

## in the clinic

# Osteoarthritis

<b>Prevention</b>	page ITC8-2
<b>Diagnosis</b>	page ITC8-3
<b>Treatment</b>	page ITC8-7
<b>Practice Improvement</b>	page ITC8-14
<b>Patient Information</b>	page ITC8-15
<b>CME Questions</b>	page ITC8-16

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Osteoarthritis (OA) is the leading cause of disability in elderly persons (1). Recent estimates suggest that symptomatic OA of the knee occurs in 13% of persons 60 years of age and older (2). The prevalence of OA is expected to increase as the U.S. population ages and the prevalence of obesity rises. By 2020, the number of people with OA may double (3, 4). Despite its growing prevalence, OA remains poorly understood, and recent concerns about the safety of several medications that are commonly prescribed for treatment have highlighted the deficiencies in OA management.

OA can be viewed as the clinical and pathologic outcome of a range of disorders that causes structural and functional failure of synovial joints with loss and erosion of articular cartilage, subchondral bone alterations, meniscal degeneration, limited synovial inflammatory response, and bone and cartilage overgrowth (osteophytes) (5). OA occurs when the dynamic equilibrium between the breakdown and repair of joint tissues become unbalanced (6). This progressive joint failure can cause pain and disability (7), although many persons with structural changes consistent with OA are asymptomatic (8).

OA can occur in any synovial joint in the body but is most common in the knees, hips, and hands. OA may affect 1 or several joints. A diagnosis is usually made by assessing the constellation of presenting clinical features on the history and physical examination. The diagnosis can be confirmed by imaging.

This article will primarily emphasize prevention, diagnosis, and treatment of OA of the knee, but many of the diagnostic and therapeutic recommendations also apply to OA of the hip and hand.

## Prevention

### What are the major risk factors for OA?

OA is perhaps best understood as resulting from excessive mechanical stress applied in the context of systemic susceptibility. Susceptibility to OA may be increased in part by genetic inheritance (a positive family history increases risk), age, ethnicity, and female gender (9).

Although OA has worldwide distribution, geographic and ethnic differences have been reported and can provide further insights into disease etiology (10). For example, the prevalence of hand and knee OA is similar among Europeans and Americans. However, there is great variation in the distribution of hip OA, with markedly lower rates in African blacks, Asian Indians, and Chinese persons from Beijing and Hong Kong.

In persons vulnerable to knee OA, local mechanical factors, such as malalignment, muscle weakness and alterations in the structural integrity of the joint environment (such as meniscal damage), facilitate the progression of OA. Loading can also be affected by obesity and joint injury, both of which may increase the likelihood of development or progression of OA.

As few as 5 degrees of genu varum (bow-legged) malalignment results in an estimated 70% to 90% increase in compressive loading of the medial knee compartment (10). This increase corresponds to a 4-fold increase in the risk for worsening OA of the medial knee over 18 months (11). Conversely, genu valgum (knock-kneed) malalignment markedly increases compressive load on the lateral compartment of

the knee, elevating the risk for lateral OA progression 5-fold (11).

### What should clinicians advise patients about diet and physical activity to prevent OA of the knee?

Obesity is the single most important modifiable risk factor for severe OA of the knee (12, 13). Obesity has also been increasing in prevalence in the United States over the past 4 decades (14, 15). Thus, it is critical to counsel patients to lose weight, particularly women with a body mass index (BMI) of 25 or more.

*In the Framingham study, among women with a baseline body mass index (BMI) > 25, weight loss was associated with a significantly lower risk for knee OA. For a woman of normal height, for every 11-lb weight loss (approximately 2 BMI units), risk for knee OA dropped > 50%. A similar weight gain was associated with an increased risk for knee OA (odds ratio, 1.28 for weight gain of 2 BMI units). If obese (BMI > 30) elderly men lost enough weight to fall into the overweight category (BMI 26–29.9) and overweight men lost enough weight to move into the normal-weight category (BMI < 26), the incidence of knee OA would decrease by 21.5%. Similar changes in weight category in women would result in a 33% decrease in knee OA (16).*

**Prevention...** Obesity is the single most important modifiable risk factor for OA of the knee. People participating in sports should be advised to engage in proper training and conditioning to avoid injury, and all patients should be encouraged to exercise to maintain quadriceps strength.

