Hindfoot

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1 Arthritis

Take-Home Message
- Post-traumatic arthritis is the most common etiology.
- Classified based on etiology.
- Nonoperative management (Arizona brace or AFO) is the first-line treatment strategy.
- Arthrodesis is indicated when nonoperative management is not effective to control pain and disability. No role for arthroplasty in the hindfoot.

Definition

- Inflammation and degenerative disease of the hindfoot articulations that includes the talonavicular, subtalar, and calcaneocuboid joints.

Etiology

- Post-traumatic arthritis is the most common cause. Mechanical degeneration of the articular surface as a result of post-traumatic articular injury, incongruity, or collapse (Fig. 1).
- Osteoarthritis is the second most common and is commonly associated with a deformity (pes planus secondary PTTD or cavus).
- Others include inflammatory arthropathy, osteonecrosis, gout, or rarely septic arthritis.

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Fig. 1 Nonoperatively treated calcaneus fracture with resultant post-traumatic arthritis. Note the loss of height and obvious articular incongruity and joint space narrowing. The patient had no anterior ankle impingement or pain and was treated with an in situ subtalar arthrodesis

Pathophysiology

- Post-traumatic articular incongruity leads to abnormal joint contact forces and loading, resulting in mechanical wear.
- Deformity/instability can also lead to joint subluxation, decreased contact area, and increased contact pressure.

    - Post-traumatic deformity, end-stage posterior tibialis tendon disorder (PTTD), tarsal coalitions, and cavovarus are commonly associated.

- Inflammatory processes such RA lead to articular cartilage destruction and wear.
- Abnormal purine metabolism results in precipitation and deposition of monosodium urate crystals in joint spaces, resulting to severe inflammatory response.

Radiography

- Weight-bearing x-ray (AP, lateral, and oblique views) of the hindfoot

    - Joint space narrowing or loss (Fig. 2)
    - Subchondral sclerosis/cysts
    - Osteophytes and bony erosions
    - Hindfoot malalignment and deformity (Fig. 3)
      Computed tomography (CT) scan
    - In the setting of deformity, the obliquity if plain radiographs prevent appropriate visualization of the joints. CT is critical in these cases to accurately determine the presence of articular erosion.

Classification

Based on Etiology

- Mechanical (post-traumatic)
- Degenerative (osteoarthritis)

Fig. 2 Joint space narrowing in a patient who has isolated talonavicular (*arrow*) arthritis

Fig. 3 The severe malalignment of the hindfoot precludes visualization of the hindfoot joints in particular the subtalar joint in patients with a pes planus deformity. In these cases, this may be misinterpreted as subtalar DJD. A CT is appropriate in these cases to determine viability of the joints to determine if the patient is a candidate for joint salvage or requires an arthrodesis

- Inflammatory (rheumatoid, seronegative spondyloarthropathy)
- Metabolic (gout, pseudogout (chondrocalcinosis))
- Neuropathic

Treatment

Nonoperative

- Nonsteroidal anti-inflammatory drugs (NSAIDs)
- Activity modification (avoidance of impact and uneven ground)
- Bracing such as an ankle-foot orthosis (AFO) or rigid lace up brace such as Arizona brace with an associated rocker bottom shoe modification
- Corticosteroid/anesthetic injections: both diagnostic and therapeutic
- Treatment of underlying causes such as gout, RA

Operative

Failure of nonoperative treatment with persistent pain and disability is an indication for arthrodesis. Goal of surgery is to obtain a solid fusion and position the hindfoot in 0–5° of valgus, neutral abduction/adduction, congruent talus-first metatarsal axis (Meary line) and create a plantigrade foot (Fig. 4). In many patients a concomitant contracture of the Achilles tendon is present, and an Achilles lengthening should be considered at the time of arthrodesis if a fixed equinus contracture is present.

- Single joint fusion is indicated for isolated joint arthritis. However, isolated joint fusions, especially TN (TN > ST > CC), are associated to severe hindfoot motion limitation and higher nonunion rate. As such, fusion procedures usually involve ST and TN fusion or triple (ST, TN, and CC) fusion (Fig. 5).

Fig. 4 Postoperative clinical appearance of a patient s/p subtalar and talonavicular arthrodesis for a severe pes planus deformity. Note the neutral adduction/abduction and restoration of the longitudinal arch

- In the setting of stage 3 PTTD, a TN and ST fusion without violation of the CC joint may be employed. In many cases additional midfoot correction is required to obtain a plantigrade foot (Fig. 6).
- A triple arthrodesis is an effective strategy to treat pan hindfoot arthritis and deformity and is typically required for any congenital deformity.
- Subtalar bone block arthrodesis is an effective option to treat post-traumatic arthritis secondary to a calcaneus fracture with loss of height. This operation is only indicated if the patient has ankle impingement (pain or limited dorsiflexion). The higher risk of nonunion and technical difficulty is why this should not be performed if the patient is asymptomatic in the ankle.
- Tibiotalocalcaneal (TTC) fusion is an option for subtalar arthritis with associated tibiotalar arthritis or if there is a concern for bone stock and fixation as may seen in neuroarthropathy or sever osteonecrosis.

Fig. 5 Attempted isolated TN fusion that resulted in a nonunion (**a**) that was successfully revised with a combined TN and ST fusion (**b**)

Fig. 6 The radiographic appearance of the patient in Fig. 4 with collapse of the medial arch and subtalar DJD noted on CT (**a**). Postoperative radiograph demonstrating restoration of the arch and additional 1 TMT fusion that was required to create a plantigrade foot (**b**)

Complications

- Wound complications: Higher in a lateral approach for subtalar joint in severe pes planus deformity and bone block arthrodesis s/p calcaneus fractures.
- Nonunion risk factors: prior ankle arthrodesis, isolated TN fusion, and smoking
- Malunion: Resultant malposition of foot and abnormal joint loading. Post-traumatic talar neck fractures with varus require a triple arthrodesis to prevent persistent cavovarus deformity.
- Adjacent joint arthritis: The ankle is most common.

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2 Ankle Instability

Take-Home Message
- The lateral ligamentous complex is most commonly involved (ATFL > CFL > PTFL).
- Nonoperative management is the first-line treatment for all acute injuries → therapy (peroneal strengthening and proprioception) and functional bracing.
- Surgical reconstruction indicated for persistent instability → Anatomic repair is most appropriate first-line treatment (Brostrom-Gould).
- Concomitant hindfoot varus may require lateral slide/closing wedge calcaneal osteotomy.

