[Orthopaedic Surgery]

Patellar Tendinopathy

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Context: Patellar tendinopathy is a common condition. There are a wide variety of treatment options available, the majority of which are nonoperative. No consensus exists on the optimal method of treatment.

Evidence Acquisition: PubMed spanning 1962-2014.

Study Design: Clinical review.

Level of Evidence: Level 4.

Results: The majority of cases resolve with nonoperative therapy: rest, physical therapy with eccentric exercises, cryotherapy, anti-inflammatories, corticosteroid injections, extracorporeal shockwave therapy, glyceryl trinitrate, platelet-rich plasma injections, and ultrasound-guided sclerosis. Refractory cases may require either open or arthroscopic debridement of the patellar tendon. Corticosteroid injections provide short-term pain relief but increase risk of tendon rupture. Anti-inflammatories and injectable agents have shown mixed results. Surgical treatment is effective in many refractory cases unresponsive to nonoperative modalities.

Conclusion: Physical therapy with an eccentric exercise program is the mainstay of treatment for patellar tendinopathy. Platelet-rich plasma has demonstrated mixed results; evidence-based recommendations on its efficacy cannot be made. In the event that nonoperative treatment fails, surgical intervention has produced good to excellent outcomes in the majority of patients.

Keywords: patellar tendinopathy; eccentric therapy; jumper's knee

Patellar tendinopathy is a common condition, often presenting with anterior knee pain. A thorough history and physical are necessary, but imaging can also aid in the diagnosis. The majority of cases resolve with nonoperative treatment.[1,6,10,12,26,28,30,46,66,81,83] Once patients pass the initial inflammatory phase and remain symptomatic, treatment becomes more difficult.

PATHOPHYSIOLOGY AND PATHOMECHANICS

There are several factors that influence the development of patellar tendinopathy, with a high prevalence among athletes in jumping sports, particularly basketball and volleyball.[48] Nine factors contribute to the pathogenesis of patellar tendinopathy: weight, body mass index (BMI), waist-to-hip ratio, leg length difference, arch height of the foot, quadriceps flexibility and strength, hamstring flexibility, and vertical jump performance.[90] These risk factors may increase the strain on the patellar tendon.

There are several theories on the pathogenesis of patellar tendinopathy, including vascular,[50] mechanical,[51,52] impingement–related,[36] and nervous system[2,3,75] causes. Chronic repetitive tendon overload is the most commonly proposed theory.[38,45,67] Overload may result in weakening of the tissue, and eventually, catastrophic failure.[67] The increased strain is located in the deep posterior portion of the tendon, closer to the center of rotation of the knee and the inferior pole of the patella, especially with increased knee flexion.[38] Microscopic failure occurs within the tendon at high loads and eventually leads to alterations at the cellular level, which weaken the mechanical properties.[67] Tendon microtrauma can lead to individual fibril degeneration due to stress across the tendon,

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which can accumulate over time, leading to chronic tendinopathy,[67] which has been reproduced by complex computer models.[45]

Both in vitro and in vivo studies have shown that neovascularization and an increased quantity of specific proteins and enzymes can also contribute to tendon degeneration.[50] The increased strain affects the tenocytes, altering protein and enzyme production as well as deforming the nucleus.[51,52] Loading of the tendon fibroblasts increases prostaglandin E2 and leukotriene B4, which both contribute to tendinopathy.[67] In vitro studies have shown that vascular endothelial growth factor (VEGF) and matrix metalloproteinase (MMP) activity have also been linked to tendon breakdown.[52,75] In vivo studies show that VEGF may have a role in the neovascularization process, possibly because of new nerves following new blood vessels causing the pain.[2,3,75] Although several studies have shown neovascularization associated with tendinopathy, there are also conflicting studies showing no association with neovascularity.[17,33,35]

A second proposed mechanical etiology is inferior patellar pole impingement shown on magnetic resonance images (MRIs) during flexion.[36] Although biomechanically unproven, this may be why inferior pole patella excision improves symptoms.[31] In contrast, however, measurements of both symptomatic and asymptomatic knees in an open-configuration MRI found no significant difference in morphology, arguing against impingement.[74]

