CHAPTER **43**

DROP HALLUX: Assessment and Surgical Repair

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INTRODUCTION

Nerve palsy or isolated injury of the extensor hallucis longus (EHL) can result in the drop hallux condition, which has been rarely reported in our literature. Upon ruling out differential diagnoses and assessing the etiology of the condition, treatment options must be considered. Spontaneous recovery may occur in some neurologic cases, however, surgical repair is warranted in chronic conditions. Here, we provide background information on the drop hallux condition and present a case report and surgical technique shown to be an effective treatment.

ANATOMIC REVIEW OF THE EHL

Myology

The EHL muscle arises deep in the anterior muscle compartment of the leg. Its origin includes the crural interosseous membrane and the medial surface of the fibula at mid-shaft level (1). As the muscle courses inferior-medially toward the ankle joint, the muscle transitions into tendinous fibers (2). The EHL tendon runs underneath the superior and inferior extensor retinacula and then runs toward the first metatarsophalangeal joint (MPJ). The EHL primarily inserts onto the dorsal base of the distal phalanx of the hallux. The apponeurotic fibers of the EHL, however, form the extensor expansion that extends around each side of the proximal phalanx to ultimately insert into the plantar plate (1).

Innervation

The common peroneal nerve (CPN) wraps around the fibular neck and enters the fibular tunnel, a narrow fibrous hiatus at the origin of the peroneus longus (2). The CPN then divides into its terminal branches – recurrent articular, superficial peroneal and deep peroneal nerves (3-4). The deep peroneal nerve (DPN) innervates the muscles of the anterior compartment of the leg. The DPN sends short muscular nerve branches to innervate the tibialis anterior (TA) and extensor digitorum longus (EDL) muscles. The DPN gives off a longer motor branch, which runs inferiorly along the fibula periosteum until it reaches the EHL (Figure 1.) The EHL muscle arises lower on the fibula than the other muscles of the anterior compartment, as shown in Figure 2.

Blood Supply

The anterior tibial artery gives off 6-8 arterial branches that pass through the muscular belly of the EDL to supply the EHL (5). The EHL also receives arterial branches from the posterior tibial artery.

Tendon Sheath

The EHL tendon descends the leg in its own fibrous tunnel. The tendon sheath of the EHL originates just proximal to the upper arm of the inferior extensor retinaculum (1.75cm proximal to the ankle joint) (1). The sheath continues to the level of the metatarsal-cuneiform joint.

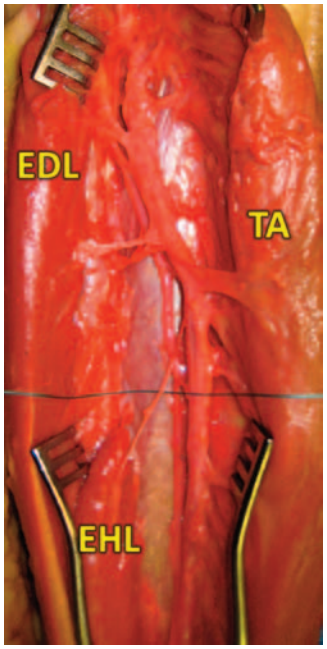

Figure 1. DPN Innervation to the Anterior Compartment Muscles. Multiple, short motor nerve branches to the TA and EDL are shown proximally. One motor nerve (isolated with green suture) is shown to innervate the EHL. This branch appears to be the longest and thinnest motor nerve of the anterior compartment.

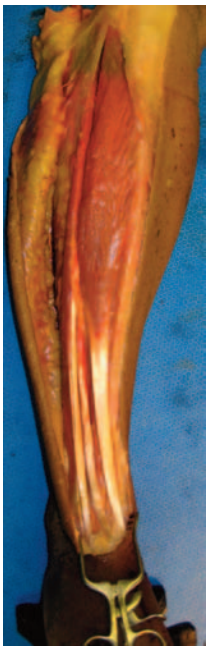

Figure 2. Muscles of the Anterior Compartment. The EHL has a more distal origin than the TA and EDL.

