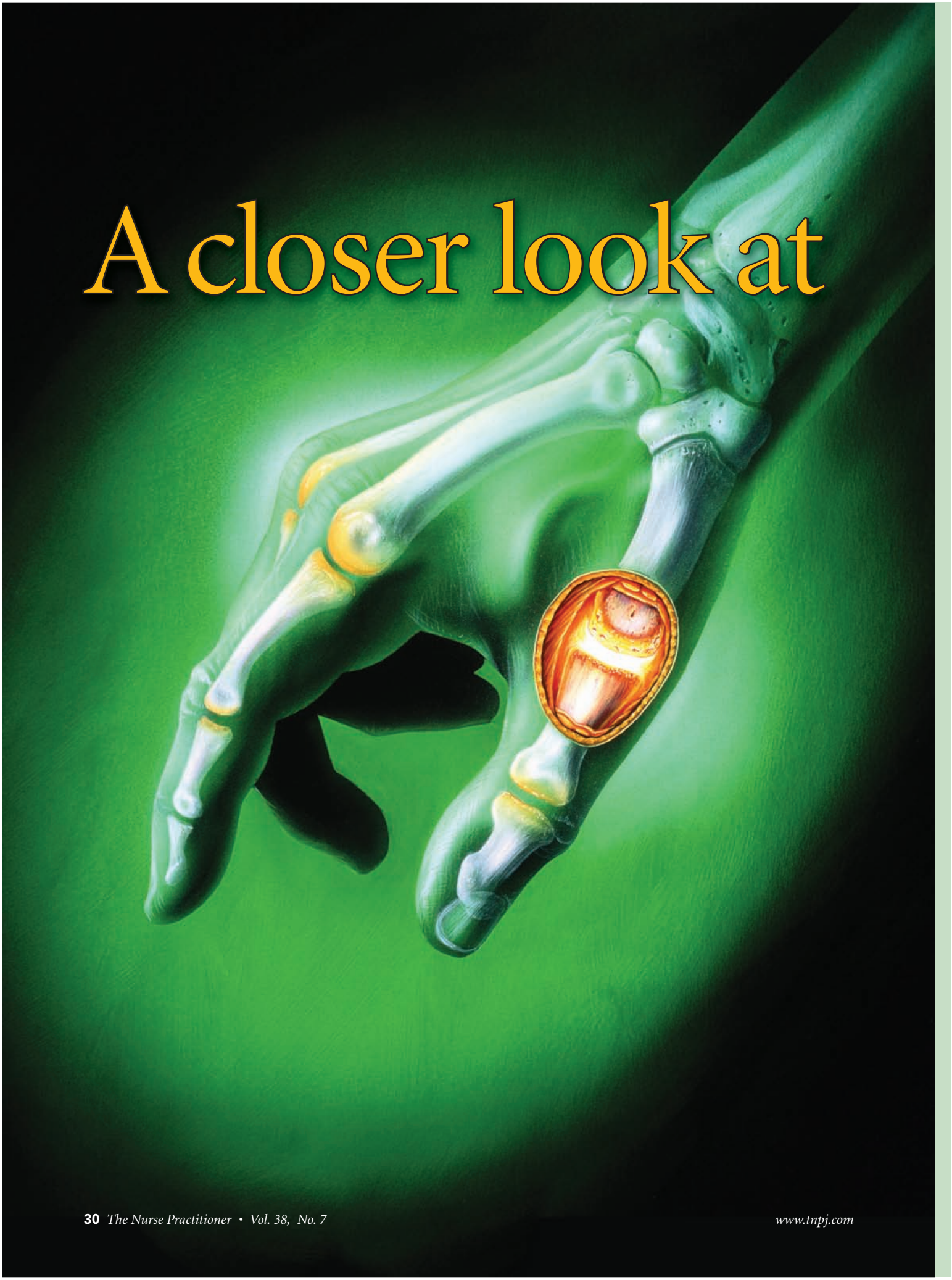
A closer look at



CE 2.0 CONTACT HOURS Rx 0.5 CONTACT HOURS

Osteoarthritis

Abstract: Osteoarthritis (OA) is the most common form of arthritis affecting 26.9 million Americans. OA is a degenerative disorder of the synovial joint that leads to pain, stiffness, and decreased function of the affected joint. An understanding of both the nonpharmacologic and pharmacologic management of OA is essential for nurse practitioners.

