Etiology of Patellar Tendinopathy in Athletes

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Objective: To review the etiology of patellar tendinopathy as it relates to clinical management of chronic patellar-tendon disease in athletes. Data Sources: Information was gathered from a MEDLINE search of literature in English using the key words patellar tendinitis, patellar tendonitis, patellar tendinosis, patellar tendinopathy, and jumper’s knee. Study Selection: All relevant peer-reviewed literature in English was reviewed. Data Synthesis: The etiology of patellar tendinopathy is multifactorial, incorporating both intrinsic and extrinsic factors. Age, muscle flexibility, training program, and knee-joint dynamics have all been associated with patellar tendinopathy. The roles of gender, body morphology, and patellar mobility in patellar tendinopathy are unclear. Conclusions: The pathoetiology of patellar tendinopathy is a complex process that results from both an inflammatory response and degenerative changes. There is a tremendous need for research to improve our understanding of the pathoetiology of patellar tendinopathy and its clinical management. Key Words: jumper’s knee, patellar tendinitis, patellar tendinosis

Many athletes suffer from anterior knee pain associated with activity. This syndrome is frequently viewed as an overuse syndrome and is often related to jumping and excessive load on the quadriceps mechanism. Some suggest that it is specifically seen with eccentric overloading of the extensor mechanism.1,2 It is commonly called jumper’s knee.3-8 It is also referred to as patellar tendinopathy, patellar tendinosis, patellar tendinitis, patellar-tendon disorder, insertion tendinitis of the patellar tendon, partial rupture of the patellar tendon, and patellar apicitis.9 The nomenclature is inconsistent because of the poor understanding of the pathology of this syndrome.10-13 Because the exact pathology of the condition is unknown, some choose not to use pathology as the basis of the nomenclature. For the intent of this article, the condition will be referred to as patellar tendinopathy, which refers vaguely to the pathology, rather than the histologic changes within the tendon.

Patellar tendinopathy has been reported most often among athletes who participate in sports with repetitive explosive movements, such as basketball, volleyball, football, soccer, high jump, long jump, tennis, weight lifting, and running.1,3,5,8,9,14-16 Most authors attribute the symptoms of patellar tendinopathy to a pathology at the bone–tendon junction, involving either the quadriceps tendon or the patellar tendon.1,17,18 The incidence of patellar tendinopathy among athletes has been found
Patellar tendinopathy has been reported as the most common injury in volleyball, representing as much as 28% of the injuries in that sport.\textsuperscript{4,14} Ferretti et al\textsuperscript{4} reported an incidence as high as 40% among high-level volleyball players. Patellar tendinopathy can be debilitating to an athlete, resulting in limited participation or cessation of play.\textsuperscript{10,12,16,19} Although it can be mild and self-limiting, it is often prolonged and recurrent.\textsuperscript{20-22} In addition, there have been several reports of complete rupture of the patellar tendon after the development of chronic patellar tendinopathy, as well as avulsion of the tendon from the bony insertion.\textsuperscript{4,18,21,23,24}

The purpose of this article is to review the pathoetiology of patellar tendinopathy as it relates to clinical management of chronic patellar-tendon disease in athletes. In order to explain the pathology, the anatomy and histology are also described.

### Anatomy and Pathology of the Patellar Tendon

In order to understand the changes in the patellar tendon’s structure, one must be familiar with the anatomy of “normal” tendon tissue. Khan et al\textsuperscript{9} have described the patellar tendon as “the extension of the common tendon of insertion of the quadriceps femoris muscle, [which] extends from the inferior pole [or apex] of the patella to the tibial tuberosity.”\textsuperscript{9}\textsuperscript{[p346]} Black and Alten\textsuperscript{21} reported that anatomists describe the patellar tendon as having “anterior superficial, medial, and lateral fibers continuous with the quadriceps tendon.” The infrapatellar fat pad serves to separate the patellar tendon from the synovial lining of the knee.\textsuperscript{21} The patellar tendon has a width of 3 cm in the coronal plane and a depth of 4–5 mm in the sagittal plane. It tends to thicken distally but does not usually exceed 7 mm.\textsuperscript{8,9} A healthy tendon appears “glistening white” with reflective properties.\textsuperscript{9,12}

