



# OSTEOARTHRITIS: A BRIEF OVERVIEW OF THE TREATMENT

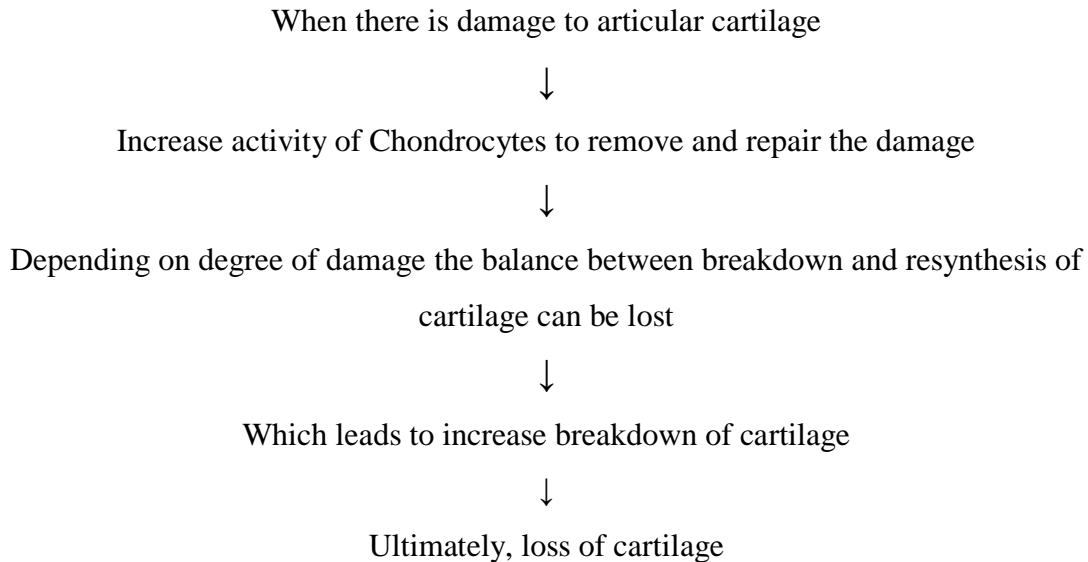
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**ABSTRACT:-** Osteoarthritis is a chronic degenerative disorder characterized by cartilage loss. It is extremely prevalent in society and is a major cause of disability. It is important to treat osteoarthritis effectively using a multidisciplinary approach tailored to the patient's needs. Pain relief, improved joint function, and joint stability are the main goals of therapy. The muscle weakness and muscle atrophy contribute to the disease process. So, rehabilitation and physiotherapy were often prescribed with the intention to alleviate pain and increase mobility. However, as exercise has to be performed on a regular basis in order to counteract muscle atrophy, continuous exercise programs is recommended in people with degenerative joint disease. Therapeutic exercise regimes either focus on muscle strengthening and stretching exercises or on aerobic activity which can be land or water based.

**KEYWORDS:-** Osteoarthritis, Knee pain, Pathophysiology, Treatment

**INTRODUCTION:-** OA begins with damage to articular cartilage, which is due to i) Trauma or other injury ii) Excess joint loading by obesity / other reason iii) Instability or injury of the joint that causes abnormal loading .OA is developed is due to Local mechanical influence, Genetic factor, Inflammation & Chondrocyte function which leads to loss of articular cartilage.



In OA, expression of hundreds of genes of cartilage tissue are affected which alters chondrocyte phenotype. In addition to articular cartilage there is also role of subchondral bone in OA. In OA, subchondral bone release vasoactive peptides and MMPs. Neovascularization and subsequent increase permeability of the adjacent cartilage occurs and contributes to further cartilage loss.

Substantial loss of cartilage cause joint space narrowing and leads to painful and deformed joints. The remaining cartilage softens and develops fibrillation (Verticle cleft) and there is splitting and further loss of cartilage and exposure of underlying bone. As cartilage is destroyed and adjacent subchondral bone undergo pathologic changes, Cartilage is eroded completely, leaving denuded subchondral bone which becomes dense, smooth and glistening. A more brittle, stiffer bone results, with decreased weight-bearing ability and development of sclerosis and micro fractures. The joint capsule and synovium also show pathologic changes in OA.

