Alexandria Journal of Medicine

ISSN: 2090-5068 (Print) 2090-5076 (Online) Journal homepage: https://www.tandfonline.com/loi/tajm20

Tarsal tunnel syndrome in patients with rheumatoid arthritis, electrophysiological and ultrasound study

Ibrahim Khalil Ibrahim, Sameh Hafez Medani, Mowaffak Moustafa Abd El-Hameed, Mohamed Hassan Imam & Mohamed Magdy Aly Shaaban

To cite this article: Ibrahim Khalil Ibrahim, Sameh Hafez Medani, Mowaffak Moustafa Abd El-Hameed, Mohamed Hassan Imam & Mohamed Magdy Aly Shaaban (2013) Tarsal tunnel syndrome in patients with rheumatoid arthritis, electrophysiological and ultrasound study, Alexandria Journal of Medicine, 49:2, 95-104, DOI: 10.1016/j.ajme.2012.05.006

To link to this article: https://doi.org/10.1016/j.ajme.2012.05.006

Alexandria Journal of Medicine (2013) **49**, 95–104

Alexandria University Faculty of Medicine

Alexandria Journal of Medicine

www.sciencedirect.com

ORIGINAL ARTICLE

Tarsal tunnel syndrome in patients with rheumatoid arthritis, electrophysiological and ultrasound study

Ibrahim Khalil Ibrahim [a], Sameh Hafez Medani [b], Mowaffak Moustafa Abd El-Hameed [a], Mohamed Hassan Imam [a,*], Mohamed Magdy Aly Shaaban [a]

[a] *Physical Medicine, Rheumatology and Rehabilitation, Faculty of Medicine, Alexandria University, Egypt*
[b] *Diagnostic Radiology, Faculty of Medicine, Alexandria University, Egypt*

Received 20 December 2011; accepted 18 May 2012
Available online 10 January 2013

KEYWORDS

Rheumatoid arthritis;
Tarsal tunnel syndrome;
Electrodiagnosis;
Ultrasound

Abstract *Background:* Tarsal tunnel syndrome (TTS) is an entrapment neuropathy of the tibial nerve at the ankle. Rheumatoid arthritis is one of the systemic causes that has been responsible for TTS.
Patients: In this study thirty feet of patients diagnosed as rheumatoid arthritis with complaints of burning pain or paresthesia on the plantar aspect of the foot and toes with 15 feet of age and sex matched control subjects were included.
The aim of this study: To detect TTS among patients with rheumatoid arthritis.
Methods: All patients included in this study were subjected to history taking, clinical examination (general and local), nerve conduction studies and ultrasonography of both tarsal tunnels. In this study, we detected the presence of TTS in rheumatoid arthritis patients group and none was found in the control group.

Abbreviations: TTS, tarsal tunnel syndrome; RA, rheumatoid arthritis

* Corresponding author.
E-mail addresses: Ibrahim_Khalil@yahoo.com (I.K. Ibrahim), Samehmedani@hotmail.com (S.H. Medani), Mowaffak_AbdElHameed@gmail.com (M.M.A. El-Hameed), imam_mohamed@hotmail.com (M.H. Imam), mohamedshaaban@yahoo.com (M.M.A. Shaaban).

Peer review under responsibility of Alexandria University Faculty of Medicine.

ELSEVIER Production and hosting by Elsevier

http://dx.doi.org/10.1016/j.ajme.2012.05.006

Results: A total of 28 cases were confirmed as having TTS. In the patients group a strong statistically significant correlations were found between ultrasonographic and electrodiagnostic findings. *Conclusion:* So it is concluded that TTS is detected in patients suffering from rheumatoid arthritis and that the use of both methods could lead to more reliable confirmed diagnosis which could lead to better management.

1. Introduction

Tarsal tunnel syndrome (TTS) is an entrapment neuropathy at the ankle. It is caused by entrapment of the tibial nerve. It is an uncommon condition predominantly affecting adults, with a slight female predominance.[1] Tarsal Tunnel Syndrome is caused by compression of the tibial nerve or its terminal branches between the proximal origin of the flexor retinaculum and its exit from the tarsal tunnel, where the plantar nerves pierce the abductor hallicus fascia. In many cases the etiology remains idiopathic, a recognizable cause can be identified in up to 80% of cases and the commonest underlying problem is of proliferation or edema of the connective tissues within the tunnel reducing its volume.[2]

Rheumatoid arthritis is a systemic rheumatic disease characterized by a symmetrical, often erosive and deforming polyarthritis.[3,4] Extra-articular manifestations occur in 10–20% of patients, especially those with high titers of rheumatoid factor.[5,6] Symptoms of neuropathy may be overlooked or overestimated in the presence of severe joint disease, restriction, pain and deformities.[7] Careful examination is thus warranted while evaluating such patients. Rheumatoid neuropathy could result from entrapment, nerve ischemia due to vasculitis or drugs used to treat this condition.[4,5,8] Clinical presentations include entrapment neuropathy which is one of the commonest types (carpal tunnel syndrome, tarsal tunnel syndrome, ulnar neuropathy at the elbow or wrist, posterior interosseous nerve syndrome, femoral neuropathy and peroneal neuropathy) mild sensory polyneuropathy; combined sensorimotor polyneuropathy and mononeuritis multiplex. Evaluation of suspected TTS is greatly simplified if one side is symptomatic and the other side is normal. This situation allows for side-to-side comparison studies. The important nerve conduction studies to perform include bilateral tibial (medial and lateral plantar) distal motor latencies to abductor hallucis brevis muscle and abductor digiti quinti pedis muscle, stimulating the tibial nerve proximal to the tarsal tunnel at the medial malleolus. Compound muscle action potential (CMAP) amplitudes and distal latencies are compared from side to side. Theoretically, if there is demyelination across the tarsal tunnel, the distal latencies on the involved side should be markedly prolonged. In axonal loss lesions, the CMAP amplitudes will be reduced, and the latencies will be normal or only slightly prolonged.[9] Consequently, absent or low amplitude potential should not be considered abnormal unless a clear side-to-side difference is found using identical distances between the stimulating and recording sites. No diagnostic significance should be attributed to bilaterally absent plantar mixed or sensory responses, especially in middle aged or older individuals. It is important to emphasize that the plantar mixed and sensory nerves are the most distal nerves in the lower extremities. As such, their conduction velocities normally are slower than those of more proximal nerves and are more susceptible to the effects of temperature and cooling. In addition to bilateral plantar motor, sensory, and mixed nerve studies, further nerve conductions should be performed routinely, especially to exclude a polyneuropathy.[9]