Introduction

- Acute injuries → Traumatic injury to the lateral ligamentous complex presenting with pain, swelling, and ecchymosis.

- Chronic instability → Repeated episodes of "giving way" or recurrent ankle sprains without significant trauma.

- These symptoms can be debilitating to athletes, and recurrent ankle sprains are felt to be an etiologic factor for ankle arthritis.

Etiology

- Injury and incompetence of the ligamentous structures (most commonly laterally)

- Certain factors put patient at risk for ankle instability:

  - Mechanical: varus hindfoot alignment, generalized ligamentous laxity
  - Functional: muscle weakness (peroneal tendons), impaired proprioception, and impaired neuromuscular control

- Suggested mechanism of injury → inverted, plantar flexed foot with an internally rotated hindfoot, and an externally rotated leg

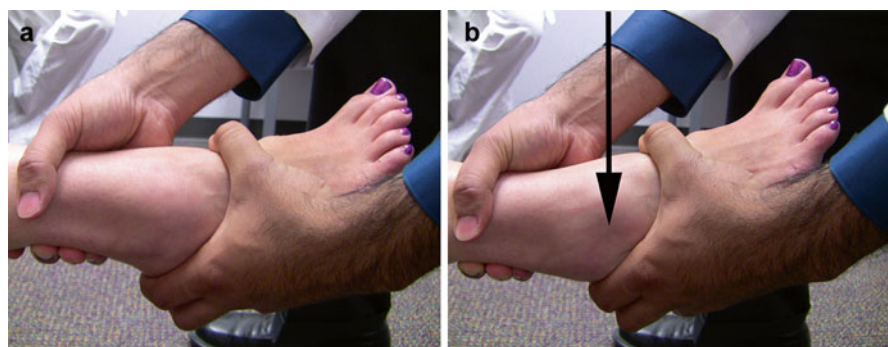**Fig. 7** Intraoperative photograph of the ATFL (*white arrow*)

Fig. 8 To perform the anterior drawer test, ne hand stabilizes the anterior distal tibia, while the other is cupped around the posterior calcaneus (**a**). The heel is translated anteriorly with respect to the tibia, and any subluxation should be noted (**b**). Note the sulcus that is created over the antero-lateral ankle with an unstable ankle (*arrow*)

Fig. 9 Intraoperative photograph of the CFL (*white arrow*). Note how the fibers become taught with dorsiflexion of the ankle

Fig. 10 Inversion stress test in a patient with severe laxity of the CFL

- Can lead to chronic instability

Pathophysiology

Ankle Joint Stability

- Lateral ligamentous structures: most commonly involved (medial side rarely injured). Always compare to contralateral lower extremity.
    - Anterior talofibular ligament (ATFL) (Fig. 7) → most commonly injured → once injured, causes stress on the remaining ligaments; tested by anterior drawer test (Fig. 8). Also assessed with inversion in plantarflexion.
    - Calcaneofibular ligament (CFL) (Fig. 9) → second most commonly injured; tested by talar tilt stress (inversion test) in dorsiflexion (Fig. 10)
    - Posterior talofibular ligament (PTFL)
- Can check joint hyperlaxity by calculating the Beighton score. Signs of ligamentous laxity should be evaluated using the Beighton scoring system. Out of a possible nine points, four points indicates a generalized ligamentous laxity.
    - 5th finger metacarpophalangeal joint extension past 90° (bilateral, 2 points)
    - Thumb to volar forearm (bilateral, 2 points)
    - Hyperextension of the elbow (bilateral, 2 points)
    - Hyperextension of the knee (bilateral, 2 points)
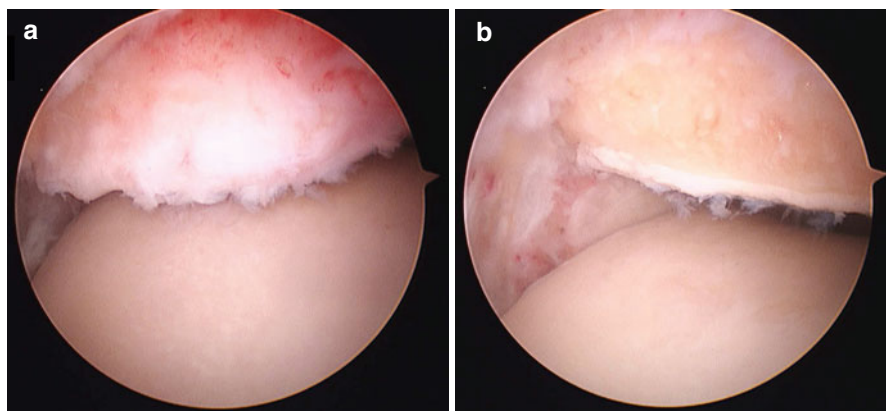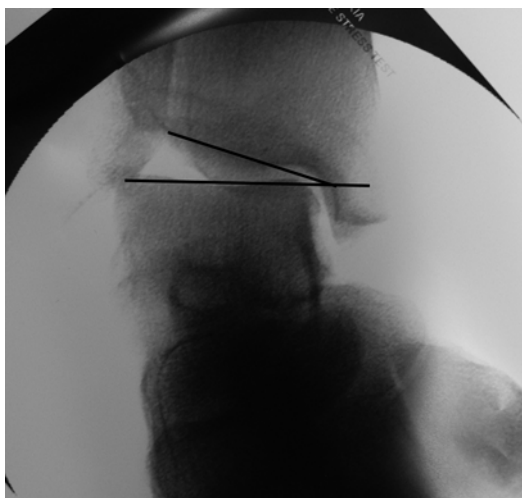   - Hands flat on floor with forward trunk flexion (1 point)

Fig. 11 Anterior osteophytes are identified by their prominence relative to the remaining tibia and by the lack of cartilage inferior to the bone (**a**). After excision of the osteophyte, the normal articular surface is easily seen (**b**)