As for changes with tendinopathy, several adaptations have been noted, but often only at the bone–tendon junction.\textsuperscript{14,15,23} Degenerative changes in the tendon are typically associated with steroid injections,\textsuperscript{17} but some authors do report evidence of tendon degeneration in the absence of steroid injection.\textsuperscript{15,25}

On gross observation, the injured tendon appears soft and yellow-brown or gray with disorganized tissue. The collagen fibers also appear thin, and the tendon loses its polarized properties. This form of degeneration is known as mucoid degeneration.\textsuperscript{5,12,16,21,26} Colosimo and Bassett\textsuperscript{23} report that mucoid degeneration is seen less than 6 months after the onset of symptoms. A hardening might also occur, which has been labeled hyaline degeneration.\textsuperscript{1,8} The changes are most evident in the deep posterior portion of the superior aspect of the tendon.\textsuperscript{5} Colosimo and Bassett\textsuperscript{23} reported fibrocartilaginous formation, fibrous-tissue degeneration, and calcified cartilage from 6 months to a year after the development of symptoms.

An overall thickening of the insertional fibrocartilage has also been reported.\textsuperscript{1,5} Popp et al\textsuperscript{27} found a “focal thickening in the proximal one-third of the patellar tendon with an average depth of 12.2 mm”\textsuperscript{27}\textsuperscript{[p219]} in all subjects with patellar tendinopathy. Thickening was also seen in the middle portion of the tendon in 5 of 11 symptomatic subjects. A tendon with a depth greater than 7 mm is classified as “thickened” and indicative of patellar tendinopathy.\textsuperscript{19} This apparent thickening was confirmed by Shalaby and Almekinders.\textsuperscript{28} Typically, the thickening is localized along the medial aspect of the tendon.\textsuperscript{27} Shalaby and Almekinders\textsuperscript{28} also reported a
significant increase in tendon width among those with patellar tendinopathy compared with a control group, whereas no significant changes in tendon length were found. The distinction between the posterior aspect of the tendon and surrounding tissue is often lost in patellar tendinopathy.5

Microscopically, the healthy patellar tendon is composed of thick collagen bundles that are found in a hierarchical arrangement, tightly packed in parallel bundles. Spindle-shaped tenocytes are distributed sparingly among the collagen fibrils.1 There is no evidence of extracellular matrix between the collagen bundles. In addition, there are no fibroblasts present. The vasculature is described as inconspicuous.1,12

There are many microscopic changes associated with patellar tendinopathy. The collagen bundles appear disorganized and loose within the first 6 months.8,9,13,23,27 Necrotic tissue replaces collagen.9,16,21,23 The organization and hierarchical structure seen in normal tendon are lost in patellar tendinopathy.

There are also cells present that appear different from tenocytes. Based on the characteristics of the cells, however, it is possible that they developed from tenocytes. Some authors suggest that these cells are in fact tenocytes with a mutated shape.1,8,9,13,27 Changes have also been seen in the ground substance, or extracellular matrix. With tendinosis, there is an increased glycosaminoglycan content as described by Rolf et al,13 who did not see this in their control group.

Hypercellularity of fibroblasts is seen in subjects with patellar tendinopathy.1,8,13,23 Rolf et al15 compared subjects with patella tendinosis with controls and found hypercellularity in all subjects with patella tendinosis and in none of the controls. They reported finding hypercellularity and cell proliferation, which indicates that it is an ongoing disease process and the hypercellularity will continue to increase.

