MANAGEMENT OF ACHILLES TENDON DISORDERS

A Case Review

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Abstract: A case of heel pain is presented. The clinical presentation implicates the pathological processes of acute retrocalcaneal bursitis, Achilles tendinosis, partial rupture of the tendon possibly as a result of repetitive corticosteroid injection, and subsequent calf atrophy and functional disability. Finally, aspects of predisposing factors, biomechanics, pathology, and clinical management of Achilles tendon disorders are reviewed in the discussion.

INTRODUCTION

Achilles tendinitis is one of the most common injuries in sport. It typically affects mature male athletes engaged in a high degree of running and jumping activities. These injuries account for between 6% (1) and 15% (2) of all running injuries.

The reported male to female incidence of Achilles rupture has varied from 2:1 to 12:1. Recent reports indicate that 75% of all Achilles tendon ruptures occur in athletes between the ages of 30 and 40 years old (3).

CASE REPORT

A 43 year old male presented himself with heel pain. He reported similar heel pain many times before, and each time his GP gave him an injection at the heel and the pain was relieved. The present episode of pain had lasted for one week. He was unwilling to have further injection and anti inflammatory medication offered no relief. He related this episode of pain with busy work. He reported that he had given up jogging a few years ago since the heel pain. For the last year his family had noticed that he had an abnormal gait.

On examination the posterior heel at the Achilles tendon insertion was tender on palpation. There was slight local swelling. Pain was otherwise minimal along the tendon. Thompson's test was negative, and there was active although painful plantar flexion of the foot. A large nodule could be palpated in the tendon about one inch proximal to the calcaneal insertion. The calcaneum was held in a valgus position and the foot was pronated. His limping gait showed no toe-off from the injured foot. The calf muscle had atrophied significantly. A single toe raise was tested in a subsequent visit when the pain was less severe and he failed to perform this manoeuvre successfully.

He was diagnosed as suffering from acute retrocalcaneal bursitis, secondary to chronic Achilles tendinosis and partial rupture of the tendon. Ultrasound and MRI examination would be very helpful in differentiating between tendinitis, paratendinitis, focal degeneration or a partial tear. However it was regarded as not necessary in the initial management of the case.

He was initially treated with ice, pulsed ultrasound, and a heel wedge. Stretching and strengthening exercises of the plantar flexors were given as the patient could tolerate. The acute pain subsided after two weeks (4 visits) of treatment. The ankle joint was mobilised and manipulated when the acute pain had subsided. The talocrural joint was mobilised with anteroposterior gliding motion and the talocalcaneal joint was mobilised in the pronation-supination direction. The talocrural joint was manipulated in the long axis. The patient returned two months later and reported mild calf soreness. He was advised to continue the stretching and strengthening exercises for the gastro-soleus muscle.

ANATOMY

The Achilles tendon arises as two broad sheets of tissue in the mid-calf which coalesce as an oval structure that extends to the distal aspect of the calcaneal tuberosity. The fibres of the tendon twist about on one another for a quarter turn as the tendon descends to the calcaneus (4).

There is no synovial sheath around the Achilles tendon; it is covered by the peritenon which consists of a series of thin membranes. This layer enhances tendon gliding by minimising friction between the tendon and the surrounding tissue. Inflammation of this layer with subsequent thickening and adhesion formation can result in diminished tendon flexibility and predispose to further injury (5).

Micro-vascular studies indicate that there is an area of

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relative avascularity just proximal to its insertion into the calcaneus (6). As a consequence, this area is most susceptible to tendinitis and rupture.

The Achilles tendon is the strongest and largest tendon in the body and it is subjected to the highest of forces in the body; tensile loads of up to eight times body weight are experienced during running. The medial head of the gastrocnemius is the most active during running (7). The soleus muscle does not cross the knee joint and is composed of 80% type I fibres; therefore, it has a significant potential for rapid atrophic change with disuse and immobilisation.

PREDISPOSING FACTORS

Achilles tendon overuse injuries can occur as a result of poor body mechanics, training errors, or environmental factors, or they may be a manifestation of systemic disease such as gout and ankylosing spondylitis.

In an extensive review of runners, it was found that training errors caused Achilles tendinitis in 75%, hyper-pronation in 56%, poor gastrocnemius-soleus flexibility in 39% and improper shoes in 10% of the runners (8).

