Chapter

Patellar Tendinopathy: "Jumper's Knee"

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Abstract

"Patellar tendinopathy" is also known as "Jumper's knee" and is a common cause of impaired function in athletes who participate in sports that require jumping and running activities. The exact etiology of disease is still unknown and several theories have been postulated for its pathogenesis. It usually presents as anterior knee pain that is related to the sports activity and might lead to decreased sports participation. USG and MRI are the main modality of investigation that aids in the diagnosis. Non-operative therapy forms the main stay of treatment in form of rest, brace, physical therapy and anti-inflammatory medications. Other adjuncts such as cryotherapy, corticosteroids injection, platelet -rich plasma injections and electrical therapy like TENS or ESWT have been used with some success. Operative intervention in form of open or arthroscopic procedures are reserved for chronic and refractory cases.

Keywords: patellar tendinopathy, degenerative, tendinitis, jumper's knee, enthesis, eccentric, cryotherapy

1. Introduction

Tendinopathy is a broad term encompassing painful condition in and around tendon due to overuse. It commonly affects the Extensor mechanism (patellar tendon) around the knee and is termed as "jumper's knee" [1, 2]. It is characterized by an initial reactive or inflammatory response followed by stage of degeneration. The prevalence of patellar tendinosis is seen greatest in young adults who are engaged in high demand sports that involves running, jumping and cutting movements. It has been estimated to range from 40%-50% in elite volleyball players and 35%-40% in high level basketball players [3]. It can also affect sedentary individuals and an estimated prevalence of 14.2% is seen in general population [4].

The disease pass through two stages, acute and chronic and presentation vary according to the stage of disease. It usually present as anterior knee pain, swelling and impaired function in acute stage, while in chronic stage it may presents as long standing anterior knee pain with profound muscle wasting without the sign of inflammation or impaired function. The tendo-osseous junction on the inferior pole of patella is the usual affected site. The exact etiology of disease is still unknown. However, various risk factor have been identified which may contribute in development of patellar tendinopathy such as impaired quadriceps flexibility and strength, high body mass index (BMI), leg length discrepancy, impaired hamstring flexibility and vertical jump performance, as all these factors increases the strain over the patellar tendon [5]. Several theories have been postulated on pathogenesis of patellar

tendinopathy including vascular [6], mechanical [7], nervous [8] and impingement related. However the chronic repetitive tendon overload theory is the most accepted theory for patellar tendinosis.

The term "patellar tendinitis" which is often used for patellar tendon pain appears to be misnomer for patellar tendinopathy. Multiple histo-pathological studies have reported that the primary pathology in most of painful tendon is degenerative rather than inflammatory [9–11]. However Fredberg et al. [12] challenged the concept of patellar tendon pain due to degenerative cause and stated that it is rather the presence of inflammatory process that is responsible for pain. While some histopathological studies [13, 14] have shown that pro-inflammatory chemical agent such as cyclooxygenase, growth factors, and prostaglandins are present in acute stages and macrophage and lymphocyte in chronic tendinopathy, we still need further research and more evidences to prove the theory.

The management of patellar tendinopathy has always been challenge to the healthcare professionals. It usually requires a multimodal approach and based upon the current literature and clinical practice an effective conservative intervention in the form of rest, NSAIDS and physiotherapy is indicated in acute phase and surgical procedure are reserved for chronic and long standing cases.

2. Anatomy

Patellar tendon is the continuation of the common tendon of insertion of quadriceps, extending from the inferior pole of patella to the tibial tuberosity. In adult, the patellar tendon is around 25–40 mm wide in the coronal plane and 4–5 mm deep in the sagittal plane and 4–6 cm long. Macroscopically it appears white, glistening and stringy with collagen fiber in tendon are arranged in parallel fashion.

The blood supply of the tendon is through the anastomotic vascular ring lying in the thin layers of loose connective tissue that covers the fibrous expansion of the rectus femoris. The formation of the ring is through the anterior tibial recurrent artery and genicular arteries mainly the lateral superior, lateral inferior and medial inferior artery [15, 16].