The pain in OA is not due to destruction of cartilage but arise from the activation of nociceptive nerve ending within the joint by mechanical and chemical irritants. So the slow progressive changes in OA consist of an increase in water content, loss of PG, and reduction of PG aggregates of cartilage. The cartilage is subsequently unable to repair itself. Alteration in metabolism of subchondral bone adjacent to articular cartilage appears necessary for continued cartilage destruction. Eventually, progressive loss of articular cartilage and increasing subchondral sclerosis lead to an abnormal and painful joint.

### **TYPES OF OSTEOARTHRITIS:-**

Both primary and secondary OA involve the breakdown of cartilage in joints, which causes bones to rub together. Sometimes bones grow abnormal spurs in response to cartilage breakdown. This can make joints swollen, painful and stiff.

### a. Primary osteoarthritis:

Wear and tear on joints as people age cause primary OA. Therefore it starts showing up in people between the ages of 55 and 60. Theoretically, everyone experiences cartilage breakdown as they get older, but some cases are more severe than others.

### b. Secondary osteoarthritis:

Secondary OA involves a specific trigger that exacerbates cartilage breakdown. Here are some of the most common triggers for secondary OA:

- **Injury:** Bone fractures increase a person's chance of developing OA and can bring about the disease earlier.
- **Obesity:** According to the Arthritis Foundation, every pound of extra body weight places three pounds of pressure on the knees and six pounds on the hips. The weight speeds up the wear and tear of joint cartilage.
- **Inactivity:** Inactivity can cause obesity and weaken the muscles. Weak muscles mean poorly aligned joints and greater risk for OA.
- **Genetics:** Researchers have noticed that OA runs in families, so certain genes could also put you at risk.
- **Inflammation:** Diseases that cause inflammation can also affect cartilage. One such disease is rheumatoid arthritis.

### Risk Factors:-

**i. Age:-** Incidence of OA increase as you age. Since 'wear and tear' does play a part in the development of OA, the older you are, the more you have used your joints. Although age is an important risk factor, it does not mean that OA is inevitable.

**ii. Obesity:-** Increased body weight is a serious factor in the development of OA. For every pound you gain, you add 3 pounds of pressure on your knees and six times the pressure on your hips. Since weight gain gradually increases the stress on joints, the weight you gain the decade before you have OA symptoms plays a big role in determining if you will have OA.

**iii. Injury & Overuse :-** Athletes and people who have jobs that require repetitive motion, such as typing or operating machinery, have a higher risk of developing OA due to injury and increase stress on certain joints.

**iv. Genetics or Heredity:-** Genetics appear to play a role in the development of OA, especially in the hands. Inherited abnormalities of the bones that affect the shape or stability of the joints can lead to OA. It is also more common in joints that don't fit together smoothly.

v. **Muscle Weakness:-** Studies of the knee muscles not only show that weakness of the muscles surrounding the knee can lead to OA, but that strengthening exercises for thigh muscles are important in reducing the risk.

### **PATHOPHYSIOLOGY:**

Osteoarthritis is traditionally thought of as a ‘wear and tear’ disease which occurs as we age. The pathogenesis of OA involves a degradation of cartilage and remodeling of bone due to an active response of chondrocytes in the articular cartilage and the inflammatory cells in the surrounding tissues. The releases of enzymes from these cells break down collagen and proteoglycans, destroying the articular cartilage. The exposure of the underlying subchondral bone results in sclerosis, followed by reactive remodeling changes that lead to the formation of osteophytes and subchondral bone cysts. The joint space is progressively lost over time.

### **DIFFERENCE BETWEEN OSTEOARTHRITIS & RHEUMATOID ARHRITIS:-**

	<b>Rheumatoid arthritis</b>	<b>Osteoarthritis</b>
<b>When it starts</b>	Any age.	Usually later in life.
<b>How it develops</b>	Fairly quickly, over weeks or months.	Slowly over many years.
<b>Duration</b>	A flare-up can last days or weeks, depending on how's it treated	Symptoms tends to improve substantially after 30min of moving around
<b>Risk factors</b>	Can run in families. RA is 2–3 times more common in women.	Overweight and obesity, joint deformities, traumatic joint injuries, diabetes, and gout. It's more common in men under age 45 and women older than 45.
<b>Symptoms</b>	In addition to joint pain and swelling, you may have a low-grade fever, muscle aches, and fatigue. RA can affect your entire body, including your heart and lungs. In advanced cases, hard lumps called rheumatoid nodules may develop under the skin near joints.	Mostly limited to joint pain. Excessive bone growths called bone spurs may develop on the edges affected joints.
<b>Joints affected</b>	Usually begins in smaller joints, especially in the fingers. Symptoms are	Often joints in the hands and fingers, but less symmetrical than RA. Also

	symmetrical (on both sides of the body) and may later develop in larger joints.	affects the knees, spine, and hips.
<b>Treatment</b>	Disease-modifying medications and biologics that target your immune system.	Anti-inflammatory and corticosteroid medications.

