Continuing Education

Osteoarthritis:

Overview with a Focus on OTC Therapies

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Objectives

1. Recognize the etiology, pathophysiology, and incidence of osteoarthritis and discern factors that constitute a risk for developing OA.
2. Identify the signs and symptoms of osteoarthritis and the components necessary for a diagnosis as well as the goals of therapy for individuals treated for osteoarthritis.
3. Recognize the non-drug treatments and lifestyle interventions beneficial in preventing and reducing the severity of osteoarthritis and which patients are most likely to benefit.
4. Recognize various over-the-counter and prescription drug therapies available for the treatment of osteoarthritis of the hand, knee, and hip.
5. Determine the risks and benefits of OTC and herbal therapies for the treatment of osteoarthritis and which patients in which these therapies should be avoided.

Background

Osteoarthritis is a progressive joint disease resulting from degeneration of cartilage and changes in joint structure and is one of the leading causes of disability in the United States. In 2010-2012, an estimated 52.5 million adults in the United States were diagnosed with arthritis, an increase from 49.9 million in 2007-2009 and more than double the 21 million adults in 1995. It is estimated that the rate of osteoarthritis (OA) alone will increase to around 67 million persons by 2030. The total hospital cost burden for OA was nearly $15 billion with nearly 1 million OA-related discharges in 2011, mostly from knee and hip replacement surgeries. The overall incidence of OA, defined as the number of newly diagnosed cases each year, ranges from 88-240 per 100,000 patient years, but the numbers may be somewhat unreliable due to not all OA patients seeking medical treatment, OA being relatively common in the general population (20-30% of adults), not all cases of OA being symptomatic, and many patients having multiple joints affected.[1-3]

In 2012, the United States prevalence of arthritis was lower for younger adults (22.7%) versus adults aged 65 years and older (49.7%). Arthritis is most prevalent in white (25.9%) and black (21.3%) populations, and least prevalent in Asian populations (4.9%). In particular, African-American men (35%) are more likely to have knee OA as compared to Caucasian men. African-Americans in general are more likely to have severe disease versus Caucasians. Prior to age 50, men are more likely than women to have OA, due to sports and other injuries. However, older women (26%), over age 70, have a higher prevalence of OA versus men (12%). Women are also more likely to have OA with inflammation leading to complications such as Bouchard and Heberden nodes, which are hard, bony growths or enlargements (osteophytes) within the finger joints.[1]

Who is at Risk?

The development of osteoarthritis is a complex process dependent on a number of different factors ranging from genetics to physical injury. Many OA patients have multiple known risk factors, the most common being age, obesity, sex (female), occupation, participation in certain sports, history of joint injury or surgery, and genetics. Obesity may be the most important preventable risk factor and is most directly linked to OA of more weight bearing joints such as the knee, though risk for hip and hand OA is also seen. The risk of developing knee OA is nearly 3 times higher in obese men and 4.4 times higher in obese women, versus non-obese individuals.[1-4]

Another group at increased risk of OA is individuals who work in mechanically stressful jobs. Particularly, work that involves significant amounts of time standing, kneeling, squatting, lifting, or moving of heavy objects increases a person's risk for OA. Specific jobs associated with increased risk include construction, mining, healthcare assistance, factory work, carpentry, and farming. Similarly, professional athletes of certain sports are at increased risk, though recreational participation does not carry the risk.[1,3]

Traumatic injury to joint tissues during sports or accidents greatly increases the risk of OA. Damage to these tissues decreases their ability for load bearing and shock absorption and increases the stress on cartilage and bone leading to OA later in life. In a related fashion, muscle weakness and similar conditions in the legs are associated with an increased risk of OA.[1,3]

Overall, the genetic risk for developing OA is complex and involves a wide range of potential gene sources. For example, women are ten times as likely to develop Heberden nodes, a finger joint deformity associated with advanced disease, in OA than men, and women whose mothers had these deformities are twice as likely to develop such nodes than women without this relation. However, for most genes showing a link to OA, the associated risk is typically not strong and a person's genetic risk for OA and its severity are likely determined by the combined effect of multiple genetic differences.[1-3]

Pathophysiology

Osteoarthritis is typically divided into two types, primary disease and secondary disease. Primary OA is disease that has no identifiable cause while secondary OA is disease that can be linked to either another form of arthritis (rheumatoid), a history of joint injury, metabolic disorder (obesity), or other relatable factor. Historically, onset of OA has been associated with general "wear and tear" and/or loss of joint cartilage. In reality, OA results from the combination of a number of factors including the body's inflammatory and immune responses to changes in joint tissue.[1]

