Peroneus Longus Transfer for Drop Foot in Hansen Disease

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KEYWORDS

• Peroneus longus transfer • Drop foot • Hansen disease

KEY POINTS

- Nerve injury is a central feature of the pathogenesis of leprosy because of the unique tendency of *Mycobacterium leprae* to invade Schwann cells and the peripheral nervous system, causing a mononeuritis multiplex of immunologic origin that results in autonomic, sensory, and motor neuropathy.
- Early diagnosis of leprosy, along with early detection and treatment of neuropathy, are the means to prevent permanent primary impairments.[1]
- Investigators state that up to about 4 to 5 skin lesions constitutes paucibacillary leprosy, whereas about 5 or more constitutes multibacillary leprosy.

INTRODUCTION

Leprosy, or Hansen disease, is a chronic infectious disease caused by *Mycobacterium leprae*. The skin and nervous manifestations of the disease present a singular clinical picture that is easily recognized. After India, Brazil is the country with the greatest number of cases in the world.[2] Nerve injury is a central feature of the pathogenesis of leprosy because of the unique tendency of *M leprae* to invade Schwann cells and the peripheral nervous system, causing a mononeuritis multiplex of immunologic origin that results in autonomic, sensory, and motor neuropathy. Early diagnosis of leprosy, along with early detection and treatment of neuropathy, are the means to prevent permanent primary impairments.[1]

The World Health Organization (WHO) classification system is used to differentiate the 2 clinical presentations of leprosy. The 2009 WHO classifications are based on the number of bacilli per skin lesions, as follows: paucibacillary leprosy, skin lesions with no bacilli (*M leprae*) seen in a skin smear; multibacillary leprosy, skin lesions with bacilli (*M leprae*) seen in a skin smear.

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However, the WHO further modifies these 2 classifications with clinical criteria because of the nonavailability or nondependability of skin-smear services. The clinical system of classification for the purpose of treatment includes the use of number of skin lesions and/or one or no nerve involved as the basis for grouping patients with leprosy into multibacillary and paucibacillary leprosy. Investigators state that up to about 4 to 5 skin lesions constitutes paucibacillary leprosy, whereas about 5 or more constitutes multibacillary leprosy.

The irreversible motor, sensory, and autonomic impairments caused by leprosy lead to increasing secondary impairments long after the disease process has been arrested. The progressive physical impairments caused by the disease process are compounded by the psychological and social consequences that adversely influence the participation in society of those affected.[3]

INDICATIONS AND CONTRAINDICATIONS

One of the most common secondary disabilities caused by Hansen disease is drop foot, which is found in 2% to 5% of newly diagnosed patients with leprosy.[4] Leprosy neuritis affects nerves where they are close to the skin and pass through a narrow fibro-osseus canal. In the leg, this includes the involvement of the common peroneal nerve at the neck of the fibula, which leads to foot drop, and the posterior tibial nerve in the tarsal tunnel, which produces anesthesia of the sole. When both nerves are damaged, the main impact of walking is on the anesthetic forefoot rather than on the heel and causes trophic ulceration. Unlike the clinical picture of traumatic injury of the common peroneal nerve, in which both of its branches (the deep peroneal nerve and the superficial peroneal nerve) are involved, in leprosy there is the possibility of isolated involvement of the deep peroneal nerve branch, sparing the superficial peroneal branch. The classic clinical manifestation of supinated equino varus deformity associated with injury of the common peroneal nerve commonly seen after a traumatic injury, caused by overpull of plantar flexors and inverters powered by the intact tibial nerve and loss of dorsiflexors and evertors powered by the compromised common peroneal nerve, is replaced by a selective paralysis of the anterior compartment of the leg with preservation of function of both peroneal tendons in the lateral compartment. In addition, the peroneus longus can overpower the weak anterior tibial tendon (its antagonist on the first metatarsal), and produces marked plantar flexion in the first metatarsal that can cause secondary varus in the hindfoot.

The result is a well-balanced foot, regarding the inversion and eversion moments of forces, because both tibialis posterior and peroneal brevis tendons are intact. Although many investigators still recommend the tibialis posterior tendon transfer even when there is normal function of the peroneal tendons, we do not think it is necessary to sacrifice a major tendon stabilizer of the arch of the foot in these selected cases.

