Patellofemoral Pain Syndrome: A Review of the Literature with Osteopathic Emphasis

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Abstract

Patellofemoral pain syndrome (PFPS) is a common yet complicated clinical presentation. The first objective of this paper is to review the literature related to the pathogenesis and diagnostic testing for evaluating for PFPS. The second objective is to investigate how the five models of osteopathic medicine relate to the development and treatment of PFPS. The last objective is to outline several effective osteopathic manipulative techniques that can aid in the treatment of PFPS.

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Disclosures: no conflicts of interest to disclose.

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Submitted for publication August 4, 2020; manuscript accepted for publication June 1, 2021; revised manuscript received July 16, 2021.

Keywords: patellofemoral pain syndrome, PFPS, runner's knee, anterior knee pain, chondromalacia patella, patella pain.

Fictional Clinical Vignette

A 35-year-old male runner presents to your primary care clinic with a three-week history of right anterior knee pain. The pain is moderate in severity and described as aching or sharp at times. The pain worsens with using stairs or if he gets up from sitting for a long period of time. Occasional ice application to the knee alleviates the pain. There was no known trauma or injury that occurred at the onset of these symptoms. He is a recreational runner and recently started trail running with new shoes. However, there was no change in frequency, intensity, or duration of his running. He denies any swelling, locking, or catching of the knee. He denies any numbness, tingling or weakness. He reports a past medical history of Achilles tendonitis. He has had no previous surgeries. Family history is non-contributory. He is an accountant primarily stationed at a desk throughout the day. He has tried occasional ibuprofen with some relief and does not take other medications. He has no known drug allergies. He denies fever, chills, back pain, skin rashes, erythema, and swelling. The remaining review of systems is unremarkable.

Physical examination reveals lateral patella tracking on the right knee but otherwise no obvious deformities. There is no effusion, erythema, or ecchymosis appreciated. He is tender on the superior lateral pole of the patella. The knee exhibits normal knee flexion and extension and is neurovascularly intact. He has a negative patellar grind, Lachman's, anterior drawer, posterior drawer, McMurray's tests, and there is no laxity with valgus or varus stress. He has a positive Ober test on the right. Osteopathic evaluation reveals the following somatic dysfunctions: $L4ER_RS_R$, right innominate outflare, hypertonic quadratus lumborum and tight iliotibial band on the right, right anterior talus, right posterior fibular head, and a right on left sacral torsion.

Introduction

PFPS is a frequent diagnosis in both family and sports medicine clinics. The terms "chondromalacia patella, anterior knee pain and/or syndrome, and runner's knee"[1] are often used as synonyms. PFPS is described as pain around or behind the patella that increases with intensity with certain movements or exercises.[1] These activities include squatting, climbing stairs, jogging, running, jumping, extending the knee, or standing up after sitting for an extended period, and other weight bearing activities.[1,2,3]

Epidemiology

The prevalence of PFPS is between 7% and 28% and is seen more commonly in younger populations, between the ages of 12 and 17.[4,5,6] The incidence and likelihood of developing PFPS is 2-3 times higher in females than males.[3,5,7] Myer et al found an incidence of 9.2% in young female athletes.[4] A retrospective study of 30 million orthopedic patients between the years 2007-2011 found an incidence of 6% in the patient population.[8]

Pathogenesis

The pathogenesis of PFPS is diverse. When evaluating for PFPS, it is imperative to investigate other etiologies of anterior knee pain. These would include bone tumors, Osgood-Schlatter disease, patellar stress fracture, pes anserine or prepatellar bursitis, and quadriceps tendinopathy, to name a few.[5,9]

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Normally, the patella is situated in the quadriceps tendon and courses through an S-shaped path on the trochlea of the femur when the knee goes into extension. The wedge or V shape of the undersurface of the patella and the elevated lateral epicondyle help the patella stay in the trochlea.[3,10,11] From a structural perspective, there are both biomechanical dysfunctions and other tissue imbalances that lead to the patella not being able to follow the femoral trochlea appropriately in knee extension. These factors lead to lateral tracking, increased joint stress, and damage to the joint.[3]

Risk factors that contribute to developing PFPS include extrinsic factors such as overuse injuries, sudden increases in exercise or work, or ill-suited shoes.[3] Intrinsic risk factors for PFPS will be discussed individually, along with their implications in the development of PFPS.

