**Patellar Tendon Ossification: Pathology, Prevention, and Intervention**

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**Introduction**

Although calcific tendinopathy is rather rare, it can be a debilitating condition, hallmarked by the formation and accumulation of bone crystallizations within injured tendon tissue.1 Commonly attributed to an interrupted and consequently abnormal repair process following a repetitive loading injury to the tendon, calcific tendinopathy is most predominant among individuals who are Caucasian and have a metabolic disease such as diabetes or hypothyroidism.1 Yet, it is a preventable condition, rendering its debilitation inexcusable within a modern medical framework and necessitating clinical understanding amongst primary and specialty providers alike, in order to ensure prevention and appropriate early intervention across the lifespan.

While it can occur in any tendon, calcific tendinopathy is most frequently found in the supraspinatus tendon, Achilles tendon, and patellar tendon.1 Henceforth, this paper exclusively focuses on the patella tendon (PT), distinguished from the suprapatellar or quadriceps tendon as the infrapatellar or true patellar tendon (PT), the component of the knee extensor mechanism attaching from the inferior pole of the patella to the tibial tubercle (TT). (Appendix 1) Initial injury leading to calcified tendinopathy in the PT, also referred to as patellar tendon ossification (PTO), can occur secondary to several conditions including Osgood-Schlatter syndrome, Sinding-Larson-Johansson disease, and jumper’s knee, as well as post-operative complications, all of which are examined herein. Each condition’s physiological and mechanical factors uniquely contribute to patellar tendon ossification and inform prevention and intervention.

In general, abusive loads of frequency, magnitude, and/or duration that exceeds the adaptive capacity of the PT can contribute to its injury. Unable to heal under unrelenting injurious loading, a maladaptive repair process ensues. In young myo-osseous disproportionate adolescents, whose developing bone growth surpasses musculotendinous lengthening, high loads may be better attenuated by the PT than the open apophyseal plate with which the tendon interfaces and from which skeletal growth is stimulated through loading.2 In other words, children’s apophysis suffers the brunt of abusive loads, especially during growth spurts when the apophysis to subjected to pronounced tensile force from relatively short and tight muscles, not to mention excessive contractile loads.2

However, the mechanisms contributing to PTO differ between apophysitis, which is most common in children, versus tendinopathy, which is most common in adults. In growing children, continued stresses concentrated at the PT’s interface with the open apophyseal plate chronically perpetuates local inflammation, and the body may attempt to reinforce the unit with boney ossifications. Conversely, whereas tendinopathy may begin with inflammation, unresolved healing and cessation of attempted inflammatory healing responses result in the disorganization and degeneration of tendon tissue, instigating the formation and accumulation of stronger yet far less functional heterotrophic ossifications to fill in and reinforce gaps in tendon tissue of post-pubescent adults.3

Once injured, the PT is greatly disadvantaged in its ability to heal. It is poorly vascularized, impeding the delivery of critically needed oxygen, nourishment, and reparative cells to the site of injury. Additionally, reparative cells are metabolically slow, limiting PT’s regeneration capacity and healing potential.4 Consequently, loading through activities that engage quadriceps and the extensor mechanism across a flexed knee joint to rapidly decelerate in a horizontal direction or accelerate in a vertical direction are particularly stressful and injurious to the patellar tendon and attached bone.3 More specifically, sudden forced flexion of the knee against a strongly contracted quadriceps or sudden violent quadriceps contraction acting across a flexed knee with the foot stabilized can concentrate forces in the area of the PT and it’s boney attachment sites. 3 (Appendix 2) Thus, general prevention strategies involve rest and redirection away from such aggravating activities as sprinting and jumping in sports like basketball, in order to reduce frequency, magnitude, and duration of loads and support the reparative process to promote the healing of tendon tissue instead of its replacement by ossificans.4 However, the uniqueness of each underlying condition contributing to PTO must be understood to best inform individual prevention and early intervention strategies.

**Conditions**

Osgood-Schlatter Disease (OSD)

OSD is the most common overuse injury in adolescents.2 It is characterized by chronic inflammation at the apophyseal plate of the TT, to which the PT attaches.5 See Appendix 3 for imagery. Although it’s etiology remains debated, the most accepted theory is that it is caused by repetitive contraction of the knee extensors.6 As the quadriceps contract and exert tension across the extensor mechanism, the PT pulls at its insertion site on the TT.5 Thus, signs and symptoms include swelling, edema, and pain localized to the TT with resisted knee extension, with underlying TT fragmentation, PT thickening, and infra-patellar bursitis.7 OSD can be categorized into three stages: in stage one the apophyseal plate remains non-displaced; in stage 2 the plate disrupted and loosened but still non-displaced, and at stage 3 the plate is avulsed.2

