Non-operative Treatments for Patellofemoral Arthritis

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10.1 Introduction

Anterior knee pain, also known as patellofemoral pain (PFP), affects approximately 22% of the general population, with increased prevalence in the adolescent community, as high as 28% [1]. It is twice as common among females (29.2%) than males (15.5%) [1]. The predominant demographics for PFP includes adolescents, young active adults, military recruits, and elite athletes, particularly runners, cyclists, and basketball players [2–5].

PFP is characterized by the presence of non-traumatic anterior knee pain, with and without structural damage to the patellofemoral joint [6]. In the absence of structural changes to the patellofemoral joint, such as in young patients, the presence of PFP is considered "patellofemoral pain syndrome" (PFPS), defined by retropatellar pain with actions that increase load across the joint such as ascending or descending stairs, hopping, jogging, prolonged sitting, and squatting [7]. Patients with PFPS do not typically have positive findings on examination of the knee bursa, ligaments, menisci, or plica [7]. Common causes of patellofemoral pain not due to patellofemoral pain syndrome include patellar and quadriceps tendinopathy, iliotibial band syndrome, lateral patellofemoral compression, and plica syndrome, which will be discussed later in the chapter. The presence of structural damage to the patellofemoral joint is considered patellofemoral osteoarthritis (PFOA), affecting predominantly older adults [6, 8].

It was previously believed that PFPS is a benign, self-limiting condition. This notion has been discredited, however, upon the grounds of prospective studies that have shown that patients with PFPS are more likely to decrease or stop their sports activities over a 2-year period [9, 10] and consistently report poorer scores on patient-reported outcomes for knee-specific and quality of life measures [11]. Young patients with PFPS have similar morbidity to patients with ACL injuries, without the resolution of symptoms with surgery or rest [12]. Though symptoms may improve with maturity from adolescents into adulthood, the pain usually persists. In a study by Sandow and Goodfellow, 63 patients with PFPS were followed for 16 years after initial presentation; 78% of patients reported persistent pain, with 71% of patients reporting some improvement of symptoms with age likely due to cessation of pain-inducing activities [13, 14]. Consequently, recognizing and understanding PFPS, and treating it appropriately, is critical for sports medicine physicians.

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This chapter will primarily address the etiology, history, clinical evaluation, and treatment of PFPS and its associated conditions, with a brief discussion of PFPS as a precursor to PFOA.

10.2 Patellofemoral Pain Syndrome

10.2.1 Anatomy of the Patellofemoral Joint (PFJ)

The PFJ is a diarthrodial joint with its articulation consisting of the posterior aspect of the patella and the trochlear surface of the distal femur. The proximal portion of the patella is termed the base, while the distal point is termed the apex. It is approximately 35–40 mm in length, 40 mm in width, and 20–25 mm thick [15, 16]. The articulating surface of the patella is covered in up to 7 mm of cartilage, essential to the dissipation of joint reaction forces generated with contraction of the quadriceps [15, 17].

The distal femur consists of an intercondylar groove, or trochlear sulcus, upon which the patella engages. Its lateral facet is larger than its medial facet to improve patellar stability [15]. The sulcus angle, measured at the intersection of the medial and lateral sulcus lines, is used to evaluate for trochlear dysplasia. A normal angle is approximately 138 ± 6°; a larger angle, or shallower groove, suggests dysplasia [17].

The soft tissue surrounding the PFJ helps prevent lateral translation of the patella. Static structures include ligaments about the knee, while dynamic structures include muscular attachments. The most important static stabilizer is the medial patellofemoral ligament, which has been reported to provide 60% of total lateral restraint at 20 degrees of knee flexion [18, 19]. Key lateral structures include the iliotibial band, lateral patellofemoral ligament, and joint capsule, which help to provide stability during less than 20–30 degrees of flexion due to absence of bony stability [15].

Dynamic stabilizers include the pes anserine, the biceps femoris, and, most importantly, the vastus medialis oblique (VMO). The oblique angle of the VMO creates a strong medializing force on the patella, thereby resisting lateral translation [15]. Additional soft tissue components key to PFJ stability include hip abductors, in particular the gluteus medius and minimus, and external rotators [20]. Weakness of such muscles allows for increased hip adduction and internal rotation, which together increase the Q-angle and cause increased lateral contact pressure on the PFJ and subsequent wearing of the articular cartilage [21].

10.2.2 Biomechanics and Kinematics of PFJ

The function of the patella is to serve as a mechanical pulley for the quadriceps muscle, increasing quadriceps power by 33–50% (it is most important during the last 30 degrees of knee extension).

