CLINICAL MANAGEMENT: PATELLOFEMORAL SYNDROME

Patellofemoral syndrome

S.T. Green*

Judah Street 2717, San Francisco, CA 94122, USA

Received 17 August 2003; received in revised form 1 December 2003; accepted 2 December 2003

Abstract This paper examines the condition of anterior knee pain known as patellofemoral pain syndrome. It describes the functional anatomy and biomechanics of the knee including normal movement, and factors which may destabilize the knee, and predispose it to injury. The controversial “Q” angle is discussed, as is its relationship to patellofemoral pain. The importance of balanced musculature to the integrity of the knee is emphasized as are the potential consequences of weak, or overly tight, muscles. Examination of the knee including patient history, standard orthopedic testing, observation of posture and gait, and palpation are summarized. Treatment protocols for the acute phase, recovery, and maintenance phases of treatment are described. These include gentle mobilization techniques, home exercises, and the occasional use of knee bracing and foot orthotics.

Introduction

Patellofemoral pain syndrome (PFPS) is described in the literature as anterior knee pain, caused by aberrant motion of the patella in the trochlear groove, which results from biochemical and/or physical changes within the patellofemoral joint (PFJ) (Press and Young, 1998; Tria et al., 1992). PFPS exists without gross abnormality of the articular cartilage (Travell and Simons, 1992). It is a problem of chronic overload of the muscles of the lower extremity. Discussion of PFPS often includes associated conditions such as chondromalacia patellae, runners knee, patellofemoral arthralgia, patellalgia, anterior knee pain, and patellar tendinitis (Press and Young, 1998; Kannus and Niittymaki, 1994; Beckman et al., 1989).

A number of conditions have been postulated as causes of PFPS. For instance, it has been proposed that PFPS results from overuse due to a muscular imbalance between the quadriceps and the hamstrings, the tensor fascia latae (TFL) and the gluteus medius, and vastus medialis and vastus lateralis (Liebenson, 1996; Press and Young, 1998). According to Beckman et al. (1989) abnormal length of the hip flexors, hamstrings, quadriceps, and gastrocnemius can affect the PFJ. However, PFPS may also result from a misalignment within the sulcus of the femur leading to oblique lateral tracking, or lateral and medial ligamentous imbalances leading to excessive lateral pressure in the patellofemoral articulation (Tria et al., 1992). It may also be caused by direct trauma to the knee, incorrect seat height, or new shoes. Hammer (1999). Additionally Papagelopoulos and Sim (1997) have proposed that patella alta is associated with PFPS. According to Holmes and Clancy (1998), patella alta causes the patella to enter into the
femoral sulcus late in movement of the knee, increasing lateral tracking of the patella, and contributing to anterior knee pain. Press and Young (1998) also proposed that an increased lumbar lordosis, and increased subtalar pronation or a history of ankle sprains, may contribute to PFPS. These imbalances, and their effect on lower limb kinematics, may cause the patella to move laterally on the knee.

The signs and symptoms of PFPS are: crepitus, and anterior knee pain exacerbated by running, squatting, jumping, or walking down stairs (Hammer, 1999; McConnell, 2002). According to Hammer (1999), PFPS patients may experience pain on full flexion of the knee. Thomee et al. (2002) describe catching or locking at the knee joint, and sensations of stiffness and swelling. McConnell (2002) describes a "buckling" sensation in the knee while walking. Despite this, it is common for patients to give only a vague description of the location of their pain (Press and Young, 1998).

Functional anatomy and biomechanics

The patella is a sesamoid bone. As the knee flexes (the tibia moving on the femur), it articulates with the trochlear region of the femur along the upper two thirds of its posterior surface. During knee flexion the patella moves downward. Two structures hold the patella in vertical relationship. Proximal to the patella is the quadriceps tendon, and distal to it is the patellar ligament. Moore (1995) describes this ligament as a continuation of the quadriceps tendon.