The patellar tendon attachment to bone (patella or tibia) has fibrocartilaginous enthesis with four distinct tissue zones–dense fibrous connective tissue, uncalcified fibrocartilage, calcified cartilage and bone [17]. The patellar tendon lack of well-developed proper paratenon, while the posterior surface of tendon which is in direct contact of fat pad, which is highly vascular and innervated. According to Duri et al. [18] the intensity of pain in some patient with patellar tendinopathy is due to involvement of fat pad. Patellar tendon pathology usually involve the enthesis site, it most commonly involve the inferior pole of patella but can also involve tibial tubercle or proximal aspect of patella in quadriceps tendon. Macroscopically the diseased portion of tendon become disorganized and appears yellow-brown in color.

3. Epidemiology

Jumper's knee is commonly seen in people involved in contact sports such as basketball, volleyball, high jump, long jump, tennis and running [1]. The factor contributing factor in development of patellar tendinopathy can be classified as extrinsic factor or intrinsic factor [19]. The intrinsic factor can be sex, race, bone structure, bone density, muscle length, muscle strength, joint range of motion. While extrinsic factors are training volume (duration, frequency and intensity), specific sports activity, specific movements like quick acceleration, deceleration, cutting action and

training surfaces. Ferreti [20] observed direct relation between the patellar tendinopathy with number of weekly training session and training over the hard surface.

The condition is more commonly seen in males than females [21]. This condition is not a self-limiting one and symptoms may prevail after treatment.

4. Pathogenesis

Tendons display a classical stress-strain curve, during the increased flexion the maximum load is located in deep posterior portion of patella, close to center of rotation knee and inferior pole of patella. The crimp pattern of the tendon disappears when the length of the tendon is stretched greater than 2%. With further stretch greater than 5%, the tendon fibers become more parallel and the tendon follows a linear response to stress [22, 23]. Beyond this, tendon failure starts to begin with disruption of collagen cross links.

The force experienced by patellar tendon on a level ground while walking is 0.5 kN, which increases to 8 kN during landing from a jump, 9 kN during fast running and further to 14.5 kN during competitive weight lifting. A basketball player, on an average, jumps 70 times per game where the vertical component of the ground reaction force reaches to about six to eight times the body weight. Thus, sport activities can impose high levels of stress on the tendon, enough to cause its failure. This increased strain result in alteration of cellular activity level by affecting the tenocytes and altering the protein and enzyme production with deforming of nucleus [7, 24]. The tendon fibroblast are loaded with increased prostaglandins E2, leukotrienes B4, VEGF and matrix metalloproteinase which contribute to tendinopathy. This increased strain result in alteration of cellular activity level by affecting the tenocytes and altering the protein and enzyme production with deforming of nucleus [7, 24]. The tendon fibroblast are loaded with increased prostaglandins E2, leukotrienes B4, VEGF and matrix metalloproteinase which contribute to tendinopathy.

In chronic patellar tendinopathy, there will be absent or minimal inflammation [10, 13, 21]. The diseased tendon shows hypercellularity with atypical fibroblast and endothelial cell proliferation with neovascularization [13, 21, 25, 26]. There will be loss of longitudinal collagen fibers and demarcation between collagen bundles with relative expansion of tendon. There will be abundant number of cell undergoing apoptosis with abundance of pre-apoptotic proteins and gene [27, 28]. Macroscopically tendinopathy will have disorganized appearance described as mucoid degeneration [20, 29].

5. History and physical examination

The clinical diagnosis of jumper's knee is based on the subjective sensation of the pain and restriction of activities. The symptoms are insidious in onset but usually relates to an increase in frequency or intensity of activity involving rapid repetitive ballistic movements of the knee joint. It starts with a dull aching pain in the anterior aspect of the knee after a strenuous activity and further progresses to a state where it interferes with the performance of the individual. Patients also complain of pain while ascending or descending stairs.

The key physical finding of the patellar tendinopathy is tenderness at the inferior pole of patella when the knee is at full extension with gradual decrease in the pain when the knee is flexed gradually. It is generally accompanied by few signs of soft tissue inflammation [1]. The condition often associated with abnormalities of patellar tracking, chondromalacia patellae, Osgood-schlatter disease or mechanical malalignment of the leg.

6. Imaging

6.1 Roentgenogram

Anteroposterior, lateral and tangential views of patella may show radiolucency over involved pole in early stage, while in prolonged stage, the involved pole may appear elongated. In chronic cases tendon calcification and periosteal reaction over anterior aspect of patella ("tooth sign") may be evident. While in long standing cases the stress fracture or disruption of extensor mechanism may occur.

6.2 Ultrasonography

Ultrasound and MRI is the main modality of investigation for jumper's knee. Although CT scan can image patellar tendon, it does not offer any significant advantage over the above mentioned investigations. However studies have shown CT scan to be of some prognostic value [30, 31].