At the bone–tendon junction, 4 distinct zones have been identified in healthy tendons. These zones include tendon, fibrocartilage, mineralized tendon, and bone. When a specimen is stained, a blue line separates the fibrocartilage from the mineralized cartilage. Both the mineralized-cartilage and fibrocartilage zones were noted to be no more than 2 mm thick.1,4

In contrast, there are “pseudocystic cavities” at the border between the mineralized fibrocartilage and the bone in pathologic tendon. Mineralization and ossification of the fibrocartilage are also seen, along with the disappearance of the blue line separating the fibrocartilage and mineralized fibrocartilage on staining. It is suggested that the cavities could be indicative of microtearing.1,4

The vascular supply to the patellar tendon has been proposed as a potential factor in tendinopathy.9,11 Normally, the patellar tendon is supplied by an anastomosis, which is housed in the connective tissue surrounding the rectus femoris. The vessels involved include the medial inferior genicular, the lateral superior genicular, the lateral inferior genicular, and the anterior tibial recurrent artery.9,21 The venous blood empties into the great saphenous vein.21 It is interesting to note that the vascular supply to the superior portion enters along the proximal, posterior aspect of the tendon. It is this region that is often involved in tendinopathies. Although it is counterintuitive to think that a highly vascularized region is at a greater risk for injury, it is not that simple. It has been suggested that the regional blood flow and blood supply during activity might be different than the blood flow and blood supply at rest. It is at rest that the blood flow has previously been measured.9 The
bone–tendon junction has traditionally been considered relatively avascular, but Scapinelli suggests that although the distal attachment of the patellar tendon does have an area of avascularity, the proximal attachment is surrounded by the inferior half of the patella and the infrapatellar fat pad, which are highly vascularized structures. Innervation is supplied by a branch of the saphenous nerve.

The vascular structure has also been found to be significantly different in patellar tendinopathy. Rather than having the normal parallel structure, the vessels in tendinosis are found randomly oriented and often perpendicular. In addition to the random organization, there is vascular proliferation seen in patellar tendinopathy.

It is surprising that in a symptomatic tendon there are no inflammatory infiltrates and no granulation tissue. This is the foundation of both the theory that tendinitis is a misdiagnosis and the debate about the nomenclature of patellar tendinopathy. Although most specimens are from chronic cases, Colosimo and Bassett reported a lack of inflammatory cells within the first 6 months. They did, however, find evidence of granulation tissue in cases with brief symptoms.

The histological changes seen suggest that patellar tendinopathy is a tendinosis, or degenerative condition, rather than a tendinitis, an inflammatory condition. Clinically, this is an important distinction. Although we commonly treat inflammatory conditions with modalities and pharmacologic agents to control the inflammatory process, this is most likely not appropriate in a degenerative condition in the absence of an inflammatory process. It is important to consider when the biopsies are done, however. Most often, the specimens are taken from a subject with a history of symptoms 6 months or longer. In these patients, there might have been an early inflammatory response that is not captured by the late specimen. Although some specimens are from subjects with a 6-month history or less, it is important to investigate acute specimens from subjects who have only had symptoms for 1–2 weeks before entirely ruling out an inflammatory response. In addition, it is possible that there are inflammatory mediators involved that have not previously been identified as inflammatory mediators in other disease processes or other human tissue.

Etiology

Although the exact pathology of patellar tendinopathy is not clearly understood, many report possible etiologies and mechanisms for injury. The general concept that has been supported is that patellar tendinopathy is a traction, overuse injury resulting from repetitive forces being applied to the patellar tendon. Specifically, it is most often linked to the repeated and violent stress placed on the tendon during sports activities such as jumping. This is thought to lead to focal degeneration and microtears in the tendon.

Many athletes are able to sustain these high forces and repetitive loads without developing patellar tendinopathy, but others develop this painful and debilitating syndrome. Some attribute the development of patellar tendinopathy to failed healing. Normally, when tendons are overloaded and experience tissue breakdown, spontaneous healing will occur. Tenocytes serve to increase collagen and matrix production. With repetitive, eccentric loading, however, healing might be inhibited...
or exceeded. Tenocytes might die because of the excessive strain, resulting in decreased collagen and matrix production, which predisposes the tendon to further damage⁹ (see Figure 1). This results in pain and other clinical symptoms. There are many factors that might contribute to the failed healing response, including training errors such as sudden increases in speed, distance, or resistance¹⁵,²⁰.

