Patellar Tendinopathy

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Introduction

Tendinopathy of the patellar tendon-often designated commonly, but not quite accurately, as "patellar tendinitis"- is a common source of anterior knee pain in both professional and recreational athletes. It was originally described by Blanzia et al. in 1973 and referred to as "Jumper's knee" due to its association with athletes whose sport requires frequent jumping[1]. Because the patellar tendon is subject to great strain with eccentric contraction of the quadriceps, it is difficult to rest this tendon without completely avoiding running and jumping. As such, while tendinopathy of the patellar tendon may not interfere profoundly with activities of daily living, it can be quite impairing from the perspective of an athlete's participation in sports or recreational activity. For example, the Philadelphia Phillies all-star second baseman, Chase Utley, began the 2011 season on the disabled list with this condition despite reporting no particular difficulty swinging a bat.

Anatomy

The patella is a sesamoid bone, held proximally by the confluence of the vastus medialis, vastus lateralis, vastus intermedius, and rectus femoris tendons. The patellar tendon extends from the inferior pole of the patella and attaches on to the tibial tuberosity. This tendon is also known as the infrapatellar ligament, as the attachment of bone to bone may suggest it is a ligament. Nonetheless, given that the connection of the patella to the tibia is mechanically the final connection of the quadriceps muscle group to the tibia, it can properly be designated as a tendon. In addition to the patellar tendon and the patella itself, there are other nearby structures within the knee that play in important role in the normal biomechanical functions of the knee joint. The infrapatellar fat pad, sometimes referred to as Hoffa's pad, separates the patella from the distal femur and proximal tibia and acts as a shock absorber. In some cases, impingement of the fat pad can lead to malalignment and maltracking of the patella [2]. The patellar tendon is composed of collagen fibrils arranged in bundles called fascicles that are covered by a connective tissue layer, the epitenon, which contains blood and lymph vessels and nerves. The patellar tendon lacks a true tendon sheath and is instead surrounded by a fatty areolar tissue lined by synovial cells, the paratenon, that contributes to the elasticity of the tendon and reduces friction with surrounding tissues[3].

Biomechanics

Extension of the knee is powered by the quadriceps. The power of the quads is increased by the interposition of the patella within the extensor mechanism: the patella pushes the moment arm of the extensor mechanism anterior to the axis of rotation of the knee. The degree to which the moment arm is increased range from approximately 10 percent at 120 degrees of flexion to 30 percent at full extension[4]. When the knee is flexed from zero to 30 degrees, the patellar tendon stretches approximately 10% in length; thereafter, as the knee is flexed fully, there is a negligible amount of elongation [5]. The normal alignment of the knee is slight valgus ("knock kneed"). Thus, the quadriceps mechanism courses down the thigh and inserts onto the patella at an angle relative to the patellar tendon, which is in line with the tibia. This angle is referred to as the Q angle and is approximately 15 degrees (though slightly higher values for females may be normal) [6]. The clinical importance of the Q angle is that some pull of the quadriceps exerts a laterally directed force vector the patella (Figure 1 – q angle), a force that may, in some cases cause the patella to subluxate or even dislocate. The tensile strength of the patellar tendon has been found in studies to range from 25 to 95 MPa. Similarly, the modulus of elasticity ranges from 191 MPa to 600 MPa. These properties have been shown to be independent of age and gender and are significantly related to the mass density of the tendon and moisture composition[7].

Epidemiology

Patellar tendinopathy is a relatively common cause of knee pain in recreational and professional athletes. It is typically seen in younger athletes, both adolescents and young adults. The reason is likely twofold: the likelihood of being involved in high level sporting activity is greater in this age group, and during the growth spurt the body may be unable to adequately deal with the stresses of both the activity and the growth. Males are more prone to develop

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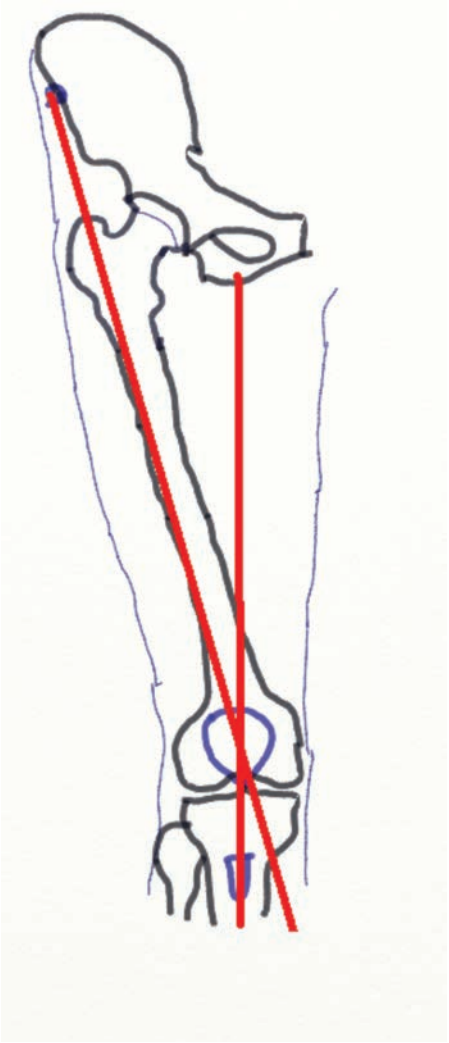

