

A Review on Osteoarthritis

Aswathy Anilkumar Sheejamol*¹, Dhanya S², Prasobh G.R³

¹Bpharm Student, Sree Krishna College Of Pharmacy And Research
Centre, Parassala, Thiruvanthapuram, Kerala, India. 695502

²Associate Professor, Department Of Pharmaceutical Chemistry And Analysis, Sree Krishna College Of
Pharmacy And Research Centre, Parassala, Thiruvanthapuram, Kerala, India. 695502

³Principal, Sree Krishna College of Pharmacy and Research
Centre, Parassala, Thiruvanthapuram, Kerala, India. 695502

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ABSTRACT: Osteoarthritis is a common, slowly progressive disorder affecting primarily the weight-bearing diarthrodial joints of the peripheral and axial skeleton. The etiology of OA is multifactorial, with inflammatory, metabolic, and mechanical causes. The Osteoarthritis cannot be cured, and appropriate management includes a combination of non-pharmacological and pharmacological measures with the ultimate goal to alleviate pain and improve functional status. No current treatment can prevent either initiation or progression of osteoarthritis. The symptoms are associated with significant functional impairment, as well as signs and symptoms of inflammation, pain, stiffness and mobility loss. Conservative treatment has documented the effectiveness of exercise in reducing pain and disability. Osteoarthritis is a common, slowly progressive disorder affecting primarily the weight-bearing diarthrodial joints of the peripheral and axial skeleton.

KEYWORDS: early knee osteoarthritis, chronic disease therapy, musculoskeletal diseases, pain, medical device, osteoarthritis, related factors

I. INTRODUCTION

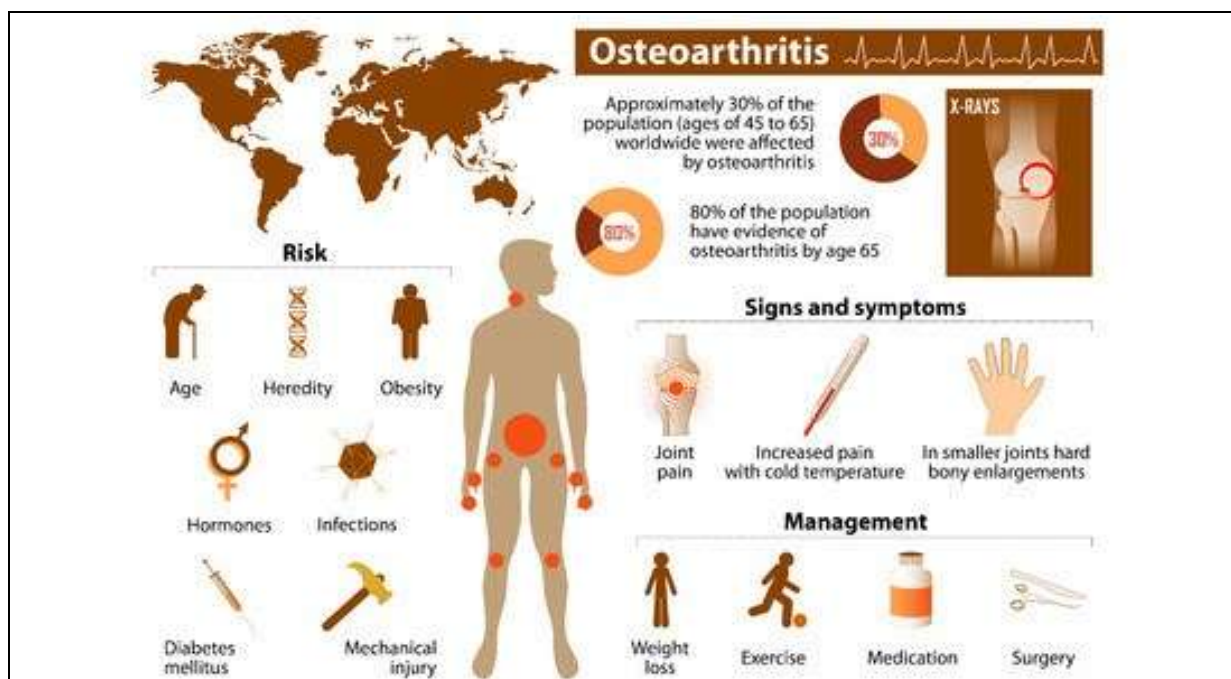
Osteoarthritis (degenerative joint disease or

degenerative arthritis or OA or wear-and-tear arthritis) is the most common form of arthritis. It's affecting millions of people around the world. Osteoarthritis is a common, slowly progressive disorder affecting primarily the weight-bearing diarthrodial joints of the peripheral and axial skeleton. It causes significant pain and functional disability, and increases costs to our health care systems. Osteoarthritis can occur in the hands, neck, lower back, knees, and hips. Knee osteoarthritis (KOA) is one of the most common types that occurs around the world [1].

Osteoarthritis is currently considered as a disease caused by dynamic reaction of joint to a variety of biomechanical and biochemical factors [2]. Cartilage has no pain sensation.

Osteoarthritis is characterized in two categories as primary and secondary. Primary osteoarthritis is age related and occurs in old age while secondary osteoarthritis may occur due to accidental injuries or as a side effect of pre-occurring diseases [2].

The best way of management of osteoarthritis is regular exercise and maintenance of diet.



Osteoarthritis Management

[5]. Osteoarthritis is a common, slowly progressive disorder affecting primarily the weight-bearing diarthrodial joints of the peripheral and axial skeleton. It causes significant pain and functional disability, and increases costs to our health care systems.

II. ETIOLOGY

The etiology of osteoarthritis is unknown. In primary OA, the causes elude determination of any particular identifiable factors. [4] In secondary OA, metabolic conditions such as hemochromatosis, acromegaly, and deposition of crystalline calcium can be identified. There are a wide variety of factors predisposing an individual to this condition including the following:

- Increasing age.
- Gender.
- Genetic predisposition.
- Congenital abnormality.
- Obesity.
- High bone mineral density.
- Chronic repetitive joint injury.
- Menopause or irregular periods.
- Sports stress.