When considering static alignment of the patella in PFPS, the frontal plane is most important as the patella has the most minimal contact with the femur in this position, allowing the patella to be most mobile and, therefore, least stable [15]. In this plane, the patella sits midway between the two femoral condyles with the knee in full extension. This is also the plane in which the Q-angle is measured, using the angle between the line extending from the anterior-superior iliac spine to the mid-patella and the line extending from mid-patella to tibial tuberosity. Normal Q-angle is 15–17° for females and 10–13° for males, with the gender difference being the results of a wider pelvis in women [22]. Increases in the Q-angle are associated with increased lateral forces on the patella and therefore increased contact stress [15]. Abnormal frontal plane motion, particularly during squatting, step-down, and jogging, is most commonly associated with PFPS [23].

In the sagittal plane, with the knee in partial flexion, the Caton-Deschamps index is measured. This is used to evaluate patella atla and patella baja. In the axial plane, the patella sits horizontally with the medial and lateral borders equidistant between the medial and femoral condyles.

Patellar tilt and tibial tubercle-trochlear groove distance are evaluated in this plane. The mean TT-TG distance is 10–13 mm, with greater values associated with increased risk for patellofemoral maltracking and instability [22].

Dynamic movement of the patella is evaluated in multiple planes including superior and inferior glide, medial and lateral glide, medial and lateral tilt, and medial and lateral rotation [15]. As the knee flexes and extends, the patella glides inferiorly and superiorly, respectively. Contact area between the patella and trochlea increases with knee flexion in order to distribute force over a greater surface area, thereby decreasing point contact pressure [15]. This is essential to preventing excessive wear on the articular cartilage that occurs with increased joint pressures in the setting of decreased contact areas.

10.2.3 Etiology of PFPS

In the absence of a single structural defect to account for the pain experienced in PFPS, its etiology is considered multifactorial. It is largely attributed to patellar maltracking, but has also been ascribed to aberrant pain pathways and psychological catastrophizing. As it is considered to be an overuse syndrome, its risk factors should be characterized. Such risk factors are generally classified as extrinsic or intrinsic risk factors, where extrinsic factors include the type of sport played, equipment used, and environment played in [24]. Intrinsic risk factors include all other components contributing to the clinical presentation of PFPS, such as quadriceps imbalance and abnormal trochlear groove. While osseous risk factors are not modifiable without surgery, all other intrinsic and extrinsic risk factors may be considered modifiable, as discussed in the treatment section of this chapter.

While such intrinsic and extrinsic risk factors contribute to the development of PFPS, it is the combination of such components in the setting of acute increases in load across the joint that likely causes the pain described in PFPS. According to Dr. Scott Dye, the typical function of the patellofemoral joint, without pain, should be viewed as within the limits of tissue homeostasis, also referred to as an "envelope of function" [25, 26]. This envelope is established by exposure of chronic loads to the PFJ and its surrounding structures, with subsequent adaptation of the joint to such loads. With the accumulation of acute increases in loading across the joint, the envelope of function may be exceeded, thereby disrupting the joint's homeostasis. This results in excessive load transfer to subchondral bone causing micro-damage and inflammation that excites nociceptive fibers, resulting in pain [27]. Though intrinsic factors themselves do not result in a painful joint, the presence of extrinsic factors, such as temporary overuse or increase in physical activity level, creating acute increases in load in the setting of pathological kinematics, results in pain. This section will discuss the intrinsic factors contributing to the envelope of function of the PFJ, as well as the extrinsic factors that result in a disruption of homeostasis and resultant pain.

10.2.3.1 Patellar Maltracking

Patellar maltracking, an intrinsic risk factor for PFJ, is typically characterized by static and/or dynamic malalignment resulting in irregular tracking of patella within the trochlear groove. Recent literature has also suggested that atypical patellar shape may also contribute to maltracking [1]. This maltracking results in abnormal contact pressures across joint, which, in the setting of acute load, can cause pain.

Static malalignment is typically characterized by osseous and ligamentous abnormalities including trochlear dysplasia, patella alta, MPFL laxity, and lateral retinacular tightness. Trochlear dysplasia, or a sulcus angle greater than 138 ± 6°, results in an increased tibial tubercle-trochlear groove distance [28], causing lateralization of the patella within the groove. Similarly, a high-riding patella, or patella alta, has been associated with maltracking due to a greater distance for the patella to travel before engaging with the trochlea. "Alta" is determined by an Caton-Deschamps ratio greater than 1.2. This ratio is determined by dividing the distance from the inferior aspect of the patellar articular surface to the tibial plateau

by the length of the patellar articular surface [15]. Such static components, when combined with dynamic valgus, result in pathological kinematics that prime the PFJ for inability to tolerate increased loads.

Dynamic malalignment is characterized by disproportionate lateral pull on patella due to vastus medialis oblique (VMO) deficiency and excessive internal rotation of the femur and tibia due to soft tissue imbalance and rear-foot eversion. Weakness of hip abductors and external rotators and iliotibial band tightness are commonly identified as causes for internal rotation of the femur, while rear-foot eversion results in internal rotation of tibia.

