Claw toes after tibial fracture in children

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Abstract

Purpose The development of claw toe deformity following fracture of the tibia in children has not been described in our review of the literature. We report on the management of the acquired claw toe deformity after tibia fracture in five children.

Methods We report on five patients, between 5 and 15 years of age, who developed clawing of the hallux following a fracture of the tibia. In two patients, the lesser toes were involved. On examination, when the ankle was passively plantar flexed, a flexion contracture of the interphalangeal joint of the hallux became fully flexible. When the ankle was dorsiflexed, the clawing became more obvious and fixed. A magnetic resonance imaging (MRI) study in two cases demonstrated fibrosis under or just proximal to the tarsal tunnel.

Results The contractures were relieved by performing a tenolysis proximal to the medial malleolus. The operative findings demonstrated that the etiology could be possibly associated with a localized subclinical compartment syndrome.

Conclusions We described five patients with a claw toe deformity following a tibia fracture associated with adhesions of the flexor hallucis longus (FHL) and flexor digitorum longus (FDL) muscles to the surrounding structures under or just proximal to the flexor retinaculum. It is the

authors' opinion that this condition may be related to a subclinical compartment syndrome localized in the distal part of the deep posterior compartment. Soft-tissue release without tendon lengthening allowed recovery in all patients.

Keywords Claw toes · Tibia fracture · Compartment syndrome

Introduction

Claw toe deformation has been widely described after compartment syndrome of the foot in children and adults [1–3]. Acquired clawing of the toes can also occur after compartment syndrome of the deep posterior muscles of the leg, which produces a fixed length phenomenon of the long flexors of the toes. If the tibialis posterior is affected, its contracture may cause pes cavus deformity [4].

The acquired clawing of the toes may also be the consequence of a fracture of the distal tibia [5] or fibula [6], in which there is an entrapment of the flexor hallucis longus (FHL) and flexor digitorum longus (FDL) in the fracture callus. A fixed tethering of the FHL tendon under or just proximal to the flexor retinaculum a few months after a fracture of the lower quarter of the tibia has also been described [5, 7]. In these situations, the principal manifestation is a flexion contracture of the interphalangeal (IP) joint of the hallux, in which plantar flexion of the ankle partially corrects the deformity and dorsiflexion increases it.

We report on five patients, less than 16 years of age, with claw toe deformities developed after tibia fracture in the absence of any evidence of a deep posterior leg or foot compartment syndrome. In addition, there was no evidence of any entrapment of the flexor tendons in the fracture site.

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Materials and methods

Between 1996 and 2006, 320 children were treated at our institution for fractures of the tibia. These fractures were primarily treated with a closed reduction and long leg cast application. Depending on the type of the fracture and associated trauma, some of these fractures underwent intramedullary nailing or an open reduction with internal or external fixation. Following the healing of the fracture, five patients presented with painful claw toes and increasing pain and pressure on the tip of the hallux with discomfort during shoe wear. There were three females and two males, with a mean age of 10.4 years (range, 5–15). All five patients sustained a fracture of the tibia, three in the middle third, one at the distal metaphyseal region, and one patient sustained a Type II Salter-Harris fracture pattern of the distal physis. In three cases, the fractures were Type I open fractures. These three patients were treated surgically with debridement and osteosynthesis, with external fixation in two and internal fixation in one. The remaining two patients underwent closed reduction and immobilization with a long leg cast. One patient had an obvious acute anterior compartment syndrome of the leg, but the posterior and deep posterior compartments, when measured, demonstrated normal compartment pressure. This patient was treated by fasciotomy of the anterior and lateral compartments at the same time as fracture reduction. The mean onset of clawing was at 7.2 months following the initial injury. All patients had a clawing of the hallux and two had a variable number of affected toes.