Figure 1. The q-angle is the angle formed by the lines drawn from the anterior superior iliac spine to the central patella and from the central patella to the tibial tubercle. The q angle is approximately 15 degrees (though slightly higher values for females may be normal).

patellar tendinopathy than females. There are some who believe that estrogen may provide a protective mechanism owing to the disparity in prevalence seen between genders. There may also be a correlation between patellar tendinopathy and muscle strength and bony alignment; however, neither of these hypotheses has been adequately tested. One study looked at the prevalence of patellar tendinopathy in elite male and female athletes across 9 different sports: athletics (sprinting and high-jumping), tennis, ice hockey, basketball, volleyball, road cycling, soccer, team handball and wrestling. The overall prevalence of patellar tendinopathy was 14.2% (87 of 613 athletes) with a difference across sports. The highest prevalence was in volleyball (45%) and the lowest was in cycling (0%) suggesting that sports in which repetitive jumping is important are more likely to have athletes suffer from this conditions. Another study done specifically on adolescent basketball players showed a prevalence of 7% (19 of 268 athletes). Women were less likely to have current symptoms compared to men in both studies.[8,9]

Etiology

In its simplest form, patellar tendinopathy is an overuse injury. Overuse, in turn, can be manifest by increased duration of exercise, increased frequency or increased intensity[10]. Also, errors in technique in sport –bending the knee too much, for example, may expose the patellar tendon to excess load. Participants involved in sports that require repetitive jumping activities are prone to develop patellar tendinopathy—hence the eponym "jumper's knee"—but many recreational athletes develop this condition without jumping. Patellar tendinopathy in the recreational athlete is often a product of sudden increases in physical activity or frequency that then puts significant stress on the tendon and overwhelms its ability to repair itself—weekend warrior syndrome. Faulty equipment that does not shield the body from stresses have been implicate in cases of tendinopathy but that is more commonly associated with upper extremity cases (i.e. racquet sports).

Other factors that increase the risk of patellar tendinopathy relate to the intrinsic biomechanics of the knee joint. Individuals with tight quadriceps muscles can increase the strain across the knee joint and thus the patellar tendon. Similarly, a weakness in the musculature about the knee joint, most commonly the vastus medialis obliquus, can lead to abnormal patellar tracking and abnormal distribution of load leading to increased strain on the patellar tendon[11]. Abnormalities of the distal pole of the patella have been implicated in an impingement theory in which irritation occurs at varying degrees of flexion[10,12]. Any misalignment of the bones such as an increased Q angle, genu varum or genu valgum, patella alta or misalignment due to fat pad edema can lead to abnormal loading and subsequently increased strain across the length of the patellar tendon. Obese and overweight individuals also add greater stresses to the knee joint and patellar tendon making these individuals more likely to develop patellar tendinopathy. Decreased vascularity might also cause patellar tendinopathy; this is typically age-related, but not always. Other age-related phenomena include cumulative wear-and-tear, with decreased healing and greater tendon stiffness. Systemic inflammatory conditions and drug reactions (floroquinolone-induced tendinopathy[13]) may also be responsible.

Pathophysiology

The name "tendinitis" suggests that inflammation is the pathological central process in this condition, yet histological examination, ultrasound imaging, and biochemical evaluation have shown inflammation can be minimal or completely absent in cases of tendinopathy [10,14-18].

A number of histological changes have been repeatedly identified in patients with tendinopathy. Micro-tears of the tendinous tissue[19] and a change in the collagen fibril orientation and fiber structure are commonly identified in surgical specimens of documented tendinopathy cases[14]. These characteristic morphologic changes result in an inability to sustain subsequent loads and further damage occurs, leading to tendon degeneration[18,20,21]. Neovascularization

