



# Management of Checkrein Deformity

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Checkrein deformity is characterized by the dynamic status of the hallux, in which flexion deformity is aggravated by ankle dorsiflexion and relieved by ankle plantarflexion. In most cases, a checkrein deformity occurs secondary to trauma or following surgery. It has been suggested that the flexor hallucis longus tendon tethers or entraps scar tissue or fracture sites. Improvement with conservative treatment is difficult once the deformity has already become entrenched, and surgical management is usually required in severe cases. Various surgical options are available for the correction of checkrein deformities. It includes a simple release of adhesion at the fracture site; lengthening of the flexor hallucis longus by Z-plasty at the fracture site combined with the release of adhesion; lengthening of the flexor hallucis longus by Z-plasty at the midfoot, retromalleolar, or tarsal tunnel area; and flexor hallucis longus tenotomy with interphalangeal arthrodesis for recurrent cases. This review aimed to summarize the overall etiology, relevant anatomy, diagnosis, and treatment of checkrein deformities described in the literature.

**Keywords:** Foot deformities, Hallux, Tendon entrapment

Checkrein deformity is an uncommon dynamic status of the hallux characterized as an aggravation of interphalangeal flexion by ankle dorsiflexion and relief of the deformity by ankle plantarflexion.<sup>1)</sup> The origin of the word “checkrein” comes from the way the horseman controls the horse, where “check” means to control and “rein” refers to a ring-shaped device that fits into the horse’s mouth. When the horseman pulls the rope, the horse’s neck is pulled, the morphology of which is similar to that of the flexed hallux when the ankle is dorsiflexed. Clawson first described the observation of claw toes following a tibial fracture.<sup>2)</sup> The flexor hallucis longus (FHL) tendon often adheres to the callus or scar tissue of the fracture after trauma. Patients may complain of discomfort when wearing shoes, have a wound on the dorsal area of the toes, or tend to trip. As

conservative treatments such as stretching are limited in significantly improving symptoms, surgical treatment is more often used. Although a few case series and case reports have reported the results of surgical treatment, there is a lack of a comprehensive review of checkrein deformities. This review aimed to summarize the overall etiology, relevant clinical anatomy, diagnosis, and treatment of this dynamic deformity described in the literature.

## ETIOLOGY

Due to its rarity, previous studies have introduced individual case reports and small case series. In most cases, checkrein deformities occur secondary to trauma, such as malleolar fracture,<sup>3,4)</sup> talus,<sup>5)</sup> calcaneus,<sup>6)</sup> or lower leg fracture.<sup>2,7,8)</sup> Previous studies reported that such deformities occur approximately 6 months after trauma.<sup>9)</sup> In addition, in some cases, deformities can occur after dislocation without fractures. Tanwar et al.<sup>10)</sup> reported a rare case of closed lateral subtalar dislocation with checkrein deformity of the hallux due to entrapment of the FHL tendon. Another study reported the occurrence of deformity caused by an incarcerated posterior tibial tendon and displaced FHL tendon following ankle dislocation without fracture.<sup>11)</sup>

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Deformities can occur after surgery, such as in fibular bone grafts. Feng et al.<sup>12)</sup> reported a case of checkrein deformity after fibular osteotomy for autogenous bone grafting for mandible surgery. Sallent et al.<sup>13)</sup> also reported two cases due to a fibular graft harvesting for the reconstruction of mandibular bone defect secondary to oral cancer treatment. When harvesting the fibular bone, the middle one-third part is typically used, leaving at least 4–6 cm from the fibular head and a minimum of 6 cm from the ankle joint.<sup>14)</sup> This area is susceptible to FHL muscle belly injuries and hematoma formation during the lateral approach, which may induce increased compartment pressure and eventually scarring adhesion leading to checkrein deformity.<sup>12)</sup>

It has been suggested that the deformity is a consequence of (1) entrapment of the FHL between calli at the fracture site,<sup>15)</sup> (2) tethering of the FHL within the scar tissue,<sup>16)</sup> (3) scarring at the musculotendinous junction,<sup>17)</sup> and (4) fibrotic contracture after subclinical compartment syndrome.<sup>9)</sup> However, it can be difficult to identify the exact cause of the deformity in each case.