## INVESTIGATIONS

**1. Imaging Plain radiograph:-** The following changes may be seen on plain radiographs • Joint space narrowing. • Osteophytes • Bony cysts • Subchondral sclerosis. Radiographs are cheap, provide a permanent record, and are easily available. They are not a good measure of disease progression as this is based on measures of joint space narrowing, which occurs at <0.1 mm per year, so it is difficult to measure accurately. Scoring systems to quantify radiological progression Several radiograph scoring systems have been employed to assist the measurement of osteoarthritis progression. Other techniques include chondrometry, where minimal interbone distance is measured using a special compass magnifying glass calibrated to 0.1 mm. A digitised image of a standard antero posterior knee radiograph is obtained and the area of the knee joint space is measured. Microfocal radiography allows magnification of the image (usually 4–10 times) with high spatial resolution, sharply defined joint margins, allowing accurate and reproducible measurements of radiographic features. Relationship between radiography findings and symptoms Results have been conflicting, probably due to the differences in populations studied and radiographic and clinical criteria used. The presence of osteophytes had a very strong association with knee pain, whereas the absence or presence of joint space narrowing was not associated. Knee pain severity was a more important determinant of functional impairment than radiographic severity of osteoarthritis. There was no correlation between joint space narrowing and a disability score at a single time point.

**2. Magnetic resonance imaging:-** This is already well established for use in assessing ligament and meniscal tears in the knee. It has no place in routine clinical assessment of osteoarthritis, but may be a specific and sensitive way of quantifying cartilage loss. Currently, magnetic resonance imaging has not proved to be sensitive enough in the detection of preclinical osteoarthritis. Changes in surface morphology and full thickness cartilage defects can be seen, but fibrillation cannot yet be evaluated.

**3. Other imaging techniques:-** Computed tomography is thought to have little advantage over plain radiographs unless an axial joint view is required. Radionuclide imaging is considered inadequate in assessing disease progression as it lacks sufficient anatomical detail. However studies have found that retention of technetium labelled diphosphonate in the knee predicts subsequent cartilage loss in patients with advanced osteoarthritis.<sup>30</sup> Thus far, radionuclide imaging has not been recommended as a routine imaging

modality due to worries about radiation exposure. Ultrasound is good for assessing cartilage integrity and destruction, but in most weight bearing joints, cartilage is not easily accessible.

## CURRENT STRATEGIES IN THE TREATMENT OF OSTEOARTHRITIS

**1. Lifestyle Modifications:-** Because of the multitude of pain generators and nociceptive mechanisms involved in the propagation of OA, it is unlikely that a single treatment modality will target the entirety of the disease process. Weight loss is important in reducing symptoms as well as for palliating the progression of OA in weight-bearing joints such as the knee and hip. Modest weight reduction using proper diet and exercise routines should be stressed in overweight patients. Periodic monitoring for compliance is important, and referrals to weight loss programs may be used to improve compliance. Sleep hygiene, regular exercise, smoking cessation, and moderate consumption of alcohol are all recommended for general health. These lifestyle modifications may sound like common sense; however, they may be more challenging to patients than simply taking a pill and going to see a therapist.

**2. Traditional Pharmacotherapy:-** Oral medications traditionally have been important to control pain in OA. Acetaminophen is the first-line oral agent in OA and is chosen for its effectiveness against mild to moderate pain as well as its low side effect profile.. There is an inflammatory component to OA, and oral non steroidal anti inflammatory drugs (NSAIDS) also are commonly used. Cyclooxygenase-2 selective inhibitors are equal in efficacy compared to nonselective NSAIDS and may have superior gastrointestinal side effect profiles, although they may have increased risk of cardiac complications such as ischemia and myocardial infarction compared to nonselective NSAIDS.

