Management of Common Tendinopathies in Basketball

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40.1 Introduction

Tendinopathies are a common cause of disability in the basketball player and account for a substantial proportion of overuse injuries in the sport. The term "tendinopathy" is defined as a clinical syndrome characterized by a combination of activity-related pain, diffuse or localized swelling, and impaired performance [1–3]. Often a sequalae of overuse phenomenon, tendinopathy may less commonly occur as a consequence of medical condition. Midportion and insertional tendinopathy (enthesopathy) should be distinguished as unique clinical entities.

Tendinosis and peritendinitis are two common phenomena that are integral to understanding tendinopathy. Tendinosis entails histopathological disorganization and collagen fiber separation which leads to haphazard proliferation of tenocytes, increase in mucoid noncollagenous matrix, and nerve and vessel ingrowth without evidence of intratendinous inflammation [3, 4].

Peritendinitis is an acute or chronic inflammation of the paratenon which can be induced by repetitive loading activities and is characterized by local inflammatory infiltration. The insertion site to bone and surrounding bursal structures are commonly susceptible to inflammation as a consequence of their greater density of blood vessels and nerves. The tissue of the tendon proper is primary aneural and avascular and does not primary exhibit classical inflammatory responses [1, 3, 4].

Tendinopathic tendons often have an increased rate of matrix remodeling, leading to a mechanically less stable tendon that is likely more susceptible to damage [4]. Histologic studies of surgical tendon specimens consistently demonstrate absent or minimal inflammation, but there is a loss of tightly bundled collagen architecture, increase in proteoglycan content, and neovascularization [5]. The inflammatory cascade is thought to play a major role in the initiation of the disease process [6].

The tendons most vulnerable to overuse in the jumping athlete entail the Achilles and patellar tendons, which will be the focus of this chapter.

40.2 Epidemiology

Basketball is a very dynamic sport that requires a combination of agility, speed, technical skill, and strength. As a consequence of many repetitive maneuvers such as jumping and pivoting, the incidence of tendinopathy is quite prevalent. Due to increased participation in recreational sports, the total exposure of hours to sport practice and competition, a known risk factor for tendinopathy, has

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been on the rise. In addition to basketball (12%), patellar tendinopathy is common in volleyball (14%), track and field (7%), and soccer (2.5%) [7].

Although physical activity is the primary source of tendinopathy, it is important to consider medical and pharmacologic causes of tendinopathy. Perturbation of glucose metabolism and atherosclerosis has been identified as underlying factors in tendinopathy [1, 8]. Obesity, hypertension, diabetes mellitus, hypercholesterolemia, and metabolic disease contribute to the incidence of tendinopathy. Inflammatory arthropathies, such as rheumatoid arthritis, contribute to connective tissue healing malfunction and homeostasis [9].

Medication therapy with statins [10] and fluoroquinolone antibiotics [11] can increase the risk for tendinopathy and should be avoided. The use of immunosuppressive corticosteroids, particularly intratendinous injection, can have detrimental effects on tendon metabolism and should be used with caution [1].

40.3 Pathophysiology (Fact Box 1)

The mechanical loading of tendon tissue leads to the upregulation of collagen gene expression and increased synthesis of collagen proteins. This anabolic process has been observed to peak around 24 h after exercise and remains elevated for up to 80 h [12, 13]. During the first 24–36 h after exercise, there is a net loss of collagen due to degradation of collagen proteins [3]. The interplay between anabolic and catabolic processes of collagen merits a restitution time interval in between exercise to mitigate tendon breakdown.

Fact Box 1 Pathophysiology of Tendinopathy

- Mechanical loading of tendons facilitates protein synthesis and degradation of collagen at the cellular level.
- Adequate tendon unloading (~24 h) after exercise is helpful in preventing net loss of collagen that leaves the tendon vulnerable to injury.
- Tendinopathy is associated with neovascularization and ingrowth of nerves that serve as pain generators.
- Chronic inflammation of a tendon can lead to increased pain signaling and hyperproliferation and fibrosis, which may ultimately become an irreversible process.

Repetitive mechanical loading leads to microruptures of collagen fibrils and the production of inflammatory molecules by tenocytes, this is evidenced by increased levels of proinflammatory cytokines [14]. Findings of inflammatory infiltrate consisting of macrophages, mast cells, and lymphocytes suggest a role for the intrinsic immune system in mediating early tendinopathy. Tenocytes and fibroblasts can transform into myofibroblasts under repetitive stress and a proinflammatory environment. Myofibroblasts facilitate tendon healing and are triggered to undergo apoptosis after the mechanical stresses abate. Although, under constant stress that does not permit this apoptosis or tissue hypoxia, myofibroblasts continue to propagate resulting in fibrosis, a feature of chronic tendinopathy [15].

Another feature of tendinopathy is microvascular angiogenesis into the tendon; this is consequence of hypoxia-induced upregulation of vascular endothelial growth factor (VEGF) [16]. This neovascularization has been ascribed as a pain generator as sensory nerve fibers can grow adjacent to the blood vessels and lead to the release of nociceptive substances, including substance P [17]. Sensory nerve ingrowth is observed in repetitive loading as well as response to injury; in non-pathologic tendon, the repair process involves autonomic nerve ingrowth and ultimate decreased nociception with subsequent nerve retraction, whereas in tendinopathy, the ingrowth nerves do not retract leading to increased pain signaling and hyperproliferative changes. Substance P has a role in potentiating tendinopathy with proinflammatory and trophic actions leading to vasodilation, plasma extravasation, and the stimulation of TGF-β in fibroblasts [1].