The occurrence of flatfoot after harvesting the posterior tibial tendon for drop foot in which both the anterior and lateral compartments of the leg are paralyzed is rare in the literature, and this may be because, in the palsied foot, loss of peroneus brevis function (as a result of nerve injury) and loss of posterior tibial tendon (as a result of tendon transfer) result in a new dynamic balance,[5] preventing the arch breakdown. In contrast, in the presence of normal function of the superficial peroneal nerve, the primary evertor of the foot, the peroneus brevis muscle, is unopposed after the posterior tibial tendon (PTT) transfer, leading to a higher possibility of development of flatfoot, because the deforming force exerted by the peroneus brevis is a well-recognized feature in the development of adult acquired flatfoot, leading to hindfoot valgus and midfoot collapse caused by insufficiency of the posterior tibial tendon.[6]

Yeap and colleagues[7] reported on the results of 12 patients undergoing posterior tibial tendon transfer through the interosseus membrane in patients without leprosy, and found 4 patients with some flattening of the medial longitudinal arch. In addition, the use of the anterior transfer of the tibialis posterior tendon in cerebral palsy to treat spastic equinovarus has led to collapse of the talonavicular joint with significant calcaneus and valgus deformities of the foot in children.[8,9]

To prevent this complication, Klaue and colleagues[10] recommended the simultaneous transfer of the flexor digitorum longus to the medial cuneiform during the PTT transfer in cases of posttraumatic drop foot, pes equinus, and cerebral palsy to restore dorsiflexion.

The objectives of tendon transfer for treatment of drop foot are to improve functional deficit by restoring or reinforcing lost functions, to neutralize deforming forces, and to gain stability, eliminating the need for bracing during gait.[2,5]

The correction of drop foot in leprosy with PTT transfer is well established in the literature, although there is still some debate concerning the need to use the interosseus route. Srinivasan and colleagues[11] recommend the anterior transposition of the PTT subcutaneously, dividing the tendon into 2 slips and suturing the medial slip to the extensor hallucis longus (EHL) and the lateral slip to the extensor digitorum longus (EDL). Advocates for the circumtibial (CT; ie, subcutaneous) transfer claim that is unnecessary to use the interosseus route, because the CT transfer is easier and safer to perform, there is no risk of injury or tethering of the neurovascular bundle, the tendon is allowed to glide smoothly, and it diminishes the possibility of adhesions of the tendon or muscle belly in the interosseous (IO) membrane. In contrast, advocates for the IO transfer claim advantages such as the direct pull of the tendon, producing less inversion deformity, and the possibility of a greater length of tendon for insertion, thus preventing the tenodesis effect related to an overly high tension of the suture transfer. Hall[12] reported that the subcutaneous route gave greater dorsiflexion (25°–30° compared with only 17°) than the interosseus route, but Soares,[4] in a long-term study comparing PTT transfer using the CT route with the IO route for drop foot in patients with leprosy, reported an unacceptably high rate of recurrent inversion in the CT group even in those cases with the peroneal tendons intact, leading to ulceration of the lateral border of the foot, and recommended that the CT route should be reserved for patients with a calcified and unyielding IO membrane. He also reported better active dorsiflexion in the IO group, although there was less active plantar flexion than with the CT route.

Another controversial issue is the point of reinsertion of the tendon, which varies according to the foot deformity present. In a cadaver study,[13] comparing the CT and IO routes for PTT transfer to the dorsum of the foot, in which the insertion sites were tested separately for the medial, intermediate and lateral cuneiform, and the cuboid, concluded that the optimal insertion site for PTT transfer to produce maximal dorsiflexion with minimal pronation was the IO route, with the insertion site in the lateral cuneiform.

