Review Article

Patellar Tendinopathy: Diagnosis and Treatment

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Abstract

Patellar tendinopathy is a common cause of pain in athletes' knees. Historically, it has been related to jumping sports, such as volleyball and basketball. Repetitive jumping generates a considerable load of energy in the extensor mechanism, leading to symptoms. The main pathophysiologic phenomenon in patellar tendinopathy is tendinosis, which is a degenerative disorder rather than an inflammatory disorder; therefore, the other popular term for this disease, tendinitis, is not appropriate. The nonsurgical treatment of patellar tendinopathy is focused on eccentric exercises and often has good results. Other experimental options, with variable levels of evidence, are available for recalcitrant cases. Surgical treatment is indicated for cases that are refractory to nonsurgical treatment. Open or arthroscopic surgery can be performed; the two methods are comparable, but arthroscopic surgery results in a faster recovery time.

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Patellar tendinopathy is a clinical condition characterized by activity-related pain in the distal pole of the patella or in the proximal patellar tendon.[1] Symptoms can be so intense that they lead to functional impairment in sports. This condition is thought to result from repetitive stresses in the extensor mechanism of the knee; therefore, it is more prevalent in sporting activities that involve jumping, which explains why this condition also is known as "jumper's knee."[2]

Epidemiology

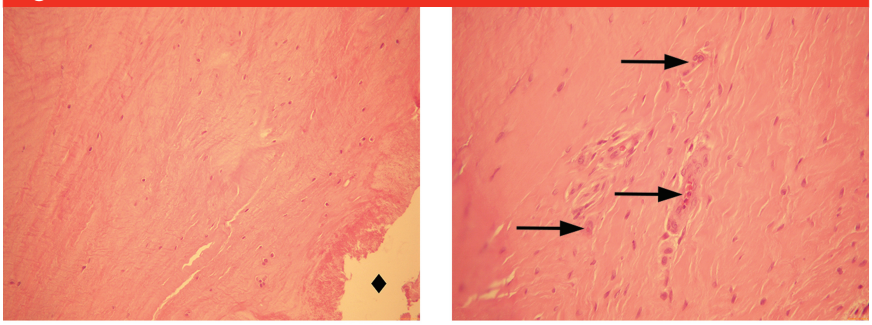
Historically, patellar tendinopathy has been shown to be related to jumping sports. Lian et al[2] reported a 45% prevalence of patellar tendinopathy in professional volleyball players and a 32% prevalence in professional basketball players. In other sports, such as soccer, in which jumping is not the main activity, repetitive stress of the extensor mechanism also can lead to the development of this disease, which has been reported in up to 2.4% of professional soccer players in a single season.[3]

Zwerver et al[4] reported a prevalence of 8.5% in amateur athletes. Volleyball, handball, and basketball players more frequently have patellar tendinopathy (range, 11.3% to 14.4%) than do soccer players (2.5%).

Risk Factors

A recent systematic review of the risk factors for developing patellar tendinopathy showed that anthropometric factors, such as a high body mass index, a large abdominal circumference, limb-length discrepancy, and flatfoot arch, are independent risk factors associated with the development of this condition.[5] The review also noted that other factors, such as weak quadriceps muscles

Figure 1



A B

A, Histologic specimen of a patellar tendon showing connective tissue with increased cellularity and stromal mucoid degeneration (diamond; hematoxylin-eosin stain, magnification × 200). **B,** Histologic specimen showing areas of vascular proliferation (arrows; hematoxylin-eosin stain, magnification × 400). (Courtesy of Mauricio Oyarzo, MD, Santiago, Chile.)

and low flexibility of the quadriceps and hamstring muscles, are associated with patellar tendinopathy. Sex and factors associated with the sport itself, including training time, the training surface, and the type of training, were not found to be correlated with the development of patellar tendinopathy.

Pathophysiology

Among the causes of patellar tendinopathy, extrinsic factors (eg, patellar tendon loading) and intrinsic factors (eg, malalignment, patella alta, abnormal patellar laxity, muscular tightness, imbalance) have been proposed. Frequently, these two types of factors overlap as causes of the tendinopathy. Extrinsic factors have been mentioned most commonly, with mechanical overload in the extensor apparatus discussed most frequently. The key pathophysiologic phenomenon of patellar tendinopathy is tendinosis, which refers specifically to the degenerative origin of this disease. Tendinosis typically is located in the posterior portion of the proximal end of the patellar tendon adjacent to the inferior patellar pole.[1] It is characterized by progressive degeneration of the tendinous tissue, an inability of the tissue to repair itself, and the absence of inflammatory cells. Macroscopically, the affected region appears yellow and commonly is described as "mucoid degeneration"[6] (Figure 1, A). This appearance contrasts with the normal bright, white, fibrous appearance of tendinous tissue.