40.4 Patellar Tendinopathy

40.4.1 Etiology

Patellar tendinopathy, or "jumper's knee," is highly prevalent in sports with a high demand for speed and power, such as basketball, volleyball, and soccer. The hallmark of patellar tendinopathy is progressive patellar tendon pain that leads to recurrent or long-standing impairment of athletic performance [17]. Overall prevalence of patellar tendinopathy has been reported across multiple sports by Lian et al. [18] as 14%. Estimated prevalence of current or prior patellar tendinopathy was 55% in profession basketball players with a mean duration of symptoms of 32 ± 25 months, suggesting that most athletes participate in their sport with active symptoms. The condition affects men approximately twice as frequently as women. Basketball often leads to maximal muscle forces generated eccentrically that are 1.5–2.0 times higher than the maximal isometric forces. Additionally, ground reactive forces can be 10 times body weight in a long jump takeoff [19, 18], explicating the connection between the loading pattern of the knee extensors and jumper's knee. Cook et al. [20] evaluated patellar tendons of a cohort of elite junior basketball players using ultrasonography and demonstrated that 29% of the tendons had hypoechoic regions with intra and peritendinous changes of collagen disorganization although not all findings correlated with clinical symptomology.

Quadriceps tendinopathy, a less common entity, is important to consider as part of the extensor mechanism spectrum. The prevalence is estimated to range from 0.2% to 2% in athletic patient populations. This entails approximately one in four patients with extensor mechanism pain who report pain localized to the superior pole of the patella with symptoms more pronounced in deep flexion [18, 21]. Although the patellar and quadriceps tendon work in concert as part of the extensor mechanism of the knee, they differ anatomically as the quadriceps tendon connects muscle to bone while the patellar tendon connects bone to bone. The quadriceps tendon is comprised of a coalescence of four muscle tendons that may variably contribute to the ultimate insertion onto the superior pole of the patella. Notably, this results in a transition of stiffness that is variable between the quadriceps and patellar tendons [22, 23]. The focus of this chapter is on the more common patellar tendinopathy in the jumping athlete although it is important to consider quadriceps tendon involvement and acknowledge that a rehabilitation protocol taking into account similar principles but localization to the quadriceps tendon is merited [21].

Ferretti et al. have demonstrated a linear relationship between training volume and the prevalence of jumper's knee and with the firmness of the floor type for training [19]. Additional extrinsic contributors to patellar tendinopathy include inappropriate training equipment, excessive loading, or repetitive high-intensity loading [24]. Patellar tendinopathy most frequently affects the inferior pole of the patella in the deeper portion of the tendon; this zone is subjected to the highest tensile forces during impact activity [25]. Patellar tendinopathy may affect the midportion zone or the insertion at the tibial tuberosity and often irritation of the infrapatellar bursa at the distal insertion of the patellar tendon coexists. Additionally, inflammation and pathology of the Hoffa fat pad may contribute to patient symptoms of tendinopathy and merit consideration in the therapeutic protocol [26, 27].

Anthropometric factors such as high body mass index (BMI), large abdominal circumference, limb-length discrepancy, and flatfoot arch are independent risk factors for the development of patellar tendinopathy [28, 29]. Weakness of the quadriceps muscles and low flexibility of the quadriceps and hamstrings are associated with patellar tendinopathy. Intrinsic factors including patellar malalignment, patella alta, abnormal patellar laxity, muscular tightness, and imbalance have been proposed as risk factors although it is their complex interplay with the aforementioned extrinsic factors that result in tendinopathy [30]. The classification systems of Jumper's knee are detailed in Table 40.1.

Table 40.1 The different classifications for "Jumper's knee" [19, 92–94]

Stage/ grade	Blazina classification	Ferretti classification	Roels classification
0		No pain	
I	Pain only following activity without functional impairment	Pain only following intense sports activity with no functional impairment	Pain at the infrapatellar or suprapatellar region following training or event
II	Pain during and following activity with satisfactory performance levels	Moderate pain during sports activity with no sports performance restriction	Pain at the beginning of activity, disappearing after warm-up and reappearing after activity completion
III	More prolonged pain during and following activity with progressively increasing difficulty performing at a satisfactory level	Pain with slight sports performance restriction	Pain during and after activity. The patient is unable to participate in sports
IV		Pain with severe sports performance restriction	Complete rupture of the tendon
V		Pain during daily activity and inability to participate in sport at any level	

40.4.2 Diagnosis

Patellar tendinopathy presents with activity-related anterior knee pain typically localized to the distal pole of the patella or proximal patellar tendon. Pain is insidious and can be precipitated by an increase in sports activity. The pain may initially commence with activity but can progress to being present even at rest. Physical examination yields tenderness to palpation at the distal pole of the patella with the knee in terminal extension; with pain reduced with knee flexion. Load-related pain that increased demand on the knee extensors, such as the single-leg decline squat maneuver to approximately 30° of flexion reproduces the symptoms of patellar tendinopathy [31]. Weakness of the quadriceps and a lack of hamstring flexibility may be observed as well. Pain may improve with repeated loading (the "warm-up" phenomenon), but there is often increased pain the day after energy-storage activities of the patellar tendon. This dose-dependent pain is a key feature, demonstrating that the patient's pain increases as the magnitude and rate of load application to the tendon increases [25, 32, 33]. Comprehensive evaluation necessitates a thorough examination of the entire lower extremity is necessary to identify relevant deficits at the hip, knee, or ankle/foot region.