During times of developmental growth, tensile loads can inflame the apophysis and disrupt the apophyseal end plate, as well as stimulate osteoblasts in constructing boney ossifications in a maladaptive attempt to reinforce the inflamed apophysis. Additionally, local inflammation at and around the apophysis may stimulate heterotrophic bone formation, which may fuse to the TT or accumulate within the PT and other soft tissue within the vicinity of the inflammation.2 After puberty, the abnormally developed PT-TT interface can lead to altered stress concentrations and biomechanics that contribute to patellar tendinitis in later years, due to similar quadricep-dominant running, jumping, and squatting loads. Again, if tendinitis is left untreated under continued abusive loading, tendinosis may develop, and heterotrophic bone may form within the injured tendon as part of the interrupted healing process.2 Thus, a critical component to prevention and early intervention is activity modification, to optimize healing of initial tendinitis and disallow progression to tendinosis or ossification. If the condition goes untreated and does progress to ossification, then more aggressive interventions may be needed to remove boney deposits and reinitiate tendon healing. See Interventions section for details.

Sinding-Larson-Johansson Disease (SLJD)

Far less prevalent than OSD but occurring under similar conditions, SLJD may be unknowingly mistaken for OSD. However, while OSD occurs at the distal PT where it inserts into the TT, SLJD occurs at the proximal PT where it inserts into the inferior pole of the patella. See Appendix 3 for imagery. Patients who develop SLJD instead of OSD may simply have a more reliant TT apophysis than the apophysis of their inferior patellar pole, which is another area in which stress can concentrate from across the extensor mechanism. Hence, while symptoms of SLJD are similar to OSD, pain, swelling, and edema are localized to the inferior pole of the patella. This is the area that heterotrophic ossification can develop within, potentially accumulating within the proximal PT. Also like OSD, activity modification is a critical component of prevention and early intervention, to disallow tendonitis from progressing to tendinosis or calcification.2

Jumper’s Knee (JK)

JK is another chronic and disabling conditions, resulting in 53% of athletes quitting their sports career within 15 years after onset.8 Unlike OSD and SLJD, it affects the PT directly, as opposed to indirectly through an apophyseal interface. More prevalent in adult males and elite athletes, JK can affect both the inferior PT and superior quadriceps tendon, but injury within the inferior PT is most common.9 Similar to OSD and SLJD, this pathology is also caused primarily through PT loading in jumping and landing activities.10,11

Progression of the disease can be categorized into four phases, with phases 1 and 2 responding well to conservative interventions.12 However, during phase 3, microtears evolve into pseudocystic cavities within the tendon’s transitional zone, as it transitions from mineralized fibrocartilage to bone.12 Additionally, tendinous fibrocartilage thickens and necrotic debris within pseudocystic cavities is replaced by ossificans, due to lack of granulation tissue containing reparative cells.12 During the rare occurrence of phase 4, tendon rupture occurs due to increased stress concentrations. This risk increases with the use of oral or injected corticosteroid medications, which catabolically destroy tendon’s collagenous contents.12 Unfortunately, corticosteroids remain a heavily utilized treatment during initial phases of patellar tendinitis such as JE.

Post-Trauma Complications

Forty four percent of patients who have surgery involving their PT end up with PTO.13 Examples of such surgical procedures include intramedullary nailing of the tibia following fracture and anterior cruciate ligament reconstructive surgery utilizing an autologous PT graft.14,15 In rare cases, pediatric patellar sleeve fractures caused by sudden high tensile forces dislodging a fleck of bone from the patella may result in a large sleeve-like cartilaginous formation around the patella.16 If unaddressed, this cartilage is likely to ossify, potentially concomitant with an elongated patella or patella alta.16,17

**Interventions**

If, in the absence of prevention or even despite early intervention, the aforementioned conditions and associated presentations should persist, then efficacy and limitations of multiple interventional approaches must be understood and compared to determine best practice at various points throughout the injury’s course. Each of the following interventions aim to decrease pain and improve function, but they do so with varying efficacy during differing phases of PTO rehabilitation. During initial onset, apophysitis and patellar tendinitis are generally best treated conservatively with rest, ice and electrotherapy for pain control, massage and taping for edema control, anti-inflammatory medication – excluding corticosteroids – heat and stretching for extensibility, and activity modification and redirection.18 In the case of patellar tendinitis, initial intervention may be progressed to include eccentric loading, to reorganize and strengthen tendon tissue.4 For refractory patellar tendinitis that is resistant to conservative treatment and progresses to tendinosis, cross friction massage to remove irritating adhesions or reinitiate the inflammatory process as appropriate. In the case that the condition has progressed to PTO, more invasive methods may be required to remove calcifications, reduce pain, and restore function.