Fig. 12 Varus stress radiograph of a patient with significant talar tilt >15°

Radiography

- Standard weight-bearing anteroposterior, lateral, and mortise views to assess avulsion fractures, arthritis, or osteochondral lesions. A common finding is anterior tibial osteophytes in patient with chronic instability; this must be addressed at the time of surgical intervention (Fig. 11).
- Anterior drawer stress x-ray and talar tilt views are helpful in cases of suspected ankle instability. Absolute values for normal talar tilt vary widely; however, the talar tilt is less the 15° in 95 % of patients (Fig. 12).
- MRI → Not required to make the diagnosis of an acute injury. May consider to evaluate for OCLT or syndesmotic injury if appropriate clinical suspicion is

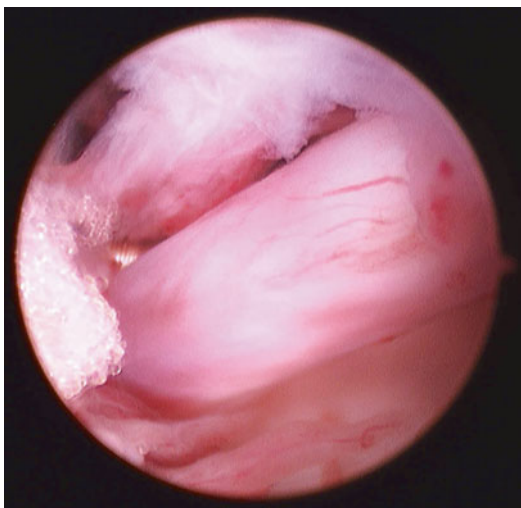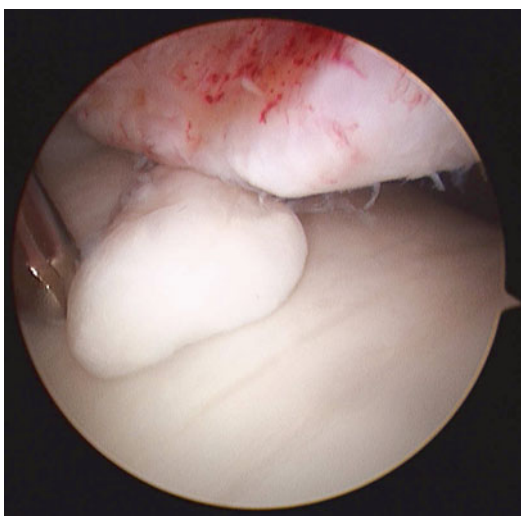**Fig. 13** Large adhesion in a patient that obliterates the anterolateral aspect of the ankle

Fig. 14 Loose bodies can be the source of instability in patients who have functional instability without any mechanical laxity. Arthroscopic excision of the loose body is critical in order to relieve the symptoms

given. Recommended in the setting of chronic instability with ankle pain to evaluate for intra-articular pathology (OCLT, loose body, anterior tibial osteophyte, synovitis) given the high incidence of concomitant intra-articular pathology (Figs. 13 and 14).

Classification

- Malliaropoulos classification of acute ankle sprains (Table 1)
- There is no relevant classification for chronic instability.

Table 1 Malliaropoulos classification of acute ankle sprains

Grade	I	II	III
Injured structures	Partial	ATFL	ATFL and CFL
Decrease in range of movement	<5	5–10	>10
Edema	up to 0.5 cm	0.5–2 cm	> 2 cm
Stress radiographs	Normal	Normal	>3 mm laxity

Treatment

Nonoperative: **initial treatment for most cases**

- Acute injury (nonoperative in all cases as first-line treatment)

    – Rest, immobilization, and protected weight bearing immediately, then early mobilization with functional bracing and early rehabilitation (peroneal strengthening and proprioception). Demonstrated superior to cast immobilization

- Chronic instability

    – Physical therapy (including peroneal strengthening and proprioceptive training), functional bracing
    – Orthotic treatment for cavovarus deformity (lateral heel wedge, decreased arch, well-out for first metatarsal)

Operative: following failure of nonoperative management with persistent instability

- Two methods:

    – Gould modification of Brostrom (anatomic repair)→Imbrication of elongated ATFL and CFL into distal fibula. Additional reinforcement with a flap of the inferior extensor retinaculum that improves stability of the subtalar joint (Fig. 15).
    – Non-anatomical reconstructions: reserved for revision cases, long-standing instability, or patients with generalized ligamentous laxity.

        • Watson-Jones, Evans, and Chrisman-Snook: all involve peroneus brevis transfer. These all create significant subtalar stiffness and less restriction of anterior translation compared to an anatomic reconstruction.
        • Autograft or allograft reconstructions have been utilized for revision cases or for patients with ligamentous laxity as these can be placed into a more anatomic position (Fig. 16).

- Arthroscopic exam: controversial, high percentage of intra-articular pathology found, but unknown clinical benefit proven. Consider if MRI demonstrates intra-articular pathology.

Complications

- Recurrence (especially with hindfoot malalignment that is uncorrected)
- Persistent pain
- Post-traumatic osteoarthritis (associated with significant long term instability)