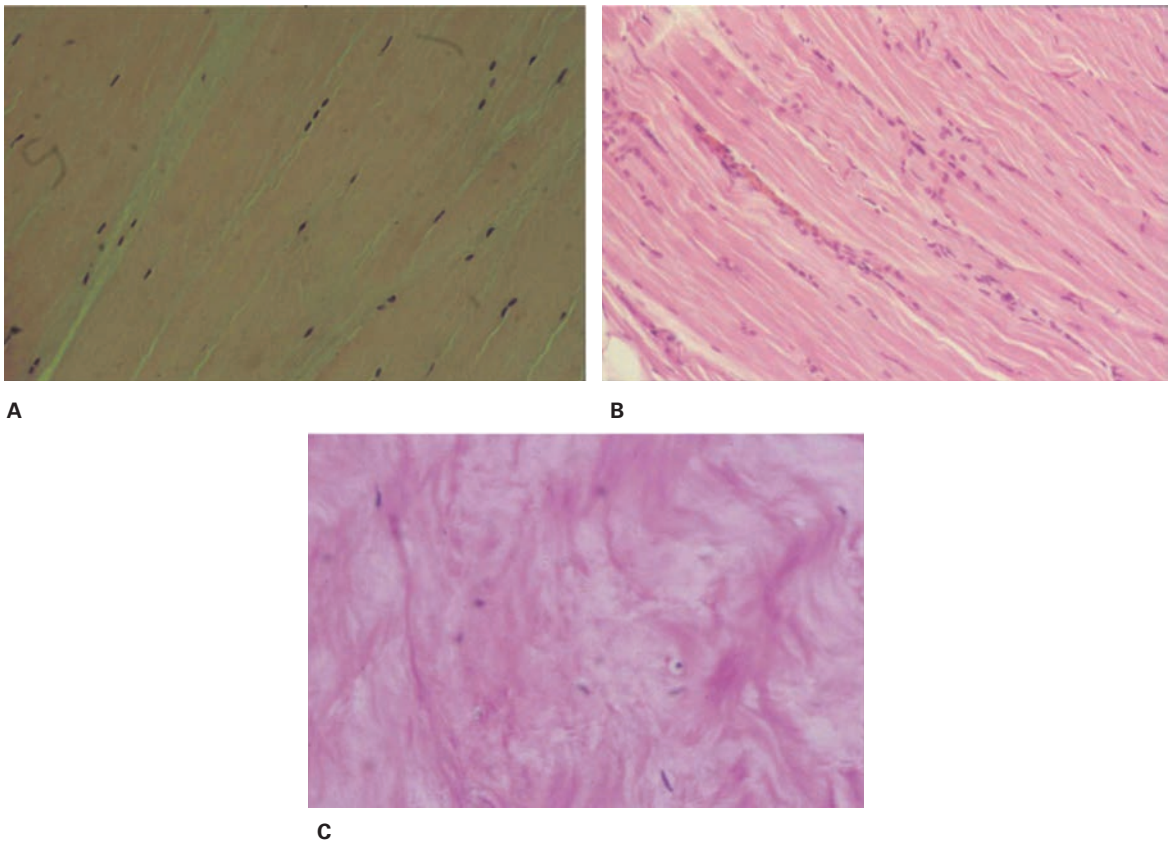

Figure 2. Histologic progression of patellar tendinopathy. A) Normal appearance of organized tenocytes. B) Partial degeneration and disorganization of tenocytes with areas of relatively hypercellularity. C) Complete disruption of normal organization and distinct lack of inflammatory infiltrates.

Rees, J. D.; Wilson, A. M.; and Wolman, R. L.: Current concepts in the management of tendon disorders. *Rheumatology,* 45(5): 508-521, 2006.

in the epitenon demonstrated by the angiofibroblastic infiltration can readily be seen in effected tendons [22]. Doppler ultrasound imaging techniques have supplemented these findings and have shown increased color flow suggesting neovascularization[23,24]. Other researchers have demonstrated this process occurring primarily at the site of pain while other non-painful sites showed no neovascularization[25,26]. It is believed that these vessels can lead to a significant source of pain as nerve fibers have been found to accompany them [27-29](Figure 3).

While the current evidence does not support inflammation as the primary process in patellar tendinopathy, it should not be completely dismissed as a possible participant either. Biochemical studies of tissues samples of patients with patellar tendinopathy have shown increased levels of cyclooxygenase-2 (COX2) and transforming growth factor-beta 1 (TGF-B1) as well as increased production of prostaglandin E2 [30]. Expanding upon this, later studies showed that cyclic loading of tendon fibroblasts increased the expression of cyclooxygenase (COX) in a frequency dependent manner which in turn increased the production of prostaglandin E2 (PGE2) [31]. Induction of cyclooxygenase increases the production of PGE2, which is a known mediator in the process of inflammation. PGE2 is a vasodilator which increases blood flow to the target area further increasing the delivery of other proinflammatory mediators to the site. Furthermore, cyclic loading of fibroblasts also increases the expression level of cytosolic phospholipase-A2 (PLA2) and the subsequent activity level of secretory phospholipase-A2. PLA2 catalyzes arachidonic acid from cellular membranes which is a precursor to many proinflammatory mediators[32].

Due to the disparate data, tendinopathy should remain the term that is used to describe the clinical conditions seen in patients with patellar tendon pain. Tendonitis (indicating inflammation) and tendinosis (indicating degeneration) should be avoided as default or generic descriptors.