III. EPIDEMIOLOGY

[3]. Osteoarthritis of the knee is the most common form of joint diseases and prevalence of both radio graphically evident and symptomatic. The women are suffering knee OA more than men's at the age of 50. The female have higher prevalence than males (11.4% vs. 6.8%). The gender difference in prevalence has recently been emphasized in meta-analyses. OA is ranked tenth, slightly below diabetes, among causes of disability-adjusted life years (DALYs). Pain is likely cause disability more when the weight-bearing joints are affected. An inherited defect in type II collagen genes is linked to the development of early onset polyarticular osteoarthritis [5].

IV. CLASSIFICATION

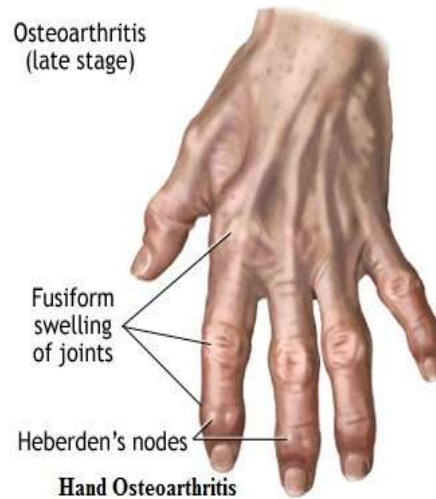
The Types of Osteoarthritis Based on the classification of India's Osteoarthritis literature by type of research,

It was found that:-

- Knee Osteoarthritis.
- Hip Osteoarthritis.
- Spine Osteoarthritis.
- Wrist Osteoarthritis
- Finger Osteoarthritis.



Osteoarthritis (late stage)



Osteoarthritis of Spine



V. PATHOGENESIS

Osteoarthritis is also known as a “wear and tear” disease of cartilage. The articular cartilage is get affected by the dynamic interactions between biochemical, morphologic, and mechanical processes. The diseases are affected clearly to the subchondral bone, ligaments and the entire joint. Disrespect to the articular cartilage can result from repetitive damage with time. Traumatic injury to articular surfaces initiates the cascade leading to release of inflammatory cytokines (tumor necrosis factor [TNF], IL-1), nitricoxide, and enzymes that break down the extracellular matrix. The breakdown of extracellular matrix leads to the cartilage that is less elastic and not able to support joint loads, and stiffening of subchondral bone. The cartilage is not able to support forces, with diminished efficacy for providing joint lubrication and weight distribution across the joint. Cartilage is a vascular, however, and contains chondrocytes that under normal conditions are

responsible for cartilage breakdown and repair.¹⁵ In early osteoarthritis, chondrocytes attempt to repair joint damage by forming osteophytes, which try to stabilize the joint to alter the biochemical properties of cartilage. An increased surface area over which to distribute the forces across the joint by the formation of osteophytes.[6]. Bony outgrowths may be responsible for the patient’s pain and limited mobility. The less viscous cartilage is structurally weaker than normal cartilage. The proteoglycan concentrations make less with shorter glycosaminoglycan side chains resulting in decreases of net aggregate proteins. Type I collagen in the extracellular matrix increases, and keratin sulphate will be decrease. Aggrecanases and collagenases enzymes are degrading proteoglycans and collagen. The control of the seen zymes is complicated by enzymatic activation of latent proteins and inactivation by proteinase inhibitors. In OA, production of proteinases is increased. Matrix metalloproteinases

(MMP), MMP-1, MMP-8, and MMP-13 are cleaved collagen. Of the three, MMP-13 maybe the most important in OA because it preferentially degrades type II collagen. Up regulated by IL-1 and TNF, MMPs cleave collagen and breakdown other important elements of the extracellular matrix. Eventual cartilage destruction and erosion occurs due to the imbalance between cartilage maintenance and degradation[11].

VI. PATHOPHYSIOLOGY OF OSTEOARTHRITIS

Osteoarthritis is a complex, chronic inflammatory disease of synovial joints, involving the articular cartilage, a unique tissue between the ends of bones in the joints. Osteoarthritis is metabolically active, dynamic process including both cartilage destruction and repair. These processes may be initiated by several biochemical and mechanical insults. The first osteoarthritis change occurring articular cartilage include a decrease in the superficial proteoglycan content, deterioration of superficial collagen fibrils, and an increases in the water content. The loss of proteoglycans and collagen results in make less cartilage stiffness. Subsequently, the chondrocytes increase the synthesis of cartilage matrix accelerates, and the thickness of cartilage may even increase [17].

At the same time, the increased formation

and resorption of the subchondral bone may leads to the calcified cartilage and subchondral bone to get thicker.

Ultimately, due to diminished repair capabilities of chondrocytes, the concentration of proteoglycans decreases and collagen fibrillation declines. This may leads to splits of the cartilage extending down to bone.

The disrupted collagen network cannot be regenerate, and this pushes the OA tissue to the point of no return. On the other hand, postulated that the repetitive impulsive loading my first induce trabecular micro fractures in the subchondral bone in an attempt to dampen impact forces. As a consequence, the overlying cartilage may become overloaded and breakdown resulting in cartilage degeneration and loss[18].

VII. RISK FACTORS

The cause of osteoarthritis remain unknown, though there is a clever evidence for major risk factors, such as obesity, joint, trauma, and heavy work load. The risk factors can be divided into systemic (for example: age, gender, genetics and overweight) and local biochemical factors, such as joint injury and malalignment, overweight and muscle weakness[7].



Risk Factors Of Osteoarthritis

- **Aging** is the most significant risk factors for knee osteoarthritis. Knee osteoarthritis is more common in obese subject than in subjects of

normal weight, for example , obese women with body mass index(BMI) of 30-35kg had a four times higher risk for knee Osteoarthritis

than non-obese women.