Rest and Activity Modification

Appropriate rest and activity modification is key to decreasing abusive loads upon the PT and its insertions, creating an environment for the tissues to heal through an innate reparative process. However, rest does not equate immobilization, as this leads to the additional weakening through atrophy of collagen. Rather, frequency, magnitude, and duration of PT loading should be modified.18 Particularly, for quadricep dominant individuals, gluteal activation and integration into functional activities from walking to squatting can play a critical role in long-term cessation of aforementioned conditions.19

Anti-Inflammatory Drugs

In the case of tendinitis, anti-inflammatory drugs can be helpful in mitigating secondary damage from acute inflammation. However, it is important to remember that inflammation is the process by which reparative cells are delivered and activated within injured tissue. Thus, in the case of tendinosis, in which the inflammatory healing process has ceased despite degenerative injury, anti-inflammatory drugs are both unnecessary and contraindicated.3 In all cases, corticosteroids are ill-advised, as they decrease strength and increase rupture risk by atrophying collagen within the tendon and surrounding tissues.10,11 Therefore, if anti-inflammatory drugs are utilized, they should be non-steroidal in nature, used as prescribed (i.e. taken with food) to reduce risk of adverse effects, closely monitored, and stopped when no longer needed.20 Alternatively, anti-inflammatory medications may be delivered via iontophoresis or phonophoresis.

Stretching

As previously mentioned, tight quadriceps place additional forces through the extensor mechanism, contributing to patella alta and stresses through the PT and its insertions. While stretching is necessary to improve quadricep extensibility and reduce noncontractile loads on the PT, it is important to conduct stretching properly, as vigorously stretching un-warmed muscles can actually exacerbate PT loads, without even creating sustained extensibility gains. Thus, it is advisable to prepare quadriceps and the patella tendon with moist heat prior to stretching, to increase tissue’s capacity to strain and deform before failure.2,4 For best results in increasing extensibility and decreasing ultimate tensile forces on the PT, a gentle and prolonged stretch of 1-3 minutes may then be applied to the quadriceps,2 guided by multiple principles including creep, stress-relaxation, contract-relax, and reciprocal inhibition. Stretching is especially important in adolescents whose immature skeletons are still outpacing their muscle length during continued growth.2

Eccentric Loading

Following inflammation and proliferation that is either natural or provoked by cross friction massage, eccentric loading currently appears to be the most effective form of initial loading during tendinopathy rehabilitation, with progressive eccentric and then concentric loads influencing collagen reorganization and strengthening along the tendons line of tension.21

Deep Friction Massage

Also known as cross friction massage, this technique was propelled into wide practice by Dr. James Cyrriax decades ago and remains a commonly used conservative intervention in tendinopathies. In tendonitis, the technique can release adhesions surrounding the tendon, decreases irritation and allow the tendon to heal unencumbered by continued microtrauma. By applying deep pressure perpendicular to the tendon’s line of tension via the practitioners overlapping thumbs, this technique can also beneficially irritate the tendon and reinitiate an inflammatory healing response. Variations of applied pressure and application duration tailored to individual patients’ injury area and pain tolerance appear similarly effective, as long as they adhere to Cyriax’s general concept of reinitiating the inflammatory process at the site of injured tendon. Likewise, this perpendicular stimulus along healing tendon may be continued to be used during proliferation and remodeling phases, in which the stimulus helps organize collagen fibers along the tenon’s line of tension.22

Iontophoresis

For patellar tendonitis, iontophoresis can be used to deliver anti-inflammatory dexamethasone, which is a steroid and must therefore again be cautioned against. Additionally, for patellar tendinosis, iontophoresis can be used to deliver acidic acid to ossification sites with the PT. However, this treatment’s effects may require further investigation and validation, especially specific to the PT. Thus far, research does not demonstrate a significant difference between experimental and control groups, attributing any decreases in mineral deposits to natural reabsorption over time.23

Ultrasound

Similar to iontophoresis, evidence that ultrasound significantly increases reabsorption rates of ossificans remains inconclusive. Even the combined intervention of ultrasound and acetic acid iontophoresis in a controlled trial did not demonstrate significant results, but research investigating varying parameters may be warranted.24 Regardless, ultrasound is still often used in an attempt to gain even the smallest advantage prior to stem cell therapy for PTO.

