

Physiotherapy management of tibio-femoral degenerative joint disease

DR. P. P. MOHANTY, ASSOCIATE PROFESSOR, DEPT. OF PHYSIOTHERAPY

Introduction:

Osteoarthritis is the most common joint disorder causing disability affecting more than millions of people all over the world. Its prevalence after the age of 65 years is about 60% in male and 70% in female. Osteo-arthritis/ degenerative joint disease of the knee may occur in the medial and/or lateral compartment of tibio-femoral joint and/ or patello-femoral joint. Investigation in people with knee pain revealed the most common radiographic pattern to be a combined TFJ and PFJ disease. Individuals with combined PFJ and TFJ OA had more symptoms, lower function in sports and recreation and worse knee related quality than individuals with isolated TFJ OA.

WHO estimates that 10% of the worlds population over the age of 60 years suffers from OA and that 80% of people with OA have limitation of movement and 25% can not perform the major daily activities. The prevalence of OA rises with age, and with increasing longevity, the incidence is certain to rise The prevalence of radiographic knee OA rises in women from 1% to 4% in those aged 24 to 45 years to 53% in those 80 years and older. In men, the prevalence rises from 1% to 6% in those 45 years and younger to 22% to 33% in those 80 years and older.

Osteo-arthritis/ degenerative joint disease is characterized by progressive degenerative changes in articular cartilage and subchondral bone. The tissue changes may be of a compensatory hypertrophic nature such as bony proliferation that typically occurs at the margin and the subchondral bone or capsular fibrosis.

Etiology

Primary osteo-arthritis is distinguished from secondary by the elimination of predisposing factors and relevant pre-existing diseases of the knee such as involving tibial plateau and/ or femoral condyles, instability after ligamentous injury, post inflammatory states-TB, septic arthritis etc. and deformity - genu valgum, genu varum, foot or hip problems, following meniscectomy or patellectomy etc. Primary OA is considered to be a result of aging.

Tibiofemoral joint is most incongruent in flexion and in extension the joint becomes most congruent. The mobility and deformability of the fibrocartilagenous menisci allow them to conform to the shape of the contacting femoral surface and reduce joint incongruity.

The exact etiology of osteoarthritis is not known.

With aging changes in the cartilage is seen in the non-articular area of the cartilage, whereas in OA changes occur in those areas of cartilage undergoing most frequent contact doing weight bearing. Changes takes place in the region undergoing increase stress with normal activities. Changes in subchondral bone will over time result in changes in articular cartilage and vice versa. Normal attenuation of force applied to a joint is dependent on the elasticity of subchondral bone as well as those of articular cartilage. Subchondral bone sclerosis reduces its energy attenuation capacity, so the articular cartilage is subjected to more load and degeneration. Habitual disuse hampers the nourishment of the avascular cartilage, which gets its nutrition from the synovial fluid.

Systemic risk factors - These factors include age, gender, hormonal factor, genetic factor, bone density, nutritional factor, obesity and other factors which have yet to be identified.

Local Biomechanical factors - Local biomechanical factors include altered joint biomechanics due to ligament laxity, malalignment, impaired proprioception and muscle weakness, prior joint injury, occupational factors, effect of sports & physical activities and result of developmental abnormalities etc.

Pathophysiology

The first change is loss of mucopoly-sacharides with the loss of elasticity and reduced ability to protect the cells by absorbing shock, leading to further damage. When loss of cartilage is full thickness the bone get exposed. On cellular level there is an imbalance between the destructive and reparative or synthesis process of the articular cartilage. The mechanism responsible for progressive loss of cartilage in OA includes alteration of the cartilage matrix, decline of the chondrocytic synthetic response and progressive loss of cartilage

The degenerated detached articular cartilage fragment needs to be removed from the joint. So there develops mild to moderate inflammatory process and the synovium gets inflamed. As inflammatory process subside the synovial fluid become thick and glue the synovial tissue and ligaments. Adhesion formation limits joint mobility. In chronic stage of disease fibrosis and thickening of capsule and intracapsular structures lead to stiffness. Fibrosis or thickening of the outer fibrous layer of the joint capsule is caused by an increased production of collagen and by a decrease in the ground substance.

As bone get involved in disease process and there is formation of new extra bone on trabeculae in the subchondral bone, gradually it leads to subchondral sclerosis, formation of cyst like bone cavities and development of osteophytes. Development of osteophyte due to changes in articular cartilage develop around the periphery of the joint and also along insertions of the joint capsule. Subchondral bone alterations are thought to be result of abnormal osteoblast function.

