The patellar tendon
in junior elite volleyball players
and an Olympic elite weightlifter

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Abstract

The principal aim of the present thesis was to prospectively follow (clinical status and ultrasound + Doppler findings) the patellar tendons in the young elite volleyball players at the Swedish National Centre for high school volleyball in Falköping. In an Olympic weightlifter with chronic painful jumper´s knee, the effects of treatment with sclerosing injections followed by early instituted very heavy weightlifting training, was also evaluated.

First, in a prevalence study, we demonstrated that the clinical diagnosis patellar tendinopathy-jumper's knee, together with structural tendon changes and vascularisation in the painful area of the tendon, was demonstrated in 12/114 tendons in Swedish junior elite volleyball players, but not in any tendons of individually matched (age, height and weight) not regularly sports active controls. Structural tendon changes alone was demonstrated among the volleyball players but also among the controls.

In a 7 months prospective study of a total of 120 tendons, we demonstrated that the clinical diagnosis patellar tendinopathy-jumper's knee was associated with neovessels/vascularity in the area with structural tendon changes in 17/19 tendons. Seventy tendons that at start were clinically normal, and had normal ultrasound + Doppler findings, remained clinically normal after 7 months with intensive training and playing volleyball.

In a 3-year prospective study it was demonstrated that normal clinical tests and normal ultrasound + Doppler findings at school start, indicated a low risk (8%) for these players to sustain patellar tendinopathy-jumper's knee during the 3 school years with intensive training and playing.

In a case study, involving an Olympic elite weightlifter with chronic painful patellar tendinopathy-jumper's knee, successful treatment with ultrasound and Doppler-guided injection of the sclerosing agent polidocanol, allowed for pain-free very heavy weight training two weeks after treatment. Further heavy

weightlifting training on a daily basis, preparing for European Championships, was done without causing tendon rupture and/or pain.

Key words: Jumper's knee, Patellar tendinopathy, Chronic pain, Ultrasonography, Doppler, Neovascularisation, Volleyball, Weightlifting

List of Original papers

This thesis is based upon the following papers, which will be referred to by there Roman numerals:

I. Gisslén K, Gyulai C, Söderman K, Alfredson H. High prevalence of Jumper's knee and sonographic changes in Swedish elite junior volleyball players compared to matched controls. Br J Sports Med 2005;39(5):298-301.

II. Gisslén K, Alfredson H. Neovascularisation and pain in Jumper's knee: a prospective clinical and sonographic study in elite junior volleyball players. Br J Sports Med 2005;39(7):423-428.

III. Gisslén K, Gyulai C, Nordström P, Alfredson H. Normal clinical and ultrasound findings indicate a low risk to sustain Jumper's knee- patellar tendinopathy: a longitudinal study on Swedish elite junior volleyball players. Br J Sports Med In press 2006.

IV. Gisslén K, Öhberg L, Alfredson H. Is the chronic painful tendinosis tendon a strong tendon? A case study involving an Olympic weightlifter with chronic painful jumper's knee. Knee Surg Sports Traumatol Artrosc 2006;14(9):897-902.

Abbreviations

US   Ultrasound

DP   Doppler

CD   Colour Doppler

PD   Power Doppler

SP   Substance-P

CGRP   Calcitonin-Gene-Regulated-Peptide

+   Plus

Mm   Millimeters

Cm   Centimeters

Introduction

Volleyball

History

Volleyball was invented in USA during the late 1880´s, and is an Olympic sport since 1964. The first World Championships were held in 1949 for men, and in 1952 for women. Volleyball is one of the five biggest international sports, and the FIVB (International Volleyball Federation), including 218 affiliated national federations, is the largest international sport federation in the world. Volleyball includes two Olympic disciplines. Since the Atlanta Olympic Games (1996), beach volleyball is also included as a medal sport (FIVB). Beach volleyball is not covered in this introduction.

Game characteristics

The volleyball playing court is a rectangular shaped area measuring 18x9 m, surrounded by a 3m (minimum) wide free zone on all sides. The net is 243 cm high for men and 224 cm high for women, and placed vertically over the centre line of the court (Figure 1). The attack line on each court is drawn 3m from the centre line, and marks the front zone (Figure 2).
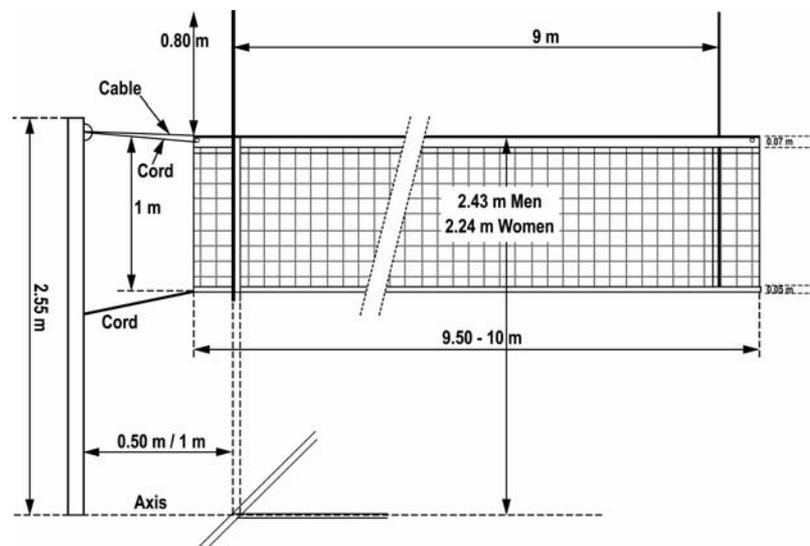

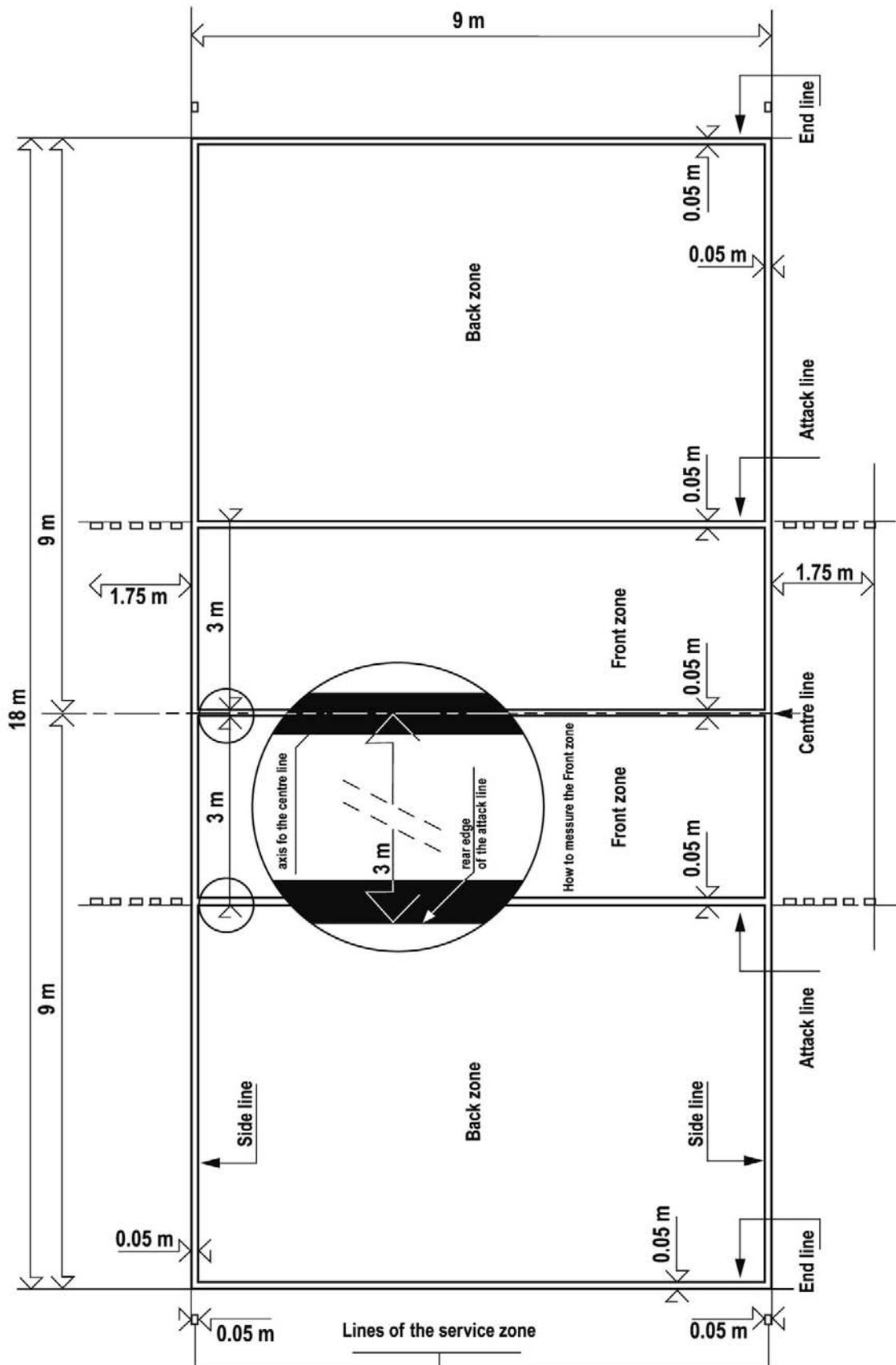
Figure 1. Design of the net.

Figure 2. The playing court.

A volleyball team consists of six players (and a maximum of six substitutes). Three players are located in the "front row" (in front of the three-metre attack line), and three players are located in the "back row". The object of the game is for the two teams (six players in each team), divided by a net, to send the ball over the net in order to ground it on the other teams court, or make the other team fail to return it properly. The ball is put in play (the serve) over the net onto the other teams court. The other team most use a combination of no more than three contacts, before returning the ball over the net. The attacker (offence) may "spike" the ball to return it over the net. The opposite team (defence) attempts to prevent the attacker to direct the ball into their court by having front row players at the net trying to jump and block the attacked ball. The team winning a rally gains a point (Rally Point system) and get´s to serve. There is a clockwise rotation (change position) of the players on the court. A game is played in sets. The first 4 sets are finished at 25 points (or more if there is not a two point difference), and if there is a 5th set it are finished at 15 points (or more if there is not a two point difference). The team that wins three sets wins the match.