Routine peroneal and tibial motor studies and their respective F responses should be obtained along with the sural sensory response. If the sural sensory response is abnormal, any abnormalities in the plantar nerves are likely secondary to either a polyneuropathy or, less often, a sciatic or lumbosacral plexus lesion. In some situations, assessment of bilateral H reflexes can yield useful information. H reflexes are normal in TTS but may be abnormal in polyneuropathy, proximal tibial neuropathy, sciatic and lumbosacral plexus lesions, and S1 radiculopathy, all of which may cause sensory abnormalities over the sole of the foot.[9] Surface sensory and mixed nerve studies are difficult to perform, even in normal healthy subjects, but they increase the sensitivity of making the electrodiagnosis of TTS. Orthodromic surface sensory studies can be performed stimulating the great and little toes (medial and lateral plantar sensory nerves, respectively) and recording over the tibial nerve at the medial ankle proximal to the tarsal tunnel. The potentials usually are extremely small in amplitude, making it necessary to average much potential. Although it is more painful for the patient, near-nerve recording of the tibial nerve with a needle electrode at the medial ankle with averaging may yield sensory responses that are missed on surface studies. Antidromic surface sensory studies also can be performed, but they have the same technical limitations.[2,9] Surface recording of the mixed plantar nerves is slightly easier. Both the medial and lateral plantar mixed nerves can be stimulated in the sole, recording over the tibial nerve at the medial ankle (proximal to the tarsal tunnel). Averaging is still required to measure these small potentials.[2,9]

Ultrasound (US) is particularly useful in the study of tendon involvement in early rheumatoid arthritis, which often accompanies and in some cases precedes evidence of the disease at joint level. The range of tendon change in rheumatoid arthritis is wide and includes distension of the tendon sheath, loss of 'fibrillar' echotexture, loss of definition of tendon margins and the partial or complete loss of tendon continuity.[10] Tendon sheath widening is the hallmark of early tendon involvement in rheumatoid arthritis and other conditions characterized by synovial inflammation. Analysis of tendon echotexture is one of the fundamental aims of US examination. Circumscribed abnormalities of the homogenous distribution of the intra tendinous connective fibers are the unequivocal expression of anatomical damage mediated by the process of chronic inflammation. In the early phases of inflammation the morphological picture is that of 'tendon erosion' that can precede a more extended 'loss of substance' and evolve into a partial or complete tendon tear. Where tendon erosion is suspected, this diagnosis should always be confirmed by dynamic investigation and comparison with

images taken on longitudinal and transverse scans. This is in order to exclude the possibility of artifacts due to altered inclination of the probe rather than a real anatomical alteration. It may be difficult to differentiate between partial tendon tear and tendon degeneration. The term 'intra substance abnormality' or intra substance tear is often used to describe irregular areas of very low echogenicity within the tendon. More commonly, partial tendon tears appear clearest on transverse views, but the possibility of an artifact should always be kept in mind and the suspicion of a tendon tear on a single field of observation must be verified along contiguous slices with the US beam held perfectly perpendicular to the tendon. Inadequate transducer positioning is the most frequent source of false diagnosis of tendon tear. Complete tendon tear is easily detectable especially if tendons with synovial sheaths are involved (empty sheath sign). The edges of the torn tendon are frequently retracted and curled up. Power Doppler studies make it possible to document hyperemia associated with the phases of active inflammation, also at the level of the tendon. In several rheumatologic disorders, such as rheumatoid arthritis, polyarteritis nodosa, Wegener's granulomatosis, Churg–Strauss and Sjogren syndrome, one of the clinical landmarks of vasculitis is the appearance of neurological findings.[11,12]

From the pathophysiological point-of-view, the vasculitis-related neuropathy affects large nerve trunks, producing a multifocal degeneration of fibers as a result of necrotizing angiopathy of small nerve arteries, the so called ''multiple mononeuropathy''.[13] In these patients, the neuropathy does not correlate with disease parameters (disease activity, rheumatoid factor and functional and radiological scores), and there is sequential involvement of individual nerves both temporally and anatomically.[14] Nerve conduction velocities are usually not markedly reduced from normal, provided that the compound nerve or muscle action potential is not severely reduced in amplitude.[15] Although multiple mononeuropathy is the most common manifestation, nerve entrapment syndromes may also occur at sites where nerves pass in close proximity to either a synovial joint (i.e. cubital tunnel, tarsal tunnel, Guyon tunnel) or one or more synovial-sheathed tendons (i.e. flexor tendons at the carpal tunnel, flexor hallucis longus at the tarsal tunnel) or para-articular bursae (i.e. iliopsoas bursa at the hip). The clinical evaluation of nerves is often made difficult in these patients by symptoms resulting from pain in the joints and limitations of movement, US imaging can contribute in distinguishing entrapment neuropathies related to derangement of joints and tendon abnormalities (joint effusion, synovial pannus, tophi) from non-entrapment neuropathy. This is based on the fact that multiple mononeuropathy does not lead to an altered morphology of the affected nerve, whereas entrapment neuropathies do. At the medial ankle, the tibial nerve and its divisional branches (plantar nerves) travel in the tarsal tunnel between the flexor hallucis longus and the flexor digitorum longus tendons covered by the flexor retinaculum.[16] Because the synovial sheath of the flexor hallucis longus tendon often communicates with the ankle joint, an effusion surrounding this tendon more likely reflects the joint disease rather than a tendon abnormality, especially when considerable ankle joint involvement is present. In these cases, the nerve may be stretched and entrapped by the distended sheath in the retromalleolar region. Marked distension of the medial recesses of the subtalar joint by synovial pannus and effusion may also cause extrinsic compression and disturbances of tibial nerve function. As it had been shown that many causes could lead to foot pain in patients with rheumatoid arthritis so in our study electromyography together with musculoskeletal ultrasonography were used to detect tarsal tunnel syndrome and to evaluate the usefulness of a combination of electrodiagnosis and ultrasound (US) assessments in diagnosing tibial nerve involvement at the tarsal tunnel in rheumatoid arthritis patients. It is hypothesized that in some cases, when the clinical or neurophysiological picture is unclear, the simultaneous study of the tibial nerve at the tarsal tunnel through both US and electrodiagnosis may provide pathologic information not obtainable through electrodiagnosis alone, and this may influence therapeutic decisions.

2. The aim of this work

The aim was to detect tarsal tunnel syndrome among patients with rheumatoid arthritis.

3. Patients

Thirty feet of patients fulfilling at least 4 of the 7 ACR criteria for classification of rheumatoid arthritis (1987)[17] with complaints of burning pain or paresthesia on the plantar aspect of the foot and toes, were collected among the patients attending the Physical Medicine, Rheumatology, and Rehabilitation Department, Faculty of Medicine, Alexandria University. Patients were excluded if there were clinical, electrophysiological or radiological signs of: Peripheral neuropathy, diabetes mellitus, S1 radiculopathy, space occupying lesions at the tarsal tunnel, foot trauma and fractures, post traumatic foot deformity, varicose veins and DVT, severe obesity (by body mass index BMI), lower limb edema. Fifteen feet of age and sex matched normal control subjects had been included to constitute the control group, after an informed consent from all subjects.