End-stage patellar tendinopathy shows absent or minimal inflammation[16,27,40,92] because the underlying pathology is degenerative. The diseased tendon shows hypercellularity with atypical fibroblast and endothelial cell proliferation along with neovascularization.[27,40,65,69,92] Loss of the longitudinal collagen fibers, demarcation between the collagen bundles, and relative expansion of the tendon are also seen.[16,27,40,65,92] There are occasional gaps in the collagen fibers, signifying ruptures of the individual collagen fibrils.[16,27,40,65,92] Diseased tendons also have a higher percentage of cells undergoing apoptosis, as well as multiple pro-apoptotic proteins and genes present.[7,47] Macroscopically, tendinopathy has a disorganized appearance described as "mucoid degeneration."[19,25,55]

TREATMENT AND OUTCOMES

Nonoperative Treatment

There is no consensus regarding the best treatment plan. There are no studies of rest or cessation of activity. Avoidance of jumping activities with stretching after physical activity may help in early disease.[23] Relative rest is preferred to complete immobilization, as the latter would cause tendon and muscle atrophy.[64]

Cryotherapy is primarily used for analgesic effect. Cold counteracts the neovascularization process, which may be a cause of tendinopathy.[68] Many variables influence ice application: temperature, duration, depth of cooling, and so on.[50] Icing should not be used before athletic participation; it may impair motor function and mask pain, leading to reinjury.[54,68]

A patellar counterforce strap has been used for more than 30 years in the treatment of anterior knee pain, including patellar tendinopathy. The strap may decrease patellar tendon strain by altering the angle between the patella and patellar tendon.[44] Some studies have shown poor results,[20,55,82] where others have shown improvement in pain and symptoms.[1,10,12,28,30,46,66,81] These studies assessed patellofemoral pain in general. No level 1 studies had been performed specifically related to patellar tendinopathy.

The use of nonsteroidal anti-inflammatory drugs (NSAIDs) to treat tendinopathic pain has been a mainstay of treatment; however, their use has come into question. The histopathology of chronic tendinopathy shows a lack of inflammatory cells, further fueling the debate regarding NSAIDs. A systematic review of 17 randomized, placebo-controlled clinical trials evaluating NSAIDs in the treatment of tendinopathy concluded that NSAIDs were useful in the short term (7-14 days), particularly in shoulder pathology.[5] There was no long-term benefit from anti-inflammatories. Some question the effect of NSAIDs on tendon healing as well.[4,43,51,87] Studies have shown healing,[13,21,87] while others highlight negative effects.[4,43,51,87]

The most popular nonoperative treatment involves eccentric exercise (EE). While there are several exercise programs,[83] there is no consensus on which is best. Opinions differ on duration and frequency of therapy, drop squats versus slow eccentric movement, a decline board, and exercising until tendon pain. In a level 2 study, at 12 weeks, patients undergoing an EE program improved significantly compared with those undergoing a concentric exercise program.[37]

The load on the patellar tendon can be maximized by squatting on a 25° decline board.[66] In a comparison of traditional EE with a decline board program[91] at 12 months, both groups showed improvement, with the decline group showing a 94% chance of a positive result versus only 41% in the traditional group. In a comparison of primary surgery with an EE program on a decline board, at 12 months there was significant improvement in both, without difference between the groups.[6]

Conversely, a decline board program was compared with normal training in elite volleyball players during the season and found no significant difference at 6 weeks or 6 months.[84] In a comparison of a decline board program to an eccentric squat training program, there was improvement in both groups at 3 months without significant difference.[26] The success rate with an EE program has been noted to be between 50% and 70% (Appendix 1, available at http://sph.sagepub.com/content/by/supplemental-data).[83]

Corticosteroids have been used in the treatment of various tendinopathies.[13,22,24,39,42,58,78] Corticosteroids can affect synthesis of extracellular matrix (ECM), collagen production and deposition, scar formation, and tenocyte proliferation and viability.[89] Corticosteroids induce a vasoconstrictor effect on smooth muscle cells, thereby decreasing the production of nitric oxide and possibly decreasing pain.[80] Most studies on local