In addition to the traditional theory on the etiology of patellar tendinopathy, other ideas have been proposed. Laduron et al³³ suggested that there might be an impingement between the deep fibers of the proximal patellar tendon and the lateral part of the femoral trochlea during full extension. Johnson et al,³⁴ however, found conflicting results in a magnetic-resonance-imaging study. They found suggestion of this impingement during knee extension in only 2 of the 24 knees they studied and therefore discounted Laduron’s theory. In contrast, they propose that the cause is an impingement of the inferior pole of the patella on the patellar tendon in flexion. The central fibers of the tendon might be displaced around the inferior pole of the patella in terminal ranges of knee flexion, resulting in impingement. Johnson et al suggest that damage to the superficial fibers of the tendon would be seen if patellar tendinopathy were an overload injury. They argue that the damage to the posterior aspect of the tendon is more suggestive of an impingement mechanism.³⁴ There is also some suggestion that patellar tendinopathy can result from trauma, but because

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Figure 1 — Tendinosis cycle. Modified from Leadbetter.⁵⁷
of the small proportion of cases of patellar tendinopathy associated with trauma, it is not viewed as the most likely cause of patellar tendinopathy.\textsuperscript{3}

### Extrinsic Factors

When looking for factors linked to patellar tendinopathy, researchers have considered both intrinsic and extrinsic factors. Some authors suggest that extrinsic factors are the most influential on the incidence of patellar tendinopathy.\textsuperscript{3} Some of these potential extrinsic factors include frequency of training, years of play, playing surface, type of training, stretching and warm-up practices, and type of shoe worn.\textsuperscript{4,7,14} As sport has changed, extrinsic factors have become more important. Athletic performance has become increasingly important in society. As a result, certain trends have developed, including year-round practice and more intensive training sessions. These trends have significantly increased the physical strain on athletes.\textsuperscript{23,31}

Ferretti et al\textsuperscript{3,14} studied 407 volleyball players in order to identify the roles of extrinsic factors in the occurrence of patellar tendinopathy. The athletes’ age range in this study was described as extending from subjects less than 15 years of age, through greater than 25 years of age. The researchers examined correlations among frequency of play, years of play, playing surface, type of training, and the incidence of patellar tendinopathy. They found that frequency of play was the most significant factor. Among athletes who practiced or competed 5 times a week, there was a 41.8% incidence of patellar tendinopathy, compared with a 29.1% incidence in those who participated 4 times a week, a 14.6% incidence in those who participated 3 times per week, and a 3.2% incidence among those who participated only twice a week. The authors were unable to establish a strong linear association between years of play and patellar tendinopathy. The highest incidence of patellar tendinopathy was in the third year of play, with a decreasing occurrence after the third year. Playing surface was also found to correlate with patellar tendinopathy. It was reported that only 4.7% of the athletes playing on parquet developed patellar tendinopathy, whereas 37.5% of those playing on cement developed patellar tendinopathy. Other authors have reported significantly fewer knee injuries among senior players who play on clay surfaces than among those who play on hard courts.\textsuperscript{35,36} In contrast, no significant correlations between type of training (weight lifting, plyometric exercises, and no weight training or plyometrics) and patellar tendinopathy were found. That was a limited study, however, looking at only 2 types of training. There might be an association between type of training and patellar tendinopathy that was not discovered in this study because of its limited array of training techniques.

### Intrinsic Factors

Although many athletes are exposed to the same extrinsic factors, they do not all develop patellar tendinopathy. Therefore, Witvrouw et al\textsuperscript{7} suggest that there are important intrinsic factors that contribute to the development of the injury. Many others have investigated the role of intrinsic factors in the development of patellar tendinopathy, as well.\textsuperscript{3,15,37,38} Some examples of intrinsic factors include lower
extremity malalignment, leg-length discrepancies, muscle imbalance, anthropometric variables, muscle length, and muscle strength.

