Achilles Tendinopathy
The name ‘Achilles Tendon’ is derived from the Iliad by Homer.

Achilles was made invulnerable by his Goddess mother Thetis, who plunged him into the river Styx, while holding him by one heel. This part was not bathed and hence not protected.

This heel was the site were he was mortally wounded by a poisoned arrow launched from the bow of ‘Paris’ during the Trojan war.
Introduction

- Previously known as “Achilles Tendinitis”
- Minimal acute inflammation
- Better referred to as “Achilles Tendinopathy”
- Clinical syndrome of Pain, Swelling and impaired performance in the Achilles Tendon
Epidemiology and Incidence

- Non-I AT is most common in 30-40s, M > F
- Seen in 7-9% of top level mid-long distance runners
- Also seen in Soccer, track and field, tennis
- In athletes with tendinopathy – 66% have non-insertional, 23% insertional
- I AT is seen in an older less active population
Aetiology of Pain

• Not yet completely determined
• Is it?
  – intratendinous degenerative changes
  – expansion of tendon from increased ground substance and GAG concentration
  – neovascularisation
• Up to 34% of asymptomatic tendons had histopathological changes in one study
• Ingrowth of sensory/sympathetic nerves from paratenon plays a role.
Achilles Tendinopathy - Histology

- “Degenerative and Overuse condition with attempts of Repair”
- Collagen degeneration, fibre disorganisation and thinning with hyalinisation and fibre separation by increased ground substance (GAGs)
- Scattered vascular ingrowth with areas of necrosis and calcification
- Nerve ingrowth occurs
- Zone of hypovascularisation
Classification

- Midportion Achilles Tendinopathy
- Paratendinopathy
  - Acute or Chronic
- Insertional Achilles Tendinopathy
- Retrocalcaneal Bursitis
- Superficial Calcaneal Bursitis

— Others — Pudda classification
Clinical

- Initially - Start up pain, at end of activity
- Progresses to during activity
- Then interferes with ADLS, shoe wear problems
- Acutely can be swollen, erythematous and tender +/- crepitations
- Chronically – activity related pain, crepitations and swelling decrease, with tender nodular swelling
Examination

- Paratendinosis Vs Tendinosis
  - Arc test – swelling in the tendon will move with plantar/dorsiflexion of the ankle
  - Royal London Hospital test – swelling that is most painful becomes less tender with maximal dorsiflexion indicates tendinopathy

- Insertional Vs Non-insertional

- Calcaneal exostosis (Haglunds deformity)
Examination

• Retrocalcaneal bursitis
  – Pinch test – pinch in front of distal TA may reproduce pain, and with dorsiflexion may intensify the pain.
• Sensitivity 50-60%, Specificity 80-90%
• Exclude
  – posterior impingement
  – Acute ruptures
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Imaging

- **XR**
  - Parallel pitch lines
- **Ultrasound**
  - User dependant
  - Colour doppler can show areas of neo-vascularisation
- **MRI**
Treatment

• Relieve symptoms initially
  – Rest, cross train, ice, shoe modifications, simple analgesia, heel raise/brace (AirHeel)
  – NSAIDS (little evidence in chronic tendinosis)
  – Corticosteroids (controversial- risk of rupture small and little evidence to support it)
    • Better for peritendinopathy/retrocalcaneal bursitis
  – Correct training errors
    – MLA orthotics in hyperpronators
Treatment

- Eccentric stretch and strength program
  - Less effective in insertional AT
- Transcutaneous Glyceryl Trinitrate (GTN)
  - Some evidence to support its use (Paoloni 2007)
  - Watch out for headaches
Treatment

• Sclerosant injections (Polidocanol)
  – Destroys neovessels and nerves
  – Small risk of rupture

• Volume injections (Brisement)
  – High volume saline/LA/hydrocortisone
  – Destroys neovessels and nerves, releases adhesions
  – Some evidence to support its use (Chan 2008)
  – Better for peritendinosis
Treatment

• Prolotherapy
  – Hyperosmolar dextrose and LA peritendinous injection, creates an inflammatory response
  – No good supportive evidence
• Shock-wave therapy
  – May be more beneficial in insertional/calcific
  – Comparable results have been seen to eccentric loading (Rompe 2007 AmJSM)
Treatment

• PRP injections
  – No evidence to justify its use (De Vos 2010 JAMA)
  – He compared eccentric program with either saline or PRP injections
Treatment

• Low-level laser therapy
  – Insufficient evidence to support its use
Treatment - Surgical

• Usually involve removal of inflamed or diseased tissue and decompression of mechanical pressure from adjacent calcaneus +- tendon transfer

• Complication rate up to 11%
  – Mainly wound healing
  – Therefore skin and vascular compromise are contraindications
Treatment - Surgical

- Percutaneous longitudinal tenotomies
  - Better for non-insertional
- Achilles Tendonscropy with release of adhesions, tendon debridement
- Plantaris excision + ventral Achilles tendon scraping
- Calcaneoplasty (Haglund's deformity debridement)
  - Open or endoscopic
Treatment - Surgical

• FHL muscle belly transfer
  – Used in non-insertional tendinopathy
  – bring blood supply to tendon

• FHL tendon transfer
  – If more than 50% of tendon debrided
  – Patients notice Hallux weakness, esp when bearfooted

• Other options include FDL and Plantaris
Treatment - Surgical

• Reconstruction
  – Debridement with a large gap remaining
  – Options include
    • Primary repair (less than 3cm)
    • V-Y lengthening or Turndown flap (3-6 cm)
    • Tendon transfer (usually FHL) (when > 6cm)
• Thank you