Tarsal Tunnel Syndrome Following a Total Knee Replacement

A Case Report

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Tarsal tunnel syndrome (TTS) is defined as an entrapment neuropathy from compression of the tibial nerve and/or distal terminal nerves in the tarsal tunnel, the medial foot, and/or ankle. While the incidence of TTS following a total knee replacement (TKR) could not be found, we present the case of a 78-year-old white female who developed symptoms of TTS 2 weeks after a left knee replacement. The history, examination, imaging, and electrodiagnostic (EDX) studies were consistent with the diagnosis of left-sided TTS. After a course of conservative care failed to alleviate her symptoms, the patient underwent nerve decompression surgery for the affected nerves. Her pain score prior to the surgery was 5 out of 10 when walking, and 10 out of 10 at night. Immediately after surgery, her pain at its worst was 3 out of 10, and soon thereafter she reported no pain whatsoever (0 out of 10). All TTS-related symptoms subsequently resolved. This case presents an unusual extrinsic cause of TTS, highlights the need for a thorough history when diagnosing TTS, and contributes to the scientific community's knowledge of knee complications and possible etiologies of TTS. (J Am Podiatr Med Assoc 115(2), 2025; doi:10.7547/24-021)

Tarsal tunnel syndrome (TTS) has been described as an entrapment of the tibial nerve as it courses beneath the laciniate ligament[1] with additional compression from the abductor hallucis muscle[2] and/or from the fascia of the lower leg.[3,4] Persons with TTS may experience burning, numbness, paresthesia, cramping, a cold or swollen sensation, or a sandpaper-like feeling on the sole of the foot and/or toes.[5-7] Causes of TTS include abnormal biomechanics, such as pronation; variations in anatomy, such as accessory muscles, tumors, varicosities, and coalitions; as well as trauma or precipitation by systemic disease.[5,6,8-15]

Cases of a proximal-located issue causing the development of TTS are uncommon.[16-18] The only reports that we could find where TTS occurred due to a proximal issue involved emboli occluding the femoral artery, fracture of the proximal tibia and fibula, lumbar disc disease, and deep vein thrombosis.[17,18] To our knowledge, this is the first known case of TTS occurring subsequent to a total knee replacement (TKR). The patient provided consent to be included in this report.

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Case Report

A 78-year-old active white female presented to the private practice of Dr. Nirenberg describing a history of left foot pain with symptoms consistent with TTS during the previous 6 months. These symptoms included numbness and tingling in the foot's arch, ball, and toes. The patient also described the pain as stabbing, burning, shooting, and as a feeling of "rocks" in her arch. She noted that she had lost the ability to move or spread apart her toes since the TKR, and that her toes were weaker than on her right foot. The severity of the pain varied between 5 out of 10 when walking to 10 out of 10 at night. The patient said that the issues began 2 weeks after she underwent the TKR procedure on her left knee. There were no complications with the procedure, and she reported no history of knee injury.

Prior to presenting to the podiatric clinic, the orthopedic surgeon who performed the knee surgery referred her to a pain management physician, where she was treated with pain medication injections to her back, a night splint, and physical therapy for plantar fasciitis. The pain management physician

also prescribed gabapentin, but this medication had recently been discontinued due to the drug's side effects.

The patient's medical history was significant for hypertension. Her body mass index was 25.5 kg/m^2, and she had previously undergone a TKR on her right knee. Her daily prescriptions were 12.5 mg of hydrochlorothiazide and 40 mg of simvastatin; she also took 1200 mg of fish oil daily. She had no allergies and reported no back or calf pain.

Physical examination of the patient's feet and ankles found normal circulation, integument, and range of motion at the ankle and subtalar joints. Also, mild edema was noted on the left foot and ankle compared to the right. Musculoskeletal examination found normal ankle strength. The toes on the left foot were weaker than the right and also were limited in their ability to spread apart. There was no calf pain on palpation, and there was a negative Homan's sign. Her gait was antalgic, placing more weight on the contralateral foot. Pain was elicited on palpation of the plantar fascia at the area of the medial tubercle of the calcaneus consistent with plantar fasciitis. Neurologic examination revealed she was alert and oriented and had normal reflexes at the knees and right ankle; however, reflex at the left ankle could not be elicited. Vibration sensation to the left toes were reduced by 30% compared to those on her right. There was a negative Babinski sign, and no provocative sign could be elicited over the area of the soleal sling. There was also a positive Tinel sign, provocative sign, dorsiflexion-eversion test, and triple compression test at the left tarsal tunnel. These were negative on the right side.

Diagnostic testing included radiographs of the left knee, along with magnetic resonance imaging (MRI) and EDX testing. The radiographs and MRI were negative, which assisted in ruling out other causative factors for the symptoms. Electrodiagnostic testing of both extremities was performed by a clinical neurophysiologist (R.P.S.) incorporating both conventional and also short-segment testing.[19-25] The examination found tibial nerve motor conduction studies with borderline distal latencies of 6.2 msec and low normal evoked responses of 3 mV. In addition, the test showed that detailed stimulation of the nerve at multiple levels (or segments) above the laciniate ligament revealed focal slowing of 10 m/sec at 14 cm from the abductor hallucis muscle (the "pick up" muscle) and 20 m/sec at 12 cm and 28.5 m/sec at 10 cm from the muscle. Sensory conduction studies had normal latencies and evoked responses for the sural and superficial peroneal nerves. The medial and lateral plantar responses were absent. An electromyogram showed no alterations at rest or under voluntary control. As such, the findings were consistent with TTS and entrapment of the tibial nerve at, and also above, the level of the ankle, at the high tarsal tunnel.

