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PRS GLOBAL OPEN

CASE REPORT

Pediatric/Craniofacial

# Idiopathic Pediatric Tibial Nerve Palsy

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**Summary:** Tibial nerve entrapment is uncommon in the pediatric population, and presents diagnostic and treatment challenges. We present the unusual case of a 3-year-old male child with progressive lower leg atrophy of an unknown etiology. Preoperative electrodiagnostic testing and magnetic resonance imaging suggested proximal tibial neuropathy. Surgical exploration showed compression of the tibial nerve at the inferior fascial edge of the long head of the biceps femoris and at the soleal sling. Release and external neurolysis led to improvement of distal leg motor function. *(Plast Reconstr Surg Glob Open 2021;9:e3484; doi: 10.1097/GOX.0000000000003484; Published online 15 March 2021.)*

Symptomatic tibial nerve entrapment is rare in children.[1] Entrapment of the tibial nerve can occur in the tarsal tunnel beneath the flexor retinaculum or, less commonly, in the tendinous arch at the origin of the soleus muscle, the so-called soleal sling.[1] Manifestations of proximal tibial nerve entrapment include pain and paresthesias in the foot, leg, or popliteal fossa, and variable motor weakness in foot plantarflexion and toe flexion and abduction.[2,3] Reports are limited, but symptoms tend to improve after surgical decompression.[1–3] We present the evaluation and successful surgical management of a toddler who presented with a progressive lower leg atrophy caused by proximal tibial nerve compression.

## CASE REPORT

A 3-year-old male child was referred for progressive tibial neuropathy. His parents reported that his motor development was unremarkable during his first year of life and that he began walking at the age of 13 months with no visible limp or difficulty. At the age of 20 months, his parents noted that he was ambulating on the outside of his left foot and, upon further evaluation, that his left lower leg was thinner that the contralateral leg. His left foot was also visibly shorter in length (Fig. 1) and cooler to touch than the right side foot. He denied pain. The patient was evaluated at several centers, and he was eventually referred to our clinic.

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*Received for publication December 12, 2020; accepted January 22, 2021.*



DOI: 10.1097/GOX.0000000000003484

Physical examination at the age of 30 months demonstrated atrophy of the calf muscles of the left leg, especially the posterior compartment, and a high longitudinal arch of the foot. There was no toe clawing. The left foot was smaller in length and overall size when compared with the right side. The left leg had very weak ankle plantar flexion and inversion, intact ankle dorsiflexion and eversion, and normal sensation to pinprick in the distributions of the tibial, common peroneal, superficial peroneal, sural, and saphenous nerves. Distal pulses were intact, as was capillary refill, but the left foot was notably cooler to touch than the contralateral side.

Magnetic resonance imaging (MRI) of the left leg revealed atrophy and fatty infiltration of the deep, and superficial posterior compartments musculature was consistent with tibial nerve compression distal to the bifurcation of the sciatic nerve but proximal to the branches to the gastrocnemius and the sural nerve. Visualization of the soleal sling was limited. Electromyography (EMG) demonstrated fibrillation potentials in the gastrocnemius, and nerve conduction studies showed reduced motor amplitude in the abductor hallucis brevis, consistent with chronic isolated tibial nerve denervation.

Because the neuropathy appeared progressive and incomplete, surgical decompression was advised to prevent further atrophy and undergrowth of the foot. The patient was positioned prone, and the left tibial nerve was exposed through a longitudinal incision spanning the left popliteal fossa. The tibial nerve was identified and traced proximally to its bifurcation from the sciatic nerve. The common peroneal nerve appeared normal, whereas the tibial nerve was narrowed and pale. Proximally, the nerve was found to be constricted by anomalous fascial bands spanning from the medial margin of the biceps femoris to the lateral margin of the semitendinosus (Fig. 2). After release of these fibrous bands and an external neurolysis, there was significant visual improvement in the appearance of the nerve. Dissection was then carried distally to the tendinous arch of the soleus (Fig. 3), where the nerve