4. Methods

The following data were obtained from each patient:

4.1. Personal data

Name, age, sex, occupation, marital status, menstrual history, and parity.

4.2. History of the present condition

Onset, duration, course, progression, relieving factors, aggravating factors and medications received.

4.3. Clinical examination

This had included:

Musculoskeletal examination including signs of rheumatoid arthritis.

Neurological examination including symptoms and signs of tibial nerve entrapment at the tarsal tunnel:

(A) Symptoms: Pain and numbness in the sole of the foot, cramping pains, sensation of tightness, worsening of symptoms with prolonged standing and walking.

(B) Signs: Hypoesthesia, Tinel's sign, Valleix sign.[18] Ankle eversion and dorsiflexion (stretching of the tibial nerve) causing reproduction of symptoms,[18,19] and ankle inversion

(decreasing the volume of the tarsal tunnel) causing reproduction of symptoms.[18,19]

The clinical diagnosis of cases in our study depended upon the main subjective symptom (pain and/or paresthesia in the sole).

4.4. Electrophysiological study

Neuropack 2 electromyograph (EMG) apparatus from Nihon Kohden (Japan) was used to perform the electrophysiological studies of this work. During electrophysiological examination, the skin was kept warm by using hot packs whenever needed.

4.4.1. Sensory conduction studies (SCS)

Of the sural, medial plantar and lateral plantar nerves.[20]

4.4.2. Motor conduction studies (MCS)

Of the medial and lateral plantar nerves.[20]

4.4.3. Late responses

F-response of posterior tibial nerve and H-reflex.[20]

4.5. Ultrasonographic study

The Ultrasonographic study was performed by the Nemios XG. A 10 MHz linear array probe was used to detect the following inflammatory findings in each case: Facial plane fluid accumulation, hypervascularity of inflammation and cellulites were detected through Doppler. Tendon girth swelling altered echogenicity of flexor hallucis longus tendon, flexor digitorum longus tendon, and the tibialis posterior tendon. Signs of plantar fasciitis (edema and increased thickness of plantar fascia).

5. Results

Thirty rheumatoid arthritis patients having pain and/or burning sensation in their feet with a mean age of 39.8 years (ranging from 28 to 57) were included in the study. The control group consisted of 15 feet of healthy individuals with a mean age of 44 years (ranging from 27 to 58). There was no statistically significant difference between age of patients and control groups ($p = 0.166$).(Table 1).

Electrophysiologically, Table 2 shows the medial plantar motor distal latency in the patients group that showed a mean latency of 4.4 ms (ranging from 3.4 to 5.8), while in the control group the mean was 3.62 ms (ranging from 3.0 to 4.4). There was significant difference between patients and control groups ($p = 0.000^*$). In addition, the lateral plantar motor distal latency in the patients group showed a mean latency of 5.057 ms (ranging from 3.9 to 7.4), while in the control group the mean was 3.967 ms (ranging from 3.3 to 4.8). There was a significant difference between the patients and the control groups ($p = 0.000^*$). The medial plantar sensory distal latency in the patients group showed a mean latency of 3.8 ms (ranging from 2.0 to 9.6), while in the control group the mean was 2.6 ms (ranging from 2.0 to 3.2). There was a significant difference between the patients and the control groups ($p = 0.005^*$). The medial plantar sensory conduction velocity (SCV) in the patients group showed a mean SCV of 42.73 m/s (ranging from 21.9 to 66.0), while in the control group the mean was 54.53 m/s (ranging from 43.8 to 77.8). There was a significant difference between the patients and the control groups ($p = 0.001^*$). The lateral plantar sensory distal latency in the patients group showed a mean latency of 4.57 ms (ranging from 2.1 to 9.8), while in the control group the mean was 2.86 ms (ranging from 2.3 to 3.4). There was a significant difference between the patients and the control groups ($p = 0.000^*$). The lateral plantar sensory conduction velocity (SCV) in the patients group showed a mean SCV of 39.8 m/s (ranging from 19.0 to 66.0), while in the control group the mean was 51.45 m/s (ranging from 42.1 to 67.1). There was a significant difference between the patients and the control groups ($p = 0.000^*$).

Table 3 shows that there was no statistically significant difference between the patients and control groups regarding the H-reflex, F-wave latencies and sural nerve SCVs. Table 4 shows the cutoff points of abnormality of the measured electrophysiologic variables. The cutoff point was calculated as the mean ± 2SD. The most frequently abnormal measurement was the sensory latency of the lateral plantar nerve (abnormal in 76.6% of the cases) while the least frequently abnormal was the conduction velocity of the medial plantar nerve (abnormal in only 20% of the cases). Motor abnormalities were encountered in medial plantar nerve in 8 (26.6%) cases and in lateral plantar nerve in 18 (60%) cases. All cases with motor abnormalities had sensory abnormalities, while not all cases with sensory abnormalities had motor abnormalities.

As shown in Table 5, none of the studied cases showed isolated medial plantar nerve, while isolated lateral plantar nerve abnormality was detected in only 5 (16.6%) cases. Affection of both medial and lateral plantar nerves together was detected in 23 (76.6%) of the cases. According to suggested electrophysiologic criteria, the electrophysiologic study could define 28 (93.3%) cases as having TTS.

Table 6 demonstrates the association study between the different electrophysiologic variables and the cases clinically diagnosed as TTS. There was statistically significant association between the abnormalities of the measured electrophysiologic variables and the clinical diagnosis of TTS with the exception of the medial plantar sensory conduction velocity abnormality that was not significantly associated with the clinical diagnosis.

The study of association of the cases diagnosed electrophysiologically as having TTS with the clinical diagnosis (Table 7) showed highly significant association between both where $\chi^2 = 37.059$, and $p = 0.000^*$ ($p < 0.05$).

Regarding the facial plane fluid accumulation (Fig. 1) in the patients group; 20 (66.7%) cases were found positive compared to the control group where only 5 (33.3%) were found positive. There was a significant association between the

Table 1 Frequency of clinical signs and symptoms among cases diagnosed as TTS using different clinical examination tests.

Clinical test	Feet affected $n = 30$
Pain and/or paresthesia	30 (100%)
Tinel's sign	12 (40%)
Valleix sign	17 (56.6%)
Ankle inversion	9 (30%)
Ankle eversion and dorsiflexion	8 (26.6%)

Table 2 Comparison of the different electrophysiological parameters (latencies and nerve conduction studies) between the patients and control groups.

