including: the posterior compartment, retro-malleolar flexor retinaculum coverage, and both the anterior and posterior fibro-osseous tunnels branching to the plantar margins

of the foot [6,14]. The incidence of tarsal tunnel syndrome is not known, however there is a greater prevalence in females than males, predominately in the adult population [15–17]. The condition is a relatively uncommon clinical entity, and can often be misdiagnosed in both children and adults due to the clinician's low index of suspicion [4,6,18].

### 2.3. Anatomy

The tarsal tunnel primarily constitutes the osteo-fibrous canal coursing beneath the fan-shaped flexor retinaculum, extending from the postero-inferior medial malleolar region of the ankle joint to the mid-foot [5,19,20]. This structure is composed of both osseous and muscular anatomy and can be defined by its anatomical boundaries [21]. The floor or inferior border of the tarsal tunnel is comprised of the medial walls of the distal tibia, talus, sustentaculum tali and calcaneum bodies, respectively [5,22]. The superficial margin is marked by the flexor retinaculum which courses infero-posteriorly adjacent to the medial malleolus [23]. The distal margin of the tunnel is referred to as the porta pedis [24]. The respective anatomy that channels within the tarsal tunnel from antero-medial to postero-lateral is inclusive of: the tibialis posterior tendon, flexor digitorum longus tendon, posterior tibial artery and veins, posterior tibial nerve and flexor hallucis longus tendon [5]. As the tarsal tunnel is essentially an enclosed structure, both the anatomical volume and overall pressure levels within its borders are particularly decisive in the event of pathology [25]. Accordingly, reductions in the tunnel's overall volume capacity and/or increases in pressure from within the confines will result in subsequent compression of the neuro-vascular bundle [26,27].

The distal end-point of the tarsal tunnel is narrow in width and merges with both the superficial and deep fascia of the abductor hallucis muscle [23]. The posterior tibial nerve, a branch of the sciatic nerve, lies deep to the soleus muscle in the posterior compartment of the lower-leg, and within the tarsal tunnel passes deep to the flexor retinaculum between the calcaneus and medial malleolus [11,23]. It is noted that this nerve lies posterior to the tibial artery and anterior to the flexor hallucis longus muscle, and further branches into the medial plantar nerve, lateral plantar nerve and medial calcaneal nerve, as it tracks infero-posteriorly to the medial malleolus [21,23,28–30]. The studies of Bilge et al. classify the branching of the tibial nerve according to three divisional locations, with specific reference to the central point between the medial malleolus and medial calcaneal tubercle [31]. They acknowledge the divisional parameters may be either proximal, upon or distal to this focal margin [31]. Depending on anatomical variation, the distal trifurcation of the posterior tibial nerve branches usually occurs proximal to, or within, the tarsal tunnel itself [32].

The medial plantar nerve descends deep to the abductor hallucis and flexor hallucis longus musculature, prior to dividing further into three digital nerves [33]. Accordingly, the lateral plantar nerve takes a direct route through the abductor hallucis muscle belly itself, before traversing to the lateral border of the foot and terminating its divisional branches [33]. These peripheral nerves supply autonomic, sensory and motor fibres to the plantar aspect of the foot [11,33]. The medial calcaneal nerve pierces the flexor retinaculum in providing sensory innervation to the posterior and medial margins of the calcaneus [33]. A varied and specific involvement of the differing nerve branches accounts for the diverse presentation and extent level of symptoms observed with the condition of tarsal tunnel syndrome [32]. Fig. 1 illustrates the tarsal tunnel and depicts the tibial nerve with its derivative branches.

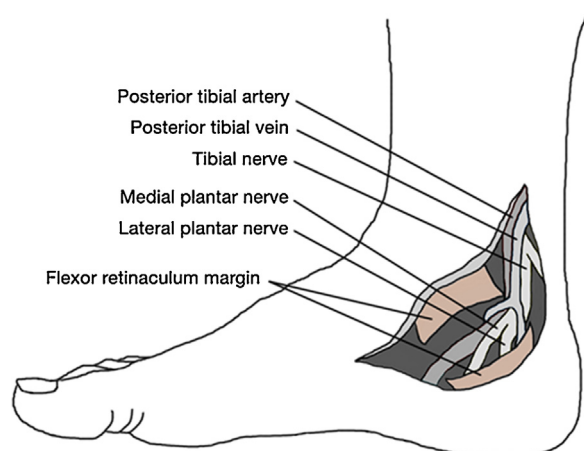


**Fig. 1.** 'The tarsal tunnel'—medial view of the right foot/ankle demonstrating relevant anatomical structures. Posterior tibial artery and vein, the tibial and derivative plantar nerves (medial and lateral), flexor retinaculum margin.