On examination, a flexion contracture of the hallux interphalangeal joint was seen (Fig. 1). When the ankle was passively plantar flexed, the deformity became fully flexible and completely corrected. When the ankle was dorsiflexed, the clawing became more obvious and fixed. A magnetic resonance imaging (MRI) study of the ankle was performed on two of the patients. In addition, two patients underwent an electromyography (EMG) of the foot (Table 1). The two MRIs were performed in order to localize the area of adhesion between the flexor tendons and the surrounding structures. The EMGs were performed in order to assess a potential entrapment of the posterior tibial nerve.

The patients were initially treated for at least 3 months by physiotherapy with stretching. Because of failure of this conservative method, they were referred for a surgical treatment.

Operative technique

General anesthesia and a tourniquet was used in all cases. The long flexor tendons were approached through an incision along the posteromedial border of the distal tibia, which extended distally along the retromalleolar region,

Fig. 1 Flexion contracture of the hallux interphalangeal joint

if necessary. The superficial posterior compartment was incised and the soleus was detached from the tibia, exposing the deep compartment aponeurosis.

The neurovascular bundle was protected from injury. The FHL and FDL could be identified and dissected free. All patients showed strong adhesions between the tendons and tibia proximal or within the tarsal tunnel. In cases with distal tibia fracture, the area of adhesions was below the fracture level. The FHL had significant fibrosis in three cases and demonstrated a necrotic aspect in two (Fig. 2). At the proximal part of the wound, above the zone of adhesions, the muscle bellies demonstrated normal anatomy. A biopsy was made at this level in one case. None of the flexor tendons were entrapped in callous at the fracture site.

After the FHL and FDL were dissected free, the hallux metatarsophalangeal and interphalangeal extension was restored and could be held in extension with the ankle in maximum dorsiflexion. Z-lengthening of the involved tendon was not necessary.

A walking cast with an extended sole to maintain extension of the toes was applied for 4 weeks and physiotherapy was starting during the immobilization period.

Results

In both of the preoperative MRI studies, fibrosis was demonstrated around the FHL and FDL under or just proximal to the flexor retinaculum. In neither of the EMGs performed, there were no signs of posterior tibia nerve compression demonstrated. The biopsy performed above the zone of adhesion demonstrated a normal histology of the muscle.

Three patients underwent release of the FHL alone and two required release of both the FDL and FHL (Table 1). After 18 months follow-up (range, 12–40), all patients

Table 1 Summary of case histories

Case	Gender	Age at the time of tendon surgery (years)	Tibial fracture	Fracture treatment	Compartment syndrome	Onset of clawing after initial trauma (months)	MRI	EMG	Tendons release
1	F	15	Closed Salter-Harris II	Open reduction	No	3	Yes	No	FHL + FDL
2	M	12	Open Distal 1/3	Closed reduction	No	12	Yes	No	FHL
3	M	5	Open Middle 1/3	External fixation	No	5	No	Yes	FHL
4	F	8	Closed Middle 1/3	External fixation	No	4	No	Yes	FHL + FDL
5	F	12	Open Middle 1/3	External fixation	Yes (anterior)	12	No	No	FHL

Fig. 2 Necrosis of the distal portion of the flexor hallucis longus (FHL) (arrow) proximal to the flexor retinaculum **Fig. 3** Postoperative active hallux metatarsophalangeal and interphalangeal joints extension

reported relief from pain, improvement of gait, and greater ease with shoe fitting. There were no complications. Active metatarsophalangeal and interphalangeal joints extension

with the ankle dorsiflexed was restored in all cases (Fig. 3). There were no recurrences of the deformity at the final follow-up.