Physical demands

The physical demands in elite volleyball can be described from an average indoor match during the 2005 FIVB World Grand Champions Cup. The game lasted about 95 minutes, and included nearly 165 rallies (Reeser et al. 2006). A volleyball game involves approximately 60 maximal jumps per hour of play (Lian et al. 1996b). Biomechanical studies reports that elite volleyball players are subjected to ground reaction forces up to 11.6 times body weight during landings from spiking (having a spike jump height of approximately 1m). The average knee flexion angle is 94° during landing from spiking, and 85° during landing from blocking (Richards et al. 1996). During landing from a jump the patellar tendon is exposed to high forces (8kN), compared with

walking on flat ground (0.5kN) (Curwin and Stanish 1984). Volleyball on elite level includes speed and power that generates high impact forces acting on the knee extensor mechanism. As a result of repetitive spiking and serving (estimated to perform more then 40 000 spikes in a season), the shoulder girdle in elite players is also exposed to high loads (Kugler et al. 1996, Reeser et al. 2006).

General injury pattern in volleyball

There are few prospective studies on injury pattern in volleyball (Bahr and Bahr 1997, Wang and Cochran 2001, Verhagen et al. 2004), and there is no reliable estimate of the injury incidence in international elite level volleyball (Bahr and Reeser 2003).

Norwegian senior players had an overall injury incidence rate over one season that was 1.7/1000 hours, with an incidence of 3.5 injuries/1000 hours during competition and 1.5 injuries/1000 hours during training (Bahr and Bahr 1997). Among elite and recreational volleyball players in Denmark, the overall injury incidence rate was 4.2/1000 hours (Aagaard et al. 1997), and in the Netherlands (2nd and 3rd division males and females) it was 2.6/1000 hours (Verhagen et al. 2004). It is well known that ankle sprains accounts for up to half of all acute injuries in volleyball, with an estimation of one ancle sprain/1000 playing hours (Bahr et al. 1994, Bahr and Bahr 1997, Verhagen et al. 2004). These data suggests that indoor volleyball is a relatively safe sport, especially if compared with ice hockey and team handball (Reeser et al. 2006). Also, reports conducted during the 2004 Olympics in Athens, revealed that volleyball had the lowest acute injury rate of any team sport (Junge et al. 2006).

Overuse injuries in volleyball are reported to bee most frequently occurring in the knee. Patellar tendinopathy-jumper's knee has been shown to be very common among senior volleyball players (40-50 % of high level volleyball

players) (Ferretti et al. 1984, Ferretti 1986, Lian et al. 1996a and 2005), and has been suggested to be the typical overuse volleyball injury (Ferretti 1990). Shoulder pain syndromes, such as impingement and functional instability are also common overuse injuries among male and female players, accounting for 8-32 % of all injuries (Kugler et al. 1996, Aagaard and Jorgensen 1996, Aagaard et al. 1997, Wang et al. 2000, Wang and Cochrane 2001, Verhagen et al. 2004, Reeser et al. 2006). Other overuse injuries mentioned in the literature are low back pain (Verhagen et al. 2004) and suprascapularis neuropathy (Ferretti et al. 1998). The aetiology to suprascapularas neuropathy is debated, but it is interesting that there is a high occurrence (up to 45 %) among elite volleyball players (Ferretti et al. 1998, Reeser et al. 2006). The overuse injuries are associated with pain, and sometimes a decreased activity level, but do not necessarily stop the player from training and playing volleyball (Ferretti et al. 1984, Ferretti 1986, Lian et al. 1996a, Verhagen et al. 2004). Although there is an increased knowledge about the sport specific risk factors in volleyball, there is relatively sparse knowledge about methods for prevention of volleyball injuries (Bahr 2003, Reeser et al. 2006)

Olympic weightlifting

History

Weightlifting has old traditions. The first World Championships in weightlifting for men was staged in London 1891, and there was Olympic weightlifting already at the first modern Olympic games in Athens 1896. For women, World Championships in weightlifting was introduced 1987, and at the Olympic games in Sydney 2000, women weightlifting was for the first time included in the program. The International Weightlifting Federation (IWF) comprises 167 affiliated nations.

Characteristics

In Olympic weightlifting there are two disciplines-the clean and jerk (a two-stage movement) and the snatch (a single-stage movement). The weightlifter has three attempts in each of the disciplines, and the sum of the best snatch and the best clean and jerk adds up the total score. The weightlifters (youth, junior and senior in men and women) compete in weight categories in a sequence of body weight (44 kg to + 105 kg). The lifts are performed with a barbell, consisting of a steel bar (weighting 20 kg for men and 15 kg for women) onto which rubber-coated weight discs are fastened (with collars weighting 2.5 kg each).

The weightlifter performs the lift on an elevated stage on a 4x4m platform coated with non-slippery material. Three referees judge if a lift is correctly completed, and a good lift requires acceptance from two of the three judges.

Physical demands

World records in Olympic weightlifting indicate that the weightlifter is exposed to loads that exceed body weight by as much as two to three times in some weight categories (IWF website 2006). The power output is reported to increase

with body-weight for a given movement, jerk drive range from 2140 watts in the 56kg class to 4786 watts for a 110kg lifter (Garhammer 1980). Based on a biomechanical analysis of a patellar tendon rupture during a competition, the patellar tendon is calculated to be exposed to very high forces (17 times the body weight) (Zernicke et al. 1977). During snatch and clean and jerk, the ability to successfully execute the pull- double-knee bent (initial displacement of the barbell from the floor to approximately waist height-a movement lasting almost 1second), is reported to depend on the weightlifters ability to both generate a sufficient magnitude of joint power and to organize the phase of power production and absorption into an appropriate temporal sequence (Enoka 1988).

General injury pattern

There is very sparse information about injury patterns in elite Olympic weightlifting.

The literature indicates that most injuries occur in the knees, followed by the shoulder and lower back (Kulund et al. 1978, Raske and Norlin 2002). This was confirmed in a retrospective study (six years) on Olympic elite weightlifters, were injuries in the knees, shoulders, and lower back accounted for 64.8 %. The majority of these injuries were referred to as strains and tendinitis. Typical injuries were characterised to be overuse injuries (Calhoon and Fry 1999). The prevalence of radiographically defined knee arthrosis in former weightlifters is reported to be 31 % in the tibiofemoral joint and patellofemoral joint, to be compared with 29 % among former soccer players, and 14 % in former runners (Kujala et al. 1995).

The normal patellar tendon

Anatomy

The quadriceps muscle is the main extensor of the knee, and the patellar tendon transmit the force created within the muscle to the bone insertion in the proximal tibia (Norkin and Levange 1983). The quadriceps muscle includes the vastus medialis, vastus lateralis and vastus intermedius, originating at the femur and inserting via the quadriceps tendon into the basis of the patella, and the rectus femoris muscle that originates at the spina iliaca anterior superior and inserts via the quadriceps tendon into the basis of the patella. The patellar tendon has a flat structure and is an extension of the quadricep muscle (primarily from the central fibres of the rectus femoris muscle), inserting into the tuberculum of the tibia (Figure 3) (Andrikoula et al. 2006). The patellar tendon is about 30 mm wide in the coronal plane, 4-5 mm thick in the sagittal plane, and 40-50 mm long (Khan et al. 1998, Peers and Lysens 2005, Andrikoula et al. 2006). In the resting state, the patellar tendon has a wavy configuration that appears as regular bands across the surface of the tendon. When the tendon is stretched, the wavy pattern disappears. If the tendon is stretched no more than 4 % it will immediately resume its normal length if the force is released (Stanish et al. 1985, Hess et al. 1989).
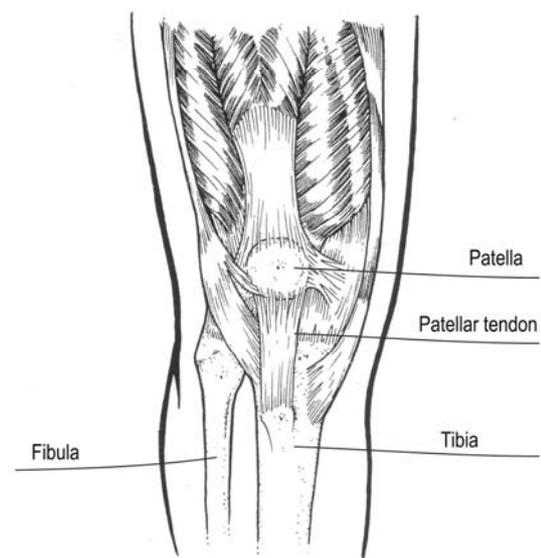

Figure 3: The extensor mechanism of the knee.

The extracellular tendon matrix is composed of collagen fibres, elastic fibres (elastin), ground substance, and anorganic components (Kannus 2000). Collagen (mainly type I) and elastin are embedded in the proteoglycan-water ground-substance, with collagen accounting for 65-80 % and elastin for about 1-2 % of the dry tendon mass (Hess et al. 1989, O´Brien 1992, Kirkendall and Garrett 1997, Kannus 2000). The complex collagen structure enables the tendon to withstand high loads (Curwin and Stanish 1984) and the elastic fibres (elastin) may contribute to the recovery (back to wavy configuration) after tendon stretch (Butler et al. 1978). The tendon ground substance, surrounding the collagen, consists of 60-80 % water, proteoglycans, glucose-aminoglycans (GAGs), structural glyco-proteins, and other small molecules (O´Brien 1997, Kannus 2000). The tendon cells, tenoblasts and tenocytes, comprice about 90-95 % of the cellular elements, and are located between the collagen fibres (Kannus 2000).
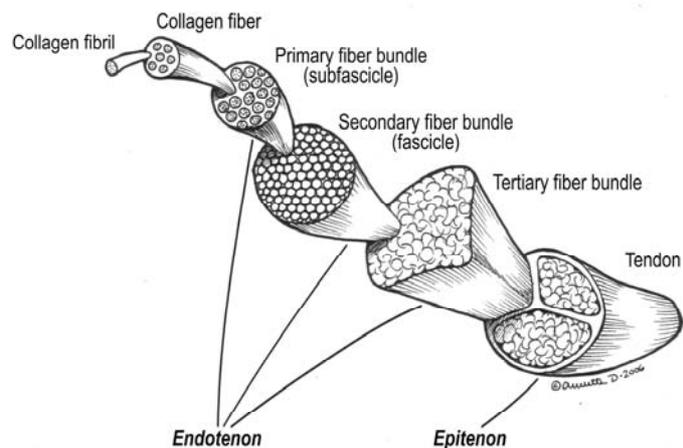
Figure 4: Schematic image of the tendon structure from collagen fibrils to the entire tendon.

