REVIEW

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Achilles Tendon Enthesopathy. Current Therapeutic Treatments.

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ABSTRACT

Achilles tendon enthesopathy (AE) involves four different pathologic entities of the tendon's distal insertion to the calcaneus. These entities are described as (a) insertional Achilles tendinitis, (b) retrocalcaneal bursitis, (c) Haglund's deformity, and (d) intrasubstance calcification. There are many causative factors that lead to the development of AE, such as overuse, trauma, inadequate training or sport equipment, metabolic disorders and autoimmune diseases. The treatment can be initially conservative and in refractive cases surgical.

KEYWORDS: Achilles tendon, enthesopathy, retrocalcaneal bursitis, Haglund's deformity, calcified tendonitis

Introduction

Approximately 6% of the general population reports Achilles tendon pain during their lifetime (1) but only one third of them will develop AE (2). The condition can affect both male more than female population, young athletes, middle-aged long-distance runners and elderly patients with a tight heel cord. It is also found as clinical manifestation in metabolic bone disorders and in rheumatologic diseases such as ankylosing spondylitis.

Anatomy

The Achilles tendon consists of the aponeuroses of the gastrocnemius, plantaris longus, and soleus muscles (3) and it is primarily composed of type I collagen, surrounded by a paratenon (4). The tendon inserts 2 cm distal to the posterosuperior calcaneal prominence with an anterior-posterior diameter of 5 to 6 mm with medial and lateral projections (5). The tendon's blood supply is primarily provided from an arterial plexus along the calcaneus, supplied by the fibular and posterior tibial arteries (6). Additionally, the tendon is surrounded by subcutaneous and retrocalcaneal bursae in order to reduce friction between its surface and the adjacent tissues (7). The sural nerve is in close proximity to the Achilles tendon, stretching across the lateral border of the sheath (3). Neuroma formation or other sural nerve disorders should be included in the differential diagnosis of AE.

Pathophysiology

Achilles tendon degeneration is characterized by loss of parallel collagen structure, fatty infiltration,

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loss of fiber integrity, and capillary proliferation (8), manifested by increased thickness and abnormal appearance of the tendon. A tendon thickness over 6 mm has been used as a diagnostic criterion for AE (2). Nicholson et al (2007) developed a grading system for Achilles tendon pathology based on MRI findings, which guided selection of the optimal treatment option. The grading system was based on the tendon's diameter and the presence of degeneration within its substance: (I) grade I, anteroposterior diameter of 6 to 8 mm and non- uniform degeneration, (II) grade II, diameter of >8 mm with uniform degeneration of <50% of tendon width, and (III) grade III, tendon diameter >8 mm and uniform degeneration of >50% tendon width (9). The authors concluded that individuals with grade I disease were much less likely to require surgery (13%) than individuals with grade II and III pathology (91% and 70%, respectively).

The presence of bony spurs appears to be more common in patients with AE (65%-80%) (10) when compared to people without foot pain (25%-35%). Chimenti et al, found that on average, the spurs were significantly longer on the symptomatic side (12.9 mm) than the asymptomatic side (8.9 mm, P = .01) and controls (3.5 mm, P = .03) (11). All the above findings indicate that the size of the enthesophytes, rather than the presence of enthesophytes, may be a contributing factor to the development of symptoms in AE.

Causative factors

Predisposing conditions associated with AE include running on hard surfaces, walking gait abnormalities, which exert excessive pressure on the calcaneus and/or its ligaments and nerves, obesity and inappropriate shoes. It is also sometimes associated with gastrocnemius tightness and plantar fasciitis. Mechanical overloading may also contribute to AE symptoms. Running imposes loads of 4 to 6 times the force of body weight on the Achilles tendon (12) and approximately 8% strain along the entire length of the tendon (13). Although the load on the tendon is less with walking, this task still imposes approximately 7% strain along the tendon length (14).

The posterior-superior calcaneal prominence known as Haglund's deformity is mostly an idiopathic condition. Heredity also plays an important role since intrinsic foot anatomy, such as pes cavus, can often be a predisposing risk factor. Plantarflexion of the calcaneus leads to constant irritation of the overlying bursa, which is the main causative factor of redness and swelling associated with Haglund's deformity. Varus deformity of the hind foot represents another risk factor. The tendon protects itself by forming a bursa, which eventually.

