INSERTIONAL TENDINOPATHY OF THE ACHILLES TENDON

Heel pain is a common complaint and is often poorly managed.

Non-operative treatments are highly effective for the vast majority of patients, and surgery is reserved for recalcitrant cases.

The first distinction to make clinically is between plantar heel pain and posterior heel pain. The former is commonly caused by plantar fasciitis.

Posterior heel pain:

Baxter [1] classified Achilles pain as arising from the insertional portion of the tendon or from the noninsertional region. This distinction is helpful clinically.

Noninsertional Achilles tendon pathology is more common than insertional tendinopathy and is due to degeneration within the substance of the tendon, thickening of the paratenon, or a combination of the two.

Box 1. Differential diagnosis of heel pain

Posterior heel pain
Insertional tendinopathy
Retrocalcaneal bursitis
Pump bumps
Os trigonum
Flexor hallucis longus
Gout
Seronegative arthropathy

Plantar heel pain
Plantar fasciitis
Os calcis stress fracture
Tarsal tunnel syndrome
Rheumatoid arthritis
Infection (especially in diabetics)

Maffulli proposed a logical and easy-to-use nomenclature for describing Achilles tendon pathologies. This has reduced the use of many confusing synonyms that were previously seen in the literature.

The emphasis is upon tendon degeneration rather than inflammation.

The clinical picture of pain, swelling, and impaired function is best referred to as Achilles tendinopathy. This terminology may also be applied to the rotator cuff, patellar tendon, and other tendons that have painful overuse

Nomenclature in Achilles tendon pain

Clinical

Tendinopathy pain, swelling, and reduced function

Paratenonopathy affects paratenon clinically

Panatendinopathy affects both tendon and paratenon clinically

Histological

Tendinosis mucoid degeneration and collagen disorganization

Paratenonitis hyperemia and inflammatory cells; fibrosis and thickening; more common in specimens

from younger patients symptoms. The term tendinopathy does not define the underlying pathological processes responsible for the symptoms.

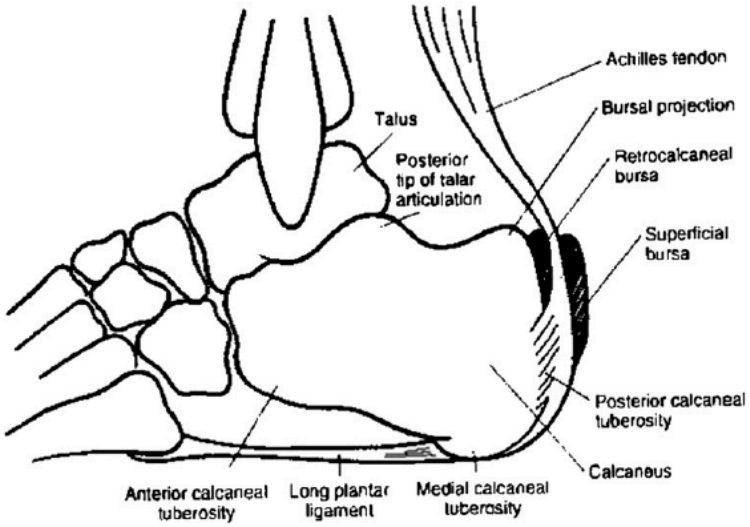
In a chronic tendinopathy, there is no inflammatory response and granulation tissue is rarely seen when tissues are examined histologically. For this reason, the term tendinitis is considered inappropriate.

Local anatomy

Triceps surae

The Achilles tendon is the conjoint tendon of the soleus and the two heads of gastrocnemius. The gastrocnemii originate from the posterior aspect of the femoral condyles and the soleus does not extend above the knee, taking its origin from the posterior aspect of the tibia, fibula, and interosseous membrane.

Posterior heel anatomy. (Orthop Clin North Am 1994;25(1):43)



The fibers of the conjoint tendon rotate through 90° as they progress distally, such that the medial fibers proximally become the most posterior fibers distally at the insertion.