VMO Deficiency

Deficiency of the VMO weakens the medial quadriceps vector, thereby allowing greater pull of the lateral quadriceps vector with a resultant increase in the dynamic Q-angle. Due to this loss of the medial force, the patella is pulled laterally, out of its normal tracking. Additionally, studies have shown delayed activation of the VMO as compared to the vastus lateralis, at 15, 30, and 45 degrees of knee extension, using electromyography [29]. Such delayed activation contributes to VMO dysfunction relative to vastus lateralis function, with subsequent further lateralization of the patella.

Hip Abductor and External Rotator Weakness

The gluteus medius and minimus, two primary hip abductors, are frequently weak in the setting of PFPS. While not directly related to PFJ kinematics, weakness of such muscles allows the femur to adduct/internally rotate more than normal, thereby increasing lateral patellar contact pressure causing subsequent increased pain [30]. Ireland et al. reported that female patients with PFPS had 26% less hip abduction strength and 36% less hip external rotation strength than their non-painful counterparts [30, 31]. Such pathomechanics result in a greater portion of the absorption load to be transferred to passive lower limb structures, with subsequent "out-of-plane" loading with greater control on the frontal and transverse planes [32]. As a result, the patella is forced to dissipate higher levels of force via less efficient control mechanisms [32].

Iliotibial Band Tightness

The IT band has been associated with PFPS due to its attachment to the lateral retinaculum and patella. In the setting of such connections, a tight IT band will increase the lateral force vector on the patella, increasing joint stress [33].

Rear-Foot Eversion

Patients with PFPS have been shown to have reduced range of rear-foot eversion, increased rear-foot eversion during heel strike, and delayed timing of peak rear-foot eversion, compared to controls [6, 34]. Such foot abnormalities contribute to internal rotation of the tibia in PFPS, with resultant increased lower extremity valgus.

Hamstring tightness is also commonly seen in patients with PFPS, with resultant co-contraction of the quadriceps and hamstrings relatively to controls [6, 35]. This co-contraction results in increased joint forces on the PFJ, contributing to greater contact stress. Additionally, the lateral hamstrings of patients with PFPS have been shown to contact earlier than medial hamstrings during isometric exercises, increasing patellar maltracking [6, 36].

10.2.3.2 Overuse

While patellar maltracking may "prime" a knee for PFPS, overuse of the knee is essential to the development of the syndrome, as it is this acute stress that results in surpassing its envelope of function and presentation of pain. Patients with PFPS are classically athletes participating in sports that repetitively load the knee, such as running, cycling, and basketball. Fairbank et al. evaluated involvement in competitive sports in female patients with PFPS and found that those with PFPS were more likely to be involved as compared to age-matched controls [30, 37]. He noted that the patient's associated pain onset was correlated with an increase in activity level.

10.2.3.3 Aberrant Pain Pathways

Neurodynamic causes of PFPS have gain popularity in the last two decades. This theory suggests

that minor nerve damage, or altered mechanosensitivity of nerves about the knee, contributes to PFPS. For example, Sanchis-Alfonso and Rosello-Sastre have demonstrated that excessive pressure on the patella causes periodic episodes of ischemia that trigger neural proliferation and cause pain [38, 39]. This group also observed that patients with PFPS often possess hyperinnervation in the lateral retinaculum, which may also contribute to the patients' pain experience [40]. Such hyperinnervation the lateral retinaculum has been corroborated by additional studies that examined the incidence of free nerve endings in the soft tissue structures of the knee and found the highest amounts of afferent nerve fibers type IVa in the retinacula, as well as the patellar ligament, pes anserinus, and ligaments of Wrisberg and Humphrey [41].

The femoral nerve which gives rise to the medial and lateral patellar nerves has also been shown to play a key role in PFPS symptoms. Blocking of the femoral nerve with local anesthetics significantly reduced pain intensity among 20 patients with PFPS, in a study by Maralcan et al. [42]. Further support for the importance of the femoral nerve is demonstrated in a study by Lin et al., in which patients with and without PFPS were subjected to the femoral slump test (FST), the neurodynamic test used to assess mechanosensitivity of the femoral component of the nervous system [39]. The research revealed that patients with PFPS had a smaller hip extension angle during the FST as compared to the control group, suggesting that mechanosensitivity of the femoral nerve may play a role in the development of anterior knee pain.

10.2.3.4 Psychological Impact: Catastrophizing and Fear Aversion

Many patients with PFPS experience psychological distress due to their chronic knee pain [6]. Thomee et al. demonstrated that patients with PFPS cope and experience pain similarly to other chronic pain patients; however those with PFPS tend to catastrophize more than other chronic pain patients [43]. Such catastrophizing likely contributes to perpetuation of pain and cessation of sports activities. This fear-avoidance belief regarding physical activity has been shown to be a psychological risk factor for pain and function in those with PFPS [44].