Tropocollagen molecules form cross-links to create the insoluble collagen molecule, then progressively aggregating to form definable groups (Hess et al. 1989) (Figure 4). The basic units of a tendon are the fibrils, and the smallest collagenous structures that can be tested mechanically are the collagen fibres (Hess et al. 1989). The collagen fibres are aligned from end to end in a tendon. The endotenon is a sheath of lose connective tissue that surrounds collagen fibrils in primary fibre bundles. Primary fibre bundles form secondary fibre

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bundles that form tertiary fibre bundles (Kannus 2000). The endotenon network allows the fibre bundles to glide on each other, and carries blood vessels, nerves and lymphatics (Hess et al. 1989, Józsa and Kannus 1997, Kannus 2000). The tertiary fibre bundles forms the actual tendon that is surrounded by the epitenon-loose connective tissue (Kannus 2000). A loose connective tissue-the paratenon, is the outermost tissue and functions as an elastic sleeve, composed of loose randomly occurring collagen fibrils permitting free movement of the tendon against the surrounding tissues, and surrounds the epitenon (Hess et al. 1989, Józsa and Kannus 1997, Kirkendall and Garrett 1997, Kannus 2000, Danielson et al. 2006a).

Blood supply

The arterial blood supply to the patellar tendon originates from the descending and inferior medial genicular arteries, the lateral genicular arteries and the recurrent tibial anterior arteries. There is a peritendinous arterial network. Intra-tendinous vessels pierce the tendon resulting in a bipolar pattern of tendinous arterial supply in the middle third of the tendon (Scapinelli 1967 and 1968, Soldado et al. 2002).

Metabolism

Tendons have been demonstrated to be more metabolically active than previously thought, demonstrated (using microdialysis outside the Achilles tendon) as clear circulatory responses and collagen turnover changes related to activity (Langberg et al. 1998 and 2001). However, after exercise it appears that it takes 48-72 hours for the type-I collagen formation to peak (Langberg et al. 2000). This findings have been reproduced by Miller et al. (2005), who showed (by studying biopsies from the patellar tendon) that there is a rapid increase in collagen syntesis up to 72 hours after strenuous exercise.

Intra-tendinous microdialysis has been used to study prostaglandin E_2 and glutamate levels over time (4 hours), under resting conditions, and showed relatively stable levels in normal and chronic painful (jumper´s knee-patellar tendinopathy) patellar tendons (Alfredson et al. 2001).

Innervation

Until recently there has been limited information about the innervation patterns of the human patellar tendon. However, in new studies there is information about the sensory and autonomous innervation. It has been shown that the human patellar tendon is richly innervated by nerve structures coursing through the loose paratendinous connective tissue that surrounds the tendon (Danielsson et al. 2006a). In this tissue, large nerve fascicles, as well as the walls of some of the larger arteries and a few of the smaller blood vessels, display distinct immunohistochemical reactions for the general nerve marker PGP 9.5 (protein gene product 9.5) (Danielsson et al. 2006a). When typing the nerve fibers of the fascicles and perivascular innervation, it has been shown that part of these correspond to sensory afferents, as seen via staining for the sensory nerve markers SP (substance P) and CGRP (calcitonin gene-related peptide) (Danielsson et al. 2006a). However, it has also been found that this innervation contains markers of the autonomous nervous system as well, both parasympathetic (vesicular acetylcholine transporter) (Danielsson et al. 2006b) and sympathetic (neuropeptide Y [NPY] and tyrosine hydroxylase [TH]) (Danielsson et al. 2006c). Thus, there are immunohistochemical findings favoring the occurrence of both a sensory and an autonomous innervation of the patellar tendon.

In contrast to the loose paratendinous connective tissue, the tendon tissue proper (i.e. the actual tendon tissue) is sparsely innervated (Danielsson et al. 2006a). The innervation that is found in this tissue, is mainly seen in narrow zones of loose connective tissue and blood vessels, zones interspersed between the

collagen bundles (Danielsson et al. 2006a). The innervation is made up of thin nerve fascicles and perivascular nerve fibers, and only very few of these display positive reactions for the sensory nerve markers. This latter observation, put together with findings of autonomous nerve markers (Danielsson et al. 2006a and c) seem to indicate that the innervation of the deep parts of the tendon is mainly autonomous and not sensory in its type.

The general (Danielsson et al. 2006c) sensory (Danielsson et al. 2006c) sympathetic (Danielsson et al. 2006c) and parasympathetic (Danielsson et al. 2006b) innervations of the patellar tendon in tendinosis patients have been found in principle not to differ particularly from the corresponding innervations in the normal patellar tendon. SP-positive nerve fibres, seen as free nerve endings, have been observed interspread between collagen fibres in tendons of athletes with or without pain symptoms indicating patellar tendinopathy (Lian et al. 2006).

Recent findings show a local production of signal substances within the tendon cells (tenocytes) themselves, signal substances that are traditionally found in the neuronal system; this anomaly seemingly being particularly pronounced in tendinosis tendons (Danielsson et al. 2006a and c). Thus, evidence of an occurrence of both an acetylcholine production and muscarinic receptors (Danielsson et al. 2006b), as well as of a catecholamine production and adrenergic receptors (Danielsson et al. 2006c), have been shown for the tendon tissue proper of both normal and in particular, tendinosis tendons.

Neurokinin-1 (NK-1) receptor (the primary SP receptor) immunoreaction has been demonstrated in the walls of blood vessels and nerve fascicles/nerve fibres (Forsgren et al. 2005).

Biomechanics

Tendons are flexible. They can act as shock absorbers and can bend at joints. During the lengthening phase of the tendon there is a degree of extensibility,

were elastic energy can be stored and used as elastic recoil. Forces that place the highest stress on the tendon occur during eccentric muscle contractions- such as landing from a jump (Komi 1984, Stanish et al. 1985, O'Brien 1992, Fyfe and Stanish 1992, Kirkendall and Garrett 1997, Ishikawa et al. 2005).

The patellar tendon is exposed to high forces in certain sports. In volleyball, forces up to 8kN have been calculated when landing from a jump. In Olympic weightlifting, forces up to 14.5 kN (equals about 17 times the body weight) have been calculated. For comparison, walking on flat ground generates 0.5kN load on the tendon (Zernicke et al. 1977, Curwin and Stanish 1984).

The chronic painful patellar tendon

Patellar tendinopathy-Jumper´s knee-Patellar tendinosis

Definitions

In most studies a chronic (long-term) condition is defined as a condition with a duration of symptoms longer than 3 months (Kettunen et al. 2002). For many years there has been a confusing terminology for the chronic painful tendon. Despite that histological evaluations of biopsies repeatedly have shown the absence of inflammatory cell infiltrates, the chronic painful patellar tendon has been considered to include an inflammatory component, and the terms tendinitis and tendonitis have often been used. However, recently intra-tendinous microdialysis (Alfredson et al. 2001) have shown that there are no signs of a prostaglandin-mediated inflammation in the chronic painful patellar tendon. Consequently, with the information from histological analyses of biopsies together with the recent findings using microdialysis, it cannot be justified to use the terms tendinitis and tendonitis for this condition. It is now a common opinion that for chronic pain symptoms from a tender area of the tendon, the term tendinopathy should be used (Maffulli et al. 1998). The combination of chronic pain-symptoms from a tender area in the tendon, and were histology and/or images show corresponding changes in the tendon, is commonly named tendinosis (Khan et al. 1996, Movin 1998, Alfredson and Lorentzon 2000). The term jumper´s knee has been used for many years, implying that this condition most often is seen among jumping athletes. Generally, this is the most commonly used term.

Patellar tendinopathy-jumper´s knee-patellar tendinosis is used for a chronic painful condition in the proximal or distal patellar tendon. Jumper's knee is also used for a chronic painful condition in the quadriceps tendon. In this thesis, all studies refer to the chronic painful condition localized to the proximal patellar

tendon (insertion into the apex of the patella), and the nomenclature patellar tendinopathy-jumper´s knee is used.

Aetiology

The aetiology to patellar tendinopathy- jumper's knee is unknown (Roels et al. 1978, Raatikainen et al. 1994, Johnson et al. 1996, Järvinen et al. 1997, Khan et al. 1996 and 1998, Alfredson et al. 2001, Hamilton and Purdam 2003). However, there are multiple theories, and most commonly the condition is considered to be an overuse injury of the knee joint extensor mechanism (especially from jumping activity) (Roels et al. 1978, Ferretti 1986, Kujala et al. 1989, Witvrouw et al. 2001, Richards et al. 2002, Lian et al. 2005 and 2006), possibly causing micro-lesions in the cross link structure, followed by a failed healing response (Roels et al. 1978, Curwin and Stanish 1984, Kujala et al. 1989, Kannus 1997). Cumulative fibre micro-trauma occurs when 4-8 % strain is repeatedly applied to the tendon (Kannus 1997). A recent theory suggests that the condition is an adaptive response secondary to compressive forces within the tendon (Hamilton and Purdam 2003).

Pathogenesis

There is sparse knowledge about the pathogenesis to patellar tendinopathy-jumper´s knee (Khan et al. 2000, Almekinders et al. 2002, Peers and Lysens 2005). Histological examinations of tendon biopsies have shown tenocyte changes, increased amount of ground substance, an irregular tendon structure and an irregular fiber-bundle arrangement, and a vascular ingrowth in the chronic painful tendon, compared to the normal pain-free tendon (Roels et al. 1978, Martens et al. 1982, Ferretti et al. 1983, Karlsson et al. 1991, Khan et al. 1996, Cook et al. 1997, Järvinen et al. 1997, Alfredson et al. 2001).

Also, injured tendons have been reported to consist of more type III collagen compared to normal tendons (Järvinen et al. 1997).

