—Review—

Tarsal Tunnel Syndrome: A Clinical Review

Hiroto Yoshida[1,2], Kyongsong Kim[3], Takato Tajiri[2], Fumiaki Fujihara[2], Juntaro Matsumoto[1,2], Hiroshi Abe[2] and Toyohiko Isu[4]

[1]Department of Neurosurgery, Matsumoto Hospital, Fukuoka, Japan
[2]Department of Neurosurgery, Faculty of Medicine, Fukuoka University, Fukuoka, Japan
[3]Department of Neurological Surgery, Nippon Medical School Chiba Hokusoh Hospital, Chiba, Japan
[4]Department of Neurosurgery, Kushiro Rosai Hospital, Hokkaido, Japan

Tarsal tunnel syndrome (TTS) is elicited by compression of the tibial nerve in the tarsal tunnel. TTS in the absence of a lesion tends to be idiopathic, and most TTS is idiopathic. Patients complain of several plantar symptoms, and TTS affects their quality of life. The symptoms tend to worsen with walking, and ankle joint movement and arterial distortion may also be involved. Because TTS symptoms are similar to those of diabetic neuropathy and lumbar disease, clinical symptoms are diagnostically important. While magnetic resonance imaging reveals nerve compression, it is difficult to identify causative factors, and false-positive results are a concern. Wound-related complications after TTS surgery may be reduced by a zigzag skin incision. Surgery for carpal tunnel syndrome yields better outcomes and greater patient satisfaction than TTS surgery. (J Nippon Med Sch 2025; 92: 132–137)

Key words: tarsal tunnel syndrome, diagnosis, entrapment neuropathy, neurolysis, surgical outcome

Introduction

Compression of the tibial nerve in the tarsal tunnel leads to tarsal tunnel syndrome (TTS). TTS affects patient quality of life but can be treated by less invasive surgery[1,2]. TTS may be underdiagnosed and its true incidence is unknown. Opinions on its diagnosis and surgical treatment vary[3].

Epidemiology and Etiology

TTS appears to be slightly more common in women[4,5]. Suspected causes include space-occupying lesions such as ganglia and schwannomas, post-traumatic adhesions, compression due to dilated vessels, hypertrophy of the flexor retinaculum, and accessory muscles. Pulsation due to tortuous arteries may contribute to TTS pathogenesis, as pulsatile compression may occur in a closed space[4,6–9].

Kim et al.[8] reported MRI findings for 28 feet treated by TTS surgery and found a tortuous artery in 22 feet, a varix in 3 feet, and a ganglion, connective tissue, and a small vascular branch in 1 foot each. Vascular dilation and distortion within the tarsal tunnel and hypertrophy of the flexor retinaculum are age-related physiological changes. Because adhesions may be present in the tarsal tunnel in the absence of trauma, TTS in the absence of a lesion tends to be considered idiopathic[4,5,10], and the incidence of idiopathic TTS was reported to be 18-96% of all TTS cases[3,8,11]. According to Reichert et al.[11], among 31 TTS patients, 11 (35.5%) were diagnosed with idiopathic or trauma-related TTS and 9 with space-occupying lesions. Samarawickrama et al.[12] reported that 3 of 6 TTS patients had flat feet. An ultrasound study by Doneddu et al.[13] to identify the primary compression site in patients with idiopathic TTS suggested that it was located where the nerve had become larger at the middle of the tarsal tunnel. Tajiri et al.[9] reported MRI findings indicating that in 14 of 15 sides the most severe compression point was in the proximal half of the tarsal tunnel.

Clinical Anatomy

The medial and lateral plantar nerves (MPN, LPN) branch off the tibial nerve (TN) near the tarsal tunnel. Although the branching sites vary, they most commonly

Correspondence to Kyongsong Kim, MD, PhD, Department of Neurological Surgery, Nippon Medical School Chiba Hokusoh Hospital, 1715 Kamagari, Inzai, Chiba 270–1694, Japan
E-mail: kyongson@nms.ac.jp
https://doi.org/10.1272/jnms.JNMS.2025_92-206
Journal Website (https://www.nms.ac.jp/sh/jnms/)

branch at the proximal tarsal tunnel[14–16]. The MPN provides sensation from the medial side of the sole and the first 3.5 digits[3,16]. It also supplies muscles on the medial side of the sole, e.g., the abductor hallucis, flexor digitorum brevis, and flexor hallucis brevis. The LPN provides sensation from the lateral side of the sole to the lateral side of the fourth and little toe and mainly supplies plantar muscles such as the adductor hallucis, flexor digiti minimi brevis, and quadratus plantaris.