Role of ultrasound:

1. Detect preclinical lesion in athletes

2. Detect patellar tendon pathology and assess its severity

Ultrasonographic appearance:

Patellar tendons in patients suffering from jumper's knee have decreased echogenicity, containing either a sonolucent region or diffuse hypoechogenicity [31–33]. The tendon envelope is irregular, and there may be erosion of patellar tip and intratendinous calcification may be present.

6.3 Role of MRI

1. Identification of exact location and the extension of the tendon involvement

2. Exclusion of other condition such as bursitis, chondromalacia

3. Quantification of the size of patellar tendon to be excised during surgery

Appearance in MRI:

The abnormal patellar tendon contains an oval to round area of high signal intensity in T1- and T2-sequence at the tendon attachment site or focal zones of high signal intensity in the deeper zones of the tendon [34, 35]. The T2-weighted sequences (particularly the T2*-weighted GRE sequences) have greater sensitivity than the T1-weighted protocols. However, the T1-weighted signal can image most cases of patellar tendinopathy.

Ferrati et al. [36] classified jumper's knee into six stages according to symptoms:

Stage	Symptoms
0	No pain
1	Pain only after intense sports activity, no undue functional impairment
2	Pain at the beginning and after sports activity, still able to perform at a satisfactory level
3	Pain during sports activity, increasing difficulty in performing at a satisfactory level
4	Pain during sports activity, unable to participate in sports at as satisfactory level
5	Pain during daily activity, unable to participate in sport at any level

7. Management

Unfortunately the management used for chronic tendon disorders has a very little scientific backdrop and varies considerably among the surgeons and across countries. Thus the treatments listed below are at best empirical.

7.1 Rest

This modality is useful in athletes presenting for the first time with patellar pain but becomes a concern in individuals involved in competitive sports.

7.2 Straps, braces and exercises

One approach to help in healing of the tendon tissues is unloading. This can be achieved either by the modification of the activities for e.g. decreasing the number of jumps or landing on the ground with proper orientation of the foot, or use of knee braces and straps such as chopart straps that share and alter the load on the tendon. Chopart strap [10] is a tape attached just proximal to tibial tendon. The most popular non-operative treatment involves eccentric exercise.

7.3 Cryotherapy and physical modalities

Cryotherapy helps in controlling initial tissue response to the injury. It is thought to act by decreasing the blood flow and metabolic rate, thereby decreasing the rate of inflammation. Electrical modalities that have been used in patellar tendinopathy include ESWT, ultrasound, heat, interferential therapy, magnetic fields, pulsed magnetic and electromagnetic fields, transcutaneous electrical nerve stimulation (TENS), and laser [10, 37, 38]. The true effects of the above mentioned modalities still remain unknown and further studies are required to support their use.

7.4 Remedial massage

It tends to treat the tendon tissue by having an effect on the muscle stretch and direct effect on the tendon cells. Muscle belly massage is thought to increase the compliance of the muscle and decrease the load on the muscle. Deep friction massage is thought to activate the mesenchymal cells to stimulate the healing response. "Fibrolysis", a form of deep frictional massage originally developed in Finland, has been successful in Achilles tendinopathy. However a controlled study failed to provide any evidence of healing in patellar tendinopathy and further evidence is required to warrant its use [39].

7.5 Rehabilitation

The key treatment nowadays to chronic tendinopathy is the stretching and strengthening programme of the whole muscle tendon unit. A staged program for tendinopathy corresponding to the stages of worsening severity of the condition [1] was outlined by Stanish et al. [40] outlined in **Table 1**.

Drop squat forms one of the key exercise for this condition in which patients are asked to sit to about 100–120° of knee flexion from a standing position and are advised to perform three sets consisting of 10 repetitions per session. It was observed that this regimen brought about complete relief in 30% of patients with reduction in the symptoms in further 64% of the patients [41]. Worsening symptoms were seen in the remaining 6% of patients. Cannell [42] observed that eccentric squats were better as compared to leg curl/extension exercises in treatment of the condition.

Stage	Program
Stage 1	Adequate warm up Ice after activity Local anti-inflammatory treatments, including non-steroidal anti-inflammatory drugs (NSAIDs) Physiotherapy(isometric quadriceps exercises and an elastic knee support)
Stage 2	Addition of period of rest and heat before activity
Stage 3	Addition of prolonged period of rest

Table 1.
Program for patellar tendinopathy.