BIOMECHANICS OF THE EHL

All muscles in the anterior compartment of the leg course anterior to the ankle joint axis and therefore dorsiflex the foot on the ankle. The EHL courses medial to the ankle joint axis so it also supinates the foot (6).

The EHL's insertions to the hallux's distal phalanx and plantar plate allow it to provide stability to the first MPJ and hallux interphalangeal joint. This is necessary during propulsion; as the heel is lifted from the ground, the hallux dorsiflexes on the plantarflexed metatarsal and the EHL provides extensor stability that allows the first ray to function as a rigid lever (6). Initially in swing phase, the TA and EHL contract to accelerate ankle dorsiflexion and this helps to clear the foot from the ground. By maintaining the hallux in a dorsiflexed position throughout swing, the EHL also stabilizes the hallux so it can clear the ground. During swing, the foot is in a pronated position and the hallux would otherwise drag across the ground surface. In a drop foot or drop hallux condition, the patient can accommodate via a steppage gait pattern to achieve clearance (6).

DROP FOOT

Weakness or paralysis of the anterior compartment muscles results in impaired foot clearance during the swing phase of gait. This drop foot condition is the hallmark of CPN palsy.

CPN palsy accounts for approximately 15% of all peripheral nerve injuries (7). The frequency of this condition is a consequence of CPN's superficial anatomic course, confinement within the fibular tunnel, and its intimate relation to the fibula (3).

Various etiologies may lead to CPN palsy. Compression of the CPN can occur secondary to cast pressure, sitting in a cross-legged position for a prolonged period, improper positioning or ineffective padding on the operating room table, a hematoma, popliteal cyst, enlarged fabella, etc. (3). Traumatic injury, causing complete or partial disruption of the CPN, can result from knee dislocation, a Maisonneuve fibular fracture, or could be iatrogenic (3).

Treatment of drop foot is dependent on the severity, etiology, and duration of the condition. In general, acute CPN palsies presenting in open injuries should undergo emergent surgical exploration (8). In closed injuries, acute CPN palsies may spontaneously recover via neuronal regeneration; however, surgical repair should be considered after 4 months of no improvement (8). Surgical treatments involve nerve decompression, nerve repair, nerve transfer, tendon transfer, or ankle fusion (9). Mild cases of drop foot, however, may only necessitate an ankle foot orthosis to hold the foot in a neutral position until healing occurs. Spontaneous recovery has been reported after a period of months to over a year (10). Ankle dorsiflexion typically returns prior to hallux dorsiflexion. In some cases, neuronal regeneration is incomplete with continued impairment isolated to the EHL (10).

DROP HALLUX

Drop hallux is a condition similar to that of drop foot; however, instead of all muscles of the anterior compartment being affected, only EHL function is impaired. Due to an intact TA, ankle dorsiflexion is adequate for foot clearance but the patient has no active hallux dorsiflexion. This condition is problematic since the non-functional EHL cannot stabilize the first ray or accelerate and decelerate the foot throughout the gait cycle.

Similar to drop foot, various etiologies for drop hallux have been reported. Incomplete drop foot recovery can result in a drop hallux condition as described above. The CPN trunk contains a separate identifiable fascicle to the EHL (11), which may be susceptible to injury as it wraps around the fibular neck. A direct lesion of the motor nerve to the EHL could also result in a neurologic drop hallux.

Isolated EHL weakness or paralysis can occur iatrogenically during surgical procedures that expose the proximal one-third of the tibia or fibula. This has been reported after proximal tibia and fibula osteotomies,

application of external fixation, knee reconstruction, or intramedullary nailing of tibia fractures, etc. (12-20). Iatrogenic injury may result from trauma to the popliteal artery, tibial nerve, or the CPN and its branches (12). Incidence of neurologic damage varies with the surgical procedure performed; however, it is nearly always present (21). A prospective study by Shingade et al reported 10 of 26 patients (38.5% incidence) sustained isolated EHL injury after autograft harvest of the proximal fibula (15). Georgoulis et al observed 4 cases of drop hallux (not proceeded by drop foot) following 250 high tibial osteotomies; 1.6% incidence (12). In general, proximal fibular osteotomies are reported to have a higher postoperative incidence of neurologic injury (4.1-11.9% incidence) (16-17) compared to more distal fibular osteotomies (1.0% incidence) (18).