Increased neovascularization in the tendon has been proposed to accompany the lack of reparative capacity and to be directly related to pain[7] (Figure 1, B). Another factor that has been linked to pain in patients with patellar tendinopathy (in the absence of inflammatory cells) is the presence of inflammatory mediators, in particular cyclooxygenase-2.[8]

Although extrinsic factors are the most widely accepted causes of patellar tendinopathy, they alone cannot explain why the condition does not develop in all persons with the same activity level and similar risk factors. Therefore, intrinsic causes have been proposed.[9] Nevertheless, the relevancy to tendinopathy of the proposed intrinsic factors, including malalignment, patella alta, abnormal patellar laxity, muscular tightness, and imbalance,[9] has not been established and their importance remains a subject of debate. The most commonly accepted theory is that patellar tendinopathy is caused by an interplay of extrinsic and intrinsic factors.

History and Physical Examination

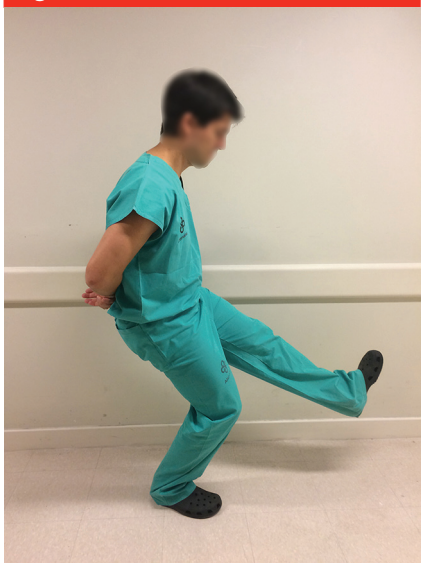
Patellar tendinopathy presents as activity-related anterior knee pain highly localized to the distal pole of the patella and the proximal end of the patellar tendon.[1] The pain is insidious and can be precipitated by an increase in sports activity. Pain initially starts after physical activity but can progress to a point at which it is present during any activity or is continuous, existing even at rest.

On physical examination, pain occurs at the proximal patellar tendon when it is palpated and at the distal pole of the patella when the leg is fully extended; pain at the latter site decreases with knee flexion. One test to reproduce the symptoms associated with patellar tendinopathy is the decline squat test, in which a single leg squat is performed to 30° of knee flexion with the other knee extended. This test produces a substantial load on the patellar tendon, causing the onset of pain in the region[10] (Figure 2). A weak quadriceps and tightness of the quadriceps and hamstring muscles also can be seen on physical examination.

Imaging

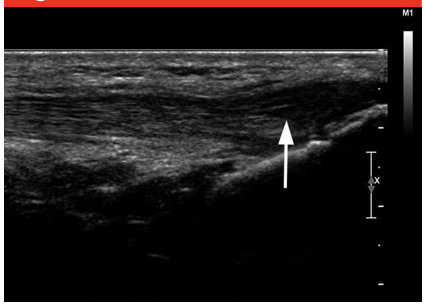
The imaging techniques of choice for the study of patellar tendinopathy are ultrasonography and MRI. Ultrasonography can locate intratendinous lesions that appear as zones of lower echogenicity, typically in the posterior portion of the patellar tendon adjacent to the

Figure 2



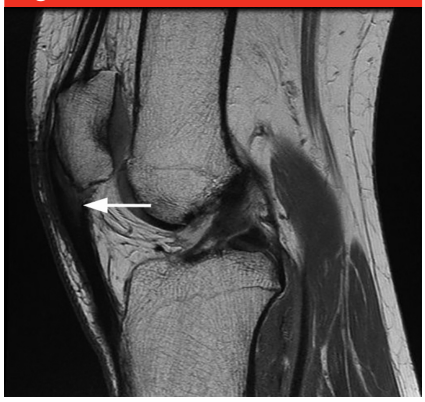
Clinical photograph demonstrating the decline squat test.

Figure 3



Sonogram showing zones of lower echogenicity (arrow), typically in the posterior portion of the patellar tendon adjacent to the inferior pole of the patella.

