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Etiology and Epidemiology of Achilles Tendon Problems

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Introduction

Rupture of the Achilles tendon is on the increase,[1,2] and appears more common in men in the white-collar professions.[3,4] Various hypotheses have been put forward as to why the Achilles tendon ruptures.[3,5–8] Tendinopathy of the Achilles tendon is common both in athletic and nonathletic individuals.[9–12] As in ruptures of the Achilles tendon, its etiology is likely to be multifactorial.[13,14]

Acute Ruptures

Two main theories are advocated, the "degeneration theory" and the "mechanical theory." According to the degeneration theory, chronic degeneration of the tendon leads to a rupture without excessive loads being applied. Degenerative changes can result from several factors, including age-related alterations in the tendon, chronic overloading with microtrauma, drug treatment, and in association with other diseases.

Kannus and Jozsa[5] evaluated specimens obtained from the biopsy of spontaneously ruptured tendons in 891 cases; 397 of these ruptures were of the Achilles tendon. They compared the histopathology of the 397 Achilles tendon ruptures with 220 control tendons using conventional and polarized light microscopy, and also scanning and transmission electron microscopy. The control specimens were age- and sex-matched for 445 tendons taken at the time of death from the cadavers of previously healthy individuals who died accidentally. A healthy structure was not seen in any spontaneously ruptured tendon, whereas two-thirds of the control tendons were structurally healthy. There were characteristic histopathological patterns in the spontaneously ruptured tendons. Most (97%) of the pathological changes were degenerative, with hypoxic (45%), mucoid (19%), tendolipomatous (6%), and calcifying tendinopathy (3%), either alone or in combination. These changes were also found in 31% of the control tendons. The findings indicated that, at least in an urban population, degenerative changes are common in the tendons of subjects older than 35 years and that these changes are associated with spontaneous rupture.[5]

Disruption of the homeostasis of extracellular matrix components such as the fibrillar collagens and the proteoglycans may predispose to rupture. Type I collagen comprises 95% of collagen in normal tendons. This parallel arrangement imparts high tensile strength to the tendon. Maffulli et al.[6] showed that Type I collagen is the main collagen in normal tendons with type III collagen being present in small amounts. However, in ruptured Achilles tendons there was a significantly greater proportion of type III collagen, which is less resistant to tensile forces and may predispose the tendon to spontaneous rupture.[6] An in vitro model was used to determine whether tenocytes from Achilles tendons that are ruptured, nonruptured, tendinopathic, and fetal exhibit different behavior. Samples of Achilles tendon were digested with collagenase and the released tenocytes were collected. Primary tenocyte cultures were established and subsequently cultured onto

glass coverslips. Once a confluent monolayer was obtained, the cell populations were "wounded" by scraping a pipette tip along the surface. The cultures were further incubated for either 1, 4, 8, 12, 16, or 24 hours, and production of the collagen types was assessed by immunostaining. Cultures from ruptured and tendinopathic tendons showed increased production of type III collagen.

Athletic participation places excess stress on the Achilles tendon, which could potentially lead to areas of localized microtrauma within the tendon. These areas may heal by the production of type III collagen, an abnormal healing response. Accumulation of such episodes of microtrauma could result in a critical point where the resistance of the tissue to tensile forces is compromised, and tendon rupture occurs.[6] This leads to the conclusion that ruptured tendons produce and exhibit significantly greater proportions of type III and reduced type I collagen, together with significantly higher degrees of degeneration, than nonruptured tendons.

Birk and Mayne[15] investigated the localization of collagen types I, III, and V during different stages of tendon development. The tendon fascicles and their connective tissue investments (endotendenium) were studied. The data show a changing pattern of type III collagen expression in the developing tendon. The increases in diameter are associated with a decrease in type III collagen reactivity. During all stages of tendon development there is a constant, small but detectable amount of type V collagen. However, no correlation between type V reactivity and fibril diameter was observed at any stage of development. These results indicate an inverse relationship between type III collagen reactivity, and fibril diameter in the developing tendon.[15]

Magnusson et al.[7] tested the hypothesis that collagen fibril diameter and crimp angle in ruptured human Achilles tendons differed from that of intact ones. Although crimp morphology is unchanged, there appears to be a site-specific loss of larger fibrils in the core and periphery of the Achilles tendon rupture site. Moreover, the lack of symptoms prior to the rupture suggests that clinical tendinopathy is not an etiological factor in complete tendon ruptures.[7]

The role of versican, the principal large proteoglycan expressed mid-tendon (with regard to

tendon pathology), has recently been investigated. Corps et al. attempted to define the expression of versican isoform splice variant messenger ribonucleic acid (mRNA) in normal Achilles tendons, in chronic painful tendinopathy, and in ruptured tendons.[16] Changes in versican expression relative to that of collagen observed in ruptured Achilles tendons may have contributed to the changes in matrix structure and function and therefore contributed to the etiology of the rupture.