**Disclosure:** *The authors have no financial interest to declare in relation to the content of this article.*

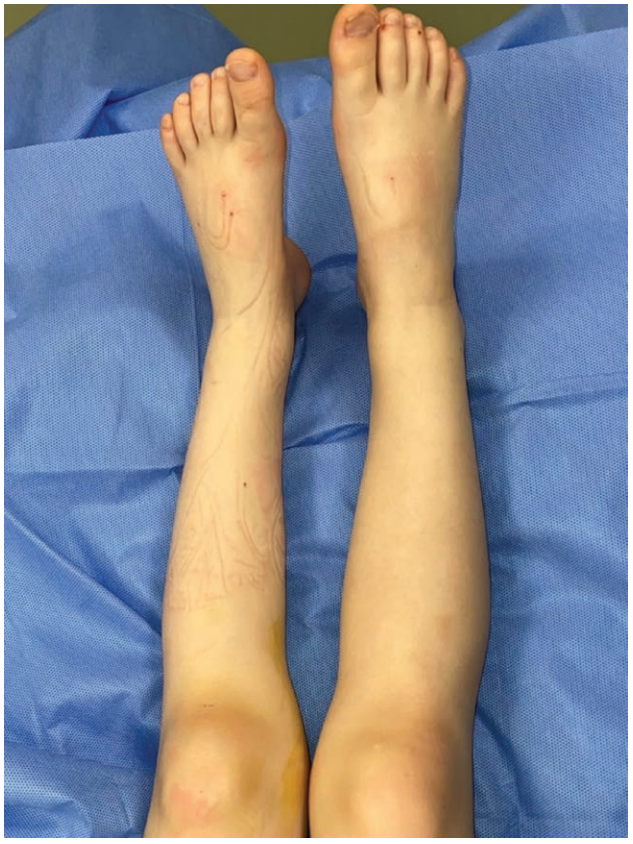

**Fig. 1.** Decrease in the length and overall size of the left leg when compared with that of the right leg.

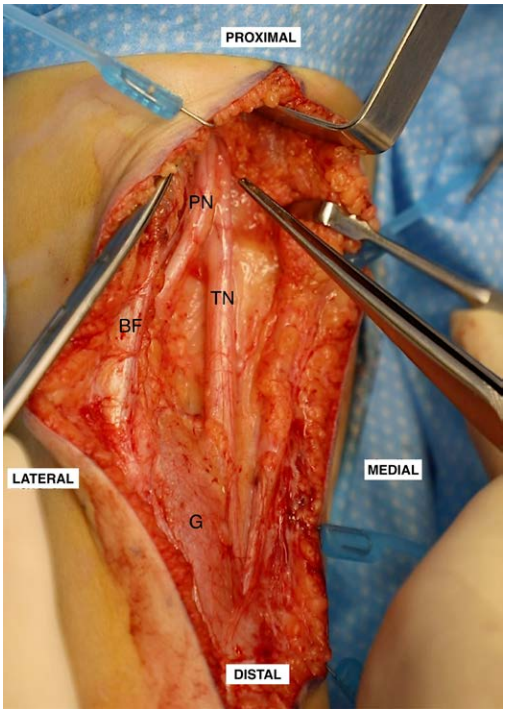


**Fig. 2.** Released proximal compression point beneath fascial band spanning from biceps femoris to semitendinosus. BF, biceps femoris; G, gastrocnemius muscle; PN, peroneal nerve; TN, tibial nerve.

