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Claw Toes: A Rare Complication Following Tibial Shaft Fracture

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Abstract

Dynamic claw toe deformity after a tibial shaft fracture classically manifests as a checkrein effect caused by isolated entrapment of the flexor hallucis longus. Combined involvement of the flexor hallucis longus and flexor digitorum longus causing multi-toe clawing in adults is rare and presents a diagnostic challenge, as static imaging may not identify the adhesions.

An 18-year-old female sports student sustained a tibial shaft fracture in a pedestrian-vehicle accident and was treated with intramedullary nailing. Two months after injury, she developed clawing of all toes, followed by plantar pain and hallux paresthesia. Physical examination revealed a strictly dynamic deformity that resolved completely with ankle plantarflexion and severely worsened during passive ankle dorsiflexion, alongside a positive Tinel's sign over the tarsal tunnel. Electromyography was inconclusive, and magnetic resonance imaging showed no clearly identifiable adhesion. Physical therapy and a trial of botulinum toxin injections provided no clinical improvement. Nine months after injury, surgical exploration revealed dense adhesions at the level of the fracture callus, between the flexor hallucis longus and flexor digitorum longus and predominantly between these muscles and the hypertrophic callus. Myolysis, tenolysis, and tarsal tunnel decompression achieved intraoperative restoration of full toe extension. Passive mobilization began on the first postoperative day. At six months, the deformity and symptoms had resolved, the overall score on the patient-reported Foot and Ankle Outcome Score questionnaire had improved from 52 to 89 out of 100 points, and the patient reported returning to sports.

Multi-toe clawing after a tibial shaft fracture may result from combined flexor hallucis longus and flexor digitorum longus adhesions. In this patient, the diagnosis relied primarily on dynamic physical examination, as magnetic resonance imaging did not show a clearly identifiable adhesion and electromyography was inconclusive. Myolysis, tenolysis, and tarsal tunnel decompression, performed nine months after injury and followed by passive mobilization from the first postoperative day, were associated with a favorable patient-reported outcome at six months.

Categories: Trauma, Orthopedics, Anatomy
Keywords: checkrein deformity, claw toes, intramedullary nailing, tendon entrapment, tenolysis, tibial shaft fracture

Introduction

Intramedullary nailing is a widely used treatment for displaced tibial shaft fractures [1,2]. However, it is associated with a spectrum of potential complications. Early sequelae include acute compartment syndrome and deep infection, whereas late complications typically encompass malunion, axial or rotational deformity, nonunion, anterior knee pain, and implant failure [2]. While most of these structural complications are routinely monitored during postoperative follow-up, the insidious development of muscular and tendinous adhesions within the lower leg compartments may be overlooked.

Checkrein deformity is an uncommon delayed manifestation following tibial shaft fractures [3]. Classically, it is described as an isolated flexion deformity of the hallux caused by mechanical tethering of the flexor hallucis longus (FHL) tendon within the deep posterior compartment [4,5]. Unlike a fixed claw toe, checkrein deformity is dynamic: toe flexion increases with ankle dorsiflexion and corrects with plantarflexion. Because the FHL and flexor digitorum longus (FDL) muscles lie in close anatomical proximity along the posterior aspect of the tibia, trauma at this level may result in adhesions involving both muscle-tendon units. However, combined FHL and FDL involvement causing dynamic clawing of all toes appears to be rare, with cases described in children [6] and few reports in adults [7], potentially leading to underdiagnosis and progressive gait impairment.

We report a delayed complication presenting with progressive clawing of all toes. The deformity was recognized before fixed contractures developed, allowing correction through soft-tissue release alone, without tendon lengthening or transfer. This report highlights the diagnostic challenges of multi-tendon involvement, the limitations of static imaging in detecting dynamic tendon tethering, and the importance of recognizing the deformity while it remains dynamic.

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Case Presentation

An 18-year-old female sports student was involved in a pedestrian-vehicle accident and sustained fractures of the left tibial and fibular shafts, classified as Arbeitsgemeinschaft für Osteosynthesefragen/Orthopaedic Trauma Association (AO/OTA) 42-B2 and AO/OTA 4F-2A (Figure *1*). On admission, distal pulses were palpable, the skin over the affected limb was intact, and there were no clinical signs of acute compartment syndrome. Compartment pressure measurements were not performed given the complete absence of clinical suspicion.