Lower extremity alignment has been studied extensively. It is proposed that it could predispose an athlete to patellar tendinopathy as a result of an abnormal distribution of forces in the extensor mechanism. Ferretti reported no significant abnormalities in foot characteristics including forefoot alignment and longitudinal arch structure among athletes with patellar tendinopathy. It has also been reported that no significant abnormalities or significant differences in tibial rotation, femoral rotation, Q angle, or knee alignment (genu valgum or genu varum) are related to patellar tendinopathy. When such static or dynamic malalignments are present, there might be a greater demand placed on the quadriceps in order to dynamically stabilize the knee. The roles of patellar mobility and position are unclear. Some authors report greater patellar mobility in subjects with patellar tendinopathy than in subjects without knee pathology. Ferretti reported that 22 of 26 subjects with patellar tendinopathy had normal patellar mobility. In the same study, 20 of 26 symptomatic subjects had a normal tibial tuberosity, and 6 presented with a prominent tibial tuberosity. When studying leg-length discrepancies and medial tibial intercondylar distance, Witvrouw et al found no significant differences between symptomatic and asymptomatic athletes.

Morphology has also been proposed to contribute to the development of patellar tendinopathy. When looking at the relationship between weight and patellar tendinopathy, Witvrouw reported no significant relationship, whereas Lian et al found that heavier athletes are more likely to develop patellar-tendon pathology. In order for one to make a strong claim about the correlation between weight and patellar-tendon pathology, more evidence must be provided. The role of height in patellar tendinopathy has also not been agreed on. Blazina et al report that patellar tendinopathy is usually seen in tall athletes, and Johnson et al reported that athletes with patellar tendinopathy have longer patellar tendons. Several authors, however, report no significant relationship between height or patellar-tendon length and patellar tendinopathy.

Many authors have also tried to link muscle properties to patellar tendinopathy. Muscle flexibility, muscle strength, and muscle imbalances have all been suggested as potential factors in patellar tendinopathy. Witvrouw et al found no significant association between the concentric strength of the quadriceps-muscle group and patellar tendinopathy. Concentric muscle strength of the hamstring-muscle group was also similar between symptomatics and asymptomatics. Witvrouw did demonstrate decreased flexibility in both the hamstring- and the quadriceps-muscle groups among athletes with patellar tendinopathy when compared with asymptomatic athletes. The correlation between musculotendinous flexibility and patellar tendinopathy might be explained by Fyfe and Stanish's report that decreased flexibility results in an increase in strain on the tendon during movement.

Gender and age have also been studied by multiple authors. It has been suggested that aging might result in tendon degeneration, a decreased healing response, increased tendon stiffness, and decreased vascularity, predisposing older individuals to patellar tendinopathy. Ferretti, however, reports that patellar tendinopathy occurs at any age over 15. Ferretti suggests that during growth the stress is on the nucleus of the bone as opposed to the bone–tendon junction. As a result, children under the age of 15 present with Osgood-Schlatter or Sinding-
Larsen-Johansson disease rather than patellar tendinopathy. Witvrouw et al. and Lian et al. found similar results and reported no significant peak age. Despite reports by Järvinen that most tendon injuries occur in men, Ferretti reports no significant differences between men and women in regard to the occurrence of patellar tendinopathy.

The Role of Jumping in “Jumper’s Knee”

Jumping is an essential component of many sports. McClay et al. report that on average, basketball players jump 70 times per game. Volleyball players have been reported to perform 60 maximal jumps per hour of play. Although jumping is essential to sport, many authors assert that it is the most dangerous factor for patellar tendinopathy because it repeatedly exposes the knee to “violent functional stress,” specifically stressing the knee-extensor mechanism. With each jump, the ground-reaction forces delivered to the body can be up to 8 times one’s body weight. With explosive activities such as jumping, the knee-extensor mechanism is subject to large torsional stresses over a few thousandths of a second between landing and explosive upward thrusts.