In a normal, healthy joint, normal cartilage is involved in a number of functions including lubricating motion, shock absorption, and load bearing. In synovial joints, such as the knee, normal cartilage is found attached to bone, separating it from the synovial fluid in the synovial cavity of the joint. Normal cartilage is easily compressed when bearing weight and spreads out the force of weight evenly across bone, muscle, and other joint tissues. Normal cartilage is also smooth and frictionless to allow for smooth movement across the joint as well as overall joint stability.[1,3]

Additionally, articular cartilage lacks direct blood supply and relies on nutrient supply by the synovial fluid. With normal movement, nutrients are able to flow into the joint, but lack of movement reduces supply. This is one way in which routine physical activity is beneficial in maintaining joint health. With proper nutrients, routine turnover of normal cartilage helps ensure its continual repair and restoration.[1,3]

Development of OA typically begins with damage to normal cartilage as a result of physical injury, excess weight bearing (obesity), or other instability of the joint leading to poor joint function. Chondrocytes, repair cells normally present for healthy cartilage turnover, become over-active and the balance of creating and breaking down cartilage is lost leading to overall degeneration and remodeling, with joint capsule stretching and weakness of muscles around the joint. Overall, the progression to OA involves numerous biological molecules and body processes involving cartilage function and structure.[1,3,4]

Abnormalities in other joint tissues also contribute to OA and its complications. Changes in underlying bone results in increased production of vasoactive peptides and other signal proteins that contribute to cartilage damage. Joint space narrowing leads to painful movement and deformation. Finally, further changes in bone structure after cartilage loss, such as becoming more brittle and stiffer, results in misalignment that decreases weight bearing ability and increases the development of sclerosis and microfractures. The pain of OA is created by activation of nerve endings by chemical irritants released as a result of increased joint fluid, microfracture, bone irritation, or damage to other joint tissues.[1-4]

Presentation and Diagnosis

Often the most apparent symptom of osteoarthritis that leads to patients seeking treatment is pain that is deep and/or aching as well as pain with joint motion. The most

common sources of pain are bone, synovial inflammation, and overstretching of the joint capsule due to fluid. The cartilage itself lacks pain receptors and is typically not a source of pain.[1-5]

Joint motion is frequently impaired, often with swelling, and may lead to loss of stability in weight-bearing joints. Another hallmark symptom of OA is joint stiffness that lasts less than 30 minutes and resolves with motion, but returns with rest. Particularly, joint crepitus (crackling) is a frequent complaint of OA patients. These symptoms are often reported by the patient to improve and worsen with the weather. Inflammation of the synovium or cartilage may or may not be present in OA; however, the presence of inflammation may indicate some other condition such as rheumatoid arthritis.[1-5]

In advanced hand OA, some patients present with bony osteophytes or bony growths in the distal finger joints called Heberden nodes. Hard or soft growths that present on the proximal finger joints are called Bouchard's nodes. Growths present in the metacarpal joint (where the fingers meet the hand) lead to a square appearance of the hands often seen in hand OA.[1,2]

Diagnosis of osteoarthritis ought to be made with consideration of patient history, physical exam, radiology (x-ray, CT), and lab testing. In the course of diagnosis, the primary goals are to discern between primary and secondary OA disease and identify what joints are involved and their extent of involvement. In the course of clinical evaluation, it is recommended clinicians evaluate a patient's ability to perform activities of daily living. Radiography is useful for confirming a diagnosis of OA, however, not all cases of OA have visible changes with radiology. The usefulness of lab testing is limited to ruling out other conditions such as rheumatoid arthritis or gout. The diagnosis criteria are outlined in Table 1.[1,3,4,6]

Table 1: Requirement for Diagnosis of OA

Hip OA

• Requires
  ○ Pain in hip
• Plus two of the following:
  ○ Erythrocyte sedimentation rate < 20 mm/h
  ○ Femoral or acetabular osteophytes on radiography
  ○ Joint space narrowing on radiography

Knee OA

• Requires
  ○ Pain in the knee
  ○ Osteophytes on radiography
• One of the following:
  ○ Age > 50 years
  ○ Morning stiffness lasting < 30 min
  ○ Crepitus on motion
  ○ Bony enlargement
  ○ Bony tenderness
  ○ Palpable warmth