Fig. 15 Brostrom anatomic reconstruction for lateral ankle instability. The ATFL and CFL are incised from the fibular origin (**a**). Utilizing suture anchors in this case the ligaments are imbricated (**b**). Final appearance of the ligaments shows them taut (**c**)
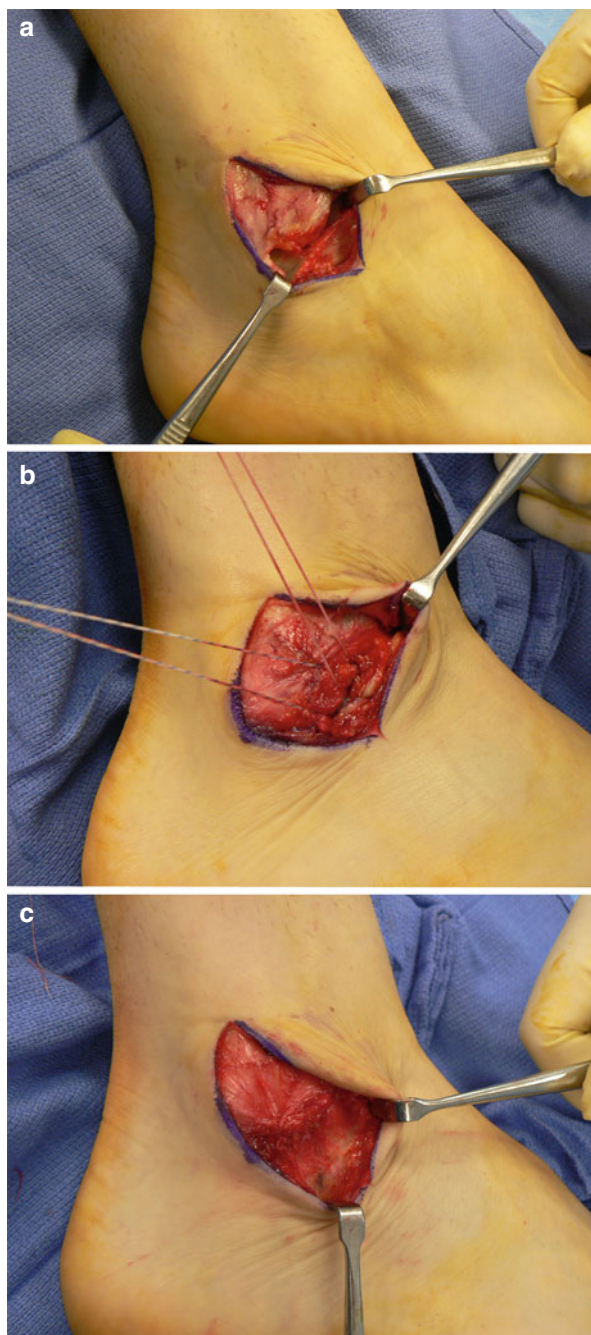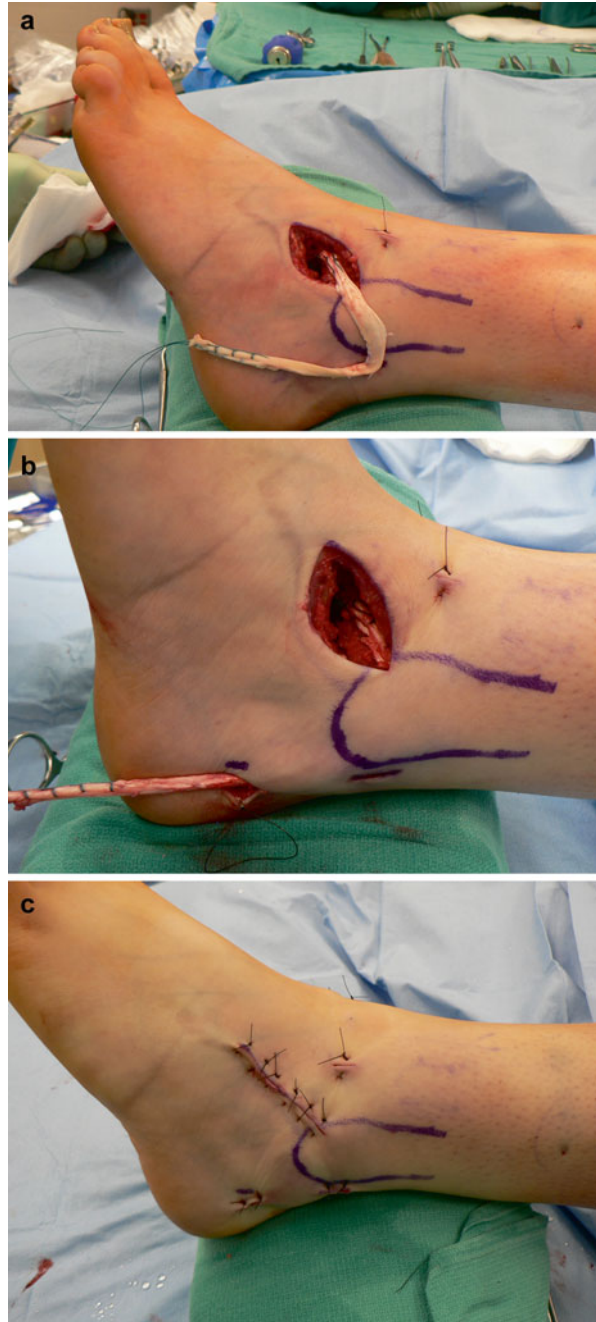
Fig. 16 Anatomic allograft reconstruction of the lateral collateral ligaments. The graft is fixated within the talus and routed from anterior to posterior through a fibular drill hole (**a**). Subcutaneous passage to the calcaneal insertion is then performed superficial to the peroneal tendons to prevent subluxation (**b**). Some authors prevent passing the tendon deep to the peroneal tendons to recreate the CFL's anatomic origin. There is no data to demonstrate superiority of one technique over the other. Final appearance of the reconstructed ankle (**c**)

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3   Talar Osteochondral Defects

> **Take-Home Message**
> - Lateral OCLT are usually of traumatic etiology, smaller, more shallow and more symptomatic than medial lesions. However, lateral lesions have superior surgical outcome.
> - Persistent ankle pain after acute sprain → obtain MRI to rule OCLT.
> - Lateral lesions have less potential for spontaneous resolution.
> - Choice of operative treatment is dependent on lesion size.
> - Arthroscopic verse open procedure has comparative results.

Definition

- Osteochondral lesions of the talar dome is a cause of ankle pain and disability. Presenting symptoms includes pain, swelling, and mechanical symptoms such as catching and locking. Up to 10 % are bilateral.

Etiology

- Acute trauma
- Repetitive microtrauma

Pathophysiology

- Osteochondral lesions of the talar dome may result from acute trauma or repetitive microtrauma. Pathophysiology differs based on lesion location. Recent data has demonstrated that both medial and lateral OCLT are found most common in the central talar dome in the sagittal plane.
- Lateral talar dome
  - Tends to be traumatic etiology, typically inversion or inversion-dorsiflexion mechanism
  - Lesions usually smaller, more shallow and more symptomatic than medial lesions (Fig. 17)
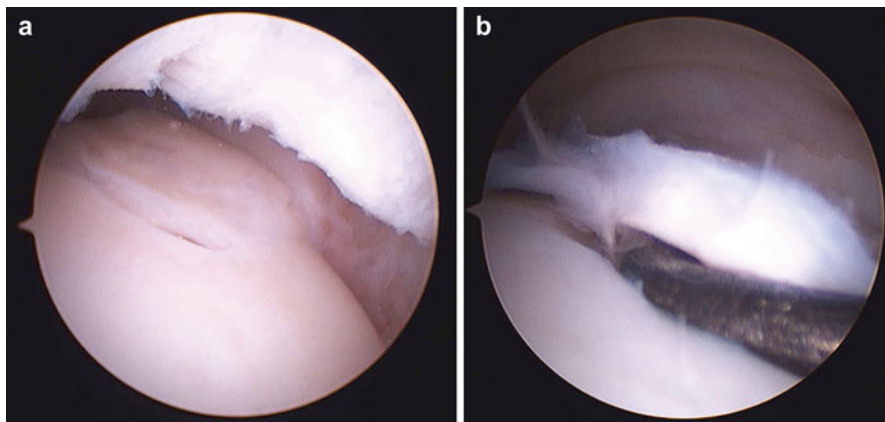 - Superior surgical results