Decorin is a prototype member of the family of the small leucine-rich proteoglycans (SLRPs). It plays a significant role in tissue development and assembly, as well as playing both direct and indirect signaling roles. It modulates collagen fibrillogenesis, and is a vital player in maintaining tendon integrity at the molecular level.[17] The glycosaminoglycans bound to decorin act as bridges between contiguous fibrils, connecting adjacent fibrils every 64–68 nm. This architectural arrangement suggests a possible role in providing mechanical integrity of the tendon structure. Laboratory evidence suggests that fluoroquinolone antibiotics decrease decorin transcription,[18] which may alter the viscoelastic properties of the tendons and induce increased fragility. Bernard-Beaubois suggested that perfloxacin, a fluoroquinolone, does not affect transcription of type I collagen, but decreases the transcription of decorin at a concentration of only 10^{-4} millimoles.[18] The resulting decrease in decorin may modify the architecture of the tendon, leading to altered biomechanical properties and increased fragility.

Clinical painful tendinopathy is not common before complete Achilles tendon ruptures.[7] Most patients who sustain an acute Achilles tendon rupture are asymptomatic prior to injury. Nine (5%) of the 176 patients presenting with a rupture of the Achilles tendon in Aberdeen, Scotland, between January 1990 and December 1995 had had previous symptoms.[3] However, Nestorson et al. reported that among 25 Achilles tendon rupture patients over 65 years age, 11 (44%) had had Achilles tendon symptoms, and 7 of those had received local cortisone injections.[19] Although clinical painful tendinopathy seemingly is a risk factor, patients with chronic Achilles tendinopathy in reported nonsurgical and surgical series have had a long duration of symptoms (several months or years) without sustaining a rupture.[20,21]

On the other hand, at the time of Achilles tendon rupture, degeneration and necrosis were present in 47 of 50 and 42 of 50 of the contralateral asymptomatic Achilles tendons, respectively. Spontaneous rupture of the Achilles tendon seems to be preceded by widespread, bilateral tendon damage.[22] Additionally, the patients' asymptomatic contralateral Achilles tendons showed a greater prevalence of intratendinous alterations at ultrasonography.[23] Simultaneous bilateral ruptures without preceding factors are very rare.[24] However, ruptures of both Achilles tendons at different times have been reported in up to 6% of the patients with no preceding factor.[25]

Achilles tendon rupture may result from a drug adverse event. Fluoroquinolone antibiotics such as ciprofloxacin have been implicated in the etiology of rupture of the Achilles tendon during the last decade.[8,26] In France, between 1985 and 1992, 100 patients treated with fluoroquinolones had tendon disorders that included 31 ruptures.[26] Szarfman et al. demonstrated disruption of the extracellular matrix of cartilage as well as depletion of collagen in animals that received fluoroquinolones.[27] This may also apply to humans.

Local and systemic corticosteroids are administered for a variety of diseases and have been widely implicated as a risk factor for tendon rupture.[28] Both oral and peritendinous steroid injections have been associated with Achilles tendon rupture.[29,30] Balasubramaniam and Prathap injected hydrocortisone into the calcaneal tendons of rabbits, thus inducing necrosis at the site of injection 45 minutes after the injection.[31] Tendons injected with corticosteroid had a delayed healing response compared with those injected with saline solution. Newnham et al. reported a series of 10 patients attending a respiratory outpatient clinic taking oral corticosteroids, who subsequently ruptured their Achilles tendon in the course of 12 years.[29]

