Arch Rheumatol 2019;34(x):i-viii
doi: 10.5606/ArchRheumatol.2019.7134

ORIGINAL ARTICLE

Assessment of the Frequency of Tarsal Tunnel Syndrome in Rheumatoid Arthritis

Mehtap KALÇIK ÜNAN[1], Nevsun PIHTILI TAŞ[2], Rabia AYDOĞAN BAYKARA[3], Ayhan KAMANLI[4]

[1]*Department of Physical Medicine and Rehabilitation, Cizre State Hospital, Şırnak, Turkey*
[2]*Department of Physical Medicine and Rehabilitation, University of Health Sciences, Elazığ Training and Research Hospital, Elazığ, Turkey*
[3]*Department of Physical Medicine and Rehabilitation, University of Health Sciences, Malatya Training and Research Hospital, Malatya, Turkey*
[4]*Department of Physical Medicine and Rehabilitation, Sakarya University Faculty of Medicine, Sakarya, Turkey*

ABSTRACT

Objectives: This study aims to investigate the frequency of tarsal tunnel syndrome (TTS) in rheumatoid arthritis (RA) patients.

Patients and methods: Thirty RA patients (1 male, 29 females; mean age 41.9 years; range, 26 to 65 years) providing the American College of Rheumatology classification criteria and 20 healthy volunteers (1 male, 19 females; mean age 41.9 years; range, 25 to 65 years) without any complaints were included in the study. The demographic characteristics of the study group were assessed and neurological examinations were performed. The Tinel sign was checked to provoke the TTS symptoms. Disease severity was measured with Visual Analog Scale (VAS), Disease Activity Score 28 (DAS28), erythrocyte sedimentation rate, and C-reactive protein; while the health-related quality of life and disability status were determined by questioning with Health Assessment Questionnaire, Short Form-36, Foot Function Index, and VAS (0-100 mm). In addition, the positional relationship of foot pain was questioned with VAS. The 100-meter (m) walking time of the patient and control groups was calculated.

Results: Bilateral TTS was detected in 10 patients (33.33%) with RA. No relationship was found with the TTS disease duration, seropositivity, rheumatoid nodule, joint deformities, corticosteroid use, and DAS28 score. In correlation with TTS, foot and ankle were the first involved joints at the beginning of RA disease ($p<0.005$). The Tinel sign was 45% positive in patients with TTS. The 100-m walking time of the study group was longer and significantly different compared to the control group ($p<0.0001$).

Conclusion: Tarsal tunnel syndrome is commonly seen in RA. The incidence increases in patients with primary foot involvement. For this reason, caution should be taken against entrapment neuropathies in RA patients whose diagnosis should be supported by electrophysiological studies when the medical history and physical examination are not sufficient.

Keywords: Entrapment neuropathy; rheumatoid arthritis; tarsal tunnel syndrome.

Rheumatoid arthritis (RA) is a chronic, multisystemic, inflammatory, rheumatic disease that affects the synovial joints primarily and develops from unknown causes.[1-4] The disease affects mainly the hand, wrist, and foot joints, while it can also be seen in all other synovial joints such as the knee, ankle, hip, elbow, and shoulder.[1]

In patients with RA, foot involvement is as frequent as hand involvement.[5] Foot involvement was reported as 38% by Bukhari et al.[6] It was shown to be frequent particularly in the early periods of the disease. As the disease progresses, the incidence of foot involvement increases.[7] Metatarsophalangeal joints of the feet are affected mostly in RA.[8] It was reported that the early forefoot involvement is an indicator of aggressive disease, and early erosions in metacarpophalangeal joints were reported in 10% of the patients.[5-7]

Received: August 02, 2018 ***Accepted:*** October 19, 2018 ***Published online:*** January 28, 2019

Correspondence: Nevsun Pıhtılı Taş, MD. SBÜ Elazığ Eğitim ve Araştırma Hastanesi Fizik Tedavi ve Rehabilitasyon Kliniği, 23280 Elazığ, Turkey.
Tel: +90 533 - 663 35 69 e-mail: nevsunpihtili@gmail.com

Citation:
Kalçık Ünan M, Pıhtılı Taş N, Aydoğan Baykara R, Kamanlı A. Assessment of the frequency of tarsal tunnel syndrome in rheumatoid arthritis. Arch Rheumatol 2019;34(x):i-viii.

