Original Article

Value of Motor Nerve Conduction Studies in the Diagnosis of Idiopathic Tarsal Tunnel Syndrome: A Single-center Prospective Observational Study from India

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Abstract

Background: Nerve conduction studies are considered to be the gold standard for diagnosing secondary tarsal tunnel syndrome (*s*TTS), but their utility in the diagnosis of idiopathic tarsal tunnel syndrome (*i*TTS) is largely unknown. **Objective:** We sought to investigate the value of motor nerve conductions studies (MNCS) in the diagnosis of clinically suspected *i*TTS. **Materials and Methods:** Twenty-six (52 limbs) adult patients of clinically suspected *i*TTS were subjected to motor nerve conductions of posterior tibial nerve, and its branches and motor conduction parameters were compared with those of 45 healthy controls. **Results:** Symptoms were bilateral in 70% ($P = 0.02$), with heel pain in 95% of symptomatic limbs. MNCS was abnormal in 32 (80%) of symptomatic limbs and 8 (66.6%) of asymptomatic limbs ($P = 0.004$). Out of electrophysiologically abnormal nerves ($n = 67$), the pathological process could be identified in all the nerves with abnormal MNCS ($P = 0.02$). Probable demyelination was seen in 58.2% of the electrophysiologically abnormal nerves. **Discussion:** The present study shows that *i*TTS are gender and Body Mass Index neutral with bilateral symptoms being common. Tinel's sign was inconsistent. Heel pain did not correlate with abnormal inferior calcaneal nerve conductions. Motor nerve conduction study was abnormal in a significant number of symptomatic limbs. "Probable demyelination" was more frequent in symptomatic limbs. **Conclusion:** MNCS is significantly abnormal in symptomatic limbs of subjects with *i*TTS. Demyelination is slightly more common than axonopathy in *i*TTS. With a sensitivity of 80% and specificity of 33.3%, MNCS seems to be useful as a screening tool in clinically suspected *i*TTS. This study is Level II: Lesser quality randomized controlled trial or prospective comparative study.

Keywords: Heel pain, motor nerve conduction studies, plantar neuropathy, tarsal tunnel syndrome

Introduction

Posterior tarsal tunnel syndrome (TTS) is characterized by symptoms over the plantar aspect of the foot, which exacerbate either on standing, prolonged walking, or wearing tight footwear. The symptoms of TTS include pain, numbness, tightness, or burning sensations over the sole and tend to vary in severity and distribution between the patients to such an extent that it is recommended to consider the possibility of TTS in all those who present with sensory symptoms in the sole.[1]

The TTS is often secondary to foot deformity, local trauma, diabetes, accessory muscle, arthritis, sports injuries, nerve tumor, or obesity. However, in up to 25% of the patients no specific cause can be identified, these are often called *i*TTS.[2-5]

The tarsal tunnel (TT) is a 2.5–3 cm wide osteofibrous canal roofed by a strong flexor retinaculum.[4] Tibial nerve branches out in TT and posterior tibial nerve (PTN) could be entrapped behind the medial malleolus, but there are several other potential compression sites along the course of its branches in the foot, where they are in a proximity of tough fibrous septae of the foot.[4,6] Tawfik *et al*. and Kim *et al*. have measured the cross-sectional area of TT using neuromuscular ultrasound and MRI respectively, in the controls and the patients with *i*TTS and discovered that it was significantly small in the later.[7,8] A clinical syndrome similar to TTS, called "distal TTS," can

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result from compression of one (or more) of plantar nerves in fibroosseous tunnels under abductor hallucis.[1,2,9] Isolated heel pain could be due to PTN entrapment in TT, apart from the isolated involvement of its branches, namely, medial plantar (MPN), lateral plantar (LPN), and inferior calcaneal nerve (ICN).[10]

Various investigative modalities including plain radiography, neuromuscular ultrasound, and MRI have been used to substantiate the clinical diagnosis of TTS; however, the electro-diagnostic tests are considered to be a gold standard for diagnosing the entrapment of PTN in TT.[4]

A view in contrast to that of Patel *et al*., who reviewed the usefulness of electrodiagnostic studies in the evaluation of suspected TTS, discovered some associations between abnormal nerve conductions and clinical symptoms. They, however, failed to identify any definite role of electrophysiology in the diagnosis of TTS. Procedural differences and methodological flaws have been cited as the cause of failure to derive any conclusive evidence to support the use of electrodiagnostic studies in the evaluation of suspected TTS. Not elaborating the protocols, used for measurement of the electrodiagnostic findings, has made the comparison and the evaluation of the published work even more challenging.[11]