#### 2.4. Signs and symptoms

Characteristic clinical manifestation of tarsal tunnel syndrome includes poorly localized paraesthesia, dysesthesia and hyperaesthesia radiating from the retro-malleolar region to either the sole, heel or digits of the forefoot, or a combination of these areas. It usually worsens with the progression of the day and may involve subsequent cramping in the medial longitudinal arch of the symptomatic foot. Symptoms are typically unilateral, and rarely present bilaterally [1,3,34–37]. It is noted that in some cases pain may also extend proximally to the mid-calf region in response to percussion of the nerve at the site of entrapment, a condition known as Valleix phenomenon [11]. Dependent on the pathologic aetiology, patients may display a distinct localized tenderness, mass or swelling over the medial malleolar region, and are unable to abduct, adduct, flex or extend the hallux [16,38].

It is imperative to adequately rule out systemic or referred causes of neuropathy during clinical investigation (i.e., diabetes, vitamin B12 deficiency, nerve root avulsions) [39]. The digital abductor and flexor muscles of the symptomatic foot can weaken, atrophy or even paralyse in some chronic circumstances [6]. However this is often difficult to detect clinically, and may require subsequent referral for nerve conduction studies [40]. It must be acknowledged that symptoms are most often described by patients as being worst at night [11]. In significantly severe circumstances, the intensity of nocturnal pain in this region may disturb sleep [1].

#### 2.5. Aetiology

The intrinsic and extrinsic aetiologies of tarsal tunnel syndrome predominately stem from post-traumatic, biomechanical, inflammatory and morphological conditions [4,6]. Symptoms may also occur through the formation of mass lesions occupying space within the tarsal tunnel; alternatively it can be classified as an idiopathic and iatrogenic syndrome [6]. Examples of aetiological influences can be broadly grouped into the following categories: space occupying (osteophytes, osteochondromas etc.); soft tissue irregularities (hypertrophic flexor retinaculum, hypertrophic tendinopathies, tumours and anatomical variations); trauma; biomechanical; inflammatory arthropathies (rheumatoid arthritis and seronegative spondyloarthropathies); obesity and lower leg oedema [5,6,11,32,41–49]. Tarsal tunnel syndrome tends to be more common in athletes and individuals whom are subjected

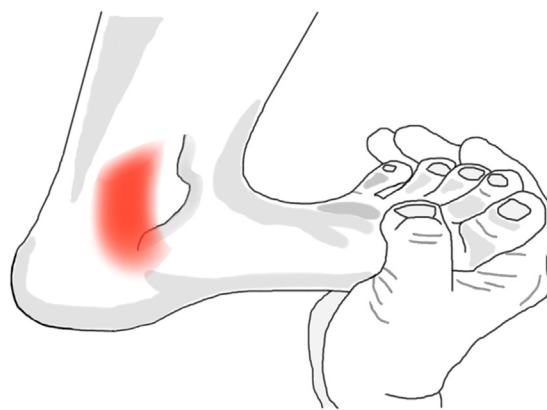


**Fig. 2.** The dorsiflexion-eversion test—the testing clinician passively maximally everts and dorsiflexes the ankle whilst maximally dorsiflexing the metatarsophalangeal joints.

to prolonged weight-bearing periods inclusive of standing, walking or intense physical activity [4,50].