Non-Drug Treatment

Overall, the treatment of osteoarthritis should be tailored to the individual patient. The goals of therapy are typically (1) to educate the patient, family members, and caregivers; (2) to relieve pain and stiffness; (3) to maintain or improve joint mobility; (4) to limit functional impairment; and (5) to maintain or improve quality of life. Individual treatment of OA depends on joint involvement and severity, other medical conditions, other medications, and allergies beginning with patient education, personal activity and exercise, and weight loss, as appropriate.[1]

Of the current common treatment practices with OA, non-pharmacologic therapies are the only preventative therapies shown to delay progression to OA. Patient education in regards to the disease, its progress and extent, overall prognosis, and therapy options is vital to encourage patients to seek proven beneficial therapies and should be provided to all patients. Overall, patient education should be personal and encourage the patient in the benefits of their overall treatment program as

patient motivation is critical for achieving treatment goals. Patients should also be provided with walking, stability, and other mechanical aids as appropriate to promote patient mobility and ability to perform normal daily activities, such as supportive gloves or splints.[1-3,6]

Weight loss is consistently linked to improved OA outcomes as modest (5%) weight loss has been shown to provide some pain relief in obese patients while weight loss of 10% or more may lead to significant reductions in pain. Weight loss, often combined with regular exercise, is recommended in all overweight patients to improve joint function and reduce pain. The recommended exercises for OA are low-impact, range-of-motion aerobics and strength building resistance exercises, including water-based exercise. Particularly, regular exercise is recommended in all patients at least two to three times weekly involving a variety of exercises and is associated with improved outcomes resulting from muscle strengthening and increased joint stability.[1,2,4,6]

Heat therapy is another non-drug therapy that is beneficial in providing temporary relief of pain associated with OA. The use of over-the-counter (OTC) heating pads and wraps is typically safe and beneficial, particularly in hand and knee OA when inflammation is not of primary concern. Heat therapy, in the form of warm wet compress, heating pad, or hot-water bottle, should be applied for 15-20 minutes 3-4 times per day. Heat therapy should not be used when inflammation is present, with topical analgesics (such as nonsteroidal anti—inflammatory drugs [NSAIDs]), or over broken skin. Many adhesive products are available over-the-counter for ease of use. Heat wraps ought to be worn over a towel or clothing in persons older than 55 years and should not be worn during sleep to avoid skin burning.[2,5]

Surgery for knee and joint replacement is a consideration with advanced disease leading to functional disability or severe pain that is not improved with medication therapy. While most patients who undergo joint-replacement surgery do see improvement in pain, disability, and quality of life; benefits are not universal. Additionally, joint replacement surgery accounts for a large portion of total medical costs attributed to treating OA. Other surgical options such as arthrodesis (joint fusion) and osteotomy (removal of bony tissue growth) are available and suitable for specific situations. Surgery for hand OA is often less beneficial and frequently not recommended.[1,2]

Drug-Based Treatment

Medication therapy often begins with scheduled acetaminophen (Tylenol®) in knee and hip OA. Other topically applied therapies such as NSAIDs (diclofenac gel) for knee and hand OA or capsaicin for hand OA are also recommended choices for initial treatment. If additional pain control is required, NSAIDs or Cyclooxygenase-2 (COX-2) Selective Inhibitors (celecoxib) are appropriate based on risk versus benefit analysis under the supervision of a clinician. Tramadol (Ultram®) may also be considered if further pain relief is required. If initial therapies are not sufficient or not feasible, other opioid pain medications may be considered for pain relief. Other secondary therapies that may be considered include duloxetine (Cymbalta®) or hyaluronic acid injections for knee OA.[1,3]

Hand OA

For hand OA, the recommended medication therapies are either topical or oral NSAIDs/COX-2 inhibitors, topical capsaicin, or tramadol. However, oral NSAIDs, such as ibuprofen (Motrin®) and naproxen (Aleve®), and COX-2 selective inhibitors, such as celecoxib (Celebrex®), may be used as an alternative therapy. If first choice of therapy is ineffective, a combination of two recommended therapies is acceptable. Treatment with acetaminophen lacks quality studies for recommended use in hand OA as in knee and hip OA but may be considered in certain cases as an alternative therapy. Treatment with opioid analgesics and intra-articular therapies (drug injections directly into the joint) is not recommended.[1,2,6,7]