Anatomic data provided by cadaveric studies have been important, specifically in explaining neurologic injury as an etiology of drop hallux (4, 12-13, 22-23). Various innervation patterns to the EHL have been reported. Past studies report that 25-100% of EHL muscles are innervated via a single motor nerve (12, 15, 22-24). If the EHL does receive motor innervation via a single motor nerve, complete paralysis or weakness would result from its injury.

The motor branch given off from the DPN to innervate the EHL is vulnerable to injury. It has been shown to be the longest (ranging from 5-10cm in length) and the thinnest of the DPN's motor branches (4, 11, 13, 23). Since the DPN gives off its motor branches at about the same level and the EHL has a more distal origin than the other muscles in the anterior compartment, this makes sense intuitively. As the nerve courses inferiorly along the fibular periosteum, it is vulnerable to injury from osteotomy or periosteal stripping (15, 21). If the nerve is retracted away from bone where the osteotomy is to be performed, it is still tightly bound to the fibula via periosteum proximally and distally. Therefore, the nerve is vulnerable to stretching forces caused by retraction (21). Ischemic conditions caused by use of a pneumatic tourniquet may make the nerve more sensitive to this stretch injury (21). Compartment syndrome is another possible etiology of drop hallux (20). Increased anterior compartment pressure can result from poor surgical hemostasis, hematoma formation, or reperfusion after deflation of a tourniquet, etc. (21). Elevated anterior compartment pressures may first present as a pressure palsy of the motor nerve to the EHL since it is the longest and thinnest motor nerve in the compartment and it is tightly bound by fibro-osseous structures. The EHL may also be directly vulnerable to increased compartment pressures since it is the deepest muscle in the compartment and it would be sandwiched between the TA, EDL, and intramuscular septum (22).

Spontaneous drop hallux (isolated EHL impairment not proceeded by a trauma, surgery, or drop foot) has been reported twice in the literature. Moorman et al presented a patient in which drop hallux resulted from CPN compression after a prolonged period of abnormal foot positioning (25). After 3 months, EHL strength was measured as 1-2/5 and the patient chose to continue wearing a customized hallux brace. Spinner et al reported a possible ischemic etiology of spontaneous drop hallux in which a patient with hereditary spherocytosis presented without other pertinent history (26). In this patient, full recovery was reported after 3 months of conservative treatment.

Assessment of Drop Hallux

Diagnosis of drop hallux is made via clinical assessment. The patient will have an isolated EHL weakness or a complete inability to dorsiflex the hallux. The differential diagnosis of drop foot must be ruled out by thoroughly assessing the muscle strength of the other muscles in the anterior compartment. Barefoot gait assessment may reveal the hallux dragging across the ground or it may demonstrate a compensatory steppage gait. During nonweight-bearing assessment with the foot in a gravity-dependent position, the hallux will be in a relatively plantarflexed position. The contralateral hallux serves as a control for assessment of the affected hallux.

A history of present illness and a medical history should also be analyzed. Etiologic factors should be identified and the duration of injury should be calculated. If the patient presents with the clinical signs of compartment syndrome, the compartment pressures should be measured and immediate fasciotomy may be indicated. Patients who present with drop hallux and a history of trauma necessitate radiographic evaluation to rule out avulsion of the EHL (Figure 3.) Magnetic resonance imaging can assess the continuity of the EHL tendon when it is necessary to rule out laceration, rupture, or aplasia, etc. (Figure 4).