There has recently been some evidence to support peritendinous steroid injections, providing the needle does not pass directly into the Achilles tendon body. Gill et al., in a retrospective cohort study of 83 patients,[32] established the safety of low-volume injections of corticosteroids for the management of Achilles tendinopathy when the needle is carefully inserted into the peritendinous space under direct fluoroscopic visualization. In this study, although 23 patients (53%) did not report any improvement, only 3 patients (7%) felt that their condition was any worse. Alfredson found normal prostaglandin E2 (PGE2) levels in chronic painful tendinosis (Achilles and patellar) tendons, showing that there is no PGE2-mediated intratendinous inflammation in the chronic stage of these conditions.[33] The neurotransmitter glutamate (a potent modulator of pain in the central nervous system) was, for the first time, found in human tendons. Microdialysis showed significantly higher glutamate levels in chronic painful tendinosis (Achilles and patellar) tendons, compared with pain-free normal control tendons. A specially designed treatment, using ultrasound and Doppler-guided injections of the sclerosing agent Polidocanol, targeting the neovessels outside the tendon, has been shown to cure tendon pain in pilot studies in a majority of the patients. Many authors feel injections of corticosteroids in patients with established Achilles tendinopathy are to be avoided.[34] Speed reviewed the literature on corticosteroid injections involving tendinopathies and concluded that there was no good evidence to support corticosteroid injections.[35] It is possible that the anti-inflammatory and analgesic properties of corticosteroids may mask the symptoms of tendon damage, and individuals will continue to maintain high levels of activity even when the tendon is damaged.[36]

Spontaneous rupture of the Achilles tendon has been associated with many disorders, such as inflammatory and autoimmune conditions,[37] genetically determined collagen abnormalities,[38] infectious diseases,[39] neurological conditions,[3,40] and hyperlipidemia.[41,42] A disease process may predispose the tendon to rupture from minor trauma.[43] Blood flow into the tendon decreases with increased age[44] and the area of the Achilles tendon more prone to rupture is relatively avascular compared with the rest of the tendon.[45–47]

High serum lipid concentrations have been reported in patients with complete ruptures of the Achilles tendon.[41,42] Although there is uptake and excretion of sterols by the enzyme sterol 27-hydroxylase (CYP27A1) in the Achilles tendon,[48] histopathological evidence of lipomatosis was only found in 6% of specimens from Achilles tendon ruptures.[5] Further, patients with familiar hypercholesterolemia and Achilles tendon xan-

thomata do not appear to be at greater risk of ruptures.

When considering the "mechanical theory" in relation to acute rupture, McMaster proposed that a healthy tendon would not rupture even if subjected to severe strain.[43] Barfred (in three 1971 papers)[49–51] investigated this hypothesis, and noted that, if straight traction were applied to the tendon, the risk of rupture would be distributed equally to all parts of the muscle-tendon-bone complex. However, if oblique traction is applied, the risk of rupture is concentrated on the tendon. Subjecting a 1.5-centimeter-wide Achilles tendon to traction in 30° supination on the calcaneus leads to elongation of the fibers on the convex aspect of the tendon by 10% before the fibers on the concave side are strained.[49] This means that the risk of rupture would be greatest when the tendon is obliquely loaded with the muscle in maximum contraction, and when the initial length of the tendon was short. In sports requiring rapid push-off, the above factors are likely to be present.[52] Even if a tendon is healthy, there is still a chance of rupture if the strain on the muscle is strong enough, especially in the presence of certain functional and anatomical conditions.[3] Sports are commonly associated with problems of the Achilles tendon, and training errors will increase the risk of these problems.[9,12,53] Clement et al. investigated 109 runners with Achilles tendinopathy.[54] In this series, the three most prevalent causes were overtraining (82 cases), functional overpronation (61 cases), and gastrocnemius/soleus insufficiency (41 cases). Clement et al. speculated that runners would be susceptible to Achilles tendinopathy due to microtrauma produced by the eccentric loading of fatigued muscle.[54] Excess pronation produces a whipping action of the Achilles tendon. Vascular blanching of the Achilles tendon is produced by conflicting internal and external rotatory forces imparted to the tibia by simultaneous pronation and knee extension. Equipment can also be linked with Achilles tendon problems; the flared heel on some sports shoes can force the hindfoot into pronation when the heel strikes the ground.[29]

Intrafibrillar sliding is the process by which tendons are initially damaged at the submicroscopic fibrillar level. This may apply to tendons that rupture without previous degenerative changes.

This process occurs a few seconds before macroscopic slippage of collagen fibers, implying that tendons unaffected by degenerative changes may rupture due to accumulation of fibrillar damage. This supports the theory of complete rupture being due to multiple microruptures and the tendon reaching a critical end point prior to rupture. Knorzer et al. illustrated this by using X-ray diffraction spectra to study the behavior of the structure of collagen during tendon-loading.[55] Not only slow or very fast elongation, but also very fast unloading of stretched fibers seems to be responsible for disseminated damage, which reduces the stability of a fiber.