Fig. 17 Arthroscopic view of a lateral talar osteochondral defect. (**a**, **b**) Note the shallow nature of the defect with the clear detached articular surface (**a**, **b**)

Fig. 18 Ankle radiograph demonstrating an obvious lucency of the medial talar dome (*arrow*) consistent with an OCLT

- Medial talar dome
    - Tends to be atraumatic etiology
    - Lesions usually larger and deeper than lateral lesions

Radiography

- Ankle x-ray
    - Maybe normal or subtle radiolucency, articular surface irregularity, or subchondral bone fragmentation (Fig. 18)

- CT/MRI
  - Further evaluate lesions apparent on x-ray or for persistent ankle pain after trauma such ankle sprain/fracture despite appropriate management (Fig. 19)
  - Helpful for classification of lesions

Classification

- The Berndt and Harty radiographic staging classification (Table 2)
- The Ferkel and Sgaglione CT staging classification (Table 3)
- The Hepple and associates MRI staging classification (Table 4)
- Arthroscopic evaluation is the reliable means of determining lesion size and the intactness of articular surface of lesions.

Fig. 19 MRI demonstrating a medial OCLT with fluid inferior to articular surface (*arrow*) that denotes instability of the lesion
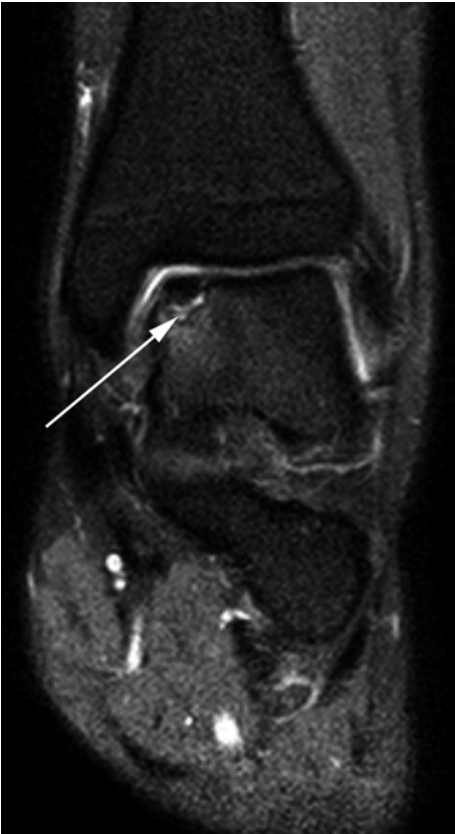
Table 2 The Berndt and harty radiographic staging classification of OCLT

Stage 1	Small area of subchondral compression/depression
Stage 2	Partial fragment detachment
Stage 3	Complete fragment detachment, non-displaced
Stage 4	Complete fragment detachment, displaced

Table 3 The Ferkel and Sgaglione CT staging classification of OCLT

Stage 1	Cystic lesion within talar dome with an intact roof in all views
Stage 2a	Cystic lesion with communication to talar dome surface
Stage 2b	Open articular surface lesion with overlying non-displaced fragment
Stage 3	Non-displaced lesion with lucency
Stage 4	Displaced fragment

Table 4 The Hepple and Associates MRI staging classification of OCLT

Stage 1	Articular cartilage
Stage 2a	Cartilage injury with underlying fracture with surrounding bone edema
Stage 2b	Cartilage injury with underlying fracture without surrounding bone edema
Stage 3	Detached but non-displaced fragment
Stage 4	Displaced fragment
Stage 5	Subchondral cyst formation

Treatment

Nonoperative

- Indication: Non-displaced fragment. No proven risk of DJD with nonoperative management.
- Short leg cast immobilization
- Restricted weight bearing

Operative

- Indication: displaced fragment in the acute setting. Persistent pain or mechanical symptoms in the chronic setting.
- Treatment choice is based on size of lesion.
- Results of arthroscopic verse open procedures are comparable.
- Lesion with intact cartilage cap: retrograde drilling ± bone grafting.
- Lesion < 1.5 cm^2: debridement and microfracture/drilling (Figs. 20 and 21)
- Lesion > 1.5 cm^2 cm with displaced cartilage cap: joint restorative procedures – osteochondral autograft, chondrocyte implantation, or osteochondral allograft
- Lesions >3 cm^2: osteochondral allograft (Fig. 22).

Complications

- Nonunion of autograft/allograft
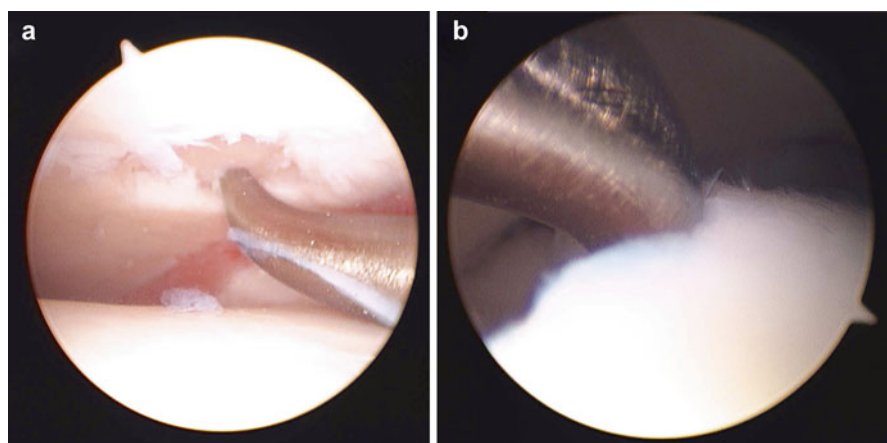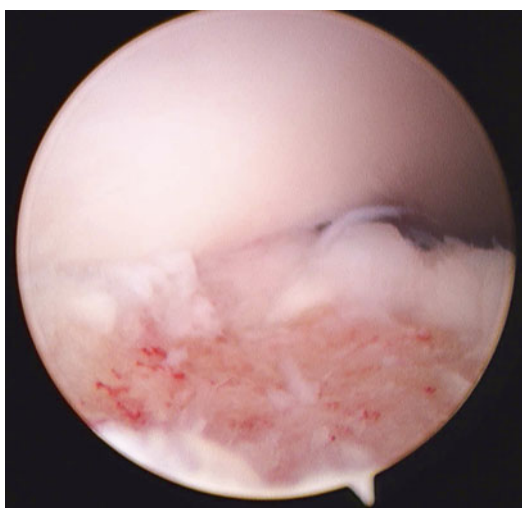- Persistent pain
- Tibiotalar arthritis

