Review Article

An overview of patellofemoral pain—from a tissue homeostasis perspective

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Abstract: The etiology of patellofemoral pain (PFP) has been misunderstood for decades, leading to the broad acceptance of therapies that are not only ineffective, but often harmful. The solution to the problem of PFP eventually came from the discovery and development of a new musculoskeletal paradigm: tissue/joint homeostasis. This new conceptualization of the cause of anterior knee pain (AKP) looks beyond the mere structural, anatomic and biomechanical factors, deeper into the underlying perturbed biology. Loss and restoration of tissue homeostasis of innervated PF components such as synovium and bone, best accounts for the symptoms of PFP, independent of factors such as chondromalacia patella, or patellofemoral (PF) "malalignment".

Keywords: Patellofemoral pain (PFP); synovitis; tissue homeostasis

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Introduction

Patellofemoral (PF) symptoms represent perhaps the most common clinical complaint seen by orthopedic surgeons world-wide. Yet, despite its extreme prevalence and years of dedicated research, conditions of the anterior aspect of the knee surprisingly remain a source of continued controversy. Disagreement exists in the explanation of the etiology as well as which methods of treatment are likely to result in symptom resolution (1). Typically, the symptoms consist of patellofemoral pain (PFP)/anterior knee pain (AKP), and/or "Instability" (2). Patients with complaints of AKP—despite existing in far greater numbers than those with complaints of PF "instability"—often get much less attention from the orthopedic surgical community than they deserve. Most orthopedic surgeons will just "pass on" the patient to the physical therapist, in essence deferring to their perspective of the problem. This should not be the case and needs to be corrected. This work will concentrate on an overview of the vast majority of these patents—those that experience PFP—so the orthopedic clinician can be exposed to the latest concepts of pathophysiology and treatment.

The enigma of PFP

For well over a century, the clinical condition of PFP has been considered to be a classic musculoskeletal enigma. Why is this so? It seems likely that the primary cause results from approaching it as due to exclusively structural/anatomic/biomechanical (S/A/B) pathology—as is assumed to be true in most every other orthopedic condition. The standard 20th Century treatments based on a S/A/B diagnosis such as aggressive physical therapy (PT) to strengthen the quadriceps to treat patellar chondromalacia or correct maltracking; lateral releases to unload a "tilted" patella; or various proximal and distal "realignment" procedures to correct "malalignment". None have proved to be predictably effective. In fact, the worst cases of AKP that this and many other clinicians have witnessed have resulted from multiple surgical attempts to correct chondromalacia patella, or "PF malalignment" (3). Often patients whose initial, pre-operative symptoms were merely mild and intermittent discomfort end up, after 1–17 surgeries, left with permanent, chronic discomfort and on occasion,

complex regional pain syndrome and disfigurement! There are exceptions, of course, where improvement of PF tracking can actually be beneficial. Often these cases are properly assessed and treated by true experts in the field such as Drs. Merchant, Fulkerson, Andrish, Post, Sanchis-Alfonso, Arendt, Fithian, and Teitge, who understand the knee and patellofemoral joint (PFJ) in depth (4,5). The typical orthopedic surgeon often misjudges the underlying pathophysiology, and so performs surgeries that are inherently dangerous. It is important to remember that the PFJ is unforgiving of treatment that does not respect its unique biological characteristics.

Pain cannot be measured in any manner similar to objective structural factors (e.g., Q-Angle, femoral anti-version, TT-TG measurement, presence of chondromalacia, etc.). It is crucial to understand that the experience of pain is inherently and irreducibly a subjective phenomenon. Pain is a function of central nervous system (CNS) events in the opposite cerebral cortex to the symptomatic knee. Therefore, factors such as emotional and psychological stress can often accentuate the symptoms.

One must have an accurate diagnosis in order to provide safe and effective treatment for any condition (6). The primary factor underpinning the past failure of PFP treatment was an utter misunderstanding of the actual etiology of the condition. In a way, it was like treating fever with blood-letting or head ache with trephination. The treatments made perfect sense from the perspective of the medical paradigms of their era and just like the various 20thC approaches for PFP, many patients eventually improved, not because of the treatment, but in spite the treatment.