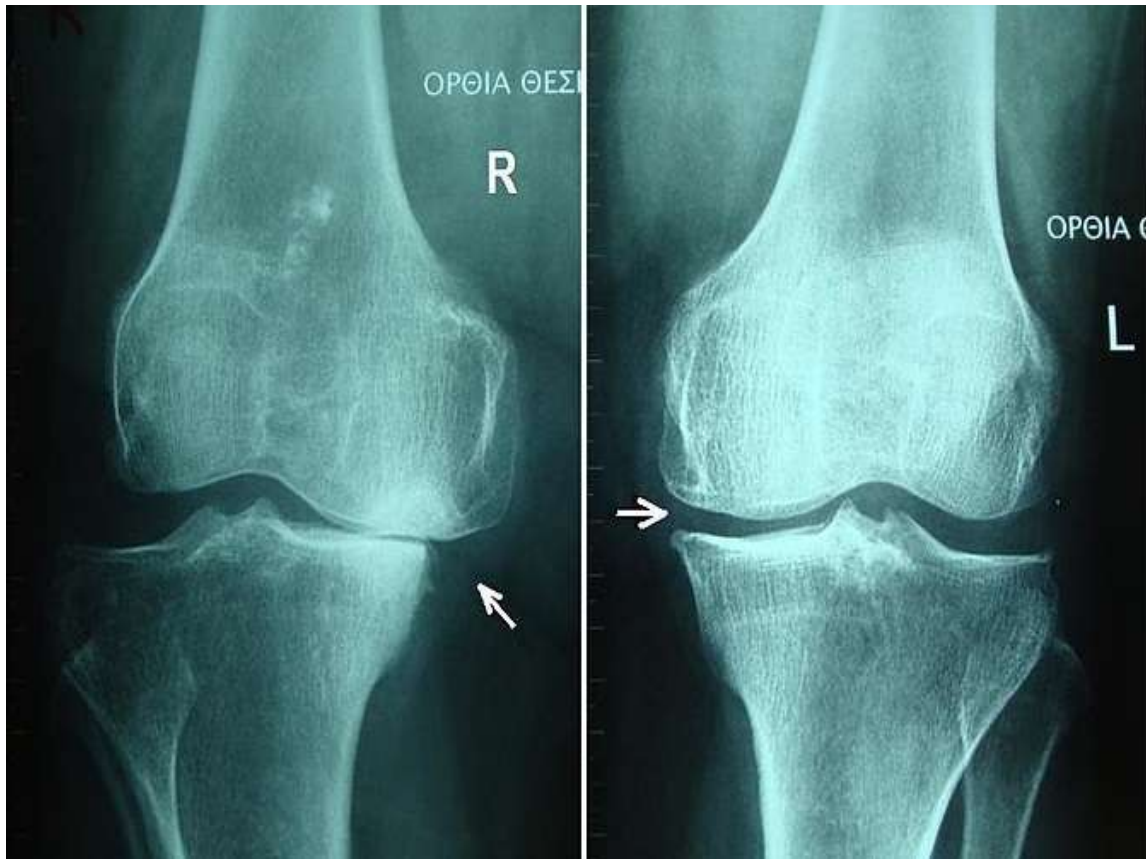
- **Obesity** is also a major risk for the incidence of bilateral knee osteoarthritis. The effect of obesity on OA has been mediated through the increased mechanical loading of the knee and hip. This would leading cartilage damage in these weight bearing joints.
- **Joint injury.**
- **High bone density**
- **Reduced muscle strength**
- **Malalignment of joints**
- **Excessive mechanical stress**
- **Sports and other trauma to the joints**
- **Genetic predisposition** – a study of monozygotic twins aged 48 to 70 years, having identical genes, showed 65% influence of genetic factors in developing osteoarthritis
- **Meniscus deficient knees** – the prevalence of OA seems to be high among former athletes from team and individual sports when compared to the general population and other occupational sectors.
- **Childhood hip disorders** e.g. acetabular dysplasia, congenital hip dislocation, epiphysiolysis (abnormal separation of an epiphysis from the bone shaft), and Legg-Calve²-Perthes (a disorder initiated by a disruption of blood flow to the head of the

femur) have been implicated in the development of early onset OA of the hip.

- **Coxa valga** (a deformity of the hip) with acetabular dysplasia.
- **Degeneration** of the acetabular rim triggered by femoroacetabular impingement (the ball shaped femoral head rubs abnormally or does not permit a normal range of motion in the acetabular socket).

VIII. DIAGNOSIS

- Diagnosis is made through patient history, physician examination, radiologic findings, and laboratory testing.
- American College of Rheumatology (ACR) criteria for classification of OA of the hips, knees, and hands include presence of pain, bony changes on examination, normal erythrocyte sedimentation rate (ESR), and radiographs
- For hip OA, the patient must have hip pain and two of the following: (1) ESR less than 20mm/h, (2) radiographic femoral or acetabular osteophytes, and/or (3) radiographic joint space narrowing.
- For knee OA, the patient must have knee pain and radiographic osteophytes in addition to one or more of the following: (1) age more than 50 years, (2) morning stiffness lasting 30 minutes or less, (3) crepitus on motion, (4) bony enlargement and bony tenderness, and/or (5) palpable joint warmth.
- ESR may be slightly elevated if inflammation is present. Rheumatoid factor is negative.