10.2.4 History

Patients with PFPS typically report pain around or behind the patella that is worse with activities that load the patellofemoral joint, including ascending and descending stairs, squatting, running, and jumping. Patients may also describe pain after prolonged sitting, called the "theater sign" [25, 45]. Additional symptoms include crepitus within the patellofemoral joint during knee flexion, tenderness to palpation around the patella, and the presence of a small effusion [46]. Pain is often bilateral, though it is typically worse on one side than the other. Patients often have trouble localizing their pain at a precise location on the patella and therefore typically place their hand on the knee or circumscribe the patella, known as the "circle sign" [45].

Important to note, a history of dislocation or subluxation is exclusion criteria for the diagnosis of PFPS.

10.2.5 Clinical Evaluation

As patellofemoral pain syndrome is considered a diagnosis of exclusion, clinical evaluation of anterior knee pain should be systematically approached and should include inspection, palpation, gait, and special tests for anterior knee pain.

10.2.5.1 Inspection

The general appearance of each knee should be considered, taking into account any erythema, swelling, or additional skin changes that may be present. Significant swelling with or without contusion, in the setting of patellar injury, may suggest a patellar dislocation or fracture, both of which are separate entities to PFPS. Any bony abnormalities should be noted, as well as the presence of genu varum or genu valgum. Obvious muscular deformities

should be appreciated as well, such as a bulge in the anterior thigh suggesting quadriceps tendon rupture.

10.2.5.2 Palpation

Each knee should be palpated for tenderness around the four poles of the patella, as well as the medial and lateral joint lines, tibial tubercle, pes anserine, patellar and quadriceps tendons, and pre-patellar and supra-patellar bursas. Patients with patellofemoral pain syndrome may present with pain at the superior or inferior poles; however tenderness along the joint lines, tibial tubercle, pes anserine, and supra-patellar bursa typically suggest alternative etiologies for anterior knee pain such as meniscal pathology, patellar tendinitis, pre-patellar bursitis, or apophysitis.

10.2.5.3 Gait

Due to pain and altered biomechanics, patients with PFPS often present with an abnormal gait. Fox et al. examined gait kinematics in patients with acute (<3 months of symptoms) and chronic (>3 months of symptoms) PFPS and found that both groups had greater knee flexion across stance and greater ankle dorsiflexion during early stance as compared to age-matched controls [47]. Interestingly, patients with acute PFPS exhibited greater transverse plane hip motion across stance, while chronic PFPS patients demonstrated greater frontal plane hip motion. Patients with chronic PFPS also exhibited greater knee abduction, and reduced ankle eversion, as compared to acute PFPS and age-matched controls [47].

10.2.5.4 Special Tests

Merchant et al. describe five physical exam maneuvers, and two radiographic measurements, to evaluate anterior knee pain. The physical exam tests will be discussed here:

1. *VMO deficiency*: This is assessed by having the patient actively maintain an unsupported leg at 30° flexion while sitting. Deficiency is observed when the VMO inserts higher into the medial edge of the quadriceps tendon.
2. *MPFL ligament laxity*: This is assessed using the lateral glide test, where quadriceps are relaxed and the leg is supported at 30° flexion, while sitting. The physician then pushes the patella laterally, allowing the patella to translate approximately 1 finger-breadth. The lateral glide test is positive if the patient demonstrates apprehension during this motion.
3. *Lateral retinacular tightness*: This is assessed by attempting to centralize the patella in the trochlear groove and everting the patella to neutral. If this cannot be completed, excessive tightness is likely present.
4. *Q-angle measurement*: The patient should be supine with their leg in neutral rotation and the knee in full extension.
5. *Hip abductor weakness*: This is assessed with the step-down test, in which the patient stands on a stool/stair and slowly steps down with the opposite limb, allowing their heel to touch the ground, before slowly rising up. The test is considered positive if a Trendelenburg sign is seen.

Additionally, the iliotibial band may be examined for tightness, as a possible contributor to PFPS, by using Ober's test [48]. This test is performed with the patient in the decubitus position with the non-affected lower leg flexed to 45° to maintain a neutral spine [48]. The knee of interest is flexed to 90° with the upper leg brought into abduction and extension. The physician then lowers the leg into adduction, observing for abnormal hip rotation [48].

10.2.5.5 Radiographic Evaluation

Plain Radiographs

While PFPS can typically be diagnosed without imaging, plain radiographs are often useful in diagnosing osseous abnormalities, including patella alta and trochlear dysplasia, and in excluding other items in the differential diagnosis for anterior knee pain such as meniscal pathology and plica syndrome.