Researchers have struggled to discover the more dependable electrodiagnostic procedure and an easily recordable parameter for confirming the clinically suspected TTS. Reports are indicating that frequent abnormalities are present in the sensory nerve parameters, rather than the motor nerve conductions.[12] However, obtaining a sensory nerve action potential (SNAP) from the nerves in the foot is not only time-consuming but also technically challenging, and often invasive techniques are required. Oh *et al*. could record SNAP from LPN nerve only after inserting a needle electrode close to the PTN in the ankle. In another study, the same researcher reported that the averaging up to 256 stimuli were required to obtain a reliable SNAP.[12] Recording a SNAP using surface electrodes by stimulating the mixed nerves in the medial or lateral sole, has also been reported to be difficult and unreliable.[13,14]

Keskin *et al*. studied the reliability of medial and LPN nerve recordings with bar electrodes in the healthy elderly individuals and noted that the responses were absent in up to 53% of the patients.[15] In contrast, Schon *et al*. evaluated the electrodiagnostic support for nerve entrapment in the patients with heel pain and found motor nerve conduction studies (MNCS) to be the most helpful diagnostic test.[13] A significant number of investigators have also found NCS to be a good diagnostic test, as it is easy to perform and less time-consuming.[1,14] Kaplan and Kernahan studied MNCS in the assessment of TTS and compared the findings with the control population. They did not find any difference in NCS parameters between the control group and the unaffected side of patients with TTS while the affected side showed abnormalities either in the distal latency or the duration of compound muscle action potential (CMAP).[16]

Several case series are on record describing the findings of electrodiagnostic tests in *s*TTS[3,12-15,17,18] and establishing the usefulness of electrodiagnostic tests in the diagnosis of *s*TTS, but its value in the diagnosis of *i*TTS has been sparingly studied.

In this study, we prospectively investigated the value of motor conductions of the PTN and its branches in the diagnosis of *i*TTS. Based on the MNCS findings we also attempted to classify the underlying pathology into demyelination or axonopathy.

Materials and Methods

This prospective study was conducted at Sri Aurobindo Institute of Medical Sciences and Postgraduate Institute, Indore, a teaching hospital in the rural setting. Patients referred between April 2014 and January 2016 to our electrodiagnostic facility with clinical suspicion of *i*TTS formed the study group. Controls were drawn from the healthy subjects invited through posters placed in the hospital and the medical college. Patients and controls underwent a detailed history and clinical examination.

The history explored the foot pain characteristics, distribution of the pain (lateral sole, medial sole, heel, or more than one location), aggravating, and relieving factors of the pain. Medical history of diabetes, arthritis, and claudication was obtained. Physical examination included the recording of the weight, height, palpation of distal pedal pulses, elicitation of Tinel's sign over the TT.[1,19] The body mass index (BMI) was calculated using Du Bois method.[20] The examination also included the elicitation of deep tendon reflexes and sensory evaluation of the soles with a 10 G monofilament (Darco), at four sites (heel, the base of the toes, and the plantar aspect of great toe).[21] The distribution of sensory symptoms and signs were mapped on a template and grouped as per the location into the medial sole, lateral sole, only heel and more than one site.

Motor nerve conduction study was performed by one of the investigator (MD) on Nicolet Viking (version 12.0). Surface disk electrodes were used for recording the response. The MPN,[12] LPN,[22] ICN,[23] tibial[24] and sural[24] nerves were evaluated on both the sides, as per the previously described methods. The sural nerve was stimulated on the calf slightly lateral to the midline in the lower third of the leg behind 140 mm proximal to the lateral malleolus and recorded by placing the recording electrode behind the lateral malleolus. The tibial nerve was stimulated behind medial malleolus 80 mm proximal to the active electrode, placed over the most prominent part of the belly of abductor hallucis muscle and in the popliteal fossa.