Achilles enthesopathy may be also an expression of ankylosing spondylitis. Finally, fluoroquinolone antibiotics increase the risk of Achilles tendinopathy or tendon rupture especially in people over age 60.

Clinical Presentation and diagnosis

The diagnosis of AE is based primarily on past medical history, physical examination and radiographic findings. Patients typically report tenderness upon palpation of the terminal 2 cm of the Achilles tendon. A well-defined area of swelling and redness can often be seen at the posterosuperior aspect of the calcaneus. Ankle range of motion should always be assessed, since limited ankle dorsiflexion and plantarflexion weakness, are common findings in patients with AE. Lastly, pain that is aggravated by physical activity as well as stiffness that is associated with prolonged rest represent common complaints.

The lateral weight bearing radiograph reveals the presence of a bony prominence (Haglund's lesion) at the posterosuperior aspect of the calcaneal tuberosity, enthesophytes and intratendinous calcifications. (fig.1) Calcaneal bursal swelling and increased density in pre-Achilles bursa may also be present (fig.2) (15). The shape and lucency of the Kager triangle on radiographic imaging can also be used to assess the presence of retrocalcaneal bursitis (16).

Apart from the presence of the posterosuperior spur on the Achilles tendon, MRI findings may also include synovial thickening of the retrocalcaneal bursa as well as thickening and high signal at the site of Achilles tendon insertion. (fig.3) Ultrasound

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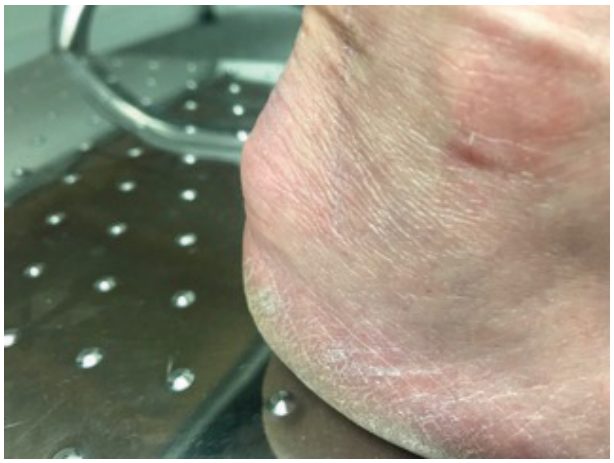

Fig.1 *A retrocalcaneal prominence may be either soft (retrocalcaneal bursitis) or hard (retrocalcaneal exostosis).*

imaging can be also used to evaluate soft tissue changes such as tendon degeneration, neovascularization, bursitis, paratendinitis, as well as bony changes namely enthesophytes and intratendinous calcification (11).

Conservative Treatment

1.Eccentric Exercises

Eccentric exercises such as open chain ankle dorsiflexion led to reduced pain and a higher level of patient satisfaction (Grade B recommendation). Closed chain eccentric exercises such as lowering the heel below the step, lead to less favorable clinical results with only 28% to 33% of patients with AE rating the intervention as excellent or good (Level IV evidence) (17). An alternative eccentric exercise program that utilizes a limited ankle range of motion with heel lowering to a past neutral standing position has been more successful for patients with AE. In a single case series study, Jonsson et al (18) had 67% (18/27) of patients reporting excellent or good results with pain rating decreasing from 72 to 33 on the VAS at 4 months (Level IV evidence).

Since 2010, 2 RCTs have compared traditional eccentric exercises to other treatment modalities such as extracorporeal shock wave therapy (19) and stretching (20). Traditional eccentric exercises do carry some therapeutic benefit, with an average decrease in pain ranging from 1.8 to 2.2 on the VAS