- Analgesics/anti-inflammatory agents
- Glucocorticoids
- Opioids
- Symptomatic, slow-acting drugs for osteoarthritis (SYSADOA)
- Anti-cytokines.

The specific risks associated with the use of classic non-steroidal anti-inflammatory drugs (NSAIDs) are due to their mechanism of action, i.e., the inhibition of prostaglandin secretion through the inhibition of cyclooxygenase (COX) in one or both of its two isoforms, COX-1 and COX-2. Specific inhibitors of COX-2 have a selective anti-inflammatory effect but are still markedly nephrotoxic. Nonselective COX inhibitors also have renal side effects. When signs of inflammation arise, intra-articular glucocorticoid injections can very rapidly eliminate a joint effusion. The most suitable type of glucocorticoid for injection has been found to be one with a long half- life, in crystalloid solution, with a small crystal size (e.g. triamcinolone acetonide or hexacetonide, at a dose of 10 mg or 40 mg, respectively). In addition to the types of medications mentioned above, there is a heterogeneous group of medications that, unlike the COX-2 inhibitors, do not inhibit

prostaglandin synthesis. This group includes hyaluronic acid, D-glucosamine sulphate, chondroitin sulphate, and diacerein. These medications are collectively called slow-acting drugs for osteoarthritis (SADOA).

**3. Complementary Alternative Medicine and Naturopathic Pharmacotherapy :-**A number of patients prefer to avoid traditional pharmaceuticals and interventional procedures. These patients are drawn to popular complimentary and alternative therapies, which include chondroitin, glucosamine, collagen, plant-derived substances (i.e., phytotherapy), vitamins, antioxidants, ayurvedic medicine, traditional Chinese medicine, and homeopathic preparations. Another treatment often grouped in the above category is acupuncture. Acupuncture has been applied in various traditional and modified forms in the non pharmacological treatment of OA.

**4. Orthotics and Assistive Devices:-** New treatment guidelines suggest the use of non pharmacologic therapy before instituting medications. Braces (orthotics) and assistive aids have been used for centuries and are now being utilized with increasing frequency in patients with OA. Bracing reduces pain by limiting excessive painful motion around a joint.

Assistive devices are used to reduce the load on weight-bearing joints such as the knee and hip. These devices may be used as an alternative to or complementary to, medications and potentially costly interventions. A single-point cane held in the contra lateral hand is useful in knee and hip OA and may reduce disease progression in OA of the knee.

**5. Physical Therapy:-** Exercise is considered by many to be the most important maintenance therapy for chronic OA and should be encouraged in all patients. Physiotherapy/physical therapy is the science of using specific exercises to strengthen joint-stabilizing muscles as well as applying therapeutic modalities to aid in healing tissues. These allow improved joint biomechanics and gait, thereby relieving pain and preventing joint dysfunction. The short-term effects of in-office physiotherapy on OA have been well proven. After completion of a therapist supervised course, the patient is given an individual home exercise program (HEP) that should be performed regularly.

**6. Physical Modalities:-** Physical modalities are noninvasive, time-honored approaches to pain and should be considered for OA. The most common are heating and cooling modalities. Heating can be superficial (heat packs or compresses) or deep (ultrasound, diathermy, microwave, laser) and is delivered via conduction (conventional heat packs), convection (whirlpool, baths, paraffin), or conversion (radiant heat). Therapeutic cold has an anti-inflammatory and primary analgesic effect and is excellent for reducing acute inflammation and slowing down the speed of nerve conduction. Heat relieves pain via a relaxation effect of the tissues as it is thought to increase collagenase activity. However, heat can cause vasodilation and may worsen inflammation. Limitations to the use of heat and cold modalities include neuropathic sensory loss, metallic

foreign bodies or implants, certain surgical procedures, and temperature-sensitive hematologic or rheumatologic conditions.

**7. Manual Medicine/Manipulation Therapy:-**Manual medicine or manipulation is employed to improve alignment and allow the body to utilize its self-healing capabilities. Manipulation has been described in the treatment of acute pain such as spinal facet sprain and muscular strains, but there remains a paucity of data in the literature on its long-term efficacy in chronic arthritic pain. Manipulation is postulated to act directly on the affected tissues or indirectly through neurological or vascular mechanisms.