Moreover, entrapment neuropathies are frequently seen in RA. The involvement is usually in the form of mononeuritis multiplex. Median, ulnar, peroneal, and tibial nerves are affected most commonly.

Tarsal tunnel syndrome (TTS) is the entrapment neuropathy of the posterior tibial nerve and/or its terminal branches (calcaneal, medial plantar [MP], and lateral plantar [LP] branches) which develop in the fibro-osseous tunnel, under the flexor retinaculum and behind the medial malleolus of the ankle.[9] The posterior tibial nerve, tibial artery, tibial vein, and also the tendons of tibialis posterior, flexor digitorum longus and flexor hallucis longus muscles pass through the tarsal tunnel (Figure 1).[10-14]

Tarsal tunnel syndrome is clinically characterized by local tenderness behind the medial malleolus, pain in the feet and heels, paresthesia, and heat followed by numbness and tingling. Pain and paresthesia progressively become more permanent and severe and may spread towards the posterior or medial (Valleix phenomenon) or distal (Tinel sign) of the leg proximally.

Patients' detailed history and physical examination as well as electrophysiological studies are used in the diagnosis of TTS.[15] Foot and ankle radiographs are useful in the evaluation of bone deformities and abnormalities. Computed tomography is also helpful in diagnosis. Magnetic resonance imaging (MRI) and ultrasonography (USG), which are popular today, are used in the evaluation of soft tissues.[16,17]

Treatment modality of TTS should include targeting the etiology.[11] Conservative treatment should be initiated first. In conservative treatment, nonsteroidal anti-inflammatory drugs (NSAIDs) and other agents that reduce and prevent neuropathic pain are used. Also, local steroid injections are useful. Arthrosis that limits the pronation of the ankle help in reducing nerve compression.

Surgical treatment should be indicated if symptoms persist for six months and conservative treatment is insufficient. Negative electrophysiological studies do not exclude the diagnosis of TTS. Electrophysiological studies are generally supportive in the diagnosis of TTS; however, patient's clinical symptoms and signs are the most significant in the diagnosis and treatment.[18] In this study, we aimed to investigate the frequency of TTS in RA patients.

PATIENTS AND METHODS

Thirty patients (1 male, 29 females; mean age 41.9 years; range, 26 to 65 years) diagnosed with RA according to the American College of Rheumatology classification criteria and