The necessity for bone fixation instead of tendon-to-tendon reattachment is also debatable. Tendon-to-tendon suture addresses the difficulties related to tendon-to-bone procedures and donor tendon length. It allows the surgeon to adjust and modulate tendon tension, appropriately verifying foot posture before suturing is completed .Bone fixation provides a stronger attachment site and theoretically prevents the stretching that might occur over time when a tendon-to-tendon transfer is performed. However, the occurrence of a Charcot arthropathy as a consequence of a creation of a bone tunnel in patients with leprosy is possible.[14–16] Some investigators state that the presence of leprosy is a contraindication for bone fixation, and that all those cases should be managed with tendon-to-tendon procedures. Warren[16] in 1968 advised against bone fixation in correction of foot drop in leprosy, because “it has been assumed that surgical intervention with the tarsal bones must increase the tendency to bone breakdown after surgery.” Our recommendation is that, in paucibacillary cases, the transfer should be to bone, and, in multibacillary cases, we do not recommend bone fixation because of the risk of Charcot arthropathy, anchoring the tendon to soft tissues in the dorsum of the foot.

Carayon and colleagues[17] use both the PTT and the flexor digitorum longus (FDL) through the interosseus membrane with tendon-to-tendon insertion, in which the PTT is sutured to the tibialis anterior and the FDL to both the tendons of EHL and EDL. They reported 31 cases, of which 26 cases were drop foot caused by Hansen disease. The results were excellent in 11 (active dorsiflexion of 15° or more and active plantar flexion of more than 30°), good in 6 (active dorsiflexion of 5°–10° and active plantar flexion of 15°–20°, fair in 2 (no active dorsiflexion but correction of drop foot and plantar flexion to 10°), and poor in 3 (the foot held in a disabling degree of plantar flexion and no dorsiflexion). In an analysis of the fair and poor results, they noted that most failures were caused by loss of tension in the transferred tendons at the point of fixation, which is one of the problems with tendon-to-tendon fixation.

The use of the peroneal longus tendon as the single dorsiflexor of the foot in leprosy was first reported by Srinivasan and colleagues,[11] who wrote that “with the peronei tendons intact, removal of the tibial posterior from the medial side of the ankle might cause instability of the foot.” This method has the advantage of leaving the PTT intact on the medial side, whereas the peroneus brevis was left to balance it on the lateral side. They also stated that, if the patient later developed paralysis of the peroneals, the tibialis posterior could still be used as a motor. In his original technique, he split the tendon into 2 halves and performed a tendon-to-tendon transfer into the EHL, EDL, and peroneus tertius. In our surgical procedure, we use the entire tendon, use a subcutaneous route, and anchor it to the intermediate cuneiforme with a drill hole or to the intercuneiform ligaments. Two benefits are derived from this tendon transfer. A deforming force (plantar flexion of the first metatarsal producing forefoot-driven hindfoot varus) is eliminated, because there is an unopposed pull of the peroneus longus against the weak tibialis anterior tendon, and a correcting force (dorsiflexion in the ankle) is established. Morton[18] pointed out that the peroneus longus had been a lateral dorsiflexor in early evolutionary stages, justifying its use as a dorsiflexor muscle.

The bridle procedure was described by Riordan and later modified by Rodiguez.[19] It also uses the peroneus longus tendon, combining the transfer of the peroneus longus with the PTT[19] to balance the foot in dorsiflexion. However, in this surgery the peroneus longus is not used as the primarily active dorsiflexor, because it is divided proximally, rerouted anteriorly to the lateral malleolus, and anastomosed with the anterior and posterior tibialis tendons in the anterior leg.

Despite the peroneus longus transfer for drop foot being described more than 40 years ago, there is still some question of its efficacy. Sigvard Hansen[20] stated in his textbook that, “transfer of the peroneus longus seems to violate too many rules to be effective. In this procedure, the peroneus longus is removed from its normal bed, carried a long distance, and expected to go from plantar flexion in stance phase to dorsiflexion in swing phase.”

In addition, the peroneus longus transfer alone may not be as strong as the tibialis posterior. **Tables 1** and **2** show the different moments of rotation of the extrinsic tendons around the ankle and subtalar joints, and show that the tibialis posterior and peroneus longus tendons have similar amounts of force.

