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Review Article

Patellofemoral Pain: Current Concepts

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Abstract

Patellofemoral pain (PFP) is common, especially in women who are physically active. The diagnosis can be challenging, as the etiology of PFP is complex and is unclear. The purpose of this paper is to discuss the current etiological understandings both microscopically and macroscopically and provide an overview for the evaluation and treatment. It is essential to approach each patient presenting with PFP systematically such that the correct causative factors are identified and an appropriate treatment protocol is devised that is tailored to the patient's individual needs.

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- Runner's knee
- Q angle
- TT-TG

ABBREVIATIONS

PFP: Patellofemoral Pain; VMO: Vastus Medialis Obliquus; TT-TG: Tibial Tubercle-Trochlear Groove Distance; Q Angle: Quadriceps Angle

INTRODUCTION

Although patellofemoral pain is a common condition, it remains a complex challenge for the treating clinician. The 4th International Patellofemoral Pain Research Retreat in 2016defined PFP as retropatellar or peripatellar pain "aggravated by at least one activity that loads the patellofemoral joint during weight bearing on a flexed knee", including tasks, such as squatting, running, jumping, or stairs ascending and descending. It was also reported that patellar instability, including recurrent dislocation and subluxation, should be regarded as a separate clinical condition from PFP [1]. This review was performed to provide an overview for the patient presenting with PFP, with the goals of aiding the evaluating clinician in the diagnosis of PFP, the identification of the correct etiological factors, and the prescription of an effective treatment regimen.

EPIDEMIOLOGY

Establishing the accurate incidence of PFP in the general population remains a challenging issue. Callaghan et al. performed a review of studies on PFP from 2000 to 2005 and found that only 40/136 papers cited an incidence or prevalence rate. Of these, the most frequently cited studies were performed in the 1980s and 1990s and were based on military populations and patients presenting to sports medicine clinics [2].

More recently, Glaviano et al. found that using the two ICD-9 codes commonly used to diagnose PFP (717.7-Patella Chondromalacia and 719.46-Pain in joint, lower leg), PFP

represented 7.3% of all diagnoses in orthopedic clinics from 2007-2011. There were higher incidence rates in females and increasing incidence with age, with peak incidence rates in the 50-59 age group [3]. This study is more applicable to the general population than previous epidemiologic studies that were limited to sports medicine clinics or military populations [4-6] and found higher incidence rates among older patients than previously suggested [6]. However, there were still limitations to this study given the lack of a stand-alone ICD-9 code for PFP. In addition, there is difficulty in distinguishing between PFP and patellofemoral arthritis in older patients, and the study utilized insurance data from physicians' offices, which excludes uninsured individuals, young athletes who are treated by athletic trainers, and military personnel with TriCare insurance. This could account for the higher incidence rates among older patients [3]. Given the lack of a stand-alone ICD-10 code for PFP, there will be continued difficulty in establishing the true incidence in the general population.

Etiology

The etiology of PFP is complex and multifactorial in nature. Multiple sources of pain at the cellular level have been identified. In addition, numerous biomechanical abnormalities have been associated with the development of PFP.

At the cellular level

Histologic studies of the knee have suggested both soft tissue and bony origins of PFP. Free nerve endings of nociceptive afferent fibers are found in high quantity in theretinacular tissue, the synovium, and the infrapatellar fat pad [7]. Studies have shown evidence of demyelination and fibrosis of nerves of the lateral retinaculum with increased number of myelinated and unmyelinated nerves with predominantly nociceptive roles in patients with PFP [8,9]. Witonski and Wagrowski-Danielewicz



found that patients with PFP had significantly higher numbers of substance P-containing nerve fibers in the fat pad and medial retinaculum when compared with patients with osteoarthritis or ACL rupture [10]. Bohnsack et al. also found high numbers of substance P nerve fibers in the fat pad and the synovium [11]. While peripatellar synovitis could be a potential source of pain given the rich innervation of synovial tissue, histologic studies have not shown significant changes in patients with PFP [12,13].