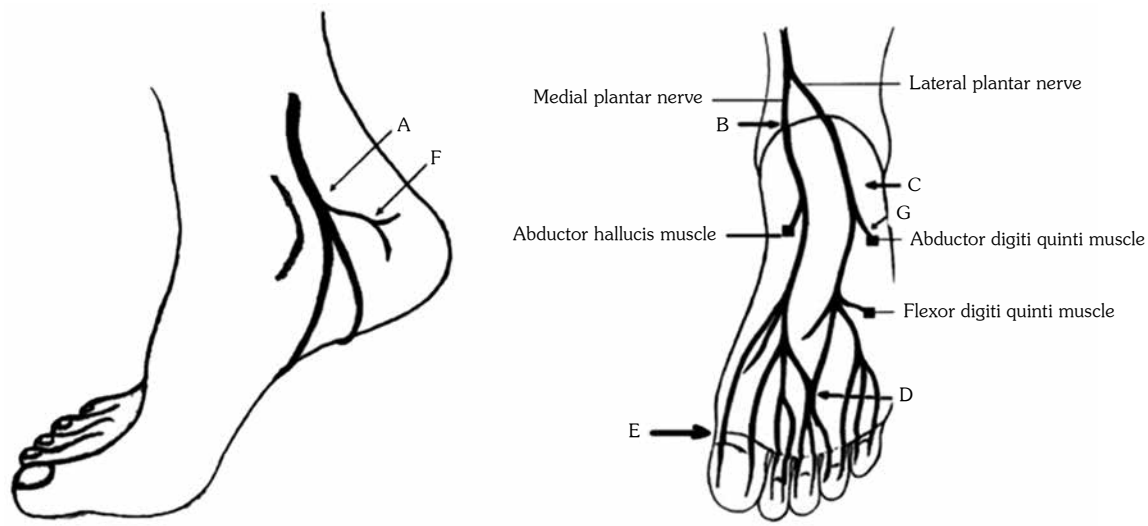

Figure 1. Trapping sites of tibial nerve.[11]
A: Tarsal tunnel (before leaving branches); B: Medial plantar neuropathy; C: Lateral plantar neuropathy; D: Morton's neuroma; E: Joplin's neuroma; F: Medial calcaneal neuropathy; G: Inferior calcaneal neuropathy.

followed-up in the outpatient clinic of Fırat University, Department of Physical Medicine and Rehabilitation, Rheumatology Unit between February 2006 and May 2008 were included. Also, 20 healthy individuals (1 male, 19 females; mean age 41.9 years; range, 25 to 65 years) who did not have any foot complaints were included as the control group. The study protocol was approved by the Fırat University Ethics Committee (approval number: 2005-2006/584). A written informed consent was obtained from each patient. The study was conducted in accordance with the principles of the Declaration of Helsinki.

Patients with a history of other metabolic diseases known to affect the electroneurophysiology such as diabetes mellitus; who were pregnant or had an autoimmune disease such as hypothyroidism; those who underwent hip arthroplasty or surgery for the lumbar region; or those with a history of fracture, trauma, and/or operation for feet and ankles were excluded.

Patients were selected cross-sectionally: Patients with or without foot pain, active or non-active, and those using disease-modifying antirheumatic drugs (DMARDs) and steroids were randomly included.

Demographic information such as age, sex, weight, and height for both groups as well as the time of the disease's onset; duration of morning stiffness; other comorbid diseases; the use, dose and duration of steroids; DMARDs, and NSAIDs for the study group were registered in detail. Hemoglobin, hematocrit, platelet, white blood cell, erythrocyte sedimentation rate, C-reactive protein, and rheumatoid factor levels were measured.

The 100-meter (m) walking time for both the patient and control groups was calculated in seconds. Foot pain, severity, and its relationship with position were assessed using the Visual Analog Scale (VAS). Health Assessment Questionnaire (HAQ), Foot Function Index, Short Form-36 (SF-36) and VAS were performed in both groups by a single clinician who was not informed of the laboratory results of patients.

Neurological examinations were carried out in both groups. During the examination, the Tinel sign was evaluated in detail to provoke the tibial nerve. The Tinel sign is pain and numbness in the medial one-third of the foot base after the percussion of the tibial nerve at the upper border of the tarsal tunnel behind the medial malleolus. The positive percussion mark can be seen in all entrapped neuropathies.[19]

Symptoms are exacerbated by the pronation of anterior foot. Another provocative test is the compaction of the posterior tibial nerve by compressing the ankle to dorsiflexion and eversion position.[15]

The electrophysiologic examination was performed in the electrophysiology laboratory using Medelec Synergy Electroneuromyography device (Medelec, Surrey, UK). Room temperature was kept between 26°C to 32°C. TTS electromyography (EMG) protocol was administered bilaterally to the lower extremities of individuals in both groups by the same researcher:

1. Distal tibial nerve was studied as motor MP nerve (MPN) and LP nerve (LPN).
2. In the routine tibial motor study, the tibial nerve was supramaximally stimulated from the medial ankle and popliteal fossa, as it was recorded from the abductor hallucis brevis muscle.
3. The peroneal nerve was stimulated via the lateral ankle, under the fibula head and popliteal fossa in the routine peroneal motor study. The record was taken from the extensor digitorum brevis muscle.
4. Medial plantar and LP mixed or sensory studies were performed orthodromic.

The nerve was stimulated from the heel's MPN in the MP mixed sensory studies. The nerve was stimulated from the heel's LPN in the LP mixed sensory studies.