In a recent study, a temporal pattern of changes, from cellular abnormalities, to increased ground substance, longitudinal collagen separation, and finally neovascularisation, was suggested to proceed to tendinopathy (Cook et al.

2004a). Also recently, in studies using ultrasound and color/power Doppler, findings of an increased vascularity in the area with structural tendon changes have been reported (Weinberg et al. 1998, Terslev et al. 2001, Cook et al. 2004b and 2005, Alfredson and Öhberg 2005), and immunohistochemical analyses of tendon biopsies have shown sensory nerves (SP and CGRP) in close relation to the vessels (Danielsson et al. 2006a-b, Lian et al. 2006). Interestingly, the occurrence of neovascularisation has in chronic midportion Achilles tendinosis been found to be associated with pain (Öhberg et al. 2001). An association between neovessels (increased blood flow) in the area with structural tendon changes and pain has also been suggested for the chronic painful patellar tendon (Terslev et al. 2001, Cook et al. 2004b and 2005, Alfredson and Öhberg 2005, Lian et al. 2006, Danielson et al. 2006a-b). However, it appears that not all tendons with neovessels (increased blood flow) are painful (Terslev et al. 2001, Cook et al. 2004b and 2005). The importance of possible nerve related effects have been highlighted recently. Using microdialysis technique, the neurotransmitter glutamate was found in significantly higher concentrations in the chronic painful patellar tendinosis tendon, compared to the pain-free normal patellar tendon (Alfredson et al. 2001). Glutamate is a potent modulator of pain in the central nervous system, but the importance of glutamate in the periphery is not known.

Of considerable interest are recent findings of a local production of signal substances within the tendon cells (tenocytes) themselves, signal substances that are traditionally found in the neuronal system; this anomaly seemingly being particularly pronounced in tendinosis tendons (Danielsson et al. 2006a and c). Thus, evidence of an occurrence of both an acetylcholine production and muscarinic receptors (Danielsson et al. 2006b), as well as of a catecholamine production and adrenergic receptors (Danielsson et al. 2006c), have been shown for the tendon tissue proper of both normal and, in particular, tendinosis

tendons. There appears to be an upregulation of the cholinergic system in the patellar tendinosis tendon (Danielson et al. 2006b).

Other immunohistochemical studies have suggested that neuronal changes, like nerve-sprouting and an increased perivascular innervation, might occur in the tendon when it develops tendinosis (Sanchis-Alfonso et al. 2001). Recently, Lian et al. (2006) showed the occurrence of sprouting non-vascular SP-positive nerve fibres, and decreased occurrence of vascular sympathetic nerve fibres, in patellar tendinopathy. Substance-P has well known vasodilatory effects, and a possible nerve-mediated regulation of blood vessels is supported by the findings of neurokinin-1 (NK-1) receptor (SP receptor) immunoreaction in the walls of blood vessels and nerve fascicles/nerve fibres (Forsgren et al. 2005).

Risk factors-Predisposing factors

There are many theories around predisposing factors to patellar tendinopathy-jumper´s knee. However, the scientific background to most theories is sparse. The relationship between biomechnical abnormalities and patellar tendinopathy-jumper´s knee have been investigated in several studies. Biomechanical abnormalities such as: patellar hypermobility (Roels et al. 1978, Martens et al. 1982), patella alta (Kujala et al. 1986 and 1989, Kannus 1997, Kettunen et al. 2002), prominent tip of the patella causing impingement between the patella and the proximal patellar tendon (Johnsson et al. 1996), reduced antero-posterior patella tilt angle (Tyler et al. 2002), hyperlaxity syndrome (Kannus and Natri 1997), leg length discrepancy (Kujala et al. 1986), decreased flexibility of the quadriceps and hamstrings muscles (Mariani et al. 1978, Witvrouw et al. 2001), reduced ankle dorsiflexion range (Malliaras et al. 2006b), a combination of high ankle inversion-eversion moments, high external tibial rotation and plantar flexion moments, large ground reaction forces, and high rate of knee extensor moment development (Richards et al. 1996 and 2002), have been suggested to be linked to patellar tendinopathy. There seems to be a low correlation between

height (Martens et al. 1982, Witvrouw et al. 2001), weight (Witvrouw et al. 2001), types of training in volleyball (Ferretti et al. 1984, Ferretti 1986, Lian et al. 2003) and patellar tendinopathy-jumper´s knee. But, Lian et al. (2003) showed that greater body weight might be of importance. There is no conclusive data that strength deficits, or imbalances, are causative factors for tendon injury (Almekinders and Temple 1998). However, in volleyball players, it appears that there is an association between high jumping capacity and having patellar tendinopathy-jumper's knee (Lian et al. 1996b and 2003). Playing on hard surface (Ferretti et al. 1984, Ferretti 1986), and playing volleyball more than four times per week (Ferretti et al. 1984, Ferretti 1986) has also been showen to be associated with having patellar tendinopathy-jumper´s knee.

During the last years, possible relationships between patellar tendinopathy-jumper's knee and antropometric factors have beeb evaluated. Gaida et al. (2004) studied antropometry, body composition and muscle strength in a small group of female basketball players, and found indications that unilateral and bilateral tendinopathy may have somewhat different aetiology. Cook et al. (2004c) studied elite junior basketball players and found a poor posterior thigh muscle flexibility (males and females) and a high vertical jump ability (females), to be associated with abnormal patellar tendon imaging (US). Recently, Malliaras et al. (2006a) investigated 113 competitive volleyball players, and found waist girth (> 83cm) in men to be the only antropometric factor related to abnormal patellar tendon imaging (US).

There is no conclusive data considering gender differences for the condition (Ferretti et al. 1984, Ferretti 1986, Cook et al. 1997, Witvrouw et al. 2001, Lian et al. 2005).

Classification

Patellar tendinopathy-jumper's knee is characterised by activity related pain and/or soreness (variable intensity) most commonly located in the patellar

tendon insertion into the tip of the patella (Blazina et al. 1973, Mariani et al. 1978, Ferretti et al. 1985, Lian et al. 1996a and 2005, Khan et al. 1998). The condition has been described and classified according to symptoms (Blazina et al. 1973, Roels et al. 1978, Lian et al. 1996a) (Table 1).

Table 1. Classification of patellar tendinopathy according to symptoms as outlined by Blazina et al. 1973, modified by Roels et al.1978, and Lian et al. 1996a.

Patellar tendinopathy-jumper's knee	
Grade I	Pain at the infrapatellar or suprapatellar region after practise or after an event
Grade II	Pain at the beginning of the activity, disappearing after warm-up and reappearing after completion of activity
Grade IIIa	Pain during and after activity, but the patient is able to participate in sports at same level
Grade IIIb	Pain during and after activity and the patient is unable to participate in sports at same level
Grade IV	Complete rupture of the tendon

Epidemiology

Patellar tendinopathy-jumper's knee is commonly seen among athletes engaged in explosive jumping sports (Blazina et al. 1973, Martens et al. 1982, Raatikainen et al. 1994, Cook et al. 1997 and 2000a). In a recent epidemiological study on elite athletes from different ports, the overall prevalence of patellar tendinopathy-jumper´s knee was reported to be 14 % (Lian et al. 2005). There was a lower prevalence in women (5.6 %) compared to in men (13.5 %), studying team handball and soccer. The condition is the most common overuse injury in volleyball (28 %), and the prevalence is 40-50 % among elite (senior) level players (Ferretti et al. 1984, Ferretti 1986, Lian et al. 1996a and 2005). In elite (senior) basketball players the prevalence is 32 % (Lian et al. 2005), and in junior basketball players the prevalence is 7 % (Cook et al. 2000a).

In a 2-year prospective study, on students (physical education) training various sports 12-14 hours/week, patellar tendinopathy-jumper´s knee was diagnosed in

13.8 % (19/138 students) (Witvrouw et al. 2001). Other sports where this condition is seen are team handball, soccer, athletics/running, football, ice hockey and tennis (Martens et al. 1982, Karlsson et al. 1991, Raatikainen et al. 1994, Lian et al. 2005, Shelbourne et al. 2006). The condition is also reported to be relatively common among military recruits, representing 15 % of all soft tissue injuries (Linenger and West 1992).

Prevention

There is, to the best of my knowledge, no study presenting an evidence-based strategy that prevent patellar tendinopathy-jumper´s knee. However, reducing known predisposing factors for the elite volleyball player/athlete have been recommended (Bahr 2003, Reeser et al. 2006).This was based on epidemiologic and biomechanical studies. The only published longitudinal material is an abstract, where prophylactic eccentric training reports to have reduced the ultrasonographic intra-tendinous changes (Fredberg and Bolvig 2005 abstract ref). Previously, the same authors reported a 17 % risk to develop patellar tendinopathy-jumper´s knee in asymptomatic tendons with ultrasonographic intra-tendinous changes (Fredberg and Bolvig 2002).

Diagnostics-Clinical

The diagnosis is based on history and clinical examination. Activity-related pain and/or soreness in the proximal patellar tendon is characteristic (Blazina et al. 1973, Mariani et al. 1978, Ferretti et al. 1985, Khan et al. 1998, Cook et al. 2000a). Palpation of the tendon shall be performed with the knee in full extension (Mariani et al. 1978, Lian et al. 1996a, Cook et al. 2001a). With the knee flexed, tenderness might be difficult to elicit (Colosimo and Basset 1990, Khan et al. 1998). The point of tenderness at palpation is located in the tendon insertion into the patella (Blazina et al. 1973, Mariani et al. 1978, Roels et al. 1978, Martens et al. 1982, Ferretti et al. 1985, Kujala et al. 1986, Colosimo and

Basset 1990, Lian et al. 1996a, Cook et al. 2001a). Mild tenderness in the patellar tendon insertion, in asymptomatic (no pain during tendon loading activity) jumping athletes, should be considered as a normal finding (Lian et al. 1996a, Cook et al. 2001a). Recently, a single leg decline squat test has been recommended as a clinical test in the physical assessment of patellar tendinopathy (Purdam et al. 2003). This test is supposed to be the best tool to detect changes in pain intensity, and is relatively easy to standardise compared to other body weight bearing squat tests or jump tests. This was confirmed in a recent report showing significantly greater patellar tendon strain during a single leg decline squat, compared to single leg squat on flat surface (Kongsgaard et al. 2006).