7.6 NSAIDS

Although the benefits of NSAIDs are dubious, they are the most common drug used for symptomatic relief [43]. Although the use of "anti-inflammatory" medication seems paradoxical for a condition that is essentially degenerative, it is believed NSAIDs might act via mechanisms different from their conventional anti-inflammatory actions [44]. In vitro studies in human cartilage have revealed a variable interaction of NSAIDs with glycosaminoglycans (GAGs) where some have been shown to stimulate and some to inhibit, its synthesis [45]. This mechanism also sheds light on its effect on the synthesis of extra-cellular matrix. In a double blinded placebo controlled study, the use of NSAIDs in tendinopathy, piroxicam did not benefit patients with Achilles tendinopathy however topical ketoprofen reached the target tissue in patients with patellar tendinopathy, but the clinical efficacy was not assessed [46].

7.7 Corticosteroids

Corticosteroids are known for reducing the symptoms arising from the inflamed synovial structures. However the role of corticosteroids remains controversial in management of tendinopathy. According to Jozsa and Kannus [47] steroids are contraindicated in acute phase of tendinopathy and in the late chronic phase of tendinopathy when the tendon degeneration is advanced which may lead to tendon rupture. However it has proved beneficial when diluted with anesthetic for diagnostic reasons and to minimize adverse effects and in conditions where 1–6 week rest period combined with a programme of gradual strengthening is required before returning to activity.

7.8 Other medical treatments

Aprotinin, an 85 amino acid 65 kDa basic polypeptide extracted from bovine lungs has shown to offer better pain relief than steroids at least in short term. However aprotinin which is a strong inhibitor MMP (matrix metalloproteinase) is less effective in insertional tendinopathy as compared to main body [48]. Another non-surgical treatment option includes use of sclerosing agent with chemical irritant (e.g. polidocanol) [8, 49, 50]. These targets the neovascularization and accompanying nerves. The use of platelet-rich plasma injection has been tried in tendinopathy and favorable outcome have been found [51–53]. However still there is no level 1 or level II studies about role of PRP in patellar tendinopathy. The glyceryl trinitrate (GTN) patch [54, 55], which delivers nitric oxide (NO) to pathological tendon which play role in tendon healing. But we still need level1or level II evidence to support it.

7.9 Surgical treatment

Patellar tendon surgery is indicated in patients who have failed conservative management more than 6 weeks [56–58]. A variety of surgical procedures have been described such as resection of the tibial attachment of the patellar tendon with realignment, drilling of the inferior pole of the patella, macroscopic necrotic area excision [59], repair of macroscopic defects, longitudinal tenoplasty/tenotomy of the tendon [60] percutaneous longitudinal tenotomy, percutaneous needling [61] and arthroscopic assisted decompression [62, 63] of the tendon, possibly with excision of the inferior pole of the patella however the effectiveness of any single procedure has not been elucidated.

8. Conclusions

Patellar tendinopathy is essentially a degenerative condition and the management should be based on the clinical assessment. Imaging appearances, although aids in the diagnosis but should not determine the treatment. Conservative treatment forms the mainstay of management, while surgery is indicated only after a dedicated period of appropriate conservative measures have been instituted, usually around 6–9 months. These include physical modalities such as local application of ice and graduated strengthening physiotherapy protocol such as functional exercises and eccentric strengthening; the latter are done only after the patient is pain free. Although there is a lack of level I evidence, eccentric training appears to be the most promising modality. Peritendinous corticosteroid or aprotinin infiltration may also be useful as an adjunct for the treatment of this condition. Although scientific consensus is lacking percutaneous needling appears to be the least invasive procedure, followed by percutaneous longitudinal tenotomy. Arthroscopic debridement has been proposed, but, although early results are encouraging, its efficacy is still under scrutiny.

Conflict of interest

No conflict of interest.

Acronyms and abbreviations

BMI	body mass index
NSAID	nonsteroidal anti-inflammatory drug
VEGF	vascular endothelial growth factor
MRI	magnetic resonance imaging
CT	computed tomography
GRE	gradient echo sequence
ESWT	extracorporeal shockwave therapy
TENS	transcutaneous electrical nerve stimulation
MMP	matrix metalloproteinase
PRP	platelet rich plasma
GTN	glyceryl tri-nitrate
NO	nitric oxide

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