While the primary movement of the knee joint is flexion and extension of the tibia on the femur, rotational movements of the tibia are also normal. As it extends, the tibia rotates externally, and as it flexes, the tibia rotates internally. These movements are controlled by the soft tissue structures of the knee. Damage to these soft tissue structures challenges the integrity of the knee, allows for inappropriate, exaggerated movement at the knee, and affects flexion and extension of the knee (Greenman, 1996).

Increased internal rotation of the femur may disrupt normal knee mechanics causing increased torsion at the PFJ. According to Press and Young (1998), internal rotation may be caused by a tight TFL and a weak gluteus medius and piriformis. However, Hoppenfeld (1972) also suggests the cause may be associated with femoral neck anteversion.

Fibers from the iliotibial band (ITB) and vastus lateralis stabilize the patella laterally. According to Zappala et al. (1992) and Press and Young (1998), fibers of the vastus medialis (the primary medial dynamic stabilizer), and adductor magnus stabilize the patella medially. The adductor magnus does not attach directly to the patella. However, Zappala et al. (1992) argues that it acts as a patellar stabilizer because its fibers attach to the patellar retinaculum, which attaches to the femoral condyles and capsulomeniscal tissues. Press and Young (1998) describes the adductor magnus tendons as the insertion for the vastus medialis obliquus (VMO), and therefore a critical part of its stabilizing force. Both passive and dynamic stabilizers affect function. Passive stabilizers such as the retinaculum, and the shape of the patella itself, along with the dynamic influence of the quadriceps femoris, ITB, adductor magnus and longus, pes anserine, biceps femoris, and VMO, allow for proper patellar tracking (Tang et al., 2001; Press and Young, 1998). The VMO, which is the designation of the distal obliquely angled fibers of the vastus medialis, keeps the patella in alignment in the femoral sulcus (Zappala et al., 1992; McConnell, 2002). The vastus lateralis, intermedialis, medialis and rectus femoris act on the patella to extend the knee. This action is known as the extensor mechanism (see Fig. 1).

According to Hammer (1999), there are two important biomechanical functions of the PFJ: lengthening the lever arm of the quadriceps muscle, and increasing the area of contact between the femur and the patellar tendon. Lengthening of the lever arm allows for increased mechanical advantage of the quadriceps. Increasing the contact area, allows for greater distribution of compressive strength.

The literature frequently refers to the "Q" angle (quadriceps angle) which is formed by the intersection of a line extending from the ASIS, through the midpoint of the patella, and another line from the tibial tubercle, through the midpoint of the patella as shown in Fig. 2. This angle represents the line of pull of the quadriceps and patellar tendons. The angle will vary with foot pronation and supination (increased with hyperpronation), and therefore, may be best measured standing without shoes (Holmes and Clancy, 1998). Women tend to have greater "Q" angles than men due to differences in pelvic shape. The normal angle for men is about 10° and the normal angle for women averages about 15°. This difference is often given as the rationale for the tendency of women to have a higher incidence of PFPS than
men. However, there is no consensus in the literature regarding the functional importance of the "Q" angle. An increased "Q" angle is often described as an important factor predisposing patients to patellar tracking problems, because the patella tends to track more laterally as the angle increases (Chaitow, 2002; Tria et al., 1992). McConnell (2002) sites external tibial torsion, increased femoral anteversion, and lateral displacement of the tibial tubercle as causes for increased "Q" angles. As the "Q" angle is a static measurement, it may have little bearing on the actual function of the knee (McConnel, 2002; Tang et al., 2001). In a small study of 10 subjects diagnosed with PFPS and 10 healthy, non-symptomatic volunteers, Tang et al. (2001) found that "Q" angle did not vary significantly between the asymptomatic and the symptomatic subjects. Post et al. (2002) described several recent studies of the relationship of "Q" angle to PFPS, concluding that there is little relationship between "Q" angle measurements and patient complaints, and that it is of "uncertain and limited clinical value".