FIGURE 1: Anteroposterior radiograph of the left tibia and fibula fracture, classified as AO 42-B2 and AO 4F-2A.

AO: Arbeitsgemeinschaft für Osteosynthesefragen.

Within 24 hours of injury, the patient underwent closed reduction and reamed, locked intramedullary nailing of the tibia using a T2 nail. The immediate postoperative course was uneventful.

At follow-up two months after injury, wound healing and radiographic progression were satisfactory. However, clinical examination revealed the onset of a claw toe deformity affecting all toes of the left foot. Over subsequent follow-up visits, the patient progressively reported diffuse plantar pain and paresthesia extending from the hallux to the medial plantar aspect of the foot, which worsened with prolonged walking and improved with rest.

Physical examination demonstrated plantar callosities and interphalangeal flexion deformities of all toes with the ankle in a neutral position. The deformity was strictly dynamic; it resolved completely during ankle plantarflexion but became severely pronounced with passive ankle dorsiflexion (Figure *2*). Clinical assessment of the left ankle revealed a limitation in passive dorsiflexion, which was restricted to 10°. A positive Tinel's sign was elicited over the left tarsal tunnel. Motor strength was preserved (5/5) in all muscle groups of the left lower limb, with no deficit in the peroneal or tibial nerve territories. During follow-up, the patient developed a progressive gait disturbance characterized by an impaired toe-off phase and compensatory ankle mechanics during ambulation.

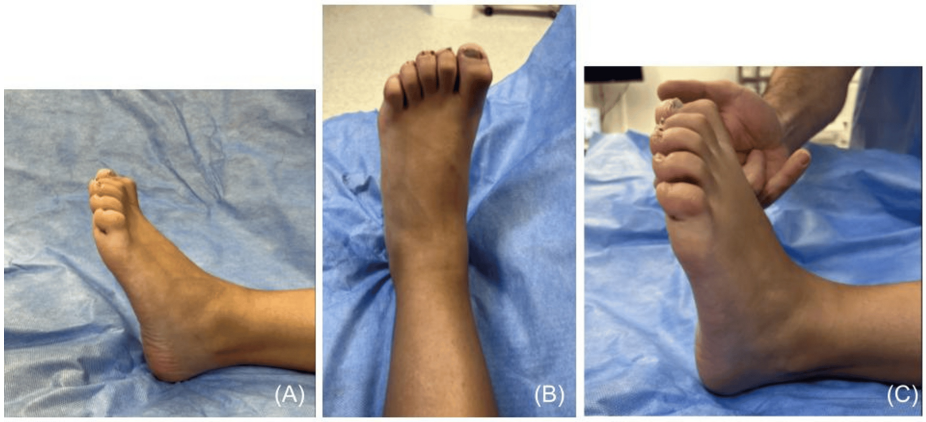


FIGURE 2: Claw toes of the left foot.

Lateral view in a neutral ankle position showing metatarsophalangeal extension and interphalangeal flexion (A); frontal view demonstrating multi-toe clawing (B); severe exacerbation of the deformity during passive ankle dorsiflexion (C).

Serial radiographs showed satisfactory fracture healing without implant-related complications (Figure *3A*, B). Because the deformity persisted, further diagnostic workup was performed six months after injury. Electromyography (EMG) of the left lower limb was inconclusive, suggesting possible lesions of the tibial nerve at the distal leg or tarsal tunnel and of the common peroneal nerve, and further investigation was recommended. Magnetic resonance imaging (MRI) of the lower leg was performed on a 1.5-T scanner and included axial T1-weighted, coronal T2-weighted turbo spin-echo, and axial and coronal fat-suppressed proton density-weighted sequences. It showed expected post-fracture changes and a healing callus, with no hardware protrusion into the deep posterior compartment. Edema-like signal in the deep flexor compartment was interpreted as a nonspecific post-traumatic change. Metal-related artifact partially limited assessment around the nail and fracture site. No clearly identifiable adhesion was observed, although this did not exclude adhesions (Figure *3C*-F).