The PTN was stimulated at the ankle (proximal to the flexor retinaculum, 1 cm posterior to the medial malleolus) and the response was recorded 80 mm distal to the stimulation point over the abductor hallucis muscle (MPN).[12] The PTN stimulated sequentially, at the site as mentioned above and

responses were recorded over abductor digiti quinti (ICN)[23] -halfway between the tip of the lateral malleolus and sole and stimulated behind medial malleolus 80 mm proximal to the active electrode, placed as while testing abductor hallucis muscle, known as Fu's method and over flexor digitiminimibrevis muscle-along the midpoint of the inferolateral edge of the 5th metatarsal (LPN), the stimulation point being the same as was for the recording from abductor hallucis muscle.[22] During the electrodiagnostic study, the skin temperature of the limb was maintained between 29ºC and 31ºC. In the case of no response or a low CMAP amplitude, the procedure was repeated after altering the electrodeposition and or the inter-electrode distance; best response was included in the analysis.

The following electrodiagnostic parameters were noted as follows:

a. Terminal latency or distal latency (l):[24] stimulus artifact to the take-off of the negative peak of the motor response, in ms. For measuring the latency of ICN CMAP, the initial negative peak, if present, was ignored
b. CMAP amplitude (a):[24] measured from the baseline to negative peak, millivolts (mV)
c. Duration of the negative peak of CMAP (d):[24] from the onset to the first base line, cross of negative peak
d. Sural nerve sensory action potential latency
e. SNAP amplitude
f. Tibial nerve motor conduction velocity between the knee and the ankle.

Based on the above observations, following parameters were further computed

1. Medial plantar latency (MPl)
2. Duration of negative peak of the medial plantar CMAP (MPd)
3. Lateral plantar nerve latency (LPl)
4. Duration of the LPN CMAP (LPNd)
5. Amplitude of the medial plantar CMAP (MPa)
6. Amplitude of the LPN CMAP (LPa)
7. ICN latency (ICNl)
8. Amplitude of the ICN CMAP (ICNa)
9. Duration of the ICN CMAP (ICNd).

Values of terminal latency, duration, and amplitude of the CMAP were used for identifying the demyelination or axonopathy in peripheral nerves. The former reflects in MNCS as delayed distal latency in prolonged duration of the negative peak of CMAP; the later reflects a reduction in the amplitude of negative peak.[25,26] Accordingly, nerves with delayed DL, (>2 standard deviation [SD] of the upper limit of normal[ULN]) with or without prolongation of the duration of the CMAP, were considered to have "probable demyelination" while nerves showing low CMAP amplitude with normal DL were considered to be having "probable axonopathy."

Only those who fulfilled the following criteria were included in the study:

a. The presence of sensory symptoms (pain, tingling, burning, tightness, and jabs) restricted to the plantar aspect of the foot. The symptoms, not relieved by analgesics, should have persisted for more than 4 weeks, preceding the day of inclusion
b. Symptoms should be persisting most of the day, with aggravation on standing, walking, prolonged standing or wearing footwear
c. Absence of tenderness at the medial calcaneal tuberosity and middle portion of plantar fascia, low back pain, pain in legs, diabetes, rheumatoid arthritis, history of surgery in the symptomatic limb or spinal surgery, foot deformity, gout, family history of burning feet, signs of distal symmetric neuropathy (graded distal sensory loss, and absent ankle jerks), and abnormal dorsalispedis pulsations
d. Should not have received local steroids or analgesic injections.

Those who had abnormal knee and ankle tibial motor conductions (slow conduction velocity, conduction block, or temporal dispersion[27]) and sural SNAPs (low amplitude) were excluded.

Both the controls and the patients were explained the procedure in detail and were required to give written consent, failing which, they were excluded from the further study. The study protocol was approved by the institutional ethical committee.

Statistical analysis

For analysis, the data were grouped according to their gender, age, and BMI from both control and the study population. The patients with unilateral and bilateral symptoms were divided into two groups. The limbs were segregated, according to the presence or absence of the symptoms, into symptomatic and asymptomatic groups. The study population was further grouped as per the distribution of symptoms over the sole, MNCS findings, pathology (probable demyelination or probable axonopathy) and abnormalities in different nerves. The symptomatic and the asymptomatic limbs were compared to calculate the sensitivity and specificity of MNCS in picking up abnormal motor conductions in the symptomatic limbs.