There are 2 main nerve branches in the tarsal tunnel: the medial and inferior calcaneal nerves (MCN, ICN). The MCN, which provides sensation to the heel, mainly branches from the LPN but may also branch from the TN[16]. There are 1-3 MCNs, and branching has been reported proximal to[14,16,17] and within the tarsal tunnel[14–18]. The ICN, which provides sensation to the anterior surface of the calcaneus, may also arise as a branch of the TN or LPN[16,18].

Clinical Symptoms

Patients complain of sensory plantar symptoms such as numbness, pain, burning, or cold or foreign-body sensations. When the MCN branches proximal to the tarsal tunnel, TTS symptoms are absent or weak in the heel area[6,10]. The MCN can branch within the tarsal tunnel and be entrapped[14–18].

Unlike carpal tunnel syndrome (CTS), TTS is usually not primarily caused by a pressure increase inside the tunnel, but rather by direct compression of the nerve, so even if the MCN runs through the tarsal tunnel, there are no symptoms in that area unless there is compression. The symptoms are similar to those of diabetic neuropathy, and this should be considered in the differential diagnosis. According to the prospective Rotterdam Diabetic Foot Study[19], TTS is common in diabetic patients and bilateral in 61% of this group. Therefore, TTS symptoms must be differentiated from diabetic neuropathy[19]. The rate of diabetic foot ulceration due to sensory impairment associated with diabetic neuropathy is significantly higher than in non-diabetic persons[19]. In patients with TTS, atrophy of innervated muscles, e.g., the abductor hallucis muscle, may not elicit subjective symptoms.

The symptoms elicited by idiopathic TTS tend to worsen with prolonged standing or walking, suggesting that ankle movement may play an etiologic role[4,9,11,13,20,21]. The tarsal tunnel narrows in forefoot pronation and in the presence of hindfoot varus. Flexor retinaculum tension in the tarsal tunnel increases because of ankle valgus[22,23]. Tajiri et al.[9] reported that nerve compression is worsened by the significantly increased distortion of the posterior tibial artery upon plantar flexion. TTS symptoms may be misdiagnosed as lumbar disease. Other studies[20,21] found that 4.8% of lumbar spine diseases are associated with TTS and that 5% of cases of failed back surgery syndrome involve TTS. Consequently, when plantar symptoms persist after spinal surgery, it is necessary to rule out TTS[4,11,13,20,21].

Diagnosis

Diagnosis of TTS is primarily based on clinical symptoms[5,12,13], although electrophysiological and imaging studies may be useful. Elicitation of a positive Tinel sign is the most straightforward clinical test and is useful for predicting surgical outcome, although it is difficult to reproduce and of low specificity. Therefore, the possibility of false-positive results must be considered[4,5,10,11].

Diagnostic imaging may be more helpful for diagnosing TTS than other nerve entrapments, e.g., CTS, which can be evaluated by both electrophysiological and diagnostic imaging studies[13]. In ultrasound examination, the cross-sectional area of the posterior TN in the central region of the tarsal tunnel was notably larger in TTS patients than in those without TTS. Furthermore, the ratio of the cross-sectional area of the posterior TN at the proximal and central regions of the tarsal tunnel yielded a sensitivity of 74% and a specificity of 100%[13,24]. MRI is useful for detecting trauma, space-occupying lesions, and foot deformities in symptomatic TTS; no imaging abnormalities were observed in 40% of patients with idiopathic TTS[25]. Kim et al.[8] documented results from 1.5-T MRI and intraoperative findings for 28 idiopathic TTS patients: although 1 patient presented with a ganglion, nerve compression was found in the other 27 patients. To visualize nerves in the tarsal tunnel, axial T2* fat-suppression images were useful. Although the nerve compression site could be visualized on MRI scans, other details responsible for nerve entrapment, e.g. varices, small vessel branches, and connective tissue, were not. Because the rate of false-positive finding on such scans is not known, a diagnosis of TTS should not be based on MRI findings alone. Rather, the consistency of MRI and neurological findings should be ascertained and evaluated.