Patellar tendinopathy is a primary clinical diagnosis that does not require confirmatory imaging although imaging can be helpful for inclusion/exclusion of potential alternative diagnoses in a convoluted clinical picture. In addition to the inferior pole of the patella, tendinopathy of the extension mechanism of the knee can involve the quadriceps tendon or the distal insertion of the patellar tendon at the tibial tuberosity [25]. Preferred imaging modalities are ultrasonography and MRI. Ultrasonography can locate intratendinous lesions evidenced by hypoechogenicity that may be adjacent to the inferior pole of the patella. Thickening of the tendon, intratendinous calcifications, and erosions in the inferior pole of the patella may be observed. Ultrasonography has a sensitivity of 58% and specificity of 94% for diagnosing patellar tendinopathy [31]. MRI typically demonstrates hyperintensity of the proximal patella tendon with observed tendon thickening as with ultrasonography. MRI provided the added benefit of assessing intra-articular pathology over ultrasonography. The sensitivity and specificity are 78% and 86% for MRI to diagnose patellar tendinopathy, respectively. In the high-level basketball player, MRI should be the first-line imaging option due to the ability to rule out additional pathologies. It is important to note that each imaging modality guides the clinical based on a diag-

nosis of patellar tendinopathy that is diagnosed based on history and physical examination. The main differential diagnosis to be distinguished is patellofemoral pain syndrome and fat-pad syndrome which can often be done on the basis of clinical examination.

40.4.3 Management

40.4.3.1 Nonoperative Treatment (Fact Box 2)

Eccentric Exercises

The mainstay of management of patellar tendinopathy is nonoperative management and the support of high-level evidence [34]. Eccentric exercises lead the musculoskeletal unit to adapt to the stresses by remodeling the collagen fibers in the patellar tendon. This is likely a consequence of the tendon stretching more during eccentric loading as opposed to concentric loading, resulting in more mechanotransduction of collagen fibers leading to greater number of blood vessels and a larger quantity of collagen ultimately resulting in improved tendon healing [35–37].

Universal positive effects are noted without adverse effects [38]. There are multiple types of eccentric loading exercise programs, and in a systematic review Visnes and Bahr [22] concluded that a particular modality is not isolated. A treatment program should include a decline board and should be performed with a mild level of discomfort for the athlete. Assessing pain irritability is a fundamental part of configuring a training regimen with regard to determining the duration of symptoms aggravation following energy-storage activities. It is suggested that up to 24 h of pain provocation after a training session is acceptable during rehabilitation [25, 32]. A minimum of 20 training sessions, performed at a 15-repetition maximum [39], appears to be necessary to ensure effective therapy with a program duration of 6 weeks to 1 year depending on the severity to reduced symptoms of tendinopathy [35, 40].

It is important to recognize that eccentric exercise for the treatment of patellar tendinopathy may be too aggressive for patients that are highly symptomatic, particularly during the basketball season. As a consequence, rehabilitation should progress in a stepwise fashion. The first phase of this should entail load modification to reduce pain and avoid aggravation of symptoms. Following this, we recommend a gradual progression of rehabilitation from isometric loading, to isotonic loading, to ultimate eccentric loading with a culmination of return to sport-specific training activities [25]. It is important to acknowledge that progression of rehabilitation, particularly with the demands of returning to elite-level competition, may require 6 months or longer.

Fact Box 2 Nonoperative Management Modalities for Tendinopathy

- Eccentric exercises lead to tendon stretching and mechanotransduction of collagen fibers leading to increased vascular inflow and ultimately improved tendon healing.
- Extracorporeal shock wave therapy (ECWT) improves tendon healing by hyperstimulation analgesia to reduce pain signal transmission and the production of growth factors via mechanical stimulation.
- Ultrasound-guided galvanic electrolysis technique (USGET) may treat tendinopathy by electoral destruction of degenerative tissue and facilitating a controlled, localized inflammatory healing response.
- Platelet-rich plasma (PRP) injections have shown clinical improvement in tendinopathy due to improved cellular remodeling. Multiple injections offer an improvement over single injection.
- Corticosteroid injections should be used with caution for the management of tendinopathy.

Extracorporeal Shock Wave Therapy

Extracorporeal shoulder wave therapy (ESWT) is gaining vogue as a safe and promising treatment for patellar tendinopathy. The basis of utility of ESWT is based on the theory that pain relief is achieved by hyperstimulation analgesia, and overstimulation of the painful area leads to a diminished transmission of pain signals to the brain stem. Additionally, that the mechanical load developed by ESWT stimulates tissue regeneration and destroys calcifications via a mechanical disintegration [41, 42]. Mechanical stimulation of the tendon increases the expression of growth factors such as insulin-like growth factor 1 (IGF-1), transforming growth factor β1 (TGF-β), and interleukin [43]. A 2017 randomized controlled trial comparing ESWT to conservative treatment (physiotherapy, exercise program, non-steroidal anti-inflammatory drugs (NSAIDs), and knee strap) provided some valuable insight to patellar tendinopathy management. Patients did not require local or regional anesthesia and were treated with 1500 impulses of shockwave at 14 kV (equivalent to 0.18 mK/mm^2 energy flux density) at the point of maximal tenderness. ESWT demonstrated superior pain relief, Victorian Institute of Sport Assessment (VISA) scores, and sonographic vascularity at 2–3 year follow-up [44]. Additionally, recurrence of patellar tendinopathy symptoms occurred in 13% of the ESWT group and 50% of the conservative group (p = 0.014). Ultrasonography showed a significant increase in the vascularity of the patellar tendon and a trend of reduction in the patellar tendon thickness with ESWT without any risk of local to systemic complications. The particulars of ESWT use, including energy level, number of treatments, and impulses, are varied among the literature.