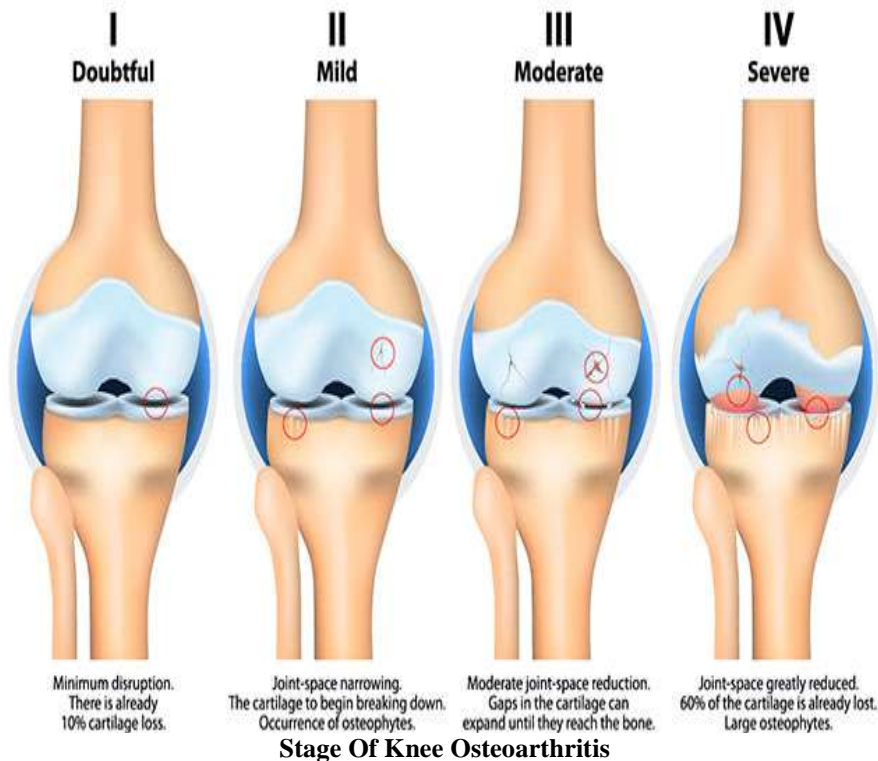
Diagnosis Of Osteoarthritis By X-Ray

The diagnosis can be established by clinical examination, and it can be confirmed by X-rays.

Knee OA can be sub-divided into 5 grades:

- Grade 0: This is the stage where “normal” knee health shows.
- Grade 1: this is the stage where Very minor bones spur growth and is not experiencing any pain or discomfort.
- Grade 2: This is the stage where people experience symptoms for the first time. It shows pain after a long day of walking and also senses a greater stiffness in the joint. Grade 2 is a mild stage of the condition, the X-rays already shows the greater bone spur growth. But the cartilage will remain at a healthy condition.
- Grade 3: this is the stage of Moderate OA. This stage shows frequent pain during movement, joint stiffness, especially after sitting for long periods and in the morning. The cartilage between the bones shows obvious damage and the space between the bones are getting smaller.
- Grade 4: This is the severe stage of OA. The joint space between the bones will be reduced, the cartilage will be completely gone and the synovial fluid will be decreased. This stage is associated with high levels of pain and discomfort during walking or moving the joint.

STAGE OF KNEE OSTEOARTHRITIS



IX. CLINICAL MANIFESTATION

Osteoarthritis is traditionally classified by **etiology into idiopathic and secondary forms.**⁵ **Idiopathic** can be further divided into **localized and generalized** depending on the number of joints involved. **Localized osteoarthritis** most commonly affects the hands, feet, hip, knees and spine, and less commonly the shoulder, temporomandibular, sacroiliac and wrist joints. The movement and loading on the joint is increasing the pain, and radiate beyond the joint itself, as in leg pain associated with spinal disease, and knee pain radiating from the hip. Stiffness in the early morning lasts for less than 30 min, unlike that due to rheumatoid arthritis; it may also occur after periods of rest and throughout the day. In the hands, the most commonly affected joints are the distal interphalangeal joints or Heber den's nodes, the proximal interphalangeal joints (Bouchard's nodes) and the base of the thumb, the first carpometacarpal joint[9].

X. INVESTIGATIONS

Osteoarthritis is primarily diagnosed by its clinical presentation. There is a lack of conformity between symptoms and radiological

findings. Normal cartilage is smooth, white and glistening, while osteoarthritic cartilage is yellowed, irregular and ulcerated¹. Synovial fluid analysis should be carried out if one suspects infection or crystal arthropathy such as gout or pseudo gout. Blood tests reveal a normal ESR and CRP. Inflammatory markers are including C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR). It's not always elevated in active disease and is useful for monitoring response to treatment. Rheumatoid factor (RF) is an auto antibody directed against the host immunoglobulin and is most commonly found in rheumatoid arthritis.⁽¹²⁾ Routinely performed tests only detect immunoglobulin M rheumatoid factor (IgM RF) which is present in 75–80% of patients with rheumatoid arthritis (termed seropositive disease) and 5% of normal subjects. The Patients who do not have a detectable RF are called as 'seronegative' Anti-cyclic citrullinated peptide antibodies (anti-CCP antibody) are a more specific test for rheumatoid arthritis with a specificity of 90–96% compared with the specificity of IgM [8].

RF of 85 %.[10].Antinuclear antibodies (ANA) and extractable nuclear antigens (ENA) are useful for

establishing the differential diagnosis, such as other connective tissue diseases presenting or associated with arthritis. ANA is almost universally positive

in systemic lupus erythematosus and only positive in 20% of patients with rheumatoid arthritis.

XI. SIGNS AND SYMPTOMS OF OSTEOARTHRITIS

Areas commonly affected by osteoarthritis:

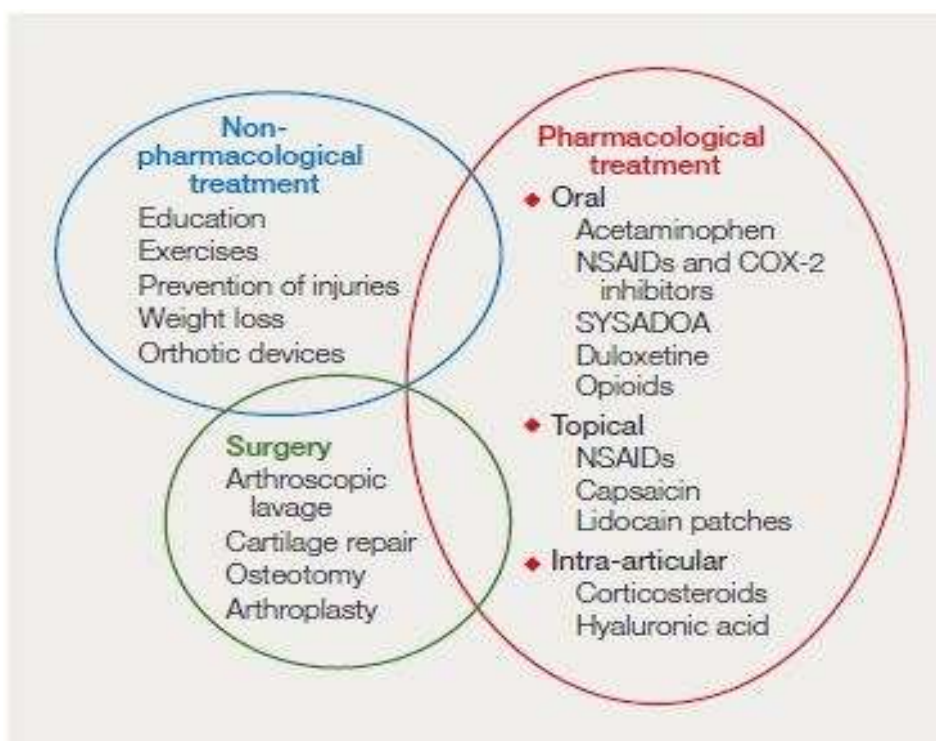


Signs And Symptoms Of Osteoarthritis

<u>JOINTS AFFECTED</u>	<u>SIGN AND SYMPTOMS</u>
<u>Hand:-</u>	Heberden nodes at distal interphalangeal joints. Bouchard’s nodes at proximal interphalangeal joints, Osteophytes at first metacarpal joint, Give characteristic square appearance to hands Tenderness over carp metacarpal joint of the thumb.
<u>Hip Pain:</u>	In groin during weight lifting exercises Stiffness, Pronounced following activity Pain in gluteal region Limited joint movement, especially internal rotation.
<u>Knee Pain:</u>	Associated with climbing stairs Transient joint effusion Lateral instability Genu varum, Crepitus associated with range of motion.

<p><u>Spine:</u></p>	<p>Typical lumbar involvement at L3 and L4 Paraesthesia Loss of sensation at lower extremities Motor weakness from compression of nerve root Loss of reflexes Pseudoclaudication from spinal stenosis.</p>
<p><u>Shoulder:</u></p>	<p>Limited range of motion, especially external rotation Crepitus associated with range of motion.</p>

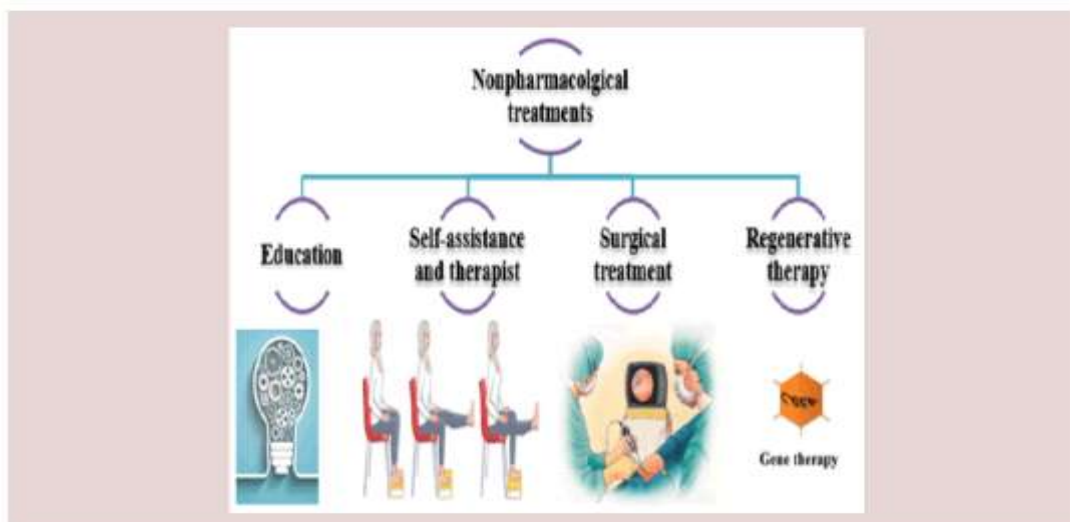
XII. MANAGEMENT OF OSTEOARTHRITIS



Management Of Osteoarthritis

Osteoarthritis cannot be cured, and appropriate management includes a combination of non-pharmacological and pharmacological measures with the ultimate goal to alleviate pain and improve functional status. No current treatment can prevent either initiation or progression of osteoarthritis[13].

Non-Pharmacological Management:



Non-pharmacological treatment of osteoarthritis

Daily use of the joints actually preserves rather than “wears out” articular cartilage and inadequate use is the commonest cause of cartilage degeneration.¹⁸ An OA-patient should agree to manage themselves by ensuring positive behavioural changes e.g. exercise, weight loss, and use of suitable footwear (including those with shock-absorbing properties). Physical activity, e.g. walking, can reduce pain and help to maintain (or attain) a healthy weight. Excess weight adds additional stress to weight-bearing joints and by losing weight. It can reduce OA pain and limit further joint damage. Strengthening exercises build muscle around the OA-affected joints – this can ease the burden on the affected joints.⁴ Exercise also improves joint flexibility and reduces joint stiffness. Gentle stretching of joints can further improve flexibility, decrease stiffness and lessen pain. Other non-pharmacological interventions include patient education, occupational therapy, and heat and cold therapies[16].

- **Education:-**
 Patient education is an important integral part of management. The practitioner should address aspects of the disease process, benefits and risks of treatment options. Empowering the patient, by involving them in shared decision making and providing them with positive skills directed at lifestyle changes, goes a long way to ensure treatment adherence.
- **Social support:-.**
 In patients with osteoarthritis of the knee

controlled studies have shown that regular telephone contact from a healthcare worker produces significant improvement in pain and functional status. Furthermore, education of family members can improve their ability to provide social support, which also benefits the patient. These specific effects complement the generally observed improvements in wellbeing and reduced use of health care associated with social support networks.

- **Reduction of adverse mechanical factors: -**
 In knee and hip, Obesity is a risk factor for the development and progression of OA. Obesity is one of the strongest modifiable risk factors for OA and weight reduction is an effective primary and secondary disease prevention strategy. Weight loss improves pain and function. The correct dietary habits (eat correctly, regularly and less) and exercise can reduce the weight loss. Many osteoarthritis patients having other chronic cardiac and metabolic disease and the benefits of weight loss are substantial. The patients with lower limb OA should be advised about appropriate footwear, i.e. a shoe with soft thick soles are used and no raised heel is recommended. Lateral or medial wedged insoles can be used to reduce pain and improve function in patients with medial or lateral tibiofemoral OA, respectively.
- **Assistive devices: -**
 Patients with osteoarthritis reduce mechanical loading and pain by the use of a cane, frame or wheeled walker. Patient should be

educated about the usage of canes. The cane or crutch should be held in the hand contra lateral to, and moved together with, the affected limb. This should result in elbow flexion of about 200. Knee braces can be used in patients. Overuse of braces worsens joint instability by contributing to muscle atrophy. They should be used only when there is a flare of inflammation, to protect the joint during unusual activity and when all other treatment modalities have failed[17].