Pathophysiology

Articular cartilage is not pain sensitive tissue (Papagelopoulos and Sim, 1997; Beckman et al., 1989). Papagelopoulos and Sim (1997) suggest that PF pain is produced when cartilaginous lesions irritate the highly innervated synovium. McConnell (2002) describes PF pain as "most likely caused by either tension or compression of the soft-tissue structures".

Little objective data exist in the literature regarding PFPS. However, many clinicians propose that tightness of the adjoining tissues to the patella significantly affect its function (Beckman et al., 1989, Liebenson, 1996; Press and Young, 1998; Hammer, 1999; McConnell, 2002). For instance, Zappala et al. (1992), suggest that a tight rectus femoris may prevent full inferior excursion of the patella. Tight ITB may force the patella into lateral tracking. Tight hamstrings or triceps surae (gastrocnemius-soleus) may restrict talocrural dorsiflexion promoting subtalar pronation, as well as causing increased knee flexion (Beckman et al., 1989). As the knee flexes, compression of the patella on the femur increases.

In addition, lower limb kinematics have been proposed to influence patellofemoral biomechanics.
Hyperpronation of the subtalar joint may be quite significant in PFPS: the greater the amount of pronation in this joint, the greater the internal rotation of the tibia. This in turn increases stress on the periarticular soft tissues surrounding the knee, which can lead to an increase in knee pain.

**Differential diagnosis**

Pain and dysfunction of the anterior knee may be caused by several conditions. PFPS must be differentiated from fibobifular subluxation, Osgood-Schlatter disease, patellofemoral arthritis, plica syndrome, patellar tendinitis, bone tumors, or bursitis. Box 1 delineates these conditions.

**Box 1 Differential Diagnosis**

- **Tibiofibular subluxation:** Palpatory pain at the fibular head. Leg "gives way" with one leg stance at 30°. There may be swelling of the lateral or medial structures. Positive apprehension sign, probable pain on resisted knee extension, increase of laxity in the joint (Hammer, 1999).

- **Osgood-Schlatter disease:** Inflammation of the tibial tubercle, primarily found in pubescent athletes. Complaints include gradual onset pain localized at the patella or at the tibial tubercle with resisted knee extension. Painful and limited flexion. Pain on resisted knee extension. (Hammer, 1999). Increased "Q" angle and patella alta may be associated with Osgood-Schlatter disease (Thabit and Micheli, 1992).

- **Patellofemoral arthritis:** Difficulty using stairs, squatting, with symptoms worse at the end of the day. Limited and painful flexion, crepitis, tight hamstrings, loss of joint play, may have deformity present. May have positive Lachman’s or anterior drawer tests (Chaitow, 2002). Test blood for elevated ESR.

- **Patellar tendinitis:** Inflammation where quadriceps attaches to the patella. Pain is localized and often burning, aggravated by activity, better with rest. To test, pain is likely present on knee extension (Hammer, 1999).

- **Pesch anserine Bursitis:** Swelling at medial knee, inferior joint space, localized tenderness. Restricted internal or external rotation with pain. Restricted flexion. Contraction of the semitendinosus, sartorius, and gracilis will aggravate bursitis (Chaitow, 2002).


- **Bone tumor:** Constant unilateral pain. Diagnose using radiographic findings, MRI.

Useful orthopedic tests for the diagnosis of PFPS include: compression, patellar inhibition, Wladron’s, apprehension signs, and retinaculum tests.

