Flexor Hallucis Longus Transfer for Foot Drop without Functioning *Tibialis Posterior*

Přenos šlachy *m. flexor hallucis longus* u peroneální parézy bez využití *m. tibialis posterior*

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SUMMARY

For a foot drop resulting from peroneal nerve palsy transferring the *tibialis posterior* tendon is a standard surgical treatment. The situation of foot drop with no functioning *tibialis posterior* presents a challenge. We describe a case of successful *flexor hallucis longus* transfer in such a case.

Key words: foot drop, *flexor hallucis longus*, peroneal nerve palsy; tendon transfer.

INTRODUCTION

In case of a foot drop requiring surgical treatment, a standard approach is transferring the *tibialis posterior* (TP) tendon to the dorsum of the foot. Patient without working TP presents a special challenge and usually requires an arthrodesis. We present a case, where active dorsiflexion of the foot has been restored by means of transferring a *flexor hallucis longus* (FHL) tendon.

CASE DESCRIPTION

A 29-year-old patient sustained posterior dislocation of the left knee due to the fall from height while biking. Patient received emergency treatment which consisted of popliteal artery reconstruction, fasciotomy and knee external fixation. Secondary split- thickness skin grafting of fasciotomy wounds was performed. From the time of the injury complete palsy of tibial and peroneal nerves was observed. Ultrasound revealed preserved continuity of peroneal nerve and hematoma around it, electromyography studies confirmed full nerve palsy.

While tibial nerve presented first signs of motor recovery after 6 weeks and eventually patient regained full function of tibial nerve, no signs of peroneal nerve recovery were observed neither clinically nor electromyographically.

Thirteen months post-injury a neurolysis of the peroneal nerve was performed. No signs of nerve recovery clinically or in conduction studies were observed up to one year after this procedure.

Physical examination revealed extremity with signs of complete peroneal palsy, scars after fasciotomy and palpable foot pulses. Muscle function testing of *tibialis anterior* (TA), *extensor hallucis longus* (EHL), *extensor digitorum longus* (EDL), *peroneus longus* (PL) and *peroneus brevis* (PB) revealed no function (MRC=0 (15)), while testing of FHL and FDL revealed full (MRC=5) strength. The TP was not functioning (MRC=0) with contracture limiting pronation beyond neutral. The gastrocnemius strength was 4 with contracture limiting dorsiflexion. Supination of the foot was unrestricted. The passive range of ankle motion was (0)-5-45° (equinus contracture of 5°). There was checkrein phenomenon of the toes at the limit of dorsiflexion. On x-ray there were no signs of arthritis in the hindfoot. Patient scored 74 and 71 points in FAOQ (2) and AOFAS (8) scales, respectively.

Decision process

With no return of peroneal nerve function in two years observation, neither improvement in conduction studies despite neurolysis, the nerve injury was deemed irreparable. The patient was unwilling to accept the brace and he opted for operative treatment. With no voluntary contraction of TP this tendon was deemed unsuitable for transfer. Fibrotic degeneration of TP is visible in MRI (Fig. 1). With no functioning TP, two potential motors were available – FHL and FDL. We chose to transfer the FHL tendon to the dorsum of the foot.

Operative technique and aftertreatment

The patient was operated supine without a tourniquet (due to previous vascular reconstruction). Z-lengthening of the Achilles tendon was performed through stab incisions (18). By this maneuver 10 degrees of dorsiflexion was achieved. Dorsiflexion exacerbated the checkrein deformity of the toes. Medial approach to the midfoot

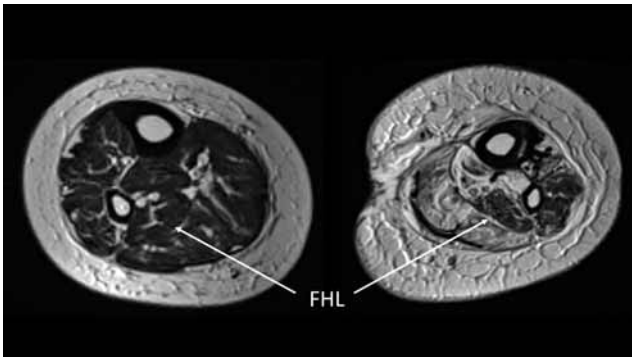

Fig. 1. MRI comparison of the muscles of the right and left lower leg. Note global degeneration of the muscles sparing the FHL and FDL.