When using the PTT as an active dorsiflexor, the tendon transfer should act partially as a tenodesis, producing a decreased range of motion in the plantar direction. After

Table 1
Moments of rotation of the extrinsic tendons in relation to the ankle joint

Tendon	Moment of Rotation (Nm)
Flexor Tendons	
Achilles tendon	164
Flexor digitorum longus	3.9
Tibialis posterior	3.9
Peroneus longus	3.9
Peroneus brevis	2.9
Flexor hallucis longus	8.8
Extensor Tendons	
Tibialis anterior	25
Extensor hallucis longus	3.9
Extensor digitorum longus	7.8
Peroneus tertius	4.9

Data from Sarrafian SK. Anatomy of the foot and ankle. 2nd edition. Philadelphia: Lippincott; 1993.

bridle PTT for drop foot, Gellman and colleagues[21] reported an average ankle dorsiflexion of 10° and plantar flexion most commonly measuring 0°. The subtalar joint range of motion was significantly reduced to 0% to 25% of the nonoperative side.

Because it is not necessary to reroute the peroneus longus tendon through the interosseus membrane, a better excursion of the tendon is possible, avoiding the tenodesis effect related to adhesions in the IO space. This option potentially increases the range of motion after the tendon transfer. However, according to biomechanical principles of tendon transfers, when a stance-phase muscle (both the posterior tendon and peroneus longus are stance-phase muscles) is converted to a swing-phase muscle (nonphasic transfer), there is a chance that it will function more as a tenodesis than as an active muscle transfer, regardless of the route used for the transfer.

Table 2
Moments of rotation of the extrinsic tendons in relation to the foot plate and the subtalar joint axis

Tendon	Moment of Rotation (Nm)
Supinator Tendons	
Achilles tendon	48
Flexor digitorum longus	7.8
Tibialis anterior	9.8
Tibialis posterior	18
Flexor hallucis longus	7.8
Pronator Tendons	
Peroneus longus	17
Peroneus brevis	13
Extensor digitorum longus	7.8
Peroneus tertius	4.9
Extensor hallucis longus	1
Tibialis anterior	2.9

Data from Sarrafian SK. Anatomy of the foot and ankle. 2nd edition. Philadelphia: Lippincott; 1993.

PREOPERATIVE PLANNING

It is of paramount importance that the disease process is controlled and the patient is adequately treated with multidrug therapy. In addition, it must be established that there has been at least 1 year without inflammatory reactions (reverse reaction or erythema nodosum). This is because the surgical procedure itself might cause those reactions, compromising the surgical results. The neurologic damage must be irreversible, which is considered after 1 year without improvement of the motor function or established earlier with eletroneuromuscular studies. Equinus contracture of the Achilles tendon must be tested for. Patients with passive dorsiflexion of less than 20° should have percutaneous lengthening of the Achilles at the same time as the transfer. It may be advisable to perform percutaneous lengthening in all patients, because the more powerful Achilles tendon will eventually overcome the weaker peroneus longus. The ideal patient for the peroneus longus transfer has normal motor strength in the peroneus longus muscle, but motor strength of +4/+5 is also acceptable and provides enough power for ambulation out of a brace. As in all tendon transfers, any fixed joint deformities must be corrected to gain a successful result, requiring arthrodesis, osteotomy, or soft tissue releases. The presence of clawing of the toes should be noted and treated if necessary.

We always instruct our patients how to contract the tendon to be transferred in an isolated manner in the preoperative period to maximize our results. Factors such as motivation, self-care, and whether the patient is capable of following the postoperative recommendation are important issues.

SURGICAL TECHNIQUE

With the patient lying supine on the operating table, a pneumatic tourniquet is applied to the thigh. The entire lower limb is prepped and draped in a regular fashion. If the limb is overly externally rotated, an ipsilateral bump is place under the buttock to internally rotate the leg. Patients are given preoperative intravenous antibiotics (**Fig. 1**). This procedure may be performed with the patient under spinal, epidural, or general anesthesia. Testing for equinus contracture is performed. If present, a percutaneous lengthening of the Achilles is done. The peroneal tendons are accessed through a short incision at the level of the base of the fifth metatarsal directed approximately 3 cm toward the lateral malleolus, parallel to the peroneus longus tendon. The dissection is carried down to the level of the paratenon. The sural nerve usually runs more cranially; if identified, it is retracted. The peroneal tendon sheath is then opened and the peroneus