We hypothesized that MNCS would be abnormal in symptomatic feet and would be normal in the asymptomatic feet. Furthermore, the MNCS results would enable us to identify the underlying process (axonopathy or demyelination). Results found in line with the above hypothesis were considered as a good outcome of the intervention (MNCS), and a 2 × 2 contingency table was used to calculate the two-tailed *P* value. The sensitivity and specificity were calculated using Medcalc diagnostic test calculator.[28]

The ULN for the distal latency and lower limit of normal for CMAP amplitude were calculated from the control data at 95%. CI and the values beyond 2SD were considered abnormal. Various MNCS parameters from each of the three nerves (tested in each limb) were evaluated against the computed normal values from the controls. Based on this the limbs were classified

into electro-diagnostically "normal" (when distal latency, CMAP amplitude, and duration were normal in all the three nerves) or "abnormal" (when distal latency or CMAP amplitude or CMAP duration was abnormal in one or more nerves).

We used Medclac,[28](Medcalc Software) Microsoft Excel 2010,[29] (Microsoft Corporation) and GraphPad Software,[30] (GraphPad Software, Inc.)for statistical analysis. The descriptive analysis of numerical variables was shown as an arithmetic mean ± 2SD and the analysis of categorical variables as a percentile. Unpaired *t*-test was applied to compare the mean of two groups and the *P* values at 95% CI ($P < 0.05$, as per Fisher's exact test) were calculated.

Results

Initially, 52 clinically normal persons were recruited to serve as controls, out of them, seven had to be excluded due to abnormal sural SNAPs. The remaining 45 patients (90 limbs) were included as controls.

During the study period, 40 patients were referred for evaluation of clinically suspected *i*TTS. Twelve of them were excluded due to various reasons (4-foot deformity, 6 with diabetes, one history of local trauma, and one lack of consent). After NCS 2 more patients were excluded, due to abnormal sural SNAPs. Remaining 26 patients fulfilled the inclusion criteria, giving 52 limbs (40 symptomatic and 12 asymptomatic) and 156 nerves (120 nerves in symptomatic limbs and 36 in asymptomatic). The clinical characteristics and electrodiagnostic findings of the control and patients are provided in Tables 1 and 2, respectively.

The age, gender, and BMI did not show statistically nonsignificant difference between the control and the study population. BMI of patients with abnormal MNCS ($n = 24$)

Table 1: Demography and nerve conduction parameters in controls ($n=45$)

Parameters	Values	*P**	Upper limit of normal	Lower limit of normal
Age (mean±2SD)	36.3±12.8			
Gender (number/percent)				
Male	22 (48.9)			
Female	23 (51.1)			
BMI (mean±2SD)	22.5±4.5			
NCS findings				
Right MPl (ms)±2SD	4.34±0.65	0.57 (NS)	4.99	
Left MPl (ms)±2SD	4.42±0.71		5.13	
Cumulative †MPl (ms)±2SD	4.38±0.68		5.06	
Right MPa (mV)±2SD	13.13±3.96	0.74 (NS)		9.17
Left MPa (mV)±2SD	12.88±3.44			9.44
Cumulative MPa (mV)±2SD	13±3.69			9.31
Right MPd (ms)±2SD	5.68±0.91	0.52 (NS)		4.77
Left MPd (ms)±2SD	5.81±1.02			4.79
Cumulative MPd (ms)±2SD	5.75±0.96			4.79
Right LPl (ms)±2SD	6.41±1	0.58 (NS)	7.41	
Left LPl (ms)±2SD	6.54±1.22		7.76	
Cumulative LPl (ms)±2SD	6.47±1.1		7.57	
Right LPa (mV)±2SD	6.29±2.61	0.50 (NS)		3.68
Left LPa (mV)±2SD	6.66±2.59			4.07
Cumulative LPa (mV)±2 SD	6.47±2.59			3.88
Right LPd (ms)±2SD	4.44±1.07	0.79 (NS)		3.37
Left LPd (ms)±2SD	4.50±1.09			3.41
Cumulative LPd (ms)±2SD	4.47±1.07			3.4
Right ICNl (ms)±2SD	6.42±1.08	0.81 (NS)	7.5	
Left ICNl (ms)±2SD	6.36±1.31		7.67	
Cumulative ICNl (ms)±2SD	6.39±1.2		7.59	
Right ICNa (mV)±2SD	4.83±2.58	0.83 (NS)		2.25
Left ICNa (mV)±2SD	4.95±2.92			2.03
Cumulative ICNa (mV)±2SD	4.89±2.74			2.15
Right ICNd (ms)±2SD	3.71±0.86	0.95 (NS)		2.85
Left ICNd (ms)±2SD	3.70±0.76			2.94
Cumulative ICNd (ms)±2SD	3.70±0.80			2.9