- **Manual Therapy:-**
- **Taping: -**Taping the knee, in particular the patella is a physiotherapy treatment strategy recommended in the management of knee OA by some clinical guidelines. The mechanism by which taping reduces pain is not clear, but may include changes in patellar alignment and enhanced function and activation of muscles.
- **Electrotherapy: -**Transcutaneous electrical nerve stimulation (TENS) is recommended in most guideline as safe adjunctive modalities for pain relief. The acupuncture may provide relief to patients and there is less universal support for its use.

- **Therapeutic Exercises:-**
It is a form of physical activity that is provided under the supervision of appropriate health professional for specific treatment goals.[18] The effectiveness of exercise and physical activity for individuals with knee osteoarthritis may help to reduce the pain and improve disability.

It decreasing risk for a wide range of disease and conditions, such as cardiovascular disease, osteoporosis, falling, cancer, diabetes, blood pressure and osteoarthritis[4].

Main reasons for prescribing exercise in general include:

- (1) Achieving therapeutic goals
- (2) Improving general health and reducing secondary disability.
- (3) Modifying possible risk factors in disease progression.

Minor summarized the potential benefits of physical activity and exercise on osteoarthritis as follows:

1. Minimizes the pathological process that takes place in the OA joint.
2. Decreases or reduces the impairments that occur

from OA by reducing the main impairment factors. Exercise will helps in decreasing the pain, improving the strength and endurance, and improving the range of motion and connective tissue elasticity.

3. Decreases the functional limitation by improving walking speed, gait and physical activity and decreasing the depression and anxiety.

Although, activity avoidance by knee osteoarthritis patients is common, exercise is an effect non-pharmacological treatment for knee OA. The systematic reviews of non-pharmacological interventions have documented the effectiveness of exercise in reducing the pain and disability. The muscle endurance increased, improves proprioceptive acuity and decrease arthrogenic muscle inhibition of the quadriceps by the help of exercise. Quadriceps weakness and disabling impairments is the most common seen in individuals with knee osteoarthritis. For undertaking basic activities of daily living such as standing and walking, sufficient quadriceps and hamstrings strength, both isometric and dynamic, is essential[15].

3 factors are present in which contribute to knee extension and flexion weakness in those with knee OA:

- **Muscle atrophy,**
- **Failure of voluntary muscle activity**
- **Apparent weakness from increased antagonist muscle co contraction.**

- **Occupational therapy:**
Occupational therapy provides a means of social support and educating patients.¹⁰ There are few evaluations of specific interventions including the provision of walking aids, orthoses, and splints. In a single trial of patients with osteoarthritis of the hand the combination of a hand exercise programmers, provision of splint age, and non-steroidal anti-inflammatory drugs improved disability in 49% of treated patients, but the study could not dissect the relative benefits of occupational therapy from those of the drugs[17].

Pharmacological Management:

- **Simple analgesia.**
Examples of simple analgesia:
 - Acetaminophen (Tylenol)
 - Duloxetine (Cymbalta)
- **Non-steroidal anti-inflammatory drugs.**
Examples of NSAIDs include:

- Aspirin
- Ibuprofen
- Naproxen sodium and naproxen
- Diclofenac

Other prescription NSAIDs for OA:-

- celecoxib (Celebrex)
- piroxicam (Feldene)
- indomethacin (Indocin)
- meloxicam (Mobic Vivlodex)
- ketoprofen (Orudis, Ketoprofen ER, Oruvail, Actron)
- sulindac (Clinoril)
- diflunisal (Dolobid)
- nabumetone (Relafen)
- oxaprozin (Daypro)
- tolmetin (Tolmetin Sodium, Tolectin)
- salsalate (Disalcid)
- etodolac (Lodine)
- fenoprofen (Nalfon)
- flurbiprofen (Ansaid)
- ketorolac (Toradol)
- meclofenamate
- mefenamic acid (Ponstel)
- **COX-2 inhibitors. (cyclo-oxygenase-2 selective non-steroidal anti-inflammatory drugs)**
- **Topical analgesic.**

Examples of topical analgesic:

- Capsaicin.
- Diclofenac sodium gel and solution (Voltaren, Flector Patch, Solaraze, Pennsaid).
- Lidocaine patch.
- Methyl salicylate and menthol (Bengay).
- Trolamine (Aspercreme)
- **Chondroprotective Agents.**

Analgesics, non-steroidal anti-inflammatory drugs, and cyclo-oxygenase-2 (COX-2) inhibitors:

Several short term studies shown that non-steroidal anti-inflammatory drugs are more effective than placebo in reducing pain and improving function, but there have been few studies that have lasted longer than two years[7].

Non-steroidal anti-inflammatory drugs are poor because of adverse effects, while those with paracetamol are poor because of sub optimal pain relief. [8]. 20-30% of deaths from peptic ulcer disease in elderly people may be related to use of

non-steroidal anti-inflammatory drugs. There is evidence that misoprostol and proton pump inhibitors reduce the risk of serious upper gastrointestinal injury induced by non-steroidal anti-inflammatory drugs.

Adjunctive use of H2 blockers has been shown only to reduce the incidence of duodenal ulceration. The cost utility of prophylactic use of any of these agents, however, is controversial. It is recommended that non-steroidal anti-inflammatory drugs are initiated only after consideration of side effects and counselling of the patient; the prescription should be reviewed every six months [10].

Cyclo-oxygenase, an enzyme involved in the conversion of arachidonic acid to prostaglandins, exists in two isoforms:

COX-1, a constitutive isoform, predominates in the stomach and produces cytoprotective prostaglandins.

COX-2, an inducible isoform, predominantly involved in the inflammatory cascade, gives rise to articular pain, swelling, and stiffness.