Furthermore, in cases of equinus deformity caused by foot drop or Achilles contracture, checkrein deformity may appear due to the shortening of the deep compartment flexor tendons following Achilles tendon lengthening. Costa et al.<sup>18)</sup> demonstrated a linear relationship between the length of the Achilles tendon and the range of ankle dorsiflexion in a cadaveric study. Thus, when the Achilles tendon is lengthened and ankle dorsiflexion range of motion is increased, a newly developed checkrein deformity should be checked and treated accordingly.

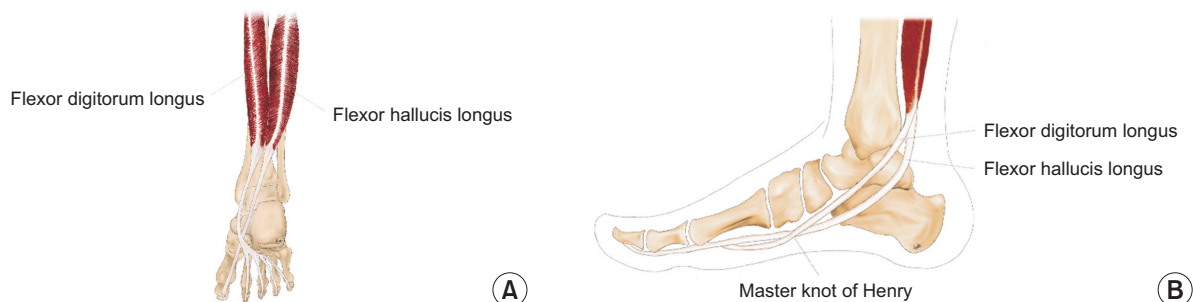
In summary, checkrein deformities can occur following trauma to the lower extremities or surgical procedures around the ankle. Therefore, it is important to closely observe patients during the follow-up period and inform them about the possibility of such a condition occurring.

## ANATOMY

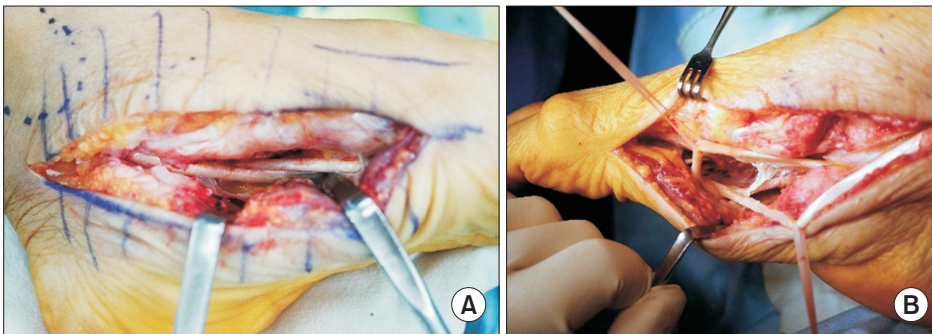
The FHL is a deep-compartment muscle of the lower leg (Fig. 1A). It originates from the distal half of the posterior part of the fibula, passes inferomedially over the posterior distal tibia and grooves posterior to the medial malleolus, and inserts at the base of the distal phalanx.<sup>19)</sup> Its main action is great toe flexion. The tendinous portion starts just above the posterior aspect of the medial malleolus and is located posterolateral to the tibialis posterior and flexor digitorum longus (FDL) tendons. Meanwhile, FDL is another deep-compartment muscle in the lower leg. It originates from the distal tibia medial to the FHL origin, splits into four tendons in the midfoot, and inserts into the bases of the second to fifth distal phalangeal bases.<sup>20)</sup> The main action is flexion of the lesser toes.