## CLINICAL BOTTOM LINE

### What are the characteristic symptoms that should alert clinicians to the diagnosis of OA?

OA typically presents with joint pain. During a 1-year period, 25% of people over 55 years have a persistent episode of knee pain, and 1 in 6 consult their general practitioner about it (23). Approximately 50% of these persons have radiographic

Before age 50, OA is more common in men than in women. This is attributed to joint injury. In an effort to reduce the potential for injury and subsequent OA, sports participants should be advised to use graduated training schedules, participate in appropriate conditioning programs, and avoid intense loading of previously injured joints (17). Persons involved in contact sports are at greater risk for meniscal tears and cruciate ligament injury, which are known to predispose to OA (18, 19).

Quadriceps weakness decreases the ability of muscle to distribute load across the knee joint and maintain joint stability. Quadriceps weakness may result from the pain of OA (20); however, some have suggested that quadriceps weakness precedes the onset of knee OA and is itself a risk factor for knee OA, particularly in women (21, 22). Patients should be encouraged to maintain quadriceps muscle strength through strengthening exercise, as this may diminish the risk for both radiographic knee OA and symptomatic knee OA (22).

knee OA (24). Symptomatic knee OA, defined as pain on most days and radiographic features consistent with OA, occurs in approximately 12% of persons older than 55 years (23).

OA of the hand usually affects the distal and proximal interphalangeal joints and the base of the thumb.

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## Diagnosis

When symptomatic, especially at the base of thumb, hand OA is associated with functional impairment (25, 26). OA of the thumb carpometacarpal joint is a common condition that can lead to substantial pain, instability, deformity, and loss of motion (27). Approximately 5% of women and 3% of men over the age of 70 years have symptomatic OA affecting this joint with impairment of hand function (25).

The prevalence of hip OA is about 9% in Caucasian populations (14). In contrast, studies in Asian, black, and East Indian populations indicate a very low prevalence of hip OA (28). The prevalence of symptomatic hip OA is approximately 4% in those populations (2).

The joint pain of OA is typically exacerbated by activity and relieved by rest. More advanced cases of OA can cause rest and night pain. The source of pain is not particularly well understood and is best framed in a biopsychosocial framework in which biological, psychological, and social factors all play a significant role (29). Of the local events in the joint, cartilage loss itself probably does not contribute directly to pain because cartilage is not innervated. In contrast, the exposed subchondral bone, periosteum, synovium, and joint capsule are all richly innervated and can be the sources of nociceptive stimuli in OA.

### What physical examination findings should clinicians look for in diagnosing OA?

The features on physical examination that suggest a diagnosis of OA are shown in Table 1. In addition to evaluation of the joint, it is

important to assess muscle strength and ligament stability of the joints. Evaluation of joint involvement of the lower limb should include assessment of body weight and BMI and postural alignment during standing and walking (30).

To assess alignments, a goniometer can be used to visually bisect the thigh and lower leg along their lengths. The centers of both the patella and ankle should be located and marked with a pen. The center of the goniometer is placed on the center of the patella, and the arms of this goniometer are extended along the center of the thigh and along the axis of the lower leg to the center of the ankle.

### When should clinicians order imaging studies and other diagnostic studies in patients with suspected OA?

Bearing in mind that radiographs are notoriously insensitive to the early pathologic features of OA, the absence of positive radiographic findings does not rule out symptomatic disease. Conversely, the presence of positive radiographic findings does not guarantee that an osteoarthritic joint is the active source of the patient's current knee or hip symptoms; other sources of pain, including periarthritic sources, such as pes anserine bursitis at the knee and trochanteric bursitis at the hip, often contribute (8).

