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Mini Review

Painful Knee Osteoarthritis & Iliotibial Band Syndrome, New Approach

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Abstract

OA can be viewed as structural and functional failure of synovial joints, the pathophysiological response of a synovial joint to mechanical insult, and the attempt of the joint to repair the damage, therefore, thinking about OA is moving from biochemistry of the particular cartilage to the mechanobiology of the whole joint.

The hallmark symptom of knee OA is pain yet the etiology of pain in OA is not entirely clear.

It is clear that the severity of the joint damage on the radiograph bears little relation to the severity of the pain experienced.

According to multiple researches and articles the most prevalence and consistent signs from all the MRI findings in painful knee osteoarthritis are active effusion/synovitis and large bone marrow lesions BML (often associated with effusion and some degree of synovitis) suggesting these features may indicate the main aetiopathology of painful knee osteoarthritis compared with patients with no symptoms.

As well as the high incidence of iliotibial band friction syndrome ITBS and advanced medial compartment knee osteoarthritis with genu varum, this is due to cartilage loss associated with advanced degeneration of the medial meniscus led to reduced medial knee joint space created a varus knee deformation, and thus putting extra tension into the iliotibial band (Vasilevska s`group & Farell et al.), this alter bio mechanic may contribute to the development of painful fibro vascular tissue between ITB and lateral epicondyle.

Accordingly to presence or not of effusion/synovitivit we simply attribute the aetiopathology of pain in symptomatic knee OA into intra articular pathology with presence of effusion/synovititis or extra articular & periarticular pathology (iliotibial band syndrome) with no effusion/synovitis and different methods of treatment, after thorough history, clinical, radiological examination and always ultrasound with power Doppler scan we divide patients into two groups:

- (Wet KOA) Patients with signs of effusion/active synovitis and or MRI finding of large bone marrow lesion, BML (often associated with moderate effusion/synovitis) if visible, the ateiopathology is an intra-articular one and to be treated accordingly.
- (Dry KOA) Patients with no signs of effusion/active synovitis the aetiopathology is extra articular & periarticular one, (most probably iliotibial band friction syndrome) which is treatable.

Keywords: Osteoarthritis; Wet knee; Dry knee

Introduction

It is important to identify whether patients with knee OA are at increased risk for periarticular lesions, and which lesions are likely to be present.

After thorough history taken, careful clinical and radiological examination including X-ray, MRI and most important outpatient ultrasound with synovial power Doppler scan.

We group patients into two categories

Wet knee: Swollen, synovial hypertrophyactive synovitis, and large effusion confirmed with ultrasound and synovial power Dopplerscan as well as large bone marrow lesion seen in MRI if visible.

This groupof patients theetiology of pain is due to advanced osteoarthritic active intra-articular lesionsfor which we treat accordingly.

Dry knee: With No signs of active synovial reaction, hypertrophy and large effusion confirmed by ultrasound & Doppler scanor large bone marrow lesion in MRI if available.

This groups, of older patients , young age, athletes and even a

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dolescents, with all their local complain, clinical and radiological signs of advanced knee osteoarthritis, the etiology is coming from extra articular cause mostly if not always tight Iliotibial band syndrome ITBS associated with chronic myofascial pain syndrome. We are of the view that treatment of acute iliotial band syndrome with local steroid injection and magnesium supplements may suppress the pain and improve the patient's ability to mobilize in the presence of advanced varus osteoarthritis knee changes.

It is our believe that there are a considerable amount of patients receiving unnecessary treatment due to incomplete detailed history taking ,examination and investigation done by the health provider who is distracted by clinically and radio logically apparen tsigns of sever osteoarthritis with easy readily diagnosis.

Discussion

Osteoarthritis has been ranked number 5 in the top 10 most costly medical conditions after heart disease, cancer, mental and trauma related disorders with an estimated cost burden of $185.5 billion in the USA. According to the American Academy of Orthopedic Surgeons, every year 9 million adults in the United States are diagnosed with arthritis and 600,000 total joint replacements are performed.

OA can be viewed as the clinical and pathological outcome of a range of disorders that results in structural and functional failure of synovial joints.

The joint is a mechanical structure, and the key to understanding OA is abnormal mechanical stress: joint failure is the patho physiological response of a synovial joint to mechanical insult, and the attempt of the joint to repair the damage caused by local abnormalities in force/unit area.