Diagnosis

History

Anterior knee pain is a common complaint seen in orthopaedic surgery and the differential diagnosis can be quite extensive (Table 1). In order to correctly identify cases of patellar tendinopathy it is essential to carry out a complete history and physical exam, bearing in mind of course that a patient can have two concurrent conditions (eg, patellar tendinopathy as well as patello-femoral arthrosis). With all types of pain, it is beneficial to follow a systematic way of gathering details. Inquiring about the onset, severity, location, quality, presence of radiation of the pain and exacerbating and/or alleviating factors is imperative. In patients with patellar tendinopathy, the onset is generally

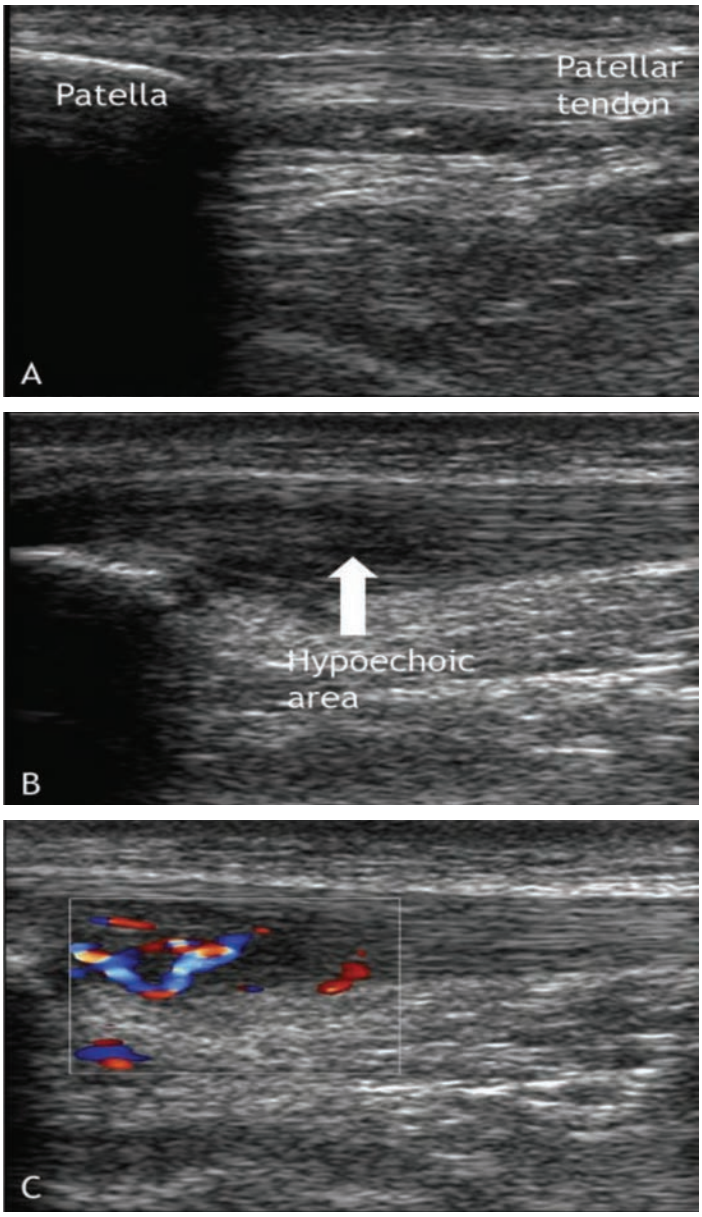

Figure 3. US image of the patellar tendon. A) Demonstrates the normal appearance of the patellar tendon in the absence of any pathology with no hypoechoic areas or increased vascular flow. B) Demonstrates a hypoechoic area with associated thickening of the tendon structure. C) Again seen is a hypoechoic area with localized thickening and now seen is increased vascular flow in the affected area.

Aasne Hoksrud, Lars Öhberg, Håkan Alfredson, and Roald Bahr. Color Doppler Ultrasound Findings in Patellar Tendinopathy (Jumper's Knee) *Am J Sports Med* September 2008 36 1813-1820.

insidious, described as an aching quality and usually located at the inferior pole of the patella. Pain is usually worsened with activity, particularly movements that place significant force on the patella and patellar tendon, e.g. jumping, climbing stairs, rising from a seated position. The classification of patellar tendinopathy is related to the experience of pain in relationship to activity with stage I signifying pain with activity only and stage IV representing complete disruption of the patella tendon (Table 2)[1].