Variable	Patients $n = 30$	Controls $n = 15$	Statistical significance
Medial plantar latency (motor) (ms)			
Min.–max.	3.4–5.8	3.0–4.4	$t = -4.193$
Mean ± SD	4.4 ± 0.5954	3.627 ± 0.5574	$p = 0.000^*$
Lateral plantar latency (motor) (ms)			
Min.–max.	3.9–7.4	3.3–4.8	$t = -4.939$
Mean ± SD	5.057 ± 0.8054	3.967 ± 0.3904	$p = 0.000^*$
Medial plantar latency (sensory) (ms)			
Min.–max.	2.0–9.6	2.0–3.2	$t = -2.946$
Mean ± SD	3.817 ± 1.5654	2.600 ± 0.4018	$p = 0.005^*$
Medial plantar conduction velocity (sensory) (m/s)			
Min.–max.	21.9–66.0	43.8–77.8	$t = 3.570$
Mean ± SD	42.730 ± 10.6993	54.533 ± 9.9294	$p = 0.001^*$
Lateral plantar latency (sensory) (ms)			
Min.–max.	2.1–9.8	2.3–3.4	$t = -4.041$
Mean ± SD	4.570 ± 1.6174	2.860 ± 0.2849	$p = 0.000^*$
Lateral plantar conduction velocity (sensory) (m/s)			
Min.–max.	19.0–66.0	42.1–67.1	$t = 4.033$
Mean ± SD	39.800 ± 9.78193	51.4533 ± 7.63197	$p = 0.000^*$

* $p \leqslant 0.05$ (significant).

Table 3 Comparison of the H-reflex and F-wave latencies and sural nerve SCVs between the patients and control groups.

Variable	Patients $n = 30$	Controls $n = 15$	Statistical significance
H-reflex (ms)			
Min.–max.	27.0–31.0	27.4–30.9	$t = 0.001$
Mean ± SD	28.96 ± 1.18862	29.0 ± 1.14808	$p = 0.973$
F-wave (ms)			
Min.–max.	45.7–51.0	45.3–50.4	$t = 0.010$
Mean ± SD	48.22 ± 1.48612	48.03 ± 1.49220	$p = 0.920$
Sural nerve SCV (m/s)			
Min.–max.	41.2–51.1	41.1–52.4	$t = 0.714$
Mean ± SD	45.41 ± 2.99805	45.59 ± 3.59036	$p = 0.403$

$^*p \leqslant 0.05$ (significant).

Table 4 Frequency of abnormality of each of the studied electrophysiological variables using calculated cut off points.

Variable	Cut off point	Cases $n = 30$ (%)
Medial plantar latency (motor)	4.7 ms	8 (26.6%)
Lateral plantar latency (motor)	4.7 ms	18 (60%)
Medial plantar latency (sensory)	3.4 ms	18 (60%)
Medial plantar conduction velocity (sensory)	34.7 m/s	6 (20%)
Lateral plantar latency (sensory)	3.4 ms	23 (76.6%)
Lateral plantar conduction velocity (sensory)	36.2 m/s	8 (26.6%)

ms: millisecond, m/s: meter/s.

Table 5 Frequency of medial and lateral plantar nerves affection in TTS.

Nerve affected	Frequency
Isolated medial plantar nerve affection (any variable)	0
Isolated lateral plantar nerve affection (any variable)	5 (16.6%)
Both medial and lateral plantar nerves affection	23 (76.6%)
Total No. of cases diagnosed as TTS by EDX	28 (93.3%)

presence of facial plane fluid accumulation and the clinical diagnosis of TTS ($p = 0.034^*$).

Among the studied patients, cellulites and hypervascularity (inflammation) by Doppler study (Fig. 2) were found in 19 (63.3%) cases compared to the control group where they could be detected in none. There was a significant association between the detection of cellulites and hypervascularity (inflammation) by Doppler study ($p = 0.000^*$).

The study of tendon girth swelling (Fig. 3) in the patients group showed that 13 (43.3%) cases had increased girth compared to the control group where none could be detected. There was a significant association between the tendon girth swelling and diagnosis of TTS ($p = 0.002^*$).

Altered echogenicity of tendons (Fig. 4) in the patients group was identified in 24 (80%) cases compared to the control group where none could be detected. There was a significant association between identification of altered echogenicity of tendons and diagnosis of TTS ($p = 0.000^*$).

Twenty-two (73.3%) cases were found positive for plantar fasciitis (Fig. 5) compared to the control group where 8 (53.3%) were found positive. There was no significant association between plantar fasciitis and the diagnosis of TTS ($p = 0.180$).

Fig. 6 shows that 10 cases (33.3%) were found positive regarding all the ultrasonographic studies compared to the control group where none could be detected. There was a significant association between these findings together and the clinical diagnosis of TTS ($p = 0.011^*$).

Table 6 Association between electrodiagnostic parameters and the clinical diagnosis of TTS.

Electrodiagnostic variable (cut off value)	Cases detected n (%)	χ^2	Statistical significance
Motor medial plantar distal latency (>4.7 ms)	8 (26.6%)	$\chi^2 = 4.865$	$p = 0.027^*$
Motor lateral plantar distal latency (>4.7 ms)	18 (60%)	$\chi^2 = 15.000$	$p = 0.000^*$
Sensory medial plantar latency (>3.4 ms)	18 (60%)	$\chi^2 = 15.000$	$p = 0.000^*$
Sensory medial plantar conduction velocity (⩽34.7 m/s)	6 (20%)	$\chi^2 = 3.462$	$p = 0.063$
Sensory lateral plantar latency (>3.4 ms)	23 (76.6%)	$\chi^2 = 23.523$	$p = 0.000^*$
Sensory lateral plantar conduction velocity (⩽36.2 m/s)	8 (26.6%)	$\chi^2 = 4.865$	$p = 0.027^*$

* $p \leqslant 0.05$ (significant).

Table 7 Association of the electrophysiologic diagnosis with clinical diagnosis of TTS (Chi-square).

EDX	Clinical diagnosis	Statistical significance
28 (93.3%)	30 (100%)	$\chi^2 = 37.059$ $p = 0.000^*$

* $p \leqslant 0.05$ significant.

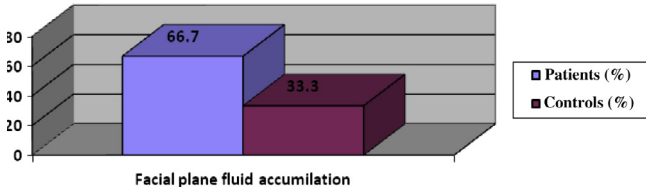

Figure 1 Frequency of facial plane fluid accumulation in the patients and control groups.

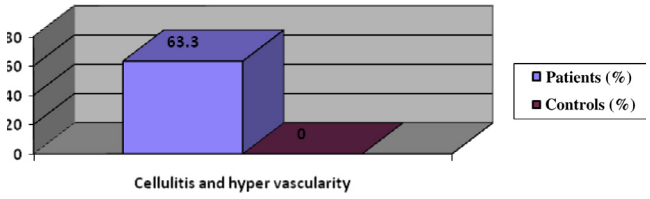

Figure 2 Frequency of cellulites and inflammation in the patients and control groups.