Novel therapeutic agents have been developed that act as specific inhibitors of the cyclooxygenase-2 isoform (COX-2 inhibitors).

Treatment algorithm for osteoarthritis. (COX, cyclooxygenase; GI, gastrointestinal; IA, intraarticular; NSAID, non-steroidal anti-inflammatory drug; OA, osteoarthritis; PPI, proton pump inhibitor.)

Topical Treatment:

The patients with osteoarthritis who have inadequate pain relief or who cannot tolerate systemic therapy are doing the topical treatment. Non-steroidal anti-inflammatory drugs and capsaicin are the two best topical agents for the treatment[11].

A recent meta-analysis concluded that 65% of patients allocated to active treatment with topical non-steroidal anti-inflammatory drugs had a good response compared with only 30% of patients receiving placebo.

The topical non-steroidal anti-inflammatory drugs are effective and safe for patients with osteoarthritis. Capsaicin is a natural compound. ⁶ It

reversibly depletes the stores of the neurotransmitter substance P from sensory nerve endings. Thereby, it attenuates the transmission of painful stimuli from the peripheral nerve fibers to higher centres. It is well tolerated and has significantly greater analgesic effects than placebo.

➤ **Intra-Articular Therapy:**

● **Corticosteroids :**

There are important and maintained responses to the intra-articular placebo injections and arthrocentesis incorporated in these studies, such that the group treated with corticosteroids often show sustained benefit over baseline for several months.[13].

● **Hyaluronic Acid:**

It is a linear polysaccharide found naturally in synovial fluid, where it is thought to facilitate shock absorption and lubrication. Patients with osteoarthritis have a reduced concentration of hyaluronic acid, resulting in low viscosity synovial fluid and an increase in cartilage loading.

The results of most randomized controlled trials suggest superior pain relief to placebo and equivalent relief to corticosteroid injections but with a greater duration of action. The high molecular weight preparations seem to produce greater benefit than the low molecular weight preparations, although this observation needs confirmation in a parallel group randomized controlled trial. A substantial proportion of patients (up to 20%) experience a joint flare after injection, which, although transient, may cause considerable discomfort [1].

● **Tidal Irrigation:**

Irrigation of the knee joint with saline by using a wide bore needle emerged as a potential treatment for osteoarthritis⁴. A first trial has shown considerable improvement after this procedure when compared with standard medical management. A second trial has shown the compared use of tidal irrigation with formal

arthroscopic lavage and suggested the similar improvements in pain and function at three months, but the presence of a meniscal tear predicted a better response to arthroscopic intervention.

➤ **Chondroprotective Agents:**

All the pharmacological interventions aim to relieve pain and thereby improve function in osteoarthritis.³ No measures have been shown to modify the rate of structural change in cartilage or subchondral bone, which constitute the underlying disease process.

Several putative chondroprotective agents may modify structure, including chondroitin and glucosamine compounds, other glycosaminoglycan derivatives found in mammalian articular cartilage, and tetracycline.

Clinical trials are providing some justification for the use of chondroitin and glucosamine preparations but it's only for their analgesic or anti-inflammatory effects.

➤ **Surgical Treatment:**

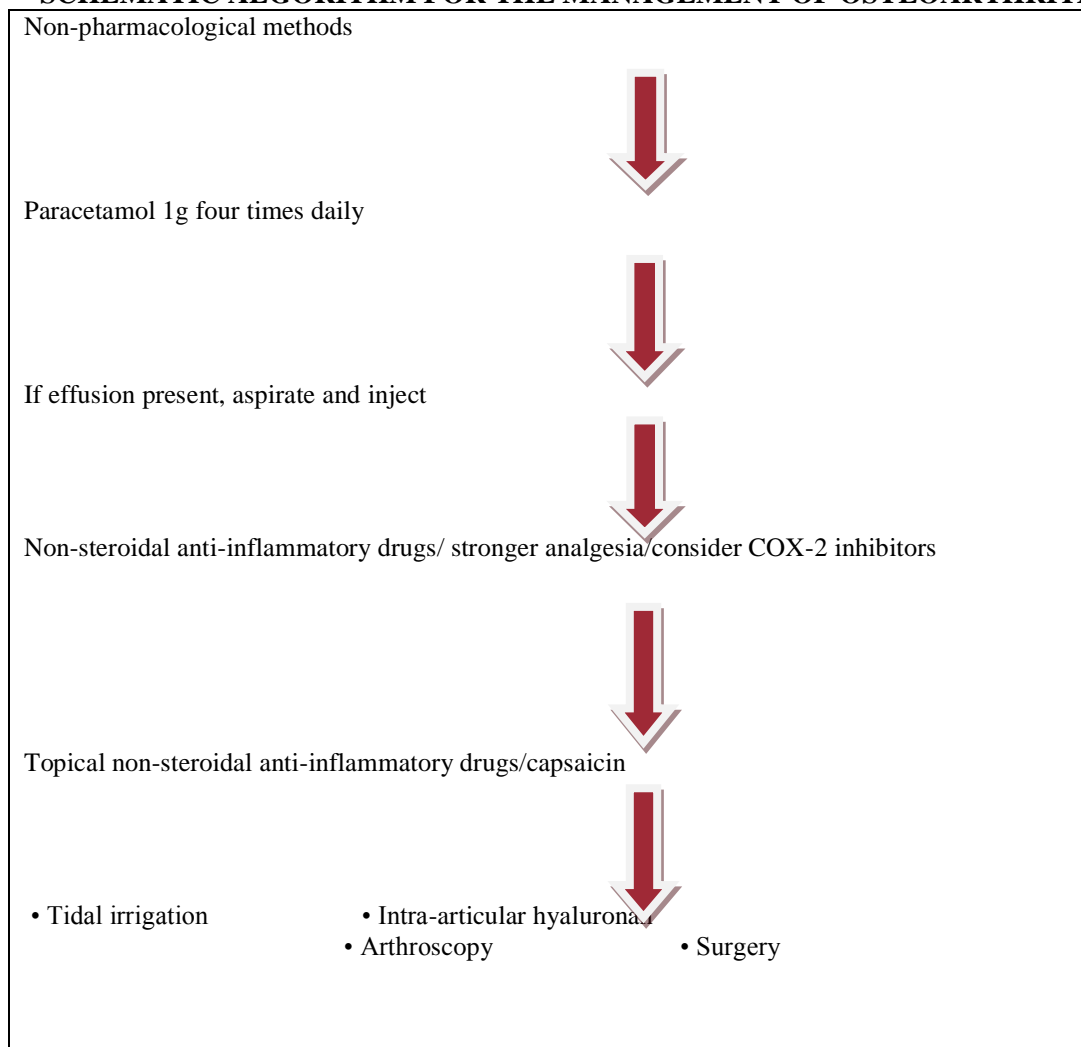
Joint replacement surgery should be considered in patients who have persistent pain and reduced function that are refractory to non-surgical therapies, and which impact markedly on their quality of life[5].