Physical Exam

A full physical exam of the knee should be carried out in all patients regardless of the location of their pain. In cases of patellar tendinopathy a number of physical exam findings may be present. Bassett's sign refers to tenderness to palpation of the insertion of the patellar tendon to the distal pole of the patella in full extension and no tenderness with full flexion[33]. While this is one of the most common findings in patellar tendinopathy, it is only moderately sensitive and relatively nonspecific[34] and physical exam alone can incorrectly diagnose this condition[35]. Other clues include quadriceps atrophy, hamstring tightness, patellar hypermobility, and patella alta/baja however these are relatively nonspecific and little correlation exists. If physical exam and history alone are unable to clearly make the diagnosis and rule out the other possibilities, then imaging techniques can be utilized to aid in the diagnosis.

Imaging

Plain films do not generally reveal patellar tendinopathy, however are important to evaluate other potential causes of pain. Patellar subluxation can easily be identified on a Merchant view. On a lateral view evidence of Osgood-Schlatter and Sinding-Larsen-Johanssen disease can be seen. An elongated distal pole of the patella may be clearly on plain films evident although its role in the development is still under controversy.

Ultrasound imaging is being utilized increasingly in clinical practice to aid in the diagnosis of patellar tendinopathy. The benefits of ultrasound include its non-invasiveness and cost; on the other hand, the quality of an ultrasonographic diagnosis depends critically on the quality, ie skill, of the ultrasonographer. Ultrasound images are commonly referred to as either grey scale ultrasound (GS-US) or color Doppler ultrasound (CD-DS) and are obtained in both the sagittal and axial planes. GS-US images are often obtained while the knee is flexed to maximally stretch the tendon in order to fully evaluate fiber arrangement and to look for the existence of hypoechoic areas indicating pathology. Color Doppler ultrasound is performed while the knee is extended to prevent mechanical constriction of the blood vessels and falsely hide the presence of neovascularization. An ultrasound image of normal, disease-free tendons shows regular and smooth fibers without hypoechoic areas or vascular flow. Abnormal tendons can show localized widening, irregular arrangement of fibers, and hypoechoic areas on GS-US[36]. Tendons that have undergone neovascularization will show increased blood flow on CD-US. When compared with MRI, ultrasound has been shown to be equally specific, yet more sensitive in confirming clinically diagnosed patellar tendinopathy [37]. Furthermore, ultrasound has been shown to have a high intraobserver and interobserver reliability under experienced ultrasonographers therefore making it a valuable study in the diagnosis of this condition [38](Figure 4).

MRI is another valuable modality with many potential benefits. It can evaluate surrounding structures including cartilage, ligaments, and bone more effectively than ultrasound. Also, MRI is less operator-dependent. On MRI the normal patellar tendon should be a homogenous band with

Table 1: Differential Diagnosis of Anterior Knee Pain

Children and adolescents	Adults	Older adults
Sinding-Larsen Johansson Syndrome	Patellofemoral pain syndrome (chondromalacia patellae)	Osteoarthritis
Tibial apophysitis (Osgood-Schlatter)	Osteoarthritis	Crystal-induced inflammatory arthropathy: gout, pseudogout
Patellar subluxation or dislocation	Patellar subluxation or dislocation	
Osteochondritis dissecans	Osteochondritis dissecans	
Referred pain: slipped capital femoral epiphysis, others	Mensical tears	

Table 2: Classification of Patellar Tendinopathy

Stage 1	Pain only after activity, without functional impairment
Stage 2	Pain during and after activity, although the patient is still able to perform satisfactorily in his or her sport
Stage 3	Pain during and after activity, although the patient is still able to perform satisfactorily in his or her sport
Stage 4	Complete tendon tear requiring surgical repair

relatively low signal intensity on all sequences with a clear distinction between the posterior border of the tendon and the underlying infrapatellar fat pad (Figure 5). MRI images of abnormal tendons show increase signal intensity on both T1-

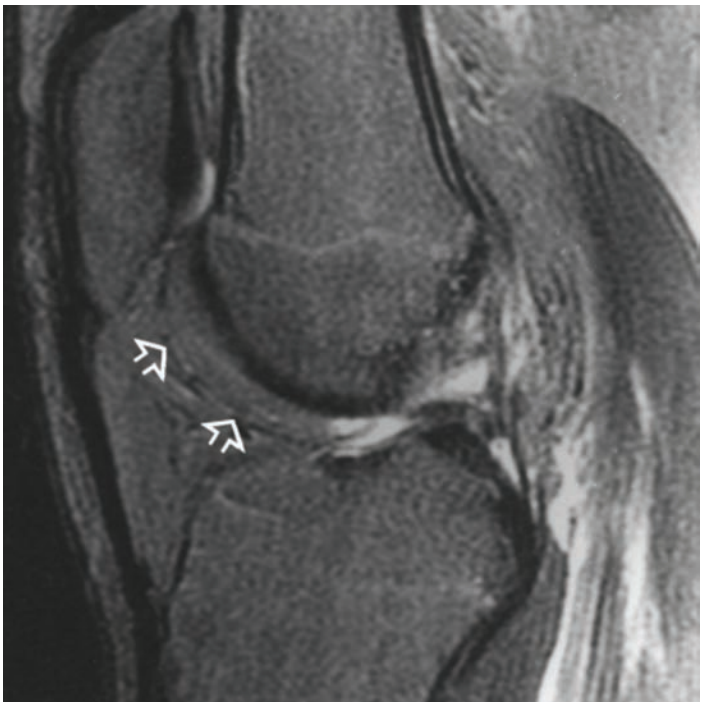

Figure 4. T2 weighted-MRI image of a normal patellar tendon demonstrating a homogenous band with relatively low signal intensity with clear distinction between the posterior border of the tendon and the underlying infrapatellar fat pad indicated by the arrows.