## **8. Interventional Therapy :-**

**i) Traditional Injection Therapy:-** Intra-articular joint injections and procedures are a popular modality used to deliver medications directly into the joint space or to aspirate an acutely inflamed joint. Today, corticosteroid injections (although not without controversy and potential side effects) and viscosupplementation are common intra-articular procedures performed for symptomatic relief of painful OA. Viscosupplementation involves intra articular injections of hyaluronate , which is derived from naturally occurring glycosaminoglycan molecules. When corticosteroid and viscosupplementation treatments are compared, one study suggests that better short-term relief is provided by corticosteroid injection (at 4 weeks after treatment) but that better long-term relief is provided by viscosupplementation (from weeks 8–26).

**2. Empirical Interventional Therapy:-**Pulsed radiofrequency (PRF) neuromodulation is a treatment modality under investigation that may be considered for the management of OA refractory to more traditional options. This technique uses non ablative radiofrequency energy targeted to either intra articular neural structures within the joint or to the extra articular sensory nerves. The intra-articular approach typically targets its radiofrequency waves at the articular periosteal surfaces to modulate nociceptive input via changes in cellular genetics and metabolism. There are several limitations of PRF as a treatment option, including limited evidence based on observational data alone and limited insurance coverage.

I. Surgical Treatments Types of traditionally performed surgical procedures for OA are (a) Arthroscopic procedures (b) Decompressive procedures (c) Arthrodesis (surgical fusion as in the carpal bones) (d) Osteotomy and (e) Partial or total joint arthroplasty.

**a. Arthroscopic Procedures:-** Arthroscopic procedures involve joint irrigation/ lavage with normal saline. The efficacy of arthroscopy, however, is still controversial. Another component of arthroscopic procedures is debridement, which is known to be beneficial when OA is associated with the radiographic finding of an intra-articular loose body. Other arthroscopic procedures include arthroscopic abrasion of sclerotic bones.



**b. Decompressive Procedures:-** In the spinal canal, the impingement of nerves caused by degeneration and subsequent overgrowth of bone may occur (typically causing spinal stenosis or foraminal stenosis), resulting in pain, paresthesias, and potential sensory and/or motor dysfunction. In an effort to alleviate pain caused by spinal stenosis, interventional pain management techniques or decompressive surgical procedures can be employed to alleviate nerve impingement or create space in the spinal canal.

**c. Arthrodesis:-** Arthrodesis has largely fallen out of favor for large joints because of its subsequent limitation in range of motion, such as in the hips or knees, in light of the higher success rate of current arthroplasty procedures. However, fusion still plays a role in treatment of OA of smaller joints, such as in the hands, feet, and ankles. Spinal fusion surgery may correct instability from multilevel decompressive spine surgery..

**d. Osteotomy:-** Osteotomy, particularly the technique known as high tibial osteotomy, has been employed as a successful treatment option for unicompartamental OA of the medial knee in younger (usually <60 years old) active patients. In this procedure, a wedge is taken out of the tibia to relieve unequally distributed weight bearing (patients with varus deformity of the knees) over arthritic joint surfaces. However, osteotomy may have only temporary effects.

**e. Joint Replacement:-**The most commonly performed surgical procedures for OA in large joints are arthroplasties or joint replacements. Current U.S. estimates count 150,000 hip arthroplasties and 300,000 total knee arthroplasties per year. It should be noted that the success rate and complication rate for the replacements have strong correlations with the numbers of cases performed by an individual surgeon and with the volume of cases seen at a hospital. One recent study demonstrated a higher functional outcome 2 years postoperatively and a lower risk of postoperative complications with higher numbers of cases per surgeon/ hospital.

## CONCLUSION:

There are many treatments for knee osteoarthritis. Prevention is important: If the influences that can potentially damage the knee are eliminated early enough, then the development of osteoarthritis can be prevented, or at least the progression of any changes that are already present can be slowed. Patient education and counseling are the first step in any treatment plan and should include information about the course of the disease and the range of treatment options. A stepwise treatment algorithm should be applied, in order to slow the progression of the disorder and thereby grant the patient the best possible quality of life. The best treatment for each patient should be chosen after an individual assessment of the severity of knee osteoarthritis and an individual evaluation of the risks. An important general principle is that surgery should be performed only when conservative treatment has failed.

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