One of the many intrinsic factors to consider is iliotibial (IT) band tightness. This tightness leads to improper tracking since the lateral retinaculum of the knee has fibers that originate from the IT band. Increased tightness and pull from the IT band through the lateral retinaculum increases stress on the patella and the patellofemoral joint. The stress causes a pulling of the patella laterally across the joint, resulting in adverse patellar tracking, subsequent pain, and damage.[6,7,10]

Another important intrinsic factor is weakness of the quadriceps muscle, specifically of the vastus medialis. The weakened superomedial pull of the patella via the quadriceps tendon leaves the patella to move more freely in the lateral direction.[5] Conversely, increased quadriceps muscle tightness can bring the patella into increased contact with the femur, causing increased joint stress and injury.[5,10]

Boling et al mention other medial stabilizers such as the medial patellofemoral ligament (the most influential structure to prevent lateral movement),[12] medial meniscopatellar ligament, medial retinaculum, and the medial collateral ligament.[6] If injured, the lateral pull on the patella through the lateral patellofemoral ligament, lateral retinaculum, and IT band, as mentioned above, can lead to the abnormal lateral tracking.[3,6]

An additional intrinsic factor to examine is the Q angle. An increased Q angle (more than 10-13 degrees in males and more than 15-17 degrees in females) leads to genu valgum, resulting in increased lateral forces on the patella like a "bow string effect."[4,6,7] Excessive foot pronation can result in increased tibial internal rotation. The increased tibial internal rotation prevents the knee from locking during the gait cycle, leading to compensatory femur internal rotation during the gait cycle. Internal rotation of both the tibia and femur lead to increased lateral stress on the patella, increased Q angle, and increased joint stress between the patella and femur, which also leads to joint damage and pain.[3,5,10,13]

Zarei et al have also investigated how quadratus lumborum and gluteus medius and their associated myofascial trigger points play into the pathophysiology of PFPS. Other studies showing these core muscles have a role in PFPS, and that trigger points in these muscles have a higher prevalence in people with PFPS.[14] Both muscles help stabilize the pelvis and the trunk, as well as limit hip adduction during the gait cycle. Inappropriate muscle activation and myofascial trigger points affect the muscle's ability to function properly, leading to improper alignment of the pelvis and acetabular joint, causing increased stress and damage of the patellofemoral joint.[15] Other intrinsic risk factors include, but are not limited to, weakness in other gluteal muscles, hip, pelvic, and sacral somatic dysfunctions, lateral patellar tilt, and hamstring muscle tightness.[1,3,5,6,7]

The loss of tissue homeostasis is an intrinsic risk factor that is of great importance to the osteopathic physician. The aspect of tissue imbalances is discussed in the work of Scott F. Dye, MD.[16] Dye stresses that the homeostasis of the innervated muscles, fat pads, and other tissue/synovial structures have a greater role than the structural issues found in the disease process. Fat pads such as Hoffa's, quadriceps, and the pre-femoral fat pads are richly innervated and can become irritated with acute high loading and repetitive loading conditions. Loading conditions can be "a single loading event of sufficient magnitude or a series of repetitive loading events of a lesser magnitude" causing inflammation and metabolic disturbances of biochemical processes.[16] The combination of these factors leads to tissue "homeostasis loss." Dye defines homeostasis as "the maintenance of consistent conditions such as blood glucose, serum calcium, and other physiologic processes in living cells." The loss of these conditions in the muscles and tissue surrounding the patella leads to anterior knee pain and the development of PFPS.[16,17]

Dye used technetium scintigraphy to investigate the metabolic activity of the tissue and muscles surrounding the knee. He found an increase in dye accumulation in patients suffering from anterior knee pain indicating increased metabolic activity of the tissues. After non-surgical treatment (rest, NSAID use, and/or rehabilitation) and the patient's pain symptoms being resolved, Dye reperformed the scan and found less dye accumulation in the knee area even though the Q angle and other structural features had not changed.[16] This finding is consistent with some recent literature suggesting the Q angle may not be as significant a risk factor for PFPS as once thought.[3]

Based on the proceeding information, PFPS is not a simple clinical picture. Patients can present with many or some of these factors/symptoms, and some might not present with any structural malalignment at all. Simply evaluating and treating structural causes and symptoms might not be applicable for each patient. The osteopathic physician is trained to identify tissue texture abnormalities strain of the tissue by palpation. By restoring homeostasis and structural alignment, osteopathic physicians can diagnose and treat PFPS quickly, accurately, and non-invasively.