Fig. 1. Marking of skin incisions.

longus can typically be seen beneath the peroneus brevis. The peroneus longus is pulled using an elevator and secured by a suture.[22] Before cutting the tendon, it is important to tenodese the distal stump of the peroneus longus with the peroneus brevis tendon using a side-by-side suture (Ethbond no. 2; **Fig. 2**), which prevents elevation of the first metatarsal (dorsal bunion) because of the unopposed pull of the tibialis anterior tendon (although this tendon is theoretically weak in drop foot, I always tenodese the distal stump of the peroneus longus to the peroneus brevis tendon). Now the peroneus longus tendon can be sectioned as distally as possible in the cuboid tunnel and the proximal stump tagged with a whipstitch suture. A second incision is then made proximally on the lateral compartment in the distal third of the leg, about 8 cm proximal to the tip of the fibula at the level of the musculotendinous junction of the peroneus longus. Protect the superficial peroneal nerve. The tendon sheath of the peroneus longus is incised and the peroneus longus tendon is identified and pulled proximally in the wound (**Fig. 3**). The tendon is kept moist with a sponge or allowed to remain in its sheath during dissection of the dorsal tarsus. A third 3-cm transverse incision is made on the dorsum of the foot over the intermediate cuneiform (**Fig. 4**). Care is taken with the superficial peroneal nerve and extensor tendons. Protect the deep neurovascular bundle, which is usually encountered in this approach; it is directly deep to the muscle of the extensor hallucis brevis. Identify the intermediate cuneiform, leaving the periosteum and capsular tissue intact. A tunnelization clamp is passed subcutaneously from this incision to the proximal lateral wound and the tagged peroneal longus tendon is delivered distally (**Fig. 5**). Using a fluoroscopic image is helpful to confirm the position for creation of the bone tunnel in the intermediate cuneiform. The bone tunnel is made from dorsal to plantar using drill bits and curettes as necessary to accommodate the tendon. A straight Keith needle is used to pass the tag suture through the bone tunnel, exiting the sole of the foot. The foot is then held in maximal dorsiflexion (if possible, in 30° of dorsiflexion) and the tag suture in the plantar aspect of the foot should be pulled distally, thereby pulling the tendon end into the tunnel. The proximal stump of the peroneus longus is then fixed to the bone tunnel using staples, biotenodesis screws, or anchors. If possible, we augment the anchor point with several nonabsorbable sutures from the periosteum surrounding the tunnel to the tendon directly at the entrance of the tunnel. In multibacillary cases, we avoid using the bone insertion because of the potential for Charcot arthropathy. In these situation, we insert the peroneus longus transfer in the lateral margin of the intertarsal ligaments (**Fig. 6**).

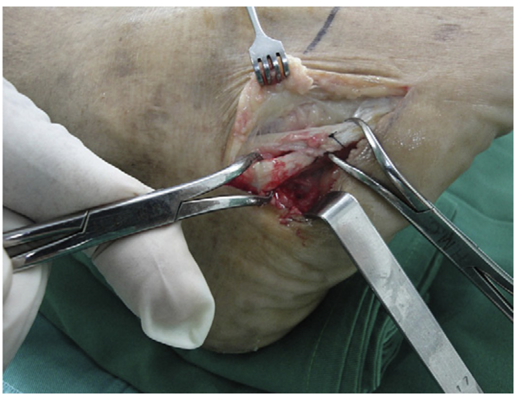

Fig. 2. Tenodesis of the distal stump of the peroneus longus to the peroneus brevis before cutting the tendon.

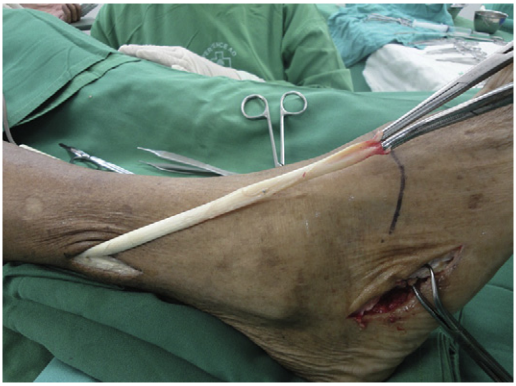

Fig. 3. Delivery of the peroneus longus tendon through the lateral incision in the lower leg.

