Osteoarthritis

Osteoarthritis (OA) is a common, progressive disorder affecting primarily weight-bearing joints, characterized by progressive destruction of articular cartilage, osteophyte formation, pain, limitation of motion, deformity, and disability

Epidemiology

Osteoarthritis is the most common form of arthritis and is strongly related to age (OA is uncommon in adults under age 40 and highly prevalent in those over age 60). The prevalence of OA is greater in women by 1.5- to 2-fold, and they tend to have more generalized disease.

Normal joint anatomy and physiology

The synovial joint consists of two bone ends covered by articular cartilage. The roles of articular cartilage include

1. Enabling frictionless movement of the joint.
2. Distributing the load across the joint (shock absorber), to prevent damage.

The joint capsule is a fibrous outer layer that encapsulates the joint. The joint capsule is lined by synovium, a membrane that produces a viscous fluid (synovial fluid) which fill the space between the surfaces of articular cartilage.

Synovial fluid lubricates the joint and reduces friction between cartilage surfaces, thereby serving as a major protector against friction-induced cartilage wear.

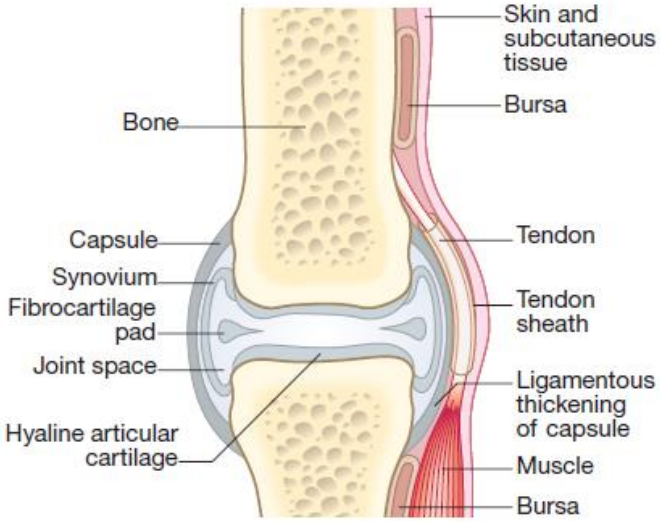


Fig. 25.3 Structure of a synovial joint.

Pathophysiology

Primary OA, the most common type, has no known cause. Secondary OA is associated with a known cause, such as trauma, metabolic or endocrine disorders, and congenital factors.

The pathophysiology is multifactorial and typified by progressive destruction of joint cartilage, erratic new bone formation, thickening of subchondral bone and the joint capsule, bony remodeling, development of osteophytes, variable degrees of mild synovitis, and other changes.

OA usually begins with damage to articular cartilage through injury, excessive joint loading from obesity or other reasons. The destruction of protective cartilage leads to exposure of underlying bone. Without cartilage, dispersion of weight across the joint no longer occurs (permitting bone-to-bone contact), thereby leading to microfractures.

New bone is formed (osteophytes: bony outgrowths) at joint margins (distant from cartilage destruction) as an attempt by the body to repair and stabilize the joint; however, this leads to further disfiguration and friction within the joint.

Progressive loss of joint cartilage, bone damage, narrowing of joint spaces, and formation of osteophytes result in deformed, painful joint.

The release of cartilage components, as well as bony formations, leads to inflammation within the synovium. The degree of inflammation within the joint is significantly less than that seen in rheumatoid arthritis.

Risk factors:

Risk factors include age, gender, genetic predisposition, and nutritional status. Age is the strongest predictor of OA, although advanced age alone is insufficient to cause OA.

Joints exposed to biomechanical factors are at increased risk (e.g knee and hip joint). Occupational and recreational activities involving repetitive motion can provoke OA.

Heavy physical activity is a stronger predictor of subsequent OA than light-to moderate activities, especially for older individuals, in whom the joint structure is less capable of coping with highly stressful activities.