#### 2.6. Diagnostic investigations and testing

The Hoffmann–Tinel sign is the most commonly used clinical test of tarsal tunnel syndrome. The nerve is percussed or tapped at the suspected site of compression [4,40]. A positive diagnosis will cause paraesthesia either locally or radiating along the course of the nerve [4,11]. Tinel postulates that this described sensation is mediated through young nerve axis cylinders during the process of neural regeneration [51]. In addition to this maneuver, symptoms of tarsal tunnel syndrome may also become diagnostically apparent through the dorsiflexion-eversion test [40]. As seen in Fig. 2, to perform this test the clinician both passively maximally everts and dorsiflexes the ankle whilst maximally dorsiflexing the metatarsophalangeal joints [36]. This position is held for 5–10 s and will display further intensification of the symptoms if positive [4]. On occasions pain may also ascend to the thigh following this means of testing [6].

Kinoshita et al. have reported that the dorsiflexion-eversion test reproduced or aggravated symptoms in 36 out of 44 feet (82%) in their study, with no replication evident in the healthy control group [36]. Cadaveric studies by Daniels et al. and Lau and Daniels depicted a substantial tensional increase of both the tibial and lateral-plantar nerves within the tarsal tunnel, following dorsiflexion, eversion, and a combination of these actions to the ankle and foot [52,53]. Accordingly, extension of the metatarsophalangeal joints elicited further strain upon the medial-plantar and tibial nerves, respectively [54]. It is postulated that greater than 50% of patients with compressive neuropathy of the tarsal tunnel will portray a positive Tinel's sign of the posterior tibial nerve [6]. Another specific test to confirm tarsal tunnel syndrome is the plantar flexion-inversion or Trepman test. This maneuver also increases pressure on the tibial nerve within the tarsal tunnel confines [55]. Through intra-operative observation, Hendrix et al. acknowledged this combined movement not only reduced the overall width of the tarsal tunnel, but also compressed the lateral-plantar nerve [56].

Aboulela et al. investigated the sensitivity and specificity of their proposed 'triple compression stress test' in diagnosing tarsal tunnel syndrome. This test involves combining the Tinel's test with the Trepman test for an interval of approximately 30 s [1]. Their study found that clinical signs and symptoms of tarsal tunnel syndrome were apparent within a matter of seconds for 93.8% of the symptomatic feet [1]. The more recent studies performed by Trepman et al. and Barker et al. further support the concept of heightened intra-compartmental pressure in the tarsal tunnel, in

accordance to the external influences of dorsiflexion, plantarflexion, supination and pronation [55,57].

Electrodiagnostic investigations can assist in the diagnosis of tarsal tunnel syndrome [3]. These tests include nerve conduction studies that assess sensory conduction velocities of the tibial nerve or one of its branches, as well as the amplitude and duration of motor-evoked potentials [11]. There is limited quality evidence-based research that demonstrates high sensitivity and specificity of the electrophysiologic techniques in tarsal tunnel syndrome. Reduced amplitude and increased duration of the motor response are the more sensitive indicators of the presence of pathology [58,59]. Unfortunately these investigations often yield an unacceptable level of false negative results, and should be utilized as an adjunctive assessment to confirm physical examination findings [1]. Saeed discusses evidence of false positive readings in his study of 70 asymptomatic subjects, by which 10% of abductor hallucis muscles and 11% of abductor digiti minimi muscles depicted this unacceptable level [60]. Electroneuromyographic investigation characteristically highlights that prolongation of distal motor latencies of abductor hallucis correlate to medial plantar nerve entrapment [3]. Accordingly, prolongation of distal motor latencies of abductor digiti quinti corresponds to lateral plantar nerve entrapment [3].

Generally, abnormal nerve conduction study testing values for applicable clinical reference include the following:

- (1) Prolonged distal motor latency (DML)
  - Terminal latencies of the abductor hallucis muscle (medial plantar nerve innervation) longer duration than 5.8 milliseconds (ms).
  - Terminal latencies of the abductor digiti minimi muscle (lateral plantar nerve innervation) longer duration than 6.3 ms.
  - Side-to-side difference of the same branch derivative more than 1 ms.
- (2) Conduction velocities record of less than 41 ms.
- (3) Recorded amplitude of compound muscle action potential (CMAP) less than 4 millivolts (mV) for abductor hallucis muscle or less than 3 mV for the abductor digiti quinti muscle.
- (4) Side-to-side variation of recorded amplitude decrement more than 50% of the same branch derivative [61].