Richards et al. studied knee-joint dynamics in 10 Canadian men’s national volleyball team players. He looked at both spike jumping and block jumping. All subjects were right handed. Using a logistic-regression model, they identified several differences in knee-joint dynamics between symptomatic and asymptomatic athletes. Athletes with tenderness on palpation of the inferior pole of the patella without signs of bursitis were classified as having patellar tendinitis. All correlations were positive.

During spike-jump takeoff, maximum ground-reaction force and the time derivative of vertical ground-reaction force were predictive of patellar tendinitis in the right knee. Both factors individually predicted 8 out of 10 cases correctly. Peak tibial external-rotation moment during spike-jump takeoff was another reliable indicator of patellar tendinitis in the right knee, again predicting 8 out of 10 cases correctly.

When looking at spike-jump landing, the maximum knee-flexion angle was a significant predictor of patellar tendinitis, predicting 10 out of 10 cases correctly for the left knee. Eight out of the 10 cases were predicted correctly using the peak time derivative of knee-extensor moment for the right knee.

During block-jump takeoff, Richards et al. demonstrated a significant relationship between maximum ground-reaction force and patellar tendinitis in the right knee, predicting 9 out of 10 cases correctly. Peak tibial external rotation during block-jump takeoff was found to be indicative of left-knee patellar tendinitis.

When interpreting the results presented by Richards and colleagues, it is important to consider the retrospective nature of their study. Although the dynamics differ between the symptomatic and asymptomatic athletes, one cannot determine whether the difference contributes to patellar tendinopathy or the differences result from patellar tendinopathy. Although this can only be determined by a prospective study, it seems reasonable to believe that these differences are a result of the patellar tendinitis. There were several athletes in the study who had a history of
patellar tendinopathy but were asymptomatic at the time of the study. If the knee-joint dynamics were a factor in predisposing athletes to patellar tendinopathy, one would expect all athletes with a history of patellar tendinopathy to demonstrate the same abnormal knee-joint dynamics. That was not seen in this study.

Lian et al also looked at jumping performance in volleyball players. They compared 12 symptomatic and 12 asymptomatic “well-trained” Norwegian volleyball players and found that volleyball players satisfying the diagnostic criteria for patellar tendinopathy performed better on jumping tasks than did matched controls. The athletes with patellar tendinopathy were able to jump higher during the countermovement jump and the standing jump with a 20-kg load. They also performed better during the 15-second rebound jump test. In addition, the symptomatic athletes produced more work than the asymptomatic group during the standing jump and the countermovement jump. Lian et al also looked at the difference between the standing jump and the countermovement jump. The difference allows us to identify the eccentric component of the jump. The greater the difference, the greater the contribution from eccentric muscle force. There was a significantly greater difference between the standing jump and the countermovement jump in the symptomatic group than in the asymptomatic group. This suggests that the symptomatic group had a much greater eccentric force output. This eccentric-component difference was the greatest difference in jumping performance identified in this study.

The extensor mechanism is commonly considered the problem site in patellar tendinopathy. More specifically, eccentric contractions are thought to be detrimental to the tissue. Stanish et al explained that eccentric-force production is often responsible for microruptures, because eccentric contractions exceed concentric and isometric forces by 300%. This in turn might exceed the strength of the tendon, resulting in tissue breakdown. Therefore, one would suspect the quadriceps performance, especially the eccentric quadriceps control, of athletes with patellar tendinopathy to be impaired.

Lian’s results do not support this theory. He suggests that the load placed on the extensor mechanism during ballistic activities such as jumping is responsible for injury. Lian explains that “players who jump well load their tendons more than others, and this may lead to greater risk for injury.” This explains why injured athletes jump better. Again, the interpretation of these results is limited by the study design because the study was retrospective. Lian et al believe that patellar tendinopathy is a result of the jumping mechanics of the athletes, rather than the jumping mechanics being dependent on the condition of the knee.

The tactics of volleyball have changed over time, resulting in an increased incidence of patellar tendinopathy. Middle blockers are required to jump more than other players and also have the highest incidence of patellar tendinopathy. This serves as supporting evidence for the theory that patellar tendinopathy is in fact a jumping injury.