Consideration of temperature in relation to rupture is important, as 10% of elastic energy stored in tendons may be released as heat.[56] Although research has not been performed on human tendons, Wilson and Goodship[14] used equine models to mathematically model tendon thermodynamics. They predicted that the temperature of the central core of the equine superficial digital flexor tendon would plateau at 11°C above the tendon surface temperature during a sustained gallop. Peak intratendinous temperatures in the range 43–45°C were recorded. Temperatures above 42.5°C may result in fibroblast death in vitro.[57] These in vivo recordings provide a possible etiology for the degenerative changes observed in the central core of tendons in both equine and human athletes, and a link with exercise-induced hyperthermia.

Epidemiology of Acute Rupture

Achilles tendon rupture usually occurs in middle-aged men working in a white-collar profession during sports activities.[58] Its incidence has increased during the last decades, at least in Northern Europe and Scotland.[3,59] Leppilahti et al.[2] found an increased incidence of Achilles tendon ruptures in Oulo, Finland from 2/100,000 in 1979–1986 to 12/100,000 in 1987–1994, and also demonstrated a bimodal age distribution. The incidence was highest in the age group 30–39 years with a smaller peak incidence between 50 and 59 years. The mean age was significantly lower for patients experiencing rupture during activities.

Patients with Achilles tendon rupture can be classified into two subgroups, namely young or middle-aged athletes and older nonathletes. Epidemiological data from Malmö, Sweden[59,60] have shown an incidence curve with two peaks:[60] a larger one in young, middle-aged individuals and a smaller one in patients in their seventies. Compared with the age-specific incidence in 1950–1973, there was a marked increase in both sports and nonathletic injuries, with patients in the latter group older than in the former period. Of all spontaneous tendon ruptures, complete Achilles tendon tears are most closely associated with sports activities, with 60% to 75% of all Achilles tendon ruptures related to sports.[61,62] In 430 tendon ruptures, the number of sports-related Achilles ruptures was approximately 62%, similar to the two studies mentioned earlier, while only few (2%) ruptures on other tendons were sports-related.[63] The increase in the athletic group is mostly explained by increased participation in recreational sports. The cause of increase in the elderly group is unknown, though 13% of ruptures occur in patients older than 65 years.[19] Patients with a spontaneous Achilles tendon rupture are at increased risk of sustaining a contralateral Achilles tendon rupture.[25]

The distribution of Achilles tendon ruptures in different sports varies considerably from country to country, according to the national sports traditions. In Scandinavian countries, badminton players are particularly at risk.[64] In a study of 111 patients, 58 (52%) had a rupture of the Achilles tendon while playing badminton.[65] In Northern and Central Europe, soccer, tennis, track and field, indoor ball games, downhill skiing, and gymnastics are the most common sports accounting for Achilles tendon ruptures.[3,66] In North America, American football, basketball, baseball, tennis, and downhill skiing predominate.[3,66]

Achilles tendon rupture is predominantly a male disease and the dominance of males is evident in all studies, with a male:female ratio of 2:1 to 12:1, probably reflecting the higher prevalence of males involved in sports.[3,67,68] Almost all studies report a dominance of left-sided Achilles tendon ruptures. In a review by Arndt,[69] 57% of 1,823 Achilles tendon ruptures were left-sided, probably because of the higher prevalence of right-side-dominant individuals who push off with the left lower limb.[3,58]

Hungarian and Finnish studies showed a higher prevalence of rupture of the Achilles tendon in patients with blood group O.[63,70] These findings have not been confirmed in another Finnish area and in Scotland,[2,71] probably due to peculiarities in the distribution of the ABO groups in genetically segregated populations.

Among U.S. military personnel who underwent repair of Achilles tendon ruptures between 1994 and 1996, blacks were at increased risk for undergoing repair of the Achilles tendon compared with nonblacks.[72]

Etiology of Achilles Tendinopathy

Overuse injuries of the Achilles tendon are well documented and fairly common.[34,73] Repetitive overload of the Achilles tendon to a level beyond its physiological threshold can lead to inflammation of its sheath, degeneration of its body, or a combination of both.[74]