The standard 20thC diagnoses for PFP that made the most sense from the perspective of that era, based on a pure S/A/B paradigm, were CMP and some form of "malalignment/maltracking". These diagnoses could be readily determined with the commonly available structural studies: X-ray, ultra-sound, CT, MRI, and direct observation of the PFJ at surgery.

Most patients and even the current majority of orthopedic surgeons, believe that an MRI reveals the *cause* of the problem leading to an *accurate diagnosis* from which to make logical, therapeutic decisions. This is no longer believed to be true by an increasing number of orthopedic surgeons who have come to understand a deeper, more biological perspective of living joints (7). This, in essence, represents an entirely new M/S paradigm from which to make improved diagnoses leading to more effective therapeutic decisions. What is this new, emerging orthopedic paradigm?

Tissue/joint homeostasis

The older paradigm viewed joints as assemblages of anatomic parts where structural damage of a component (e.g., CMP) was presumed to be the etiology of PFP. However, it has been shown by us and others, that even grade 3+ CMP (which is T1 rho positive on MRI, indicating a substantial diminishment of proteoglycan content) can be completely asymptomatic to probing without intra-articular anesthesia (8). In fact, despite having had documented bilateral grade 3+ CMP for at least 18 years—the lead author still remains completely pain-free in all his activities even though he has experienced millions of load cycles placed across his knees during this time. (This observation is critical for knee surgeons to understand. Finding the presence of CMP on an MRI does not mean the symptom of PFP is caused by the structural damage of articular cartilage. The finding should NOT be used by itself, as a pretext or rationale to perform aggressive patellar surgery.)

The new homeostasis paradigm views joints as living organ systems, evolutionarily designed to safely accept, transfer and dissipate repetitive loads in the manner of a biological transmission. Joints are composed of a variety of tissues each one represented by a volume of cells, each with their own range of normal physiological processes—termed tissue homeostasis (6). In combination the term is Joint Homeostasis.

The patellofemoral joint acts as a type of large "slide bearing" within the biologic transmission. The PFJ withstands the highest forces both in compression and tension of any human musculoskeletal component. It therefore has a low threshold for loss of homeostasis and great difficulty in healing —restoration of homeostasis following injury. If a joint is in homeostasis, it is, by definition, pain free. The opposite is also true. If a patient is experiencing discomfort, then that tissue/joint is not in homeostasis. A symptomatic knee typically represents a mosaic of pathophysiology, composed of potentially, multiple tissues. Two of the primary "Tiles" of the that potential mosaic in PFP patients are: symptomatic synovitis and bone overload (objectively manifested by a positive Tc Bone Scan). The senior author has directly experienced severe pain from palpation of normal, un-anesthetized synovium and experimentally produced increased intra-osseous pressure within his own normal right patella.

This new perspective was developed in order to provide a more rational explanation for the enigma of PFP. However, this same insight has brought clarity to a number of M/

S conditions beyond the PFJ, including the early natural history of osteoarthritis of the knee and other joints (9).

The treatment goal from the perspective of the old S/A/B paradigm was restoration of anatomically normal structures, e.g. reconstruction for every ACL tear and attempting to replace the missing segment of medial meniscus removed in a partial medial meniscectomy. However, many patients do remarkably well without surgical intervention—if they stay within their joints' envelope of function (EOF): a load/frequency distribution that represents the range of load that is pain-free and compatible with induction/restoration of tissue/joint homeostasis (10).

The PFJ is special because the normal loading environment is the harshest of any M/S component in the human body, typically multiples of body weight. These very high loads with normal activities of daily living (ADLs) often exceed the load transference capacity of the PF Joint (out of the EOF)—resulting in loss of homeostasis of the PF tissues (e.g., synovium and bone), independent of the radiographic appearance of the joint (11).

In other words: tissue/joint homeostasis is more fundamental to knee function than any structural characteristic and needs to be high on the conceptual priority list for the orthopedic surgeon and physical therapist when approaching a symptomatic joint.

Principles of evaluation and treatment of the PFP patient from a tissue homeostasis perspective

First, it's critical to establish an *accurate diagnosis* as best as one can—each symptomatic knee represents a unique mosaic of PFJ pathophysiology. Importantly—one must rule out non-PFJ sources of pain: esp., ipsilateral hip pathology (e.g., synovitis, infection, and osteoarthritis) as well as saphenous nerve neuropathy, particularly of the infra-patellar branch (by the use of a Tinel's test—palpation over the nerve induces the sensation of tingling/discomfort). Neuropathy of the saphenous nerve is much more prevalent than is commonly believed. If this diagnosis is missed, no matter what therapy may be directed towards the knee proper, symptoms of discomfort are likely to indefinitely persist.