*Unpaired *t*-test at 95% CI, †Sum total of right and left limbs (n=90). MPl = Medial plantar distal latency, LPl = Lateral plantar distal latency, MPa = Medial plantar amplitude, MPd = Medial plantar CMAP duration, LPa = Lateral plantar CMAP amplitude, LPd = Lateral plantar CMAP duration, ICNl = Inferior calcaneal nerve distal latency, ICNa = Inferior calcaneal nerve CMAP amplitude, ICNd = Inferior calcaneal nerve CMAP duration, NCS = Nerve conduction study, BMI = Body mass index, NS = Nonsignificant, SD = Standard deviation

did not differ significantly than that of the control ($P = 0.26$, N−1 Chi-squared test).

In our study population, bilateral symptoms were significantly more frequent (n = 28, 70%) than unilateral (n = 12, 30%, $P = 0.02$, N−1 Chi-squared test). We found the pain or sensory symptoms restricted to any single foot location to be rare. In only nine limbs (22.5%), the pain was restricted to a single foot location (7 in the heel area and 1 each in the medial and the lateral sole). The heel pain was common ($n = 38$, 95%), hence was the pain in multiple locations of the foot ($n = 31$, 77.5%). The mean total duration of symptoms was 13.78 months.

Table 2: Clinical characteristics of study population ($n=26$)

Parameters	Values	P
Age years, mean±2SD (control)	42±15 (36.3±12.8)	0.1 (NS)†
Gender, *n* (%)		
Male; control	12 (46); 22	0.8 (NS)†
Female; control	14 (54); 23	0.8 (NS)†
Limbs (*n*=52), *n* (%)		
Symptomatic	40 (77)	
Asymptomatic	12 (23)	
Symptom and signs in symptomatic limbs		
Distribution, *n* (%)		
Unilateral	12 (30)	0.02 (S)*
Bilateral	28 (70)	
Medial sole	1 (2.5)	
Lateral sole	1 (2.5)	
Only heel	7 (17.5)	
More than one location	31 (77.5)	
Signs, number of limbs, *n* (%)		
Tinel's	3 (7.5)	
Sensory loss	7 (17.5)	
BMI versus control mean±2SD		
Total patients	24.23±4.7	0.1 (NS)†,‡
Patients with abnormal NCS	23.75±4.52	0.26 (NS)†

*S (*n*-1 Chi-square test); †NS (*n*-1 Chi-square test); ‡Compared with control. NCS = Nerve conduction study, BMI = Body mass index, NS = Nonsignificant, S = Significant, SD = Standard deviation

The MNCS was abnormal in 32 out of 40 (80%) of the symptomatic and 8 out of 12 (66.6%) of asymptomatic limbs. This difference was statistically significant (two-tailed $P = 0.004$, Fisher's exact test). The MNCS test had a sensitivity of 80% and specificity of 33.3% for picking up an abnormality in limbs symptomatic of *i*TTS. The apparently low specificity could be to the ability of MNCS to pick up subclinical abnormalities in the asymptomatic limbs [Table 3].

MNCS abnormalities in more than one nerve were found in 40% of symptomatic limbs and 25% of the asymptomatic limbs. Decreased CMAP amplitude abnormality was the most common MNCS finding in symptomatic as well as asymptomatic limbs, occurring in isolation as well as in combination with altered latency and/or CMAP duration (29% and 22%, respectively; annexure 1 and 2). Out of 156 nerves, 67 (43%) showed abnormal MNCS [Table 3]. Based on the abnormalities of terminal latency, CMAP amplitude, and CMAP duration either a demyelination or axonal process could be identified in each of the abnormal nerves. MNCS was abnormal in 46.6% (56 out of 120) nerves of the symptomatic limbs. Out of 56 nerves, 35 (62%) had "probable demyelination" and 37.5% (21 of 56) nerves showed features suggestive of "probable axonopathy." In asymptomatic limbs, 11 out of 36 nerves showed electrophysiological abnormalities, and they could be classified as "probable demyelination" ($n = 4$) or "probable axonopathy" ($n = 7$).

In our patients, Tinel's sign ($n = 3$) and sensory deficits ($n = 7$) were not very common. Two of the patients with Tinel's and 6 with sensory deficits had abnormal MNCS. We found heel pain to be a common symptom, with 38 out of 40 (95%) symptomatic limbs having it. The ICN abnormality was detected in only 17 limbs (44.7%).