Diagnostics-Ultrasound+Doppler-MRI

To use of ultrasonography (US) and MRI for the evaluation of the patellar tendon have been questioned due to a low prediction value in diagnosing patellar tendinopathy- "structural tendon changes alone does not necessarily mean that there is pain in the tendon" (Lian et al. 1996a, Khan et al. 1996, 1997 and 1999, Cook et al. 1998, 1999, 2000b and 2001b, Shalaby and Almekinders 1999, Malliaras et al. 2006c-d). However, an objective measure such as US or MRI, is of value to verify if the tendon is normal (Kälebo et al. 1991, Khan et al. 1996, Peace et al. 2006).

In chronic patellar tendinopathy-jumper´s knee US examination usually shows a local widening of the tendon, irregular collagen fibre structure, and hypo-echogenic areas in the patellar tendon insertion into the patella (Fritschy and De Gautard 1988, Khan et al. 1996, Alfredson and Öberg 2005). MRI shows a local widening of the tendon and high signal intensity in the widened part of the tendon (Johnson et al. 1996, Schmid et al. 2002).

The occurrence of calcifications have previously been reported in athletes (Fritschy and De Gautard 1988, Karlsson et al. 1992), but no strict correlation

with pain symptoms have been demonstrated (Kujala et al. 1989, Khan et al. 1999). Remaining sonographic abnormalities can be seen after successful patellar tendon surgery (Fritschy and De Gautard 1988, Khan et al. 1999), and sonographic abnormalities have been shown to not correlate with clinical outcome (Coleman et al. 2000a). Consequently, tendon changes alone, without pain symptoms, should not be interpreted as an indication for surgery (Khan et al. 1997, Cook et al. 1998, Testa et al. 1999).

Recently, Colour Doppler (CD) and Power Doppler (PD) have been used to study vascularity in tendons (Weinberg et al. 1998, Terslev et al. 2001). To combine US with CD or PD provides information about vascularity in relation to the tendon structure. Studies on the chronic painful patellar tendon (Terslev et al. 2001, Alfredson and Öberg 2005, Cook et al. 2004b and 2005) indicate an association between pain during tendon loading activity and the occurrence of vascularisation in the area with structural tendon changes. In symptomatic patellar tendons, the amount of vascularity has been shown to be associated with the intensity of pain (Cook et al. 2004b and 2005). Consequently, US+CD or PD is considered to be an important tool in the evaluation of the patellar tendon.

Figure 5. Longitudinal scan of a patellar tendon from a patient suffering from jumper´s knee. US show structural changes, including hypo-echoic areas, mainly in the dorsal tendon. PD show neovessels-vascularity (coloured structures) in the areas with structural changes.
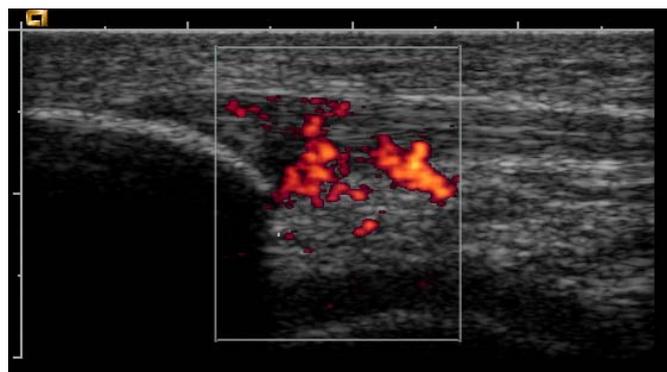
Differential diagnoses

A painful condition in the proximal patellar tendon can be difficult to differentiate from a painful condition in the femuro-patellar joint (Mariani et al. 1978, Kujala et al. 1986 and 1989, Kälebo et al. 1991, Brukner and Khan 2001,

Lian et al. 2005). Patellar cartilage injuries are not uncommon, and similar to patellar tendinopathy-jumper´s knee, they are often associated with knee-loading activity such as jumping. Again, US examination can be helpful to find out whether the tendon is normal or not. Other possible differential diagnoses are fat pad impingement, causing pain in the patellar tendon region during knee-loading activity (Brukner and Khan 2001) and symptoms from synovial or retinacular abnormalities and bursitis (Kujala et al. 1989, Brukner and Khan 2001).

Treatment

Patellar tendinopathy-jumper's knee is considered to be difficult to treat. Approximately 1/3 of the athletes who consult sports medicine clinics with patellar tendinopathy, are unable to return to sports after non-surgical treatment because of recurrence or persisting pain, and need surgical treatment (Cook et al. 1997). There are relatively few scientific studies, and there is no treatment of choice for patellar tendinopathy, neither conservative, nor surgical (Ferretti et al. 1990, Cook et al. 1997, Khan et al. 1998, Coleman et al. 2000b, Cook 2001c, Bahr et al. 2006).

Conservative treatment

Pain intensity in patellar tendinopathy-jumper's knee has been shown to decrease in periods of rest, however, high recurrence rates of symptoms have been reported in high-level athletes when resuming training at their previous activity level, as after summer recess or periods of rest up to 3-6 month (Ferretti 1986, Colosimo and Basset 1990).

An extensive literature review investigated the efficiency of pharmacotherapy, as with oral or topical nonsteroidal anti-inflammatory drugs (NSAID), without a clear efficacy in athletes with patellar tendonitis (Weiler 1992). Other reviews conclude that NSAIDs appears to be effective only when there are short-term

symptoms, though in the chronic stage it's a non-inflammatory condition (Blazina et al. 1973, Sandmeier and Renström 1997, Almekinders and Temple 1998, Cook 2001c). It remains to be determined whether NSAIDs actually change the natural history, or whether they merely have analgesic effects (Weiler 1992, Almekinders and Temple 1998). Steroid injections remain controversial. In the literature it is frequently recommended that steroid injections should be avoided because of risks for further tendon degeneration (Martens et al. 1982, Curwin and Stanish 1984, Sandmeier and Renström 1997). However, some studies suggest the use of steroid injections (Blazina et al. 1973, Mariani et al. 1978, Kelly et al. 1984, Ferretti et al. 1990, Fredberg 1997, Fredberg et al. 2004). Aprotinin injections have been suggested, but there is no scientific evidence for its use (Capasso et al. 1997). Recently, a new treatment method called sclerosing injection therapy was introduced (Öhberg and Alfredson 2002). This method is based on the new findings of ingrowth of vessels and sensory nerves in the dorsal side of the proximal tendon. Ultrasound and colour Doppler-guided injections of the sclerosing substance polidocanol, targeting the area with vessels and nerves dorsal to the area with structural tendon changes (just outside the tendon), have been shown to give promising short-term clinical results in a pilot study (Alfredson and Öhberg 2005), and in a randomised controlled trial (Hoksrud et al. 2006).

Non-surgical treatment has generally been considered to be effective on minor to moderate symptoms of patellar tendinopathy-jumper's knee (Roels et al. 1978, Martens et al. 1982, Ferretti et al. 1985 and 1990, Karlsson et al. 1992, Panni et al. 2000, Cannell et al. 2001). There are numerous of conservative treatment methods, unfortunately most are based on clinical experience and not tested systematically in scientific studies (Cook et al. 1997, Khan et al. 1998, Cook 2001c, Peers and Lysens 2005). Treatment methods include, rest (Mariani et al. 1978, Gecha and Torg 1988, Colosimo and Basset 1990, Khan et al. 1998), ice (Blazina et al. 1973, Curwin and Stanish 1984, Gecha and Torg 1988),

transverse frictions massage (Stanish et al. 1985, Pellecchia et al. 1994, Fritschy and De Gautard 1998, Brukner and Khan 2001, Stasinopoulos and Stasinopoulos 2004), iontophoresis (Pellecchia et al.1994), ultrasound (Stanish et al. 1985, Stasinopoulos and Stasinopoulos 2004), and stretching (Mariani et al. 1978, Curwin and Stanish 1984, Ferretti et al. 1985, Witvrouw et al. 2001). Taping or the use of a brace with pads that pushes the patella to decrease stress on the tendon has also been recommended (Blazina et al. 1973, Gecha and Torg 1988, Tietz 1988, Sandmeier and Renström 1997, Khan et al. 1998).

The use of eccentric strengthening programs has been advocated in the treatment of patellar tendinosis (Curwin and Stanish 1984, Stanish et al. 1986, Jensen and Di Fabio 1989). There are six randomised trials that have evaluated the effect of an eccentric quadriceps training regimen (Jensen and Di Fabio 1989, Cannell et al. 2001, Young et al. 2005, Visnes et al. 2005, Jonsson and Alfredson 2005, Bahr et al. 2006). Jensen and Di Fabio (1989) reported a tendency to increased eccentric quadriceps work capacity, but it was unclear whether there was pain reduction with eccentric training. Cannell et al. (2001) evaluated different types of strength training (eccentric drop squats or leg extension/leg curl exercises). The study outcome showed pain reduction in both groups, but no difference between groups in the numbers returning to sport, or in muscle moment force in the hamstrings or quadriceps muscles. In four randomised studies the effects of using a 25 degrees decline board during the exercise was evaluated. Young et al. (2005) evaluated the effects of painful eccentric decline squat compared to pain-free traditional eccentric leg squat, in elite volleyball players, and found no significant differences between the groups after three months. However, after 12 months the decline group had superior results. Visnes et al. (2005) showed, in a randomised study on male and female Norwegian elite volleyball players, that there was no difference in knee function between the group who did 12 weeks of eccentric training on a decline board, compared to the group who trained normal. In a recent randomised control trial, treatment with 12 weeks of

eccentric quadriceps training on a 25 degrees decline board gave similar results as surgical treatment (open tenotomy) (Bahr et al. 2006). Notably, only about 50 % of the patients in each group were able to return to sports one year after treatment. Johnsson and Alfredson (2005) found 12 weeks of painful eccentric, but not painful concentric, quadriceps training, to give a significant improvement in the VAS and VISA scores on a group of mixed athletes. In that study, and the study by Bahr et al. (2006), but not the other studies evaluating eccentric training on a decline board, the individuals were taken out from training and competition during the first 6-8 weeks of treatment.