Anatomy at the Achilles insertion

The tendon inserts onto the middle third of the posterior surface of the tuberosity of the os calcis. Like all tendons, insertion into bone is via transitional tissues: tendon, fibrocartilage, mineralized fibrocartilage, and bone.

The distal tendon immediately proximal to the insertion is closely related anteriorly to the superior third of the posterior surface of the os calcis. This bone surface may be covered by fibrocartilage. The synovially lined retrocalcaneal bursa lies between tendon and the os calcis at this level.

A subcutaneous bursa lies posterior to the distal Achilles tendon.

Blood supply

The poor blood supply to the noninsertional region of the Achilles tendon is implicated in the

pathophysiology of tendinopathy and rupture. However, the blood supply to the insertion of the tendon is widely held to be good. The anterior plexus, periosteal vessels, and osseous branches supply this portion of the tendon. Diminished blood flow at the insertion of the tendon has been demonstrated, however, and may play a part in the development of insertional tendinopathy.

Bony anatomy

The shape and alignment of the os calcis is relevant to the management of pain or swellings of the posterior heel. Distal to the insertion of the Achilles tendon, the os calcis gives attachment to the fascia that runs in continuity with the plantar fascia. An enlarged posterosuperior margin of the calcaneal tuberosity is called a bursal projection (Haglund deformity) and may impinge against the tendon. The impingement may result in retrocalcaneal bursitis or degenerative change in the tendon itself. It may also rub against the heel counter of shoes, producing local swelling and tenderness (pump bumps).

Multiple pathology

Posterior heel pain may be due to degenerative change in the insertional portion of the tendon itself, to enlargement of the retrocalcaneal bursa, or to both. Pump bumps are usually posterolateral and should not be confused with retrocalcaneal bursitis or insertional tendinopathy.

Insertional tendinopathy

Insertional tendinopathy presents with posterior heel pain. It is maximal in the central region and at the insertion of the tendon.

There is often calcification within the central portion of the Achilles insertion. This calcification can be seen as a spur arising from the middle third of the os calcis on a lateral radiograph.

Surgical findings in humans, however, demonstrate that the tendon does not gain attachment from this spur.

Histology and histochemical studies have shown that an insertional tendinopathy is characterized by mucoid degeneration, necrosis, hemorrhage and calcification.

Retrocalcaneal bursitis

In many cases, this is seen on MRI in conjunction with tendinopathy at the insertion.

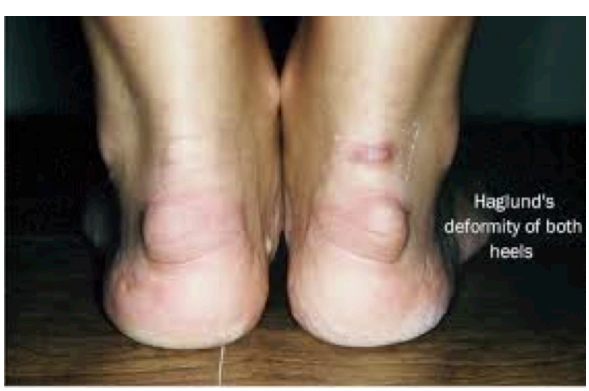
Haglund deformity [pump bump]

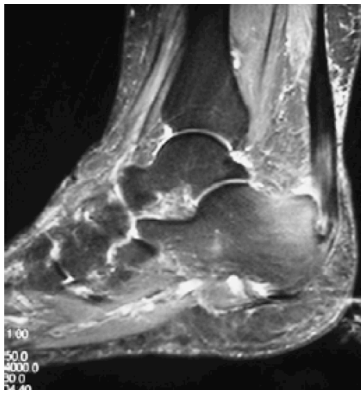
A large posterosuperior prominence of the lateral side of the calcaneal tuberosity rubs against poorly fitting shoes. This produces swelling of the subcutaneous (not retrocalcaneal) bursa with a tender, erythematous lump on the heel., emphasizing the association with certain shoes.

This condition should be considered as a specific entity, distinct from insertional tendinopathy, although in some cases there is associated enlargement of the retrocalcaneal bursa.