Magnetic resonance imaging (MRI) is the gold standard in identifying suspected compression of the tarsal tunnel caused by the presence of obstructive foreign masses, lesions or tumours [4,11,62]. Such imaging not only confirms the presence of a suspected lesion, but also defines the depth, extent and margins of the lesion for accurate characterization [63]. In a study undertaken by Frey, MRI was deemed to have shown significant findings in 88% of symptomatic tarsal tunnel candidates, thus assisting with etiological reasoning and surgical planning if required [64]. MRI and high-resolution ultrasound have the diagnostic capability to detect and demonstrate the thickness of the flexor retinaculum, overall depth and contents within the tarsal tunnel, including the posterior tibial nerve cross-sectional area and its terminal branch derivatives [30,65].

In a study performed by Numkarunarunrote et al., both the flexor retinaculum and three root components (medial, intermediate, and lateral) of the stem ligament of the inferior extensor retinaculum were all well seen in the coronal plane following MRI investigation. The respective average thicknesses of these structures were reported to be 0.9 millimeters (mm), 1.5 mm, 1.0 mm, and 0.9 mm [66]. Accordingly, it is acknowledged that diagnostic ultrasound may provide direct evidence of nerve compression by demonstrating focal nerve enlargement and change in nerve echogenicity. By means of ultrasonography, Therimadasamy et al. contrasted the bilateral posterior tibial nerve cross-sectional area

values for a presumed healthy patient presenting with unilateral tarsal tunnel syndrome. Comparative imaging revealed the respective posterior tibial nerves to appear normal above the ankles, however showed a marked enlargement at the tarsal tunnel depicting a cross-sectional area of  $0.17 \text{ cm}^2$  on the pathological side vs.  $0.10 \text{ cm}^2$  on the unaffected side [67]. In deriving comparative perspective from these values, it is interesting to acknowledge that Lee et al. (2005) report a mean cross-sectional area of  $24 \text{ mm}^2$  in patients with diabetic tibial nerve pathology vs.  $12 \text{ mm}^2$  in people without a neuropathy [99].

In a study performed by Bracilovic et al., MRI of thirteen ankles in nine healthy subjects was obtained. Imaging of these ankles was captured with respect to three positional changes (neutral, eversion, inversion) [68]. The volume of the tarsal tunnel was calculated as described by Bracilovic et al. with the tarsal tunnel defined as the area inside a circle created by the distal tip of the anterior colliculus of the medial malleolus, the medial tubercle of the posterior calcaneal tuberosity, and the lateral tubercle of the posterior calcaneal tuberosity on serial MRI sections [68,69]. The tarsal tunnel is assessed from the superior aspect of the calcaneus to a distance 3 cm distal from this margin, and encompasses 10 sequential 3 mm cuts [69]. These sections are then multiplied by the distance between sections, and subsequently added to determine a complete volume measurement of the tarsal tunnel [69]. In this study, the parameter of the tarsal tunnel on MRI was traced with a computer digitizing apparatus in calculating the cross-sectional area of the tarsal tunnel on each image [68]. Following this process, the slice thickness and interspace distance for the seven central images were then utilized to calculate tarsal tunnel volume. Results of the study revealed: the mean tarsal tunnel volume to be significantly greater when the foot and ankle were in the neutral position ( $21.5 \pm 0.9 \text{ cm}^3$ ) in comparison to either full eversion ( $18.0 \pm 0.9 \text{ cm}^3$ ) or inversion ( $20.3 \pm 1.0 \text{ cm}^3$ ) [68].

Plain weight-bearing radiographs and/or computed tomography of the foot and ankle should be acquired if suspecting morphological influences or structural anomalies from bony abnormalities [32]. It is recommended that all tests should ideally be performed bilaterally for adequate observation and comparative study of the contralateral joint [11]. Gait factors such as hyperpronation may compromise the anatomical structures within the tarsal tunnel through physically decreasing the cross-sectional area. Furthermore, the importance of noting valgus and varus foot deformities during assessment is emphasized [70].