By Lora R. Shelton, DNP, RN, FNP-BC, CNE

There are currently 50 million adults (1 in 5) who report provider-diagnosed arthritis.[1] It is estimated that as the age of the U.S. population increases, there will be 67 million adults affected with arthritis by the year 2030.[1] Osteoarthritis (OA), the most common form of arthritis, affects an estimated 26.9 million Americans, including 13.9% of adults 25 and older and 33.6% of adults 65 and older.[2] OA begins asymptomatically in the 20s and 30s, with symptoms beginning in the 40s through 60s, and becoming more common by the 70s.[3] OA is a leading cause of disability, and the economic impact related to hip and knee replacements alone is estimated at $7.9 billion.[2]

Etiology and pathophysiology

Articular cartilage plays an important role in joint physiology by serving as a smooth, weight-bearing surface. Along with synovial fluid, cartilage decreases the friction associated with joint movement. In OA, cartilage becomes weaker when the normal process of cartilage remodeling becomes altered. Injury to chondrocytes within the cartilage, along with subsequent protease release, destroys synovial joint structures and increases stress to subchondral bone. As OA progresses, subchondral bone becomes sclerotic and thickened, joint space narrows, and osteophytes (or spurs) may form at joint margins (see *Joint changes in OA*).[4,5]

Multiple factors have been linked to the development of OA, including: aging, obesity, female gender, genetics, prior joint trauma, repetitive activities, and metabolic, neurologic, or hematologic conditions.[3,6,7] While OA is not a normal part of the aging process, age is a risk factor for disease development with evidence of disease in at least one joint present on X-ray in more than half of patients over 65.[6] Women have an increased incidence of OA[2,4,6] which may be due to a decrease in estrogen production at menopause.[6] There is a greater incidence of OA in women than men, especially after age 50.[2]

Obesity is a strong, modifiable risk factor for OA with evidence showing a strong relationship between obesity and knee OA and less consistent data showing a relationship between obesity and hip OA.[7] Occupations that involve frequent kneeling and stooping or repetitive stress on the joint increase the development of OA.[6,7] Involvement in sports, such as those that require quick stops and pivoting, may lead to an increase in OA as well.[6] Genetics also play a role in the development of OA. A classic twin study comparing 130 pairs of identical and 120 pairs of nonidentical twins showed that 39% to 65% of OA of the hand and knee could be attributed to genetic factors.[8] The development of Heberden's and Bouchard's nodes of the hands may have a genetic component. Genetic alterations in cartilage or bone metabolism, as well as genetic abnormalities in collagen, may lead to an increased incidence of OA.[7]

Clinical presentation

OA can occur in single or multiple joints, and it usually affects joints asymmetrically. The most commonly affected joints include the joints of the hand, such as the

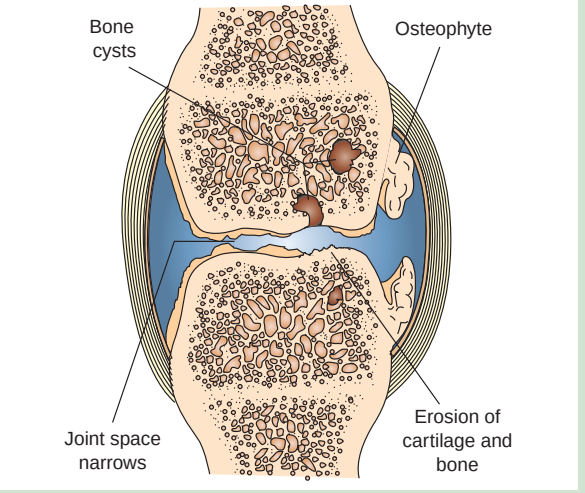
Key words: degenerative disorder of the synovial joint, osteoarthritis, pharmacology

distal interphalangeal (DIP), proximal interphalangeal (PIP), and the first carpometacarpal joint of the thumb; weight-bearing joints such as the hip and knee; first metatarsophalangeal joint of the foot; and cervical and lumbar spine.[3,4,6,9,10] Previously injured joints may also be affected.[3]