Bilateral AP views are useful for evaluating the tibiofemoral joint, but may also show patellar abnormalities such as gross patella alta, and lateromedial subluxation. Lateral views are

used to evaluate patellar height, and the presence of patella alta or baja, using the Caton-Deschamps ratio.

Merchant view, particularly the standing loaded Merchant, has been shown to be the gold standard for representing joint kinematics. Axial views are preferable for measuring patellar translation or the lateral or medial displacement of the patella with respect to the trochlear groove where >2 mm is considered abnormal [45]. Similarly, the sulcus angle at the intersection of the lines drawn from the medial and lateral femoral condyles is used to evaluate degree of trochlear dysplasia, using axial radiographs. The normal range is 138 ± 6°, where >144° is considered diagnostic of trochlear dysplasia.

Advanced Imaging: CT and MRI

The primary advantage of CT and MRI over plain radiographs is the ability to evaluate soft tissue abnormalities that may be contributing to anterior knee pain such as chondral defects, patellar and quadriceps tendinopathy/tendinitis, bursitis, plica, and integrity of the MPFL. Additionally, certain measurements such as the tibial tubercle-trochlear groove distance are better measured on MRI or CT. Furthermore, dynamic MR imaging allows for assessment of patellofemoral kinematics with real-time tracking of patellar movement and surrounding muscle function [45].

Drew et al. conducted a systematic review investigating which PFJ imaging features are associated with PFPS as compared to asymptomatic controls [49]. MRI bisect offset at 0-degree knee flexion under load and CT-derived congruence angle at 15-degree knee flexion with and without load were shown to both be strongly associated with PFP. Increased patellar tilt and decreased patellofemoral contact area were also shown to be suggestive of PFP radiographically [49].

10.2.6 Treatment

10.2.6.1 Non-operative

The mainstay of treatment for PFPS is currently strengthening and gait retraining. Additional non-operative measures include cortisone injection, hyaluronic acid injection, orthobiologics such as platelet-rich plasma or stem cell injections, and passive correction of patellar maltracking with bracing and taping.

Strengthening

Strengthening exercises for PFPS management originally focused on strengthening the knee via quadriceps strengthening as VMO weakness is a known factor in the etiology of PFPS. In recent years, however, the importance of hip strengthening, in particular the hip abductors and external rotators, has been identified as a potentially more important treatment for PFPS. Two recent systematic reviews that investigated the importance of hip and knee strengthening as compared to hip strengthening alone found that the combination therapy significantly reduced pain in patients with PFPS as compared to knee strengthening alone [50, 51]. When comparing hip strengthening alone to knee strengthening alone, earlier reduction of pain has been shown in hip strengthening groups as compared to knee strengthening groups [52]. It is believed that the relative importance of hip strengthening over knee strengthening is due to the change in hip and knee biomechanics during functional activities which addresses the underlying cause of PFJ loading, with hip strengthening [52]. Knee strengthening, in comparison, helps to relieve lateral joint stress on the joint, but does not alter the biomechanics of the hip and knee as significantly as hip strengthening.

In addition to strengthening of the hip and knee, core strengthening has been recently discovered to be an important component to add to PFPS treatment regimens [53–55]. As neuromuscular deficits of the core muscles have been associated with greater risk for knee injury, strengthening of these muscles is useful in the treatment of PFPS [56]. Additionally, patients with PFPS have been shown to have impaired trunk postural control as compared to age-matched controls [53]. Furthermore, patients with PFPS have been shown to have abnormal postural control in both static and dynamic balance positions, as well as core muscle contraction with voluntary heel raise [54]. Such weakness of core musculature contributing to postural imbalances likely contributes to

the altered biomechanics and resultant pain seen in patients with PFPS. Therefore, strengthening of the core musculature should improve posture and reduce pain in patients with PFPS. In a recent study by Foroughi et al., patients with PFPS were treated with either hip, knee, and core strengthening or hip and knee strengthening alone [54]. While both groups demonstrated reduced pain after 4 weeks of treatment, those that received the additional core-strengthening regimen reported greater reduction in pain.

Gait Retraining

Patients with PFPS have greater incidence of rear-foot foot strike pattern as compared to controls. This pattern of foot strike causes greater shock attenuation, loading rate, and patellofemoral joint stress, thereby contributing to the pain experience. As a result, gait retraining has gained popularity as an adjunct or an alternative to strengthening for treatment of PFPS. In a recent prospective study of Roper et al., PFPS patients with rear-foot strike patterns were retrained to adopt forefoot strike patterns [57]. This gait retraining produced significant reductions in pain according to a visual analog scale. Support for pain reduction with retraining to forefoot strike pattern is corroborated by additional studies that have shown a 10–27% reduction in peak patellofemoral joint stress, as compared to rear-foot strike patterns [58–61]. In addition to retaining strike patterns, increasing the step rate, or number of steps per minute, and increasing forward trunk lean are gait retraining methods that have been associated with reduced patellofemoral joint stress [58, 62, 63]. Dos Santos [64] compared these three methods of gait retraining and found that forefoot strike training most significantly reduced patellofemoral pain in patients with PFPS compared to the other two methods. They attributed this improvement in pain with forefoot running to an increased plantarflexor moment during stance phase, allowing greater control of ankle dorsiflexion from ground reactive forces [58]. Additionally, dos Santos found that forefoot running allows for a reduction in peak knee flexion angle; increased knee flexion has been associated with higher patellofemoral joint stress and subsequent pain.