Table 3: Classification of study population as per the nerve conduction study findings

Variables	MNCS classification	Values, *n* (%)	Significance level
Total limbs (*n*=52)			
Symptomatic limbs (*n*=40)	Abnormal	32 (80)	*P*=0.004 (S)* sensitivity 80%, specificity 33.3%
	Normal	8 (20)	
Asymptomatic limbs (*n*=12)	Abnormal	8 (66.6)	
	Normal	4 (33.4)	
Total nerves (*n*=156)			
Symptomatic limbs (*n*=120)	Normal	64 (53.3)	
	Abnormal	56 (46.7)	
	Probable Demyelination	35 (29.1)	
	Probable Axonopathy	21 (17.5)	
Asymptomatic limbs (*n*=36)	Normal	25 (69.44)	
	Abnormal	11 (30.6)	
	Probable Demyelination	4 (11.1)	
	Probable Axonopathy	7 (19.4)	

*Significant two-tailed *P* value comparison between symptomatic and asymptomatic. MNCS = Motor nerve conduction study

Discussion

The present study shows that *i*TTS are gender and BMI neutral and that these patients rarely have pain restricted to single sole location. Moreover, a significant number of our cases had bilateral symptoms. This is in contrast to *s*TTS where unilateral symptoms are more common. Oh *et al*., Kaplan and Kernahan., and Urgüden *et al*. studied 13, 8, and 21 patients of *s*TTS, respectively, and did not find bilateral symptoms in anyone.[3,16,31] An and Kim *et al*. reported bilateral *i*TTS in 12 of their 21 patients and concluded that bilateral *i*TTS were more common in their study.[18] Kohno *et al*. did neurovascular decompression for 9 patients of suspected *i*TTS and found that 3 (33%) of their patients had bilateral symptoms.[32]

Although Tinel's sign is considered to be an important physical sign of TTS, it is not consistently present in all the patients. The surgically oriented studies of *s*TTS[12,17,31] and *i*TTS[32] report it to be present in 90%–100% the cases, in contrast, a medically oriented retrospective study of *i*TTS has reported this sign in 51% feet.[18] In our prospective study, we found Tinel's sign to be present only in 7.5% of symptomatic limbs. Since its description, the clinical value of Tinel's sign has remained in question.[33] The similar sign can be elicited by tapping a nerve distally in proximally injured nerves. Tinel's sign is a feature of renervation rather than of nerve compression. After focal nerve injury and later renervation of the nerve fibers at the site of the lesion is required for the generation of spontaneous and reverberating bursts of ectopic impulses on tapping the nerve locally.[34,35] It is therefore not surprising for the Tinel's to be absent if the pathological process in the nerve is predominantly restricted to myelin sheath or if the site of percussion and that of injury do not match. We elicited this sign by tapping behind the medial malleolus, and it is possible that in many of our patients the TTS like symptoms might have been due to compression of branches of PTN in the fibroosseous tunnel of the foot rather than compression of PTN itself in TT.

We found that heel pain does not correlate with abnormality ICN conductions. Ninety-five percent of the symptomatic limbs in our study had heel pain, but corresponding ICN abnormality was seen in 44.7% limbs only. The ICN may be the common cause of heel pain but not specific which has been found in subjects with the involvement of tibial, plantar, or calcaneal nerves.[10]

The value of electrodiagnostic tests in *s*TTS is well-established,[12,13,36] but its place in the diagnosis of *i*TTS has not received a focused attention. In this study, MNCS was abnormal in a significant number of symptomatic limbs [Table 3]. However, because we also found a subclinical abnormality in 66.66% of asymptomatic limbs the sensitivity of this test seems to be erroneously low. Nerve conduction abnormality in the asymptomatic limbs of *i*TTS has been reported previously.[5,13]

In TTS, the nerves have been reported to show demyelination as well as axonal degeneration.[25,26,37] Histological evaluation of the nerve samples of patients with TTS has been reported to show a marked loss of myelinated fibers.[38] We could identify either a demyelinating or an axonal process in all the nerves with abnormal MNCS and that the "probable demyelination" was more frequent in symptomatic limbs.