With the progressive degeneration of articular cartilage and subchondral bone, the joint space reduces resulting in capsular laxity, hypermobility and instability. With the

destruction of the smooth articular surface, there will be crepitus during the movement from increased friction between the articulating surfaces.

With pain and decrease willingness to move, contracture eventually develops in portions of the capsule and overlying muscles as disease progress, movement becomes more limited. Decrease use of the joint lead to muscle atrophy with concomitant loss of joint protection.

Osteoarthritis pain develops mainly from synovium, joint capsule, periarticular tender points and subchondral bone as all of these structures have large number of group IV nociceptors. Group IV nociceptors in the synovium and capsule become mechanically stimulated when the pressure in the joint increases. They become chemically stimulated when inflammation induced tissue damage leads to the liberation of nociceptor sensitizing substance such as bradikinin, prostaglandin, serotonin, histamine and neuropeptides. In addition pain may develop as a result of the calcium crystal induced inflammatory changed.

Tender points are found commonly in anteromedial aspect of upper part of tibia, where the lower part of the collateral ligament and superficial to this the tendon of the sartorius, gracilis and semitendinosus muscle insert into the tibia. Tenderness in this site may also be due to inflammation of the anserine bursa situated between the ligament and these tendons. Tenderness may also be located along the edges of the patella, particularly along its lateral upper border.

Posterior tender points are frequently found to be present in the popliteal fossa, particularly at the centre of it and on its insertion.

Clinical features:

Insidious onset of pain following weight bearing activity such as walking, stair climbing, getting up from sitting etc. Later as low grade inflammation develops, the patient complains of morning pain and stiffness, which subsides following movements

and activity and returns back with increase intensity following activity due to fatigue. At the onset the pain following activity is due to fatigue. As the inflammation set in pain becomes continuous. Pain during activity is because of stretching of the tight capsule. With the erosion of subchondral bone which is highly pain sensitive pain becomes continuous.

Once joint instability develops due to gross destruction of the articular cartilage, strain on the periarticular capsule-ligamentous structures gives rise to pain. Impingement of soft tissue structure by the marginal osteophyte also give rise to localized inflammation and pain.

Observation - Walks with limping and finds difficulty in squatting, getting up from sitting, stair climbing etc

Inspection - Atrophy of thigh, more of vastus medialis.

Deformity – knee flexion deformity due to capsular contracture and varus due to destruction of medial compartment of tibio-femoral joint.

Movements - Active and passive knee movements limited in capsular pattern i.e. flexion beyond 90° and terminal extension beyond 20° is limited. Crepitus present. Resisted isometric knee flexion-extension is painless.

Joint play: - Passive patellar movements are limited. Tibial gliding over femur are also limited. Internal rotation of tibia is limited and painful due to tightness of postero-medial capsular tightness.

Palpation - Warmth, swelling and joint line tenderness present in joint effusion stage. Localized pain may be present due to irritation of the soft tissue by the osteophyte.

Management

Avoid or minimize weight bearing activities with knee bend and reduce body weight to reduce joint loading.

In the presence of joint effusion high voltage pulsed galvanic stimulation, Interferential therapy, strong faradic electrical stimulation or TENS can be used for pain relief.



Electrotherapy modalities (Electrical stimulation, Ultrasound therapy)

When knee movements are limited in capsular pattern traction, capsular stretching, soft tissue massage, mobilization techniques etc are useful to stretch the tight capsule, which is the source of pain. Ultrasound or short wave diathermy before manual technique is helpful to improve the extensibility of the short collagen tissues. Tight posterior capsule can be stretched in supine position by placing one hand under the ankle and other hand over the anterior aspect of lower thigh; with the hip in neutral rotation press the knee down while raising the heel up.



Tibio-fibular Mobilization technique

In prone position with pillow under the lower thigh and ankle out of the bed, fix the thigh by one hand and hold above the ankle by the other hand, rotate the leg inward and extend the knee with traction so that patient will experiences discomfort on the back of the knee due to stretching of tight capsule.



Self posterior capsule stretching



posterior

In prone position with pillow under the lower thigh, leg and foot out of the bed, fix the thigh by one hand and leg is stabilized between Therapist's legs. Rotate the leg inward, apply traction to the leg and move the proximal tibia backward to forward with the other hand. It mobilizes the knee to restore full knee extension.

Ankle traction with pillow under the ankle, hip in neutral rotation and fixation of the lower thigh by a belt stretches the short periarticular structures. Soft tissue massage

around the patella and on the back of the knee, more over the postero-medial aspect is effective in reducing pain and improving joint mobility.