Obesity increases load-bearing stresses on hip and knee joints. The risk of OA increases by 10% for each kilogram of body weight above ideal body weight.

Clinical presentation

The frequently used and weight-bearing joints (hands, hip, knee, and spine) are principally affected and contribute to disability.

Generally, patients usually are more than 50 years old. Presentation may range from asymptomatic to severe joint pain and stiffness with functional limitations. In contrast to some other arthritic conditions (eg, rheumatoid arthritis, gout), inflammation usually is absent or mild and localized when present.

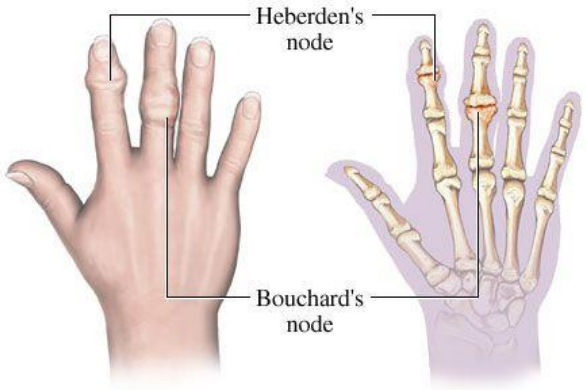
The predominant symptom is deep, aching pain in affected joints. Pain accompanies joint activity and decreases with rest.

Limitation of motion, stiffness, crepitus, and deformities may occur. Patients with lower extremity involvement may report weakness or instability.

Morning stiffness (joint stiffness) is also common; usually lasts less than 30 minutes and resolves with motion.

Presence of warm, red, and tender joints suggests inflammatory synovitis. Physical examination of affected joints reveals tenderness, crepitus, and possibly enlargement.

Nodules on the distal interphalangeal joints (Heberden's nodes) or proximal interphalangeal joints (known as Bouchard's nodes), are due to the bony overgrowth of osteoarthritis



Diagnosis

Osteoarthritis is primarily diagnosed by its clinical presentation and physical examination (diminished range of motion, crepitus, abnormalities in joint shape).

[The American College of Rheumatology (ACR) has defined criteria for OA of the hip, knee, and hand].

Confirmation and progression can be achieved by radiography. Narrowing of joint space (owing to loss of cartilage), and osteophytes are seen. Also, there is no specific lab tests are diagnostic for OA.

Goals of treatment

1-Relieve pain and stiffness.

2-Maintain or improve joint mobility

3-Maintain or improve quality of life.

Treatment

A-Non pharmacological treatment

Nonpharmacologic therapy is the cornerstone of treatment. weight loss a pivotal treatment goal in overweight and obese patients. Weight loss should be pursued through a combination of dietary modification and increased physical activity. The patient's physical capabilities should be considered when implementing an exercise program.

Aerobic exercise programs to increase muscle strength. Patients should be encouraged to participate in aerobic exercises, such as swimming or walking. Aerobic activity also reduces pain and disability from OA. Exercise can help overcoming the depression that may accompany the chronic pain of osteoarthritis.

Application of heat or cold to involved joints improves range of motion, reduces pain, and decreases muscle spasms. Applications of heat include warm baths or warm water soaks.

Physical therapy—with heat or cold treatments and an exercise program—helps maintain range of motion and reduce pain and need for analgesics. Assistive devices (e.g., canes, walkers) may help decrease joint load.

Surgery is indicated for patients with severe impairment of function as a result of end-stage osteoarthritis. Two common surgical procedures present. These are osteotomy and arthroplasty. Osteotomy (cutting or sectioning of bone) to relief pain. Arthroplasty which is joint replacement Surgery.

B-Pharmacologic therapy

Simple analgesics such as acetaminophen and nonsteroidal anti-inflammatory drugs (NSAIDs) are first-line agents for treating OA

Acetaminophen

Acetaminophen is a centrally acting analgesic that inhibits prostaglandin production in the brain and spinal cord. Acetaminophen is an effective and inexpensive analgesic with a favorable risk–benefit profile. For treatment of mild-to-moderate pain, acetaminophen should be tried initially (first line) at an adequate dose and duration before considering an NSAID.