Inadequate warm-up and improper stretching techniques predispose to injury by failing to increase muscle flexibility. Ruptures occur with rapid eccentric loading as the foot and ankle are in dorsiflexion and the knee is extended with a contracted soleus muscle. The tensile strength of the tendon has been calculated to be approximately 50-100 N/mm^2. The elongation at rupture has been calculated to be approximately 10%. With Achilles tendon rupture, these calculation should imply a tensile strength of 4000 to 5000 N and an elongation of 1 to 2 cm (9). Protective measures such as warming up are important to allow for creep (increased deformation with time under constant load), stress relaxation (decreased stress with time under constant deformation) and a temperature rise in the tendon and the peritendinous tissues, which decreases the stress level in the tissues (9).

Exercising surface characteristics have been traced to the onset of symptoms. Achilles tendon injuries are commonly seen in recreational runners who train on a banked or hard surface or whose routine includes significant hill running.

The classical ballet positions of en pointe and revele exert tremendous force on the Achilles tendon by demanding extreme ankle plantar flexion. The demi pile and grand pile position require marked ankle dorsiflexion with eccentric contraction of the gastroc-soleus complex that can result in tendon overuse.

Improper footwear, in particular, worn or poorly designed running shoes can predispose to the development of Achilles tendon injuries. A good running shoe should have a slightly flared and wedged heel and a firm heel counter to prevent excessive hindfoot varus and valgus rotation.

Subtalar pronation is associated with tibial internal rotation and increased midfoot flexibility. Pronation is normally initiated immediately following heel strike, and it progresses during the early stance phase of gait. Subtle malalignment of the lower extremity can prolong the duration of pronation and increases the degree of twisting that the Achilles tendon undergoes and results in increased rotational and shear forces within the structure (8,10).

Increased femoral anteversion increases the duration of subtalar pronation by requiring persistent internal rotation of the limb to optimally centre the hip. Varus deformity of the knee, tibia, heel or forefoot results in the heel striking the ground in a varus position and promotes a compensatory pronation to maintain the foot in a more plantargrade position.

A tight triceps surae creates a functional heel varus that is accompanied by increased compensatory pronation. Achilles tightness results in a functional ankle equinus contracture and induce tendon overuse (2). Age-related changes in tendon include increased tissue stiffness combined with diminished tendon tensile strength and strain to failure (4).

PATHOLOGY

A popular theory of tendon overuse injury is that the inability of the tendon to resist a repetitively applied load results in micro tearing (4). The theory was supported in a rabbit model experiment where an Achilles tendinosis was induced (11).

Injuries of the Achilles tendon can vary from mild inflammation of the surrounding tissue (paratendinitis) to structural alteration of the tendon (tendinosis).

Overuse injuries of the Achilles tendon progress through several well-defined stages that are characterised by the site of involvement (12).

The paratenon is initially involved with increased vascular permeability and formation of immature scar tissue (13). Thickening of the paratenon impairs the gliding function of this tissue, thereby intensifying the inflammatory stimulus. If the stimulus persists, such as when an athlete tries to work through pain, scarring of the paratenon and structural disruption of the tendon can occur.

Chronic Achilles tendinitis demonstrated nodules with hypoxic and mucoid degeneration and calcific tendonopathy, as well as fissuring within the substance of the tendon (6). Collagen breakdown and subsequent release of proteolytic enzymes predispose the tendon further to degradation. Thickening of the peritenon occurs; with continued overuse, there is a relative increase in the avascularity of the tendon proximal to its calcaneal insertion.

The spiralling architecture of the Achilles fibres supports the hypothesis that within the Achilles a wringing effect can produce, resulting in vascular compromise to the most central region of the tendon.

Tendon rupture is usually associated with tendinosis, and typically occurs 4 to 6 cm proximal to the calcaneal insertion site (14). This anatomical area is the narrowest portion of the tendon, has the smallest cross-sectional diameter and has the greatest cross-sectional fibre rotation. Langerfren and Lindholm (15) demonstrated that this area has the poorest blood supply and that the blood supply decreases with age. In a controlled study of 891 patients, Kannus and Jozsa (6) showed the presence of degenerative changes in all ruptured tendons. Corticosteroid injection for Achilles Tendinitis has been implicated for the increased risk of tendon rupture (16, 17). Intra-articular as well as intra-tendinous injections have been shown to cause weakening of ligamentous and tendinous structures, possibly by the corticosteroid's inhibitory effect on the synthesis of proteins and collagen (18-20).

CLINICAL FEATURES

The diagnosis of Achilles tendinitis should be soundly based on a thorough history along with a detailed clinical examination that includes observation of the patient's gait and inspection of their shoes.