The FHL can easily adhere to the callus at the fracture site, especially the distal tibia or sometimes the fibular fracture site.<sup>4)</sup> Furthermore, the FHL is more commonly involved because the muscle bulk of the FHL is larger than that of the FDL behind the distal tibia.<sup>3)</sup> The tendon of the FHL forms a bowstring structure between this site and the insertion site of the tendon in the great toe.<sup>12)</sup> Therefore, when tendon entrapment occurs, the distal phalanx base is bent, resulting in claw toe deformity.

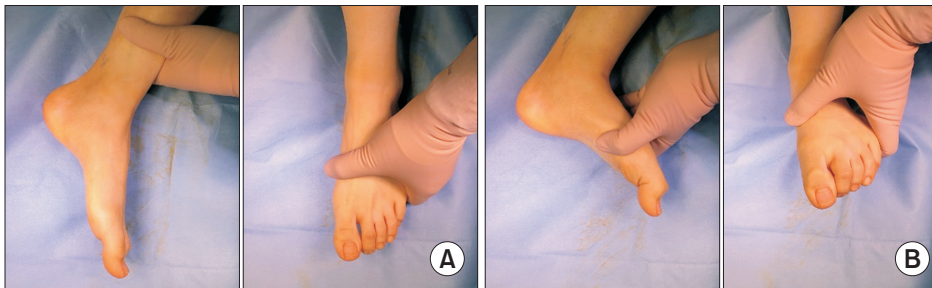
Distal to the sustentaculum tali, the FHL and FDL tendons cross at the site called “the master knot of Henry” (Figs. 1B and 2A). This chiasmatic structure was first described by Henry<sup>21)</sup> in 1970. In the vicinity of the knot, interconnections between FHL and FDL often exist (Fig. 2B), where Beger et al.<sup>22)</sup> reported various types of interconnections in a cadaveric study. Of seven different types, type 1, which is one slip from FHL to FDL, accounted for the majority (75%). In another cadaveric study by Plaass et al.,<sup>23)</sup> they also reported that 67% of the specimen showed tendon slip branching proximally from the FHL to the distal FDL. Owing to this anatomically interconnected structure, shortening of the FHL may also lead to checkrein deformity of the lesser toes.



**Fig. 1.** (A) Anatomy of the flexor hallucis longus and flexor digitorum longus tendon. (B) Master knot of Henry.



**Fig. 2.** Master knot of Henry (A) and nearby interconnection between the flexor hallucis longus and flexor digitorum longus tendon (B).



**Fig. 3.** Dynamic checkrein deformity of the hallux during ankle plantarflexion (A) and dorsiflexion (B).

## DIAGNOSIS

The diagnosis of a checkrein deformity is usually made by a thorough history taking and physical examination. Patients should be asked about a history of fractures that could potentially lead to tethering or entrapment of the FHL or FDL tendons, compartment syndrome, and operations such as Achilles tendon lengthening. Patients may complain of toe clawing during gait and pain at the distal ends of their toes. Pain in the posteromedial ankle may develop by ankle or hallux dorsiflexion, which induces FHL tension.<sup>5)</sup>

The pathognomonic sign of the checkrein deformity is as follows: when the ankle joint is passively dorsiflexed, the interphalangeal flexion deformity of the hallux becomes more prominent and is not extended by passive force. However, when the ankle joint is plantarflexed, the flexion deformity is completely corrected (Fig. 3). As described in the anatomy section previously, flexion deformity of the lesser toes may become more visible when the ankle is dorsiflexed owing to the interconnection between the FHL and FDL.

Mallet toe, hammer toe, and claw toe should be considered as differential diagnoses for the lesser toe. The mallet toe involves the distal interphalangeal joint and is fixed in a flexed state while sparing the metatarsophalangeal joint. By contrast, the hammer toe mainly involves the proximal interphalangeal joint and is in a flexed state. Flexion deformities can be in a fixed state; however, in

some cases, they can be manually corrected. If the proximal interphalangeal joint deformity is corrected while dorsiflexing the ankle joint by lifting the plantar side of the metatarsal head with a finger (push-up test), it is considered a flexible hammer toe. However, this is in the opposite direction to confirming the checkrein deformity described above. For the claw toe, the metatarsophalangeal joint is in a hyperextended position, while the proximal and distal interphalangeal joints are in a fixed flexion position. Therefore, clinicians should keep in mind that a checkrein deformity is a dynamic deformity. During physical examination, it is essential to not only assess the curled toes but also manipulate the ankle joint.