Haglund deformity may also be seen in up to 60% of patients with insertional Achilles tendinopathy. This does not mean that all patients presenting with pump bumps have insertional

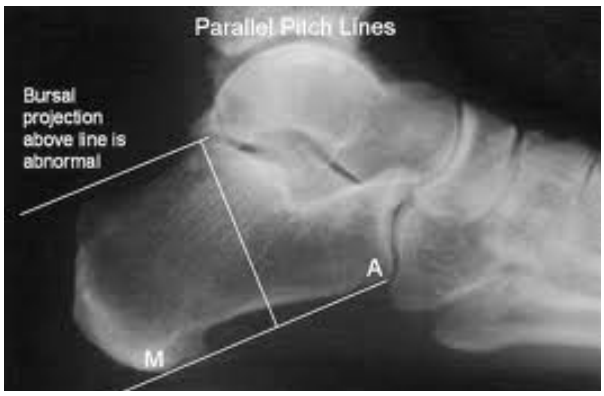
tendinopathy.



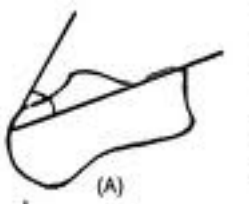


Existing studies consistently show that noninsertional tendinopathy is four times more prevalent than symptomatic insertional tendonopathy.
The large study by Paarvolaand colleagues [27] from Finland further distinguished between isolated insertional tendinopathy and cases of retrocalcaneal bursitis with insertional tendinopathy. The latter group, with mixed pathology, was much larger, accounting for 20% of all Achilles tendinopathies.

Measuring Haglund deformity
Pavlov: The parallel pitch lines determine the extent to which the bursal projection rises above the rest of the superior surface of the os calcis



Philip :The superior calcaneal angle described by Philip has been the most enthusiastically adopted by clinicians.



Neither method has proven reliable as a guide to treatment.

Assessment

1. Careful history taking determines the relationship of the symptoms to activity, to new training regimens, to poor warm-up technique, or to specific shoes.
2. Atypical features in the history, such as night pain, should prompt investigations to rule out a rare neoplastic cause for the heel pain.
3. Patients with pain arising from the insertion of the tendon, from retrocalcaneal bursitis, or from mixed pathology give a history of pain that is exacerbated by activity.

4. Local examination reveals the site of maximal tenderness. Midline tenderness is indicative of insertional tendinopathy, whereas retrocalcaneal bursitis causes maximal tenderness to the lateral. Erythema with localized swelling over a superolateral prominence

5. Increased calcaneal pitch with heel varus renders the bursal projection of the os calcis more prominent and is often a contributory factor in Haglund's disease.

6. Patients with this planovalgus foot posture invariably have adaptive shortening of the gastrocnemius in isolation. This examination technique, described by Silfverskiold, must be performed with the forefoot held to reduce the talonavicular joint.
7. ROM
8. Pulses and sensory examination.
9. Shoes must be inspected for excessive asymmetrical wear and any orthotic device already in use

Imaging

1. Plain radiographs should include a lateral weight-bearing view of the foot and ankle and an axial view of the heel.
2. Ultrasound scan
3. MRI can both provide useful information

Treatment

Nonoperative treatment

Orthotics and shoes

Modification of shoes, and occasionally an orthotic to lift the affected part of the heel away from the upper margin of the heel counter.

There may be some benefit from anti-inflammatory medication or gel, but steroid injection should be avoided wherever possible.

Corrective orthotics for planovalgus deformity can be helpful, but overcorrection is not well tolerated, particularly in runners.

Stretching

Stretching regimens for noninsertional tendinopathy are extremely effective, **with 90% of patients** responding when the stretches are performed properly. The results in cases of insertional pain are not as good because **only one third of patients** respond. Stretches are still worth pursuing, particularly

where adaptive shortening of the gastrocnemius is pronounced.

A high proportion of patients presenting with rupture at the insertion of the tendon report prior injection with corticosteroids.