In electrophysiological tests, increased terminal latencies of the MPN and LPN, along with a diminished conduction velocity and amplitude, are diagnostic markers. However, sole reliance on electrophysiological findings for a TTS diagnosis poses challenges due to the possibility of false-positive and false-negative results[3,5,10–12,21].

Lalevee et al.[26] studied 45 surgical TTS cases, and electrophysiological studies were positive in only 13 (28.9%). Oh et al.[27] observed prolonged motor latencies in 9 of 17 patients (52.9%) and abnormal sensory action potential responses in 90%. Galardi et al.[28] reported that the absence of sensory action potentials was the most frequent abnormality; 92% involved the lateral plantar nerve and 77% the medial plantar nerve. Only 21% of TTS limbs had prolonged distal motor latencies to the abductor hallucis. These results indicate that sensory action potentials are more sensitive for an electrophysiological diagnosis[10,27,28]. On the other hand, the results were false-positive for 10-43% of asymptomatic subjects, and this rate was higher in those older than 60 years[10,29–31]. Seidel et al.[32] reported that preoperative electrophysiological findings predicted postoperative symptom improvement in TTS patients. Other studies[33,34] found that negative electrophysiological findings did not correlate with surgical outcomes and cannot be used to rule out surgical indication; rather, electrophysiological tests ought to be utilized for supplementary evaluation[5,10,13]. Therefore, we usually measure sensory nerve conduction velocity at the tarsal tunnel and consider as a positive finding a terminal latency of the abductor hallucis muscle exceeding 5.8 ms and a difference in side-to-side amplitude of more than 50%[1,8,19,35,36].

Treatment

Conservative treatment options are available for patients with TTS. They include rest, physical therapy, massage, taping, stretching, and medications aimed at alleviating neuropathic pain. For TTS patients with symptoms that are exacerbated by walking, use of appropriate footwear or a sole plate may prove beneficial[13].

Less invasive surgery can be performed under local anesthesia. Tarsal tunnel decompression by simple opening of the flexor retinaculum is inadequate for treating TTS because affected nerves within the tarsal tunnel must be decompressed. The MPN and LPN are enclosed within the same compartment as the posterior tibial artery and vein (neurovascular band). Their release is important for nerve decompression; artery transposition may be useful[4,6,7]. Intraoperative observations indicated that the MPN was involved in 82% of cases and the LPN in 68% of cases[11]. Abductor fascia release may be required for distal decompression of the MPN and LPN in the tarsal tunnel[37]. Complications, mostly wound related, were reported in 6.2-12% of operated patients[38,39]. The tarsal tunnel is located near the ankle, and walking places a dynamic load on the site. Ankle joint movement may inhibit wound healing, so stretching forces around the wound must be considered. Shirokane et al.[35] placed zigzag skin incisions to reduce stretching and the dynamic load on the skin. This method is simple and convenient and reduces wound complications after TTS surgery.

Treatment Outcome

The outcome of surgery for TTS after a mean postoperative follow-up of at least 3 years was favorable in 44-96% of cases, although some symptoms may persist[4,10,11,26,28]. According to Lalevee et al.[26], 57.8% of operated patients who were followed for a mean of 3.6 years reported symptom improvement. The outcome was favorable in 93.3% of patients with space-occupying compressive structures and poor in patients with venous dilations (53.3%) and idiopathic (26.7%) TTS.

According to outcomes based on the numerical rating scale (NRS) after nerve decompression with posterior tibial artery transposition, the symptoms of operated patients improved from 6.6 ± 1.5 to 2.7 ± 1.5; the NRS for surgical satisfaction was 6.6 ± 2.1.