History

When taking a history of the symptoms from a patient there is very often an identifiable initiating event or series of events that triggers the onset of persistent PFP. Examples are: repetitive squats and lunges, hiking up and down hills, stairs or inclines, lifting and carrying heavy objects, kneeling, and possible direct trauma (e.g., dashboard injury).

AKP is also typically aggravated by activities that highly load the PFJ. Often the initial event (overload out of the knee's EOF) will be on its way towards improvement when a 2nd or even 3rd overload event occurs shortly thereafter that makes the symptoms worse—where the EOF for that knee diminishes to the point that ADLs become symptomatic. This is the essence of most chronic PFP. Often patients experience increased warmth and on occasion at least some degree of swelling (effusion), the hallmark of symptomatic PF synovitis.

The symptoms of AKP are often episodic—interspersed with periods of clinical silence, depending on the loading environment placed across the knee. Recurrent synovial impingement and inflammation are, in the lead author's opinion, the most common underdiagnosed condition of clinical significance in the knee, world-wide. One can conceive of PF synovial impingement as equivalent to repetitively biting the inside of one's cheek.

The symptoms can be in different geographical regions of the PFJ at different times due to synovial impingement and bone overload in different areas.

Improvement secondary to rest (loading within the knees EOF) and icing is common.

Complaints of 'giving way' and 'instability' are often due to impingement of inflamed peri-patellar tissues with resultant reflex inhibition of the quadriceps. This phenomenon is a protective mechanism equivalent to dropping a hot pan—and, N.B.—should not be confused with "malalignment/maltracking". David Dejour of Lyon France defines surgically significant 'instability' as recurrent documented dislocations, not mere episodes of "subluxation".

Physical exam

The presence of peri-patellar tenderness is frequently found at the anterior medial joint line and patellar facets.

Warmth—often above 88 degrees F, or 2–3 degrees warmer than the asymptomatic knee, if unilateral.

A small, but detectable effusion.

Increasing pain with loaded flexion.

Muscle tightness, stiffness.

Occasionally a positive Tinel's sign, especially over the infra-patellar branch of the saphenous nerve indicative of

neuropathy. One should perform this test on every PFP patient.

Imaging

Radiographs—AP, lateral and Merchant views.

Ultra sound.

CT.

Cine CT.

MRI to look for evidence of synovial impingement pathology—i.e., presence of an effusion, detectable synovial swelling, and very often, neovascularization of the fat pad.

Tc99m-MDP Bone Scan—3rd delayed phase, focal or diffuse up-take in the PF Joint.

SPECT—(Single Photon Emission Computed Tomography) is rarely needed with a good standard Bone scan, although many clinicians prefer its higher resolution.

CT-SPECT-rarely needed.

PET.

CT-PET—highest resolution of loss of osseous homeostasis—but very high radiation exposure (27 millisieverts) compared to standard Tc Bone Scan (4 millisieverts).

Treatment

Goal of treatment

Restore the joint's normal EOF—painless load transference capacity by achieving and maintaining joint homeostasis (3)!

Rest—decrease the loading across the knee until the joint becomes asymptomatic—in other words: get the knee within its current diminished EOF! This is 80% of what is generally required for substantial, persistent improvement of symptoms. This is a simple but powerful principle, and crucial for both patients and physical therapists to understand.

Consider what one is asking the knee to do—heal itself while continuing its primary function of high load repetitive high load transference. These are, of course, completely opposite concepts. It's therefore not surprising when symptoms of AKP become chronic, secondary to ADLs exceeding the knee's diminished EOF (load acceptance capacity). These consist of activities such as stair climbing, squatting, kneeling, getting in and out of chairs and prolonged walking are often sufficiently great so as to subvert the normal biological mechanisms of healing (equivalent to putting lighter fluid on a fire one is wishing to extinguish).

One needs to help create the internal environmental conditions to allow the strained/inflamed PF tissues to heal themselves and restore joint homeostasis!