Qualitative sensory testing primarily aims to determine pain mechanisms through the assessment of large and small sensory nerve fibre functionality [71]. By this means of diagnostic investigation, qualitative sensory testing devices generate thermal, vibratory, and painful stimuli, amidst varied electrical impulses in monitoring and deriving a measured sensory impairment [72]. A study performed by Tassler and Dellon has revealed higher cutaneous pressure thresholds in 22 patients diagnosed with tarsal tunnel syndrome compared with an age-matched control group [73]. Continuation of basic sensory testing to the symptomatic foot should be repeated periodically throughout the entire course of treatment [40]. This will allow a patient's condition to be properly monitored and is achieved by means of two-point discrimination testing to the plantar surface of the foot [40]. Semmes–Weinstein monofilaments are commonly used for comparative review of conservative and surgical treatment management effectiveness, in clinically evaluating sensory peripheral neuropathy of tarsal tunnel syndrome sufferers [74].

## 2.7. Differential diagnoses

Establishing a comprehensive subjective history is integral in formulating the diagnosis of tarsal tunnel syndrome, as there are

numerous other potential pathologies of the foot/ankle paralleling the symptomology of this condition [75,76]. When collating a thorough subjective history the assessing clinician should aim to derive the following information: mechanism of injury, location of pain, type/description of pain, changes/pattern of symptoms over time, associated footwear, and presence of bruising or swelling [75,76]. Deciphering between musculoskeletal overuse issues, nerve or vascular entrapment is crucial, as the overall treatment management regime for each will differ considerably [75,76]. A precise understanding of the relevant anatomy and physiology of tarsal tunnel syndrome also provides the foundation for increased recognition and identification of this condition [77]. Due to the diffuse and varied nature of presenting symptoms, the potential list of differential diagnoses is quite broad [11]. Other conditions depicting similar clinical symptoms to that of tarsal tunnel syndrome may include: Polyneuropathy, L3–S1 nerve root syndromes, proximal tibial nerve injuries, tumour, osteomyelitis and bone cysts, Morton's metatarsalgia, plantar fasciitis, gout, heel pad atrophy, radiculopathy, ischemia, drug toxicity, nerve entrapment beneath the piriformis, nerve entrapment behind the knee or at the head of the fibula, calcaneal stress fractures, neuroalgodystrophy, compartment syndrome of the deep flexor muscle group, calcaneal spurs and bursitis, posterior tibial tendon dysfunction, flexor hallucis longus and flexor digitorum longus tenosynovitis, arthrosis and inflammatory changes of fascia and ligaments [4,6,39,78–81]. With reference to above, it is highlighted that plantar fasciitis (one of the more common differential diagnoses) results from repetitive use, increased tension, or excessive loading on the plantar fascia and does not typically resemble paresthesia which is otherwise depicted in tarsal tunnel syndrome.

In eliminating differential diagnoses of tarsal tunnel syndrome, it must be acknowledged that proximally located nerve lesions are normally easy to distinguish from peripheral nerve lesions [6]. Collectively, reflex findings and the applicable distribution of motor and sensory deficits may assist this process of differentiation [6]. Intermittent neurogenic disturbance seen in lumbar spinal stenosis frequently presents bilaterally, as opposed to the typical unilateral pain presentation of tarsal tunnel syndrome [6]. Accordingly, paresthesia of the forefoot usually appears bilaterally secondary to polyneuropathic interference [6]. The classic lancinating symptoms of Morton's metatarsalgia arise from mechanical stress and compression of the transverse forefoot arch, making it distinctively different from the residual burning dysesthesia of the retro-malleolar and plantar margins of the foot/ankle typically seen with tarsal tunnel syndrome [6]. Should symptoms be left untreated, permanent nerve damage can occur [4]. Patients may ultimately be left with persistent foot pain, motor deficits, and severe numbness, potentially leading to plantar ulcerations and complex regional pain syndrome [82].