Fig. 4. Third incision over the dorsum of the foot, exposing the extensor tendons and taking care with the neurovascular structures.

Fig. 5. After rerouting the peroneus longus tendon through the subcutaneous route.

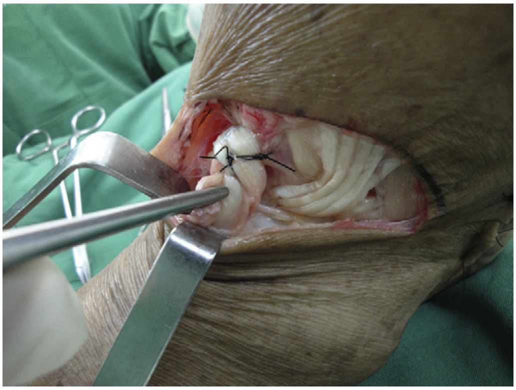

Fig. 6. Suture of the peroneus longus tendon to the intertarsal ligaments with nonabsorbable stitches.

Fig. 7. Wounds closed, the transferred peroneus longus can be seen under the skin.

Fig. 8. Final postoperative position of the foot.

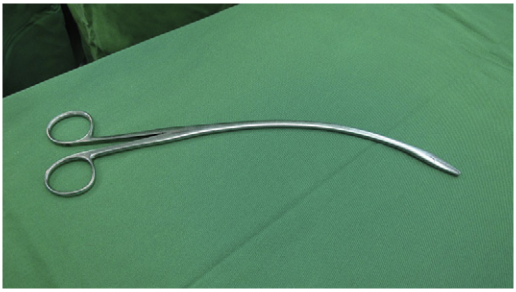

Fig. 9. Tunnelization clamp.

Fig. 10. Postoperative example: active plantar flexion.

Fig. 11. Postoperative example: active dorsiflexion.

The wound is irrigated and closed in a regular fashion. At the end of the procedure, the foot should rest in 20° to 25° of dorsiflexion and a well-padded short leg cast is applied, also holding the foot in the desired dorsiflexion position (**Figs. 7–11**).

POSTOPERATIVE MANAGEMENT

The cast is maintained for 2 weeks. The cast is then removed and stitches taken out. The patient is then placed in a weight-bearing cast and is allowed partial weight bearing with use of crutches. Six weeks after the procedure, the cast is removed and the patient is instructed to ambulate in an ankle-foot orthosis brace or walker boot. At 8 weeks, the rehabilitation program to improve strengthening, proprioception, gait, and range of motion is started, but the protected ambulation is continued for 4 months. In addition, the patient should use a cam boot for sleeping until 4 months after surgery. Physical therapy is beneficial to train the peroneus longus in its new phase, and the patient is encouraged to walk as much as possible. If the muscle fails to adapt to its new function, electrical stimulation should be added to the physical therapy program.

RESULTS

From 1998 to 2008, we operated on 57 cases of drop foot caused by Hansen disease, with 61 feet, 59% men and 41% women, with an average age of 39 years. Among these, 19 cases were considered to have a selective paralysis of the anterior compartment of the leg, with functioning peroneal tendons. The postoperative results showed that average dorsiflexion was 10° and plantar flexion was 32°. Fifteen cases were very satisfied with the procedure, being able to walk without any kind of supportive brace. Two cases were satisfied with the result, but still wear a brace for walking long distances. We had 2 poor results, with weak dorsiflexion after the transfer. These cases may not have been good candidates for the procedure because of undetected weakness of the peroneal tendons.

POSSIBLE CONCERNS, INDICATIONS, AND FUTURE OF THE TECHNIQUE

We are concerned that the peroneus longus transfer for drop foot might result in varus deformity of the hindfoot in the middle to long term, because the remaining force provided by the peroneus brevis may not be enough to counteract the powerful pull of the tibialis posterior tendon. Other possible indications for the peroneus longus transfer are posttraumatic anterior or anterior and deep posterior compartment syndromes. Both of these conditions are characterized by drop-foot deformity and absence of anterior tibial tendon function.

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