Cothran, R. Lee, McGuire, Philip M., Helms, Clyde A., Major, Nancy M., Attarian, David E. MR Imaging of Infrapatellar Plica Injury. *Am. J. Roentgenol.* 2003 180: 1443-1447.

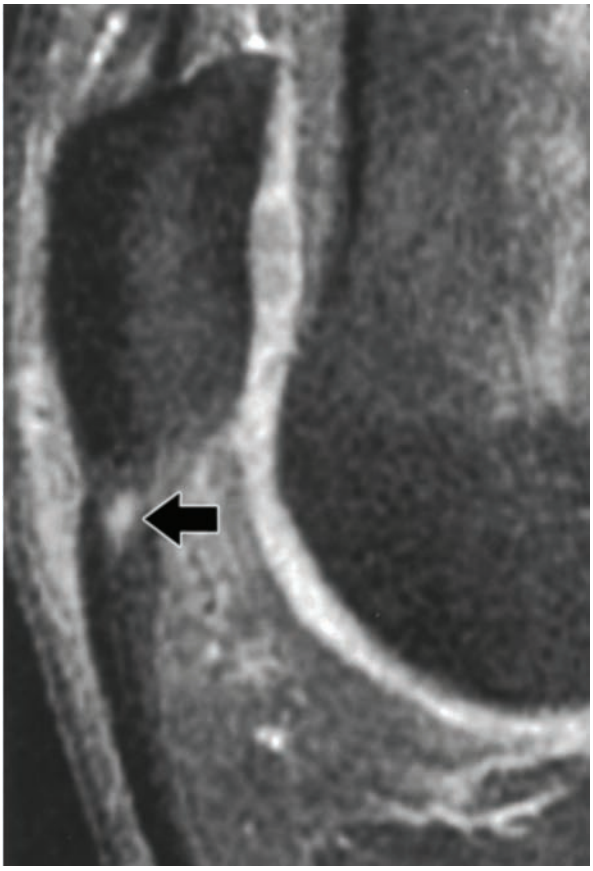

Figure 5. Sagittal fat suppressed T2 weighted MRI image of the patellar tendon demonstrating diffuse thickening with a focus of high signal intensity at the insertion of the patellar tendon to the inferior pole of the patellar in a patient with patellar tendinopathy.

Major, Nancy M., Helms, Clyde A. MR Imaging of the Knee: Findings in Asymptomatic Collegiate Basketball Players. *Am. J. Roentgenol.* 2002 179: 641-644.

weighted and T-2 weight images[36](Figure 6). Furthermore, loss of a clear delineation of the posterior border with the fat pad can be visualized in patients with clinically diagnosed patellar tendinopathy[39,40]

Management

Management of patellar tendinopathy is aimed at decreasing pain, improving function and returning the individuals to their sport or recreational activity. To achieve these goals, multiple modalities have been used with varying degrees of success. When patients present with acute pain, the progression of therapy typically includes a short period of relative rest and activity modification to limit the repetitive stresses to the effected tendon. Overuse injuries should be treated with "underuse". Nonetheless, complete rest and certainly complete immobilization imposes biological and psychological costs as well. The amount of time one should rest depends from person to person and no certain length is universally accepted. Those with stage III symptoms, pain during and after activity that interferes with competition, should expect to have a longer period of rest (3 months or more) than those with stage I symptoms (3 weeks). Complete immobilization of the tendon should be avoided to prevent muscle atrophy. If pain persists following this initial course, one of the following modalities is selected.