Foot Orthotics

Similar to bracing and taping, the use of orthotics in PFPS has been frequently debated. Recent studies have demonstrated some benefit of orthotics over short-term interventions [65]. In a 4-week intervention using semi-custom orthotics in runners with PFPS, reduction in pain due to decreased patellofemoral loading was observed [66]. This study attributed the reduction in loading to reduction in knee flexion via proprioceptive effects produced by shock attenuating properties of the orthotics. Similarly, foot orthoses have also been shown to improve joint stability and reduce work by dorsiflexors such as the abductor hallucis and tibialis anterior [67]. Such altered biomechanics improves stability of the knee and reduces pain-producing components seen in PFJS. Important to note, the usefulness of foot orthoses may be limited to those who have greater peak rear-foot eversion during walking and greater midfoot flexibility [65, 68, 69]. Additionally, strong evidence suggests that foot orthoses do not improve outcomes by 12 or 52 weeks compared to placebo, but may improve outcomes in the short term, over 6 weeks [65].

Bracing and Taping

Both bracing and taping to passively correct patellar malalignment have been explored extensively; however, the literature remains inconclusive regarding long-term benefit of such interventions [70, 71]. Patellar bracing has shown some short-term benefit in small studies evaluating the effect of knee bracing in PFPS [66]. According to a systematic review by Saltychev [71], however, of the 37 studies included in their review, 30 did not demonstrate a significant benefit with patellar bracing. Kinesio taping of the VMO has been shown to decrease pain and improve function of the quadriceps in athletes with PFPS [72]; however these results were among only 15 patients with PFPS, limiting the power of the results. In a systematic review by Logan et al., five studies were evaluated for the effect of taping on patellofemoral pain syndrome [73]. The review found

that knee taping may be beneficial in reducing PFPS but only as an adjunct to strengthening therapy.

10.2.6.2 Operative

Surgical treatment for PFPS is uncommon and is reserved for cases due to severe osseous and ligamentous abnormalities that prevent normal patellar tracking despite non-operative treatment programs. Arthroscopic lateral retinacular release may be performed in those with excessive lateral retinacular release; however we do not recommend this treatment. The release has been shown by some authors to relieve lateral tension and decrease surface pressure, as well as denervate the hyperinnervated lateral retinaculum which is believed to contribute to the etiology of PFPS [74]. More typically, lateral release procedures may also be combined with a tibial tubercle osteotomy to unload the lateral aspect of the patellofemoral joint or in dislocators with a medial patellofemoral ligament reconstruction as part of general soft tissue balancing [6, 75].

10.3 Additional Causes of Anterior Knee Pain

10.3.1 Patellar Tendinopathy

10.3.1.1 Pathology

Patellar tendinopathy, commonly referred to as jumper's knee, is an overuse injury affecting the patella tendon. This condition can be very painful and often affects young patients, particularly athletes. Athletes who participate in sports that involve a lot of jumping, such as basketball and volleyball, are more likely to present with patellar tendinopathy [76]. The other common name for this condition, patella tendinitis, is misleading as it refers to an inflammatory disorder, when tendinopathy actually describes a degenerative disorder. However, studies involving more modern research tools have shown evidence that inflammatory responses may be an important component to chronic tendinopathy [77]. There is no consensus on the pathogenesis of tendinopathy, which is a main reason there is no consensus on an effective treatment for this disease. The most widely accepted theory for pathogenesis of tendinopathy describes cellular and mechanical property changes as a result of repetitive microtrauma to the tendon [78]. An alternative theory is the neural theory, which describes the release of pain-generating neurotransmitters and substance B due to cellular changes within the nerves themselves [78]. The last major theory is the vascular theory, which is studied more in regard to other tendons rather than the patella tendon. This theory blames the degeneration and substandard healing of tendons to the poor blood supply tendons receive [79].