Common symptoms of OA include pain and stiffness in one or more joints; this is a primary reason for seeking medical care. In early stages of the disease, patients may experience joint pain with activity that is relieved by rest. As OA progresses, pain may occur with minimal activity, at rest, or cause sleep disruption.[6,10] If present, early morning joint stiffness is often improved after 30 minutes. This phenomenon is termed "gelling," and relates to stiffness of the joint, which may occur after periods of inactivity.[6,10,11] Decreased range of motion, joint swelling, bony enlargement, effusions, and deformity, or instability (laxity) of the joint may also be seen.[9]

Clinical manifestations are specific to the joint affected. In OA of the hand, Heberden nodes (deformity of DIP joint) and Bouchard nodes (deformity of PIP joint) may be present and tend to occur in family members (see *OA of the hands*). These deformities are an indication of osteophyte formation and loss of joint space, which occurs with breakdown of cartilage.[6] In OA of the cervical or lumbar spine, neuropathy and radiculopathy may develop as nerves are compressed.[3] OA of the hip may cause groin or buttock pain that can radiate to the medial thigh or knee. Over time, a loss of joint space may occur, leading to one leg being shorter than the other and an altered gait.[6] Crepitus, present in more than 90% of those with OA of the knee,[6] is a popping or grating sound, or feeling of bone rubbing against bone, that indicates a loss of cartilage integrity. OA of the knee may cause joint malalignment as a result of cartilage loss. Most commonly, a varus deformity (bow-leg) may develop as the medial joint compartment is affected, but over time, the lateral joint compartment may be affected, leading to a valgus deformity (knock-knee).[12] Pain may be present on palpation of the medial and lateral joint line. Joint effusion and quadriceps muscle atrophy may also be seen on the side of the affected knee.[3]

Joint changes in OA



Source: Porth CM. *Essentials of Pathophysiology: Concepts of Altered Health States.* 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2011:1149.

Diagnosis

OA is a clinical diagnosis based on history, physical exam, and radiography. It can be classified as idiopathic, which includes localized disease, or generalized, affecting three or more joints.[13] OA may also be classified as secondary, meaning disease is related to another condition, increasing the risk of OA or prior joint trauma.[13] OA is a nonsystemic disease[3] and does not cause manifestations, such as fatigue, fever, or organ involvement.[6] This is helpful in distinguishing OA from other forms of arthritis. Joint pain is a common symptom of many conditions, which makes it necessary to rule out other causes of joint pain in the differential diagnosis. OA that is present in nontypical joints such as the elbow, wrist, and ankle should also lead the nurse practitioner (NP) to look for a secondary cause or another form of arthritis that may be superimposed on OA.[3,13]

No lab tests are specific to OA, but lab findings may help rule out other conditions prior to a diagnosis. Lab tests that may be performed include rheumatoid factor, cyclic citrullinated peptide, antinuclear antibody, complete blood count (CBC) with differential, erythrocyte sedimentation rate (ESR), uric acid, joint aspirate for crystals and white blood cells (WBC), and C-reactive protein.[3] A synovial effusion, if present, has mild inflammatory characteristics such as WBC less than 2,000 cells/mm,[3] and synovial fluid analysis will yield clear, yellow fluid with little signs of inflammation.[6,14] CBC, renal, and liver function tests may be useful in screening for other conditions or to determine baseline values for beginning pharmacologic therapy.[6]

Radiography can detect changes with OA, such as joint space narrowing, osteophyte formation, alterations of subchondral bone, and synovial effusion.[5,6,10] These findings do not necessarily correlate to the significance of patient symptoms. Radiographic findings may show extensive disease while the patient may have minimal symptoms. The inverse is also true in that a patient may have significant symptoms without the presence of extensive findings on radiography.[5,6] The American College of Rheumatology (ACR) has guidelines available for the diagnosis of hand, hip, and knee OA.[14-16]

OA that follows a typical presentation of joint pain, morning stiffness, low ESR, noninflammatory synovial fluid, and radiographic findings of joint space narrowing

and osteophyte formation may not be difficult to diagnose, but the patient with an atypical presentation or unusual joint involvement may present more challenges in diagnosis.[13] Rheumatoid arthritis may be confused with OA, particularly when involving the hand joints, although the differing clinical presentation of the two conditions should help distinguish the diagnosis. The differential diagnosis of OA may also include such conditions as calcium pyrophosphate dihydrate (CPPD) crystal deposition disease (also known as pseudo gout) infectious monoarticular disease (septic arthritis); psoriatic arthritis; connective tissue diseases, such as systemic lupus erythematous or scleroderma; Reiter syndrome; and referred pain from the hip and spine or bursitis.[11,13]