Stretching of hamstrings, TA, hip adductors, hip external rotators is also essential to relief pain and re-orientation of the joint. Strengthening of the quadriceps and hamstrings also helps in deloading the joint and provides joint protection.



Self stretching of Hamsting & adductors



Strengthening of the quadriceps muscle



Strengthening of the Hamstring muscle

Associated causative factors such as pronated foot, leg length discrepancy, genu varum etc. can be managed by modified foot wear that will realign the knee and deload the involved compartment of tibio-femoral joint.

Knee orthosis realign the medio-lateral malalignment i.e. varus so that medial compartment can be deloaded and further degeneration can be minimized. One can use walking stick to reduce transmission of body weight through the joint.

Nancy et al studied the effectiveness of manual physical therapy & exercise in OA of knee joint. In a randomized controlled clinical trial 83 subjects were assigned into 2 groups. The treatment group received manual therapy to the knee as well as to the lumbar spine, hip & ankle as required and performed a standardized knee exercise programme in the clinic & at home. The placebo group had sub therapeutic ultrasound to the knee joint for 4 weeks. The treatment group showed significant improvement clinically & statically at 4 weeks & 8 weeks but no improvement occurred in control group.

Huang et al examined the effect of ultrasound in isokinetic muscle strengthening exercises on functional status of 120 subjects with OA knee joint

The author concluded that pulsed US could enhance the therapeutic effect of isokinetic strengthening exercises for training periarticular soft tissue pain in patients with knee OA.

Snyder –Mackler L et al 1995 suggested that closed kinetic exercise alone may not provide an adequate stimulus to permit normal function of the knee. Subjects who performed OKC knee extension with high-intensity electrical stimulation demonstrated greater increases in quadriceps femoris muscle torque compared to subjects performing CKC alone

Deyle et al 2000 examined the effectiveness of manual therapy and exercise in patients with knee osteoarthritis. . A total of 83 subjects (mean age 60 years) were

assigned randomly to treatment group or a placebo-control group (sub therapeutic ultrasound) who received treatment 2 times a week for 4 weeks

The intervention group received a variety of treatments including active range of motion, stretching, strengthening, and manual physical therapy techniques. These techniques were done primarily to the knee, but also to the back, hip, and ankle if limitations were found. The treatment group showed significant improvements in 6 minutes walk distance and WOMAC scores at 4 and 8 weeks. The control group did not show any changes. The final measurement taken one year later indicated that WOMAC scores remained lower than baseline values in the exercise group

O' Reilly et al examined the effects of a home exercise quadriceps strengthening program on knee pain and 191 subjects with knee joint osteoarthritis. All the subjects were randomized into intervention and control group. The intervention group performed exercise program for 6 months which includes quadriceps sets, short arc knee extension and flexion, resisted knee extension and stepped up and down. Results showed improved quadriceps strength, reduced pain during activity, improved in WOMAC score for pain and physical function and improvement in anxiety and disability.

Hurley & Scott investigated the effect of relatively brief exercise regime on knee joint proprioception, quadriceps strength and disability in patients with knee joint osteoarthritis. 60 patients participated in the exercise program with a control group of 37 patients who did not receive any exercise. The exercise regime was consists of 2 exercise sessions/week for 5 weeks with each session lasted for 30 minutes. Exercise consists of maximal voluntary isometric quadriceps contractions, 2 minutes cycling on static cycle , 1minute isotonic knee extension and flexion using thera band, 3 functional exercise (sit-stand, step up, step down) and 3 balance and co-ordination exercise(unilateral stance, balance board) for 1 minute. Results indicated statically significant improvement in all variables in experimental group.

PATELO-FEMORAL DYSFUNCTION

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There are two common dysfunctions of the patellofemoral joint - patella becoming too compressed against the femur and moving too far laterally in the intercondylar groove. Both conditions cause abrasion of the cartilage, leading to inflammation and degeneration. It becomes too compressed due to increased flexion of the knee, which is caused by sustained tension in the hamstrings, iliotibial band (ITB), and gastrocnemius or by a shortened joint capsule. This compression force is dramatically increased when a person climbs stairs or gets up from chair.

If the patella is compressed laterally in the trochlear groove, it is called patellar tracking dysfunction. The resultant of quadriceps muscle force in the frontal plane faces upward and outward. Therefore with active quadriceps contraction patella tracks upward and outward. With increase in Q-angle there is more risk of lateral tracking and subluxation. Lateral subluxation of patella is prevented by anterior prominence of femoral condyle, deep patellar groove and dynamically by the force of contraction of vastus medialis obliquus. Genu valgus, IT band tightness, femoral anteversion, external tibial torsion, foot pronation, high riding small patella, weakness of vastus medialis, an overdevelopment of the vastus lateralis, lateral retinaculum, and tensor fascia lata (TFL) etc. are the factors that precipitate to lateral tracking. This restricts the medial glide of patella and increasing the pressure of the patellofemoral joint.