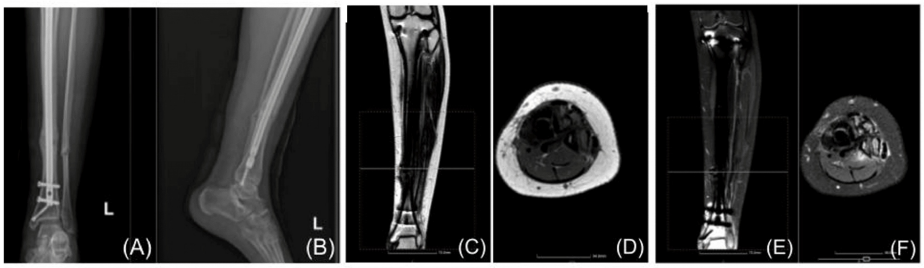


FIGURE 3: Left leg imaging.

(A) Anteroposterior and (B) lateral radiographs obtained nine months after injury showing fracture union. (C-F) Magnetic resonance images of the distal third of the left leg, at the level of the fracture callus, obtained six months after injury: (C) coronal T2-weighted, (D) axial T1-weighted, (E) coronal fat-suppressed proton density-weighted, and (F) axial fat-suppressed proton density-weighted images. The orange line indicates the level of the axial images. Edema-like signal is visible in the deep posterior compartment on the fat-suppressed images, without a clearly identifiable adhesion.

After the onset of the deformity, the patient underwent physical therapy, which continued until surgery. Approximately six months after the onset of the deformity, a trial of botulinum toxin injections into the affected muscles was performed without clinical improvement. There was no meaningful reduction in toe clawing during gait, and plantar pain persisted. Before surgery, the overall result of the patient-reported Foot and Ankle Outcome Score (FAOS) questionnaire was 52 out of 100 points. Given the significant impact on her daily activities as a sports student, surgical exploration was performed nine months after injury, seven months after the onset of the deformity.

Surgical intervention

Under general anesthesia and with a tourniquet applied at the proximal thigh, a longitudinal incision was made along the posteromedial border of the distal tibia, extending distally toward the retromalleolar region. This single incision was used to access the tarsal tunnel and, indirectly, the level of the fracture callus. The

superficial posterior compartment was incised, and the aponeurosis of the deep compartment was exposed and opened. The neurovascular bundle was carefully isolated and protected throughout the procedure.

The FDL and FHL muscles were identified. Dense adhesions were found along the length of the fracture callus, between the FHL and FDL muscles and predominantly between these muscles and the hypertrophic callus. The posterior tibial nerve was free of adhesions at the level of the callus. The FDL was directly exposed and released (Figure *4A*, C). The FHL, which lies deeper and more laterally, was released from the callus indirectly through the same approach. Both muscles and their respective tendons exhibited a normal macroscopic appearance. Intraoperatively, after complete release of the adhesions, a dynamic test was performed by placing the ankle in maximal passive dorsiflexion. Full extension of all toes was achieved, and the pre-existing dynamic contracture resolved completely. Consequently, isolated myolysis and tenolysis were deemed sufficient, and further tendinous lengthening (such as Z-plasty) was not required.

Distally, the flexor retinaculum within the tarsal tunnel was incised (Figure *4B*), followed by neurolysis of the posterior tibial nerve, given the plantar paresthesia and positive Tinel's sign over the tarsal tunnel. At the end of the procedure, the skin was closed using an intradermal suture technique, and the limb was immobilized with a Robert-Jones dressing.

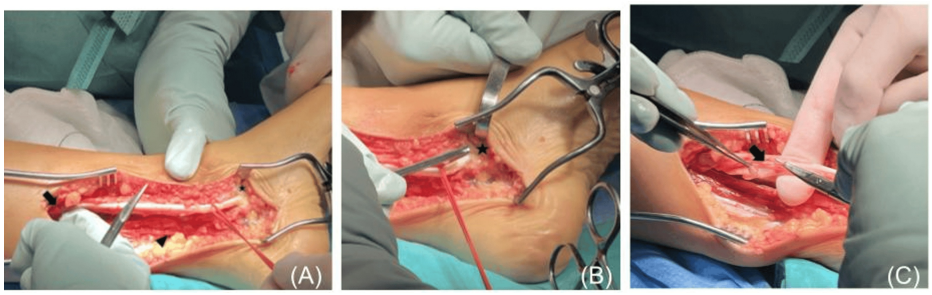


FIGURE 4: Posteromedial surgical approach to the left distal leg showing release of the flexor adhesions and tarsal tunnel decompression. The images show the distal part of the exposure (proximal to the left of the image); the release at the level of the fracture callus, located more proximally, is not directly visible.