Acetaminophen is generally considered to be as effective as NSAIDs for mild-to moderate joint pain with a more favorable adverse effect profile. Acetaminophen should be administered initially on an as-needed basis in daily doses up to 4 g. Single doses should not exceed 1 g.

Despite being one of the safest analgesics, acetaminophen can cause significant adverse effects, including hepatic and renal toxicity. Doses greater than 4 g are associated with an increased risk of hepatotoxicity. Acetaminophen does not appear to exacerbate stable, chronic liver disease; it can be used with caution and vigilant monitoring of liver function in this population.

Acetaminophen may worsen kidney function and increase blood pressure. Nevertheless, acetaminophen remains the preferred oral analgesic for mild-to-moderate pain in patients with hypertension or kidney disease because of the greater risks associated with NSAID use.

Nonselective NSAIDs and cyclooxygenase-2 (COX-2) selective inhibitors:

NSAIDs are a reasonable first-line therapy in patients with moderate-to-severe OA; or, as adjunctive or alternative therapy when acetaminophen fails to provide an acceptable analgesic response. NSAIDs significantly reduce pain and improve functioning in patients with OA, although individual responses can vary widely. Some clinicians recommend NSAIDs over acetaminophen for the initial treatment of severe pain or when signs and symptoms of inflammation are present.

At equipotent doses, the analgesic and anti-inflammatory activity of all oral NSAIDs, including COX-2–selective inhibitors, are similar. The selection of a specific oral NSAID should be based on patient preference, previous response, tolerability, dosing frequency, cost, and considering underlying GI and cardiovascular risk. Some patients respond to one NSAID better than to another. If an insufficient response is achieved with one NSAID, another agent should be tried.

Pain relief occurs rapidly (within hours), but anti-inflammatory benefits are not realized until after 2 to 3 weeks of continuous therapy. This period is the minimal duration that should be considered an adequate NSAID trial.

All NSAIDs are associated with adverse GI, renal, hepatic, cardiovascular, CNS, and blood pressure effects, particularly in the elderly. Inhibition of the COX-1 enzyme is thought to be responsible primarily for the adverse effects on the gastric mucosa, kidney, and platelets. Direct irritant effects also may contribute to adverse GI events. Celecoxib (COX-2 inhibitor) has less gastrointestinal toxicity and adverse effects on platelet aggregation.

COX-2 inhibitors should be reserved for patients at high risk for GI complications and low risk for cardiovascular events. For patients at particularly high GI bleeding risk (eg, previous history of NSAID-induced GI bleed), a selective COX-2 inhibitor combined with a proton pump inhibitor is better in reducing risk for GI bleeds compared to a selective COX-2 inhibitor alone, which in turn has lower risk for GI bleeds compared to a nonselective NSAID combined with a proton pump inhibitor.

Both nonselective and selective NSAIDs are associated with an increased risk for cardiovascular events (hypertension, stroke, myocardial infarction, and death).

Although selective COX-2 inhibitors reduce GI events, these agents have been associated with increased risk of cardiovascular events relative to many nonselective NSAIDs.

Naproxen appears to have the lowest incremental cardiovascular risk of the nonselective NSAIDs and should generally be considered first in such patients.

Concomitant use of low-dose aspirin mitigates some of the increased cardiovascular risk of selective COX-2 inhibitors but also obliterates the GI safety of COX-2 selectivity.

In patients at risk for GI events and cardiovascular disease and requiring oral NSAID therapy, a nonselective NSAID plus a proton pump inhibitor is a reasonable option.

Topical NSAIDs: Topical administration of NSAIDs minimizes systemic exposure while providing pain relief comparable to that of oral NSAIDs, but topical NSAIDs are only appropriate for patients with OA of superficial joints, including hands, wrists, elbows, knees, ankles, and feet. Systemic absorption of topical agent is significantly less than that of oral agent. Thus, GI, cardiovascular, and renal adverse effects are rare. The most common adverse effects include application site dermatitis, pruritus, and phototoxicity.

