Surgical correction of checkrein deformity following malaligned distal tibia fracture

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INTRODUCTION

Checkrein deformities are rare and involve entrapment or fixed tethering of the flexor hallucis longus (FHL) tendon in the posterior foot, just proximal to the flexor retinaculum of the ankle (1-3). The deformity occurs secondarily to non-surgical or surgical correction of fractures of the distal tibia and has been observed after fibular, calcaneal, and talar fractures or the removal of fibular grafts (1-4). Radiographic imaging has suggested that the deformity can result from entrapment of the FHL in callus formation at a fracture site or within scar tissue (5). In general, deformities present with flexion contracture at the interphalangeal (IP) joint of the hallux with extension contracture of the metatarsophalangeal joint and occasionally affect the second and third toes. Complications of compartment syndrome can also cause contracture of the deep posterior muscle compartment and produce a fixed length phenomenon of the long toe flexors inducing the checkrein deformity (6). Pain related to the deformity is normally elicited in the postero-medial ankle upon palpation of the FHL at the midfoot without release of adhesions (5). A paucity of information exists on this rare deformity and available surgical techniques used for correction. The aim of this study was to report the etiology of a checkrein deformity case, describe the method of treatment, and provide and compare operative results of the case with other treatment modalities in the literature.

CASE STUDY

A 19-year old male presented at a foot and ankle clinic with a flexion deformity at the IP joint of his left hallux and proximal IP joints of the left second and third toes. The deformity had progressed over the past five years since treatment of a distal tibia fracture, which had healed in a malaligned position. Patient denied sudden lock up', snapping sensation, clicking sensation, 'catching' during movements, feeling of instability, joint looseness, or feelings of displaced joints in anterior toes. The patient was unable to actively straighten the deformed joints, and admitted to occasional pain upon palpation and ambulation. Stiffness was noted in the left great toe joint and left second toe joint. The IP joint of the hallux as well as the proximal IP joint of second and third toes were only partially reducible with passive reduction. The flexion deformity was enhanced with dorsiflexion of left ankle and was partially corrected with plantarflexion. All remaining joints of left foot and ankle maintained normal range of motion. No neurological deficits were noted upon examination and no visible swelling of left forefoot.

Bilateral foot and ankle radiographs were obtained, with left foot lateral radiograph (Fig.1) demonstrating hallux IP dislocation and hammering at second digit. Clinical examination of non-weight bearing left foot (Fig.2) illustrated a checkrein deformity while loaded foot when dorsiflexed (Fig.3) revealed increased deformity. Surgical correction of deformity was explained to patient and parent as well as risks and benefits of surgical procedures. Both parties agreed and elected to proceed with surgical intervention.

The tendon was transected and retracted giving rise to the joint. All cartilaginous surfaces were removed from joint and it was reduced (Fig. 5). A 4.0, 46 mm cannulated screw was placed and retrograded back down through toe providing compression. Light fluoroscopy demonstrated appropriate screw placement and position of toe. Extensor tendon was re-approximated with fibrotire and skin was closed in layers. Post closure the forefoot was loaded (Fig. 6) demonstrating correction of the checkrein deformity. An ankle block was administered using 30cc of 0.5% bupivacaine hydrochloride and foot and ankle was wrapped in a Robert Jones compression dressing. Patient was transferred to a Post Anesthesia Care Unit with stable vital signs and vascular status intact.

LITERATURE REVIEW

The FHL muscle belly arises from the inferior two-thirds of the posterior surface of the fibula and the interosseous membrane (7). Since Cloward (1974) first described claw toes following fractures of the tibia, checkrein deformity has been observed in other circumstances (8). Typical causes for tendon entrapment include, but not limited to, talar fractures, calcaneal fractures, and ankle fractures. These fixed flexion deformities of the hallux cause significant pain for patients, particularly when the ankle is passively dorsiflexed, causing the deformity to become prominent and rigid (6). Due to the rarity of this deformity, few reports have been cited in the literature. The reported case described a malaligned tibia fracture resulting in a progressing deformity requiring surgical correction. Currently there is little available information in the literature on etiologies and standard treatments of deformity.

Compartment syndrome has been reported as an etiology for developing clawing of the toes from delayed or inadequate treatment with decompression. Feeley et al. (6) reported contracture of multiple digits in nine patients, of which only one had an acute compartment syndrome present. The absence of any contracture of tibialis posterior in the case suggested the etiology may not have been related to compartment syndrome. The surgical treatment involved lengthening the FHL alone in four of the patients, and the FHL and flexor digitorum longus (FDL) in the remaining five patients through a retromalleolar incision. All patients had full correction of the deformities with no recurrence. Follow-up at 12 months or longer revealed all patients reported relief from pain, ease of shoe-fitting and subjective improvement of gait. Inter-tendinous connection between the FDL and the FHL ultimately determines whether lengthening the FHL alone is sufficient. Feeley et al. suggested lengthening the FHL first if clawing of the hallux is present, and if there is persistent clawing of the lesser toes on maximum passive ankle dorsiflexion, then lengthen FDL additionally (6).

Lee et al. (5) reported on surgical repair of 11 patients with checkrein deformities of hallux, providing the largest available patient sample in the literature. Five patients underwent release of adhesions with Z-plasty lengthening at the musculotendinous junction above the ankle at fracture site, and the remaining six had lengthening of the FHL in midfoot. Of these five patients with lengthening sites above the ankle, three patients had a recurrence and one required a secondary operation. No patients in the midfoot group had recurrence. At final follow-up of 20 months, 9 out of 11 patients had lost some flexion of the IP joint, but without significant loss of function. Though the patient groups in the study were small, authors suggested lengthening the FHL tendon by Z-plasty in midfoot, due to a relatively simple incision with less chance of adhesions recurrence.

These authors assert that release of adhesions can improve movement but tendon lengthening or release needs to be performed to correct deformity. Tenotomy of flexor tendon at the ankle may be an option to treat checkrein deformity, particularly in recurrent cases (9). Some authors suggest soft tissue correction distal to the retromalleolar level or arthrodesis on individual digits does not correct the deformity (5,6), however there is no evidence-based studies to substantiate these claims. Due to the rarity of the deformity, single surgical technique has been defined as standard. In the presented case, the checkrein deformity developed after a distal tibia ankle fracture that healed malaligned, which resulted in adhesions of the FHL and FDL to the callus of the tibial fracture. The case received flexor tenotomy to digit 1 and IP arthrodesis to hallux. Immediate correction of deformity was achieved intraoperatively. As of two months post-surgery the patient had no recurrence of deformity or complaints.

CONCLUSIONS

• Checkrein is a rare deformity caused by traumatic injury to the posterior aspect of the foot and ankle leading to impingement of FHL and occasionally FDL tendons.

• Previous surgical corrective intervention have been described in the literature as a release of adhesions associated with or without Z-plasty lengthening of tendons in midfoot and supramalleolar region.

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