The infrapatellar fat pad has received recent attention as a probable source of pain [1,14]. Bennell et al. found that injection of the fat pad with hypertonic saline in asymptomatic patients produced pain[15], and Hodges et al. found that pain induced by injection of the fat pad with hypertonic saline was associated with altered coordination of the quadriceps muscles, with delayed vastus medialis obliquus firing relative to vastus lateralis [16]. This could be a potential explanation for the association of altered quadriceps activity with PFP (discussed below).In addition, studies have shown increased volume of the fat pad in patients presenting with anterior knee pain [14] and patellofemoral osteoarthritis with pain [17].

Bony involvement is also likely. Subchondral bone is richly innervated, and while cartilage is aneural, cartilage degeneration could precipitate pain of subchondral bone with overloading of the patellofemoral joint [18]. Numerous studies have correlated increased patellar intraosseous pressure and metabolic activity with PFP. Schneider et al. found that 40 of 69 adult patients with PFP failing to respond to conservative treatment had a positive "pain provocation test", where increasing patellar intraosseous pressure replicated pain symptoms. In addition, 90% of these patients reported symptom relief over 3 years after the study following intraosseous drilling and decompression [19]. Miltner et al. performed a similar study on an adolescent population with even more convincing results, demonstrating a positive pain provocation test in all 27 participants in the study with statistically significant reduction in intraosseous pressure and pain 1 year post intraosseous decompression [20]. While not every patient in these studies had a positive pain provocation test, it suggests one potential cause of PFP, termed "patellar hypertension syndrome". In addition, there have been studies indicating increased metabolic activity of bone in patients with PFP. Draper et al. performed [18] F NaF PET/CT scans as a method to measure bone metabolic activity based on uptake of the tracer and found that out of 20 knees with chronic PFP, 17 showed increased metabolic activity at the patellofemoral joint: 15 at the patella, 4 at the femoral trochlea, and 2 at both the patella and trochlea. No asymptomatic knees showed increased uptake of tracer [21].

Mechanics

On a macroscopic level, numerous physical abnormalities have been associated with development of PFP, including quadriceps atrophy with vastus medialis obliquus (VMO) deficiency, increased tibial tubercle-trochlear groove distance (TT-TG distance), patella alta, hip abductor weakness, foot pronation, and decreased ankle range of motion. While malalignment and maltracking of the patella are not synonymous alone with PFP, these mechanical issues can certainly predispose to overload at the patellofemoral joint and thus precipitate pain.

Quadriceps atrophy/VMO deficiency

The patella is the largest sesamoid bone in the human body. The quadriceps tendon, formed by the rectus femoris, the vastus lateralis, the vastus intermedius, and the vastus medialis, inserts at the proximal aspect. The patella tendon connects the distal aspect of the patella to the tibial tubercle anchoring the patella inferiorly. The patella functions to increase the moment arm of the patellar tendon, thereby improving the ability to extend the leg [22]. The extensor mechanism of the knee exerts a net lateral moment on the patella given the normal physiologic valgus alignment of the femur and tibia. The vastus medialis obliquus (VMO), the distal obliquely oriented fibers of vastus medialis, plays an important role as an active stabilizer of the patella medially.

Quadriceps atrophy, particularly that of the VMO, has been associated with PFP [23,24]. Jan et al. utilized ultrasonography to evaluate characteristics of the VMO in patients with PFP compared to demographic-matched controls and found more proximal VMO insertion levels on the patella, smaller VMO insertion angles, and reduced VMO volume in the PFP group [25]. Pattyn et al. found similar VMO atrophy utilizing MRI [26]. In addition, electromyographic studies have measured reflex response times in response to patellar tendon tapping and found that while in asymptomatic patients the VMO fired faster than the vastus lateralis, the opposite was found to be true in patients with pain [27]. Despite this association, it is not known whether VMO atrophy is the cause or the effect of PFP. More recently, Giles et al. found that atrophy was not limited to the VMO in patients with PFP, reporting that all portions of the quadriceps muscle were significantly smaller in the symptomatic limb when compared to the asymptomatic limb [28].