Figure 4



Sagittal T1-weighted magnetic resonance image showing increased signal in the posterior region of the proximal patellar tendon and the inferior pole of the patella, with tendon thickening (arrow).

inferior pole of the patella (Figure 3). Other common sonographic findings include thickening of the patellar tendon, irregularity of the paratenon, intratendinous calcifications, and erosions in the inferior pole of the patella. The disadvantages of ultrasonography include its operator dependence and the limited ability to rule out intra-articular disease with this modality. The sensitivity and specificity of ultrasonography for patellar tendinopathy are 58% and 94%, respectively.[10]

A common finding on MRI is increased signal in the posterior region of the proximal patellar tendon and the inferior pole of the patella, with tendon thickening (Figure 4). An advantage of MRI over ultrasonography is its capacity to delineate intra-articular pathology, allowing a broad spectrum of diseases to be included in the differential diagnosis. Disadvantages of MRI include higher cost, less availability, and a longer examination time. The sensitivity and specificity of MRI for patellar tendinopathy are 78% and 86%, respectively.[10] Given its ability to rule out other pathologies and because of its greater sensitivity, we recommend that MRI be the first imaging option, if available. Ultrasonography should be reserved for cases in which MRI is not an option.

It is important to note that neither imaging modality is diagnostic. Each should be used only as a guide for the physician because patellar tendinopathy is diagnosed based on patient history and physical examination. Various studies have found no association between findings on ultrasonography or MRI and the severity of the disease or its evolution after treatment.[11-13] The only proven association between imaging findings and disease severity was reported by Cook et al;[14] the risk of future development of symptoms was 4.2 times greater in subjects with abnormal findings on ultrasonography than in control subjects with normal findings.

Classification

In 1973, Blazina et al[15] classified patellar tendinopathy based on its clinical features alone (Table 1). His classification system includes four progressive stages graded according to disease severity. Stage I is characterized by pain that occurs only during sports. In stage II, pain occurs at the beginning of sporting activities, disappears after warm-up, and reappears when fatigue presents. Stage III is characterized by constant pain during activity and at rest, and stage IV disease is defined as patellar tendon rupture. Other classifications based on sonographic or MRI characteristics of the patellar tendon exist; however, their utility is not well established, and the classification system by Blazina et al[15] remains the most referenced system in clinical studies of patellar tendinopathy.

Table 1

Classification of Patellar Tendinopathy[15]

Stage	Description
I	Pain only after sports
II	Pain at the beginning of sports, disappearing after a warm-up, but reappearing during fatigue
III	Constant pain at rest and with activity
IV	Complete rupture of the patellar tendon

Table 2

Differential Diagnoses for Patellar Tendinopathy

Patellofemoral pain syndrome
Fat-pad syndrome
Meniscal tears
Cartilage lesions
Referred pain

Differential Diagnosis

The differential diagnoses for patellar tendinopathy are listed in Table 2. The most important differential diagnosis in patellar tendinopathy is patellofemoral pain syndrome. This syndrome is usually simple to differentiate from patellar tendinopathy because the subjective and objective features of patellar tendinopathy are generally distinctive. In some cases, however, the differential diagnosis may be difficult because the two conditions may coexist.

In addition to patellofemoral pain syndrome, patellar tendinopathy needs to be differentiated from fat-pad syndrome. An assessment of other potential coexisting conditions, such as meniscal tears and cartilage degeneration, may need to be considered. Also, the potential for pain referral to the knee should not be ignored.[10]

Nonsurgical Treatment

Eccentric Exercises

Eccentric exercises have been proposed to increase the remodeling process of the collagen fibers in the patellar tendon. This process causes the musculoskeletal unit to adapt to protect itself from the stress related to physical activity. Excellent results have been reported in athletes and nonathletic patients. Findings suggest that eccentric exercises have positive effects with no adverse reactions.[16] A recent systematic review concluded that eccentric exercises are the only treatment of patellar tendinopathy that has the support of high-level evidence.[17] Therefore, we advocate the use of eccentric exercises as the initial treatment for most patients with patellar tendinopathy, especially when the disorder is in the early stages.