A careful history must be elicited in patients presenting with Achilles tendon complaint. In particular, the changes in the level of physical activity, and changes in footwear should be sought. The quality of the patient's symptoms should also be established. Morning stiffness has been noted in the more protracted cases. Pain during sprinting or acceleration almost always correlates with surgical findings of tendinosis or partial rupture (10).

Physical findings vary with the degree of tendon involvement. Decreased ankle dorsiflexion and hamstring tightness are common findings. Pain is localised to the inferior aspect of the posterior calf. Tenderness to palpation is usually present 3 to 6 cm above the tendon insertion. A tender, nodular swelling is believed to signify tendinosis and is usually present in patients with more chronic symptoms. Swelling and tenderness to palpation anterior to the Achilles tendon is more consistent with retrocalcaneal bursitis. Performance parameters such as toe raise capability and maximum vertical jump may be impaired.

TREATMENT

Tendon repair has long been recognised to follow the three stages of soft tissue healing: inflammation, repair and remodelling, and maturation.

Researchers found that early controlled mobilisation of healing canine tendons has led to a significant reduction of adhesion formation and stronger tendons upon final healing (21,22).

Effective management of the Achilles tendinitis is based on reducing the effects of intrinsic and extrinsic variables by altering training techniques and modifying footwear to accommodate the individual patient's anatomic deficiencies.

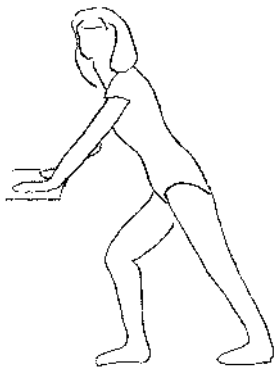
Initial management includes a course of rest, liberal use of ice, analgesics and use of modalities.

Stretching is the most important component of the treatment with overuse injury. The gastrocnemius and soleus are stretched with wall lean exercises (Figure). Concentric and eccentric strengthening of the plantar flexors is instituted in the early period of rehabilitation.

Figure

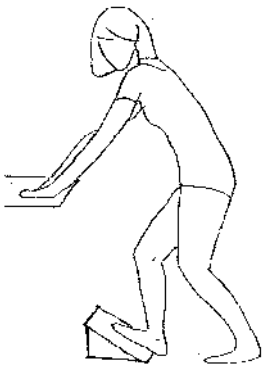
(A) Gastrocnemius stretch.

The athlete stands and leans towards a table (or wall) with the weight on the heels and the back knee straight. The feet are turned inward in a slightly pigeon-toed position and the pelvis pointed forwards. The athlete slowly leans forward until a stretch is felt in the calf, and holds for 30-60 seconds. Repeat stretch 3-4 times.



(B) Soleus stretch.

Same position as for gastrocnemius stretch. An inclined board can be used for more effective stretching. The athlete slowly leans forward with the weight on the heels and the knees slightly bent until a stretch is felt in the calf. Holds for 30-60 seconds. Repeat stretch 3-4 times.



Chiropractic mobilisation and manipulation of the talocrural and the subtalar joints help to restore proper motion and function, and aid in proper alignment of the healing tissues.

Orthotic devices may be required to correct any underlying heel valgus and help to restore the normal arch. Heel wedge (3/8 inch) can help lessen patient's symptoms during daily activities.

Patient with paratendinitis, tendinosis and partial rupture may require conservative treatment and rehabilitation for several months before full return to sports participation is possible.

Achilles tendon ruptures can be repaired surgically and cast for approximately 8 weeks. Beskin (17) showed that the operative patients had increased strength and a decreased re-rupture rate. Inglis (23) and Wills (24) demonstrated that patients who had open repair were in fact stronger. Surgical intervention in young active athletes is advocated. Nevertheless, low demand recreational athletic and non athletic patients, especially more than 50 years of age, have been shown to do well when treated non operatively acutely or early after the rupture (25-27).

CONCLUSION

Successful conservative treatment of Achilles tendon injuries is based on the thorough understanding of the operative etiologic factors and the pathological processes involved. Conservative management involves measures to control pain and inflammation, chiropractic mobilisation and manipulation of the ankle and subtalar joints, stretching and strengthening of the plantar flexors, change in athletic training program, and correction of faulty biomechanics. Corticosteroid injection in or around the tendon is not advised. Surgical repair is indicated in complete Achilles tendon rupture in young athletic candidates who demand maximum Achilles tendon strength for performance.

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