Management

Nonpharmacologic treatment

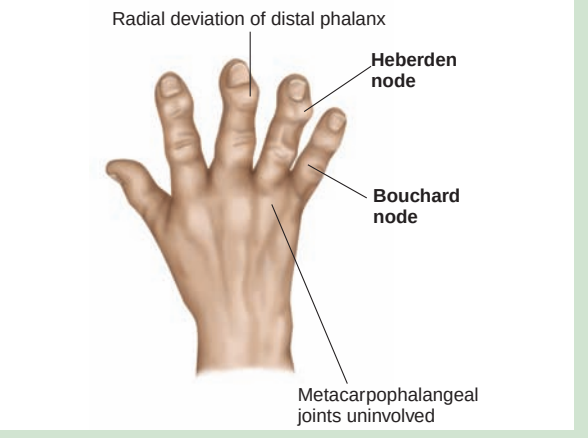
Nonpharmacologic treatment is the first-line therapy for mild-to-moderate OA. Management of OA should be individualized to the patient, considering functional level, joints involved, severity of disease, and vocational needs with the goals being to reduce pain and stiffness, improve joint mobility, decrease functional disability, and improve quality of life.[17] It is important that patients, family members, and caregivers are educated about the disease process, including the importance of self-management. Self-management programs have been shown to be effective and allow patients to play a more active role in their care by providing tools necessary to manage their condition, improve quality of life, and limit short- and long-term adverse health effects.[1,18] Self-management programs have been used effectively in diabetes and asthma, and there is evidence that these programs may provide some small benefit on pain and disability in OA.[18] Programs that have shown success in arthritis include the Arthritis Self-Management Program (ASMP)/Arthritis Self-Help Program and the Chronic Disease Self-Management Program.[18-20] The ASMP is a 6-week series of classes taught in a group setting by a trained lay leader in the community. The focus is on what individuals can do to manage arthritis-related problems such as pain and fatigue, as well as learning about varied topics, including appropriate exercise, diet, and making informed medical decisions.[20] The Arthritis Foundation offers additional programs, including tai chi, aquatics, walking, and exercise. The Arthritis Foundation's website (www.arthritisfoundation.org) easily links to state chapters where exercise and education programs can be located in the local community. If in-person ASMP programs are not available in a patient's community, the Arthritis Foundation offers an online ASMP that can be accessed from the main website under Resources/Online tools.[20] In its guidelines for the management of hand, hip, and knee OA, the ACR includes self-management programs, physical activity, and weight management among its recommendations.[21]

Rest is recommended when joint pain occurs from prolonged use. Joint rest for a short period of time (12 to 24 hours) can lead to decreased pain, but prolonged rest may lead to decreased joint mobility.[17] It may be necessary to alter activities to avoid prolonged standing, kneeling, or squatting. Assistive devices such as canes, crutches, and walkers may help to improve ambulation and decrease joint stress.[6] Other assistive devices such as jar openers, reachers/grabbers, or raised toilet seats may help the patient with activities of daily living (ADLs), thus increasing independence. Physical and occupational therapy are important for the patient with diminished functional ability. Physical therapists can advise on exercise programs and assistive devices for ambulation, while occupational therapists can assist with assistive devices for ADLs, proper joint protection, and energy conservation.[5]

An exercise program should be an integral part of OA management and may increase joint range of motion, decrease pain, and improve function. Exercise should be individualized and may include aerobic or land-based exercise, aquatic programs, strengthening programs, or tai chi. In OA of the knee, both high-intensity and low-intensity aerobic exercise appear to be effective in improving functional status, gait, and pain.[22] In addition, those with hip or knee OA should be counseled regarding weight loss.[21] Even modest weight loss may lead to improvement in pain and joint function. In addition, braces, splints, patellar taping,

OA of the hands

The Heberden nodes located on the dorsolateral aspects of the DIP joints result from bony overgrowth of arthritis. These nodes are generally hard and painless. Bouchard nodes on the PIP joints are less common. The metacarpophalangeal joints are not involved.