(A) Exposure of the FDL muscle belly (black arrow) after release of the adhesions; the posterior tibial nerve is indicated by the arrowhead and the intact flexor retinaculum by the star. (B) Flexor retinaculum (star) immediately before release using a grooved director. (C) Isolated FDL tendon (black arrow) after release of the adhesions.

Postoperatively, the limb was protected with the Robert-Jones dressing for two weeks, while passive mobilization of the ankle and toes was initiated on the first postoperative day to prevent recurrent adhesions and preserve tendon gliding. Non-weight-bearing status was maintained during this initial two-week period. Full weight-bearing was then permitted, followed by progressive rehabilitation focused on range-of-motion recovery, gait retraining, and stretching of the long toe flexors.

Follow-up and outcomes

During follow-up visits, the patient demonstrated favorable wound healing. At the six-month clinical evaluation after surgery, the dynamic claw toe deformity had resolved, with all toes achieving neutral extension (0°) (Figure *5*). Ankle dorsiflexion remained limited to 10°, as before surgery, which was attributed to joint stiffness; however, on clinical observation, the patient demonstrated a normalized gait pattern with an effective toe-off phase and reported no functional limitations (Figure *6*).



FIGURE 5: Postoperative clinical appearance of the left foot at six months.

Frontal (A) and lateral (B) views showing neutral toe extension (0º).

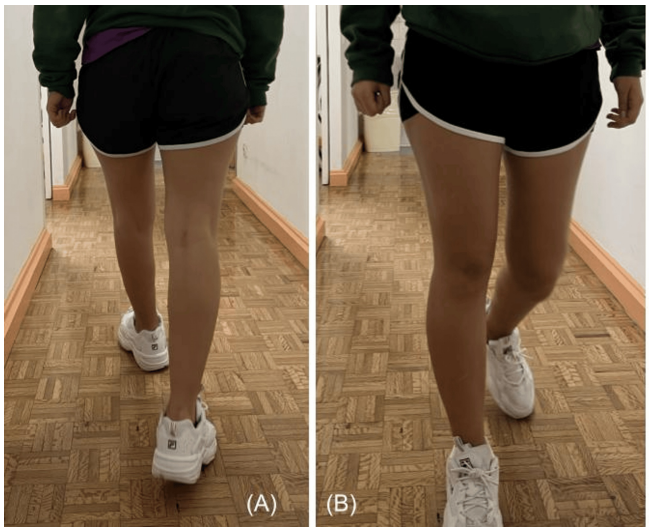


FIGURE 6: Clinical gait sequence at six months, showing a normalized gait pattern with an effective toe-off phase.

Toe alignment at the same time point is shown barefoot in Figure 5.

From a neurological standpoint, the preoperative hallux and plantar paresthesia had resolved, and Tinel's sign over the tarsal tunnel was negative. The overall result of the patient-reported FAOS questionnaire improved from 52 out of 100 points before surgery to 89 out of 100 points at six months. The patient reported returning to all activities as a sports student without pain.

Discussion

Claw toe deformity after tibial fracture was first described by Clawson [3] and has most often been reported as a checkrein deformity of the hallux caused by FHL tethering [4,5]. Given the anatomical proximity of the FHL and FDL within the deep posterior compartment, combined tethering can cause clawing of all toes. While isolated FHL involvement is well documented [4,5], clawing associated with combined FHL and FDL adhesions has been described in children [6] and in adults [7], although such reports remain scarce.