corticosteroids, although not well designed, show good short-term pain relief[13,22,24,39,42,58,78] and decreased swelling and vascularization in patellar tendinopathy.[22,24] Two level 1 studies examined corticosteroids and physical therapy in patellar tendinopathy.[22,42] In 24 patients, 12 received 2 to 3 corticosteroid injections (CSIs) and 12 received a placebo injection of lidocaine, with all patients participating in physical therapy. All patients improved, especially in the short term, with pressure and walking pain significantly improved in the CSI group at 4 weeks. However, at the 2-year follow-up, 14 patients required surgery for continued symptoms. The high recurrence rate may be because of the steroids and aggressive rehabilitation. In 37 patients (12 in CSI, 12 in eccentric training, and 13 in heavy slow resistance training), all refrained from training and sporting activities for 1 week. At the 12-week follow-up, all groups improved significantly ($P < 0.01$), with an average increase in Victorian Institute of Sport Assessment (VISA) score of 18 in the CSI group and 22 in both the eccentric training and heavy slow resistance group. At 6 months, both physical therapy groups had good results (similar VISA scores as the 12-week mark), while the steroid group had a relapse of symptoms, emphasizing that CSI is effective in the short term. Caution must be taken with patellar tendon injection because of risk of tendon rupture.[8,9,24,58] Seven cases of patellar tendon rupture have occurred after multiple steroid injections in weight lifters with patellar tendinopathy.[14]

Aprotinin injections have been used to treat patellar tendinopathy.[13,51,57] It is a strong inhibitor of MMPs.[51] In a level 1 study of corticosteroids, aprotinin, and placebo, aprotinin had 72% good/excellent results compared with 59% in the CSI group and 28% in the placebo group.[13] In 430 patients treated with aprotinin, there was a high risk of side effects, including anaphylaxis and bovine spongiform encephalopathy.[57]

Another nonsurgical treatment option is a sclerosing injection with a chemical irritant (eg, polidocanol).[2,3,32,34,75,88] These target neovascularization and the accompanying nerves.[2,3,75] Fifty patients (33 in the sclerosis and 17 in a placebo group) all underwent 2 weeks of light training, followed by training as pain permitted.[34] At 4 months, the treatment group showed a significant improvement in VISA score ($P = 0.01$) compared with placebo, which did not show significant improvement ($P = 0.86$). This study was limited by the short cross-over time (4 months) and lack of long-term follow-up.[34] Twelve of 16 patients in the control group crossed over. In 29 patients undergoing sclerosing treatment[32] at 44 months, 41% (12/29) had undergone arthroscopic treatment; the remainder had good clinical outcomes. In 26 knees undergoing sclerosing treatment and 26 undergoing arthroscopic debridement at 6- and 12-month follow-up, both groups had statistically significant reductions in their visual analog scores (VASs) at both rest and with activity ($P = 0.004$ and $P = 0.001$, respectively).[88] The arthroscopic group had less pain, higher satisfaction, and quicker return to sport.

A current treatment option is platelet-rich plasma (PRP) injection,[11,19,29,41,85] which has some good outcomes.[29,41,85] Unfortunately, there are currently no level 1 or 2 studies. There are no treatment standards for dosage, injection technique, timing, or number of injections. Case report data have shown worsening patellar tendinitis symptoms after PRP injection (Table 1).[11]

Extracorporeal shock wave therapy (ESWT), generating high forces in the tendon, may produce analgesic benefits by mechanical disintegration of calcium deposits and stimulation of tissue reparation.[15,71,72] There is no consensus on the method of application, shockwave generation, energy level, localization, number and frequency of treatments, or the use of anesthesia.[15,76] In 13 patients with surgical tenotomy and 14 with ESWT, both groups had statistical improvement at 22 to 26 months without significant difference, with the VISA scores improved to 70.7 for the surgery group and to 83.9 from 78.8 for the ESWT group. Seven patients in the surgical group and 8 in the ESWT group had returned to sports at the same level as prior to their knee pain.[63] In 30 knees treated with ESWT and 24 with NSAIDs, physical therapy, and a patellar strap at the 2- and 3-year follow-up, the treatment group had significantly decreased pain (6 to 0.59; $P = 0.001$), increased function, and improved overall VISA scores ($P < 0.001$).[86] In a level 1 study comparing ESWT to placebo in 62 symptomatic athletes who were in training or in competition with less than 12 months of symptoms, there was no significant difference between the groups at 1, 12, or 22 weeks.[94]

Another nonoperative treatment option is the glyceryl trinitrate (GTN) patch, which delivers nitric oxide (NO) to the pathologic tendon.[59-61,79] NO may play a role in tendon healing after injury in vitro[93] by stimulating fibroblast proliferation and collagen synthesis.[56] Some studies have shown the healing potential of GTN[59-61]; the amount of NO released by the patch is unknown.[79] In a level 1 study comparing GTN (n = 16) and a placebo patch (n = 17) with concurrent eccentric training at 24 weeks, both groups showed improvement, with no significant difference between the groups.[79]