Ultrasound-Guided Galvanic Electrolysis Technique

Ultrasound-guided galvanic electrolysis technique (USGET) is a novel therapeutic option that has recently been evaluated in a randomized controlled trial by Abat et al. [45]. They have demonstrated the efficacy and superiority of USGET compared to conventional electrophysiotherapy when performed with eccentric exercise for patellar tendinopathy management. USGET is a minimally invasive technique that entails treatment at 2-week intervals applying a galvanic current to the area of pathology with a 0.25 × 25 mm stainless steel acupuncture needle. This technique involves puncturing the superficial paratenon, deep paratenon, and intratendinous areas at the inferior pole of the patella with electrical current of 2 milliamps. The authors suggest that this technique is capable of minimally invasively destroying degenerative tissue and facilitating an appropriate localized inflammatory response to trigger biological collagen repair [46]. This technique merits strong consideration as a therapeutic modality for patellar tendinopathy that obviates the need for operative intervention.

Injection Therapy

Corticosteroid injections are not recommended for the treatment of patellar tendinopathy. Multiple randomized controlled trials (RCTs) have shown limited long-term relief and an increase in potential patellar tendon rupture with the use of corticosteroid (triamcinolone and methylprednisolone) injections [47–49]. Corticosteroid injections may normalized lesions in patellar tendons on ultrasonography and may have pronounced clinical effects over a short duration but do not demonstrate sustained benefits beyond 6 months.

Platelet-rich plasma (PRP) is a promising therapy for enhancing tissue repair due to delivery of platelet-derived growth fractures and bioactive molecules in hyperphysiologic doses [50]. A 2019 systematic review and meta-analysis reviewed 15 articles describing the use of PRP with good overall results in terms of clinical improvement, return to sports, and pain relief at short-term and long-term follow-up [51]. In vitro and in vivo studies of PRP demonstrate benefits that include improved cellular remodeling and decreased healing team [50, 52–54]. The details of optimal preparation and administration of PRP remain unclear in the literature. Although, there is now evidence to support multiple injections of PRP to yield a significant benefit at long-term

follow-up [55, 56]. Single infiltration of PRP might provide short-term relief but not optimal long-term results. Notably, one study comparing single injection to multiple injections of PRP found no benefit for successive injections [57]. As a consequence, although further investigation is pending—in the case of advanced patellar tendinitis we recommend 2–3 consecutive infiltrations of leukocyte-rich PRP with 5 mL delivered at an average spacing of 10–15 days. It is advised to combine PRP treatment with rehabilitation due to the synergistic effect in recovery.

Cell-Based Therapies

Stem cell-based therapies theoretically could improve the speed and quality of the healing process with the aid of autocrine and paracrine factors to enhance remodeling. This research remains in the preliminary phases including case studies and animal models. This has been evaluated with mononuclear bone marrow cells at 5-year follow-up [58] and skin-derived tenocyte-like cells [59] with initial promising results. Ultimately, further information is needed to indicate their role in patient management.

Hyaluronic Acid

High molecular weight hyaluronic acid (HA) has been hypothesized to have an anti-inflammatory effect as well as potential tendon healing impact at the bone-tendon interface [60]. A mean of two injections of hyaluronic acid has been shown to improve recovery at a mean follow-up of 25.7 months, reporting 54% excellent results and 40% good results. High-level evidence is still lacking to determine the efficacy of HA.

Additional Treatments

A variety of agents have been and are currently being investigated to aid in the management of patellar tendinopathy. Sclerosing agents inhibit blood vessel formation and counteract accompanying vasa nervorum and can have an impact on the known neovascularization that occurs in the pathophysiology of patellar tendinitis. Level IV evidence shows that ultrasound-guided injection of 5 mg/mL polidocanol to the paratenon can improve pain during activity [61]. There are multiple studies suggesting improvement over placebo [62–64] although the longevity of impact and overall usefulness remain unclear.

Injection of matrix metalloproteinase inhibitor aprotinin has been evaluated in a retrospective case series of 97 patients [65] which demonstrated differential degrees of improvement at approximately 1 year of follow-up. Further clinical data is desired in this treatment regimen.

Glyceryl trinitrate delivers nitric oxide locally and has been shown to play a role in fibroblast proliferation, collagen synthesis, and macrophage angiogenesis. Glyceryl trinitrate can be delivered via a topical patch and has shown promising early data with regard to positive effects on tendinopathy [66, 67].

40.4.3.2 Operative Treatment (Fact Box 3)

Nonsurgical treatment of tendinopathy is successful in approximately 90% of patients. Those who are refractory may ultimately respond to surgical intervention [68]. The objective of surgical intervention is tenotomy of the patellar tendon, excision of pathologic tissue, and induction of the repair process via stimulating the inferior pole of the patella. This can be done with open surgery or arthroscopic intervention.

Open approach entails a midline longitudinal incision from the inferior pole of the patella to the tibial tubercle; the paratenon is incised to expose the patellar tendon. Through a longitudinal incision in the tendon, the posterior degenerative tissue is debrided and the distal 5 mm of the patella are resected to ensure a bleeding surface for healing (Fig. 40.1). The tendon and paratenon are closed with resorbable sutures; a suture anchor may be placed into the patella if needed. Alternatively, a technique using a Beath pin to secure the tendon to the patella [69].