According to the American College of Rheumatologists (ACR) criteria for classification of OA, radiographs are less sensitive and specific than physical examination in the diagnosis of symptomatic hand OA, but more so for OA of the hip

#### Common Symptoms of Osteoarthritis

- Pain (typically described as activity-related or mechanical, may occur with rest in advanced disease; often deep, aching, and not well-localized; usually insidious in onset).
- Stiffness of short duration, also termed "gelling," (i.e., short-lived) stiffness after inactivity.
- Reduced movement, swelling, and crepitus in the absence of systemic features, such as fever.

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and knee (31). When disease is advanced, it is visible on plain radiographs, which show narrowing of joint space, osteophytes, and sometimes changes in the subchondral bone (Figure 1).

In clinical practice, OA should be diagnosed on the basis of history and physical examination. Radiography should be used only to confirm clinical suspicion and exclude other conditions.

Magnetic resonance imaging (MRI) can be used to facilitate diagnosis of other causes of joint pain that can be confused with OA, such as osteochondritis dissecans and avascular necrosis. An unfortunate consequence of frequent use of MRI in clinical practice is the frequent detection of meniscal tears. Meniscal tears are nearly universal in persons with knee OA and are not necessarily a cause of increased symptoms (32). Removal of menisci should be avoided unless there are symptoms of locking or significantly decreased knee extension (33).

Do not rely on laboratory testing to establish the diagnosis of OA. Because OA is relatively noninflammatory, laboratory findings should be normal. Instead, use tests to detect conditions that therapy could worsen. Consider obtaining a blood count, creatinine level, and liver function tests before initiating nonsteroidal antiinflammatory drugs (NSAIDs) for OA, especially in elderly persons or those with other chronic illnesses. Laboratory testing



**Figure 1:** A weight-bearing plain radiograph of the knee depicting the characteristic features—joint space narrowing, osteophytosis, and subchondral sclerosis—of osteoarthritis.

should otherwise be reserved to exclude other types of arthritis when the diagnosis is uncertain.

**What clinical factors should clinicians consider in deciding whether to perform diagnostic arthrocentesis?**

Consider aspirating a joint if effusion is present and a diagnosis other than OA is suspected. Synovial fluid from osteoarthritic joints is clear, viscous, and noninflammatory; leukocyte count is less than 2000/mm<sup>3</sup>. Always perform diagnostic aspiration to look for septic arthritis, gout, and pseudogout if the joint is red, hot, and swollen. If a diagnosis other than OA is

**Table 1. Physical Findings Suggestive of a Diagnosis of Osteoarthritis\***

- Tenderness, usually over the joint line
- Crepitus with movement of the joint
- Bony enlargement of the joint (e.g., Heberden and Bouchard nodes at the DIP and PIP joints, squaring of the first CMC joint), typically along the affected joint line in the knee
- Restricted joint range of motion
- Pain on passive range of motion
- Deformity (e.g., angulation of the DIP and PIP joints, varus deformity of the knees [bowed legs])
- Joint instability

\* CMC = carpometacarpal; DIP = distal interphalangeal; PIP = proximal interphalangeal.

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suspected, the specimen should be sent for crystal analysis, Gram stain, and culture in addition to cell count.

### What are the diagnostic criteria for OA?

When diagnosing OA of the knee, consider using the criteria from the ACR based on clinical, radiologic, and synovial fluid analysis data (31, 34) (Table 2). Similar criteria are available for classification of OA of the hip and hand.

### What is the differential diagnosis of OA?

Other forms of arthritis may present with hand, knee, or hip pain, including rheumatoid arthritis, psoriatic arthritis, other seronegative spondyloarthropathies (e.g., ankylosing spondylitis, arthritis associated with inflammatory bowel disease, and reactive arthritis), and sarcoidosis. The prognosis and treatment for inflammatory arthropathy are quite different from those of OA. If a patient presents with features suggestive of inflammatory arthritis, such as prolonged early morning stiffness, symmetrical peripheral polyarthropathy, prominent soft tissue swelling, or extensive

axial (spine and sacroiliac joint) involvement, consider these alternate diagnoses and investigate them appropriately.