Increased TT-TG distance

The quadriceps angle (Q angle) is anatomically measured as the angle formed by a line from the ASIS to the patella, and the patella to the tibial tubercle. While the Q angle has classically been used as a tool to quantify malalignment, its utility has recently been called into question. It has been shown that the static Q angle is not as useful of a predictor of PFP as measurements of dynamic knee valgus [29], the Q angle is not correlated with intensity of PFP [30], and it can be difficult to measure clinically. Given these difficulties, the Q angle has largely been replaced by the radiographic measurement of the tibial tubercle-trochlear groove distance (TT-TG), measured using CT or MRI. The patella articulates in the trochlear groove of the distal femur. As the patella tendon inserts at the tibial tubercle, the horizontal distance between the trochlear groove and the tibial tubercle provides quantification of the lateral force vector on the patella. Studies have shown the TT-TG distance, which is more reliably measured than the Q angle, to be increased in patients with PFP [31,32].

Patella alta

Patella alta, or an abnormally high position of the patella, has additionally been reported as a causative factor [33,34]. Patients with patella alta demonstrate reductions in patellofemoral joint contact area and increased patellofemoral contact stress with physical activity. In addition, this force is increased until the

quadriceps tendon comes into contact with the trochlea with flexion, and this contact time is delayed in patients with patella alta. These findings suggest a mechanical explanation for the association between patella alta and pain [35,36].

Hip abductor weakness

Cichanowski et al. found that female collegiate athletes with PFP had global weakness in hip muscles, most pronounced in the hip abductors and external rotators [37]. When patients with weak hip abductors bear weight on the weak limb, the contra lateral pelvis drops (positive Trendelenberg sign), resulting in the ipsilateral limb collapsing into valgus and contributing to the lateral force vector on the patella. Observe the patient doing a single leg squat for any collapse into a valgus alignment signifying hip weakness.

Distal mechanics

Studies have shown increased foot pronation and rear foot eversion at heel strike as well as delayed rear foot eversion to be associated with PFP [38,39]. Increased rear foot eversion has been associated with increased tibial internal rotation in patients with PFP as well as increased hip adduction in both patients with PFP and asymptomatic patients [40]. While further prospective studies are required to determine whether these are a cause of PFP or a compensatory mechanism, it is suggested that this increased tibial rotation and hip adduction could contribute to dynamic knee valgus and thus loading at the patellofemoral joint [41]. Further, it has been proposed that delayed foot eversion could be a compensatory mechanism to increase mid foot dorsiflexion given findings of reduced range of dorsiflexion in runners with patellofemoral pain [42].

HISTORY

PFP is a clinical diagnosis by exclusion. As such, the history and physical are paramount to evaluation, with emphasized importance over ancillary studies [43]. As reported, patients typically present with diffuse, anterior knee pain, worsening with activities that require flexion on the weight-bearing knee and increased loading of the patellofemoral joint [1]. Post and Fulkerson recommend asking the patient to point with one finger where exactly the pain occurs, and found that the use of knee pain diagrams filled out by patients predicted locations of tenderness on physical exam [44]. However, if a patient places his or her hand over the front of the knee this typically signifies PFP.

As noted, PFP is a diagnosis of exclusion. Differential diagnosis includes Osgood-Schlatter disease, Hoffa's fat pad syndrome, plica syndrome, quadriceps or patellar tendinopathy, traumatic injury, prepatellar bursitis, and patella stress fracture. Other sources of knee pain should be considered and excluded during evaluation.

The pain is typically described as insidious and spontaneous in onset, achy, and worsening with physical activity, especially that requiring flexion of the knee such as stairs or inclines, or start up pain after sitting with the knee flexed (termed the Theater Sign). Pain that is more constant in nature could suggest other etiology, including post-operative neuroma of the infrapatellar branch of the saphenous nerve, reflex sympathetic dystrophy, referred radicular pain, or symptom magnification [43].

As overuse and trauma can significantly contribute to PFP, recent activity changes, weight gain, and recent injury are all vital parts of the history [45].