Arthroscopic intervention begins with diagnostic knee arthroscopy to evaluate the patella for chondral lesions. The inferior pole of the patella is identified, and the adjacent synovial tissue is resected, exposing the degenerative tissue in the

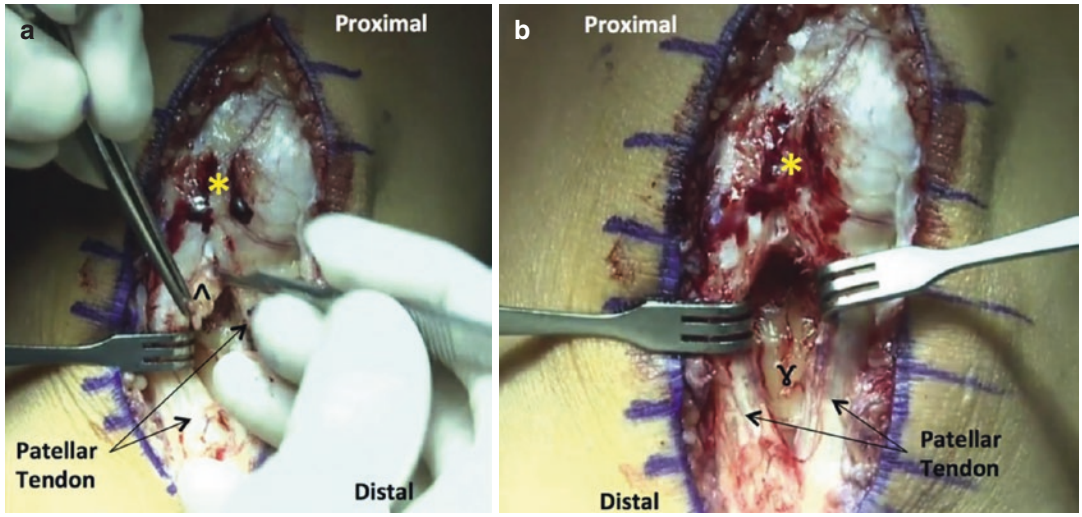

Fig. 40.1 Midline approach to patellar tendon demonstrated with posterior degenerative portion exposed and identified by abnormal color and texture of the tissue. (**a**) The tissue is excised and carefully debrided with a #15 blade, asterisk denotes the patella. (**b**) Post excision the infrapatellar fat pad is visualized by the gamma sign *(Borrowed from Kruckeberg* et al. *Arthroscopy Techniques 2017)*

posterior zone of the proximal patellar tendon, which can be debrided until healthy tissue is identified. The distal pole of the patella is resected of 5 mm using a burr.

Each of these techniques can be augmented with leukocyte-rich PRP injection at the conclusion of the procedure. Postoperative rehabilitation should not restrict weight-bearing or range of motion. After wound healing at approximately 10 days postoperatively, patients should commence initiation of eccentric squats on an inclined board and strengthening. Typical return to sport is 3 months postoperatively when patients can demonstrate functional recovery without pain during strengthening and exercise [28].

40.5 Achilles Tendinopathy

40.5.1 Etiology

The Achilles tendon (AT) structure plays a crucial part in basketball-specific movements, allowing explosive movements by storing and releasing elastic energy. Risk factors associated with Achilles injuries include advancing age, changes in player performance characteristics, altered foot and ankle biomechanics, gastrocnemius-soleus dysfunction, and anatomic variations [63, 64, 70]. Achilles tendinopathy can be attributed to excessive loading of the tendon during vigorous physical training that is exacerbated by an imbalance between muscle power and tendon elasticity. In particular the strength of the ankle plantar flexors and the amount of dorsiflexion excursion predict an Achilles tendon overuse injury [71]. Lateral ankle instability leads to excessive motion of the hindfoot with resultant excessive compensatory pronation causing a "whipping action" on the AT, predisposing tendinopathy. Forefoot varus is additionally implicated in patients with Achilles tendinopathy. It has been hypothesized that the plantaris tendon may contribute to midportion tendinopathy or chronic paratendinopathy due to adhesions with the paratenon or AT [27]. Extrinsic factors that predispose athletes include changes in training pattern, poor technique, previous injuries, footwear, and environmental factors such as hard, slippery, or slanting training surfaces [72]. Players with Achilles tendinopathy have a better change to return if they are younger in age and early in their professional career [73, 74].

40.5.2 Diagnosis

Achilles tendinopathy can be categorized into two distinct categories as insertional and non-insertional. Non-insertional is localized to the midportion which typically involves an area of 2–7 cm from the distal insertion; it may be localized to the main body of the tendon or to the paratenon. The patient typically presents with activity-limiting pain and chronic swelling of the AT. The diagnosis is mainly based on history and clinical examination. Pain is often a late symptom, with common symptoms of morning stiffness or stiffness after a period of inactivity and a gradual onset of pain during activity. Athletes often experience pain at the beginning and end of training sessions, with a period of diminished discomfort during activity. On physical examination, the both legs should be evaluated simultaneously and is best with the patient standing and prone. Tendinopathy of the main body of the AT involves tenderness, likely thickening/nodularity, and swelling that is decreased or relieved when the tendon is put under tension. Paratendonitis of the tendon is similar in the involvement of the central 1/3 although the swelling and tenderness remain fixed in relation to the malleoli from full dorsi- to plantarflexion. Insertional tendinopathy or Achilles enthesopathy is characterized by pain, swelling, and tenderness of the posterior aspect of the calcaneus that is aggravated by active to passive motion [72, 75].