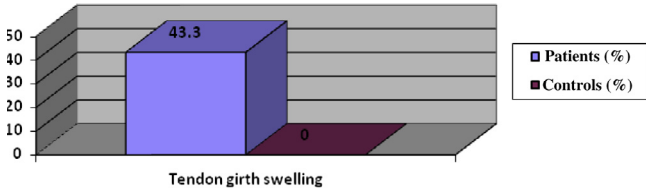

Figure 3 Frequency of tendon girth swelling in the patients and control groups.

The association study between the different ultrasonographic variables and the cases clinically diagnosed as TTS showed that there is statistically significant association between all the studied ultrasonographic variables and the clinical diagnosis of TTS where $\chi^2 = 6.429$ and $p = 0.011^*$ (Table 8).

The study of association between the ultrasonographic diagnosis and electrodiagnosis of TTS (Table 9) showed significant statistical results, where $\chi^2 = 7.806$ and $p = 0.005^*$.

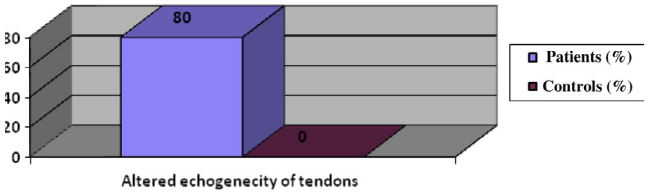

Figure 4 Frequency of altered echogenicity of tendons in the patients and control groups.

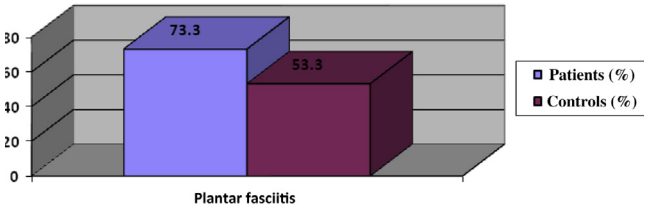

Figure 5 Frequency of plantar faciitis in the patients and control groups.

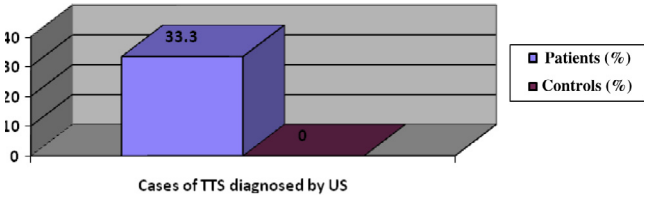

Figure 6 Frequency of cases with inflammatory findings suggestive of TTS in the patients and control groups.

6. Discussion

The aim of this study was to detect tarsal tunnel syndrome among patients with rheumatoid arthritis. Thirty feet of rheumatoid arthritis patients who had pain and burning sensation in their feet and hence suspected to have TTS were examined electrophysiologically and by ultrasound. All patients (100%) fulfilled the inclusion criteria of having pain and/or paresthesia in the sole of foot. Similar inclusion criteria were adopted by

Table 8 Frequency and association of the different US abnormalities with clinical diagnosis.

Variable	Cases $n = 30$ n (%)	Controls $n = 15$ n (%)	Statistical significance
Facial plane fluid accumulation	20 (66.7%)	5 (33.3%)	$\chi^2 = 4.500$ $p = 0.034^*$
Doppler (Inflammation)	19 (63.3%)	0 (0%)	$\chi^2 = 16.442$ $p = 0.000^*$
Tendon girth (swelling)	13 (43.3%)	0 (0%)	$\chi^2 = 9.141$ $p = 0.002^*$
Altered echogenicity of tendons	24 (80%)	0 (0%)	$\chi^2 = 25.714$ $p = 0.000^*$
Plantar fasciitis	22 (73.3%)	8 (53.3%)	$\chi^2 = 1.800$ $p = 0.180$
Cases diagnosed as TTS	10 (33.3%)	0 (0%)	$\chi^2 = 6.429$ $p = 0.011^*$

* $p \leqslant 0.05$ (significant).

Table 9 Association between ultrasonographic diagnosis and electrodiagnosis of TTS.

Ultrasonographic diagnosis	EDX	Statistical significance
10 (33.3%)	28 (93.3%)	$\chi^2 = 7.806$ $p = 0.005^*$

* $p \leqslant 0.05$ significant.

Mondelli et al.[21] who suggested inclusion of cases based on clinical history and symptoms suggesting TTS. Any kind of paresthesia and/or pain in all or part of the foot supplied by the plantar nerves was included. However, Lanzillo et al.[11] considered that pain and paresthesia in rheumatoid arthritis patients could be due to other causes such as referred pain of arthritis or tendinitis or even plantar fasciitis and be misinterpreted by patients who complains as being related to a neurological problem. This can lead clinicians to over- or underestimate the incidence of clinical sensory symptoms.

In our study, eliciting Tinel's sign was considered as an objective clinical method for possible tibial nerve entrapment at the tarsal tunnel. Positive Tinel's sign was observed in 12 (40%) of the cases. In Oh's and Mann's series it was present in 90% of cases (Oh et al.)[22] (Mann et al.)[23] However, Tinel's sign is not pathognomonic of nerve entrapment syndromes, and can also be elicited in the normal population and in patients with polyneuropathy.[24,25] Objective hypoesthesia of the sole may be of more diagnostic help, even if some patients give contradictory responses to sensory examination. Another objective sign in our study was the Valleix sign being positive in 17 (56.6%) cases. It is based on the possible presence of a damaged local area of the nerve at the tarsal tunnel resulting from nerve compression. Consequently, percussion of that damaged area could lead to the reproduction of paresthesia and pain proximally as well as distally along the course of the nerve from that damaged area.[18] With the use of ankle inversion test thus reducing the volume of the tarsal tunnel, reproduction of symptoms was found in 9 (30%) cases, and with ankle eversion and dorsiflexion, increasing the pressure in the tunnel and stretching the posterior tibial nerve, symptoms were reproduced in only 8 (26.6%) cases. Both the above 2 clinical findings depended on the fact that, unlike the carpal tunnel, the tarsal tunnel is a fully enclosed space with critical volume and pressure. Any decrease in the volume or increase in pressure by space occupying lesions, edema, or swollen tendons could compromise the neural bundle passage through the tunnel leading to the entrapment.[18,19] None of the cases included in this study suffered from weakness or wasting of the small muscles of the foot. These are findings consistent with severe TTS. Mondelli et al.[21] found 23% of the studied cases having weakness in toe flexion. On the other hand, mild weakness of the small intrinsic muscles of the foot can seldom be appreciated by clinical observation and it is difficult to obtain reliable information from examination of sensory function in some elderly patients.