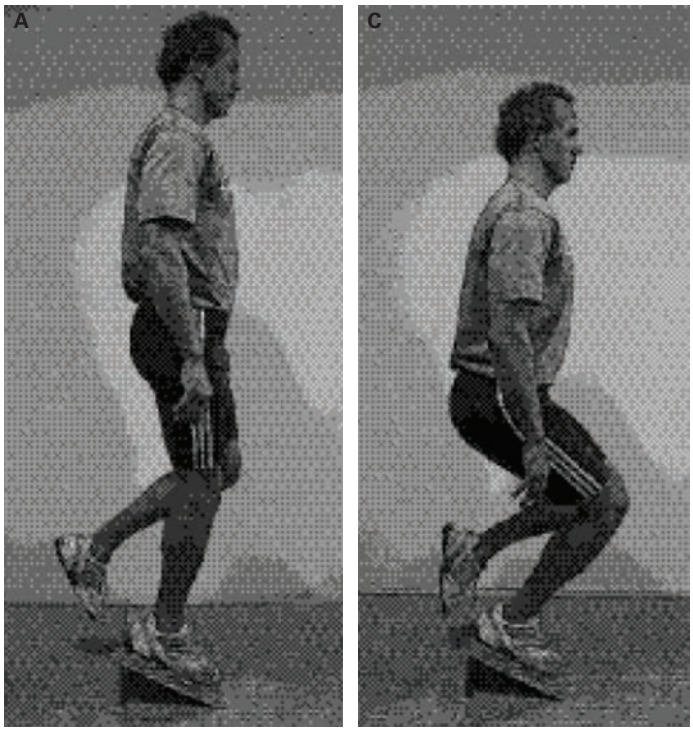

Figure 6. An eccentric contraction describes the lengthening of a muscle despite maximal contraction. In this figure, the entire weight is placed on the injured leg on a 25-degree decline board (A). The patient then slowly descends until the knee is flexed 90 degrees (B).

Visnes, H., and Bahr, R. The evolution of eccentric training as treatment for patellar tendinopathy (jumper's knee): a critical review of exercise programmes *British Journal of Sports Medicine.* 41(4): 217-223, 2007.

NSAIDs

One of the most commonly used methods is non-steroidal anti-inflammatory drugs (NSAIDs). The use of NSAIDs might be under question because inflammation has been shown to be absent in individuals with tendinopathy. Nevertheless, NSAIDs, both oral and topical, are effective for improving pain independent of inflammation for the short terms (7-14 days); beyond this period, their efficacy is much less [41-45]. Furthermore, those presenting with chronic symptoms typically respond less favorably to a course of NSAIDs indicating that their use is primarily reserved for the acute setting and for only a short period of time.

Eccentric Exercise Program

Following the initial episodes of pain, it is often recommended that patients undergo a stretching and eccentric exercise program. The goals of these programs are to prevent further degeneration of the tendon by promoting new collagen formation. An eccentric contraction describes the lengthening of a muscle despite maximal contraction (Figure 6). One study looked at progressive drop squats and knee extension exercises in patients for 12 weeks without a control group and found that most of the test subjects' pain scores improved and a large proportion of the study population were able to return to sports[46]. Another study carried out the observation until 12 months and included a control group and showed that an eccentric squat protocol is effective in reducing pain and allowing subjects to return to activity [47]. Other studies have compared eccentric exercises to concentric exercises in which the concentric group was eventually stopped as the results of poor results [48]. Overall, eccentric exercises seem effective at reducing pain and improving function.

Sclerotherapy

Neovascularization is commonly seen on ultrasound images in athletes with patellar tendinopathy. It is believed that these new vessels are accompanied by pain fibers that travel along with them, and that these pain fibers may account for much of the pain that these athletes experience. Studies have been designed to test the hypothesis that ablation of these newly formed vessels can also eliminate the accompanying nerve fibers and thereby reduce pain. Sclerotherapy (also known as prolotherapy), is one method thought to potentially be effective at ablating these newly formed vessels. It is performed by injecting a material such as polidocanol into or onto the tendon under ultrasound guidance in an area of neovascularization. Hoksurg et al used a cross-over design to evaluate the potential therapy in 33 Norwegian athletes from elite division in basketball, team handball, and volleyball. The athletes were randomly assigned to either the active therapy group or the control group; active therapy patients were injected with polidocanol under ultrasound guidance at 4, 8, and 12 months, with the control group crossing over to active treatment at 4 months. The patients had an overall improvement rating of 84%, suggesting a significant improvement in those athletes treated with sclerotherapy[49]. These results show promise and suggest that sclerotherapy should continue

to be evaluated for its efficacy in the treatment of patellar tendinopathy refractory to more conservative measures.

Iontophoresis

Iontophoresis is a technique that uses a small electric charge to deliver a medicine through the skin. Alternatively, phonophoresis uses ultrasound waves to aid in the delivery of a drug. The use of steroids with iontophoresis has been in practice since the 1950's and has been used to address problems such as plantar fasciitis, carpal tunnel syndrome, lateral epicondylitis, rotator cuff tendonitis and patellar tendonitis. The general principle is to place an electrode of a given polarity containing a drug with the same polarity as the electrode onto the skin. When an electrical current is then applied the drug's charge will cause it to be repulsed from the electrode and thus be delivered into the skin. A commonly used drug for iontophoresis is dexamethasone phosphate. A number of studies have demonstrated that following treatment with iontophoresis, dexamethasone can be detected in human skin and connective tissues[49]; however, no studies have determined if this method of delivery provides any significant subjective benefits to the patient or any relief of their symptoms. Studies still need to be done to look at the clinical benefits, if any, of this therapy.