Fig. 20 Microfracture technique of a tibial (**a**) and talar (**b**) osteochondral lesion

Fig. 21 Final appearance of a talar osteochondral defect following microfracture. Note the blood that is emanating from the microfracture holes made in the talus

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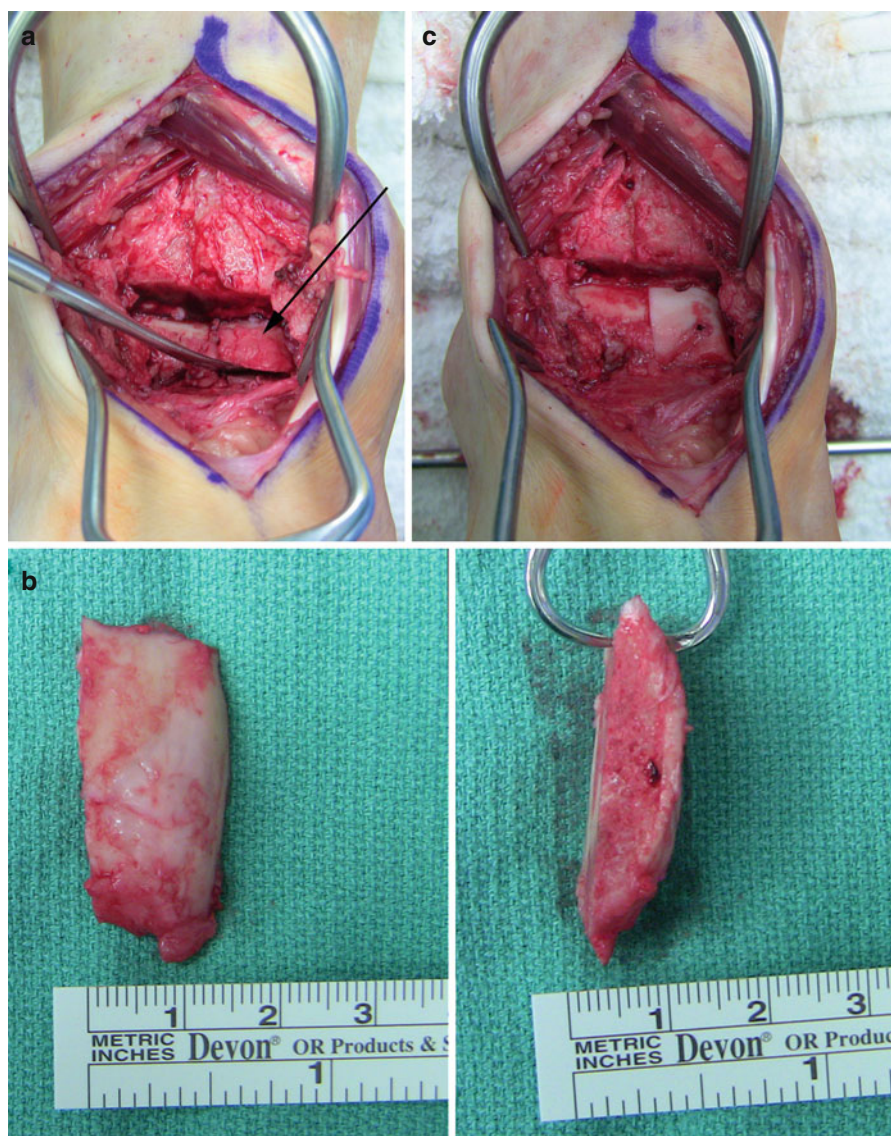
Fig. 22 Large (>3 cm^2) osteochondral defect (*arrow*) that was approached via an anterior ankle arthrotomy (**a**). The entire ½ of the articular surface was abnormal (**b**). Given the large surface area involved an allograft reconstruction (**c**) was performed

4 Heel Pain

Take-Home Message
- Plantar fasciitis is the most common cause, present classically with "start-up" pain in the AM that improves after the first few steps.
- Location of pain provides diagnostic clue.

  - Inferior heel pain ->plantar fasciitis
  - Medial heel pain→Baxter's neuritis (Lateral plantar nerve compression)
  - Posterior heel pain→retrocalcaneal bursitis, Haglund's deformity, insertional and Achilles tendinitis/tendinosis
  - Pain with lateral and medial compression→calcaneal stress fracture

- Nonoperative management is the first treatment. Plantar fascia-specific stretch is most important for PF.

Definition Heel pain is a common foot problem that causes significant discomfort and disability. The differential diagnosis for heel pain is broad and includes plantar fasciitis, calcaneal stress fracture, calcaneal apophysitis (Sever disease), central heel pain (fat pad atrophy, fat pad contusion), and nerve entrapment (tarsal tunnel syndrome, entrapment of the first branch of the lateral plantar nerve) and posterior heel pain (retrocalcaneal bursitis, Haglund's deformity, insertional Achilles tendinitis/tendinosis).

Etiology

- Repetitive microtrauma (stress fracture, Baxter's neuritis in runners)
- Inflammatory
- Degenerative
- Drug induced (fluoroquinolone use with Achilles pathology)

Pathophysiology Etiology is multifactorial, as such presentation and pathophysiology unique to each disease process.