Clinical diagnosis of a neurologic drop hallux can be supported via electrodiagnostic evidence. Electromyogram studies will show a denervation fibrillation pattern in the affected EHL and a slowed nerve conduction velocity in the motor branch to the EHL (15). Electromyogram and nerve conduction velocities will be normal in the other branches of the peroneal nerve and the muscles supplied by them in the ipsilateral and contralateral limbs.

Treatment of Drop Hallux

Conservative treatment options include an ankle foot orthosis, custom hallux bracing, and physical therapy strengthening exercises. In Shingade et al's series, 9 of the 10 patients who acquired drop hallux spontaneously recovered. Mean time to recovery was 5.5 months (ranging from 4-6

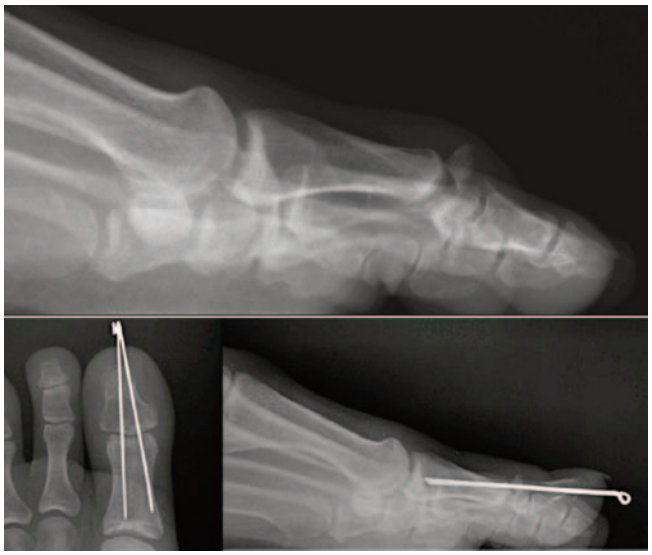

Figure 3. EHL Avulsion. Radiographs can assist in diagnosing a drop hallux caused by avulsion. Once etiology of the drop hallux is identified, appropriate surgical repair can be decided. Here, open reduction and internal fixation of the avulsion was performed.

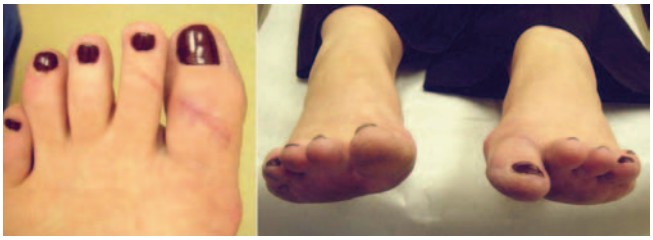

Figure 4. EHL Laceration. In this patient, a traumatic injury to the first and second digits resulted in a drop hallux due to EHL laceration. On nonweightbearing clinical assessment, the left hallux is in a plantarflexed attitude, relative to the nonlacerated hallux on the right side.

months) (15). Spontaneous axonal regeneration and recovery from drop hallux may take up to one year, but may not recover in 20% of drop hallux cases (25). Shingade et al reported one individual (10%) who did not recover after 25 months. In chronic conditions, conservative measures can be continued indefinitely or surgical intervention can be pursued (15).

Surgical repair for neurologic drop hallux has only been described in one published report, to our knowledge. In 2010, Singh et al reported a technique in which the medial half of the first slip of the EDL and the extensor hallucis brevis were anastomosed to the EHL. A good functional result and patient satisfaction was reported; n=1 (27). Similar surgical techniques have been described for EHL lacerations. If the tendon cannot be primarily repaired, the EHL can be anastomosed to the TA or the EDL to restore dynamic function (28, 29). Nerve repair and nerve transfers have not been described for the treatment of drop hallux; however, these techniques may be helpful. Fusion of the first MPJ may not be necessary for correction, but it would offer a definitive positional correction of the hallux.