**Functional examination**

The goal of the examination is to devise a functional road map, which will guide the practitioner and patient through the treatment process. This is accomplished by discovering the key links, which cause the patellar dysfunction and pain. The first step is a thorough history, which includes:

- Prior injuries throughout the kinetic chain
- Surgeries
- Fractures
- Family history of knee pain
- Prior use of knee bracings
- Description of current complaint
  - Location of pain
  - Initial onset
  - Aggravating activity
  - Activity intolerances

Observation of posture should rule out the following:

- Subtalar pronation
- Genuvarum, valgum or recurvatum of the knee
- Lateral or medial shift of patella
- Prominent ITB
- Anterior pelvic tilt with increased lumbar lordosis
- Forward drawn posture

Gait analysis should include evaluation of movement throughout the entire kinetic chain. Palpation of specific structures can determine signs of inflammation, patellar alignment, restriction of the fibular head, muscle tightness, and active hamstring trigger points. Quadriceps should be checked bilaterally for size and strength.
Observation of hip abduction and hip extension movement patterns, as described by Janda (1996), helps to identify muscle weakness or over activity. An altered hip abduction pattern reveals weakness of the gluteus medius, and compensatory activation of synergists such as TFL (Janda, 1996), quadratus lumborum or piriformis. The hip extension test can be used to help determine delayed activation and possible weakness of gluteus maximus, early recruitment and possible activity in erector spinae or hamstrings, or tightness of anterior hip structures. Using a weight-bearing test, involving one legged standing, further assessment can be performed to evaluate the strength of gluteus medius (Liebenson, 1996). Janda (1996) described a modified Thomas’ test that can identify shortened or over-active TFL, rectus femoris, or psoas muscles.

In the author’s experience, it is very common to find a weak gluteus medius and tight TFL, with concomitant over-active, or shortened, ITB in patients with PFPS. Clinical experience suggests that this often leads to a tightening of the lateral patellar retinaculum. Clinically patients with PFPS may present with failed hip abduction and hip extension tests, medial hip rotation at mid-stance in gait, and a tendency to rotate and adduct the femur medially when walking down stairs. It is also common to find a weak VMO, allowing for a lateral pull on the patella. Hyperpronation of the subtalar joint causes internal rotation at the knee, which results in further lateral pull on the patella. Hyperpronation often results from tight gastrocnemius, as explained previously.

Examination for aberrant patellar movement is achieved using the Patellar Glide Test. This test may indicate medial or lateral retinacular tightness and abnormal patellar movement. If a rise at the medial or lateral border accompanies the glide, a patellar tilt is also present.

**Method:** The test is performed with the patient either sitting or supine. The quadriceps is relaxed and the knee is supported at 20–30° of flexion. The patient is asked to tighten the thigh. A quick lateral or medial movement of the patella indicates a positive sign. Insufficient medial restraint is demonstrated by excessive lateral glide, while a hypermobile patella is suggested with excessive medial glide (Zappala et al., 1992).

**Treatment**

The treatment regime offered below is based on the clinical experience of the author, along with a collation of information from the literature (Lewit, 1965; Beckman et al., 1989; Zappala et al., 1992; Press and Young, 1998; Hammer, 1999; Liebenson, 1996; McConnell, 2002; Chaitow, 2002).

**Acute phase treatment**

In most cases, PFPS is the result of chronic overload and not acute trauma. However, when trauma is the instigating cause, initial, acute treatment may last up to 7 days requiring relative rest, elevation of the knee, and use of proteolytic enzymes or NSAIDS. Modalities such as ice, pulsed ultrasound, and micro-electric therapy can also be used to reduce inflammation. Very gentle mobilizations may be performed. Avoid activities known to aggravate the condition. Climbing, squatting, kneeling and/or jumping should be avoided whenever possible.

During the acute stage, limited range of motion exercises, exercises for the upper extremities, and exercises for the opposite leg should be continued. Zappala et al. (1992) recommend stationary bicycle exercises keeping the affected leg on the ground as shown in Fig. 3.