Surgical treatment

Failure of conservative treatment implies a choice for the patient to give up his/her sport, or to consider surgery (Blazina et al. 1973, Mariani et al. 1978, Martens et al. 1982, Ferretti et al. 1985, Orava et al. 1986, Kettunen et al. 2002). The literature suggests that surgery is indicated after failure of appropriate conservative treatment for 3 to 9 months (Karlsson et al. 1991, Cook et al. 1997, Sandmaier and Renström 1997, Khan et al. 1998, Panni et al. 2000, Bahr et al. 2006). Surgical treatment of chronic patellar tendinopathy-jumper's knee most often include open patellar tenotomy with revision of the area with tendinosis, and sometimes also curettage or multiple drilling of the inferior patellar pole (Karlsson et al. 1991, Raatikainen et al. 1994, Coleman et al. 2000a). Arthroscopic excision of the tip of the patella (Johnson 1998), and ultrasound-guided percutaneous longitudinal tenotomy (Testa et al. 1999), is also being used. Conflicting clinical results are reported after surgical treatment. Premature return to jumping sports after surgery has been considered to contribute to a poor outcome (Orava et al. 1986, Raatikainen et al. 1994). Most authors state that resumption of sporting activities after surgery often requires 3-8 months, but symptoms may persist for eight to 12 months before complete resolution (Martens et al. 1982, Colosimo and Basset 1990, Cook et al. 1997, Ferretti et al.

2002, Shelbourne et al. 2006). Volleyball players are reported to be especially difficult to treat, having a less predictable outcome after surgical treatment (Ferretti et al. 2002). Interestingly, in an extensive review article, Coleman et al. (2000b) found that studies with a low methodology score were associated with good results, and studies with a high methodology score were associated with poor clinical results.

Very recently, a new surgical approach using arthroscopic shaving of the area with vasculo-neural ingrowth in the dorsal part of the proximal tendon, showed promising short-term clinical results in a pilot study on a group of mixed athletes (Willberg et al. 2006 in press).

To the best of my knowledge, there is no study evaluating different types of surgical treatments in a randomised manner.

Aims of the thesis

The general aims were:

- To investigate the prevalence of patellar tendinopathy-jumper's knee (clinical examination + US + PD) in Swedish junior elite volleyball players, and in matched not regularly sports active controls.

- To prospectively follow, clinically and by US + PD, the patellar tendons in the junior elite volleyball players (15-19 years) subjected to intensive training and playing at the Swedish National Centre for volleyball.

The specific aims were:

- To study the possible importance of clinical and ultrasound findings in the patellar tendon of the junior elite volleyball players at the Swedish National Centre for volleyball.

- To study the possible importance of neovascularisation/increased blood flow for pain in patellar tendinopathy-jumper's knee.

- To prospectively study (repeated clinical examinations and US + CD) the patellar tendon in an Olympic elite weightlifter with patellar tendinopathy-jumper's knee, that started very heavy tendon loading strength training already two weeks after successful treatment with sclerosing injections.

Subjects

The most talented junior volleyball players in Sweden are recruited to the Swedish National Centre for high school volleyball. General physical and sport-specific tests are undertaken. If included, it is a 3-year education (age group from 15-16 to 18-19 years). During the three years at the Swedish National Centre for high school volleyball, they play and train 37-weeks/year (including 13 hours of training + a varying amount of matches/week). The majority of the volleyball players represent the Swedish Junior National Teams in international tournaments. Four teams represent the high school in the Swedish Volleyball Federation leagues, from women's top elite division, to first and second division for men and women. Physical and sport-specific tests are regularly performed during the school year.

Before entering the Swedish National Centre for high school volleyball, the individuals had played and trained volleyball on average 4 years (1-6 years), including 4-8 hours/week. Some players had represented the Swedish National Youth Teams in international tournaments.

Prevalence study (Paper I)

Controls were recruited via advertisement at the local high school. The controls were not regularly sports active (not active in organized sport), and had been recreationally active for not more than 3 hours/week during the last six months before inclusion. Individual matching for age, height, and weight was performed (Table 1a-b).

After permission from the Educational Board of the Swedish National Centre for volleyball and high school, 57 volleyball players (29 males and 28 females) and 55 controls (27 male and 28 female) from first, second, and third grades, participated in the study.

Prospective study (Paper II)

After permission from the Educational Board of the Swedish National Centre for volleyball, all 60 players (29 males and 31 females) in first, second, and third grades (15-19 years old) participated in the study (Table 2).

Prospective study (Paper III)

After permission from the Educational Board of the Swedish National Centre for volleyball, all 22 players (11 males and 11 females) in first grade participated in the study (Table 3).

Case study (Paper IV)

The patient was a 25-year old male super heavyweight (+105 kg) Olympic weightlifter, representing the Swedish National Federation team. For the last six years he had been training with weights 8-10 sessions/week, and regularly taken part in competitions in the Swedish Weightlifting Federation top elite league, and in international competitions.

In December 2003, he was given the diagnosis patellar tendinopathy-jumper's knee after having had a 9-months duration of pain symptoms from his left patellar tendon.

Characteristics of the subjects in Papers I-III.

Table 1a (Paper I). Basic data (mean and range) of the male volleyball players (n= 29) and controls (n= 27).

Variable	Year of birth -87		Year of birth -86		Year of birth -85	
	Volleyball players (n=11)	Controls (n=11)	Volleyball players (n=10)	Controls (n=8)	Volleyball players (n=8)	Controls (n=8)
Age (years)	16.6 (16.1-16.9)	16.5 (16.3-16.9)	17.6 (17.3-17.9)	17.5 (17.3-17.8)	18.5 (18.0-19.5)	18.8 (18.4-19.6)
Height (cm)	186 (176-195)	186 (176-192)	190 (182-202)	188 (185-192)	188 (179-196)	188 (184-192)
Weight (kg)	71.8 (63.0-87.0)	70.8 (61.0-87.0)	81.2 (60.5-91.7)	78.5 (63.0-96.0)	79.2 (63.4-95.0)	78.9 (67.5-91.0)

Table 1b (Paper I). Basic data (mean and range) of the female volleyball players (n= 28) and controls (n= 28).

Variable	Year of birth -87		Year of birth -86		Year of birth -85	
	Volleyball players (n=11)	Controls (n=11)	Volleyball players (n=10)	Controls (n=10)	Volleyball players (n=7)	Controls (n=7)
Age (years)	16.4 (16.0-16.8)	16.5 (16.0-16.8)	17.6 (17.0-17.9)	17.5 (17.0-17.9)	18.5 (18.2-18.8)	18.4 (18.0-18.7)
Height (cm)	177 (164-184)	177 (166-183)	174 (166-181)	173 (165-177)	177 (174-182)	175 (174-179)
Weight (kg)	68.7 (60.9-78.5)	68.1 (58.5-84.0)	66.4 (55.9-75.8)	64.0 (51.9-73.0)	66.7 (60.3-76.4)	66.7 (59.0-79.0)

Table 2 (Paper II). Basic data (mean and range) of the male and female volleyball players (n= 60).

Variabel/Gender	Grade 1		Grade 2		Grade 3	
	Male (n=11)	Female (n=11)	Male (n=8)	Female (n=8)	Male (n=10)	Female (n=12)
Age (years)	16.4 (15.6-16.7)	16.3 (15.1-16.7)	17.2 (16.8-18.3)	17.2 (16.5-17.6)	18.5 (18.2-19.5)	18.2 (17.1-18.7)
Height (cm)	190 (182-202)	174 (166-179)	187 (178-195)	178 (174-182)	184 (171-198)	174 (161-183)
Weight (kg)	80.6 (58.0-95.0)	65.9 (54.0-72.0)	77.6 (62.0-93.0)	67.7 (58.4-80.0)	79.1 (60.0-92.5)	68.6 (56.0-81.0)

Table 3 (Paper III). Basic data (mean and range) of the male and female volleyball players (n= 22).

Variabel/Gender	Baseline 2002		3-yerars follow up (134 weeks)	
	Male (n=11)	Female (n=11)	Male (n=10)	Female (n=8)
Age (years)	16.3 (15.6-16.7)	16.2 (15.1-16.7)	19.0 (18.3-19.3)	18.8 (17.8-19.3)
Height (cm)	189.6 (180-202)	173.6 (166-179)	192.4 (186.0-204)	174.3 (166.5-180.0)
Weight (kg)	81.0 (57.7-96.0)	67.6 (55.0-72.9)	85.6 (65.0-99.0)	69.5 (57.2-81.3)

Methods

Tools for evaluation

Questionnaire

All athletes answered a questionnaire when starting the education. Questions to the athletes included: date of birth, amount and type of training, present and former symptoms involving the patellar tendon, treatments, other knee injuries, and results of treatments. The volleyball players and the controls that reported pain from the patellar tendon marked the area of pain on a special knee chart.

Clinical examination

All participants underwent a clinical knee examination by a registered sports physiotherapist (K.G). The examination included palpation of the patellar tendon, and particular the tendon attachment to the inferior pole of the patella. Tests aiming to reproduce the pain for patellar tendinopathy-jumper´s knee included: one leg body weighted squats and drop jumps (plyometric jump) from 20 cm and/or 43 cm. Clinical tests and/or taping techniques to try to exclude differential diagnoses such as; patellofemoral pathology, fat pad impingement, bursitis, synovial or retinacular abnormalities, and ligament and meniscus injuries, were also performed. The following diagnostic criteria for patellar tendinopathy-jumper's knee were used: history of more than 4 weeks of exercise/volleyball-associated pain in the patellar tendon insertion into the inferior pole of the patella, tenderness to palpation and pain during provocative tests of the knee extensors. The volleyball players regularly (4 times/year) underwent clinical examination (K.G.) and US + PD examination (2 times/year). The physiotherapist responsible for the Swedish National Centre for volleyball

(K.G.) visited the school every week, and if there was pain symptoms from the patellar tendon additional clinical examinations were done.

Ultrasonography (US)

A single experienced musculoskeletal radiologist (C.G), who was blinded to the volleyball player's and the control´s clinical history, performed all US and PD examinations. A high-resolution linear array (8 MHz) ultrasound transducer (Sequoia 512, Acuson, Mountain View, CA) was used for all examinations. US was performed using longitudinal and transverse grey scale scans of the left and right patellar tendons, with the individual laying supine having the knees in a relaxed extension position, and also with the knees in slight flexion (20°). The thickness of the patellar tendon was measured (at the thickest part) and structural changes (hypo-echoic areas) evident in both the longitudinal and the transverse scans, were recorded.