Several different types of eccentric patellar tendon loading exercises are available. In a systematic review, Visnes and Bahr[18] concluded that the ability to specify which protocol component is responsible for the positive effects in patellar tendinopathy is limited. The available studies, which are small and of variable quality, indicate that the treatment program should include a decline board, should be performed with some level of discomfort, and should include the removal of athletes from sports activity.[19-25]

Extracorporeal Shock Wave Therapy

The utility of extracorporeal shock wave therapy (ESWT) for patellar tendinopathy is based on three theories.[26] The first theory is that pain relief is achieved by hyperstimulation analgesia, in which overstimulation of the painful area leads to a diminished transmission of signals to the brain stem. A second theory presumes that the mechanical load developed by ESWT stimulates tissue regeneration. The third theory asserts that ESWT destroys calcifications in tendons in the same way that lithotripsy destroys kidney stones. Today, ESWT is debated as a treatment option for patellar tendinopathy, although a recent systematic review concluded that it is a safe and promising treatment.[27]

Wang et al[28] conducted a randomized study that compared ESWT with physical therapy and NSAIDs and found substantial differences in pain, Victorian Institute of Sport Assessment scores, range of motion, functional improvement, overall outcomes, and sonographic vascularity during follow-up that favored the ESWT group. Another, more recent prospective randomized study did not find any benefit for using ESWT, however.[29] Given the controversial evidence, more controlled studies are necessary before ESWT can be recommended, and it remains an experimental treatment for patellar tendinopathy.

Injections

Steroids

We do not recommend steroid injections for the treatment of patellar tendinopathy. Three randomized studies failed to demonstrate long-term relief with the use of steroid injections, which pose a potential risk of tendon rupture.[30-32] In a randomized study of placebo versus steroid injection, Fredberg et al[30] concluded that a steroid injection can normalize the lesions in patellar tendons that are found on ultrasonography and can have dramatic clinical effects over a short-term period, but have no effect in a 4-week to 6-month follow-up period.

Kongsgaard et al[31] conducted a randomized study comparing steroid injections, eccentric training, and heavy slow resistance training for patellar tendinopathy. They concluded that all the therapies studied have good short-term effects; however, at long-term follow-up, only the eccentric training and resistance groups maintained the effects, with steroid injections having no beneficial effects at 6-month follow-up. Capasso et al[32] compared the efficacy of steroids with aprotinin versus training protocols for management of patellar tendinopathy in athletes. The authors found that steroid

treatment resulted in short-term improvement, but that improvement deteriorated by the 6-month follow-up.

Platelet-rich Plasma

Platelet-rich plasma (PRP) is thought to be capable of enhancing tissue repair because of its high concentration of growth factors. A recent systematic review showed that studies comparing PRP with other treatments had inconsistent results and that none of the studies showed marked differences between PRP and other treatments.[33] Therefore, PRP is a promising therapy, but controlled studies are necessary to determine its true efficacy.

In a recent systematic review, Jeong et al[34] reported that PRP seems to have a positive effect in treating patellar tendinopathy, but the available evidence is of low quality and, thus, this finding is not definitive. Furthermore, no standard protocol for obtaining PRP exists and the number of injections needed is variable. Some evidence shows that multiple injections are better than one injection, however.[35,36] In contrast, Kaux et al[37] found no benefit for successive injections compared with a single injection. Given the inconsistent results and the lack of uniform protocols for treatment of patellar tendinopathy with PRP, this treatment should be considered investigational.

Cell-based Therapies

Limited data are available on the use of cell-based therapies for patellar tendinopathy. It is theorized that the addition of stem cells could improve the speed and quality of the repair process and autocrine and paracrine factors could enhance tissue healing and remodeling. Much of the research in this area involves nonrandomized cases studies or series and animal models of the disease.[38]

Pascual-Garrido et al[39] conducted a study in which eight patients with patellar tendinopathy in whom nonsurgical treatments had failed were injected with mononuclear bone marrow cells. At 5-year follow-up, seven patients stated that they were satisfied with the procedure and that they would be willing to undergo the same procedure again if the disease developed in the contralateral knee.

Clarke et al[40] injected skin-derived tenocyte-like cells in 33 knees in a prospective randomized study of patients in whom nonsurgical treatments had failed. Faster and greater improvement in pain and function were observed in the group treated with the cells. Cell-based therapies for management of patellar tendinopathy are promising, but more evidence is needed to recommend them. Their use remains experimental, and patients being offered such treatments must be counseled regarding this experimental status.