The abnormalities in cytokines, degradative enzymes, toxic radicals and the like, which are being studied as the cause of OA, are rather the result of this attempted repair. Therefore, thinking about OA is moving from biochemistry of the particular cartilage to the mechano biology of the whole joint.

Knee OA is clearly a multi factorial disease and the aetiopathogenesis.

Includes local factors (trauma, misalignment, overloading, muscle Weakness around joints etc.), general conditions (old age, Female sex, obesity, physical activity level) together with genetic Susceptibility.

The relative contribution of these factors, and their importance for development and progression of Knee OA with possible implications for sub grouping remains to be clarified.

Estimates are that 20% of all patients aged 45 years or older have KOA and that this increases to35 % in subjects age 65 years or older.

Obesity is recognized as a very important risk factor for KOA, a high Body Mass Index (BMI) has been found to increase the risk of KOA and weight-loss to reduce this susceptibility.

The genetic predisposition for KOA is described in several studies and though very heterogeneous, the heritable component has been estimated to be significant.

The hallmark symptom of knee OA is pain yet the etiology of pain in OA is not entirely clear.

the European League Against Rheumatism (EULAR) concluded that in persons older than 40 years who have use-related knee pain, a clinical diagnosis of OA may be made on the basis of 3 key symptoms-persistent knee pain, morning stiffness of only brief duration, and reduced function-and 3 findings on physical examination-crepitus, reduced movement, and bony enlargement.

There were only two ways of looking at joints affected by OA: X-ray or gross anatomy, that obsession with pathology, X-rays and cartilage damage, which was led from the UK, and that stretched from the 1950s to the beginning of this century.

During the years of focus on articular cartilage, fundamental research into OA was dominated by biochemists and cellular biologists who did wonders in sorting out the biology of cartilage, but largely failed to understand that OA is primarily a mechanical problem.

In clinical practice the diagnosis of OA should be made on the basis of your history and physical examination and the role of radiography is to confirm this clinical suspicion and rule out other conditions.

It is clear that the severity of the joint damage on the radiograph bears little relation to the severity of the pain experienced.

Amin and colleagues showed that knee x-rays have a sensitivity of only about 25% for cartilage loss seen on MRI.

The main MRI findings are, anterior cruciate ligament rupture with or without meniscal lesions, Cartilage defect, Osteophytes, large bone marrow lesion, active synovitis, hypertrophy and joint effusion.

To systematically evaluate the association between MRI findings (cartilage defects, Bone Marrow Lesions (BML), osteophytes, meniscal lesion, effusion/synovitis, ligamentous abnormalities, subchondral cysts and bone attrition) and pain in patients with knee Osteoarthritis (OA) in order to establish the relevance of such findings when assessing an individual patient. Conclusions Knee pain in OA is associated with BML and effusion/synovitis suggesting that these features may indicate the origin of pain in knee OA.

Cartilage has no vascular , lymphatic or sensory supply ,pain arise from subchodral sensory supply and damage cartilage micro particle causing chemical synovitis leading to inflammation with synovial swelling ,warms and effusion leading to pain.

The synovial reaction in OA includes synovial hyperplasia, fibrosis, and thickening of synovial capsule, activated synoviocytes and in some cases lymphocytic infiltrate (B- and T-cells as well as plasma cells).

The site of infiltration of the synovia is of obvious relevance as one of the most densely innervated structures of the joint is the white adipose tissue of the fat pad which also show evidence of inflammation and can act as a rich source of inflammatory adipokines.

Synovial causes of pain include irritation of sensory nerve endings within the synovia from osteophytes and synovial inflammation that is due, at least in part, to the release of prostaglandins, leukotrienes, proteinases, neuropeptides and cytokines.

A semi-quantitative measure of synovitis from the Infrapatella fat pad is associated with pain severity and similarly change in synovitis is associated with change in pain severity.

Bone marrow lesions were found in 272 of 351 (77.5%) persons with painful knees compared with 15 of 50 (30%) persons with no knee pain ($P < 0.001$). Large lesions were present almost exclusively in persons with knee pain (35.9% vs. 2%; $P < 0.001$What may subsequently cause pain is as yet unknown. Increased trabecular bone pressure, ischemia and inflammation are all possible stimuli.

The prevalence of subchondral bone marrow edema, knee joint effusion, and synovial thickening in patients with symptomatic knee OA compared with patients with no symptoms. The primary pathologic abnormality in OA (hyaline Cartilage loss) could occur without pain.