Source: Bickley LS, Szilagyi PG. *Bates' Guide to Physical Examination and History Taking.* 10th ed. Philadelphia, PA: Wolters Kluwer Health/Lippincott Williams & Wilkins; 2009:649.

or wedged insoles may be beneficial for some patients.[17] Treatment with acupuncture and transcutaneous electrical nerve stimulation (TENS) is conditionally recommended in knee OA in the patients with moderate-to-severe pain who are not candidates or are unable to undergo total knee arthroplasty.[21] Studies on the effectiveness of TENS have shown conflicting results,[18] while a 2009 Cochrane review could not confirm that TENS led to decreased pain in OA of the knee.[23] Heat treatments such as warm paraffin wax therapy may be an effective method of soothing the stiff joints of OA. Hands and feet can be coated in the warm wax, which is then peeled off when cool. A paraffin wax system can be bought over the counter at a pharmacy or beauty supply store. It is important to instruct patients to avoid overheating the wax in order to prevent burns.

Pharmacologic treatment

When nonpharmacologic therapy is no longer effective in managing symptoms of OA, pharmacologic treatment may be added. A first-line therapy for mild-to-moderate OA is acetaminophen.[21,24] Because of the risk of accidental acetaminophen overdose, patients should be cautioned to avoid the use of other products that contain acetaminophen, including over-the-counter (OTC) medications, such as cold preparations and combination opioid analgesics.[21] Patients should also be instructed to follow the dosing instructions for acetaminophen products and not to exceed the maximum daily dose limits.[25] Caution should be used in those with existing liver disease or chronic alcohol use due to the risk of hepatic toxicity. Caution should also be used in those taking warfarin sodium because high-dose acetaminophen may prolong the half-life of warfarin sodium, increasing the anticoagulant effect.[26] A 2006 Cochrane review of 5,986 participants compared the use of acetaminophen, placebo, and nonsteroidal anti-inflammatory drugs (NSAIDs) in OA. Seven randomized controlled trials (RCTs) compared acetaminophen to placebo, and 10 compared acetaminophen to NSAIDs. Results showed that those who took acetaminophen had less pain when compared with placebo but also showed that acetaminophen was less effective overall when compared to NSAIDs at relieving pain of the hip and knee and improving functional status.[27]

NSAIDs are indicated in those who fail to respond to acetaminophen or those who have moderate-to-severe pain.[24,28] NSAIDs interact with the cyclooxygenase (COX) system and prevent the conversion of arachidonic acid to prostaglandins.[24,29] Two types of COX have been identified: COX-1 and COX-2. COX-1 isoenzyme affects platelet aggregation, gastric protection, and renal vascular regulation, and when inhibited, leads to the potential for adverse reactions.[24] COX-2 is produced in response to injury or inflammation of local tissue.[29] NSAIDs that are nonselective inhibit both COX-1 and COX-2, while selective NSAIDs inhibit COX-2. NSAIDs are effective primarily by their effect on COX-2.[26]

NSAIDs can have adverse effects on the gastrointestinal (GI), cardiovascular, and renal systems. The desire to avoid GI adverse reactions led to the development of selective COX-2 inhibitors, such as celecoxib, rofecoxib, and valdecoxib; however, two of these drugs (rofecoxib and valdecoxib) were later removed from the worldwide market due to an increased risk of cardiovascular events associated with their use.[28,30] Results of a comprehensive meta-analysis show the potential for cardiovascular adverse reactions, including myocardial infarction and stroke with the use of NSAIDs.[31] The risk of GI bleeding increases with the following: those over 60 years of age; history of stomach ulcers or bleeding problems; taking an anticoagulant or corticosteroid drug; taking other prescription or nonprescription NSAIDs; drinking three or more alcoholic drinks per day; or those who take more of the product than directed.[29] Due to an increased risk of NSAID-related adverse reactions with increasing age, ACR guidelines recommend the use of a topical rather than oral NSAIDs for those 75 or older.[21] Guidelines also suggest the use of a proton-pump inhibitor (PPI) in those with hip or knee OA when an NSAID is used for chronic management in order to reduce the risk of adverse GI events.[21] There does not seem to be data to support the use of one NSAID over another in terms of effectiveness for OA of the hip and knee. The choice of an NSAID should be individualized to the patient, including such considerations as potential for adverse reactions, cost, and dosing frequency.[28]