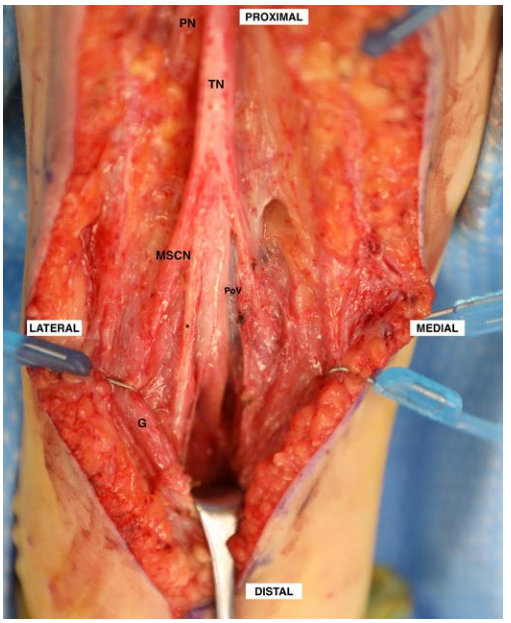


**Fig. 3.** Tibial nerve released; distal dissection to the tendinous arch of the soleus muscle.MSC, medial sural cutaneous nerve; PoV, popliteal vein; PN, peroneal nerve; S, soleus muscle; TN, tibial nerve. *Motor nerves to soleus and gastrocnemius.

was visibly compressed under the fascia of the soleus sling. The sling was released and an external neurolysis was performed. Intraoperative neurophysiological monitoring revealed an increase in EMG responses in the gastrocnemius muscle and in toe flexion. The patient was casted for 3 weeks postoperatively.

At 6-months follow up, the patient demonstrates improved strength in ankle plantar and toe flexion (4-/5). Ankle inversion and plantar flexion have also improved, although he continues to walk on the outside of his foot. Dorsiflexion and eversion remained in full strength, and he has no pain or paresthesia.

## DISCUSSION

Tibial nerve entrapment is rare in the pediatric population. Lower extremity mononeuropathies in children have been described in the setting of traumatic or iatrogenic injury involving the peroneal and sciatic nerve,[4] but reports of idiopathic lower extremity peripheral nerve compression in the pediatric population are exceedingly rare, with only a few isolated cases in the literature.[5–7] Although the soleus arch is a well-known compression point of the tibial nerve in the adult population,[2,3,8] this has not, to our knowledge, been reported as cause of proximal tibial nerve compression in a pediatric patient. Moreover, our patient had an additional area of compression (beneath anomalous fascial bands between the biceps femoris and semitendinosus), which also makes this presentation unique (Fig. 4). We speculate that the progressive undergrowth of the left foot was caused by an early loss of neurotropic input, while the leg growth was spared by continued neural inputs from the peroneal nerve. This may also have accounted for the

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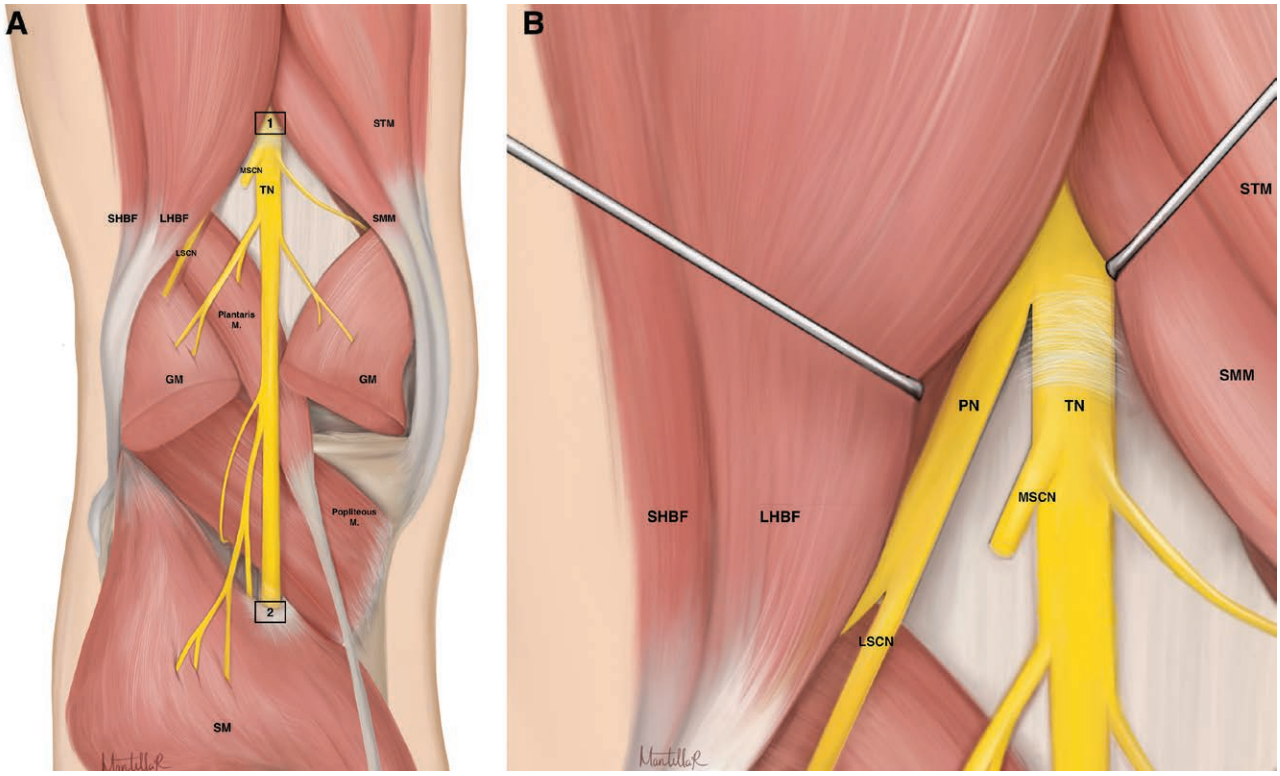