**Figure 3** The affected left leg remains on the floor, as the healthy right leg exercises on the stationary bicycle.
Recovery phase treatment

As inflammation subsides, rehabilitation becomes more active. Initially exercises should be performed in a closed kinetic chain position (feet maintaining contact with the ground or other solid surface) whenever possible (Tang et al., 2001; Press and Young, 1998). In a closed chain position, the hamstrings and quadriceps experience balanced co-activation essential for weight-bearing activities. In open chain exercises (feet moving freely through space), there is an unnecessary stress on the PFJ due to the isolated contraction of the quadriceps in extension and hamstrings in flexion (Press and Youngs, 1998).

Bracing and taping are frequently used in the treatment of PFPS. There is little agreement in the literature regarding their benefit. Over prolonged periods, bracing can lead to atrophy in the quadriceps, and should be avoided (Tria et al., 1992). Zappala et al. (1992) argue that if it must be used, a brace should allow for the free movement of the patella by utilizing a patellar cut out. This also avoids direct pressure to the patella. Press and Young (1998) and others utilize McConnell taping to correct patellar glide, tilt, and rotation (McConnell, 2002). The most effective use of bracing is as an aid during exercise. Figure 4 shows an example of McConnell taping. In cases of excessive subtalar pronation, it may be useful to use foot orthotics.

Normal patellofemoral function requires balanced postural and phasic muscle activity, as well as full joint mobility of the related bony articulations of the lower extremity and pelvis. Relaxing and stretching tight and shortened postural muscles, as well as strengthening the appropriate weaker, inhibited phasic muscles, are keys to rehabilitation of the knee. In the author’s clinic, treatment includes both manual techniques mobilizing the joints and soft tissues, and individualized home exercise programs.

Tight or shortened muscles may include the TFL/ITB, hamstrings, quadriceps, triceps surae, and hip flexors. If they are tight, or if there are trigger points present, treatment involving post-isometric relaxation (PIR), as described by Lewit (1965) is used. This is a very gentle technique whose success depends on the practitioner’s ability to discern the initial restriction barrier. Figure 5 shows PIR of the gastrocnemius muscle. PNF or myofascial techniques are used to lengthen shortened muscles.

Tight or shortened TFL/ITB is a common finding in cases of PFPS. The most effective procedures used in the author’s clinic for the TFL/ITB are described below:

- For relaxation, PIR is used in the modified Thomas position (Liebenson, 1996).

![Figure 4 McConnell taping of the right knee. By taping from lateral to medial, the medial structures are supported.](image)

![Figure 5 PIR of the gastrocnemius muscle. Patient is supine with legs extended, hips neutral. Practitioner passively dorsiflexes patient’s foot until initial resistance (barrier) is felt. Maintain calcaneal distraction. Patient inhales and gently attempts to plantarflex foot against practitioner’s hand with minimal isometric contraction. Hold 5–10 s. After patient relaxes and exhales, practitioner takes up the slack. Repeat procedure three times.](image)
To stretch the muscle, a myofascial technique is applied, in conjunction with a home treatment using a foam roller (Fig. 6).

Restricted movement of the patella on the femur often requires gentle mobilization. Two such mobilizations which have been found to be useful are:

1. Gentle mobilization as shown in Fig. 7.
   - Patient is supine with a small towel roll under the affected knee.
   - The patella is gently moved in all directions to find restriction.
   - With a double hand contact, the patella is then gently mobilized in the direction of the restriction.

2. Lewit’s (1965) Gapping Technique may also be performed.
   - For medial gapping, as shown in Fig. 8, the patient is supine with the involved leg stretched, but not brought to full extension.
   - One of the practitioner’s hands grips the patient’s ankle, lifting it slightly from the table.
   - The other hand exerts slight pressure at the level of the joint space, taking up the slack, and finding the point of initial resistance (barrier).
   - The practitioner then springs the joint medially several times.
• To perform lateral gapping, the practitioner sits on the table, between the patient’s legs, facing the knee joint, and palpates the joint to initial resistance.
• The practitioner then gently springs the joint laterally several times.