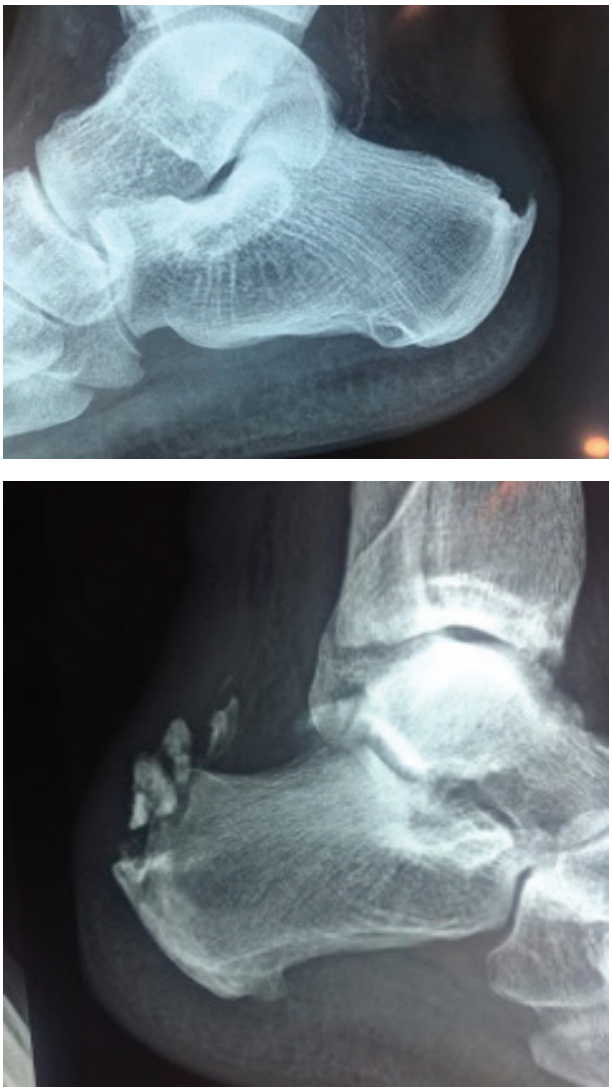

Fig. 2 *(a) Retrocalcaneal bony spur, and (b) Diffuse Achilles tendon intrasubstance calcification.*

(Level I evidence) at the 3 to 4months follow up (20). The modified eccentric exercise program also reduces pain when performed alone (average 2.4 decrease at 12 weeks and 4.4 at 52 weeks, n=8) and when combined with a soft tissue treatment (average 2.9 decrease at 12 weeks and 3.9 at 52 weeks, n=7; Level II evidence) (21). A well-established exercise regime is the Alfredson's protocol that enhances tendon eccentric loading. (34)

2. Extracorporeal Shock Wave Therapy

A randomized controlled trial by Rompe et al in 2008, concluded that extracorporeal shock wave

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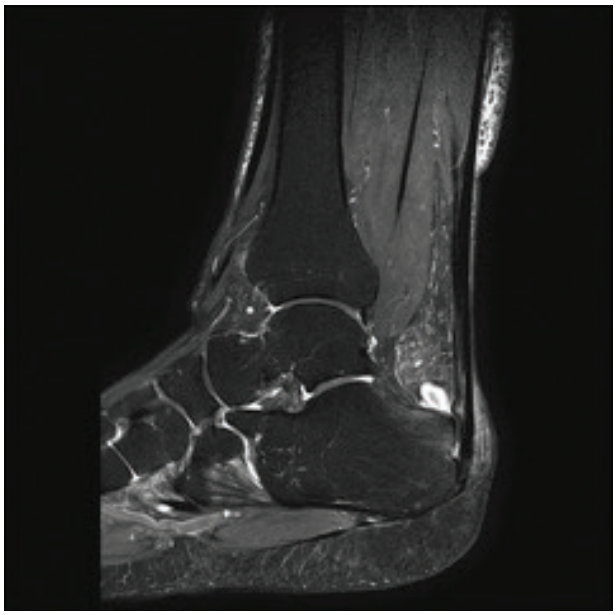