After a course of further conservative care, including custom-made foot orthotics, additional physical therapy sessions, and treatment with pregabalin, the patient continued to experience symptoms of TTS and plantar fasciitis. The patient's symptoms and clinical examination suggested that the primary issue was TTS, with plantar fasciitis being a minor contributor to her pain. A nerve block of the tibial nerve was administered, and the patient experienced temporary relief. Soon thereafter, and approximately 1 year from the date of the TKR, the patient underwent surgery for decompression of the tibial nerve, the related nerves (the medial and lateral plantar nerves and the calcaneal nerve), as well as release of the plantar fascia through the same incision. The surgery was performed under loupe magnification; the technique and routine postoperative protocol is documented in the literature.[26-28]

At the initial postoperative visit, the patient related her pain was a 3 out of 10 at most, and roughly 14 days later, her pain was a 0 out of 10. Her symptoms resolved, and the strength in her toes improved. The patient was monitored for 8 months with continued improvement, and she eventually related relief from complete pain, a normal gait, and no other complaints.

Discussion

We believe this to be the first case showing the development of TTS after a TKR. Although the onset of TTS after the TKR could be a coincidence, factors related to the TKR could be causative. The TKR procedure did require an adductor nerve block; however, this block was excluded as a contributing factor because the patient's symptoms began 2 weeks after the surgery. Had the block been a component in the TTS, the symptoms would have presented immediately after the procedure.

Though TTS after TKR appears to be a rare occurrence, common fibular nerve (CFN) entrapment and injury is well documented with an incidence from 0.3% to 4%.[29,30] Proposed risk factors and mechanisms of injury for CFN entrapment include stretching of the nerve, disruption of the blood supply due to prolonged tourniquet use, extensive blood loss and hypotension, and hematoma formation that compresses the nerve.

In the postoperative period, compression by a continuous passive motion machine has been put forth.[30]

Whereas the mechanism for the development of TTS after this patient's TKR is unclear, a known complication of TKR is that the length of the surgical limb may be longer or shorter after the procedure.[31] On this, Tipton et al[32] found 59% of TKR patients experienced an increase in length. In our case, we speculate that the surgical limb could have been inadvertently lengthened causing tension (or traction) on the tibial nerve, which contributed to the onset of TTS. Although compression has traditionally been seen as the main cause of TTS, tension on the tibial nerve has also been recognized.[33-36] However, tension alone may not be the only component to development of this patient's TTS. In this regard, Raikin and Minnich[37] have noted that surgical decompression for presumed TTS fails in cases of hip replacement or fractures of the femur or pelvis that increase tension on the tibial nerve (eg, stretch the nerve) and generate symptoms of TTS—without the actual presence of TSS. Had tension on the nerve occurred in this case (presumably from the limb lengthening as a consequence of TKR), it is likely that there was an additional factor or factors, besides the increase in tension, to cause the patient's TTS.

Augustijn and Vanneste[16] speculated that a medical problem that injures a proximal nerve may result in "decompensation" of a pre-existing asymptomatic TTS due to a double-crush mechanism. Indeed, asymptomatic TTS has been documented,[27] as has the existence of a double-crush mechanism in TTS.[17,38] This patient's TTS may have been asymptomatic, and the combination of increased tension as a result of the TKR, with existing subclinical compression in the tarsal tunnel may have acted synergistically to cause the TTS to worsen to the point of becoming symptomatic.

Alternatively, TKR is known to alter gait,[39] which may also affect the foot's biomechanicsand contribute to the development of TTS.[6,40] One known change to the foot for a significant percentage of individuals who undergo TKR is an increase in the external rotation of the tibia.[41] External rotation of the tibia would increase subtalar joint pronation,[42] which is a known factor in the development of TTS.[6,40,43] This gait alteration may have been responsible in whole or part for the patient's TTS.

Further factors that may have been contributory to TTS in this case may have been the surgical positioning of the patient during the TKR or direct stretching of the tibial nerve during movement of the patient.

Though the occurrence of TTS developing after TKR appears to be uncommon, this case report highlights the importance of considering the patient's history and in particular, their surgical history when evaluating their foot issues. It further suggests to surgeons performing TKR to be cognizant of the fact that TTS may be a remote complication. Importantly, while we have put forth possible mechanisms for the development of the patient's TTS, a definitive cause cannot be established in a lone case study.

Conclusions

To our knowledge, this is the first report that demonstrates the development of TTS following a TKR. The patient in question developed TTS 2 weeks after the procedure and later underwent nerve decompression surgery to alleviate the problem. We hope this case increases awareness among physicians performing TKR and those who treat TTS. Moreover, we hope this report promotes increased inquiry into TTS, deepening our understanding of the factors that cause the syndrome and that ultimately, leads to new methods of prevention and treatment.

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