6.1. Electrophysiological testing

In our study, electrophysiologic exclusion of associated peripheral polyneuropathy and S1 radiculopathy was among targets. In a review by Patel et al.,[27] the authors identified 317 articles on TTS and presented recommendations for the electrophysiological study of suspected cases of TTS. The recommendations assumed that history; physical examination, NCSs and needle EMG examination should exclude the possibility of polyneuropathy, radiculopathy, and other conditions that might be responsible for the patient's symptoms. Specific electrodiagnostic tests for TTS were performed measuring the motor distal latency of both medial and lateral plantar nerves as well as their sensory latency and conduction velocity. A statistically significant difference between patients and control groups was found throughout all motor and sensory studies of both medial and lateral plantar nerves. Kaplan and Kernahan[26] used motor NCSs in the assessment of TTS. No differences in nerve conduction parameters were noted between the control group and the uninvolved side of the patients with TTS. The involved side showed prolonged distal latencies both to the abductor hallucis as well as the abductor digiti quinti pedis. However, they stated that CMAP amplitude and duration were more likely to be affected than the distal latency. In another study by Oh et al.[22] sensory NCSs were used. Compared with the medial plantar nerve, the lateral plantar sensory nerve conduction velocity was slower and the response amplitude smaller. They also noted that prolonged distal motor latency was less sensitive than abnormal sensory nerve conduction velocity or amplitude. Sensory nerve conduction was abnormal in all nerves in which the distal motor latency was prolonged. In the current study we did not use needle EMG as part of the

electrophysiological study for the diagnosis of TTS. This agrees with the review of Patel et al.,[27] where none of the 317 articles reviewed mentioned the use of needle EMG in diagnosis of TTS. Another reason for not using needle EMG is a difficulty of interpretation. Intrinsic foot muscles commonly show increased insertional activity and occasionally fibrillation potentials associated with large, long duration MUAPs, as one would expect in a neurogenic lesion. Such findings are not unusual in normal subjects without symptoms, however, and have been thought to be due to everyday wear and tear on the foot.[9] The recommendations stated[27] for confirming the presence of tibial mononeuropathy at the level of the tarsal tunnel in the ankle/foot in patients with clinically suspected TTS include, first, tibial motor NCSs, with responses recorded over the abductor hallucis and abductor digiti quinti pedis muscles, demonstrating prolonged distal onset latency (Level C, Class III), second, medial and lateral plantar mixed NCSs, demonstrating prolonged peak latency or slowed conduction velocity across the tarsal tunnel (Level C, Class III), third, medial and lateral plantar sensory NCSs, demonstrating slowed conduction velocities across the tarsal tunnel and/or small amplitude or absent responses (Level C, Class III), and finally, unclear utility of needle EMG in the assessment of TTS (Level U, data insufficient). In this study, calculated cut off points of the electrophysiologic parameters were used. In a study by Mondelli et al.[21] the electrophysiological values of each subject were considered abnormal if they were 2SD below or above the mean of age-matched controls. It was proposed that affection of any parameter of the electrophysiological study reflects pathologic affection of the related nerve. So, medial plantar motor affection was found in 26.6% of cases whereas detection of abnormalities in sensory latency was found in 60% of cases and sensory conduction velocity in 20%. It was noted that medial plantar nerve affection was found in 76.6% of the cases. However, the lateral plantar nerve showed a higher incidence of abnormalities, where motor affection was detected in 60% of cases, whereas sensory latency affection in 76.6% while its sensory conduction velocity in 26.6%. It was noted that all cases with motor abnormality always had sensory abnormality. Also all cases with medial plantar affection had lateral plantar affection. It was concluded that lateral plantar nerve affection is relatively more common than medial plantar affection. In our study affection of both medial and lateral plantar nerves together was observed in the majority of cases (76.6%), while isolated affection of the lateral plantar nerve alone was encountered in only 16.6% of the studied cases with a total of 28 (93.3%) cases of TTS. In a case series report,[28] it was noted that only 3 of 13 patients had abnormal motor studies and those patients who had motor study abnormalities always had abnormalities of mixed and sensory nerves. Lateral plantar nerve sensory conduction abnormalities were observed in all limbs. Absence of the sensory nerve action potential was the most frequent abnormality, representing 92.8% of the abnormalities in the lateral plantar nerve and 76.9% in the medial plantar nerve. Sensory nerve action potentials were also absent from two unaffected limbs. Mixed NCSs were abnormal in 85.7% of TTS limbs, but normal in all asymptomatic limbs and control subjects. Mixed nerve conduction study abnormalities were always associated with abnormal sensory nerve conduction studies. Only 21.5% of TTS limbs had significantly prolonged distal motor latencies to the abductor hallucis. Like what has been noted in our study generally, the abnormalities detected in sensory studies were more frequent than those for the motor studies.

In the present study, a significant association was found between the abnormality of medial and lateral plantar nerves motor distal latencies and clinical diagnosis of TTS. Similarly, abnormality of the medial and lateral plantar sensory latencies and the lateral plantar sensory conduction velocity had significant association with the clinical diagnosis of TTS. Only the medial plantar sensory conduction velocity showed non-significant association with the clinical diagnosis of TTS. Also significant association could be detected between the cases diagnosed as TTS and the clinically diagnosed cases, such association reflects the clinico-pathologic relevance of the abnormal NCSs, where the pathology of the nerve at the entrapment site leads to the clinical problems detected in patients and diagnosed by electrophysiological parameters.

6.2. Ultrasonographic testing

Tarsal tunnel syndrome being an entrapment neuropathy of the tibial nerve at the ankle[1] US imaging can contribute in distinguishing entrapment neuropathies related to derangement of joints and tendon abnormalities (joint effusion, synovial pannus, tophi) from non-entrapment neuropathy. In our study we used Musculoskeletal US in order to identify inflammatory-caused factors that affected the medial ankle including facial plane fluid accumulation, detection of active inflammation and cellulites by Doppler study, altered echogenicity of tendons and tendon girth swelling. Clinical studies have shown that musculoskeletal US is more sensitive to the detection of inflammatory signs than a clinical examination.[29,30] In present study, 66.7% of the rheumatoid patients had facial plane fluid accumulation (effusion) showing a significant association with clinical diagnosis of TTS. Tendon girth swelling denotes tenosynovitis. It was observed in 43.3% of the studied cases but in none of the healthy control subjects. Similarly, altered tendon echogenicity was encountered in 80% of the cases. In rheumatoid arthritis the inflammatory soft tissue lesions include synovitis with effusion, synovial proliferation, tenosynovitis with effusion and/or synovial proliferation of the tendon sheath, tendinitis, as well as bursitis with effusion and/or synovial proliferation. Synovial proliferation often occurs in combination with inflammatory effusion and the combination is termed synovitis.[31] At the medial ankle, the tibial nerve and its divisional branches (plantar nerves) travel in the tarsal tunnel between the flexor hallucis longus and the flexor digitorum longus tendons covered by the flexor retinaculum.[16] Because the synovial sheath of the flexor hallucis longus tendon often communicates with the ankle joint, an effusion surrounding this tendon more likely reflects the joint disease rather than a tendon abnormality, especially when considerable ankle joint involvement is present. In these cases, the nerve may be stretched and entrapped by the distended sheath in the retromalleolar region. Marked distension of the medial recesses of the subtalar joint by synovial pannus and effusion may also cause extrinsic compression and disturbances of tibial nerve function.[32]