Sclerosant injection therapy is successful in the treatment of noninsertional tendinopathy.
Extracorporeal shock wave lithotripsy is increasingly being evaluated as treatment for chronic soft tissue complaints, including plantar fasciitis, tennis elbow, and rotator cuff injuries.
There are few reports specific to the treatment of insertional tendinopathy, but their results are promising.
McGarvey and colleagues found that nonoperative treatments were successful in 89% of cases.

Surgery
Surgery is only considered for that minority of patients for whom all nonoperative treatments have been tried and have failed to produce sufficient improvement in symptoms.
Haglund's disease alone or in combination with retrocalcaneal bursitis. Haglund's disease that is refractory to nonoperative treatment is rare, and surgical case series therefore have relatively small numbers.
The bursal projection through a medial incision, which affords a convenient approach for osteotomy of the posterolateral bony prominence. An additional advantage of this approach is the absence of risk to the sural nerve.
Insertional tendinopathy alone. Maffulli treated 21 patients for calcific tendinopathy through a central posterior incision and tendon-splitting approach. The tendon and retrocalcaneal bursa were debrided, but no bone was resected from the bursal projection.
Taylor reported excellent results in a larger number of patients where resection of Haglund deformity was performed through a posterior midline Achilles splitting approach.
Whether the tendon was detached and reattached with suture anchors or whether debridement was considered satisfactory without tendon detachment. When the tendon was detached, a proximal V-Y plasty was used in addition.

Conclusions
_ Excision of the bursal projection and debridement of the retrocalcaneal bursa give good results in patients without significant symptoms from the Achilles insertion itself. Some of these patients have calcification at the insertion that is asymptomatic.
_ Complete detachment of the Achilles tendon insertion with suture anchor repair is safe and reliable.
_ At least 50% of the attachment can be released without the need for suture-anchor repair and without the need for postoperative cast immobilization.

Angermann found no correlation between the amount of bone removed and the outcome.
_ Flexor hallucis longus transfer is a useful augmentation.

Recovery after surgery is not complete for at least 6 months.

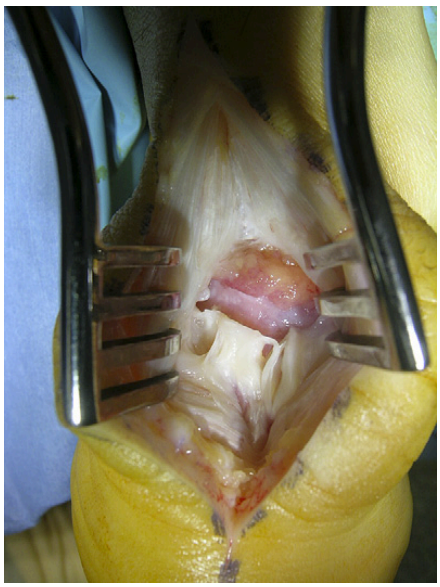
Authors' preferred treatment

Nonoperative treatment is preferred initially.

Steroid injection is avoided wherever possible and, if performed, ultrasound guidance is used.

The patient is counseled regarding the risk of rupture.

For recalcitrant cases, surgery has good results. If an isolated gastrocnemius contracture cannot be corrected by physiotherapy, a gastrocnemius



release is considered. As with eccentric physiotherapy stretches, the results are less reliable than for the treatment of noninsertional tendinopathy. Isolated Haglund deformity is treated by reduction of the bursal projection.

Retrocalcaneal bursitis with mild insertional tendinopathy is debrided endoscopically.

Where the calcific spur is large, an open debridement is preferred through a posterior midline incision and tendon-splitting approach. If less than half the insertion is detached, postoperative cast immobilization is not routinely employed. In the authors' experience, complete detachment of the tendon is not necessary.

If the patient presents with rupture at the insertion, the authors recommend augmentation with transfer of flexor halluces longus. If the distal stump is suitable, then the flexor hallucis longus is split and a side-to-side tenodesis used. If the distal stump is too diseased, a short harvest, calcaneal tunnel, and interference screw fixation is the preferred technique.

Reference

1. Foot Ankle Clin N Am 12 (2007) 597–615