**Diagnosis**

The diagnosis of patellar tendinopathy is primarily based on the patient’s history and clinical presentation. Patellar tendinopathy is most commonly manifested as chronic anterior knee pain of insidious onset, which might be described as sharp
or aching pain. The pain is typically localized to the patellar or tibial insertion of the tendon. Palpation has been found to be a reliable tool in identifying patellar tendinopathy.

In addition to pain on palpation, pain might also be present when ascending or descending stairs or with prolonged sitting or active extension of the knee. The severity of pain varies among athletes. Some report the occurrence of quadriceps atrophy, patellar hypermobility, edema of the infrapatellar tendon, nodules, and crepitus. Blazina et al explain that the report of swelling is actually “a feeling of fullness” at either the superior or the inferior pole of the patella. Blazina also reports the occurrence of momentary weakness or “giving way” of the knee with the absence of true locking or catching.

Blazina et al suggest 4 stages of patellar tendinopathy, with different symptoms at each stage. During stage 1, pain is usually reported only after activity. Characteristically, during stage 2 pain is reported at the beginning of activity and resolves during activity. The pain returns after the activity. During stage 3 pain is typically present for the duration of the activity and after activity. Stage 4 is described as complete rupture of the tendon.

When a patient is complaining of anterior knee pain it is important to rule out other pathologies including Osgood-Schlatter’s disease, a traction epiphysitis condition in active adolescents. Pain is localized to the tibial tuberosity, which might be swollen or prominent. Osgood-Schlatter’s disease is most common in girls between the ages of 8 and 13 and boys from age 10 to 15. Sinding-Larsen-Johansson disease must also be ruled out. Like Osgood-Schlatter’s disease, it is seen in active adolescents. It is characterized by pain, swelling, and tenderness over the inferior pole of the patella. There is pain on palpation of the inferior aspect of the patella with the knee in full extension. When the knee is flexed to 90°, there is less pain with palpation. Finally, one must rule out patellofemoral-pain syndrome, in which pain is typically associated with chronic activity. Whereas Osgood-Schlatter’s disease, Sinding-Larsen-Johansson disease, and patellar tendinopathy are characterized by well-localized pain, individuals with patellofemoral-pain syndrome usually complain of vague symptoms around the patella. Symptoms increase with the knee in 20° to 30° of knee flexion as the posterior aspect of the patella begins to contact the femoral condyles. Pain is elicited with lateral patellar mobilizations. Another condition that is seen in adolescents is osteochondritis dissecans, in which knee pain is generally vague and not well localized. Edema and a decreased range of motion are often reported. Finally, when evaluating adolescents with complaints of knee pain, one should investigate hip pathology including a slipped capital epiphysis.

Many authors also report the use of magnetic resonance imaging, computed tomography, ultrasound, and standard radiograph in the diagnosis of patellar tendinopathy. The reliability and validity of such imaging tools are inconclusive.

Treatment

Clinicians must consider the pain–rest–reinjury cycle when treating patellar tendinopathy (see Figure 2). It is important that athletes with patellar tendinopathy not return to sport before adequate tissue healing. Typically, patellar tendinopathy
Hale is initially managed conservatively. Specifically, conservative management is recommended during phases 1–3, as described by Blazina et al. It usually consists of relative rest, modalities, nonsteroidal anti-inflammatory drugs (NSAIDs), and progressive therapeutic exercise.

Rehabilitation is often approached based on Blazina’s phases-of-disease progression. During phase 1, treatment includes an adequate warm-up, cryotherapy after activity, NSAIDs, knee support, and isometric quadriceps exercises. During phase 2, moist heat before activity is added to the protocol. It is during phase 2 that corticosteroid injections have been administered. More recently, local corticosteroid injections have been associated with detrimental effects and have not been found to result in significant improvement, so they are no longer commonly administered. In addition to the deleterious effects of corticosteroids on tendon health, their injection can also serve to provide a temporary relief of symptoms resulting in an athlete continuing to overload an already weakened tendon. Complete tendon rupture was reported in 4 of 5 athletes after local steroid injection. For athletes classified in the phase 3 stage, the phase 2 protocol is used in conjunction with prolonged rest. It is at this stage that surgery is considered.