The two main surgical treatments for osteoarthritis are conservative treatments damaged and radical treatments. The radical treatments are the cartilage is replaced by an artificial endoprosthesis; this latter procedure is termed joint arthroplasty. These treatments are only offered to symptomatic patients. Arthrodesis is another surgical intervention in cases of osteoarthritis. It will sacrifice the joint's articular function and is performed on small osteoarthritic joints, such as wrists and ankles, for instance. Many patients who undergo **Total Knee Arthroplasty (TKA)**.



Joint replacement surgery

XIII. SCHEMATIC ALGORITHM FOR THE MANAGEMENT OF OSTEOARTHRITIS



XIV. EVALUATION OF THERAPEUTIC OUTCOMES

- To monitor efficacy, assess baseline pain with a visual analog scale, and assess range of motion for affected joints with flexion, extension, abduction, or adduction.
- Depending on the joint(s) affected, measurement of grip strength and 50-ft walking time can help assess hand and hip/knee OA, respectively.
- Baseline radiographs can document extent of joint involvement and follow disease progression with therapy.
- Other measures include the clinician’s global assessment based on patient’s history of activities and limitations caused by OA, the Western Ontario and McMaster Universities Arthrosis Index, Stanford Health Assessment Questionnaire, and documentation of analgesic or NSAID use.
- Patients about adverse effects from medications. Monitor for signs of drug related effects, such as skin rash, headaches, drowsiness, weight gain, or hypertension from NSAIDs.
- Obtain baseline serum creatinine, hematology profile, and serum transaminases with repeat levels at 6- to 12-month intervals to identify specific toxicities to the kidney, liver, GI tract, or bone marrow.

XV. PATIENT COUNCELLING

[11] Involving pharmacists in the care of patients with OA can improve utilization of treatments, function, pain, and quality of life for their patients. The Pharmacists can help patients to:

- Navigate treatment options.
- Establish and attain functional goals.
- Minimize factors that may lead to poor health outcomes.
- Adhere to treatment recommendations. Appropriate pain management to attain goals of improved function, health, and quality of life.
- Exercise to help control weight and maintain balance and mobility.
- Weight loss, which is particularly beneficial for patients with OA because it reduces strain on weight-bearing joints, and obesity itself is a cause of inflammation that can contribute to OA.
- Educational programs and ongoing support.

METHODS FOR SUPPORT:

MOTIVATIONAL INTERVIEWING:

It acknowledges the patient’s expertise about his or her problems and empowers the patient to develop intrinsic motivation. Self-motivation is crucial when considering the importance of exercise and weight-loss in care plans for OA. Examples of MI are shown in a Table.

<u>STRATEGY</u>	<u>EXAMPLES</u>
Asking open-ended questions	<ul style="list-style-type: none"> • How does your pain influence your ability to exercise? • What is something that you would like to be able to do that you can’t because of your pain?
Expressing empathy	<ul style="list-style-type: none"> • It sounds as though your knee pain has been very frustrating for you. • Your voice really upset that you haven’t been able to exercise as much as you want. Using affirmative
Using affirmative statements	<ul style="list-style-type: none"> • you are struggling to do things that are important to you, but you want to keep moving forward and that is why you are taking extra pain medication. Do I have that right?
Supporting self-efficacy	<ul style="list-style-type: none"> • You have done a great work making it to your physical therapy appointments.
Employing reflections	<ul style="list-style-type: none"> • I want to make sure I understand what you are saying and i heard that you want to do exercise more but you’re not sure you will be able to do exercises without taking more

	medication.
Summarizing statements	<ul style="list-style-type: none"> You are aware of the advantages of exercise for managing your arthritis and also have discussed about the risks associated with taking more pain medications. Is that correct?

XVI. CONCLUSIONS

Osteoarthritis is a major cause of pain and disability in the general population. Currently, most patients with osteoarthritis are managed in primary care. A detailed recent review of non-surgical methods found that education, exercise, systemic analgesics, non-steroidal anti-inflammatory drugs, and topical agents were likely to be beneficial; the review questioned the value of intra-articular treatment. The management of patients with osteoarthritis should be multidisciplinary and also include both education and physiotherapy. Patients do best if they are empowered in their own management. The figure outlines a possible management schedule for patients with knee osteoarthritis, although this must be tailored to fit the individual patient and will vary for different joint sites. The future holds promise for drugs that may genuinely modify structure, but these will require careful evaluation so that they may be appropriately positioned in the management algorithm.

Knee osteoarthritis (OA) is a major public health concern worldwide and one of the foremost causes of chronic disability in older adults. Preventive care is dependent upon identification of risk factors for development of incident knee OA. The symptoms are often associated with significant functional impairment, as well as signs and symptoms of inflammation, including pain, stiffness and loss of mobility. Conservative treatment has documented the effectiveness of exercise in reducing pain and disability. Evidence suggests that stretching, and strengthening exercise decrease pain and improve muscular strength, functional ability and psychological well-being. Exercise increases muscle endurance, improves proprioceptive acuity and decrease arthrogenic muscle inhibition of the quadriceps.

Effective management of OA is

multimodal and involves the participation of both the patient and a multidisciplinary healthcare team. Patient education plays a pivotal role in the success of therapy. Pharmacotherapy must be supported by relevant strengthening physical activity and weight reduction measures to conserve joint integrity. Occupational therapy, and surgical interventions as a last resort, form an important part of the treatment regimen to ensure a better quality of life for the patient.

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