Hyaluronic Acid

High–molecular-weight hyaluronic acid has been reported to have an anti-inflammatory effect in addition to promoting tendon healing at the bone-tendon interface and tissue regeneration.[41] In a recent level IV study of 50 patients with patellar tendinopathy that did not respond to a minimum 2-month course of nonsurgical treatment, a mean of two injections of hyaluronic acid had positive effects on recovery.[41] High-quality evidence regarding this treatment is still lacking. More studies are needed to determine the efficacy of this treatment option, which remains at an investigational stage.

Sclerosing Agents

Neovascularization is a phenomenon that is considered relevant in the pathophysiology of patellar tendinopathy, and it is present in 60% to 80% of patients with pain.[7] Therefore, sclerosing agents are used to inhibit vessel formation, collapse vessels that have already formed, and destroy the accompanying vasa nervorum, which has a denervating effect.

In a level IV study, Alfredson and Ohberg[42] reported a considerable reduction in pain during activity following an ultrasound-guided injection of a sclerosing agent (5 mg/mL polidocanol) to the paratenon, which indicates that sclerosing agents have the potential to manage pain. Hoksrud et al[43] administered an ultrasound-guided injection of polidocanol (10 mg/mL) to the paratenon in patients with painful chronic patellar tendinopathy and found a substantial difference in the Victorian Institute of Sport Assessment-P score in the group treated with a sclerosing agent versus a placebo. However, in a subsequent study of the same group with a longer follow-up (44 months), more than one third of the group treated with sclerosing agents underwent surgery for pain.[44] Therefore, the usefulness of sclerosing agents remains unclear, and the agents still are at an experimental stage.

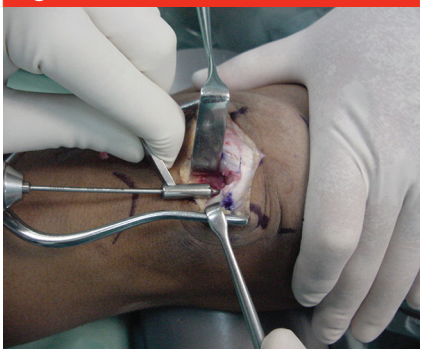
Other Injections

Various other injections have been studied for the treatment of patellar tendinopathy. Aprotinin, autologous blood, dry injections, and high-volume injections all have been used; however, studies of these treatment options are of low quality and the effects of these treatments remain unclear. Therefore, at this time, it is impossible to recommend their use for management of patellar tendinopathy. Thus, they remain experimental treatments.[45]

Additional Treatments

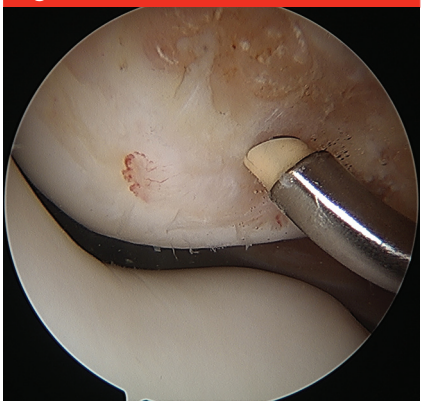
Various other treatments have been examined in level I studies. Glyceryl

Figure 5



Intraoperative photograph depicting the open surgical technique. The inferior pole of the patella is perforated using a drill to produce a bleeding bed that promotes repair.

Figure 6



Arthroscopic view showing the inferior pole of the patella after the débridement of degenerative tissue.

trinitrate delivers nitric oxide, which has exhibited a role in fibroblast proliferation, collagen synthesis, and the contraction of collagen lattices. Macrophage angiogenic activity, which is important for wound healing, also depends on nitric oxide synthase, and nitric oxide synthase activity is upregulated in tendinopathy.[46] Based on the positive effects of glyceryl trinitrate reported in three studies of other types of tendinopathy,[47-49] Steunebrink et al[46] conducted a randomized study of patients with patellar tendinopathy comparing the use of a glyceryl trinitrate patch with that of a placebo patch. The authors did not find any difference in outcome between the two groups.

Low-intensity pulsed ultrasonography can stimulate in vitro collagen production from fibroblasts and may increase mechanical strength following the repair of acute tendon injuries. Stasinopoulos and Stasinopoulos[21] and Warden et al[50] each conducted a randomized study on the effectiveness of low-intensity pulsed ultrasonography and found that this modality provided no benefit compared with eccentric exercises for management of patellar tendinopathy. These findings are supported by an earlier study by Giombini et al[51] that showed that hyperthermia was more effective than low-intensity pulsed ultrasonography for the treatment of patellar tendinopathy. All these modalities remain at an investigational stage and are not recommended for management of patellar tendinopathy.