CASE REPORT

A 22-year-old male sustained a closed tibia-fibula fracture while playing soccer. The patient initially presented to another physician who reduced the fracture via external fixation. A drop foot developed thereafter. After one year of physical therapy strength training, the drop foot resolved but a residual drop hallux persisted. The patient presented at this time for surgical evaluation. His chief complaint was uncomfortable barefoot walking and frequent falling. On barefoot gait examination, a steppage gait was observed as a compensation for a drop hallux (Figure 5). Clinical assessment demonstrated a paralytic EHL; however, the other muscles of the anterior compartment had normal strength. Surgical treatment options were considered for treatment of the drop hallux.

The senior author performed an anastomosis of the EHL to the TA to provide a functional correction. Through a single 2 cm long linear incision at the anterior ankle, the tendons of the EHL and TA were isolated (Figure 6). The tendons were anastomosed using 2-0 Fiberwire (Arthrex, Naples, FL) via a running barrel stitch. Care was taken to maintain the tendons under physiologic tension.

The patient recovered without complication. He was initially nonweightbearing for 3 weeks. After gradual increments, the patient achieved a clinically normal gait at 6 weeks postoperative. The accommodative steppage gait resolved since the hallux was now able to dorsiflex (Figure 7). The patient experienced a functional improvement and was overall very satisfied with the results of the procedure.

Similar techniques have been described for treatment of EHL lacerations; however, this is the first description of this procedure for a neurologic drop hallux, to our knowledge. This procedure provided a good functional outcome with high patient satisfaction. EHL to TA anastomosis should be considered as a surgical treatment for drop hallux.

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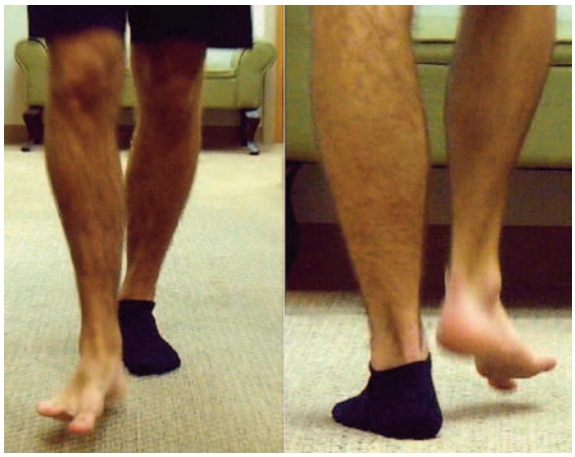

Figure 5. Drop Hallux. The EHL's inability to dorsiflex the hallux results in an accommodative steppage gait to allow the hallux to clear the ground.

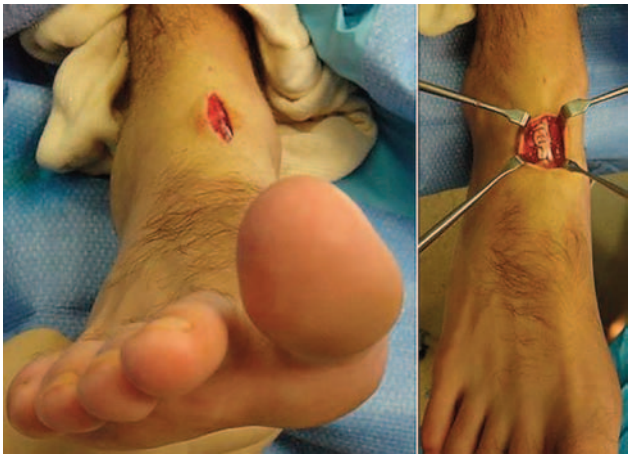

Figure 6. Anastomosis of the EHL to the TA. A 2 cm long, linear incision was created at the anterior ankle. The TA and EHL were identified an anastomosed using 2-0 Fiberwire via a running barrel stitch.

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Figure 7. Functional Improvement Following EHL to TA Anastomosis. The patient's drop hallux condition resolved. He no longer ambulates with a steppage gait.

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