Discussion

To our knowledge, claw toes after tibia fracture in children have not been described in the literature. Clawing of the toes is usually associated with neurologic conditions in which there is weakness or loss of intrinsic muscle function and a cavus foot. There are two additional causes of clawing following leg fracture:

1. Entrapment of the flexor tendons in callus [5, 6, 8] or scarring of the muscle [7] at the fracture site. Rosenberg and Sfera [5] reported a case of tethering of the FHL tendon (called checkrein deformity) and rupture of the posterior tibialis tendon after a closed Salter-Harris Type II ankle fracture. An MRI scan demonstrated tethering of the FHL and FDL tendons in the posterior tibial fragment. Leitschuh et al. [6] reported a case of hallux flexion deformity secondary to entrapment of the FHL tendon after fibular fracture. In this case, the adhesion of the FHL musculotendinous unit to the fibula fracture callus and interosseous membrane resulted in a fixed flexion contracture of the hallux. Jahss [7] noted that the hallux may become clawed after fractures of the ankle and distal tibia because of tethering of the FHL tendon under or just proximal to the flexor retinaculum.
2. The presence of a deep posterior compartment syndrome of the leg [9]. Clawing of the toes is usually associated with contracture of the tibialis posterior and pes cavus may develop [10, 11].

The development of a compartment syndrome of the leg needs a prompt diagnosis and fasciotomy in acute cases

[12, 13]. The peak pressure is usually found at the level or within 5 cm of the fracture site [14]. However, little has been written about minor or borderline compartment syndrome [15].

Four of our patients showed no clinical evidence of compartment syndrome of the leg. One was treated for an anterior compartment syndrome. The intraoperative findings, however, demonstrated either a localized fibrosis or a necrotic aspect of the distal part of the FHL muscle. Proximal to this area, the muscle was visually normal and a biopsy in one case confirmed this. For most of our cases, the best explanation for these findings was that they had developed a subclinical localized deep posterior compartment syndrome at the distal portion of the FHL or FDL muscles. As the superior border of the flexor retinaculum is not clearly demarcated [16], a localized increase in pressure can result in ischemic lesion of the distal part of flexor muscle bellies. These are located, in some patients, under the proximal part of the flexor retinaculum. As only a small portion of the muscle is involved, there is no retraction of the main muscle belly, yet, the necrotic part can become fibrotic and adhere to the surrounding structures. The immobilization of the fibrotic portion during the post-trauma period immobilization can further reduce muscle and tendon mobility. This localized involvement of the muscle itself can explain why a simple tenolysis without tendon lengthening improved the condition in our patients. Feeney et al. [15] also reported ten adults who acquired claw toes deformity. Eight of them had tibial shaft fractures, with only one who had evidence of a compartment syndrome. In order to prevent the occurrence of these deformities, early postoperative physiotherapy with passive stretching should be performed, especially in cases with high-energy trauma.

An alternative explanation could be that the condition was caused by tethering or scarring of the muscle bellies at the site of the fracture [7]. This mechanism, however, could explain the etiology of the distal tibial fracture in our series. It does not account for the claw deformities in the middle third.

For all of our patients, clawing became more obvious and fixed when the ankle was dorsiflexed. This explained why the symptoms occurred during the stance phase of the gait cycle. When the ankle is dorsiflexed, the hallux cannot dorsiflex to allow the normal rocker movement of the metatarsophalangeal joints.

Two patients showed clawing of the lesser toes. There are two explanations for this condition. The first is that the FDL was included in fibrosis tissues, with the FHL within or proximal to the tarsal tunnel. The second is the presence of tendinous connections from the FHL to FDL. Feeney et al. [15] showed improvement of the clawing of the lesser

toes by lengthening of the FHL tendon alone at the level of the medial malleolus.

Conclusion

We have described five patients with claw toe deformity following a tibial fracture due to adhesions of the flexor hallucis longus (FHL) and flexor digitorum longus (FDL) muscles with surrounding structures under or just proximal to the flexor retinaculum. The main clinical feature was an increase of the clawing with the ankle in dorsiflexion, while plantar flexion improved it. One explanation could be that the patients had developed a subclinical compartment syndrome localized in the distal part of the deep posterior compartment. Magnetic resonance imaging (MRI) can be helpful to confirm the site of the adhesions. Soft-tissue release without tendon lengthening allowed recovery in all patients.

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