Topical NSAIDs may be used in combination with, or in place of, oral NSAIDs for OA pain. Diclofenac sodium topical gel 1% and diclofenac epolamine patch 1.3% are prescription topical NSAIDs available in the United States. The use of topical NSAIDs may produce fewer adverse reactions compared to systemic preparations, although like oral NSAIDs, they also carry a black box warning related to cardiac and gastrointestinal risks. In a recent Cochrane review, topical NSAIDs were shown to be more effective than placebo at reducing pain, with the strongest data available to support the use of topical diclofenac in OA of the hand and knee. Other results showed that while mild skin reactions may occur with topical NSAIDs, there was no increase in serious adverse events and a decrease in GI events when compared to oral NSAIDs.[33] Current ACR guidelines recommend topical NSAIDs in OA of the hand and knee.[21]

Topical salicylates, of which there are many OTC preparations, have not been shown to be as effective as topical NSAIDs while also showing the potential for greater systemic exposure and toxicity.[33] Current ACR guidelines do not recommend the use of salicylates in the treatment of

OA.[21] Capsaicin, which is derived from chili peppers, works as a counterirritant on the skin surface to desensitize pain receptors.[33] Topical capsaicin is available OTC. A common adverse reaction of topical capsaicin is a local burning sensation, and patients should be encouraged to wash hands thoroughly after use. Current ACR guidelines recommend topical capsaicin for OA of the hand.[21]

Tramadol, which is a weak synthetic opioid, may be used in patients with moderate-to-severe OA pain who have demonstrated a poor response to acetaminophen or NSAIDs or who have contraindications to their use.[24] A 2006 meta-analysis of 11 RCTs showed that tramadol may decrease pain in OA when compared to placebo, and participants also had an increased likelihood (37%) of reporting moderate improvement of pain.[34] Tramadol can be used alone or in combination with acetaminophen and does not have significant potential for abuse when compared to opioids.[5] Opioid analgesics are only recommended for those with intractable pain who have not responded to other pharmacologic treatments or those who are not candidates for total joint arthroplasty. Opioids are not recommended for chronic use and should be used very cautiously in older adults due to the risk of adverse reactions.[21,24]

Other pharmacologic therapy

Intra-articular corticosteroid injections, thought to be effective because of their anti-inflammatory effect, are useful in moderate-to-severe pain when other pharmacologic therapies have failed.[26] Current ACR guidelines include intra-articular corticosteroid injections in recommendations for pharmacologic therapy of the knee and hip.[22] Results of a Cochrane review of intra-articular corticosteroids in knee OA showed that intra-articular injection is superior to placebo in reducing pain. Short-term benefit of therapy has been established with few adverse reactions, but a long-term benefit is not certain.[35] Joints should not be injected more than four times per year.[5,26] Another treatment for OA of the knee is intra-articular hyaluronates. Hyaluronate acid (HA) is present in normal joint fluid and articular cartilage, and it acts as a joint lubricant. It is thought that degradation of HA results in joint damage.[6] Depending on the preparation, hyaluronates are injected into the joint once weekly for 3 to 5 weeks with a newer hyaluronate available as a single injection. There are few adverse reactions associated with hyaluronate injections. Hyaluronates may have a slower onset of action when compared to intra-articular corticosteroids but may also provide a long-term benefit in terms of reducing pain.[5]

The popular dietary supplements glucosamine and chondroitin are not included in recommendations for OA of the knee and hip[21] and may not be any more effective than placebo.[5,28] Duloxetine is a serotonin and norepinephrine reuptake inhibitor with central nervous system activity. It has shown benefit in chronic back pain, fibromyalgia, and diabetic peripheral neuropathic pain.[36] In an RCT of 256 participants, duloxetine was compared to placebo, and results showed a decrease in pain and an increase in function in knee OA.[36] Duloxetine is conditionally recommended by ACR guidelines in knee OA when patients have not responded to other treatments.[21]

Referral

Many patients with OA can be effectively managed in the primary care setting, but consultation with a rheumatologist is necessary if diagnosis is in doubt or if assistance is needed with needle aspiration or joint injection.[28] Orthopedic consultation for surgical evaluation should be considered in patients with severe pain, symptoms not responsive to medical therapy, or a progressive decrease in functional ability.[5]

Implications for practice

The NP is well-equipped to manage patients with mild-to-moderate OA in the clinical setting. The NP should be aware of the many treatment options available that include both nonpharmacologic and pharmacologic strategies to reduce pain and stiffness, improve joint mobility, decrease functional disability, and improve quality of life. NP

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