Preoperative quality of life (QOL) was significantly lower for TTS patients than for CTS patients[1], and postoperative patient satisfaction was significantly greater for CTS patients than for TTS patients, although QOL was significantly improved in both groups. Postoperative outcomes were better for patients with compressive space-occupying lesions[40,41]. Treatment results for patients with idiopathic TTS are affected by factors such as plantar fasciitis, age, symptom duration, Tinel sign positivity, ankle disorders, electrophysiological findings, marked hindfoot valgus/varus, bone-nerve contact, and the surgical procedure[10,11,13,41]. The ankle joint position affects the tarsal tunnel pressure: it decreases in the neutral position, and ankle joint inversion or eversion increases pressure[42]. In patients with marked hindfoot valgus/varus, neurolysis may not achieve sufficient nerve decompression.

Recurrences and failed outcomes after TTS surgery may be due to several factors, such as inadequate decompression, lack of anatomic knowledge, nerve variations, scarring, nerve damage, persistent nerve hypersensitivity, and preexisting intrinsic nerve damage[43,44]. In the case of insufficient decompression, adequate and thorough decompression should be completed by revision surgery[43–45]. In patients with external scarring or traction neuritis, barrier materials may be used after neurolysis[43–45]. However, because the surgical outcomes of revision surgery can be highly unsatisfactory, it is better to prevent rather than

treat failed TTS surgery[45].

Skalley et al.[34] performed revision surgery for 12 TTS patients (13 feet) after a mean interval of 3.5 years after the initial operation. They found that TN scarring and insufficient distal release were the main problems. Adequate distal release resulted in good surgical outcomes. The patients' clinical history and physical examination findings were more helpful than electrodiagnostic studies for determining the extent and location of TN irritation.

TTS due to Ganglia

Ganglia are typical space-occupying lesions that can elicit TTS and were involved in up to 8% of TTS cases[46]. The talocalcaneal joint is the most common origin of ganglia in TTS and, among ganglion-associated TTS cases, 63-75% involved the MPN area[46]. According to Koketsu et al.[46], ganglia were palpable through the skin in only 1 of 8 patients; the other 7 were identified on preoperative MRI scans or intraoperatively. Because the symptoms of TTS due to ganglia are similar to those reported in idiopathic TTS, differentiation can be difficult, but ultrasound and MRI findings are helpful[5,10,46,47]. Ganglia tend to be hypointense on T1-weighted images and hyperintense on T2-weighted and T2* fat-suppression MRI scans[46,48].

Ganglion aspiration may be useful. However, ganglia implicated in TTS tend to be located on the ventral side of nerves and vessels, and their aspiration raises the risk of iatrogenic nerve injury[46–48]. Surgical outcome is affected by, for example, patient selection, symptom severity and duration, fibrosis and adhesion around the nerve, and surgical technique[10,13,46,48]. Nagaoka and Satou[47] recommended early surgical intervention because only 1 of 30 ganglia spontaneously resolved. To avoid incomplete ganglion excision due to bleeding and intraoperative pain, surgery under general anesthesia should be performed, especially for patients with large ganglia[46,49]. While symptom recurrence attributable to ganglia in the tarsal tunnel is uncommon, recurrent ganglia may be less likely to elicit symptoms[46,47,49].

Accessory Muscle and TTS

An accessory muscle in the tarsal tunnel can lead to TTS. Between 11-16% of cases of surgically treated TTS were associated with accessory muscles, and most such cases involved the flexor digitorum accessorius longus muscle (ALM)[26,41,50,51]. It was identified in 2-8% of legs but did not result in TTS in 14 legs. Consequently, its presence may not be a risk factor for TTS[50–53]. TTS may be attributable not only to simple nerve compression by an accessory muscle, but also to muscle edema and hypertrophy due to trauma or strenuous exercise[51]. Nerve compression by an accessory muscle may be exacerbated in plantar flexion of the ankle. MRI studies performed in the rest position may reveal only slight compression but not the actual underlying pathophysiology[50].

While nerve-compressing accessory muscles are usually resected, it can be difficult to remove the flexor digitorum ALM because it originates at the proximal third of the leg[26,41,51–53]. Even without the resection of all accessory muscles, good surgical outcomes were achieved by nerve decompression. Before muscle resection, dynamic compression by muscle contraction must be evaluated intraoperatively[50,54]. For successful treatment of TTS, not only accessory muscles but also the presence of a concomitant tortuous artery and valgus or bone-nerve contact must be ruled out[4,7–9,41]. Prolonged neuropathy from a congenital anomaly may result in an unsatisfactory outcome for TTS surgery[50,51,53].