From an imaging standpoint, X-ray radiography may demonstrate deviation of the soft tissue contours. Insertional Achilles tendinopathy at the calcaneus can result in calcifications and enthesophyte development. Ultrasonography (US) correlates the histopathologic findings very well and offers advantages over the other imaging modalities. Ultrasonography can quantify tendon structure and may demonstrate an enlarged tendon with or without fibrillation or hypoechoid areas. Additionally, power Doppler US can detect neovascularization and can serve as a reliable to tool to diagnose and follow Achilles tendinopathy. In the setting that US remains inconclusive, magnetic resonance imaging (MRI) of the tendon can be used and will further characterize the internal morphology of the tendon and surrounding tissues. MRI also clearly delineates paratendinopathy and tendinopathy of the main body of the tendon (Fig. 40.2). If considering surgical intervention, MRI may be valuable in pre-operative planning although the high sensitivity of MRI should be interpreted with caution and correlated directly to patient symptoms [5].

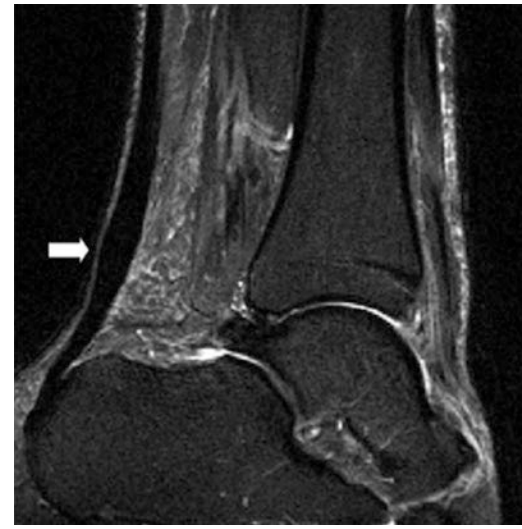

Fig. 40.2 MRI demonstrating tendinopathy of the main body of the Achilles tendon, localized thickening without involvement of the paratenon *(Borrowed from Maffulli* et al *2019. Foot and Ankle Surgery)*

40.5.2.1 Nonoperative Treatment (Fact Box 2)

The management of Achilles tendinopathy lacks evidence-based guidance and long-term morbidity is unpredictable. In a prospective observational study of patients with AT, 29% of patients required surgical intervention during an 8-year follow-up period [76]. Nonoperative management should be implemented for a minimum of 3–6 months prior to surgical consideration [77].

There is a distinct overlap in the nonoperative management of patellar and Achilles tendinopathy. This segment of the chapter is abbreviated as the same principles are in play as previously described for the management of patellar tendinopathy. Anti-inflammatory medications demonstrate a modest improvement in symptoms in the

short term as well as facilitating eccentric strengthening exercises of the gastrocnemius and soleus. There is the caveat of the analgesic effect potentially allowing patient to ignore early symptoms and imposing further damage on the affected tendon. Stretching of the gastrocnemius/soleus complex is essential in these patients as many jumping athletes are predisposed to gastrocnemius contracture, as this can often exacerbate Achilles tendinopathy [77].

Rest and eccentric exercises are the mainstay of therapy, and multiple studies have demonstrated 60%–80% of patients safely continuing with their preferred activity [78–80]. The combination of eccentric exercises with extracorporeal shockwave therapy has been shown to produce a higher success rate as compared to each modality in isolation [81].

Injections of PRP are preferred in succession as opposed to single injections for patellar tendinopathy and other tendinopathies (rotator cuff, lateral epicondylitis) [82]; this principle may be applied to the Achilles with the caveat that recent literature suggests equivocal efficacy in the management of chronic Achilles tendinopathy with PRP as compared to saline injection [83]. Further research with a large, multicenter, blinded, randomized controlled trial with a robust study population and long-term follow-up is needed to confirm these findings [84]. Corticosteroid injections are not recommended as literature has shown limited benefit with potential risk of tendon rupture [47, 85].

High-volume ultrasound-guided injections aim to produce local mechanical effects, aiming to stretch, break, or occlude neo-vessels. The injections can be localized to the area pathology, whether midportion or insertional tendinopathy. Short- and long-term pain relief along with improved daily function (VISA scores) has been observed in 70% of patients with resistant Achilles tendinopathy after injection with 10 mL of 0.5% bupivicaine, 25 mg hydrocortisone acetate, and up to 40 mL of normal saline [86]. Similar improvement in symptomology via pain and stiff values as well as VISA scores has been noted in comparison to ESWT [87].

Unique to Achilles tendinopathy, orthotics are widely used with heel pads alleviating insertional Achilles tendinopathy, but clinical evidence is limited [88]. An AirHeel brace, with applied intermittent compression to minimize swelling and promote circulation, may serve as an alternative to eccentric exercises, particularly in patients with activity-limiting pain. No clinical differences have been found in the comparison of the AirHeel brace and eccentric training program for chronic Achilles tendinopathy [89].

40.5.2.2 Operative Treatment (Fact Box 3)

Approximately one-third of patients with Achilles tendinopathy fail to improve after 3–6 months of conservative management and as a consequence surgical treatment may be indicated [75]. Surgical intervention entails a spectrum from simple percutaneous tenotomy, to minimally invasive stripping of the tendon, to open procedures that may include tendon augmentation. In systematic review of surgical intervention for chronic Achilles tendinopathy, successful results in over

Fact Box 3 Operative Management of Tendinopathy

- Open or arthroscopic debridement of the inferior pole of the patella and pathologic tissue at the postero-superior patellar tendon is the mainstay of surgical management of patellar tendinopathy.
- Gastrocnemius contracture is important to assess and can be routinely treated with gastrocnemius recession.
- Achilles paratendinopathy can be managed with minimally invasive stripping, tendoscopic debridement, or open debridement of the paratenon.
- Open debridement and tubularization of the Achilles tendon or debridement of the insertion on the calcaneus allow for the removal of pathologic tissue, an initiation of a healing response.