High Powered Laser Therapy (HPLT)

Individual trials suggest that high cost through increasingly available HPLT may influence improvements in the size of PT ossificans, likely through indirectly stimulating reabsorption versus directly breaking up formations. However, comparison of HPLT and extracorporeal shock wave Therapy (ESWT) suggests that ESWT results in greater improvements in pain and joint function, even with fewer applications.24

Extracorporeal Shock Wave Therapy (ESWT)

Better known for the treatment of kidney stones, ESWT uses an oscillating needle or barbotage to break up and aspirate calcifications, and can be used to successfully intervene in PTO. In addition to removing calcified deposits, it is believed to stimulate tissue regeneration through its sonic pulses. Therefore, it may be an applicable intervention for any patellar tendinopathy, from acute tendinitis to PTO. Specifically, three sessions of 1,000 impulses at 4Hz and 0.08mJ/mm2 has been demonstrated as an effective treatment of chronic patellar tendinosis that is resistant to conservative interventions, resulting in functional restoration that are comparable to surgical outcomes. However, it is important to note that ESWT efficacy in treating patellar tendinopathy is best optimized with other conservative treatments and may be contingent on patients receiving instruction to self-regulate activity based on pain, limiting activity that would stress the PT and impair healing.25

Platelet Rich Plasma (PRP) Injection

Under normal conditions, platelets are the first cells that arrive at an injury site, and they play a key role in releasing growth factors that mediate the repair process. Since the PT is poorly vascularized, it may not be well sourced with needed platelets and growth factors. In the event of unremitting loads that surpass the capacity of cellular repair, the inflammatory healing process may cease and lead to tendinosis. This can be combatted by PRP, which is a concentration of platelets and associated growth factors. One injection of PRP applied locally every two weeks over a six week period can reinitiate the natural healing process and positively influence PT tendon repair despite its low healing potential. PRP injection is an effective, low cost, and minimally invasive procedure. It is useful to promote PT healing that is otherwise resistant to conservative treatment, either prior to ossification or after the removal of ossificans with comparable results regardless of severity. Long term pain and function are even further improved at the six-month mark when PRP is combined with the conservative techniques listed above.18

Stem Cell Therapy

In brief, a typical stem cell therapy protocol involves harvesting mononuclear cells from the anterior iliac crest and injecting them into the patellar tendon during an outpatient procedure. Patients are instructed to limit use of their leg for 24 hours and then begin light stretching and aerobics on a stationary bike or in a pool. After a month, patients can perform recreational sports as tolerated, with up to 100% of patients making a full return to sport and stating total satisfaction at their 5 year follow up. However, this intervention has not been well studied among patients over 35 years of age. Additionally, this procedure is costly and dangerous, involving the administration of general anesthesia to harvest stem cells from bone marrow. Furthermore, the optimal timeframe for performing this procedure is still unknown, though some authors suggest that six months after unresponsiveness to nonoperative treatment is optimal to prevent calcificiation secondary to tendinosis. For patients who have already suffered calcification, aforementioned procedures should be used to remove crystals prior to stem cell therapy. Failure to do so may actually risk increased ossification, as there is no evidence that stem cell therapy is capable of replacing ossificans with new tendon tissue.26

Sclerotic Injection

Neovascular sclerosis seems to be ill-advisably offered to patients who experience unremitting pain due to patellar tendinitis, tendinopathy, or ossification. Although this procedure is highly effective in eliminating pain, it is often recommended for rapid return to activity, including competitive sports. However, there is no evidence that this procedure offers any reparative affect beyond pain control. In fact, due to its neuro-destruction, it is long-term results may likely include increased PT degeneration, rupture, and consequent dysfunction if the underlying condition is not addressed in adjunct to the injection. Yet, even for well-intentioned patients and providers seeking to use it as a method of pain control in conjunction with noninvasive or invasive methods, this intervention may still be inadvisable, as it will likely contribute to the continuation of overuse and degeneration.27

Surgery

If symptoms and functional impairment continue beyond 6 months after the start of treatment, surgery is often recommended. However, clinical results after patellar tenotomy are equivocal at best. Overall, there is a lack of reliable outcome studies with appropriate study design, and reported success rates appear to be inversely related to the methodological quality of the studies.25 Additionally, more retrospective studies have recently demonstrated suboptimal functional outcome results. Many patients never return to their preinjury level and intensity of sporting activity. Moreover, surgical treatment is accompanied by an intensive and long postoperative rehabilitation and a considerable period of sick leave.25

Still, there are several types of surgical interventions worth comparing. Most notable, surgical debridement may be used to remove calcifications, thick degenerative tendon tissue, or possible adhesions. This procedure can also be paired with the creation of microfractures in the inferior pole of the patella, to stimulate and channel blood supply from the bone to the tendon, in an effort to promote subsequent healing instead of recalcification. Although this type of debridement can be performed arthroscopically, it has far more risks than ESWT, and results are comparable at best or suboptimal in the event of post-surgical scarring or recalcification.28