Fig. 3 *MRI T2 imaging showing retrocalcaneal bursitis and Achilles enthesopathy*

therapy (ESWT) was more effective at reducing pain and AE symptoms than a traditional eccentric exercise program at 4 months (Level I evidence) (19). In 2006, Furia_reported that ESWT in conjunction with anesthetics lead to an average pain reduction of 5 points on the VAS compared to 1.4 points in the control group (Level III evidence) (22). More recently, a retrospective study found that ESWT resulted in a greater decrease in pain than a traditional eccentric exercise program at 6 months follow up (average decrease in VAS, ESWT: 3.9, Eccentrics: 1.6) and 18-month follow-up (average decrease in VAS, ESWT: 3.6, Eccentrics: 1.5; Level III evidence) (23). Wei et al, however, reported a relatively high prevalence of intolerable pain among patients undergoing ESWT that lead to discontinuation of treatment in 20% of the patients (23). Local anesthesia may help patients tolerate the treatment better, but it is unclear whether this adjunctive scheme affects patient outcomes. Two recent Level IV studies have also supported the use of shockwave therapy for IAT (24-25) yet the authors question the effectiveness for patients with enthesophytes. Based on the number of studies supporting EWST (22-24-19-25), this modality now has a Grade B recommendation.

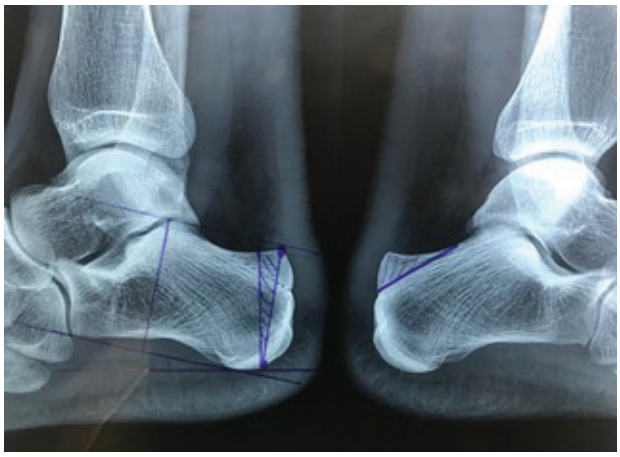

Fig.4 *The wedge osteotomy (left) and the oblique osteotomy (right).*

3. Orthotics

Night splints are less painful and better tolerated than weight bearing calf stretches; however, if a patient can tolerate weight bearing exercises, then night splints may add no additional benefit. In a Level II study, night splints did not provide any additional benefit added to eccentric exercise in patients with non-insertional Achilles tendinosis (Level II) (26). The evidence regarding night splints and insertional disease is lacking. In another Level II study evaluating the outcomes of physiotherapy, the effect of night splints could not differ from the concurrent effects of other treatment components (20). As such, this modality has a Grade I treatment recommendation.

Insoles with heel lift or shoes with a heel lift are commonly recommended. Heel lifts can reduce the amount of tendon elongation (tensile strain) and compression (compressive strain) that occurs at the tendon insertion during walking (27).

4. Local infiltrations

According to Irwin's 2010 review, there are no studies on the use of corticosteroid or glucocorticoid injections specifically for IAT (28). Corticosteroid injections have largely fallen out of favor for treatment of tendinopathy at any location, and there is particular concern around the Achilles tendon for fear of contributing to further tendon degeneration and potential tear (30). In cases of isolated retro-