It is known that the cross-sectional area of the TT in patients with *i*TTS is smaller than the control population.[5,7,8] It is noteworthy that PTN and its branches (MPN and LPN) are supplied by multiple blood vessels, often abnormally tortuous,[2] within the TT as they pass through different tight fibroosseous canals.[39] Fullerton and Gilliatt have shown that the plantar nerves are susceptible to repeated minor trauma.[40] In their elegant experimental study, they found that animals kept in confinement with the wire-mesh floor, developed demyelinating plantar neuropathy characterized by prolongation of the terminal latency and dispersion of CMAP, confirmed to be segmental demyelination in the nerve samples of those animals.[40] It is our hypothesis that the rich vascular supply, intra-TT varicose vein, tethering of nerves in the foot and multiple potential sites for compression and developmentally narrow TT make the PTN and its branches susceptible to trivial trauma and may be responsible for neurological symptoms and electrophysiological signs in the symptomatic, as well as the asymptomatic limbs of the patients with *i*TTS.

Small sample size, nonavailability of the values of the cross-sectional area of fibro-osseous TT by USG or MRI to rule out *s*TTS, study design based solely on motor nerve conductions but without across the flexor retinaculum motor study, with no sensory nerve conduction studies and EMG and finally, absence of blinding is the limitations of our study.

This study based on clinical suspicion of *i*TTS with corroborating motor nerve conduction study can indicate the PTN entrapment and can be used as a screening tool but in future, studies based on radiological parameters, sensory nerve conductions, and EMG to rule out *s*TTS will be required.

Conclusion

This single-center observational study evaluating the value of the electrodiagnostic test in the diagnosis of *i*TTS shows that MNCS is significantly abnormal in symptomatic limbs. MNCS can be used to identify the pathological process. The specificity of the MNCS for diagnosing *i*TTS appears erroneously low because of its ability to identify a subclinical abnormality in asymptomatic limbs. The value of MNCS, as a screening tool for *i*TTS, should be explored with a double-blind population-based randomized study.

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Conflicts of interest

There are no conflicts of interest.

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Annexure

Annexure 1: Motor nerve conduction study parameters of medial, lateral plantar and inferior calcaneal nerves ($n=40$) in symptomatic limbs

Symptomatic limb number	MPl	MPa	MPd	LPl	LPa	LPd	ICNl	ICNa	ICNd
1	***5.4***	11.6	5.2	5.7	4.1	4.7	6	4.9	***5.1***
2	3.3	17.3	5	5.2	7	4.2	4.9	10.8	4
3	3.8	15.5	5	5.6	5.8	3.5	5.7	7	3
4	4.3	16	5.6	6.4	8.9	3.8	5.2	3.6	***4.7***
5	4	15.9	6	6.5	4.5	4.4	6.6	7.9	3.3
6	***5.4***	***7.5***	5.5	5.5	***1.9***	1.9	6.3	2.3	2.3
7	3.9	***7.6***	4.7	***8.1***	***2.9***	5.3	7.6	2.3	3.3
8	4.3	15.1	6.3	5.6	11.5	***5.7***	7	12.1	4.1
9	4.8	11.5	6	6	8.1	***6.3***	6.4	8.1	4.1
10	4.4	13.7	6	6.3	11	5.5	6.7	8.9	3.8
11	***7.1***	***2.4***	***7.1***	***8.4***	***0.6***	3.6	***8.3***	***1.6***	***5.5***
12	4.4	***8.3***	5.9	6.5	4.6	3.5	4.7	4.8	4.3
13	4	***8***	5.9	7	***3.3***	3.8	5.1	3.3	***5.3***
14	4.3	16.3	***7***	6.5	***3.8***	5.3	6.7	3.7	3.9
15	***5.5***	***9***	***6.9***	7.3	9.1	***6.6***	***8.8***	2.7	***5.4***
16	***5.1***	***9.1***	***7.6***	***8.5***	7.2	***6.3***	***9.4***	4.6	3.8
17	4.1	***9.5***	6	5.8	4.3	***6.4***	6.9	3.4	4
18	4.1	11.5	5.8	6.3	5.5	***5.9***	7.2	4.6	3.7
19	4.6	13.3	5.9	5.2	9.9	5.3	6.9	10.6	3
20	4.6	10	5.1	5.9	7.9	***6.3***	7	6.7	4.1
21	5	***6.7***	6.5	***7.5***	***1.2***	5.3	***7.8***	2.6	***5.3***
22	5	***7.4***	***7***	5.4	***1.3***	***6***	6.7	3.2	***5.7***
23	3.8	11.1	5.7	5.8	***2.2***	4.5	6.4	6.5	3.6
24	3.8	10.5	5.8	6.3	***2.6***	***5.9***	6.2	4.6	3.2
25	4	14.7	***7.3***	6.2	5.1	4.7	6.7	3.5	2.7
26	4.7	***10.5***	6.6	7.1	7.1	4.7	6.4	7.4	4.3
27	3.9	20.1	5.3	6	6.8	3.9	5.4	6.2	3.6
28	3.8	11.6	5.1	5	7.9	4.2	5.7	5.5	3.7
29	***5.2***	10.7	4.9	6.3	***3.1***	4.2	7.3	***1.8***	3
30	4.5	10.4	5.9	***7.6***	***3.2***	4.6	***7.7***	***0.4***	3.5
31	4.3	***6.2***	5.3	7.1	5	5.4	7.2	4	3
32	4.4	13.3	5.2	5.5	5.6	4.6	7.1	***0.6***	3.3
33	4	14.6	5.4	5.8	5.7	4.2	6.4	2.4	4.1
34	4.1	15	4.7	5.7	8	3.4	6.4	***1.3***	3.6
35	4.5	15.9	5.6	6.3	10.6	4.6	4.8	***0.7***	***4.8***
36	4.3	***7.1***	4.9	6	5.4	***6.1***	4.8	***0.9***	3.1
37	3.9	***8.4***	4.3	5.8	5.3	4.7	4.7	***1.9***	2.3
38	3.8	13.8	6.4	5.1	7.2	***6.4***	5.8	3	2.5
39	3.6	14	6.5	5.3	5.2	***6.7***	6.7	2.7	2.1
40	3.9	10.6	5.2	5.4	5.4	4.4	5.8	4.1	3.2
Total abnormal MNCS parameters	6	14	6	5	11	12	5	8	8