Table 2: Recommended Therapies for Hand OA

Recommended Therapies	Topical Capsaicin Topical NSAIDs Oral NSAIDs/COX-2 Inhibitors* Tramadol
Avoid Use	Intra-articular Therapies Opioid Analgesics

**Recommended that person's ≥ age 75 avoid oral NSAIDs in favor of topical NSAIDs.*

Topical NSAIDs, such as diclofenac gel (Voltaren®), have been shown to be of similar benefit for relief of OA symptoms as compared to oral NSAIDs. As a result of less systemic exposure, topical NSAIDs demonstrate a reduced risk of potentially harmful side effects such as GI upset and bleeding versus oral therapy. Topical NSAIDs have been shown to improve pain in hand OA with reduction in pain correlating to improvement in physical function. The primary side effects noted have been application site effects such as loss of feeling at the site of application.[1,2,5-8]

The oral NSAIDs, while shown to be more effective for symptom relief than acetaminophen, pose risk of additional side effects such as GI damage, kidney impairment, and heart related events with prolonged therapy. Therefore, self-treatment with oral NSAIDs should be limited to no more than 10 days without medical evaluation. The recommended dosing of ibuprofen is 200-400 mg every 4-6 hours up to 1200 mg/day. The recommended dosing of naproxen is 220 mg every 8-12 hours up to 660 mg per day, but should not exceed 440 mg per day in persons over 65 years of age. Long term use of oral NSAIDs ought to be accompanied by GI protective therapy with a proton pump inhibitor (PPI), such as omeprazole (Prilosec®).[1,2,4-7,9]

In regards to the potential side effects, oral NSAIDs should be avoided in person's ≥ 75 years old, with more preferable alternative treatments being topical products and tramadol. Oral NSAIDs should be avoided altogether or used with extreme caution in patients with chronic kidney disease or whom are taking low-dose aspirin for heart protection. In patients who have a history of GI ulcer or bleed, but have not had an occurrence in the past year, oral NSAIDs may be considered with a preference for COX-2 selective inhibitors plus a PPI.[2,6,7,9]

COX-2 inhibitors such as celecoxib have demonstrated equivalent benefit to other oral NSAIDs for all types of OA often with less GI and renal side effects, but more risk for cardiovascular effects. Thus, COX-2 selective inhibitors may provide greater safety in patients with GI or renal complications, but should be avoided in older patients, particularly those with cardiovascular risk factors. Even with reduced risk for GI related side effects, a PPI should still be considered with long-term treatment with these agents.[1,2,6,7,10]

Table 3: Acetaminophen and Selected NSAIDs for Osteoarthritis

Drug	Class	Recommended	Dose	Side Effects	Restrictions
Acetaminophen	Analgesic	Knee and hip	650-1000 mg every 4-6 hours (Max 4 gm/day)	Skin rash	Liver failure Overdose Kidney failure
Diclofenac	Topical NSAID Oral NSAID	Hand and knee Hand, knee, and hip	 50-100 mg 2-3 times daily	Skin irritation GI upset Drowsiness/ dizziness Skin rash Fluid retention	Age > 75 years GI damage Kidney failure Cardiovascular disease Taking aspirin Liver failure
Ibuprofen	Oral NSAID	Hand, knee, and hip	200-400 mg every 4-6 hours (Max 1200 mg/day)	GI upset Drowsiness/ dizziness Skin rash Fluid retention	Age > 75 years GI damage Kidney failure Cardiovascular disease Taking aspirin Liver failure
Naproxen	Oral NSAID	Hand, knee, and hip	220 mg every 8-12 hours (Max 660 mg/day)	GI upset Drowsiness/ dizziness Skin rash Fluid retention	Age > 65 years GI damage Kidney failure Cardiovascular disease Taking aspirin Liver failure
Celecoxib	COX-2 Selective NSAID	Hand, knee, and hip	100 mg by mouth twice daily OR 200 mg once daily	GI upset Skin rash	Cardiovascular disease GI damage Liver failure Taking aspirin “Sulfa” allergy