Plantar Fasciitis

- Most common cause of heel pain. Affects both sedentary and active.
- Presents classically as "start-up" plantar medial heel pain and preference for toe walking for first few steps and improvement with progressive walking
- Repetitive microtrauma to plantar fascia→microtears and inflammatory response→reparative response
- Other risk factors: obesity, pes planus, pes cavus, gastrocnemius contracture, and excessive femoral anteversion increase the traction load at the origin of the plantar fascia during weight bearing
- About 50 % have heel spurs, however not cause of heel pain.

Calcaneal Stress Fracture

- Heel absorbs about 110 % of body weight during walking and 200 % during running.
- Repetitive loading of the calcaneus results in fatigue fractures. Common in active individual and military recruits.
- Risk factors: female, female athlete triad, hormonal deficiency, and osteopenia

Calcaneal Apophysitis (Sever Disease)

- Common cause of heel pain in pediatric population
- Overuse injury of calcaneal apophysis
- Natural history: self-limiting, resolves with closure of the apophysis

Central Heel Pain (Fat Pad Atrophy, Fat Pad Contusion)

- The heel pad provides significant shock absorber function to the hindfoot.
- Fat pad atrophy can result from inflammatory disease process, corticosteroid injection, trauma, and advanced age.
- Fat pad contusion from trauma can present as central heel pain.

Nerve Entrapment

- Entrapment of the First Branch of the Lateral Plantar Nerve (Baxter's Neuritis)

  The first branch of the lateral plantar nerve is a mixed (sensory and motor) nerve. Compression of this nerve occurs between the deep fascia of the abductor hallucis and inferomedial margin of the quadratus plantae.

  Common in running athletes and presents as plantar medial heel pain that mimic plantar fasciitis

  Tarsal tunnel syndrome can have similar presentation; however, symptoms originate more proximal and tend to involve entire foot and not just heel.

Posterior Heel Pain

Retrocalcaneal bursitis and Haglund's deformity

- Inflammation of retrocalcaneal bursa (lies between anterior surface of Achilles tendon and the posterosuperior calcaneus tuberosity).
- Haglund's deformity: enlargement of the calcaneal posterosuperior tuberosity

Insertional Achilles tendinitis/tendinosis

- Inflammatory changes at the tendon insertion site from repetitive microtrauma
- Progressive bony metaplasia and prominence at the calcaneal insertion

Fig. 23 Inferior plantar spur (*arrow*) commonly seen in patients with plantar fasciitis

Fig. 24 Lateral radiograph of a calcaneal stress fracture. Note the linear and vertically oriented increased density in the calcaneus (*arrowheads*). The finding is subtle but more easily identified as the normal lines of stress are oriented from the tuberosity towards the posterior facet, with the fracture line crossing multiple normal lines of stress

Radiography

Plantar Fasciitis

- Weight-bearing x-ray

  - Usually normal
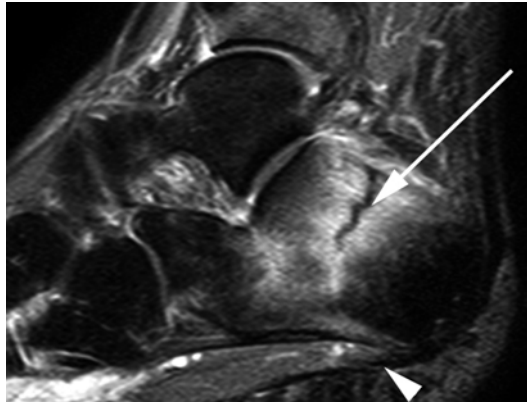 - Heel spurs (Fig. 23)

Calcaneal Stress Fracture

- Heel x-ray: Initially normal, but few weeks after onset of symptoms, radio-dense line becomes apparent (Fig. 24).
- MRI: fracture apparent in setting of normal x-ray (Fig. 25)

Calcaneal Apophysitis (Sever Disease)

- Heel x-ray

  - Apophyseal sclerosis not specific finding
  - Apophyseal fragmentation more specific finding

Fig. 25 Sagittal STIR image of a calcaneal stress fracture. Note the hypointense signal (*arrow*) delineating the fracture line with adjacent bony edema (high signal)

- MRI

  - Apophyseal inflammation, rule out other diagnosis

- Bone scan

  - Apophyseal increased uptake

Central Heel Pain (Fat Pad Atrophy, Fat Pad Contusion)

- Weight-bearing x-ray

  - Heel spurs
  - Rule out other pathology

Baxter's Neuritis

- EMG/NCV: increased motor latency of the abductor digiti quinti
- MRI: fatty atrophy of abductor digiti quinti or space-occupying lesion

Retrocalcaneal Bursitis and Haglund's Deformity

- Foot x-ray: Haglund's deformity on lateral view

Insertional Achilles Tendinitis/Tendinosis

- Lateral Heel x-ray: bone spur, intratendinous calcification/ossification (Fig. 26)
- Ultrasound/MRI: evaluate tendon degeneration

Treatment

Plantar Fasciitis

- Nonoperative

  - Plantar fascia-specific and heel cord stretching protocol
  - NSAIDs, low impact activity, physical therapy
  - Orthotics: Night splints, heel cushion inserts
  - Corticosteroid injection if failed non-op

Fig. 26 Lateral radiograph of a patient with insertional calcific Achilles tendinosis. A large posterior heel spur is diagnostic (*white arrow*)

- High-energy extracorporal shock wave therapy and PRP have demonstrated efficacy in some trials compared to standard non-op treatment in the short term. Cannot be recommended over standard non-op management at this time.

- Operative
  - Plantar fascia release. Release only medial ½. Baxter's nerve release for concomitant Baxter's neuritis symptoms
  - Gastrocnemius recession instead of a PF release if contracture is present

Calcaneal Stress Fracture

- Rest/activity modification and protected weight bearing
- Cushioned heel orthotics

Calcaneal Apophysitis (Sever Disease)