Conclusion

Although not uncommon, TTS may be overlooked in clinical practice. Patients report plantar sensory disturbances, and a diagnosis of TTS is primarily based on clinical symptoms. While the results of electrophysiological and imaging studies may be diagnostic, such tests can yield false-positive and false-negative findings. TTS can be surgically treated by less invasive procedures with the patient under local anesthesia. However, factors involved in TTS, e.g., its natural history, long-term surgical outcomes, recurrence rates, false-positive and false-negative MRI findings, and the role of dynamic MRI studies in its diagnosis, remain to be clarified, and novel diagnostic tools are needed. Advances in treatment outcomes for TTS will require additional study.

Conflict of Interest: The authors declare no conflicts of interest.

References

1. Kokubo R, Kim K, Isu T, Morimoto D, Morita A. Patient satisfaction with surgery for tarsal tunnel- and carpal tunnel syndrome - comparative study. Neurol Med Chir (Tokyo). 2023Mar 15;63(3):116–21.
2. Kokubo R, Kim K, Isu T, Morimoto D, Iwamoto N, Morita A. Quality of life effects of pain from para lumbar- and lower extremity entrapment syndrome and carpal tunnel syndrome and comparison of the effectiveness of surgery. Acta Neurochir (Wien). 2020 Jun;162(6): 1431–7.
3. Vij N, Kaley HN, Robinson CL, et al. Clinical results following conservative management of tarsal tunnel syn-