70% of cases have been reported with a particular predilection for male patients and those who classify as athletes.

Regardless of the anatomic location of the Achilles tendinopathy, patients must be first assessed for gastrocnemius contracture via the Silfverskiold physical examination maneuver. This entails assessment of ankle dorsiflexion with and without knee flexion, determining the contribution of the triceps surae to limitation of ankle dorsiflexion. Gastrocnemius recession may be an adequate surgical option to address symptoms of refractory Achilles tendinopathy. Our preferred method of gastrocnemius recession involves a medial incision at the muscle-tendon intersection of the gastrocnemius with division of the gastrocnemius fascia while taking care to avoid injury to the sural nerve. Increased ankle dorsiflexion without increased force on the tendon may alleviate further tendinopathy.

Isolated paratendinopathy can be managed with debridement of the paratenon; this may be done via a minimally invasive stripping technique (Fig. 40.3), endoscopically, or with an open incision. Minimally invasive stripping involves prone positioning with a tourniquet inflated to 250 mmHg. Two longitudinal 0.5 cm skin incisions are made at the proximal origin of the Achilles tendon, just medial and lateral to the origin, and two additional 0.5 cm incisions are made 1 cm beyond at the distal end of the AT. Using small hemostat, the AT is freed of adhesions proximally and a nonabsorbable suture is inserted, passing through the proximal incisions. This is then retrieved distally and used to carefully strip the posterior extent of the tendon from

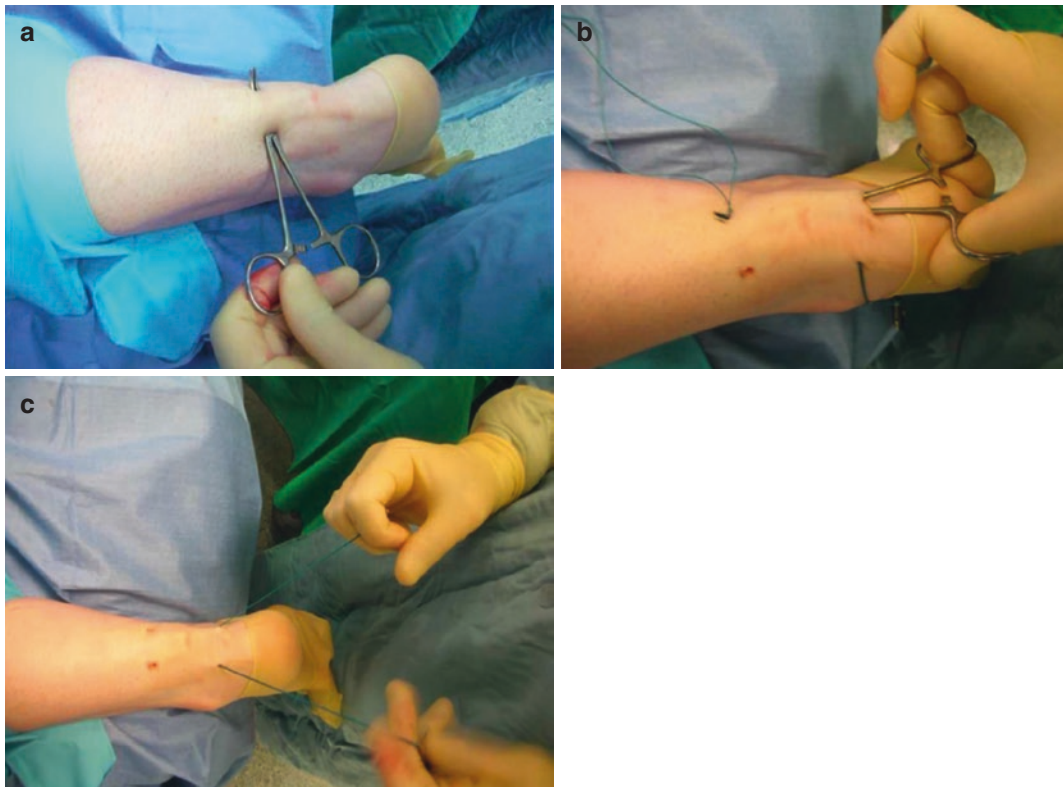

Fig. 40.3 Minimally invasive stripping of Achilles tendon. (**a**) Hemostat inserted to free AT of peritendinous adhesions. (**b**) Nonabsorbable suture inserted proximally and retrieved distally. (**c**) Using gentle sawing manipulation, the suture is translated from proximal to distal, freeing the tendon periphery [75] *(Borrowed from Maffulli* et al *2019. Foot and Ankle Surgery)*

proximal to distal [75, 77]. Endoscopic technique involves the use of a 30° arthroscopic camera and a mechanical shaver to break up paratenon adhesions.