Extracorporeal shock wave therapy

Extracorporeal shock wave therapy (ESWT) has recently been utilized in the treatment of tendinopathies and various other musculoskeletal conditions that have not responded to conservative therapies. ESWT is currently the standard therapy for lithotripsy of renal calculi. The proposed benefits for its use in musculoskeletal disorders are generally poorly understood. Much of the research has been done in Achilles tendinopathy and conditions of the rotator cough with little evidence in the use in patellar tendon. ESWT is the administration of ultrasound shock waves to the substance of a tendon as either low energy (<0.2 mJ/mm^2) or high energy (>0.2 mJ/mm^2). One proposed mechanism is these sonic pulses have the potential to generate high stress forces in the substances of the tendon and can mechanically pulverize painful calcium deposits in the substance of the tendon and thereby eliminating the pain source [44]. Another, proposed benefit of this method is that ESWT will decrease the levels of substance P there by decreasing a key component in the generation of the perception of pain [45]. Finally, there is potentially an analgesic effect from the therapy via the Malzack's concept of hyperstimulation which states that applying painful stimuli at short intervals can lead to an analgesic effect[46]. One prospective study by Vulpiani et al looked at 73 sports participating patients (16 of which were elite level) who received an average of four treatments and followed up for a period of 2 years. They obtained satisfactory results in 73.5% of cases with the elite level athletes obtaining satisfactory improvement in 87.5% of cases [50]. This study shows promise in this therapy although more studies are needed to confirm these results.

Surgery

When all nonoperative modalities have been attempted and symptoms continue to persist, surgery is generally indicated. Both open and minimally invasive arthroscopic procedures have been described with the open patellar tendon debridement of degenerative tissue being the tried and true method at this time. However, as is the trend with most procedures, arthroscopic debridement y is becoming more popular in clinical practice. With the open procedure a longitudinal or transverse incision is made and the paratenon is split exposing the patellar tendon. Any diseased or abnormal tissue is then identified, divided from healthy tissue, and excised. If there are abnormalities of the distal pole of the patella and point tenderness on physical exam some surgeons will excise this portion. Arthroscopic surgery has the ability to visualize the undersurface of the patellar tendon and fat pad. Most surgeons will debride a portion of the fat pad, any abnormal tendon tissue and shave the distal pole of the patella. The proposed benefit of the arthroscopic procedure is less invasiveness and early return to activity.

A retrospective study done by Coleman et al looked at the effectiveness of both the open and minimally invasive procedure. They had a total number of 44 tendons, 25 in the open group and 29 in the arthroscopic group. They noticed a symptomatic benefit in 81% of those treated with the open procedure and 96% improvement in those who underwent arthroscopic debridement[5147]. Furthermore, the authors also looked at the patient's ability to return to sporting activity following their procedure and obtained a success rate of 54% in the open and 46% in the minimally invasive groups [51]. This study shows that both surgical procedures provide symptomatic benefit to a large percentage of patients. Unfortunately however, both procedures were only able to allow half the patients with enough symptomatic benefit to return to their forming competing level. Despite this, the results are consistent with a meta-analysis that reviewed the surgical effectiveness of chronic patellar tendinopathy in which 10 studies were included and showed an overall success rate of 87.5%[52]. As with most cases, surgery is not 100% effective and many patients will continue to have pain. This underlines the chronic nature of this injury and highlights the need for better and more effective treatments.

Prognosis

The effects of patellar tendinopathy can range from mere nuisance to completely debilitating in some cases. Cook et al reported in their study that 33% of patients who seek out medical attention for their injury were unable to return to their sport for more than 6 months. Furthermore, they noted that 32% had two or three episodes in the past and 17% had four or more episodes showing how this process can continually recur and become a chronic problem[19]. Many athletes will often undergo multiple trials of conservative therapies and some may even resort to surgical management. Those who do not seek treatment may try and work through the pain, which runs the risk of spontaneous tendon rupture. One study looked at the long-term prognosis and determined that after 15 years, 53% of subjects gave up their sport because they were never able to obtain enough relief for their patellar tendinopathy to continue participating.

Summary

Patellar tendinopathy is a condition commonly seen in athletes that has the potential to cause significant morbidity and time away from activity. Many intrinsic and extrinsic factors play into its overall development, and it is generally the result of overuse. The term "patellar tendinitis" should continue to fall out of favor given clear histological evidence that inflammation may not always be present.The diagnosis can generally be made clinically, but various imaging techniques are available to aid in the diagnosis and rule out other causes of anterior knee pain. Treatment should begin with a conservative approach and include some form of eccentric exercises in addition to oral anti-inflammatory medications. More data is needed to support the use of other evolving conservative options; however, if conservative therapy fails, surgical intervention is a viable option.

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