drome compared with surgical treatment: a systematic review. Orthop Rev (Pavia). 2022 Sep 5;14(3):37539.
4. Kim K, Isu T, Morimoto D, et al. Neurovascular bundle decompression without excessive dissection for tarsal tunnel syndrome. Neurol Med Chir (Tokyo). 2014;54(11):901–6.
5. McSweeney SC, Cichero M. Tarsal tunnel syndrome - a narrative literature review. Foot (Edinb). 2015 Dec;25(4): 244–50.
6. Kohno M, Takahashi H, Segawa H, Sano K. Neurovascular decompression for idiopathic tarsal tunnel syndrome: technical note. J Neurol Neurosurg Psychiatry. 2000 Jul;69 (1):87–90.
7. Fujihara F, Isu T, Kim K, et al. Artery transposition using indocyanine green for tarsal tunnel decompression. World Neurosurg. 2020 Sep;141:142–8.
8. Kim K, Kokubo R, Isu T, et al. Magnetic resonance imaging findings in patients with tarsal tunnel syndrome. Neurol Med Chir (Tokyo). 2022 Dec 15;62(12):552–8.
9. Tajiri T, Kim K, Isu T, et al. Impact of dynamic change of meandering of parallel artery to the idiopathic tarsal tunnel syndrome. Neurol Med Chir (Tokyo). 2023 Apr 15;63 (4):165–71.
10. Ahmad M, Tsang K, Mackenney PJ, Adedapo AO. Tarsal tunnel syndrome: a literature review. Foot Ankle Surg. 2012 Sep;18(3):149–52.
11. Reichert P, Zimmer K, Wnukiewicz W, Kulinski S, Mazurek P, Gosk J. Results of surgical treatment of tarsal tunnel syndrome. Foot Ankle Surg. 2015 Mar;21(1):26–9.
12. Samarawickrama D, Therimadasamy AK, Chan YC, Vijayan J, Wilder-Smith EP. Nerve ultrasound in electrophysiologically verified tarsal tunnel syndrome. Muscle Nerve. 2016 Jun;53(6):906–12.
13. Doneddu PE, Coraci D, Loreti C, Piccinini G, Padua L. Tarsal tunnel syndrome: still more opinions than evidence. Status of the art. Neurol Sci. 2017 Oct;38(10):1735–9.
14. Warchol L, Walocha JA, Mizia E, Bonczar M, Liszka H, Koziej M. Ultrasound-guided topographic anatomy of the medial calcaneal branches of the tibial nerve. Folia Morphol (Warsz). 2021;80(2):267–74.
15. Joshi SS, Joshi SD, Athavale SA. Anatomy of tarsal tunnel and its applied significance. J Anat Soc India. 2006;55(1): 52–6.
16. Priya A, Ghosh SK, Walocha JA, Tubbs RS, Iwanaga J. Variations in the branching pattern of tibial nerve in foot: a review of literature and relevant clinical anatomy. Folia Morphol (Warsz). 2023;82(2):231–41.
17. Dellon AL, Mackinnon SE. Tibial nerve branching in the tarsal tunnel. Arch Neurol. 1984 Jun;41(6):645–6.
18. Govsa F, Bilge O, Ozer MA. Variations in the origin of the medial and inferior calcaneal nerves. Arch Orthop Trauma Surg. 2006 Jan;126(1):6–14.
19. Rinkel WD, Castro Cabezas M, Birnie E, Coert JH. The natural history of tarsal tunnel syndrome in diabetic subjects. J Plast Reconstr Aesthet Surg. 2020 Aug;73(8):1482–9.
20. Matsumoto J, Isu T, Kim K, et al. Impact of additional treatment of paralumbar spine and peripheral nerve diseases after lumbar spine surgery. World Neurosurg. 2018 Apr;112:e778–82.
21. Zheng C, Zhu Y, Jiang J, et al. The prevalence of tarsal tunnel syndrome in patients with lumbosacral radiculopathy. Eur Spine J. 2016 Mar;25(3):895–905.
22. Radin EL. Tarsal tunnel syndrome. Clin Orthop Relat Res. 1983 Dec;181:167–70.
23. DiStefano V, Sack JT, Whittaker R, Nixon JE. Tarsal-tunnel syndrome. Review of the literature and two case reports. Clin Orthop Relat Res. 1972;88:76–9.
24. Fantino O, Bouysset M, Pialat JB. Can the axial cross-sectional area of the tibial nerve be used to diagnose tarsal tunnel syndrome? An ultrasonography study. Orthop Traumatol Surg Res. 2021 Oct;107(6):102630.
25. Donovan A, Rosenberg ZS, Cavalcanti CF. MR imaging of entrapment neuropathies of the lower extremity. Part 2. The knee, leg, ankle, and foot. Radiographics. 2010 Jul-Aug;30(4):1001–19.
26. Lalevee M, Coillard JY, Gauthe R, et al. Tarsal tunnel syndrome: outcome according to etiology. J Foot Ankle Surg. 2022May-Jun;61(3):583–9.
27. Oh SJ, Sarala PK, Kuba T, Elmore RS. Tarsal tunnel syndrome: electrophysiological study. Ann Neurol. 1979 Apr; 5(4):327–30.
28. Galardi G, Amadio S, Maderna L, et al. Electrophysiological studies in tarsal tunnel syndrome. Diagnostic reliability of motor distal latency, mixed nerve and sensory nerve conduction studies. Am J Phys Med Rehabil. 1994 Jun;73(3):193–8.
29. Gatens PF, Saeed MA. Electromyographic findings in the intrinsic muscles of normal feet. Arch Phys Med Rehabil. 1982 Jul;63(7):317–8.
30. Falck B, Alaranta H. Fibrillation potentials, positive sharp waves and fasciculation in the intrinsic muscles of the foot in healthy subjects. J Neurol Neurosurg Psychiatry. 1983 Jul;46(7):681–3.
31. Boon AJ, Harper CM. Needle EMG of abductor hallucis and peroneus tertius in normal subjects. Muscle Nerve. 2003 Jun;27(6):752–6.
32. Seidel GK, Jamal SA, Weidert E, et al. Predictive outcome modeling of preoperative clinical symptoms and electrodiagnostic data in tarsal tunnel surgery. J Brachial Plex Peripher Nerve Inj. 2021 Jul 27;16(1):e37–45.
33. Bailie DS, Kelikian AS. Tarsal tunnel syndrome: diagnosis, surgical technique, and functional outcome. Foot Ankle Int. 1998 Feb;19(2):65–72.
34. Skalley TC, Schon LC, Hinton RY, Myerson MS. Clinical results following revision tibial nerve release. Foot Ankle Int. 1994 Jul;15(7):360–7.
35. Shirokane K, Kim K, Akimoto M, et al. Zig-zag skin incision for treatment of tarsal tunnel syndrome. J Nippon Med Sch. 2024;91(4):357–61.
36. Tawfik EA, El Zohiery AK, Abouelela AA. Proposed sonographic criteria for the diagnosis of idiopathic tarsal tunnel syndrome. Arch Phys Med Rehabil. 2016 Jul;97(7): 1093–9.
37. Heimkes B, Posel P, Stotz S, Wolf K. The proximal and distal tarsal tunnel syndromes. Int Orthop. 1987;11(3):193–6.
38. Wood WA, Wood MA. Decompression of peripheral nerves for diabetic neuropathy in the lower extremity. J Foot Ankle Surg. 2003 Sep-Oct;42(5):268–75.
39. Kim K, Isu T, Morimoto D, Kokubo R, Fujihara F, Morita A. Perioperative complications and adverse events after surgery for peripheral nerve- and para-lumbar spine diseases. Neurol Med Chir (Tokyo). 2022 Feb 15;62(2):75–9.
40. Pfeiffer WH, Cracchiolo A 3rd. Clinical results after tarsal tunnel decompression. J Bone Joint Surg Am. 1994 Aug;76 (8):1222–30.
41. Bouysset M, Denarie D, Coillard JY, et al. Predictive factors of effective tibial nerve release in tarsal tunnel syn-