So not every radiological signs of advanced osteoarthritis are symptomatic, a lot of patients present with unicompartmental disease, and the medial compartment is almost 10 times more frequently involved than the lateral compartment.

Medial compartment cartilage loss leads to varus deformity and alter the biomechanics of the knee with varus deformity leading to increase the compression of ITB over the lateral femoral condyle with old study by Farell et al. 2001 previously established association of ITBFS with genu varum in runners .and frequent presence of MR signs of ITBF in patients with isolated medial compartment knee osteoarthritis [1-11].

Vasilevska et al. [2] stated that ITBF is unrecognized cause for lateral knee pain in patients with medial compartment knee osteoarthritis. The iliotibial band is one of largest connective tissues in the body. Anatomically it is thought to have origins on the ilium, sacrum, gluteus maximus, and the Tensor Fascia Latae (TFL muscle) and insertions in the entire length femur, the patella, tibial condyles, and head of the fibula. It plays an important role in the movement of the thigh by connecting hip muscles to the tibia. It is well known that ITBFS is associated with overuse in long distance runners, cyclists, military personnel, football players, and weight lifters [4].

The study of Hariri and others calls into question whether inflammation of the ITB is actually involved in ITBFS. In their case series, the symptoms of ITBFS were alleviated by surgical excision of what they described as a bursa in the sub-ITB space. Costa and colleagues also reported on a case in which a large cyst, arising from the joint capsule, was discovered in a 28-year old runner with lateral knee pain. Nemeth and Sanders may have been describing the same tissue in their anatomical review, but they referred to it as a lateral extension of the knee synovial capsule.

It may be that different subtypes of iliotibial band friction syndrome exist, one that involves irritation of a cyst, bursa, or lateral synovial recess, and a second type arising from compression by the iliotibial band of the connective tissues that underlie the portion of the band between the lateral epicondyle and the knee joint line. Tightness of the ITB may play a role in patellofemoral syndrome.

Vasilevska's group studied patients with osteoarthritis of the medial compartment of the knee and found a high incidence of iliotibial band friction syndrome. Reduced medial joint space created a varus knee deformation, thus putting extra tension into the iliotibial band.

Greater trochanteric pain syndrome (previously known as trochanteric bursitis) may also reflect altered biomechanics of the ITB, that explain the frequent association of trochanteric pain syndrome with knee pain and mistaken as sciatica like pain.

Pelfert and others have reported the occurrence of ITBFS subsequent to repair of the anterior cruciate ligament, Farell emphasized that ITBFS usually occurs as a result of overuse. If, however, the patient has certain anatomical conditions (leg length discrepancies, varus knee alignment or excessive pronation and external tibial rotation of more than 20%), he/she will be more inclined to experience ITBFS.

And thus that ITB syndrome is a 'friction syndrome' is challenged, in 2007, John Fairclough of University of Wales Institute, with seven coauthors, issued a major challenge to the classic definition of iliotibial band syndromethe perception of movement of the ITB across the epicondyle is an illusion, in effect suggesting that the function, dysfunction and actual anatomy of the iliotibial band has been misunderstood all along., they agree that the IT band really is firmly anchored to the side of the knee.

An anatomic study disclosed that the ITB is simply a thickened, lateral part of the fascia lata. It completely surrounds the thigh, is anchored to the femoral shaft by the lateral intramuscular septum and is continuous with the patellar retinacula Thus, the ITB is unlikely to roll forwards and backwards during flexion and extension of the knee, but could move slightly in a medial–lateral direction, and converting tensile to compressive loading along its lateral aspect during knee flexion, compressing the richly innervated Vascularized fat and loose connective tissue beneath the tract [9,11].

The portion of the knee range of motion at which the ITB is most likely to rub against (old theory) or compress the underlying structures (recent theory) is with the knee flexed about 20°-30° They concluded that knee-flexion repetition was more likely to result in the onset of the overuse injury ITBFS during cycling.

An association of ITBFS with genu varum in runners has been previously established (When severe cartilage damage is associated with advanced degeneration of the medial meniscus, altered biomechanics probably, may contribute to the development of fibro vascular tissue between the iliotibial band and the lateral epicondyle on MR images as a recognized sign of ITBF [2].

Recently the iliotibial band consider part of the new fascial system interpenetrates and surrounds all organs, muscles, bones and nerve fibers, endowing the body with a functional structure, and providing an environment that enables all body systems to operate in an integrated manner. The concept of a continuum of the collagen and connective structure, the cellular diversity that makes up the fascia, is emphasized. It is this continuum itself that assures the health of the body. This is the broadest definition of the fascia by the Fascia Nomenclature Committee.