Therefore, clinical diagnosis is mostly achievable through physical examination, and the role of imaging modalities is considered limited. However, computed tomography (CT) and magnetic resonance imaging (MRI) may help identify the causative trauma or surgical history. Using CT images, entrapment of the FHL tendon near the callus at the fracture site can be easily identified.<sup>15)</sup> MRI images can visualize the contracture of the FHL and FDL tendons and the status of the FHL muscle itself.<sup>7)</sup> In addition, the electrical activity of an isolated muscle can be demonstrated by electromyography.<sup>24)</sup>

## TREATMENT

For the treatment of checkrein deformity, a conservative approach can be initially attempted, and one method is

passive stretching exercise. In addition, a patient-adjusted insole or toe brace can be used to alleviate some of the symptoms.<sup>9)</sup> Feng et al.<sup>12)</sup> reported a case of deformity following fibular osteotomy treated conservatively with 20 days of comprehensive rehabilitation therapy. Comprehensive rehabilitation therapy includes thermal therapy, medium-high-frequency electrotherapy, shock wave therapy, and manual stretching of the tendons of the lower leg. The authors insisted that rehabilitation therapy effectively alleviated the pain and improved the walking ability of the patient. Whether stretching exercises during rehabilitation after trauma can prevent checkrein deformity is not clearly understood. It is difficult to improve with conservative treatment once the deformity has already been entrenched, and surgical management is usually required for severe cases.

Various surgical options are available for the correction of checkrein deformities. It includes (1) simple release of adhesion at the fracture site,<sup>4,5,25)</sup> (2) lengthening of FHL by Z-plasty at the fracture site combined with release of adhesion,<sup>7,11)</sup> (3) lengthening of FHL by Z-plasty at the midfoot without release of adhesion,<sup>1,3,26)</sup> (4) lengthening of FHL by Z-plasty near retromalleolar or tarsal tunnel area,<sup>9,27,28)</sup> and (5) FHL tenotomy with interphalangeal arthrodesis for recurrent cases.<sup>8)</sup> Reviews of previous studies on each surgical method are as follows.

First, releasing the adhesions at the site that caused the deformity is an option. Leitschuh et al.<sup>4)</sup> reported a case where the FHL was entrapped at the musculotendinous junction in a fracture callus after operative treatment of an ankle fracture. When the adhesion was released by the medial approach, the interphalangeal joint motion of the hallux was recovered with the ankle in the neutral position, which was 8 months from the initial fracture surgery. Another case report by Rosenberg and Sferra<sup>25)</sup> demonstrated that the FHL and FDL tendons were encased in dense scar tissue, deep within the crevices of the healed distal tibial fracture, 7 months after the initial injury. Only after the tendons were completely freed of their adhesions were the hallux and second toe flexion deformities significantly improved. The other case was the release of entrapment of the FHL tendon in the posterior portion of the talus, proximal to the sustentaculum tali, after the talar fracture.<sup>5)</sup> The dynamic deformity resolved 8 weeks after the initial injury. While the solution seems intuitive, approaching the fracture site may require a large incision and dissection and is prone to neurovascular structures, which may result in secondary adhesion.

Second, if the simple release of adhesions at the fracture site is insufficient, it can be resolved by performing

additional FHL Z-plasty. Yuen and Lui<sup>7)</sup> reported a case of adhesion of the FHL at the site of a tibial shaft and demonstrated that open exploration and adhesiolysis at the adhesion site together with tendon lengthening at the distal tibial level is a feasible surgical option with satisfactory results. Bae et al.<sup>11)</sup> also reported a case of checkrein deformity caused by an incarcerated posterior tibial tendon and displaced FHL tendon following ankle dislocation. They insisted that the checkrein deformity persisted even after the release of the adhesion, and the reason was thought to result from the fibrosis of the FHL tendon. Therefore, the patient was treated by excision of the incarcerated posterior tibial tendon, adhesiolysis, and lengthening of the FHL tendon by Z-plasty, followed by reconstruction of the deltoid ligament and transfer of the FDL tendon.