Operative Treatment

Surgical intervention may be warranted for refractory cases.[18,31,49,53,62,63,73,77] The most common form of open treatment is debridement of the affected tissue.[18,63] In 11 knees that failed conservative treatment, open debridement and drilling of the inferior pole of the patella produced 7 (64%) excellent, 3 (27%) good, and 1 (9%) poor result at an average of 2.1 years follow-up.[62] A similar technique obtained 86% (6/7) excellent and 14% (1/7) fair results at a 4.2-year follow-up[31] and another obtained 70% excellent results and 82% return to play.[18]

Arthroscopically, through a direct inferior patellar portal, the retropatellar tissue at the site of the attachment of the patellar tendon was debrided, with complete resolution of pain in 2 patients.[70] Arthroscopic resection of part of the distal pole of the patella achieved 90% (18/20) good to excellent results.[49] Excision of a portion of the patella[62] had significant improvement in International Knee Documentation Committee (IKDC) score (from 51.6 to 84.2; $P = 0.023$), Lysholm, and VISA

Table 1. Summary of Injection Treatment Studies for Patellar Tendinopathy

Authors	Type of Study	Treatment (by group)	Number of Subjects (total/study group)	Results	Authors' Conclusion	Grade of Recommendation
Fredberg et al[23]	RCT	1. 10 mg/mL lidocaine and 20 mg triamcinolone 2. Placebo: 3.5 mL lidocaine and 0.5 mg 20% intralipid	24/12	Treatment groups improved, especially in the short term; 58% of patients were operated on at 2 years	Steroid injections can normalize US pathologic lesions and have dramatic clinical effects but with aggressive rehabilitation; many relapse within 6 months	A
Kongsgaard et al[42]	RCT	1. 1 mL of 40 mg/mL methylpredinose in 0.5 mL lidocaine 2. Eccentric training 3. Heavy slow resistance training	37 (12/12/13)	At 12 weeks, all groups improved significantly, but at 6 months, group 1 declined but groups 2 and 3 were unchanged	Good short-term results with steroids but poor long-term clinical effects; good short- and long-term effects with eccentric training and slow resistance training	A
Capasso et al[13]	RCT	1. 62,500 units aprotinin and 2.5 mL lidocaine 2. 50 mg methylpredinisolone acetate and 2.5 mL lidocaine 3. 5 mL 0.9% NaCl	116 (38/39/39)	At 1 and 12 weeks, significant improvements were seen: aprotinin group was better than the steroid group, and both were better than the placebo group	Aprotinin may have lasting beneficial effect; further evidence needed	A
Hoksrud et al[34]	RCT	1. 10 mg/mL polidocanol injections; 2 mL per knee maximum 2. Lidocaine with epinephrine injections	33/17	At 12 months, significant improvements in VISA and overall satisfaction in the sclerosis group	Sclerosing injections with polidocanol resulted in a significant improvement in knee function and reduced pain in patients with PT	A
Wilberg et al[88]	RCT	1. 10 mg/mL polidocanol injections; 2 mL per knee max 2. US-guided arthroscopic shaving	45 (52 tendons/26 tendons)	At 6 and 12 months, patients treated with arthroscopic shaving had significantly improved VAS scores for pain at rest and during patellar tendon loading activity and were significantly more satisfied	Both treatments showed good clinical results, but patients treated with arthroscopic shaving had less pain and were more satisfied with the treatment result; because surgical treatment is a 1-stage treatment, return to sports was faster in this group	A

PT, patellar tendinopathy; RCT, randomized controlled trial; US, ultrasound; VAS, visual analog scale; VISA, Victorian Institute of Sport Assessment.

scores, with 71% (19/27) of professional athletes able to return to sport. Similarly, an arthroscopic technique without excision of the distal pole of the patella produced an 82.6% (19/23) return to play rate.[73] Arthroscopic removal of hypertrophic synovium and fat pad without resection of patellar tendon or distal pole of the patella[53] showed a 76.7% (23/30) return to play rate and 90% good or excellent outcomes (Appendix 2, available at http://sph.sagepub.com/content/by/supplemental-data).

CONCLUSION

Initially, a nonoperative regimen consisting of physical therapy with eccentric exercises is the mainstay of patellar tendinopathy treatment. Alternative treatments ranging from oral anti-inflammatories to injectable agents have demonstrated mixed results. Evidence-based guidelines regarding their use are inconclusive. Good outcomes with both arthroscopic and open surgical intervention have been obtained in refractory cases.

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