Surgical Treatment

Although nonsurgical treatment is successful in most patients with patellar tendinopathy, approximately 10% of all patients do not respond to it and eventually require surgery.[52] Several surgical techniques have been described, including open surgery and arthroscopic surgery.[53] The goals of these techniques are tenotomy of the patellar tendon, excision of abnormal tissue, and induction of the repair process through stimulation of the inferior pole of the patella by drilling and marginal resection.

Open Surgery

A longitudinal incision is made from the inferior pole of the patella to the tibial tubercle. The paratenon is exposed and opened longitudinally to expose the patellar tendon. Through a longitudinal incision into the tendon, the posterior degenerative tissue is approached and débrided. The distal 5 mm of the patella are resected using a saw blade. Then, the inferior pole of the patella is perforated using a drill to produce a bleeding bed that promotes repair (Figure 5). Finally, the tendon and paratenon are closed with resorbable sutures.

Arthroscopic Surgery

The procedure starts with diagnostic arthroscopy to rule out intra-articular pathology. It is of particular importance to rule out patellar chondral lesions, which frequently are associated with patellar tendinopathy. The inferior pole of the patella is identified, and the adjacent synovial tissue is resected, exposing the degenerative tissue in the posterior zone of the proximal patellar tendon, which is resected until normal tendon fibers are observed (Figure 6). The distal 5 mm of the patella are resected using a burr. Finally, hemostasis is achieved, and articular lavage is performed.

We use the same rehabilitation protocol for open and arthroscopic surgery, consisting of full weight bearing and free range of motion as tolerated. When the wounds are healed at approximately 10 days postoperatively, the patients start a rehabilitation program that consists of pain management, the initiation of eccentric squats on an inclined board, and a strengthening program. Patients are allowed to return to sport when they have completed at least 3 months of supervised rehabilitation and are pain free during strengthening exercises.

Results

Comparable results for open and arthroscopic surgery have been described in the literature. A recent

systematic review reported that the mean rate of success for surgical treatment of patellar tendinopathy is 87% for open treatment and 91% for arthroscopic treatment.[54] In the studies reviewed, the mean time to return to sports was 3.9 months for arthroscopic treatment and 8.3 months for open treatment. The average rate of return to sporting activities was 82.3% after arthroscopic surgery and 78.4% after open surgery.[54] In patients who underwent open surgery with the resection of the inferior pole of the patella, clinical scores were better than in those who underwent the procedure without resection. However, no difference was observed in the rate of return to sport, and the time to return to sport was longer for the group treated with bony patellar resection. In patients treated with arthroscopic surgery, those who had resection of the inferior pole of the patella had better rates of return to sports than did those without resection of the inferior patellar pole, but there was no difference in clinical scores or the time to return to sports between the two groups.

No clear guidelines exist for the use of one technique over another. However, given that arthroscopic treatment has results comparable to those of open surgery but yields a faster return to sport, we recommend arthroscopic treatment of patellar tendinopathy as long as the surgeon has experience using this technique.

Summary

Historically, patellar tendinopathy has been associated with jumping sports. The main pathophysiologic phenomenon of patellar tendinopathy is tendinosis, which is a degenerative disorder rather than an inflammatory disorder. Therefore, the popular term for this disease, tendinitis, is not appropriate. Nonsurgical treatment primarily consists of eccentric exercises, which often have good results. If a patient does not respond to these exercises in a 4- to 6-month period, other nonsurgical options may be attempted, but all of these options are in experimental stages. ESWT and injections of PRP or other agents are promising treatments, but more evidence is required before they can be recommended. For cases refractory to nonsurgical treatment, surgery is indicated and can be performed open or arthroscopically. The two methods are comparable, but arthroscopic treatment results in a faster return to sport.

Acknowledgment

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References

Evidence-based Medicine: Levels of evidence are described in the table of contents. In this article, references 9, 19-25, 29, 30, 32, 40, 43, and 46-51 are level I studies. References 3, 4, 17, 18, 28, 31, 36, and 37 are level II studies. References 5, 7, 8, 12, 14, 20, 33, 38, and 45 are level III studies. References 2, 11, 13, 15, 34, 35, 39, 41, 42, 44, 52, and 54 are level IV studies. Reference 1, 6, 10, 16, 26, and 27 are level V expert opinion.

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