When considering the use of modalities, it is important to understand the etiology of patellar tendinopathy. One must identify the rationale behind the use of modalities. If they are being used to control inflammation, they might not be effective during later stages, when evidence suggests the absence of an inflammatory process. In contrast, modalities used to control inflammation might in fact be appropriate during the early stages of patellar tendinopathy when inflammation is active. Modalities might, however, be appropriate for pain control throughout

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**Figure 2** — Pain–rest–reinjury cycle. Reprinted from *Clin Sports Med.* ©1992;11:533-578, Leadbetter W, with permission from Elsevier.
the rehabilitation process. The role of modalities in facilitating the healing process through an increase in blood supply and inflammatory infiltrates also warrants further investigation.

Similarly, because of the absence of inflammatory infiltrates in diseased tendon, the benefits of NSAIDs have been challenged. Although NSAIDs are frequently used to control the inflammatory process, recent findings suggest additional actions of the drugs. Dingle reported effects of NSAIDs on glycosaminoglycans and, ultimately, the extracellular matrix. Therefore, in addition to the anti-inflammatory contributions of NSAIDs, they might in fact alter tissue composition. Like many modalities, NSAIDs might also be effective because of their analgesic properties.

In an effort to develop more effect progressive therapeutic-exercise programs, authors have examined the efficacy of eccentric training in managing patellar tendinopathy. Results of these studies have been positive and indicate that future research in this area is warranted.

Other less common approaches include orthotics, bracing, and extracorporeal shock-wave therapy (ESWT). Orthotics and bracing are sometimes used in an effort to decrease the tensile forces on the patellar tendon. There is little evidence to support or refute the efficacy of bracing or orthotics in the management of patellar tendinopathy. Similarly, little is known about the effectiveness of ESWT. Although it has been approved by the US Food and Drug Administration for use in patients with plantar fasciitis, this procedure has not yet been approved in the United States for patellar tendinopathy. Research regarding the use of ESWT for other chronic tendon pathology is beginning to be published in other countries, there is not sufficient evidence to make a conclusion about clinical outcomes after ESWT.

Surgical intervention should not be excluded without strong consideration. Clinicians often shy away from invasive measures such as surgery, but there are many reports of positive outcomes of treating patellar tendinopathy surgically. Conservative treatment of patellar tendinopathy is often unsuccessful, resulting in prolonged dysfunction for athletes. In such cases, clinicians must consider a surgical consult. There are several surgical procedures that are used. Many of the procedures aim to disrupt the tendon or its bony attachment in an effort to promote inflammation and healing. Others involve surgical debridement of the tendon, in which the degenerative tissue is removed. Some surgeons choose to reattach the tendon, and others do not.

Future Research

As clinicians and researchers gain a better understanding of the pathology and etiology of patellar tendinopathy, research should focus on developing sound rehabilitation programs. Future studies should examine long-term outcomes of various conservative management programs. The efficacy of a prophylactic program also should be examined.

Conclusion

When considering the pathoetiology, it is important to recognize that patellar tendinopathy, like most chronic tendon disorders, is a complex process. It appears that
both an inflammatory response and degenerative change might occur. The etiology is multifactorial, incorporating both intrinsic and extrinsic factors. Evidence suggests that jumping plays a significant role in this disease process. There is a tremendous need for research to improve our understanding of the pathoetiology of patellar tendinopathy and its clinical management.

Current work does demonstrate that patellar tendinopathy is a progressive condition and conservative management is most successful in the early stages. It is important to modify the treatment program based on the stage of the condition. In order to treat patellar tendinopathy effectively, one must consider the disease process that is taking place and whether the condition is presenting as an inflammatory process or a degenerative process. Athletes with more advanced stages of patellar tendinopathy should be considered candidates for surgery.

Acknowledgment

I would like to thank Jay Hertel, PhD, ATC, for his many contributions to the development and revision of this article.

References