Physical exam

While there are no physical exam findings that correlate perfectly well with a diagnosis of PFP, certain maneuvers have proven more useful than others. Nunes et al. found in a meta-analysis study that squatting with reproduction of pain was the most sensitive finding for PFP when compared with other findings; however, this finding was still limited in its diagnostic consistency [46]. Other notable findings when compared with control include patellar crepitation and lateral and medial retinacular tenderness, although these lack specificity [47]. By translating the patella to either side, the examiner can palpate the edge and undersurface of the patella and surrounding tissues for sensitivity. As mentioned, other etiologies of anterior knee pain should be excluded first prior to making a diagnosis of PFP.

Imaging

Imaging has a limited role in the initial evaluation of PFP. Standard radiographs including weight-bearing PA, lateral, and sunrise views are useful to exclude other joint pathology and assess patellar tilt. Bone scans, now largely replaced by MRI, could be useful in recalcitrant cases but are not necessary in initial evaluation [48]. Haim et al. found no differences in sulcus angle, patellar height, patellar tilt, or patellar displacement between subjects with pain and control subjects on radiographs. The only finding that was seen more frequently in patients with pain was subluxation of the patella on axial view (25% vs 4% in the control group) [47]. Drew et al. found moderate differences in patellar tilt and patellofemoral contact area, but otherwise found limited evidence to support association of pain with other radiographic findings [49].

TREATMENT

Conservative treatment with physical therapy is recommended for PFP and is effective [50]. Kettunen et al. found no difference in outcomes between patients undergoing athroscopy and an 8 week home exercise program compared with those who only used the home exercise program, despite both groups showing significant improvement [51]. It is essential to identify overuse patterns that could be causing undue stress on the patellofemoral joint and reduce activity levels [52]. Physical therapy should focus on restoration of strength of the extensor mechanism (particularly the vastus medialis obliquus), restoration of normal vastus firing patterns, and improvement of hip abductor/external rotator strength [52]. Patients undergoing hip strengthening before quadriceps strengthening have shown better results than those who initiate with quadriceps strengthening [53]. Physical therapy should be mostly painless to reduce further joint inflammation, but avoiding resistance exercises out of fear of pain will leave the patient stuck in a downward spiral. Quadriceps strengthening should be isometric and gradually progressive to avoid continued overload of the patellofemoral joint [54]. Strength gains take time to occur, so encouragement is essential for motivation. Closed chain exercises in which the foot is affixed to something (e.g. the floor, a pedal, a plate) are preferred because of the co-contraction of muscles in the front and back of the knee, secondarily reducing



shear or posterior translation of the tibia along with the patella. Patella mobilizations with manual translation of the patella in each direction to stretch surrounding tissues can help reduce compressive pressures.

Minimizing extra impact such as stairs, jumping or running helps the pain, especially early in the rehab process. A lateral support brace or patella taping can be a useful adjunct in the early going until the patient can make some strength gains. Evidence exists for immediate relief of pain with medially-directed patella taping, and to a lesser extent bracing, which can facilitate physical therapy and help to gain patient trust [55,56]. Foot orthoses have also shown evidence for short-term reduction in pain, although there is differential response between patients, and greater success is seen in patients with increased rear foot eversion [57], greater mid foot mobility [58], and reduced ankle dorsiflexion range of motion [59]. However, there is limited support for the efficacy of these techniques for long-term management of pain and thus active intervention should be emphasized for more lasting pain relief. Patient education is crucial. It is necessary for the patient to modify activity levels and understand the importance of physical therapy over previously mentioned passive interventions [55].

Surgery is rarely indicated, and should be directed to the specific cause of increased forces on the patella, such as a tibial

tubercle osteotomy for cases of malalignment with elevated TTTG values, or lateral retinacular release or lengthening when tight. Sometimes arthroscopic debridement can be beneficial when unstable cartilage flaps are present, usually with recurrent joint effusions and crepitus. Conservative measures should be exhausted before considering more invasive treatment methods.

CONCLUSIONS

Patellofemoral pain is a common condition. When investigating a patient's pain, it is imperative to understand both the micro and macroscopic factors influencing the symptoms. Without the appropriate understanding evaluation, it is not possible to formulate a treatment protocol for symptom resolution. Due to often vague complaints, a systematic approach to patellofemoral pain will benefit patients and clinicians alike. Physical therapy with quadriceps and hip strengthening are the keys to treatment.

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