Many diseases can predispose a person to OA, including metabolic diseases like hemochromatosis, Wilson disease, and ochronosis; endocrine diseases like acromegaly and hyperparathyroidism; hypermobility due to the Ehlers-Danlos syndrome; crystal arthropathy due to gout or calcium pyrophosphate dihydrate crystal deposition disease; neuropathic joints; and chondrodysplasias. Patients may also present with knee pain due to pes anserine bursitis, iliotibial band friction syndrome (runner's knee), patella tendonitis, patellofemoral pain syndrome, prepatellar bursitis, and semimembranosus bursitis (35).

### Under what circumstances should clinicians consider consultation with a rheumatologist or an orthopedist for diagnosis?

Patients should be referred to a rheumatologist for diagnostic consultation if the pattern of joint involvement is atypical, if the patient has symptoms that suggest an

34. Altman R, Asch E, Bloch D, et al. Development of criteria for the classification and reporting of osteoarthritis. Classification of osteoarthritis of the knee. Diagnostic and Therapeutic Criteria Committee of the American Rheumatism Association. *Arthritis Rheum.* 1986;29:1039-49. [PMID: 3741515]
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**Table 2. 1986 Criteria for Classification of Idiopathic Osteoarthritis of the Knee\***

<i>Clinical and laboratory</i>	<i>Clinical and radiographic</i>	<i>Clinical<sup>†</sup></i>
Knee pain + at least 5 of 9: - Age > 50 years - Stiffness <30 minutes - Crepitus - Bony tenderness - Bony enlargement - No palpable warmth - ESR <40 mm/hour - RF <1:40 - SF OA	Knee pain + at least 1 of 3: - Age > 50 years - Stiffness <30 minutes - Crepitus + Osteophytes	Knee Pain + at least 3 of 6: - Age > 50 years - Stiffness <30 minutes - Crepitus - Bony tenderness - Bony enlargement - No palpable warmth
92% sensitive 75% specific	91% sensitive 86% specific	95% sensitive 69% specific

\* ESR = erythrocyte sedimentation rate (Westergren); RF = rheumatoid factor; SF OA = synovial fluid signs of OA (clear, viscous, or white blood cell count <2000/mm<sup>3</sup>).

<sup>†</sup> Alternative for the clinical category would be knee pain + 4 of 6, which is 84% sensitive and 89% specific.

R. Altman, E. Asch, D. Bloch, G. Bole, D. Borenstein, K. Brandt, et al. *The American College of Rheumatology criteria for the classification and reporting of osteoarthritis of the knee. Arthritis Rheum* 1986;29:1039-49.

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inflammatory arthropathy with prolonged morning stiffness and soft tissue swelling, or if the patient has severe or atypical polyarticular OA. Patients with atypical joint involvement or inflammatory symptoms may not have OA but rather another type of arthritis, or they may have a secondary cause of OA. Similarly, if a patient presents with features less

consistent with OA and more consistent with a periarticular source of pain, such as pes anserine bursitis or trochanteric bursitis, consider referral to an orthopedist or rheumatologist if advice is needed. A red, hot, and swollen joint requires immediate joint aspiration. If synovial fluid cannot be obtained promptly, seek specialist consultation right away.

**Diagnosis...** In clinical practice, the diagnosis of OA should be made on the basis of history and physical examination. Reserve radiography and diagnostic joint aspiration to confirm suspicion in atypical cases and to exclude other conditions as needed.