Soft tissue tensions, medial and lateral retinacular, particularly the two distal expansions of the iliotibial band, joint capsule and ligaments all contribute to maintain patella alignment. It has been proposed that a tight iliotibial band

through its attachment of the lateral retinaculum into the patella could cause lateral patellar tracking, patella tilt & compression.

It has been well established that shortening of the lateral retinaculum is a common condition in patients with patella femoral dysfunction. This causes progressive tilting of the patella if the medial static stabilizers are stretched or the dynamic stabilizer (VMO) is weak. This leads to abnormal patellar alignment i.e. lateral displacement and lateral tilt. As the patellofemoral joint is subjected to elevated stress this causes patellofemoral pain. Medial glide of patella can be used extensively to stretch tight lateral retinaculum.

As the patella tracks laterally, the odd medial facet of patella, which is normally non-articular, becomes articular. The non-articular odd medial facet of patella is soft as it is not subjected to stress. With lateral tracking of patella, the odd medial facet of patella will be subjected to compressive loading and gets traumatized. Each time the knee extends, patella tracks laterally and the lateral PFJ gets compressed and degenerates over time. Gradually the lateral patellar retinaculum becomes tight and medial patellar retinaculum will be subjected to tensile stress, so there develops chronic inflammation.

RISK FACTOR FOR PATELLOFEMORAL OSTEOARTHRITIS

Risk factors can be divided into two pathogenic mechanisms. They are systemic factors influencing a generalized predisposition to osteoarthritis and local factors resulting in abnormal biomechanical loading at specific joints.

Systemic factors include person's age, sex, inherited susceptibility to osteoarthritis, obesity and other factors some of which yet to be identified.

Local factors include varus-valgus malalignment, patellar malalignment, anatomical abnormalities, chondromalacia patella, weakness of VM, increase

Q-angle, Increased anteversion, Abnormal foot pronation, and repetitive use of the joint with hyperflexion such as squatting, stair climbing etc, including that caused by occupational activities

The etiopathogenesis of osteoarthritis is widely believed to be the result of local mechanical factors along with the context of systemic susceptibility.

Clinical features

Patient complains gradual onset of anterior knee pain during activities involving loading the knee in extension due to the pull of the inflamed medial patellar retinaculum and also during activities involving hyperflexion of knee due to stretching of tight lateral patellar retinaculum and. Pain is reproduced or aggravated by stair climbing, squatting, sitting with knee bend etc. activities. Patient may present the history immobilization following any injury or surgery.

Inspection

Structural mal-alignment such as Genu valgus, femoral anteversion, external tibial torsion, foot pronation in weight bearing, high riding small patella may be present. Atrophy of vastus medialis obliquus may be present.

Movements

Active knee extension demonstrates excessive lateral tracking of patella at 30°-10° of extension. Passive medial gliding is painful and limited; reduced patellar tilting indicates tightness of lateral patellar retinaculum. Weakness of vastus medialis can be elicited by checking resisted quadriceps contraction at 30° of extension and comparing it with the sound side. Clarke's sign may be positive. Obers's test may be positive indicating IT band tightness.

Warmth and tenderness may be present on the back of medial aspect of patella and adductor tubercle, where medial patellar retinaculum is attached.

Management

Strengthening of vastus medialis is very important as it dynamically checks the lateral tracking of patella. Static quadriceps exercise, short arc quadriceps exercise from 30°-0°, multi-angle quadriceps exercise 30°, 10° and 0° , close kinetic knee extension 30°-0°, SLR are useful. Strong faradic electrical stimulation to VMO also helps for strengthening.



Straight leg raising exercise

Stretching of the tight lateral structures is very important as it pulls the patella during knee extension. Lateral to medial gliding of patella, medial patellar tilting etc. help to stretch the tight lateral patellar retinaculum. Taping of patella medially to lift the lateral border of patella may help for normal tracking of patella.



Patello-femoral mobilization

Ultrasound can be applied on the tight lateral patellar retinaculum to improve its extensibility before mobilization and on the medial patellar retinaculum to resolve the chronic inflammation.

Depending on the cause, stretching of IT band, foot wear modification to correct foot deformity and genu valgus, knee orthosis to correct excessive genu valgus, knee orthosis to normally position the patella can be considered.