Diagnosis

Proper diagnosis is obtained by performing a thorough history of present illness and physical examination. The five key components of a thorough musculoskeletal exam are inspection, palpation, range of motion/strength testing, neurovascular evaluation, and special testing. A sixth component is including an osteopathic examination.

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The exam begins with inspection of the knee for any gross deformities, erythema, swelling, or any other asymmetry in the patella or muscles surrounding it.

The second portion of the exam is palpation. Identified areas to palpate along the proximal knee includes the quadriceps tendon. Areas for examination along the lateral knee include the lateral retinaculum, attachment of the vastus lateralis and iliotibial band. Areas associated with the medial knee are the medial retinaculum and attachment of the vastus medialis. It is prudent to pay close attention to the distal knee which includes the patellar tendon, its attachment at the tibial tubercle, and the fat pads and bursa around the knee.[9]

The third portion of the exam is range of motion (ROM) and strength testing. Assessment of tight hamstring muscles should be completed, as they could increase forces on the joint during exercise. Besier et al demonstrated that patients with PFPS had greater co-contraction of quadriceps and hamstrings and greater normalized muscle forces during walking, even though the net knee moment was like the control group.[18] Passive knee extension or passive hip flexion with knee extension could be performed to assess for hamstring tightness. Hip, knee, and ankle ROM should be assessed and may be contributing to the knee pain.

The fourth portion of the exam is a neurovascular evaluation. This includes checking posterior tibial and dorsalis pedis pulses, light touch and pin prick sensation, and deep tendon reflexes.

The fifth portion of the exam is special testing. A good test to utilize for PFPS is having the patient perform a squat. The test seems to provide high reliability when it is used to test for anterior knee pain. PFPS is evident in 80% of people who are positive on this test.[19] Other tests or symptoms that can correlate with PFPS are a positive patellar grind test, a positive patellar glide test, and/or a J sign due to lateral patella tracking on knee extension, crepitus, or popping. However, Crossley et al noted that while popular, the patellar grinding and apprehension tests (e.g., Clarke's test) have low sensitivity and limited diagnostic accuracy for PFPS.[1] Other tests that can be done to assess knee stability are the valgus/varus stress tests, the anterior/posterior drawer tests, and the Lachman test. A single perfect test does not exist. Experienced clinician expertise, combined with appropriate neuromuscular examination including orthopedic special tests, may be prudent for best diagnostic confirmation.

Diagnostic imaging is not usually required unless there is a history of trauma, the patient is over 50 years of age, or pain is refractory to treatment,[9] in which case imaging may be useful to rule out other etiologies such as fracture, osteochondritis, osteoarthritis, bone tumors or other organic pathology.

Osteopathic Considerations in PFPS

The sixth component of the physical examination is an osteopathic examination. This is guided by using the five models of Patient Centered Osteopathic Care. The five models are the metabolic, neurologic, behavioral, biomechanical, and respiratory/circulatory models. Each model has a specific role in the development in PFPS; the metabolic and neurologic models are discussed in Dye's approach to PFPS.[16]

The behavioral model was evaluated by looking at psychological factors such as "pain-related fear, anxiety, depression, catastrophizing, and self-efficacy."[21] Maclachlan et al found that these factors can be elevated in individuals with PFPS and are related to pain levels and lower physical function.[21] Even if not all patients experience these symptoms, it is still pertinent to ask each patient how they are coping with their PFPS, and the limitations associated with their condition. It is also imperative to evaluate the patient's response to treatment of their psychosocial factors, whether that be through medications, counseling, or both.