was performed. Next the FHL tendon was released at Henry's notch and the FDL tendon Z-lengthened and correction of the checkrein deformity of the toes was achieved. With posterolateral and anterior approach the FHL tendon was transferred anteriorly through interosseous membrane (Fig. 2). The distal part of the muscle belly of FHL was resected to avoid entrapment in the membrane opening. Through the separate approach the tunnel has been created in intermediate cuneiform bone. The tendon has been passed subcutaneously over the extensor retinaculum and after shortening to provide appropriate tension it has been secured to the intermediate cuneiform bone with an interference screw. The foot was immobilized in posterior plaster slab latter changed to removable walker boot. The patient remained non-weight bearing for 6 weeks. Physiotherapy as well as weight-bearing was commenced at 6 weeks after the procedure.

At 10 months follow-up patient is brace free with active range of motion 5–0–10° (Fig. 3). The FAOQ and AOFAS scores are 77 and 80 respectively (Fig. 3).

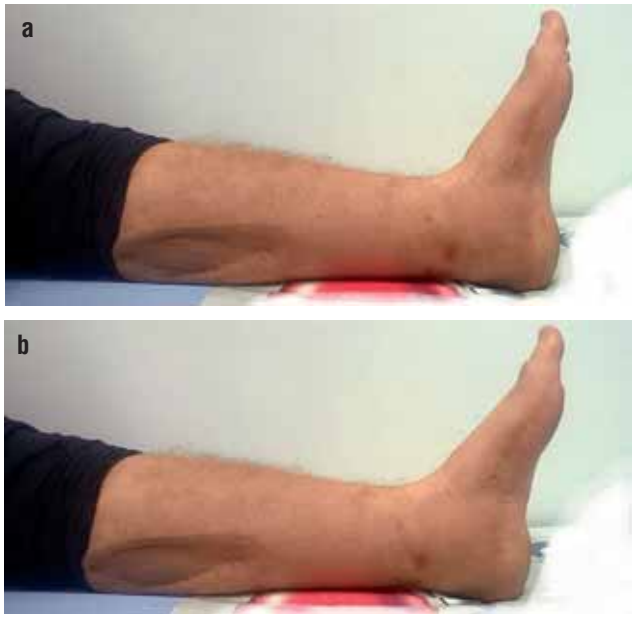

Fig. 3. The arch of active ankle dorsiflexion (a) and plantarflexion (b).

DISCUSSION

The peroneal nerve lesion results in loss of foot extensors function. Complete palsies resulting from knee dislocations carry a bad prognosis (6, 11, 17). When no recovery is observed, decision to continue with a brace can be made or if a patient is unable or unwilling to use a brace, the tendon transfer should be considered. Standard transfer is a TP transfer to the dorsum of the foot (6). Flexors are typically used in leprosy patients (FDL) (3, 4) or to supplement TP transfer (FHL) (9). Patient with no functioning TP presents a special challenge, and typically arthrodesis is an offered option (10).