Power Doppler (PD) examination

PD was used to study blood flow in the patellar tendon. As only high flows can be registered, the technique does not allow for registration of the normal circulation in the tendon because of the relatively low flow rate.

To our knowledge, there is no reliable method to quantify flow in the tendon. Therefore, we used a semi-quantitative scale as follows: 0-no vessels; 1-vessels outside the tendon; 2-one or two vessels inside the tendon; and 3-multiple vessels inside the tendon.

For analysis, only tendons with vessels inside the tendon (graded 2 or 3) were considered to have neovascularisation.

Colour Doppler (CD)

In the weightlifter, Colour Doppler velocity technique (CDV) was used to study blood flow in the patellar tendon. PD and CD are similar techniques to study blood flow, although PD is a bit more sensitive (Richards et al. 2005, Peers and Lysens 2005).

Visual analogue scale (VAS)

The weightlifter recorded the amount of pain during patellar tendon loading weight training on a 100 mm long VAS (visual analogue scale), were 0 mm is no pain and 100 mm is severe (worst) pain, at inclusion and at the 2-month and 4-month follow-ups.

Treatment-sclerosing injections

In paper IV, the weightlifter was treated with sclerosing injections. Polidocanol (10 mg/ml) was used as sclerosing agent. The active substance is an aliphatic non-ionised nitrogen-free surface anaesthetic. Before the treatment, the skin was washed with a solution of chlorhexidine and alcohol. The skin was draped with a sterile paper-cover with a hole only for the upper part of the patellar tendon. The injection was performed with a 0.7 x 50 mm needle, connected to a 2 ml syringe. The Polidocanol injections targeted the vessels entering the patellar tendon from the dorsal side of the tendon. The injection was performed dynamically, with the aid of linear high-resolution ultrasound and Colour Doppler, in order to inject polidocanol into or close to the vessels. The ultrasound probe was held on the ventral side of the patellar tendon, parallel with the fibers. It was possible to place the tip of the needle into, or close to, the vessels entering the patellar tendon (outside the dorsal part of the tendon). When the tip of the needle was positioned correctly, a small amount of polidocanol was injected in fractions until all vessels were closed. Altogether 1-2 ml was injected. It was possible to observe the immediate effect of the injection. If the position of the needle was correct (inside or very close to the vessels) the circulation in the target vessels stopped briefly. The injections against the vessels continued until the circulation had stopped in all the vessels in the region with structural tendon changes.

The patient was allowed walking and light bicycling the first two days after treatment. Thereafter, during the first week, light concentric and eccentric strength training for the quadriceps muscles was instituted. During the second week after treatment, the patient was told to gradually increase the tendon loading activity with sport specific training. However, no maximum jumping, running or weightlifting was allowed. After two weeks, maximum patellar tendon loading activity (back to sport at competition level) was allowed.

Statistical methods

SPSS 11.0 for Windows (SPSS, Chicago, IL) was used for the statistical calculations.

Mean and standard deviation was used to describe continuous data. Differences between means were calculated using Student's t-test for independent groups. For categorical values Fisher's exact test (SPSS exact routine) was used.

A p-value less than 0.05 was considered significant.

Ethics

The studies were approved by the Ethical Committee of the Medical Faculty at the University of Gothenburg, Sweden. All volleyball players and controls provided written informed consent. The studies started with a lecture about patellar tendinopathy-jumper's knee for all students.

The treatment method used in study **IV** had been approved by the Ethical Committee at the Medical Faculty, University of Umeå, Sweden.

Summary of papers

Study I: High prevalence of Jumper's knee and sonographic changes in Swedish elite junior volleyball players compared to matched controls

Background: Patellar tendinopathy-jumpers knee is known to be a common and troublesome condition among senior volleyball players, but its prevalence among junior elite players and individually matched non-sports active controls is not known.

Objective: To clinically, and sonographically (US+DP) examine the patellar tendons in junior elite volleyball players (15-19 years) at the Swedish National Centre for volleyball, and in matched controls.

Material and Methods: The patellar tendons in all 57 students at the Swedish National Centre for high school volleyball, and in 55 age-, height-, and weight-individually matched controls were evaluated clinically and by US+PD.

Results: There were no significant differences in the mean age, height and weight between the volleyball players and the controls. In the volleyball group, patellar tendinopathy-jumper's knee was diagnosed clinically and by US in 12 patellar tendons (10 males and two females). In 12/12 tendons, PD sonography demonstrated a neovascularisation in the area with structural tendon changes. In another 10 pain free tendons, there were structural tendon changes and neovessels. In the control group, no individual had the clinical diagnosis patellar tendinopathy-jumper's knee. US demonstrated structural tendon changes in 11 tendons, but there was no neovascularisation on PD.

Conclusions: The clinical diagnosis patellar tendinopathy-jumper's knee, together with structural tendon changes and neovascularisation, was seen among Swedish elite junior volleyball players but not in individually matched not regularly sports active controls. Structural tendon changes alone was seen in 10 % of the control tendons.

Study II: Neovascularisation and pain in jumper's knee: a prospective clinical and sonographic study in elite junior volleyball players

Background: The nature of patellar tendon neovascularisation associated with pain, over time, has not been studied.

Objective: To prospectively study the patellar tendons in elite junior volleyball players.

Methods: The patellar tendons in all students at the Swedish National Centre for high school volleyball were evaluated clinically and by ultrasound (US) + Power Doppler (PD).

Results: Altogether 120 patellar tendons were followed for 7 months. At inclusion, patellar tendinopathy-jumper's knee was diagnosed clinically in 17 patellar tendons. There were structural changes on US in 14 tendons, and in 13/14 PD showed neovascularisation. There were 70 clinically normal tendons with normal US and PD, 24 clinically normal tendons with abnormal US but normal PD, and nine clinically normal tendons with abnormal US and a neovascularisation on PD. At the 7 month follow up, patellar tendinopathy-jumper's knee was diagnosed clinically and by US in 19 patellar tendons, and in 17/19 PD showed neovascularisation. None of the 70 tendons that at inclusion were clinically normal, and had normal US and PD, had developed patellar tendinopathy-jumper's knee. Three of nine clinically normal tendons with structural changes and neovascularisation at inclusion, developed patellar tendinopathy-jumper's knee. Two of 24 tendons clinically normal at inclusion, with abnormal US but normal PD, developed patellar tendinopathy-jumper's knee with abnormal US and neovascularisation on PD. A total of 20 clinically normal tendons with normal US and PD at inclusion developed structural tendon changes, and 12/20 also developed neovascularisation.

Conclusions: The clinical diagnosis patellar tendinopathy-jumper's knee is most often associated with neovascularisation in the area with structural tendon

changes. The findings of neovessels might indicate a deterioration of the condition.

Study III: Normal clinical and ultrasound findings indicate a low risk to sustain patellar tendinopathy-jumper's knee: a longitudinal study on Swedish junior elite volleyball players

Background: Patellar tendinopathy-jumper's knee is well known to be a common and difficult injury in volleyball. There is sparse knowledge about the aetiology and patogenesis.

Objective: To prospectively follow clinical status, tendon structure and vascularity in junior elite volleyball players.

Methods: 44 patellar tendons (22 volleyball players, 11 males and 11 females) starting first grade at the Swedish National Centre for high school volleyball were continuously evaluated clinically and by ultrasound (US) and Power Doppler (PD) over the 3 school years.

Results: *At inclusion*: There were 44 tendons. Patellar tendinopathy-jumper's knee was diagnosed clinically in eight patellar tendons (7/8 had structural changes and vascularity on US + PD). There were 27 normal (clinical and US + PD) tendons.

At 3-years: There were 36 tendons. Four individuals (8 tendons) had been excluded. In 2/25 (2 were excluded) tendons that were normal (clinical and US + PD) at inclusion, patellar tendinopathy-jumper´s knee had developed. There was patellar tendinopathy-jumper's knee (clinical and US + PD) in six tendons.

Conclusions: Normal clinical tests and ultrasound + Doppler findings at start, indicated a low risk for these junior elite volleyball players to sustain patellar tendinopathy-jumper's knee during 3 school years with intensive training and playing.

Study IV: Is the chronic painful tendinosis tendon a strong tendon? A case study involving an Olympic weightlifter with chronic painful Jumper's knee

Background: The chronic painful tendinosis tendon is generally considered a degenerated and weak tendon. However, this has not been scientifically verified and is to be considered a hypothesis.

Objective: We here present a case study involving a high level Olympic elite weightlifter with chronic painful patellar tendinopathy-jumper's knee, that started heavy weight-training very early after successful treatment with sclerosing injections.

Methods: A 25-year old super heavyweight (+105 kg) Olympic weightlifter with 9 months duration of severe pain (prohibiting full training) in the proximal patellar tendon, where ultrasound and Doppler showed a widened tendon with structural changes and neo-vascularisation, was given one treatment with ultrasound and Doppler-guided injections of the sclerosing agent Polidocanol. The injections targeted the neovessels posterior to the tendon.

Results: The patient was pain-free after the treatment, and already after 2-weeks he started with heavy weight training (240 kg in deep squats) to try to qualify for the Olympics. Additional very heavy training on training camps, most often without having any discomfort or pain in the patellar tendon, resulted in Swedish records and 9th place at the European Championships 17 weeks after the treatment. Ultrasound and Doppler follow-ups showed only a few remaining neovessels, and less structural tendon changes.

Conclusion: This case questions previous theories about the weak tendinosis tendon, and stress the importance of studies evaluating strength in tendinosis tendons.