Based on the etiology and pathophysiology of PFPS, a systematic approach to treating the somatic dysfunctions with osteopathic manipulative medicine is essential and can be approached using the biomechanical model. The osteopathic physician should diagnose and treat somatic dysfunctions that restore normal biomechanics of the lumbosacral, hip, pelvis, sacroiliac, knee, and ankle. It is one of the authors' experience, typically, to restore related dysfunctions of the axial spine prior to normalizing dysfunctions of the extremities; treating lumbar and sacral somatic dysfunctions would initiate treatment.

In fact, there is evidence that manipulation to the SI joint proves beneficial. In a study by Suter and McMorland, a high-velocity low-amplitude (HVLA) thrust was applied to the SI joint on the side of the most affected knee. They observed increased muscle strength and decreased muscle inhibition of the quadriceps after treatment.[22] Dry needling directed at the quadratus lumborum and gluteus medius have proven effecting at relieving knee pain.[15] The success of this treatment modality should be embraced by osteopathic physicians for patients with PFPS. Osteopathic physicians that are not trained in dry needling can consider counterstrain treatments for the quadratus lumborum and gluteus medius.[23] Innominate dysfunctions are traditionally treated with muscle energy (MET), but one could utilize HVLA techniques as well.[24,25] It is critical to assess for hamstring dysfunctions, as this can potentially affect the innominate rotations. As discussed previously, tibial torsions can be a factor in PFPS and can be treated osteopathically with various techniques such as Balanced Ligamentous Tension (BLT).[25] The iliotibial band is commonly tight and can be released using soft tissue techniques.[25] The hip capsule fascia can be treated directly or indirectly using myofascial release or METs as well.[26] Treating talus, gastrocnemius and soleus dysfunctions are important in restoring proper foot mechanics. Runners with PFPS may have excessive pronation at the subtalar joint, which can lead to the fibular head is forced posteriorly. Somatic dysfunction of the fibula can be treated with METs or other modalities.[25]

To address the respiratory/circulatory model, the physician can assess for lower extremity swelling/edema along with popliteal and inguinal fullness. If there is fullness/edema noted on examination, first begin with thoracic inlet release.[20,25] Additional lymphatic techniques for the lower extremity that can be utilized are the Popliteal Fossa Release and Pedal Pump of Dalrymple.[25] By

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improving lymphatic and congested blood flow, clearance of harmful metabolites and inflammatory cytokines is promoted.

In addition to osteopathic manipulative treatment, rehabilitation exercises through structured physical therapy should be employed. These exercises help bring proper balance and alignment between the vastus lateralis and medialis muscles, with specific emphasis of retraining the vastus medialis. Correct foot biomechanics should be achieved, if possible, for the patient to help reduce further biomechanical stress. A multifaceted approach to treatment with reducing the inflammation of the patella-femoral mechanics, utilizing patella taping or braces, restoring proper biomechanics, and vastus medialis strengthening are essential for a patient with PFPS. Rarely, a patient may require retinaculum release and it is an option for patients that are refractory to conservative treatment. Since all five models are involved in the treatment and pathogenesis of PFPS, it is imperative that the osteopathic physician evaluate for and address all five models to treat the patient in a holistic manner (or consistent with the Osteopathic Patient Centered model of care).

Conclusion

The etiology and pathophysiology related to PFPS is diverse. Because of this, a careful history and physical of the patient is most important to achieving a definitive diagnosis related to the patient with PFPS. Symptoms can vary greatly from patient to patient. Symptomatology plus context of injury is needed to help narrow down a differential. Emerging trends of our understanding of PFPS demonstrate misalignment between the vastus lateralis and medialis, quadriceps, and hamstring tightness. An osteopathic approach to treatment should include review of the five models and incorporation into patient care. Future research should focus on evaluating for and incorporating treatment with all five models and measuring outcomes and comparing the effectiveness of each model to each other through randomized studies with an OMT perspective.

Acknowledgement

The resources used to research this topic listed below are textbooks which can be found on ClinicalKey, LWW, as well as in print.

Additional sources listed below were found thanks to PubMed, Google Scholar, and various journal libraries.

Thank you, Michael D. Lockwood DO, FCA, FAAO for your grammatical contribution and clinical acumen for this paper.

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