Topical capsaicin cream is another recommended OTC therapy in hand OA with potential benefit in reducing pain associated with OA. However, improvement in joint functionality is less apparent. The side effects are limited to skin irritation and burning and considered much less risky than oral NSAID therapy. However, caution should be used in diabetic patients as an increase in skin ulcers have been seen with these patients. Capsaicin therapy may be most beneficial in situations where another therapy in addition to a recommended treatment is desired. Capsaicin cream (0.025-0.25%) is recommended to be applied 3-4 times daily to the affected joint(s) for up to 7 days or longer with medical supervision. Other topical products such as Bengay® (methyl salicylate, menthol and camphor) may be considered for temporary relief of symptoms, but evidence is conflicting regarding benefit in OA[1,2,5-8]

Opioid therapy for hand OA is uncommon and avoided, if possible. However, tramadol, as a weak opioid with additional serotonin effects has shown moderate benefit in OA and can be considered in patients requiring pain relief that is not provided by other recommended therapies. The adverse effects of opioids such as sedation and dizziness are concerning, particularly among elderly patients.[2,6,7]

Intra-articular injections of corticosteroids and hyaluronic acid are not routinely recommended for use in hand OA, but may be considered if pain relief from other treatments is inadequate. Clinical trials with intra-articular corticosteroids have shown small improvements in pain lasting for 3 weeks up to 3 months. Improvements in joint function have not been clearly demonstrated. Studies with hyaluronic acid injection have demonstrated a comparable but more sustained pain response (1-6 months) as well as some potential motor function improvement versus corticosteroids. However, some conflicting studies have also shown no difference in benefit of these therapies versus placebo. Reported side effects of both therapies tend to be minimal and limited to injection site irritation and bleeding.[2,4,6,7,11,12,13]

Table 4: Alternative Therapies for Osteoarthritis

Drug	Class	Recommended	Dose	Side Effects	Restrictions
Capsaicin	Topical counterirritant	Hand	Apply 3-4 times daily to affected joints	Skin irritation/ burning	Diabetic skin ulcers
Tramadol	Opioid analgesic	Hand, knee, and hip	50-100 mg every 4-6 hours (Max 400 mg/day)	Sedation Dizziness Cognitive changes Slowed breathing GI upset Skin rash	Elderly Dependence Respiratory disease Certain anti-depressants Other sedative Seizure risk
Triamcinolone acetonide	Intra-articular corticosteroid	Knee and hip	2.5-15 mg IA (Max 80 mg in all joints per treatment)	Injection site irritation	
Hyaluronic Acid	Intra-articular hyaluronate	Knee and hip	16-80 mg IA per dose	Injection site irritation	
Duloxetine	SSRI	Knee and hip	30 mg by mouth daily, increasing to 60 mg/day	Blood pressure changes Dizziness/ drowsiness Skin rash Sexual dysfunction	Suicidal thoughts Psychosis Diabetes Seizure risk Certain anti-depressants

Knee and Hip OA

For knee and hip OA, the routinely preferred initial therapy is acetaminophen. While oral NSAIDs have been shown to have more benefit in relieving symptoms of OA, the reduced risk for serious side effects makes acetaminophen a preferable initial therapy in most patients. The recommended daily dosing of acetaminophen is 4 grams (4000 mg) divided into doses taken every 4-6 hours. Daily acetaminophen intake should not exceed 4 grams due to risk of liver toxicity with excessive dosing or chronic high dose therapy. As a result, some resources even recommend maximum daily doses of 3000-3250 mg per day with chronic therapy.[1,4-7,9]

Table 5:
Recommended Therapies for Knee and Hip OA

Recommended Therapies	Acetaminophen Topical NSAIDs Oral NSAIDs/COX-2 Inhibitors* Tramadol Intra-articular Corticosteroids
Alternative/Additive Therapies	Topical Capsaicin Duloxetine Opioid Analgesics Intra-articular Hyaluronates
Not Recommended Therapies	Chondroitin Glucosamine

**Recommended that person's ≥ age 75 avoid oral NSAIDs in favor of topical NSAIDs.*

If disease response to scheduled acetaminophen is inadequate, then consider other recommended therapies including oral and topical NSAIDs (knee OA only). Topical NSAIDs such as diclofenac gel may be preferred before oral NSAIDs due to a greatly reduced risk of harmful side effects with long term use. Topical capsaicin, as above, offers potential benefit as additive therapy in knee OA, but lacks the benefits of other recommended therapies.[5-8]