Tendinopathy of the midportion of the Achilles tendon can be managed by less invasive percutaneous longitudinal tenotomy (Fig. 40.4) If percutaneous techniques are not successful, a more

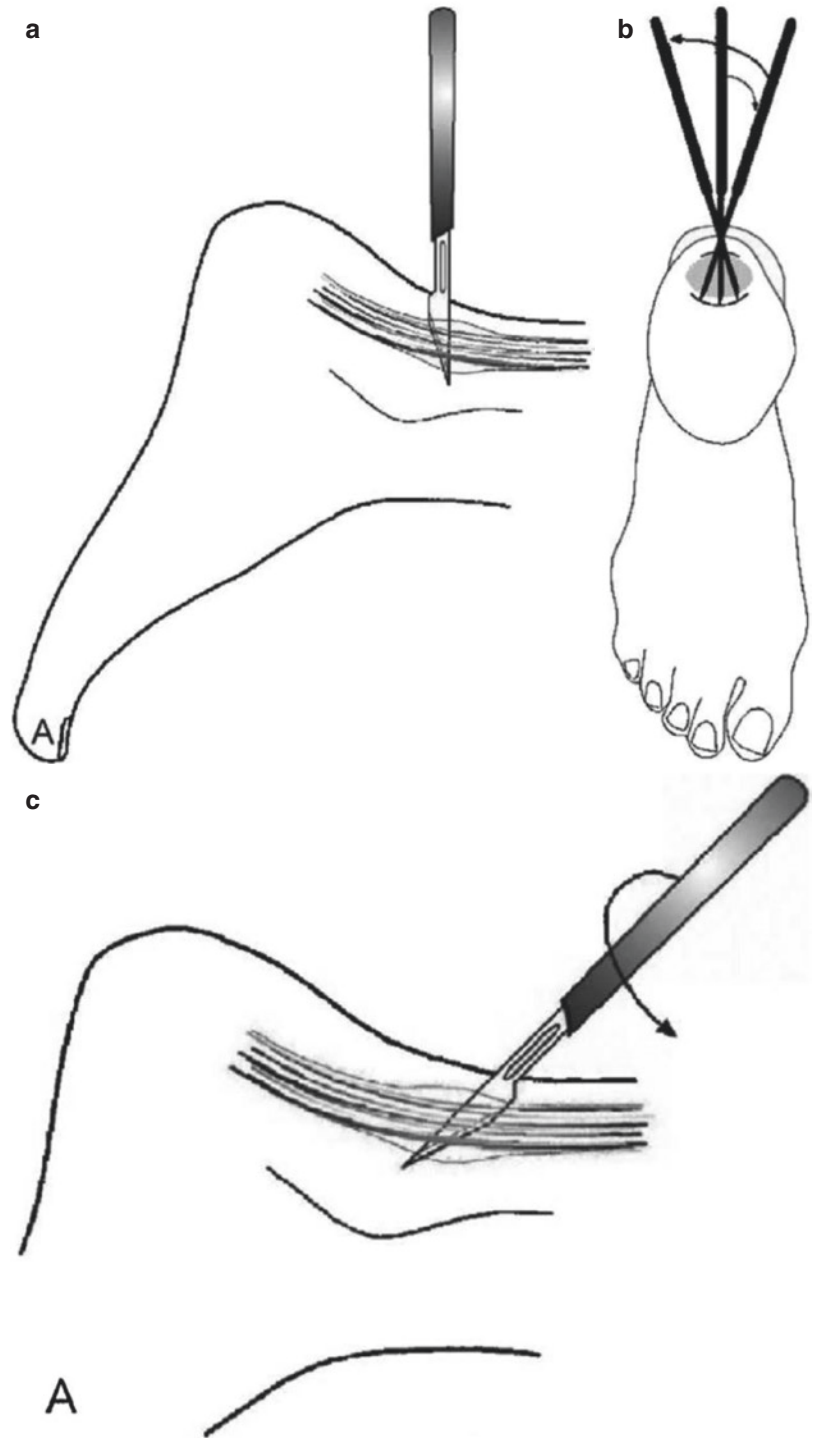

Fig. 40.4 With patient lying prone, the area of maximal tenderness is palpated, and ultrasonography identifies swelling and location is marked. (**a**) #11 scalpel blade is inserted parallel to the long axis of the tendon fibers in the center of the area of tendinopathy. The cutting edge of the blade is directed caudally and penetrates the full thickness of the tendon. With the blade in place, a full passive ankle dorsiflexion is performed. This process is repeated with the blade angled at 45° on the sagittal axis medially and laterally (**b**). Ultimately the blade is reverse 180° and the entire process repeated (**c**) *(Borrowed from Maffulli* et al *2019. Foot and Ankle Surgery)*

aggressive open debridement with tubularization of the tendon is an option. Typically, if more than 50% of the tendon is pathologic and requires debridement, we advocate for a flexor hallucis longus (FHL) tendon transfer to augment the deficit. Insertional tendinopathy is approached in a similar fashion with debridement and reattachment of the tendon. Debridement or resection of the postero-superior extent of the calcaneus is recommended to facilitate healing. Concomitant tendon transfer is indicated in greater than 50% AT resection. In cases of a nonviable Achilles tendon, with advocate for hamstring autograft as an option for reconstruction of Achilles tendon defects [90].

Surgery for insertional Achilles tendinopathy has shown to be correlated with good functional outcomes and satisfactory return to sports in 71% of patients when the surgical care was tailored to the degree of tendon involvement [91].

Take-Home Message

Management of patellar and Achilles tendinopathy in athletes requires timely diagnosis and patience in management from the provider and patient alike. Nonoperative management should be the goal and the overall mainstay of treatment. Activity modification to alleviate stresses to the affected tendon is a primary element of this approach. Due to limited level 1 evidence for the management of tendinopathy, all modalities available in our armamentarium must be used judiciously. Recent literature suggests that adjunct therapies such as extracorporeal shock wave therapy (ESWT) and platelet-rich plasma injection (PRP) are indicated to accelerate rehabilitation (eccentric exercise) and return to play. Ultimately, surgical modalities for refractory or recalcitrant symptomology are available, yet should remain avoidable with vigilant and aggressive management of tendinopathy.

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