- Rest/activity modification and heel cord stretching
- Brief immobilization with short leg cast for persistent pain
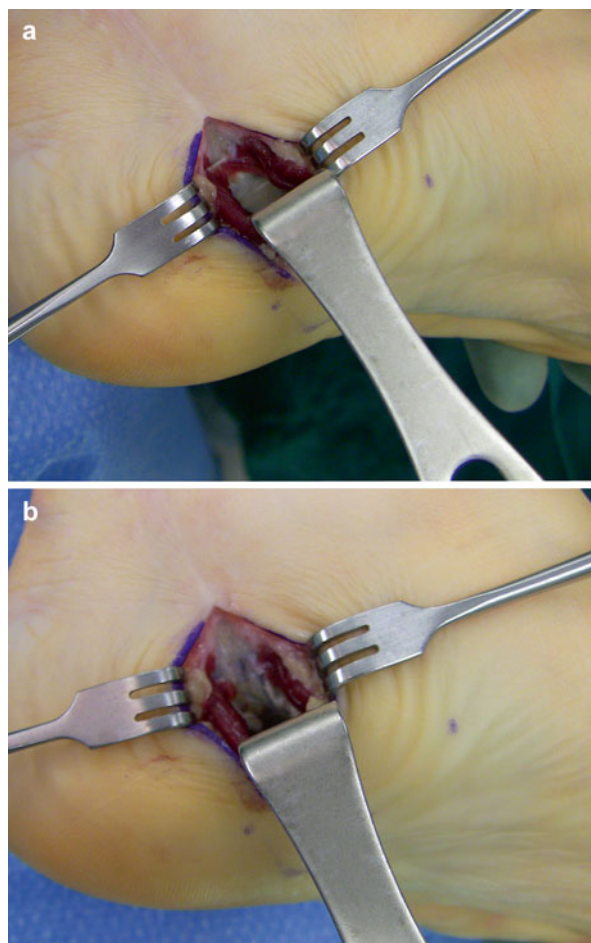- Cushioned heel orthotics

Central Heel Pain (Fat Pad Atrophy, Fat Pad Contusion)

- Cushioned heel cups

Baxter's Neuritis

- NSAIDs, cessation of aggravating activity, heel cord stretching
- Orthotics: cushioned heel inserts, correct for any underlying deformity.
- Operative
  - Surgical release and decompression of Baxter's nerve, deep fascia of the abductor hallucis must be released (Fig. 27).
  - Medial ½ plantar fascia release for concomitant plantar fasciitis symptoms

Fig. 27 Inferior retraction of the muscle belly of the abductor exposes the deep fascia (**a**). Appearance after incision of the deep fascia (**b**). The lateral plantar nerve lies directly superior to the deep fascia, and careful dissection must be performed
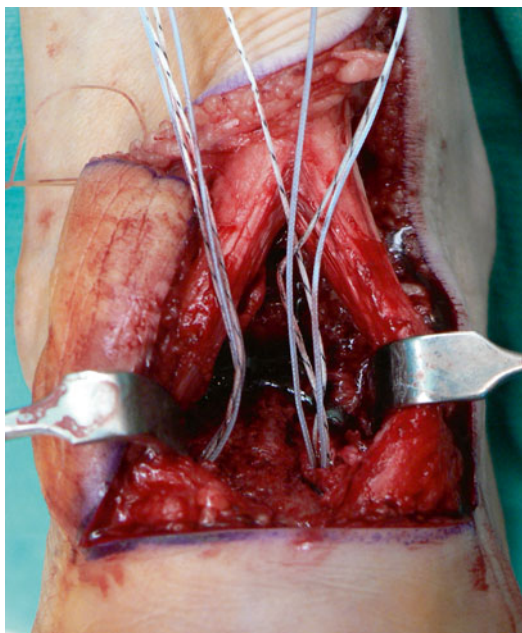

Retrocalcaneal Bursitis and Haglund's Deformity

Nonoperative

- NSAIDs
- Shoe modification (open or low back shoes) or padding to minimize mechanical irritation
- Corticosteroid injection of the bursa, avoid tendon injection → risk of rupture

Operative

- Retrocalcaneal bursectomy and resection of Haglund's deformity
- <50 % release of Achilles tendon mandates repair with anchors to calcaneus.

Fig. 28 Intraoperative photograph of a patient who had insertional Achilles tendinosis. After debridement of the diseased tissue and Haglund's deformity, more than 50 % of the Achilles was detached. Repair with suture anchors is important in preventing postoperative rupture

Insertional and Non-Insertional Achilles Tendinitis/Tendinosis

Nonoperative

- NSAIDs, activity modification (limit impact, push-off).
- Heel cord stretching and therapy with eccentric training (most critical).
- Shoe modification or padding to minimize mechanical irritation or small heel lift.
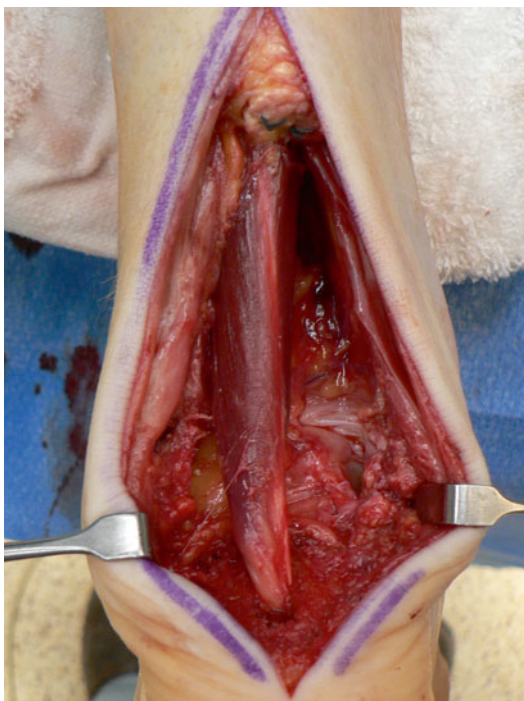- Avoid corticosteroid injection → risk of rupture.

Operative

- Tendon and prominent bone debridement with repair to bone with anchors (Fig. 28).
- Tendon augmentation with FHL, FHL transfer if > 50 % tendon debrided (Fig. 29)

Complications

Plantar Fasciitis

- Fat pad atrophy from corticosteroid injection
- Longitudinal arch collapse and chronic foot pain from excessive plantar fascia release
- Injury to the lateral plantar nerve

Fig. 29 Intraoperative photograph of a FHL transfer in a patient who had a severe insertional Achilles tendinosis. After resection of the nonviable tissue, the FHL was transferred through a drill hole in the calcaneus and fixed with an interference screw

Baxter's Neuritis

- Inadequate decompression
- Injury to the lateral plantar nerve

Retrocalcaneal Bursitis and Haglund's Deformity

- Achilles tendon rupture from corticosteroid injection or excessive debridement

Insertional Achilles Tendinitis/Tendinosis

- Achilles tendon rupture from corticosteroid injection or excessive debridement

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