## 2.8. Management

The management for tarsal tunnel syndrome can involve a variety of therapeutic interventions. Conservative or non-operative options have included: strapping the pathological foot in a neutral or inverted position, custom orthoses to support the medial and lateral longitudinal arches of the affected foot, immobilizing braces, activity modification, lesion aspiration, neural mobilization, icing, muscular stretching and strengthening of the gastrocnemius, soleus, tibialis anterior and posterior, peroneal and short toe-flexor muscles, acupuncture, cryotherapy to the affected region, TENS and Anodyne therapy, soft tissue manipulation, remedial massage therapy, local anaesthetic or corticosteroid infiltrations, non-steroidal anti-inflammatory, analgesic, opioid or GABA analog medications, tri-cyclic antidepressants and vitamin B-complex supplements [3–6,14,15,40,83,84]. Upon reviewing the literature to date, no

identifiable randomized controlled trials pertaining to the management of tarsal tunnel syndrome appear existent. Published papers have reported case series of the various treatment modalities, however quantifiable empirical evidence of their efficacy is lacking.

An indication for surgical intervention has been recommended with failure of conservative methods and management in resistant cases, but should always be assessed critically [6,40,85]. In order to maximize the visualization of the nerve, in particular its smaller adjoining branches, the utilization of a pneumatic thigh tourniquet, electrocautery and loupe magnification is rather beneficial [39]. Procedures may include: surgical decompression of the tibial nerve and its branches with division of the medial flexor retinaculum; release of the deep fascia of the abductor hallucis muscle and removal of impinging or pathological lesions [2,6,40,86]. It is understood that post-operative rehabilitation can span a duration totalling up to 24 weeks [87]. The initial post-operative rehabilitation goals are primarily aimed at controlling pain, oedema and inflammation levels, whilst protecting the integrity of the ankle joint and associated neural structures [87,88]. Intermediate rehabilitative measures prevent the occurrence of soft-tissue contractions and formation of scar tissue adhesions, to maintain regular soft tissue flexibility and joint mobility [88]. Optimizing normal, symmetric and efficient gait biomechanics, whilst re-developing strength, proprioception and balance, constitutes the end stage post-operative therapeutic rehabilitation goals [87,88].

There is a vast array of conflicting evidence relating to the efficacy of surgical treatment for tarsal tunnel syndrome, with consistent success rates remaining elusive [4,89]. With this said, many studies have in fact revealed that surgical release may actually improve and resolve the overall symptoms of tarsal tunnel syndrome in up to 85–90% of cases [33,90]. Positive surgical outcomes tended to follow a short history of foot pain, presence of a ganglion, no history of trauma or sprain, and light working duties [91]. Conversely, it is noted that several studies have also been reported in the literature, in which there has been partial or no apparent improvement from surgical intervention [35,92–94]. Five major factors are thought to strongly influence the failure ratio of surgical tarsal tunnel release and include: an incorrect initial diagnosis, incomplete surgical release of the tarsal tunnel, adhesive neuritis following initial decompression measures, intraneural damage/disruption associated with direct neural trauma or systemic disease, presence of a space-occupying lesion or double crush syndrome [95]. Requirements for small scaled randomized controlled trials in groups with homogenous etiology is needed to analyze the effectiveness of specific treatment modalities [6,40].

Takakura's study strongly supports Raikin's findings (2003), demonstrating in his investigations better surgical outcomes on symptomatic patients with aetiologies pertaining to varied definitive space-occupying lesions, compared to a traumatic or idiopathic aetiology [34]. Hence, it is acknowledged that further improvement of the surgical intervention for idiopathic or trauma-related tarsal tunnel syndrome is necessary [2]. Accordingly, patient selection, timing of the surgical procedure and specific decompressive technique also account for the broad variation in reportable successful outcome measurement [5]. In this study, co-author Tamai suggests that poor nerve recovery is more likely to occur when decompression is delayed for longer than a 10-month period, whereas early and accurate diagnosis coupled with immediate treatment intervention provides a more positive prognosis [34].

Collectively within the literature, there appears to be continual clinical dichotomy between objective and subjective surgical outcome parameters, making definitive results highly variable [39]. The most common causation of post-operative symptom recurrence is insufficient proximal nerve release, and may also be associated with scar tissue formation at the surgical site [27]. Many orthopaedic and podiatric surgeons are hesitant to perform

tarsal tunnel operations due to the high incidence of postoperative complications including that of sensory dysfunction [84]. Complex regional pain syndrome may manifest as a result of surgery in this instance, alongside that of impaired wound healing, keloid formation and infection [6]. Revised surgery following failure of the initial attempt is much less predictable, emphasizing the need to explore and evaluate a more proximal or general cause [96].