5. The recording electrodes were placed in the lateral malleolus, and the record was taken by stimulating the nerve from the posterior lateral thigh in the sural nerve sensory study.
6. Tibial and peroneal F responses were obtained.
7. Hoffmann (H)-reflex study was performed. H-reflex and F response are the most

Table 1. Reference values of lower extremity nerve conduction studies

Nerve	Amplitude (μV)	Conduction velocity (m/sn)	Distal latency (ms)
Peroneal nerve	≥2.0	≥44	≤6.5
Tibial nerve	≥4.0	≥41	≤5.8
Sural nerve	≥6.0	≥40	≤4.4
Medial plantar nerve	≥4.0	≥41	≤5.8
Lateral plantar nerve	≥3.0	≥41	≤6.3

Table 2. Reference values for MP and LP sensorial nerve conduction studies

	Amplitude (μV)	Conduction velocity (m/sn)
Medial plantar pure sensory	≥2	≥35
Lateral plantar pure sensory	≥1	≥35
Medial plantar mixed sensory	≥3	≥45
Lateral plantar mixed sensory	≥3	≥45

MP: Medial plantar; LP: Lateral plantar.

common late latency responses in electrodiagnosis laboratories. The H-reflex is often evaluated on the gastrocnemius-soleus muscle via the stimulation of the tibial nerve from the popliteal fossa and is the electrophysiological response of the Achilles reflex in this muscle. From the recording electrodes, the active electrode was placed in the gastrocnemius-soleus muscle and the reference electrode in the Achilles tendon.

The reference values of lower extremity nerve conduction studies were given in Table 1.[1] The difference in terminal latency of the MPN and LPN over 1 millisecond may be considered as TTS.[20,21]

The reference values for the MP and LP sensorial nerve conduction studies were summarized in Table 2.[18]

Statistical analysis

Statistical analyses were performed using SPSS for Windows 12.0 software (SPSS Inc., Chicago, IL, USA). The distribution of demographic characteristics of the patient and control groups was done by descriptive statistical methods. The Mann-Whitney U test was used to compare the variance to the normal curve or to compare ordinal data. Spearman rho coefficient was used for intra-group correlation. Values were given as mean ± standard deviation (SD). The values of $p<0.05$ were considered statistically significant.

RESULTS

A total of 100 feet of 30 RA patients and 20 control subjects were evaluated. Eighty percent of the RA patients (n=24) were seropositive. The demographic characteristics of the patient and control groups were summarized in Table 3. Patients with RA had a mean disease duration of 8.5 years (range, 1 to 28 years).

Among the RA patients, 26 (91.67%) had pain complaints for both feet; three patients had pain only in one foot (two right, one left). Only one patient had no foot pain (8.33%). The Tinel sign was positive in bilateral feet of eight patients and right foot of two patients (30%). Neurological examinations were otherwise normal. In RA patients with TTS, the Tinel sign was positive for bilateral feet in four patients and right foot for one patient (45%).

When the 100-m walking distance was compared between the patient and control groups, the walking duration in RA patients was longer than the control group (95.23±11.15 versus 84.35±6.74, $p<0.0001$).

There was no significant difference between peroneal motor amplitude, latency, and neural

Table 3. Demographic characteristics of patient and control groups

	Rheumatoid arthritis (n=30)		Controls (n=20)		*p*
	n	Mean±SD	n	Mean±SD	
Age (year)		41.9±10.1		39.3±10.8	>0.05
Sex					>0.05
Female	29		19		
Male	1		1		
Weight (kg)		69.5±11.6		66.3±9.5	>0.05
Height (cm)		161.1±6.5		159.7±5.0	>0.05
Body Mass Index (kg/m^2)		26.8±4.6		25.9±3.2	>0.05

SD: Standard deviation.

conduction velocity (NCV) values in the patient and control groups (p>0.05).

There was no significant difference in right tibial motor latency between the patient and control groups (p>0.05). The right tibial motor amplitude and NCV in the patient group was lower than the control group, with a significant difference (p<0.05). There was no significant difference in left tibial motor latency, amplitude, and NCV values in the patient and control groups (p>0.05).