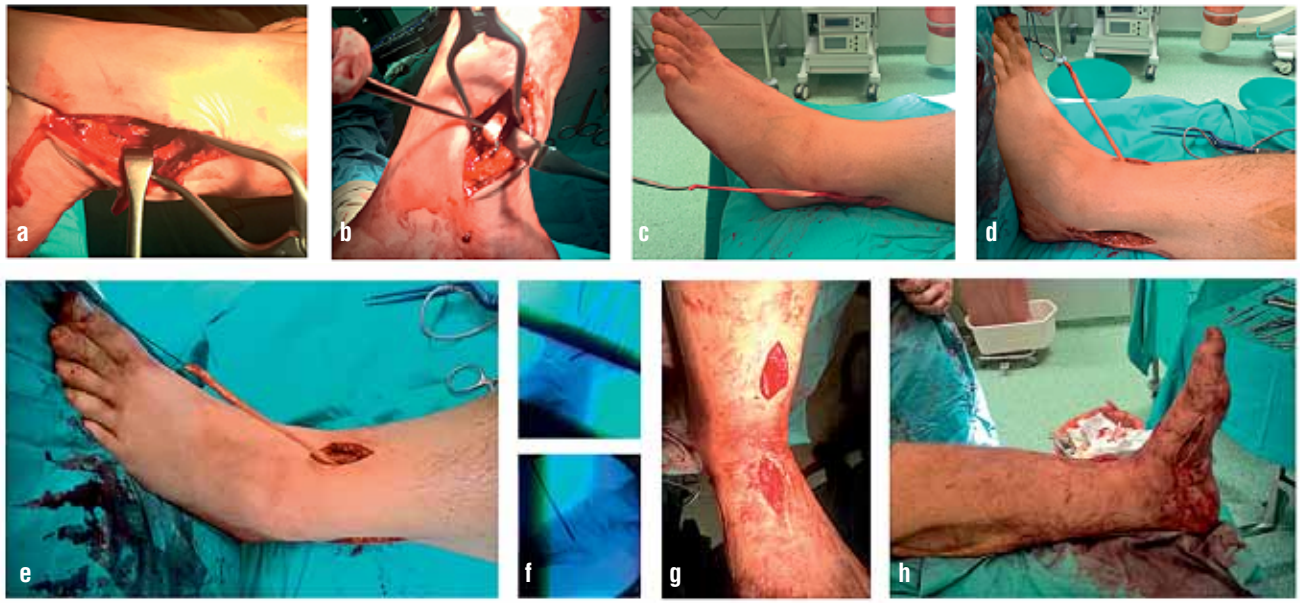

Fig. 2. Operative procedure: approach to the midfoot and identification of FHL (a and b), FHL tendon harvest (c), transfer through the membrane (d and e), fixation to intermediate cuneiforme (f), final foot position (g and h).

We present a case of successful FHL transfer to the dorsum of the foot in a patient without functioning TP. The transfer allowed the patient to ambulate brace-free and allowed for preservation of uninjured hindfoot joints.

Typical transfer was not possible in our patient, as he presented combined lesions from peroneal nerve palsy and with sequel of compartment syndrome (as illustrated by MR imaging – Fig. 1). This left the foot with functioning FHL and FDL and limited function of *gastrocnemius*.

Tibial palsy in our patient resolved completely, so we presume that observed deficiencies of the muscles of the posterior group resulted from compartment syndrome. It should be noted however, that in compartment syndrome usually the FHL is most affected (14). In our patient the FHL was spared and *gastrocnemius* and TP more affected by postischemic scarring. It is possible that in arterial damage the branches to the gastrocnemius and *tibialis posterior* may have been more damaged as they arise more proximally. The FHL receiving arterial supply more distally from posterior tibial artery was successfully reperfused.

The FHL is characterized by the muscle belly extending further distally than it is in TP. We resected the distal part of the muscle that was in danger of entrapment in the interosseous membrane opening (16).

Transfer of the FHL has been described to supplement Achilles reconstruction (12), for reconstruction of peroneal tendons (7), to supplement TP tendon transfer (9) and in flatfoot treatment (1). We are not aware of any report of using FHL for tendon transfer for the adult posttraumatic dropfoot.

Transfer of the FHL (and FDL) as a motor for dorsiflexion has been described by Hiroshima in pediatric cerebral palsy population (5). In contrast to above-described procedure, Hiroshima transferred FHL (and FDL in 62% of cases) to II metatarsal under the extensor retinaculum. Hiroshima observed limited functional conversion on transferred tendons and attributed the achieved results to tenodesis effect. To the contrary, in our patient we transferred the tendon to intermediate cuneiform, over the retinaculum, and observed full voluntary control over foot dorsiflexion. We believe lack of function in Hiroshima's series can be partially attributed to primary diagnosis of their patients and partially to subretinacular placement of the tendon which can cause overstuffing of the retinaculum (13).

The uniqueness of described case lies in observation that it is possible to successfully transfer FHL tendon to a posttraumatic foot drop. We were unable to identify any such description in the literature. Traditionally, the patient with nonfunctioning TP would be offered an arthrodesis.

CONCLUSIONS

Flexor hallucis longus can be used for restoring dorsiflexion in a foot drop without functioning *tibialis posterior*.

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