Bold and Italicized letters denote abnormal values. MNCS = Motor nerve conduction study, MPl = Medial plantar distal latency, MPa = Medial plantar CMAP amplitude, MPd = Medial plantar CMAP duration, LPl = Lateral plantar distal latency, LPa = Lateral plantar CMAP amplitude, LPd = Lateral plantar CMAP duration, ICNl = Inferior calcaneal nerve distal latency, ICNa = Inferior calcaneal nerve CMAP amplitude, ICNd = Inferior calcaneal nerve CMAP duration

Annexure 2: Motor nerve conduction study parameters of medial, lateral plantar, and inferior calcaneal nerves (*n*=12) in asymptomatic limbs

Asymptomatic limb number	MPl	MPa	MPd	LPl	LPa	LPd	ICNl	ICNa	ICNd
1	4	11.7	5.9	6.8	***3.6***	***7.3***	6.7	6.5	3.5
2	3.6	11.3	5.2	6.6	***3.6***	3	6.4	5.2	3.1
3	4	16.6	5.8	6.3	4.9	4.2	6.6	11.2	3.6
4	4.7	13.3	6.6	5.3	9.5	4.1	6.4	10.6	***5.1***
5	4.2	***8.6***	6.5	6.4	4.1	3.8	5.7	7.2	***4.6***
6	4.4	15.7	6.4	5.6	***3.5***	4.6	6.4	6.5	3.4
7	***5.4***	18.4	3.7	6	6	3.6	5.7	5.4	3.3
8	3.6	11.3	5.5	5.1	6.4	4.1	5	4.1	4.5
9	4.8	***6.2***	5.7	6.8	4	5.4	7.4	***1.5***	2.7
10	4.2	9.2	4.9	5.2	***3.1***	4.7	6.6	***0.7***	5
11	3.4	16.7	5.3	5.5	5.9	4.4	4.9	3.4	2.8
12	3.9	9.5	6.5	5.3	4.6	4.9	6.5	3.3	3.4
Total abnormal MNCS parameters	1	2	0	0	4	1	0	2	2

Bold and Italicized letters denote abnormal values. MNCS = Motor nerve conduction study, MPl = Medial plantar distal latency, MPa = Medial plantar CMAP amplitude, MPd = Medial plantar CMAP duration, LPl = Lateral plantar distal latency, LPa = Lateral plantar CMAP amplitude, LPd = Lateral plantar CMAP duration, ICNl = Inferior calcaneal nerve distal latency, ICNa = Inferior calcaneal nerve CMAP amplitude, ICNd = Inferior calcaneal nerve CMAP duration