In this study the detection of inflammation and cellulites via Doppler study revealed 19 (63.3%) cases in the patients group ($p = 0.000^{*}$) showing significant association with the clinical diagnosis. The vessels of inflammatory tissue are expanded and new vessels are constituted, all of which are detectable

with the Doppler technique. With regard to the vessel density of an inflammatory joint, it is possible to illustrate color pixels in different accumulations. The demonstration of physiological vascularization of healthy joints without signs of inflammation is only possible with very sensitive US devices and high-resolution scanning techniques, and even then only in a very few cases. The performed US studies could distinguish inflammation-relevant changes in the tarsal tunnel of 33.3% of the cases. Through this way the inflammatory conditions affecting the medial ankle structures in rheumatoid arthritis including effusion, synovitis, tendon swellings and tenosynovitis can lead to decreasing the tarsal tunnel volume or increasing the tunnel pressure which could cause tibial nerve compression and stretch. Sonographic findings in patients with tarsal tunnel syndrome depend on the etiology. For instance, with the existence of extra-articular ganglia, displacement and bowing of the tibial nerve can be visualized. When cystic enlargement of the tibial nerve is detected, a connection to the near joint should be suspected.[33] Posttraumatic changes causing external compression are easily confirmed in the same way as in any other anatomic regions, with direct nerve compression and reactive nerve edema representing a common pathway of neuropathy.[34–36] Color Doppler ultrasound can confirm venous engorgement demonstrating venous vessels surrounding the tibial nerve. After long-standing nerve compression the affected nerve shows a wavelike appearance in a longitudinal scan, similar to the median nerve in carpal tunnel syndrome. It is important to assess and compare the cross-sectional area on both nerves especially in idiopathic posterior tarsal tunnel syndrome. Direct trauma to the nerve with partial or complete dissection can easily be assessed with sonography. In our study we had been faced with multiple technical considerations for sonography of the tibial nerve at the tarsal tunnel as we used a 10 MHz probe while superficially located nerves such as the median nerve, ulnar nerve, peroneal and tibial nerves should be examined with transducers of 15–18 MHz.[37] It had been stated that a high resolution 12- to 17-MHz transducer is certainly recommended for this region (Peer et al.),[36] also ankle edema and inflammatory changes at the medial ankle soft tissue structures hinder the visualization of the posterior tibial nerve. In the study at hand we detected plantar fasciitis by US in the patients group where 22 (73.3%) cases were found positive compared to the control group where 8 (53.3%) were affected. Plantar fasciitis was not significantly associated with TTS clinical diagnosis in the present study. Clinically, plantar fasciitis is frequently considered in the differential diagnosis of TTS and vice versa. Plantar fasciitis causes inferior heel pain in up to 10% of active or sedentary adults over a range of ages. It is more likely to occur in obese people, in those who are on their feet most of the day and in people with limited dorsiflexion of the ankle.[38] The typical sufferer is most affected when starting to walk after rising from sleep or some other sedentary positions. The clinical presentation, history, physical examination and plain radiographs are usually sufficient to make a diagnosis of plantar fasciitis.[39] However, the clinical presentation of plantar fasciitis is mimicked by a number of disorders. These include other enthesopathies, traumatic and corticosteroid-induced rupture, rheumatologic and infectious processes, plantar fibromatosis, and tarsal tunnel syndrome.[40,41] Thus in the current study we detected tarsal tunnel syndrome in patients suffering from rheumatoid arthritis as part of the neurological extra articular manifestations of their chronic illness. It should be noted that whereas the interpretation of electrophysiological studies relies on validated normative data, ultrasound findings are less quantifiable. Several measurements (such as cross-sectional area) have been proposed and accepted in the literature, but normative data are so far not available for all nerves (like that for the tibial nerve). Moreover, some aspects (such as echo intensity) are not well quantifiable. The criteria adopted allowed us to proceed cautiously, giving greater importance to quantifiable measurements.[42]

A significant statistical association was found between electrophysiologic diagnosis of TTS and the ultrasonographic inflammatory relevant abnormalities. This enhances the mechanistical relationship between nerve pathology and associated neuropathy encountered in TTS of the rheumatoid patients. Thus, the present study shows that the combination of electrophysiology and ultrasonography performed in the same session (or in collaboration with an ultrasound examiner) may be useful for diagnosis of TTS in rheumatoid patients that will help in the determination of appropriate therapy. Accordingly, it was concluded that the clinical examination is the main step in identification of Tarsal Tunnel syndrome cases and the use of multiple electrophysiologic parameters in diagnosis of Tarsal Tunnel syndrome is highly appreciated rather than the use of a single electrodiagnostic test. The sensory affection of the tibial nerve branches is more common than motor affection in Tarsal Tunnel syndrome and it was realized that the lateral plantar nerve affection is more common than medial plantar nerve affection. The musculoskeletal ultrasound can detect pathologies that may predispose or lead to entrapment neuropathy and the combined use of electrophysiology with musculoskeletal ultrasonography further confirms the diagnosis of Tarsal Tunnel syndrome.

References

1. Keck C. The tarsal tunnel syndrome. *J Bone Joint Surgery Am* 1962;**44**:180–2.
2. Cimino WR. Tarsal tunnel syndrome: review of the literature. *Foot Ankle* 1990;**11**:47–52.
3. Moore PM, Richardson B. Neurology of the vasculitides and connective tissue diseases. *J Neurol Neurosurg Psychiatry* 1998;**65**:10–22.
4. Bruyn GAW, Markusse HM. Nervous system involvement in rheumatoid arthritis. In: Aminof MJ, Goetz CG, editors. *Handbook of clinical neurology. Systemic diseases, part III*, Vol. 27. Elsevier Science; 1998. p. 15–32.
5. Shannon KM, Goetz GC. Connective tissue diseases and the nervous system. In: Aminoff MJ, editor. *Neurology and general medicine. The neurological aspects of medical disorders*. Churchill: Livingstone; 1995. p. 447–71.
6. Sivri A, Guler-Uysal F. The electroneurophysiological findings in rheumatoid arthritis patients. *Electromyogr Clin Neurophysiol* 1999;**39**:387–91.
7. Dani K, Ramachandran R, Capell HA, et al. Neuropathies in the rheumatoid patient: a case of the heavy hand. *Scott Med J* 2005;**50**:125–6.
8. Gorson GC. Vasculitic neuropathies: an update. *Neurologist* 2007;**13**:12–9.
9. Preston DC, Shapiro BE. *Electromyography and neuromuscular disorders. Clinical-electrophysiologic correlations*. 2nd ed. Philadelphia, Pennsylvania: Elsevier; 2007, p 367–70.
10. Grassi W, Filippucci E, Farina A, et al. Sonographic imaging of tendons. *Arthritis Rheum* 2000;**43**:969–76.