drome. Foot Ankle Surg. 2022 Jul;28(5):610–5.
42. Bracilovic A, Nihal A, Houston VL, Beattie AC, Rosenberg ZS, Trepman E. Effect of foot and ankle position on tarsal tunnel compartment volume. Foot Ankle Int. 2006 Jun;27(6):431–7.
43. Gould JS. Recurrent tarsal tunnel syndrome. Foot Ankle Clin. 2014 Sep;19(3):451–67.
44. Gould JS. The failed tarsal tunnel release. Foot Ankle Clin. 2011 Jun;16(2):287–93.
45. Raikin SM, Minnich JM. Failed tarsal tunnel syndrome surgery. Foot Ankle Clin. 2003 Mar;8(1):159–74.
46. Koketsu K, Kim K, Tajiri T, et al. Ganglia-induced tarsal tunnel syndrome. J Nippon Med Sch. 2024;91(1):114–8.
47. Nagaoka M, Satou K. Tarsal tunnel syndrome caused by ganglia. J Bone Joint Surg Br. 1999 Jul;81(4):607–10.
48. Harbaugh KS, Tiel RL, Kline DG. Ganglion cyst involvement of peripheral nerves. J Neurosurg. 1997 Sep;87(3): 403–8.
49. Ahn JH, Choy WS, Kim HY. Operative treatment for ganglion cysts of the foot and ankle. J Foot Ankle Surg. 2010 Sep-Oct;49(5):442–5.
50. Miwa K, Kim K, Kokubo R, Dan H, Koketsu K, Murai Y. A patient with tarsal tunnel syndrome associated with the flexor digitorum accessorius longus muscle. NMC Case Rep J. 2024 May 17;11:125–9.
51. Deleu PA, Bevernage BD, Birch I, Maldague P, Gombault V, Leemrijse T. Anatomical characteristics of the flexor digitorum accessorius longus muscle and their relevance to tarsal tunnel syndrome. A systematic review. J Am Podiatr Med Assoc. 2015 Jul;105(4):344–55.
52. Cheung YY, Rosenberg ZS, Colon E, Jahss M. MR imaging of flexor digitorum accessorius longus. Skeletal Radiol. 1999 Mar;28(3):130–7.
53. Wittmayer BC, Freed L. Diagnosis and surgical management of flexor digitorum accessorius longus-induced tarsal tunnel syndrome. J Foot Ankle Surg. 2007 Nov-Dec;46 (6):484–7.
54. Ettehadi H, Saragas NP, Ferrao P. A rare case of flexor digitorum accessorius longus muscle presenting as tarsal tunnel syndrome. Arch Bone Jt Surg. 2022 Jan;10(1):112–6.

(Received, October 29, 2024)
(Accepted, December 20, 2024)