10.3.1.2 Diagnosis

Patellar tendinopathy presents itself as localized anterior knee pain at the inferior pole of the patella, as consistent with the origin of the patella tendon. When magnetic resonance imaging is performed, there tends to be an increase of signal intensity at this location [80]. It is important to note that there are patients who have imaging that reads abnormal, but who have no pain. Onset of pain is often gradual. Studies show that the following characteristics are related to increased risk of patellar tendinopathy: male gender, increased weight, decreased upper leg flexibility, decreased upper leg strength, increased fat pad size, decreased foot arch height, and increased leg length differences [81]. Extrinsically, increased training—both volume and frequency—is a risk factor for patellar tendinopathy [81]. A macroscopic look at the histopathologic changes shows that tendinopathic tendons are gray/brown instead of white and fragile instead of firm [82].

10.3.1.3 Treatment

Similarly to the lack of consensus on the pathogenesis of patella tendinopathy, there is little consensus on the most effective treatment plan for this disease. The majority of treatments for patella tendinopathy are non-operative. Physical therapy, in particular eccentric training with the addition of decline squats, is the most frequent treatment [83]. Extracorporeal shock wave therapy (ESWT), which involves using high-energy acoustic waves to deliver pressure to the symptomatic area, is also

a possible treatment [84]. A variety of injections are used to treat patellar tendinopathy. Platelet-rich plasma (PRP), aprotinin, sclerosing polidocanol, and steroid injections all show promising results—however, steroids have been shown to be harmful to the tendon in the long run. Operative options include both arthroscopic surgery, involving shaving of the tendon and tendon debridement, and open knee surgery, involving excising the abnormal tissue and drilling the inferior pole of the patella [85]. With no agreement on the correct pathogenesis nor effective treatment plan for this condition, there is much progress to be made in the study of patellar tendinopathy.

10.3.2 Iliotibial Band Syndrome (ITBS)

10.3.2.1 Pathology

Iliotibial band syndrome (ITBS) is a painful condition involving the inflammation of the iliotibial band (IT band) or deep to the IT band. It is typically inflamed by activity that incorporates recurring flexion and extension of the knee and is especially prevalent in long-distance runners. This motion causes friction which can bring about inflammation. The IT band is a band of connective tissue that runs from the proximal end of the tendons of the tensor fasciae latae and gluteus maximus muscles, across the knee joint, and into the patella, tibia, and biceps femoris tendon [86]. When magnetic resonance imaging is done, fluid is often seen in between the IT band and femoral epicondyle. In addition, the distal portion of the IT band appears thicker on an MRI [86]. The reason why some athletes are affected by ITBS and other athletes are not is still unclear. Studies have shown some internal risk factors may be muscle weakness around the knee and hip abductor weakness [87].

10.3.2.2 Diagnosis

Patients with iliotibial band syndrome present with pain along the distal portion of the lateral femoral epicondyle and/or lateral tibial tubercle. Patients are often unable to pinpoint a specific area of discomfort and describe pain spread out over the lateral knee. Pain is present while working out and after working out for most patients. As ITBS worsens, pain can become constant, even while the patient is not being active [86]. Ober's test is used to diagnose ITBS. As the patient lies on their unaffected side with their hips and shoulders in line, the bottom knee and hip are bent to 90°. The affected leg is then lowered, adducting the hip until motion is limited. A positive Ober's test occurs when the patient describes lateral knee pain and cannot fully adduct their hip, which indicates ITBS [88].

10.3.2.3 Treatment

The vast majority of treatments for ITBS are nonoperative and aim to reduce friction between the IT band and the femoral condyle. This is done by minimizing activities that involve repeated extension and flexion of the knee and stretching the iliotibial band, plantar flexors, and hip flexors. Anti-inflammatory medications also help in alleviating pain. If pain and inflammation continue, local corticosteroid injections to the greatest point of discomfort are an option. Once inflammation is reduced, patients begin strengthening the knee and hip extensors and flexors and can progress back toward normal activity. If pain persists, surgery to release the posterior aspect of the IT band over the lateral femoral epicondyle is an option [86].

10.3.3 Lateral Patellofemoral Compression Syndrome (LPCS)

10.3.3.1 Pathology

Lateral patellofemoral compression syndrome (LPCS), also known as patellar compression syndrome or excessive lateral pressure syndrome, is a condition in which overload to the lateral facet of the patella causes pressure and pain in the knee. This is exacerbated by patellofemoral malalignment, which results in a higher Q-angle. Malalignment of the patella causes increased contact between the patella and lateral femoral condyle and increased pressure on the lateral patellar facet when the knee is flexed. A tight lat-

eral retinaculum contributes to lateral pressure in flexion [89].

10.3.3.2 Diagnosis

Patients with LPCS present with localized pain to the inferolateral patella and anterolateral joint line and describe anterior knee pain during both activity and rest. Patients may have limited knee extension and test positive for the "theater sign" of worsened knee pain by prolonged flexion while seated [90]. A clinical exam to diagnose LPCS involves the seated patient attempting to extend and flex their knee. Extension is often limited, and pain heightens as the knee reaches 90 degrees of flexion. If the patella is moved medially and manually centered in the trochlea by the clinician, the patient is frequently relieved of pain and instantly may show a larger pain-free range of motion [89].