If the extent of calcification is too great to merit debridement, such that remaining tendon would be structurally insufficient, PT reconstruction may be called for, with better results for reconstruction due to proximal patellar tendinopathy than inferior patellar tendinopathy.29

Additionally, though apophysitis due to OSD can be effectively treated with early conservative interventions, a pediatric procedure exists to excise excess bone and resection the TT via tubercleplasty. This procedure may be offered for cosmetic purposes in adulthood as well, but it does not cure OSD nor is there any evidence that it improves pain or function.30

A review of 20 papers found an 80% success rate for severe patellar tendinopathies irrespective of the surgical technique used. Although effective noninvasive treatments for PTO exist, surgery does offer the benefit of simultaneously intervening in other knee pathologies, and may therefore be worth considering for patients with multiple knee pathologies, especially if those pathologies contribute to PTO such as boney overgrowths, inflamed bursas, adhesions, or tight retinacula acting upon the patella and misaligning the extensor mechanism.31

**Conclusion**

In all cases, rest and activity modification to create the conditions in which the PT can heal, as well as correction of any underlying contributor of reinjury (i.e. irritating adhesions, tight soft tissues, tensile forces exacerbated by malalignment) must be addressed either to prevent initial patellar tendinopathy or its reoccurrence. While a comprehensive combination of the above conservative techniques is optimal for treating tendinitis and preventing patellar tendinosis, patellar tendinosis and PTO are best intervened with specific treatment which stand in contrast to less effective techniques. For patellar tendinosis, deep cross friction massage is preferable to reinitiate the inflammatory healing process, to be followed by the progression of conservative techniques for tendinitis. PRP also appears to be highly effective, though expensive and slightly invasive, intervention for tendinitis or tendinosis that is resistant to conservative methods.

Variability of conclusive efficacy is even more pronounced in PTO interventions. By far, ECSW appears to be the superior method to remove calcifications, based on efficacy, cost, and relative risk. Following ECSW, PRP or stem cell therapy currently demonstrate best outcomes in precise removal of ossificans and complete replacement with real tendon tissue, which is particularly critical to the rehabilitation of severe PTO. Though stem cell therapy approximates the financial costs and health risks of more invasive open or even arthroscopic surgery, it appears to provide the best long-term results.

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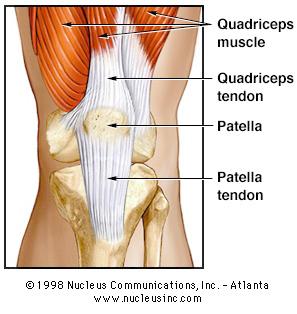
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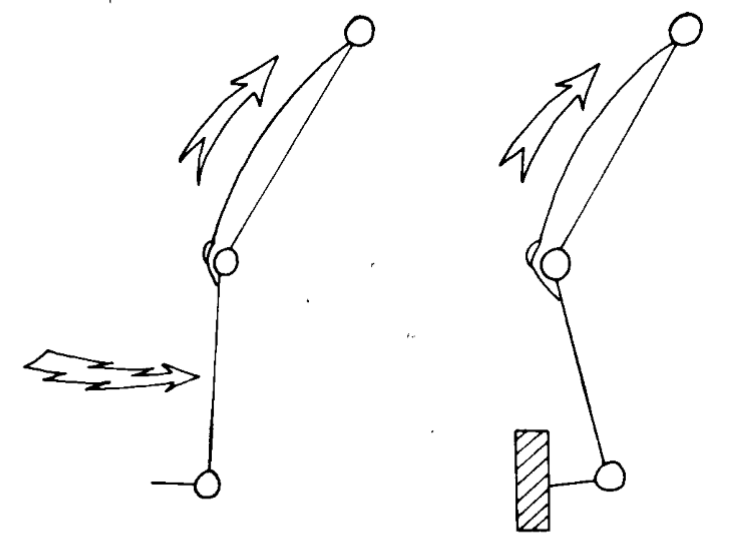
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**Appendices**

Appendix 1: Knee Extensor Mechanism31

[](https://stemcelldoc.files.wordpress.com/2008/12/patella-tendon.jpg)

Appendix 2: Image on the left depicts quadricep forces and PT loading across a flexed knee during deceleration. Image on the left depicts quadricep forces and PT loading across a flexed knee during jumping.3



Appendix 3: Radiograph on the left depicts a patient with OSD (black arrow). The radiograph on the right depicts a patient with SLJD (white arrow).2