To date, the etiology of Achilles tendinopathy remains unclear.[73,75] Tendinopathies have been linked to overuse, poor vascularity, lack of flexibility, genetic makeup, gender, endocrine or metabolic factors, and quinolone antibiotics.[76–78] Excessive loading of the tendon during physical exercise is currently thought to be a main pathological stimulus.[79] If the tendon is repetitively overloaded beyond its physiological threshold, it will respond by either inflammation of its sheath, degeneration of its body, or a combination of both.[80] Repetitive microtrauma to the tendon without adequate time for recovery and repair, even if within physiological limits, can also lead to tendinopathy.[81] Microtrauma is linked to non-uniform stresses between the gastrocnemius and soleus, due to their different individual contributions in force. This results in abnormal concentrations of load within the tendon, frictional forces between the fibrils, and localized damage to fibers.[82] It is likely to be multifactorial from a combination of intrinsic and extrinsic factors.[73,74]

Sports injuries can result from intrinsic or extrinsic factors, either alone or in combination.[83] Vascularity, dysfunction of the gastrocnemius-soleus complex, age, gender, body weight and

height, deformity of the pes cavus, and lateral instability of the ankle are considered to be common intrinsic factors.[34] Changes in training pattern, poor technique, previous injuries, footwear, and environmental factors such as training on hard, slippery, or slanting surfaces are the extrinsic factors predisposing to tendinopathy.[73,79] Extrinsic factors predominate in the acute trauma setting; however, overuse injuries and chronic tendon disorders commonly have multifactorial causation.[83]

The extrinsic and intrinsic factors associated with Achilles tendinopathy are listed in Tables 5.1 and 5.2. The basic etiology of Achilles tendinopathy is multifactorial.[83,84] In epidemiological studies, various patterns of malalignment of the lower extremity and biomechanical faults were identified in two thirds of the athletes with Achilles tendon disorders.[73,85] Kvist found malalignment of the lower extremity in 60% of patients with such disorders (Table 5.1).[73,85] However, the mechanisms by which these factors contribute to the pathogenesis of the Achilles tendinopathy remain unclear.[86] The most common malalignment in the ankle is hyperpronation of the foot. Limited subtalar joint mobility and limited range of motion of the ankle joint were more frequent in athletes with Achilles tendinopathy than in those with other complaints.[86] In addition, forefoot varus correlates with Achilles tendinopathy,[73,85,87,88] as does increased hindfoot inversion and decreased ankle dorsiflexion with the knee in extension.[88] Excessive movement of the hindfoot in the frontal

TABLE 5.1. Predisposing Intrinsic Factors Related to Achilles Tendinopathy in Sports

General Factors	Local (Anatomic) Factors on the Lower Limb
Gender	Malalignments
Age	Foot hyper- or hypopronation
Overweight	Forefoot varus or valgus
Constitution: weak or strong	Hindfoot varus or valgus
Blood group	Pes planus or cavus
HLA-types	Leg-length discrepancy
Predisposing diseases	Muscle weakness and imbalance
Blood supply	Decreased flexibility
Ischemia	Joint laxity
Hypoxia	
Hyperthermia	

TABLE 5.2. Predisposing Extrinsic Factors Related to Achilles Tendinopathy in Sports

General Factors	Sports-Related Factors
Therapeutic agents	Excessive loads on the lower extremities
Corticosteroids (local and systemic)	Speed of movement
Fluoroquinolone antibiotics	Type of movement
Weight-lowering drugs	Number of repetitions
Anabolic steroids	Footwear/sportswear
Narcotics (cannabis, heroin, cocaine)	Training surface
	Training error
	High-intensity training
	Fatigue
	Poor technique
	Poor equipment
	Environment (temperature, humidity, altitude, wind)

plane, especially a lateral heel strike with excessive compensatory pronation, is thought to cause a "whipping action" on the Achilles tendon, predisposing it to tendinopathy.[79]

In addition to hyperpronation and the other malalignments noted earlier, leg-length discrepancy is another controversial potential contributing factor.[83] The traditional orthopedic view is that discrepancies of less than 25 mm are not clinically important.[86] In elite athletes, however, a discrepancy of more than 5 mm may be symptomatic and, consequently, for a discrepancy of 10 mm or more, a built-up shoe or shoe insert has been recommended to prevent overuse symptoms. However, the true occurrence of these proposed biomechanical alterations, their magnitude, and, above all, their clinical importance is not known.[83]

The importance of muscle weakness, imbalance, and decreased musculotendinous flexibility in the development of Achilles tendon disorders is also a matter of debate. Muscle strength, power, endurance, and flexibility are an important part of physical performance, and can thus be important in the prevention of tendon injuries.[83] If the muscle is weak or fatigued, the energy-absorbing capacity of the whole muscle-tendon unit is reduced and the muscle no longer protects the tendon from strain injury, subsequent inflammation, and pain.[83] Recently, excellent short-term

improvements have been reported in patients with chronic Achilles tendinopathy using a heavy-load eccentric training rehabilitation program based on increasing the length, tensile strength, and force of the muscle-tendon unit.[20,89–92] This concept, however, is open to speculation as the studies do not provide conclusive evidence on whether muscular weakness, imbalance, and musculotendinous tightness are the causes or consequences of injuries.