Other Topical Therapies

Capsaicin

Capsaicin cream is an alternative first-line treatment. It is a reasonable option for patients unable to take oral NSAIDs. To be effective, capsaicin must be used regularly, and it may take up to 2 weeks to take effect. Although use is recommended four times a day, a twice-daily application still provide adequate pain relief. Patients should be warned not to get the cream in their eyes or mouth and to wash their hands after application to avoid burning and stinging.

Tramadol

Use of opioid analgesics may be necessary when pain is unresponsive to other pharmacologic agents or when such agents are contraindicated. Tramadol is an oral, centrally acting synthetic opioid analgesic. Tramadol effectively treats moderate pain but is lacking anti-inflammatory activity. Tramadol is a reasonable option for patients with contraindications to NSAIDs or in those who have failed to respond to other oral therapies. The addition of tramadol to NSAIDs or acetaminophen may expand the analgesic effects of a failing regimen, thereby securing sufficient pain relief in some patients.

Moreover, concomitant tramadol may permit the use of lower NSAID doses. However, the increased risk for side effects associated with tramadol may offset the benefits. Dizziness, vertigo, nausea, vomiting, constipation, and lethargy are all relatively common adverse events.

Duloxetine

Duloxetine has been approved for the treatment of chronic musculoskeletal pain. Duloxetine is a selective serotonin and norepinephrine reuptake inhibitor.

it can be used as adjunctive treatment in patients with partial response to first-line analgesics (acetaminophen, oral NSAIDs). It may be a preferred second-line medication in patients with both neuropathic and musculoskeletal OA pain.

Glucosamine and Chondroitin

The glucosamine sulfate and chondroitin sulfate are dietary supplements. Both compounds are found naturally in the body and are essential to the formation of cartilage. Glucosamine is believed to stimulate cartilage production against oxidative chemical damage. Chondroitin, often administered in conjunction with glucosamine, is thought to inhibit cartilage destruction.

The highest quality evidence to date suggests no clinically important difference in efficacy between glucosamine, chondroitin, their combination, or placebo in patients

with knee OA. Consequently, these products, used alone or in combination, are generally not recommended.

Intra-articular (IA) corticosteroid

Intra-articular (IA) corticosteroid (e.g. Methylprednisolone or Triamcinolone) injections are recommended for both hip and knee OA when analgesia with acetaminophen or NSAIDs is suboptimal but are not routinely recommended in hip OA owing to administration difficulties. Pain reliefs begins within days after the injection. Duration of action is up to 4 weeks.

Because of adverse effects on the bone (there is concern that repeated injections into the joint may lead to progressive cartilage damage, do not administer injections more frequently than once every 3 months (injections should be limited to three or four per year).

Specific instructions are given to the patient to refrain from weight-bearing activity for 3 days, except getting up for meals and going to the bathroom.

Systemic corticosteroid therapy is not recommended in OA, given the lack of proven benefit and the well-known adverse effects with long-term use.

Intraarticular (IA) hyaluronic acid derivatives

Hyaluronic acid derivatives are intended to improve elasticity and viscosity of synovial Fluid. (Healthy cartilage and synovial fluid are replete with hyaluronic acid, a viscous substance believed to facilitate lubrication and shock absorbency).

They are indicated for the treatment of knee OA when treatment failure to other therapies occur. However, IA hyaluronic acid is not routinely recommended for knee OA pain. Injections do not provide clinically meaningful improvement and may be associated with serious adverse events (e.g., increased pain, joint swelling, and stiffness).

Administration typically consists of weekly injections for 3 to 5 weeks, depending on the specific product. Most benefits are seen after the last dose.

A new hyaluronan preparation (Monovisc) has been developed, containing 4 or 5 times the amount of hyaluronan used in the usual knee injection.. It is designed to treat the symptoms of osteoarthritis with only one injection. The approach, if effective, would simplify the procedure especially for “needle shy” patients