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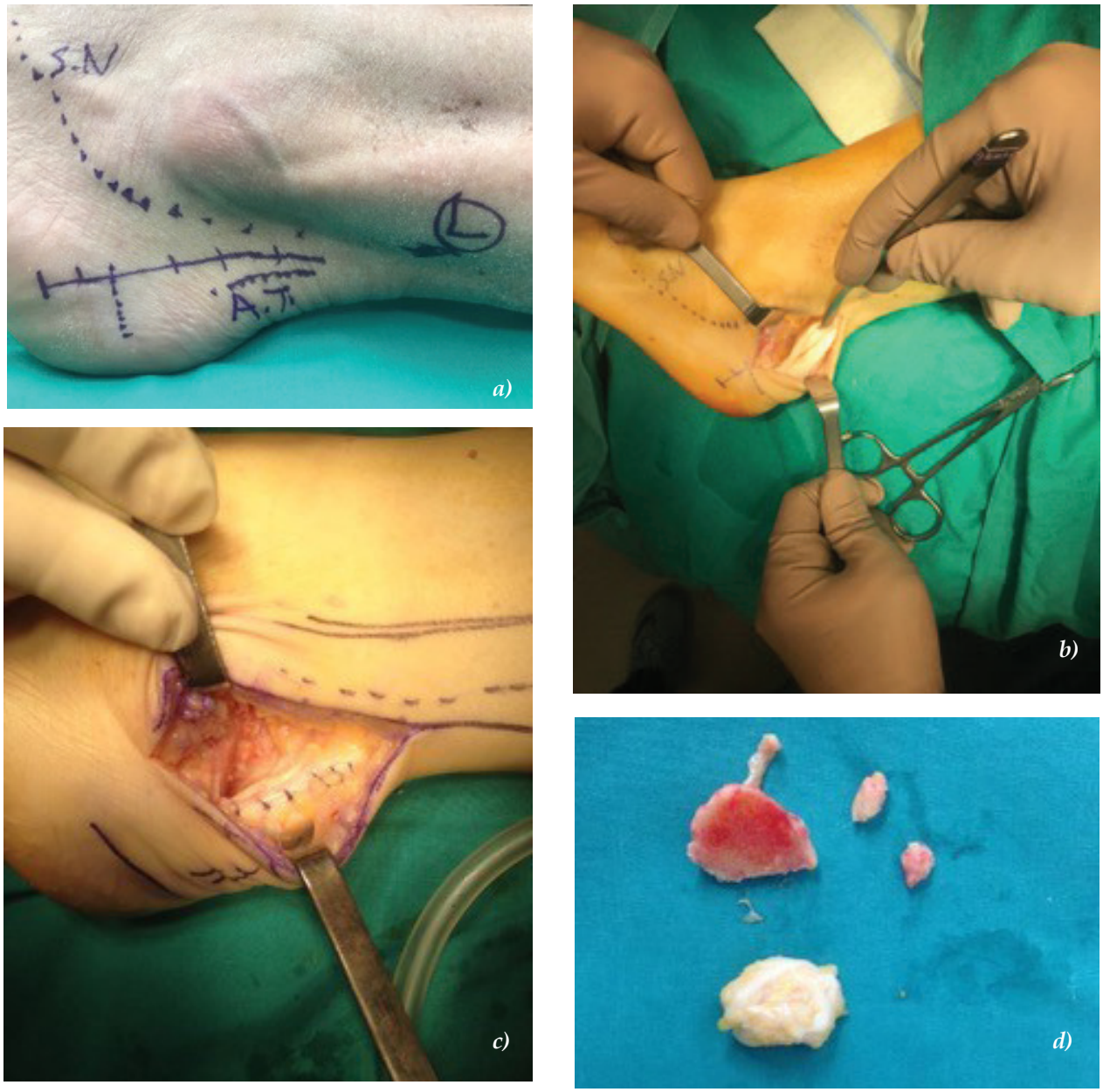

Fig.5 (a) Incision and anatomic landmarks, (b) oblique retrocalcaneal excision and longitudinal split of the tendon, (c) excision of the calcification and repair of the tendon with interrupted sutures, (d) the retrocalcaneal prominence, the intrasubstance calcification and the retrocalcaneal bursa.

calcaneal bursitis, corticosteroid injection may be considered, but care should be taken to avoid intratendinous injection.

Other potential types of injection include those targeting neovascularization, such as the sclerosing agent polidocanol or simple mechanical disruption with high volume saline and dextrose (prolotherapy). Sclerotherapy is poorly supported with only one study in literature evaluating the use of polidocanol in patients with chronic AE (Level IV evidence) (29). Although several small trials have studied these agents, most were not specific to AE and provide insufficient high-quality evidence to support their use in routine clinical practice (31). Quality evidence is therefore lacking regarding long-term efficacy of the agents for any insertional

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TABLE 1.

Proposed conservative treatment protocol for Achilles Enthesopathy in our dept.