Discussion

Chronic painful patellar tendinopathy-jumper´s knee is known to be common (28-44 %) among senior volleyball players, and 40-50 % of professional players have had pain symptoms from the proximal patellar tendon during their career (Ferretti et al.1985, Ferretti 1986, Lian et al.1996a and 2005). There are no previous studies on adolescent and junior volleyball players, but our current study showed that 11 % of Swedish junior (male and female) elite volleyball players had the diagnosis patellar tendinopathy-jumper´s knee (**I**). This is higher than in a previous study, where 7 % of 14-18 years old male and female basketball players were found to suffer from this condition (Cook et al. 2000a). In our study it was shown that in the individually matched control group consisting of not regularly sports active individuals, there was no individual with the clinical diagnosis patellar tendinopathy-jumper´s knee (**I**). The prevalence of patellar tendinopathy-jumper´s knee in an individually matched not regularly sports active control group has, to the best of our knowledge, never before been presented. Interestingly, 10 % of the individuals in the non-active control group had asymptomatic structural tendon changes. In the volleyball group, there were several players (29 %) with asymptomatic structural tendon changes. This is in accordance with other studies that have reported asymptomatic structural tendon changes in a frequency of 24 % in senior volleyboll players (Lian et al. 1996a), 18 % in senior soccer players (Fredberg and Bolvig 2002) and 21 % in junior basketball players (Cook et al. 2000a). The occurrence of structural tendon changes in asymptomatic tendons is well known, but the clinical importance of these tendon changes has not yet been clarified. However, in junior basketball players, tendons with hypo-echoic structural tendon abnormalities have been shown to have a higher risk (4.2 times) to develope clinical symptoms of patellar tendinopathy-jumper´s knee, compared to tendons with a normal structure (Cook et al. 2000b). This has also been indicated in longitudinal studies on elite soccer players (Fredberg Bolvig 2002) and basketball players (Khan et al. 1997).

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Althogether, theoretically, it might be that patellar tendons with structural changes are more prone to develope painful patellar tendinopathy-jumper´s knee if subjected to heavy tendon loading activity. However, it is not known whether the structural tendon changes are a natural response to load, increaasing the strength of the tendon. Theoretically, up to a certain limit of load the tendon might undergo adaptive changes, but above this certain limit of load, the tendon might be damaged and develop pain symptoms. During jumping, more than 50 % of the work is produced by the knee extensors- leading to high tendon load (Luhtanen and Komi 1978), and the highest prevalence of patellar tendinopathy-jumper´s knee is seen in jumping sports like volleyball and basketball players (Lian et al. 2005).

The background to pain in the chronic painful tendon has not been scientifically clarified. However, biopsies have shown ingrowth of vessels in the structurally changed tendon (Roels et.al 1978, Martens et al. 1982, Khan et al. 1996), and recent studies using ultrasound and Doppler have shown neovascularisation/ increased blood flow in the area with structural changes in chronic painful tendons (Weinberg et al. 1998, Terslev et al. 2001, Öhberg et al. 2001, Cook et al. 2004b and 2005, Alfredson and Öhberg 2005). In following studies, using immuno-histochemical analyses of tendon tissue biopsies, it was demonstrated that sensory nerves (SP and CGRP) "travel together" with blood vessels (Danielsson et al. 2006a), and that there were Substance-P (SP) receptors in the vascular wall (Forsgren et al. 2005. Injections of a local anaestetic (US + PD-guided injections of small volumes) targeting the area with vascularity just outside the tendon, was shown to temporarily cure the pain (Alfredson et al. 2003). Also, sclerosing polidocanol injections (Alfredson et al. 2005), and arthroscopic shaving (Willberg et al. 2006 in press), in the area with neovessels and nerves in the dorsal patellar tendon, significantly decreased the tendon pain in patients suffering from chronic painful patellar tendinopathy-jumper´s knee. Althogether, these findings indicate that the area with vessels and sensory

nerves, mainly located in the dorsal part of the proximal patellar tendon, is of significant importance for the pain in this condition. In Paper **II**, we found that in 13/14 patellar tendons with the clinical diagnosis patellar tendinopathy-jumper´s knee, together with structural tendon changes on US + PD showed neovascularisation-increased blood flow in the area with structural tendon changes. A similar association between patellar tendon pain and neovascularisation has been demonstrated by Terslev et al. (2001), and Cook et al. (2004b and 2005). Consequently, it seems that neovascularisation-increased blood flow demonstrated with Doppler is a frequent finding in the chronic painful patellar tendon. However, in our study 9 pain-free patellar tendons were found to have structural changes and neovascularisation-increased blood flow, showing that neovascularisation is not always associated with tendon pain (**II**). Anyhow, following these 9 tendons over 7 months with intensive training and playing volleyball, revealed that 3 of these 9 tendons developed painful patellar tendinopathy-jumper´s knee (**II**). This might indicate that Doppler findings of neovascularisation/increased blood flow in a patellar tendon with structural changes is a sign of a deterioration of the condition. Maybe junior volleyball players with such findings should rest from jumping activity and be placed on alternative training methods for a period of time? Monitoring junior elite volleyball players with repeated clinical + US + Doppler examinations might, theoretically, prevent from developing painful patellar tendinopathy-jumper´s knee?

We prospectively followed 22 players (44 tendons) with repeated clinical and US + PD examinations during their 3-years in school at the Swedish National Centre for high school volleyball (**III**). Four of the 22 players were excluded because of severe other (not patellar tendinopathy-jumper´s knee) injuries (n= 3) and social reasons (n= 1). Interestingly, only 2/25 tendons that at start were clinically normal and had normal US + PD, had developed painful patellar tendinopathy-jumper´s knee (positive clinical tests and structural tendon changes

+ neovessels on US + PD), at the 3 year evaluation (**III**). These findings indicate that there is a low risk for junior elite volleyball players to sustain patellar tendinopathy-jumper´s knee if clinically and sonographically normal when starting 3 years of intensive training and playing volleyball. This is further substantiated when studying the results in paper **II**. In paper **II** the study period is shorter compared to in paper **III**, but it is interesting to notice that none of the tendons that at inclusion were clinically normal, and had normal US+PD, developed patellar tendinopathy-jumper's knee during the 7 months with intensive training and playing volleyball.

In paper **III**, we demonstrated that in 3/6 tendons that were diagnosed to have patellar tendinopathy-jumper´s knee at school start, the condition remained at the 3-year evaluation (paper **III**). Also, in paper **II** (again during a shorter study period) we demonstrated that in several tendons with patellar tendinopathy-jumper's knee at inclusion, the condition remained at the 7 month follow-up. Althogether, it seems that to train and play with pain symptoms from patellar tendinopathy-jumper's knee is a common feature. This has also been shown by Khan et al. (1997) in a longitudinal study on female basketball players, and by Ferretti et al. (1985 and 1986) who showed that 40-50 % of professional volleyboll players had trained and played with pain symptoms from the proximal patellar tendon during their career. These findings also seem to highlight that when sustained patellar tendinopathy-jumper´s knee it is a troublesome condition to cure. From ethical and humanitarian aspects, it can be questioned whether a talented junior volleyball player with clinical and sonographic findings supporting the diagnosis patellar tendinopathy-jumper´s knee (severe symptoms), should start a 3-year volleyball education with intensive training and playing, when it appears that the painful condition offen remain during the whole school period. On the other hand, if clinically and sonographically normal at start, instead, it seems that there is a good chance for the tendon to tolerate the load without sustaining chronic patellar tendon pain.

During weightlifting training and competition very high loads are placed on the knee extensor muscles and tendons (Zernike et al. 1977). The patellar tendon is subjected to high loads during both eccentric and concentric muscle contractions. Chronic pain and structural changes in tendons is often referred to as "degenerative" changes, and these tendons are commonly considered to be weak (Kelly et al. 1984, Kannus and Natri 1997). We prospectively followed an Olympic elite weightlifter with chronic painful patellar tendinopathy-jumper´s knee, who was treated with sclerosing polidocanol injections, and returned to full training already 2 weeks after treatment (**IV**). US showed a thickened patellar tendon with advanced structural changes, and CD showed a neovascularisation-increased blood flow in the area with structural changes in the dorsal part of the proximal tendon. Interestingly, this so called "degenerative and weak tendon" was subjected to very high loads early after treatment, squats using 240 kg already two weeks after treatment followed by 4 weeks of daily and very heavy weight training to prepare for the European Championships, without pain symptoms as a sign of overload. US and CD follow-ups showed improved tendon structure. It is not appropriate to draw any strong conclusions based on a one case study, but the observations are especially interesting when related to an individual subjected to extremely high patellar tendon loads daily. Thoughts around tendon strength are being raised. Is the thick and structurally changed tendon really a weak tendon? Is the tendon degenerative, or should the term degenerative be questioned? Epidemiological studies on patients with acute Achilles tendon ruptures have shown that about 80 % of these patients had never had Achilles tendon pain before the rupture (Fahlström 2001), most likely implying that the majority of the tendons that ruptures are not the chronic painful tendons with structural changes and neovascularisation (so-called degenerative tendons). Also, from the chronic painful Achilles tendon, having a similar thickening together with structural changes and neovascularisation, a follow-up study showed that in tendons that were successfully treated with

sclerosing polidocanol injections, the tendons where significantly thinner and the structure more normal 2-years after treatment (Lind et al. 2006). These findings suggested a remodelling potential in the Achilles tendon, and not a hopeless degenerative state of the tendon. The findings in the case study (**IV**) questions previous theories about the degenerative and weak tendon, and motivate studies evaluating tendon strength.

Conclusions

The clinical diagnosis patellar tendinopathy-jumper´s knee, together with structural tendon changes and neovascularisation-increased blood flow, was seen in 11% of the players at the Swedish National Centre for high school volleyball, but not in any of the individually matched not regularly sports active controls.

For the players at the Swedish National Centre for high school volleyball

- The clinical diagnosis patellar tendinopathy-jumper´s knee is most often associated with neovascularisation in the area with structural tendon changes.
- Prospectively studied, the findings of neovascularisation in the area with structural patellar tendon changes might indicate a deterioration of the condition.
- Prospectively studied, normal clinical tests and US + PD at start, indicated a low risk to sustain patellar tendinopathy-jumper´s knee during intensive training and playing volleyball.
- Prospectively studied, having the diagnosis patellar tendinopathy-jumper´s knee (positive clinical tests, structural tendon changes on US, and neovascularisation on PD) at start, indicated a poor prognosis to get cured during intensive training and playing volleyball.

For the Olympic elite weightlifter with patellar tendinopathy-jumper´s knee

- Very heavy weightlifting training early after successful treatment with sclerosing injections was possible without causing tendon rupture.
- This case study questions previous theories about the so-called degenerative and weak chronic painful patellar tendon.

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