An overuse tendon injury is caused by repetitive strain of the affected tendon such that the tendon can no longer endure tensile stress. As a result, tendon fibers begin to disrupt microscopically, and inflammation and pain result.[83] Of the extrinsic risk factors, excessive loading of the lower extremities and training errors have been said to be present in 60–80% of the patients with Achilles tendon overuse injuries (Table 5.2).[73,84,85] The most common of these include running too long a distance, running at too high an intensity, increasing distance too greatly or intensity too rapidly, and performing too much uphill or downhill work.[73,84,85,88] Monotonous, asymmetric, and specialized training, such as running only (i.e., without cross-training), as well as poor technique and fatigue are considered further risk factors for Achilles tendon overuse injuries (Table 5.2). Poor environmental conditions, such as cold weather, hard ground surface, and slippery/icy surface may also promote Achilles tendon problems.[66,83,86,93,94] The lack of high-quality prospective studies limits the strength of the conclusions that can be drawn regarding these extrinsic risk factors.

The pathogenesis of Achilles tendon disorders includes many factors such as tissue hypoxia and resulting free radical changes to the tendon from ischemic reperfusion injury and exercise-induced hyperthermia.[13,14] If a tendon is strained to more than 4% of its original length, it loses its elasticity and is at an increased risk of a subsequent break in its collagen structure.[66]

Levels of type III collagen mRNA can be significantly higher in the tendinopathic Achilles tendon compared with normal samples.[95] The significance of this finding is still open for debate. It should be noted that most of the above factors should be associative, not causative, evidence, and their role in the etiology of the condition is therefore still debatable.

Epidemiology of Achilles Tendinopathy

In large studies, the most common clinical diagnosis with reference to Achilles disorders is tendinopathy (55–65%), followed by insertional problems (retrocalcaneal bursitis and insertional tendinopathy) (20–25%).[73,85,96–102] Kujala et al., in an 11-year follow-up cohort study,[103] found questionnaire-reported Achilles tendon overuse injury in 79 out of 269 male orienteering runners (30%), and in 7 of the 188 controls (4%). The age-adjusted odds ratio was 10.0 in runners compared with controls.[103]

There is a clear association with strenuous physical activities such as running and jumping.[34,66,93,96] In top-level runners, the annual incidence of Achilles tendon disorders is reported to be between 7% and 9%.[98,99] The occurrence of Achilles tendinopathy is highest among individuals who participate in middle- and long-distance running, orienteering, track and field, tennis, badminton, volleyball, and soccer.[73,85,96–100]

Kvist[73,85] studied the epidemiology of Achilles tendon disorders in a large group of competitive and recreational athletes with Achilles tendon problems. Following a review of 698 patients, running was the main sports activity in patients presenting with an Achilles tendon disorder (53%) and patients who were runners represented 27% of all patients studied in the sports medicine clinic where the study was performed.[73,85] Sixty-six percent had Achilles tendinopathy and 23% had Achilles tendon insertional problems.[73,85] In 8% of the patients, the injury was located at the myotendinous junction, and 3% of the patients had a complete tendon rupture. On review of the sex of the patient, only 11% were female.[73,85]

As athletes increase in age, chronic Achilles tendon disorders become more common.[104] In a report of 470 patients with Achilles tendinopathy and insertional complaints, only 25% of subjects were teenage or child athletes with 10% under 14 years of age. The majority of these younger patients were diagnosed with calcaneal apophysitis (Sever´s disease).[73]

Patients with unilateral Achilles tendinopathy have a relatively high risk of developing Achilles tendinopathy in the other leg. Initially,

the unaffected leg may show no clinical symptoms and signs of tendinopathy. However, in one study 41% developed symptoms of the Achilles tendinopathy in the contralateral leg by eight years' follow-up.[105]

Conclusion

Achilles tendon problems are increasingly common. Appropriate etiopathogenesis and epidemiological studies are lacking, and most studies do not adequately define their population.

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