There was no significant difference between the right and left sural sensory latencies and sensory conduction velocity (SCV) values in the patient and control groups (p>0.05). Right and left sural sensory amplitude values were lower in the patient group, with a significant difference compared to the control group (p<0.05).

The peroneal amplitude values of two RA patients and one healthy individual were at the lower limit (1.8, 1.8, and 1.0, respectively). Routine lower extremity neural conduction

Table 4. Routine lower extremity neural conduction values of study group

	Rheumatoid arthritis (n=30)	Controls (n=20)	
	Mean±SD	Mean±SD	*p*
Right tibial motor latency	3.4±0.6	3.4±0.7	>0.05
Right tibial motor amplitude	17.4±5.4	22.1±8.4	<0.05
Right tibial NCV	51.5±7.5	53.1±3.2	<0.05
Left tibial motor latency	3.5±0.6	3.4±0.6	>0.05
Left tibial motor amplitude	18.4±5.6	22.3±7.5	>0.05
Left tibial NCV	48.8±10.1	51.1±2.8	>0.05
Right peroneal motor latency	3.7±0.5	3.8±0.7	>0.05
Right peroneal motor amplitude	7.4±4.1	9.3±4.3	>0.05
Right peroneal NCV	50.9±4.5	49.7±11.2	>0.05
Left peroneal motor latency	3.7±0.5	3.6±1.1	>0.05
Left peroneal motor amplitude	7.1±3.5	8.3±3.5	>0.05
Left peroneal NCV	51.2±4.2	50.2±4.4	>0.05
Right sural sensory latency	3.6±0.6	3.7±0.5	>0.05
Right sural sensory amplitude	11.7±8.5	14.5±5.6	<0.05
Right sural SCV	50.6±6.5	51.6±6.0	>0.05
Left sural sensory latency	3.6±0.6	3.7±0.6	>0.05
Left sural sensory amplitude	10.5±5.9	13.9±6.5	<0.05
Left sural SCV	49.8±5.8	49.1±4.7	>0.05

SD: Standard deviation; NCV: Neural conduction values; SCV: Sensory conduction velocity.

Table 5. Hoffmann-reflex values of study group

	Rheumatoid arthritis (n=30)	Control (n=20)	
	Mean±SD	Mean±SD	*p*
Left H latency	30.0±2.2	29.9±2.1	>0.05
Left M-H latency	27.3±1.9	28.1±1.9	>0.05
Left M/H amplitude	3.6±3.2	2.0±1.8	<0.05
Right H latency	30.2±2.3	28.2±6.1	>0.05
Right M-H latency	27.7±1.8	28.0±1.4	>0.05
Right M/H amplitude	2.5±1.6	1.8±1.4	>0.05

SD: Standard deviation.

values of the study group were summarized in Table 4.

Left M/H amplitude was higher in the patient group, with a significant difference ($p<0.05$). There was no significant difference in the right and left H latencies, right and left M-H latencies, and right M/H amplitudes between the patient and control groups ($p>0.05$).

The H-reflex values of the study group were shown in Table 5.

In the patient group, right MP pure sensory sensory nerve action potential (SNAP) amplitude in three patients, left LP pure sensory SNAP amplitude in three patients, and right LP pure sensory SNAP amplitude in one patient could not be obtained.

Tarsal tunnel syndrome (33.33%) was found in 20 feet (10 patients in bilateral) of the 60 feet of RA patients. There was a decrease in both MPN and LPN velocities in TTS-detected patients. The electrophysiological evaluations of the subjects in the control group were normal.