## CLINICAL BOTTOM LINE

## Treatment

### How should clinicians manage OA?

Management of OA should be individualized to address specific findings on clinical examination, including obesity, malalignment, and muscle weakness in addition to joint pain. Comprehensive management always includes a combination of treatment options directed toward the common goal of alleviating pain and increasing tolerance for functional activity. Treatment plans should not be defined rigidly according to the radiographic appearance of the joint because structural alterations on radiographs often correlate poorly with pain and functional limitation. Treatment should instead remain flexible so that it can be altered according to functional and symptomatic responses.

Most interventions currently prescribed for knee OA involve either drugs or surgery (36), and options for conservative care of patients with knee OA are often overlooked (37). In addition, because of the known toxicity and adverse event profiles of such therapies as NSAIDs, cyclooxygenase

(COX)-2 inhibitors, and total joint replacement, primary care for OA should place greater emphasis on nonpharmacologic treatments. Only when more conservative efforts fail to improve function should pharmacologic agents be offered. Surgery should be a last resort. Consult guidelines from professional organizations for OA management that are based on evidence from trials; expert consensus supports this approach (38–40).

The nonpharmacologic approach includes education, weight loss, exercise, physical therapy and braces, and orthotics and other assistive devices.

### What should clinicians tell their patients about OA?

Education should be an integral part of treatment for any chronic disease and can affect disease outcome. All patients with OA should be encouraged to participate in self-management programs, such as those conducted by the Arthritis Foundation, or to consult videos, pamphlets, and newsletters that provide information about the natural history of the disease, resources

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for social support, and instructions on coping skills (41, 42).

*A meta-analysis showed that various educational interventions provided additional pain relief in persons with OA who were using NSAIDs (41).*

### How effective is weight loss?

Overweight patients should be encouraged to lose weight through a combination of diet and exercise.

*In The Arthritis, Diet, and Activity Promotion Trial, participants in an 18-month program of exercise and a calorie-restricted diet showed a 24% improvement in physical function and a 30.3% decrease in knee pain. These improvements were far superior to those seen in patients relegated to exercise only or to diet only as well as those seen in the control group. The greatest benefits were obtained after 6 months, and the diet-plus-exercise group maintained these benefits for an additional year, with no regression toward baseline values (43).*

### What kind of exercise should clinicians recommend for patients with OA of the knee or hip?

Exercise increases aerobic capacity, muscle strength, and endurance and facilitates weight loss (44). All persons capable of exercise should be encouraged to participate in a low-impact aerobic exercise program, such as walking, biking, or swimming (45). Quadriceps strengthening exercises also lead to improvements in pain and function. (46)

Most strengthening exercise regimens should begin with isometric exercises, then advance to isotonic resistance exercises as tolerated. Both aerobic walking and home-based quadriceps strengthening reduce pain and disability from OA.

It is important to individualize exercise therapy and provide adequate advice and education to promote increased physical activity (46). As adherence is the main predictor of long-term outcome from exercise in knee and hip OA, adopt strategies to improve adherence, such as long-term monitoring. Similarly,

encourage patients to do exercise they enjoy to promote long-term participation. Some exercises can be harmful over time to an already-injured joint, particularly those that involve high-velocity impact, such as running and step aerobics. These activities should be actively discouraged.

### When should clinicians prescribe formal physical and occupational therapy?

Refer patients with knee or hip OA to a physical therapist for active and passive range of motion exercise, muscle strengthening, instruction on joint protection principles, and manual therapy when you feel they are not obtaining maximum benefit from their own exercise program.

*A randomized, controlled trial (RCT) that compared manual therapy (passive, physiologic and accessory joint movements, muscle stretching, and soft tissue mobilization) and a standardized knee exercise program to subtherapeutic ultrasound found that patients receiving manual therapy improved more than controls. The average distance walked in 6 minutes at 8 weeks among patients in the treatment group was 170 m (95% CI, 71 to 270 m) more than in the placebo group and the average Western Ontario and McMaster Universities (WOMAC) scores were 599 mm higher (CI, 197 to 1002 mm). At 1 year, patients in the treatment group had clinically and statistically significant gains over baseline WOMAC scores and walking distance; 20% of patients in the placebo group and 5% of patients in the treatment group had undergone knee arthroplasty (47).*

Some patients with hand OA may benefit from referral to an occupational therapist for range of motion exercises, joint protection instruction, and splinting of the first carpometacarpal joint, preferably with prefabricated neoprene (48, 49).