Oral NSAIDs, again, have been shown beneficial in alleviating symptoms of knee and hip OA, but self-treatment should not exceed 10 days without medical evaluation by a clinician. If long-term treatment with an oral NSAID or a COX-2 inhibitor is used, the addition of a PPI, such as omeprazole should be considered. As above, oral NSAIDs should be avoided or alternative treatments such as tramadol, duloxetine (Cymbalta®), and intra-articular hyaluronic acid injections ought to be considered in patients ≥ 75 years old, with chronic kidney disease, taking low-dose aspirin for heart health, or have a history of GI ulcer or bleed.[2,6,7,9]

Intra-articular corticosteroid injections should be considered as another favorable option in place of, or in addition to, other recommended therapies for knee and hip OA. Clinical studies have demonstrated considerable benefit in short term (3-4 weeks) pain relief with no major side effects. Improvement in joint and motor function has not been routinely demonstrated.[4,6,7,11,13]

In patients who have failed previous recommended therapies and whom are unable or unwilling to undergo joint replacement surgery, duloxetine as well as opioid analgesics are recommended based on risks and benefits. Duloxetine, a selective serotonin reuptake inhibitor (SSRI), has been shown to be of benefit in reducing pain associated with OA. As a result of improved pain in OA, duloxetine has also shown benefit in physical functioning outcomes. Finally, the frequency of side effects with duloxetine is similar to placebo. As with hand OA, opioids have demonstrated significant reduction in pain from OA, but there is concern regarding side effects such as sedation and dizziness as well as dependence with long term use.[4,6,7,14]

Intra-articular hyaluronic acid injections are frequently not recommended for medical treatment of knee and hip OA but may be considered for patients unable to get relief through other recommended therapies. While some studies have demonstrated a small

amount OA pain relief similar to that of corticosteroid injection as well as improvement in joint function for up to 3-12 weeks, many large-scale studies have found no clinical benefit.[4,6,7,11-13]

Other Non-Prescription Therapies

Certain non-traditional therapies have mixed reviews as to their ability to treat hip and knee OA and are often not recommended as medical therapy.[6,7] Glucosamine and chondroitin are often associated with improvement in OA, but studies of their benefits are inconsistent. Glucosamine supplementation in OA is thought to be beneficial in promoting normal cartilage regeneration that fail to function normally as OA develops. Clinical data across numerous sizable studies is conflicting on joint function and pain reduction. The generally accepted dosing of glucosamine is 1500 mg per day in one or divided doses with higher doses providing no additional benefits.[4,15,16]

Chondroitin is a biological structure that acts as a flexible connector between protein units in cartilage. Supplementation with chondroitin has been thought to be beneficial in promoting the formation of healthy cartilage and countering the degenerative progress of OA disease. However, concern exists over the human body's poor ability to absorb chondroitin from the gut and then deliver it to the synovial space in joints. Substantial clinical studies of the efficacy of chondroitin have demonstrated some benefit in prevention of disease progression as well as some relief of pain. However, other similar studies have also shown no conclusive benefit. Other concerns include benefits only being seen with long term use (>1 month) and therapy ought to be avoided in patients on anticoagulants. The general recommended dosing of chondroitin is 800-1200 mg/day in one or divided doses daily.[4,17,18]

Outcomes of Therapy

The prognosis of osteoarthritis is variable and depends primarily on joint involvement. If heavy involvement of a weight-bearing joint is present, a greater risk of disability and loss of life expectancy is expected. In regards to secondary OA, overall prognosis also depends greatly on the underlying cause and its progression or regression. Most treatments of OA are limited to pain relief and do not reverse joint damage. Consideration of disease effects on quality of life is necessary in managing therapy throughout the patient's life.[1]

Regular review of disease progress and resulting complications with a clinician is necessary to monitor patient condition and disease course, review patient knowledge, assess the effectiveness and difficulties of treatment, and support self-management. An annual review should be considered for patients with troublesome joint pain, symptoms in multiple joints, multiple complicating conditions, and taking regular medication therapy.[7]

Conclusion

Osteoarthritis is a complex condition that develops as a result of many contributing and complicating factors such as physical activity, injury, body weight, and genetics. Several OTC therapies such as acetaminophen and topical capsaicin have demonstrated the potential to relieve symptoms of disease. Additionally, a number of prescription therapies and managed NSAID therapy also demonstrate benefit for patients who do not get relief with initial management. Overall, patient education and adherence to non-drug strategies remain crucial in disease management and improvement in quality of life.

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