DISCUSSION

Rheumatoid arthritis is a chronic, multisystemic, inflammatory rheumatic disease that affects the synovial joints primarily.[1-4] The most common cause of neurological involvement in RA is the neural compressions.[22]

Tarsal tunnel syndrome is an entrapment neuropathy that occurs when the posterior tibial nerve and/or one of its terminal branches is affected in the tarsal tunnel.[9,23]

The etiology of TTS is multifactorial. The most common cause of TTS is idiopathic. The cause of TTS was found to be idiopathic in 39-43% of cases in non-surgical series.[11] Among the known causes, trauma constitutes about one-third of all cases. One of the other two major causes of TTS is empty field lesions, and the other is foot deformities.[11]

Clinical evaluation, radiographic methods, MRI, and USG are used for the diagnosis of TTS. Electrophysiological studies confirm the diagnosis of TTS. These electrophysiological studies were found to be eligible and usable tests in 90-100% of patients for TTS diagnosis.[11]

Several studies have been conducted on TTS prevalence in RA. In their study, Baylan et al.[24] demonstrated the prevalence of TTS in RA as 25%. In our study, the frequency of TTS in RA was 33.33%.

Many electrophysiological techniques recommend the measurement of plantar NCV for the diagnosis of TTS. In our study, the distal motor latency (DML) of both MPN and LPN were found to be within the normal values in both RA and control groups. There was no extension in DML in any groups.

The specificity of mixed sensory conduction studies in TTS diagnosis is higher than pure sensory conduction studies, and mixed sensory conduction studies are more sensitive to the early changes than to pure sensory conduction studies.[25] In accordance with the TTS study protocol, both pure sensory and mixed sensory conduction studies of the MP and LP nerves were performed. Sensory nerve action potential amplitude in mixed sensory conduction studies

of the patient and control groups was simpler and easier to obtain than in the pure sensory conduction studies. Another averaging was not required. For this reason, when the MP and LP SCV of the study group were examined, they were evaluated especially considering the mixed sense conduction rates.[26]

Bilateral TTS was detected in 10 of the 30 RA patients (33.33%). The reason for this rate being higher than the previously conducted studies was due to the fact that TTS was mainly studied sensorially.

Tarsal tunnel syndrome is usually seen unilaterally. Galardi et al.[18] found the rate of bilateral presence as 8% in their study, and in another study conducted by Mondelli et al.,[9] this rate was found as 16%. In our study, however, all 10 patients with TTS were detected to have bilateral TTS. This result could be associated to the fact that the patients studied had RA, which is a systemic disease.

Tarsal tunnel syndrome is clinically characterized by pain, paresthesia and/or numbness in the foot and ankle. Only one of the RA patients (3.33%) who were included in the study did not have any foot pain complaints. Twenty-six patients (86.66%) had complaints in both feet, and three (10%) only in one foot.

The joint in which the disease started was associated with ankle and foot joints in more than one-third of the patients who had TTS ($p<0.05$). This suggests that the relationship between the process and severity of the affected foot joints and TTS is significant.

Only half of the patients diagnosed with TTS had very typical complaints. TTS was not detected in the electrophysiological studies of five patients with similar complaints. These results suggest that medical history taken from the patient is not enough in the entrapment neuropathy diagnosis and electrophysiological studies should support the diagnosis. Also, electrophysiological studies are helpful and significant for undelayed treatment.

Even though the tibial nerve DML and conduction velocities were similar in both the patient and control groups, the difference in the right side comparisons may be considered significant regarding showing the potential for the dominant extremity to be more affected.

Hoffmann-reflex is normal in TTS, but it may be abnormal in polyneuropathies, proximal tibial neuropathies, sciatic and lumbosacral plexus lesions, and S1 radiculopathies.[10] H-reflex latency and M-H latency values evaluated in RA patients and control group were found to be within the normal reference interval, with no significant difference between the two groups ($p>0.05$).

In the general health assessments of RA patients, there was a significant difference in all subscales of HAQ and SF-36 compared to the control group ($p<0.01$). Possibly, entrapment neuropathies contribute negatively in later periods.

Limitations of the study were the short duration of the patients' disease, non-compliance with EMG and rejection.

In conclusion, entrapment neuropathies are among the most common clinical variants in the peripheral nervous system involvement of patients with RA. In our study, the frequency of TTS electrophysiologically was higher in patients with RA than in other studies. The evaluation of RA patients should be performed more carefully in this regard. Diagnosis should be supported by electrophysiological studies when the medical history and physical examination are not sufficient.

Declaration of conflicting interests

The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

Funding

The authors received no financial support for the research and/or authorship of this article.

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