This sequela usually manifests within months of injury, although the reported onset varies [4-7]; in our patient, the deformity was identified two months after injury. Trauma at the fracture level may have

promoted the adhesions found between the FHL and FDL and, predominantly, between these muscles and the hypertrophic callus, causing progressive tendon tethering and dynamic clawing during ankle dorsiflexion, as previously described for FHL tethering at the fracture site [3,4].

A key diagnostic challenge was the discrepancy between preoperative imaging and intraoperative findings. MRI showed an edema-like signal in the deep flexor compartment, interpreted as a nonspecific post-traumatic change, but no clearly identifiable adhesion, and metal-related artifact partially limited assessment around the nail and fracture site. This case suggests that static imaging may fail to detect focal adhesions or dynamic entrapment. The adhesions found at surgery and the intraoperative restoration of toe extension after their release supported a mechanical etiology, and myolysis and tenolysis were sufficient without tendinous lengthening. A single posteromedial approach in the supine position allowed concomitant tarsal tunnel decompression but provided only indirect access to the fracture callus and the FHL.

The plantar pain and paresthesia, with a positive Tinel's sign over the tarsal tunnel, suggested posterior tibial nerve involvement, possibly related to traction transmitted along the tethered deep flexor tendons, and tarsal tunnel syndrome has been associated with FHL pathology [8]. Preoperative EMG was inconclusive, suggesting possible tibial nerve involvement at the distal leg or tarsal tunnel and common peroneal nerve involvement; however, motor strength was preserved in all muscle groups of the lower limb. At surgery, the posterior tibial nerve was free of adhesions at the level of the fracture callus. The diagnosis was therefore based primarily on symptoms and a positive Tinel's sign, and neurological symptoms had resolved six months after surgery, although the separate contributions of tendon release and nerve decompression cannot be determined.

An iatrogenic hardware-related mechanism was also considered. Bai et al. reported an FHL checkrein deformity caused by misplaced suture-button syndesmosis fixation [9]. In our patient, serial radiographs demonstrated appropriate implant positioning, and MRI showed no evident hardware protrusion into the deep posterior compartment, although metal artifact limited this assessment. This etiology was therefore considered unlikely. Furthermore, the involvement of all toes suggested combined FHL and FDL tethering rather than isolated hardware irritation.

A primary neurogenic cause of the deformity was considered less likely, given the preserved motor strength, the strictly dynamic pattern of the deformity, and the intraoperative restoration of toe extension after release of the adhesions. The muscle bellies appeared viable, without ischemic necrosis, which argued against an established Volkmann-type contracture. However, this finding does not exclude a missed subacute deep posterior compartment syndrome, in which scarring and adhesions may also occur, particularly as compartment pressures were not measured at admission.

If left untreated, dynamic clawing may impair gait mechanics, and chronic contracture may lead to secondary joint degeneration and fixed deformity, potentially reducing the success of simple release procedures. This report has limitations. Follow-up was limited to six months, and longer follow-up is required because recurrence has been reported after surgical release at the fracture site [4]. The FHL was released indirectly through a limited approach. In addition, the FAOS was orally translated for the patient, and only overall values were recorded, without individual subscale scores.

Conclusions

This case shows that dynamic clawing of all toes can arise as a rare delayed complication of a tibial shaft fracture, associated with combined FHL and FDL adhesions. MRI did not show a clearly identifiable adhesion, and EMG was inconclusive, so the diagnosis relied primarily on dynamic clinical assessment. Because the deformity was recognized before fixed contractures developed, correction was achieved through myolysis and tenolysis alone, without tendon lengthening or transfer, and tarsal tunnel decompression was performed concomitantly for the neurological symptoms. At six months, the deformity and symptoms had resolved, with a favorable patient-reported outcome on the FAOS questionnaire. This case highlights that recognizing the deformity while it remains dynamic may allow correction through soft-tissue release alone, although longer follow-up is needed.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Acquisition, analysis, or interpretation of data: Olga Tkachuk Correia, Manuel Cerdeiros, Ana M. Baía, Cláudia Oliveira, Diogo Mestre

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Critical review of the manuscript for important intellectual content: Olga Tkachuk Correia, Manuel Cerdeiros, Ana M. Baía, Cláudia Oliveira, Diogo Mestre

Supervision: Cláudia Oliveira

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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