Normal movement of the body is allowed because of the presence of the fascial tissues and their inseparable interconnection, which allow the sliding of the muscular structure, the sliding of nerves and

vessels between contractile fields and joints, and the ability of all organs to slide and move with each other as influenced by the position of the body. One of the fundamental characteristics of the fascia is the ability to adapt to mechanical stress, remodeling the cellular/tissue structure and mirroring the functional necessity of the environment where the tissue lays. (FORCE - Foundation of Osteopathic Research and Clinical Endorsement. 2013).

The diagnosis of ITB friction syndrome is based on clinical examination and radio logically with MRI with tenderness over the lateral femoral epicondyle and report a sharp, burning pain when the practitioner presses on the lateral epicondyle during knee flexion and extension. In 2004, a research group at University of Connecticut led by Michelle Devan decided to try to figure out the effect of "structural abnormalities" on overuse knee injuries like iliotibial band syndrome.

All the athletes with iliotibial band friction syndrome had a negative bilateral Ober test [their iliotibial bands were not tight]. Not one of them had tight iliotibial bands. Not even one!

Clinically, patients with medial sided osteoarthritis of the knee, occasionally also complain of laterally located pain, ITBF is unrecognized cause for lateral knee pain in patients with medial compartment knee osteoarthritis Patients with complete cartilage loss as well as patients with subtotal cartilage loss showed tendency to further increase the incidence of MR signs of ITBF, when advanced degeneration of the medial meniscus was present [2].

With a frustrating lack of progress in the development of treatments for osteoarthritis, EULAR has released recommendations to reorient research into this disease. These recommendations include focused attention on non-cartilaginous tissues, the interaction of structures within the joint, and the pathogenesis of osteoarthritic pain, new treatment strategies, and early disease.

Conclusion

Because Cartilage has no vascular, lymphatic, or sensory supply, the primary pathologic abnormality in OA (hyaline cartilage loss) could occur without pain and it is clear that the severity of the joint damage on the radiograph bears little relation to the severity of the pain experienced.

Advanced reduction of cartilage thickness combined with severe degeneration of the meniscus at the medial compartment probably leads to biomechanical changes, and varus knee alignment. It may be the cause for iliotibial band syndrome. A latest statement for the so frequent presence of MR signs of ITBF in patients with medial compartment knee osteoarthritis [2], give us a right to put this entity in the list of an important associated entities with knee osteoarthritis. We should always think about it as the reason for lateral posterior knee pain in those cases.

In MRI studies is reported an increase the prevalence of subchondral bone marrow edema, knee joint effusion, and synovial thickening in patients with symptomatic knee OA compared with patients with no symptoms. Based on these we adopt in our clinic a simple triage according to detailed history taking, clinical and radiological examination including MRI and always ultrasound examination with power Doppler scan.

First Patient group with so called WET Knee with active synovial reaction ,hypertrophy and large effusion seen with US power Doppler scan or large bone marrow lesion seen in MRI ,to be treated accordingly as intra articular advanced knee osteoarthritis pathology.

The second group so called DRY KNEE with any age group and have all signs of radiological varus medial compartment osteoarthritis but with no active synovial reaction ,effusion confirmed by US power Doppler scan or large bone marrow lesion seen by MRI, specially lateral and posterior knee pain sometime down to outer aspect of the leg and up to the trochanteric region even to the back of the hip region and because it is frequently associated together and often mistaken as sciatic pain , we treat them as Iliotibial band syndrome as well as trochanteric pain syndrome with local steroid injection (triamcinolone acetonide 40 mg)each and magnesium supplement, most of our patients felt immediate relief of pain improve mobility of the joint.

Comorbidities associated with old age especially diabetic and chronic illness as well as younger athletes, they often have some magnesium deficiency, which may contribute to the myofascial spam and tight iliotibial band associated.

Misdiagnosis often leads to omission of appropriate treatment or institution of unnecessary treatment.

Note

This article is reflecting a personal practical point of view based on my careful thorough reading and understanding of the mechanobiology of the knee joint and practicing this technique more than 7 years in busy orthopedic clinics.

This review is open for scientific discussion and criticism.

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Accordingly we simply divide patients of symptomatic knee OA after thorough history, clinical, radiological examination and always ultrasound with power Doppler scan into two groups:

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