Third, previous studies have reported good results and merits of lengthening the tendon at the midfoot level.<sup>1,3,26)</sup> The reason many authors prefer surgery on the midfoot is that surgical fields are technically simple and free of scar tissue and recurrence rates are low.<sup>3)</sup> According to a study by Sanhudo and Lompa,<sup>1)</sup> they did not need to approach the medial aspect of the ankle joint, thereby avoiding the neurovascular structures. In one of the largest case series by Lee et al.,<sup>3)</sup> 6 patients who underwent lengthening of the FHL tendon by Z-plasty in the midfoot showed complete correction of the deformity without recurrence. Meanwhile, 5 patients who underwent adhesiolysis and FHL lengthening above ankle level showed two partial and one complete recurrence.<sup>3)</sup> In addition, a recent study by Gadhavi et al.<sup>26)</sup> suggested that exploration at the midfoot should be the primary surgical intervention due to the ease of surgical correction through an operative field free of scar tissue.

When correcting procedures are performed in the midfoot, it is important to identify the intertendinous connection between the FHL and FDL tendons. Lengthening of the FHL tendon alone may not be sufficient, and separation of the connection between the FHL and FDL is necessary for correcting the lesser toes.<sup>27)</sup> If persistent clawing of the lesser toes occurs at maximum ankle dorsiflexion, even after separating the intertendinous connection, additional lengthening of the FDL tendon is required.

Fourth, case series have reported good clinical outcomes after performing FHL Z-plasty lengthening at the tarsal tunnel or retromalleolar area, which is more proximal to the midfoot. A recent case series by Polichetti et al.<sup>28)</sup> demonstrated that 14 patients with checkrein deformity were treated successfully without recurrence by Z-plasty lengthening of the FHL tendon at the tarsal tunnel. Although they admitted that the method faces a greater

risk of recurrence due to adhesion and the need for meticulous dissection to prevent injury to the neurovascular bundle, simultaneous correction of the deformities of the big toe and lesser toes is achieved. In addition, a previous case series by Lee et al.<sup>27)</sup> showed that Z-plasty of the FHL was performed at the tarsal tunnel in 8 patients, and successful results without recurrence were reported. They noted that the FHL tendon in the tarsal tunnel was surrounded by the tendon sheath and far from the injury site. These anatomical structures may decrease the likelihood of adhesion to adjacent tissues. Furthermore, they insisted that more powerful walking is possible when Z-plasty is performed at the tarsal tunnel area because the FHL tendon is connected after Z-plasty rather than being torn by tenotomy at the base of the toe.

Finally, in cases of recurrent deformities, FHL tenotomy followed by interphalangeal arthrodesis may be considered. Holcomb et al.<sup>8)</sup> reported a case that underwent operation by flexor tenotomies for digits 1 through 3. In addition, a 4.0-mm cannulated screw was inserted to fuse the interphalangeal joint of the hallux. They insisted that there was no recurrence and no complaints reported. However, caution is required because this method completely sacrifices the motion of the interphalangeal joint. Therefore, it is necessary to identify the appropriate indications, such as recurrent cases, and apply this method accordingly.

## CONCLUSION

Checkrein deformity, predominantly arising from trauma or surgical procedures, presents a challenging scenario due

to its dynamic nature and the involvement of intricate anatomical structures like the FHL tendon. Selection among the array of surgical options depends largely on the patient's specific anatomy, the severity of the deformity, and any recurrent manifestations. Although this review offers a summary to facilitate a comprehensive understanding of checkrein deformity, from diagnosis to treatment, the rarity of the condition has resulted in most of the past studies being dominated by small case series and case reports. Therefore, future research endeavors might delve into refining surgical techniques with a sufficient number of patients and devising preventative strategies to reduce the risk of this deformity following trauma or surgical procedures.

## CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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