Treatment	Effect
• Alfredson's heel stretching protocol, applied daily for 12 weeks with 180 repetitions (34).	• A series of eccentric exercises, slow movements that focus on lengthening muscle contractions of the calf.
• Use of night splinting	• Plantar fasciitis and calf stretching.
• Use of shoe insoles	• Heel lift and reduction of Achilles enthesis tension.
• Dextrose prolotherapy outside the tendon and on the paratenon x1-2 times.	• Reduction of neovascularization and triggering of healing immune response
• Application of glyceryl trinitrate patches for one month.	• For local blood vessel dilation and healing enhancement
• Oral nutraceutical supplements for two months (35).	• Per os supplement based on methylsulfonylmethane, hydrolyzed collagen, bromelain, D-glucosamine, chondroitin sulfate, L-arginine, L-lysine, plant extracts of boswellia, myrr and turmeric, and Vitamin C

Achilles disease (Grade I recommendation).

Most studies using PRP have been in patients with midportion Achilles tendinitis (AT) or are mixed cohorts with controversial results. A 2012 prospective case series by Monto, found PRP to be effective in a mixed cohort of 30 patients with AT (8 insertional, 22 midportion) leading to satisfaction in 28/30 patients at 2 years (Level IV evidence) (32). However, both treatment failures in this study occurred in patients with AE (2/8) (32).

5. Local glyceryl trinitrate patches

There is no evidence to support the application of glycerol trinitrate patches in patients with AE. Conversely, Hunte and Lloyd-Smith concluded that the local use of a glycerin trinitrate patch was more effective than placebo in patients with chronic Achilles tendinitis in the first 12 and 24 weeks of treatment (33).

6. Adjuvant therapeutic modalities

Per os administration of nonsteroidal anti-inflammatory medications, iontophoresis, and cryotherapy may be useful if substantial inflammation is present. However, they do not act on therapeutic pathways.

In our department, an holistic conservative therapeutic protocol is initially recommended before any surgical treatment. Following a thorough clinical and imaging evaluation, the patient is instructed to follow the protocol for a period of 3-6 months. (Table 1)

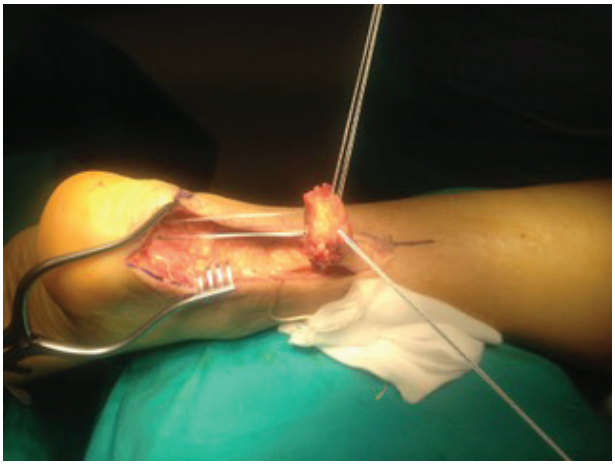

Fig.6 Detachment of the Achilles tendon insertion for removal of large intrasubstance calcification and re-attachment to the debrided calcaneus bed with anchor and heavy sutures.

Surgical treatment

If conservative treatment fails to provide adequate pain relief, surgery may be needed. In our department, we have established a certain protocol regarding the decision-making. In this protocol, surgical technique is based on the posterior morphology of

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the calcaneus and the intratendinous calcification of the tendon.

- In cases of a localized heel bump (**retrocalcaneal exostosis**) an oblique excision of the posterior tubercle would be satisfactory. (Fig. 4)
- In cases of a global posterior heel bump (**Haglund's deformity**) the Keck & Kelly calcaneal wedge osteotomy is indicated. The wedge osteotomy has many advantages such as: (a) it tilts prominence forward away from the shoe, (b) it elevates the insertion of Achilles tendon and reduces equinus stress, and (c) it provides a straight orientation of Achilles tendon fibers at the calcanea insertion. (Fig. 4)
- In cases of additional **focal intratendinous calcification,** a longitudinal incision and split of Achilles tendon fibers may lead to the accumulated calcium. The calcified tissue is removed, and the tendon is sutured. The longitudinal incision has a minor risk for Achilles tendon rupture. (Fig.5)
- In cases of additional **diffuse calcification**, the tendon is fully detached from its calcaneal insertion, debrided and reattached with special anchoring techniques. (Fig. 6)

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