Cryosurgery for tarsal tunnel syndrome is again less invasive than conventional surgical techniques, and may be a front-line option for treating this sensory peripheral nerve pathology, that has failed conservative therapy [84]. Early results published by Goldstein reveal a respectable and positive efficacy level when performing cryosurgery for the treatment of tarsal tunnel syndrome, with shorter recovery periods recorded than that of traditional surgical methods undertaken for this condition [84]. Undoubtedly, the major advantages of exploring cryosurgery for tarsal tunnel syndrome involve: the surgeon's ability to perform this procedure without the need of a surgical theatre, an absence of the potential chance of scar tissue formation using this technique, and the obvious benefits of complete functional capability for patient's after the surgery is completed [84]. It is acknowledged that due to the limited number of documented cryosurgical trial studies reported, the development of a set surgical guideline for this technique in the treatment of tarsal tunnel syndrome remains subject to ongoing refinement, natural learning and reevaluation [84]. Furthermore, it may be considered somewhat of a contentious treatment option at this point in time, with requirements for a broader range of studies that affirm its empirical effectiveness and validity as an integrated surgical option [84].

The clinical management and proposed treatment for tarsal tunnel syndrome is largely determined by the aetiological factors and pathogenesis in all individual cases [6]. Whilst the empirical effectiveness of several treatment modalities is lacking, the literature consistently highlights a uniformly structured and stepwise protocol for definitively making the actual diagnosis of tarsal tunnel syndrome [97]. Should the classic and applicable findings from a rigorous case-history and physical examination be evident, electroneurodiagnostic studies are sought in sequence with magnetic resonance imaging to eliminate the presence of a space-occupying lesion or other suspected pathological bodies within the tarsal tunnel [39]. Accordingly, it is reiterated that treatment outcomes for tarsal tunnel syndrome may routinely vary due to different reportable outcome parameters/measurements, variance in foot co-morbidities, irregular/non-uniform study grouping and variance of morphological/anatomical neural tracking/branching [98]. A conservative treatment approach to this condition is recommended prior to referral for nerve conduction studies and surgical considerations, unless there is obvious evidence of muscle atrophy or motor involvement [40]. This advocated approach could potentially take course over a 6-month period for patients reporting moderate levels of pain [6].

In some severe instances, surgery may be proposed sooner if the patient's condition is well documented as being severe and is not responding to a conservative treatment repertoire [6]. Again, it is reiterated that the indication for surgery may be regarded in the presence of neurological deficits and cases where the syndrome is due to definite space-occupying lesions [6,34,35]. In these cases, it is believed early detection and intervention has a positive prognostic outlook [34]. In the case of surgical consideration, Takakura's preoperative and postoperative functional rating scale for tarsal tunnel syndrome may be used as a standardized measuring guide for this condition [65]. The scale encompasses and in part objectively grades the presence of reported pain that is spontaneous or apparent upon movement, as well as prevailing evidence of a burning pain sensation, obvious Tinel's sign, sensory disturbance, and apparent muscle atrophy or weakness [65].

### 3. Conclusion

There is obvious reasoning for the need of continued research implementations and studies that investigate the efficacy of conservative and surgical treatment methods for tarsal tunnel syndrome. Ultimately, a structured step-wise approach to managing patients who have been correctly diagnosed with this condition needs to be formulated on the basis of empirical evidence where possible. This literature review has appraised and acknowledged the clinical significance, diagnostic investigations and varied management interventions (non-surgical and surgical) of tarsal tunnel syndrome. It is more than apparent that the prevention, clinical management and treatment of this condition is largely influenced by opinion and clinical experience. Subsequently, it has been hypothesized that the overall clinical management formulae of this condition is neither finite nor holistically defined, and thus requires ongoing clinical research in reevaluating these parameters through small-scaled randomized controlled trials in groups with homogeneous etiologies.

### Competing interests

The authors declare that they have no competing interests.

### Authors contributions

All authors (SCM, MC) contributed to the conception and design of this narrative literature review. SM drafted the manuscript submitted with critical revision and ongoing advice from MC. All authors read and approved the final manuscript.

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