Fortunately, knees have been healing themselves (by themselves) for over 360 million years of vertebrate evolution (12). The knee is typically, metabolically resilient but only within a certain specific range of loading—the unique EOF at that given moment.

Patients should avoid low chairs and sit as high as possible. The use of an elevated toilet seat can be quite helpful in decreasing the load across the symptomatic PFJ especially in females.

Anti-inflammatory Therapies as indicated can be helpful: e.g., Cool the joint 1–2 times a day × 15 minutes (cool not "frozen numb"). Plus an oral medication as tolerated (e.g., Solubilized Ibuprofen 400 mgs po bid), and if needed—an injection of an intra-articular steroid such as triamcinolone acetonide (Kenalog) 40 mgs.

Pain-Free PT: including careful muscle strengthening, gentle stretching, cooling as indicated, core strengthening, home/gym program, and patellar taping as may be helpful in decreasing perceived symptoms of PFP.

If there is evidence of neuropathy e.g. complaints of 'tingling/burning' and a positive Tinel's test then it is appropriate to begin a nerve-calming agent such as Gabapentin 300 mgs po tid.

If all else fails, consider an arthroscopic inspection and be prepared to do a careful, limited synovectomy in the region that is most symptomatic, as well as deal with whatever structural factors that may also be present and are of etiologic significance.

Understand that surgical treatment for PFP represents a new injury to the already symptomatic knee

All surgery designed to help alleviate PFP, no matter how gently performed, represents a new injury from the perspective of the knee. It has no way of perceiving, that in time, the symptoms will likely improve assuming, of course, that a correct diagnosis was made and the appropriate procedure chosen. Therefore, it is important that this concept is well understood by all those involved, including the surgeon, patient, patient's family, physical therapists, and employer, school, etc.

Healing following surgery is a rate-limited biological phenomenon that cannot be accelerated/rushed. On average

it takes about 18 months (in our experience) for operated joints to come to full homeostasis. During this time the loads across the knee must be kept well within the joint's EOF/envelope of load acceptance (i.e., pain-free). As the knee begins to heal, the capacity of the knee to take loading safely will likely increase incrementally. The natural inclination will be for the patient to "get ahead" of themselves by attempting loading activities outside of their current diminished (but expanding) EOF, such as increased walking, squatting, lunges, hiking, stair usage and the like. Any pain producing activities must be avoided. Even slight pain represents loads that subvert the normal complex biological healing mechanisms. This is true for all anatomic M/S regions but is especially important for the components of the PFJ. Mild soreness after a workout is acceptable, however.

Factors that determine clinical success following surgery

The success of surgery depends on 3 independent variables like 3 legs of a stool**.** This is an analogy that is used with all surgical patients to help them understand the various factors that determine the potential success of operative outcome.

Under the surgeon's control**:** make the correct diagnosis! PFP is rarely due to structural issues. Surgery (e.g., a synovectomy) must be done properly and carefully. Guide the patient in the post-op recovery period: when and what kind of PT to do post-op; when to come off crutches; how often to cool the knee; how often to move/exercise the knee; when to go back to work, long term PT exercises, etc.

Under the patient's control**:** don't make the symptoms worse pre-op; show up for therapy as directed, cool the knee as directed; use crutches as directed; protect the knee as it heals, in other words—be sure to stay within the knee's EOF at all times, as able.

Under nobody's control**:** the degree of pathology present pre-op and the efficiency of biological healing following whatever surgical procedure is performed.

This last factor, the efficiency of biological healing, is under the control of the patient's own DNA code. It is biologically "built in" and cannot (currently) be readily affected.

Be forbearing regarding recovery time, especially in an environment with many stairs and hills. As noted above it can often take up to 18 months or more to achieve full joint homeostasis/healing following even minimal surgery. All concerned need to respect this principle.

As previously stated, healing is a "rate-limited" biological phenomenon analogous to the maturation of a fine French Bordeaux such as a Château Lafite Rothschild. Eight to ten years are required to create a drinkable vintage from the initial grape juice. The vintners understand the chemistry (to a degree) but they don't control it. They cannot make a palatable wine in a week, month, or a year. Similarly, we cannot "accelerate" restoration of joint homeostasis/healing. Each knee must take its own unique time. Therefore, patience is a virtue in this clinical setting.

The more one helps the patient understand the fundamental principles as outlined in this work, the better will be their therapeutic outcome.

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