11. Lanzillo B, Pappone N, Crisci C, et al. Subclinical peripheral nerve involvement in patient with rheumatoid arthritis. *Arthritis Rheum* 1999;**42**:1304–5.
12. Rosenbaum R. Neuromuscular complications of connective tissue disease. *Muscle Nerve* 2001;**24**:154–69.
13. Said G, Lacroix C. Primary and secondary vasculitis neuropathy. *J Neurol* 2005;**252**:633–41.
14. Nadkar MY, Agarwal R, Samant RS, et al. Neuropathy in rheumatoid arthritis. *J Assoc Physicians India* 2001;**49**:217–20.
15. Sivri A, Guler-Uysal F. The electroneurophysiological evaluation of rheumatoid arthritis patients. *Clin Rheumatol* 1998;**17**:416–8.
16. Mahan KT, Rock JJ, Hillstrom HJ. Tarsal tunnel syndrome. A retrospective study. *J Am Podiatr Med Assoc* 1996;**86**:81–91.
17. Arnett FC, Edworthy SM, Bloch DA, et al. The American Rheumatism Association 1987 revised criteria for the classification of rheumatoid arthritis. *Arthritis Rheum* 1988;**31**:315–24.
18. Wallach DM, Katchis SD. Tarsal tunnel syndrome. In: Ranawat RG, Positano RG, editors. *Disorders of the heel, rear foot, and ankle*. Churchill: Livingston; 1999. p. 125–34.
19. Lam SJ. Tarsal tunnel syndrome. *J Bone Joint Surg Br* 1967;**49**:87.
20. Weber R. Motor and sensory conduction and entrapment syndrome. In: Johnson EW, editor. *Practical electromyography*. 2nd ed. Baltimore, Hong Kong, London: Sydney. Williams & Wilkins; 1988. p. 92–186.
21. Mondelli M, Morana P, Padua L. An electrophysiological severity scale in tarsal tunnel syndrome. *Acta Neurol Scand* 2004;**109**:284–9.
22. Oh SJ, Sarala PK, Kuba T, Elmore RS. Tarsal tunnel syndrome; electrophysiological study. *Ann Neurol* 1979;**5**:327–30.
23. Mann RA. Tarsal tunnel syndrome. *Othop Clin North Am* 1974;**5**:109–15.
24. de Krom MC, Knipschild PG, Kester AD, Spaans F. Efficacy of provocative tests for diagnosis of carpal tunnel syndrome. *Lancet* 1990;**335**:393–5.
25. Jabre J, Dillard JW, Salzsieder BT, et al. The use of multiple Tinel's sign in the identification of patients with peripheral neuropathy. *Electromyogr Clin Neurophysiol* 1995;**35**:131–6.
26. Kaplan PE, Kernahan Jr WT. Tarsal tunnel syndrome: an electrodiagnostic and surgical correlation. *J Bone Joint Surg* 1981;**63**:96–9.
27. Patel T, Gaines K, Malamut R, et al. Usefulness of electrodiagnostic techniques in the evaluation of suspected tarsal tunnel syndrome: an evidence based review. *Muscle Nerve* 2005;**32**:236–40.
28. Galardi G, Amadio S, Maderna L, et al. Electrophysiologic studies in tarsal tunnel syndrome. Diagnostic reliability of motor distal latency, mixed nerve and sensory nerve conduction studies. *Am J Phys Med Rehabil* 1994;**73**:193–8.
29. Backhaus M, Kamradt T, Sandrock D, et al. Arthritis of the finger joints: a comprehensive approach comparing conventional radiography, scintigraphy, ultrasound, and contrast-enhanced magnetic resonance imaging. *Arthritis Rheum* 1999;**42**:1232–45.
30. Szkudlarek M, Narvestad M, Klarlund A, et al. Ultrasonography of the metatarsophalangeal joints in rheumatoid arthritis: comparison with magnetic resonance imaging, conventional radiography, and clinical examination. *Arthritis Rheum* 2004;**50**:2103–12.
31. Wakefield RJ, Balint P, Szkudlarek M, et al. OMERACT 7 special interest group: musculoskeletal ultrasound including definitions for ultrasonographic pathology. *J Rheumatol* 2005;**32**:2485–7.
32. Backhaus M. Ultrasound and structural changes in inflammatory arthritis synovitis and tenosynovitis. MRI and ultrasound in diagnosis and management. *Ann NY Acad Sci* 2009;**1154**:139–51.
33. Spinner RJ, Dellon AL, Rosson G, et al. Tibial intra-neural ganglia in the tarsal tunnel: is there a joint connection. *J Foot Ankle Surg* 2007;**1**:27–31.
34. Martinoli C, Bianchi S, Gandolfo N, et al. US of nerve entrapments in osteofibrous tunnels of the upper and lower limbs. *Radiographics* 2000;**20**:199–217.
35. Nagaoka M, Satou K. Tarsal tunnel syndrome caused by ganglia. *J Bone Joint Surg Br* 1999;**81**:607.
36. Peer S, Kovacs P, Harpf C. High resolution sonography of lower extremity peripheral nerves: anatomic correlation and spectrum of pathology. *J Ultrasound Med* 2002;**21**:315–22.
37. Koenig R, Pedro M, Heinen C, et al. High-resolution ultrasonography in evaluating peripheral nerve entrapment and trauma. *Neuro Surg Focus* 2009;**26**(2):E13.
38. Cole C, Seto C, Gazewood J. Plantar fasciitis: evidence-based review of diagnosis and therapy. *Am Fam Physician* 2005;**72**:2237.
39. Williams SK, Brage M. Heel pain – plantar fasciitis and achilles enthesopathy. *Clin Sports Med* 2004;**23**:123–44.
40. Cardinal E, Chhem RK, Beauregard CG, et al. Plantar fasciitis: sonographic evaluation. *Radiology* 1996;**201**:257–9.
41. Berkoitz JF, Kier R, Rudicel S. Plantar fasciitis: MR imaging. *Radiology* 1991;**179**:665–7.
42. Bilbao A, Wilcox MS. Tarsal tunnel revisited. *Muscle Nerve* 1995;**18**:791.