10.3.3.3 Treatment

Conservative treatment for LPCS consists of closed-chain strengthening of the muscles of the upper leg, specifically the quadriceps [91]. Open-chain strengthening exercises should be limited to after 30 degrees of knee flexion [92]. Stretching should be done to improve flexibility of the quadriceps, IT band, and hip flexors. Taping the patella medially is an additional technique that can be done. The surgical treatment of LPCS would consist of a simple lateral release to correct excessive patellar tilt; however some patients may also receive a tibial tubercle osteotomy [93].

10.3.4 Plica Syndrome

10.3.4.1 Pathology

Plica syndrome occurs when synovial plica of the knee, most commonly the medial patellar plica, becomes inflamed and thus symptomatic. The mediopatellar plica runs from a supra-patellar origin to the Hoffa fat pad [94]. Plicae are very common and are thought to be the result of mesenchymal tissue from embryological development that is not fully reabsorbed after birth [95]. While most people with plicae are asymptomatic, plica can become aggravated by repetitive knee flexion and extension, blunt trauma, fat pat irritation, twisting injuries, or meniscal injuries [96]. When inflamed, plica thickens and can cause impingement on the femoral condyle. Rarely this can cause chondromalacia by abrading the far medial aspect of the trochlea [97].

10.3.4.2 Diagnosis

Patients with plica syndrome are usually young and present with dull anterior knee pain in the area anterior and medial to the patella. This pain is worsened with knee flexion and increased activity level [94]. Popping can rarely be heard with both extension and flexion [95]. When a clinical examination is performed, patients may express tenderness at the location of the plica, and clinicians may be able to feel the thickened plica as the patient extends and flexes the knee. Imaging is usually done to rule out other causes of anterior knee pain. Arthroscopy may rarely be used to provide a conclusive diagnosis [98].

10.3.4.3 Treatment

Conservative treatment for plica syndrome involves strengthening the quadriceps and stretching the muscles of the upper leg. Lowering activity levels, NSAIDs, and corticosteroid injections also may help in reducing inflammation and pain levels. When non-operative treatment fails, arthroscopy with resection of the entire plica is done.

10.3.5 Chondral Lesions

10.3.5.1 Pathology

Chondral lesions of the patella are the second most common location of cartilage lesions found during knee arthroscopy [99]. Chondral lesions of the patellofemoral joint are caused by acute trauma such as traumatic dislocation or subluxation, impaction such as a dashboard injury microtrauma due to abnormal joint loading, or osteochondritis dissecans [100]. If not treated, such defects affect the normal distribution of PFJ stress and can predispose the patient to the development of PFOA [101]. Of note, the chondral

lesion itself is not pain-generating as cartilage is aneural. Rather, the pain experienced by patients is likely due to subchondral bone overload and synovial and capsular inflammation [102].

10.3.5.2 Diagnosis

Patients typically present with anterior knee pain worsened by activity, occasional swelling, and intermittent locking or catching with knee flexion. Activities that exacerbate pain include prolonged sitting, ascending or descending stair, squatting, and running. Like the physical exam for other causes of anterior knee pain, the patient should first be evaluated for overall varus or valgus alignment and patellar position, as a large Q-angle and patella alta are risk factors for the development of chondral lesions [101]. Tibial torsion, VMO atrophy, patellar tracking, and medial and lateral stability should also be assessed. Crepitus and pain in early flexion may also be appreciated.

While initial radiographic evaluation should begin with plane radiographs, to assess for osteoarthritis, fracture, or other lesions, MRI is considered the gold standard in evaluating chondral pathology.

10.3.5.3 Treatment

Nonsurgical treatment for chondral lesions includes NSAIDs and intra-articular corticosteroid injections, platelet-rich plasma injections, as well as possible hyaluronic acid injections. All such treatment options aim to reduce pain and inflammation. Additional non-operative management includes weight loss, avoidance of painful activities, and strengthening the supporting musculature about the knee.

If patients fail nonsurgical treatment, surgical options to be considered include osteochondral autograft transplantation, autologous chondrocyte implantation, or particulated juvenile cartilage allograft. Restoring cartilage defects in the patella is particularly challenging due to the high loads seen by the PFJ, heterogeneous morphology between patients, and thickness of the patellar cartilage as compared to other articular surfaces in the body. Contraindications to cartilage restoration include PFOA, inflammatory joint disease, and low-grade lesions. If patients also have concurrent patellar malalignment or instability, such abnormalities should be surgically addressed before or during the cartilage restoration procedure. Patellofemoral arthroplasty is an additional surgical option for patients with bipolar chondral defects, but should only be considered as a salvage procedure for those who have failed cartilage restoration procedures.

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