**Fig. 4.** A diagram illustrating the tibial nerve with the 2 entrapment sites. (A), Proximal [1] with anomalous bands spanning from the medial margin of the biceps femoris to the lateral margin of the semitendinosus, and distal [2] beneath the tendinous arch of the soleus muscle. A close-up diagram (B) illustrating the sciatic nerve bifurcation to peroneal and tibial nerve, with anomalous bands entrapping the tibial nerve. G, gastrocnemius; LHBF, long head biceps femoris; LSCN, lateral sural cutaneous nerve; MSCN, medial sural cutaneous nerve; PN, peroneal nerve; SHBF, short head biceps femoris; SM, soleus muscle; SMM, semimembranosus muscle; STM, semitendinosus muscle; TN, tibial nerve. Illustration by Esperanza Mantilla-Rivas, MD.

coolness of the left foot upon touch, although the popliteal artery and vein also passed under this structure and may have experienced more subclinical compression as well. The "double crush" insult from these 2 areas of compression may account for the rapid deterioration of function observed by the child's parents.

Although tibial nerve entrapment in the soleal sling in adults often manifests as pain and numbness in the popliteal region or upper calf,[2,3] it is interesting that our patient had no pain or obvious loss of sensation in the left lower extremity. Given the child's age, we did not attempt to quantitate sensation using more sophisticated tools. It is probable that he did have concurrent sensory loss in his foot or leg, but was unable to appreciate or articulate this due to his very young age.

It is important to consider a wide differential diagnosis when confronted with a pediatric patient with leg size discrepancy, weakness, and altered gait. Traumatic injury or fracture should be considered and ruled out with physical examination and radiographs. Systemic syndromes such as muscular dystrophies, Beckwith-Wiedemann syndrome, Mafucci syndrome, CLOVES syndrome, and other genetic abnormalities should be considered and ruled out based on the absence of other accompanying signs and features characteristic of those disorders. Other neurogenic etiologies should be considered as well, such as neoplasm (neurofibroma or Schwannoma, which may show similar EMG/NCS findings but would be visible on magnetic resonance imaging), radiculopathy or polyneuropathy (which would differ from compression mononeuropathy on EMG/NCS), and central nervous system disorders, which would show upper motor neuron signs on examination (eg, upgoing Babinski reflex). A thorough history and physical examination, accompanied by magnetic resonance imaging and EMG/NCS, allowed us to elucidate the causative compression mononeuropathy and treat it appropriately.

## CONCLUSIONS

The etiology, symptoms, and sites of tibial nerve compression in children may differ from those reported in the literature for adults, specifically the lack of pain or paresthesias. The possibility of entrapment at more than 1 location is important when planning surgical decompression, and release must be continued until a normal-appearing nerve is found in both directions.

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