VMO weakness is a typical finding in PFPS. It is difficult to isolate this muscle because of its attachment to the tendons of the adductor magnus and longus. Zappala et al. (1992) suggest therefore, that hip adduction may significantly activate the VMO while leaving the rest of the quadriceps at relative rest. Early in treatment, exercising of the VMO is started by performance of a patellar self-glide (Fig. 9). This exercise progresses (without the medial self-glide) to being performed while standing, once the patient has the ability to isolate the VMO. The glide is held for 5–10 s and this is repeated 5–10 times, several times a day.

A second exercise, which focuses on the VMO and adductors, is shown in Fig. 10. This exercise may also be progressed to being performed while standing, as the patient gains strength and control.

Resistance band and tubing exercises allow for progressive strengthening of specific muscles (Hammer, 1999). In Fig. 11, the VMO is strengthened by attaching one end of the tubing just above the knee and another to the door jam. The patient stands with feet shoulder-width apart, facing away from the door and is asked to externally rotate the leg and bend the knee (keeping the foot on the floor) and to then straighten the leg to full extension.

Gluteus medius and maximus are often weak in PFPS. To train weak or inhibited gluteus maximus,

**Figure 11** VMO strengthening using resistance tubing.

**Figure 12** Gluteus medius activation using the exercise ball.

the following methods have been found to be useful (Liebenson, 1996):

• Posterior pelvic tilts.
• Bridges: progressing from the floor to the gym ball.
Quadruped single leg raises.
Rocker board, wobble board, balance shoes.
Shallow squats on the exercise ball.

To train weak or inhibited gluteus medius the following methods may be useful (Liebenson, 1996):

Quadruped single leg raises.
Rocker board, wobble board, balance shoe progressions.
One leg bridges (after patient can successfully perform bridge with both feet on the floor).

The gluteus medius is further facilitated in the exercise shown in Fig. 12.

Patient stands with the exercise ball between patient and door.
The height of the ball allows the patient’s arm to comfortably rest on it without raising the shoulder.
The leg closest to the ball is flexed at the knee with the foot off the ground.
All the weight is on the involved leg, which is slightly flexed.
The foot faces directly forward.
The leg is externally rotated, while keeping the foot, pelvis, and shoulders in a forward direction.
The buttock is slightly contracted
This exercise is similar to a McConnell exercise (McConnell, 2002), however the ball is preferred as it provides added support to the patient.

Small foot exercises, as taught by Janda, stimulate sensory motor pathways, which control the function of the muscles responsible for posture and gait. For a description of these exercises, refer to Liebenson (1996). If patients have difficulty in successfully completing the small foot exercise they are asked to stand with feet facing forward, and using a minimal movement, to externally rotate the knees, activating the longitudinal and transverse arches, while keeping their toes on the floor. This exercise can be repeated frequently during day.

**Maintenance phase treatment**

As the patient gains strength, coordination, and is able to perform exercises with no pain, eccentric exercises such as the step up (Fig. 13), may be introduced (Hammer, 1999). Leg presses and elliptical walking may also be added to the program at this time, as well as exercises with an activity specific focus.

**Conclusion**

The goal of rehabilitation of PFPS is to reestablish the functional integrity of the affected lower extremity. Each patient presents with a slightly different set of positive findings. Therefore, specific, individualized treatment for each patient must be found. To speed recovery, it is crucial to find the key elements of the patient’s dysfunction.

Exercises need to be progressed in a steady way to challenge the patient. Without inducing fatigue, so that they are able to maintain proper motor control. In doing so, the entire locomotor system can normalize.

**Appendix.**

Fig. 14

As training progresses, the patient regains motor control and prepares to return to job or sporting activities. The author has observed that traditional exercise programs, which concentrate on sagittal plane movements only, do not sufficiently prepare patients for successful reentry. Training utilizing sagittal, frontal and transverse planes creates a functional pattern allowing a patient to progress into their normal three-dimensional activities (Gray, 2001; Liebenson, 2003).
Figure 14 Overview of possible treatments for PFPS.

References