### When should clinicians prescribe devices?

Consider a cane, used in the hand contralateral to the painful joint, in patients with persistent ambulatory

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pain from hip or knee OA. A cane reduces loading force on the joint and is associated with decreased pain in patients with hip and knee OA (50).

The importance of mechanical factors may explain why knee OA occurs more often in the medial compartment, presumably because of its increased loading during gait (51). Specially designed knee braces have been shown to realign the knee, thereby reducing transarticular loading on the medial compartment with marked improvements in pain in persons with medial tibiofemoral OA (52).

Therapeutic taping of the knee may also be helpful in relieving pain and disability.

*In an RCT found of therapeutic taping in patients with knee OA, at 3 weeks, 73% (21 of 29) of patients in the therapeutic tape group reported improvement compared with 49% (14 of 29) of the control tape group and 10% (3 of 29) of the no tape group (52).*

### Which analgesic should clinicians prescribe first?

Acetaminophen in doses up to 4 g/day is the oral analgesic of choice for mild to moderate pain in OA. Table 3 presents pharmacologic treatment options for OA.

Nonsteroidal antiinflammatory drugs (NSAIDs) may be added or substituted in patients who do not respond adequately to acetaminophen. NSAIDs are considered by many physicians to be the preferred first-line agents for pharmacologic management of OA based on greater efficacy and patient preference (53, 54). However, there are disadvantages of routinely using NSAIDs in OA. For example, all NSAIDs, both nonselective and COX-2-selective, are associated with significant potential toxicity, particularly in elderly people (55). NSAIDs alone cause over 16 500 deaths and over 103 000

hospitalizations per year in the United States, predominantly related to gastrointestinal toxicity (56). Use both COX-2-selective and nonselective NSAIDs with caution in light of concern about cardiovascular risk (57). Rofecoxib and valdecoxib, two COX-2-selective inhibitors, were withdrawn from the US market in 2005 for this reason.

### When are topical analgesics useful?

Topical NSAIDs have been reported to be effective in relieving pain when compared with placebo for both hand and knee OA (58, 59), but they are not widely available. This route may reduce gastrointestinal adverse reactions by maximizing local delivery and minimizing systemic toxicity but is associated with more local side effects, such as rash, itching, and burning.

Topical capsaicin can be used as an alternative to systematic pharmacologic therapy or as an adjunct when response to conservative therapy has been suboptimal. Capsaicin in a concentration of 0.025% is better tolerated than 0.075%. It should be applied 3 to 4 times per day for at least 3 to 4 weeks.

*In a study of patients with knee OA, 80% of capsaicin (0.025%)–treated patients had pain relief after 2 weeks compared with those randomized to placebo (60).*

### What are the best strategies for avoiding drug toxicity in patients who require NSAIDs, especially those with comorbid conditions?

Patients at high risk for peptic ulcer disease or gastrointestinal bleeding include those older than 65 years, those taking anticoagulants, and those with comorbid medical conditions, or a history of peptic ulcer disease or gastrointestinal bleeding.

In patients with increased gastrointestinal risk, nonselective NSAIDs plus a gastroprotective agent, or a selective COX-2

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**Table 3. Drug Treatment for Osteoarthritis\***

<i>Agent</i>	<i>Mechanism of Action</i>	<i>Dosage</i>	<i>Benefits</i>	<i>Side Effects</i>	<i>Notes</i>
Acetaminophen	Exact mechanism is unknown but thought to block pain-impulse generation in peripheral nervous system and to inhibit CNS prostaglandin synthesis	500–1000 mg qid	Reduces pain	Hepatotoxicity if maximum daily dose exceeded or if used with ethanol	Safe for elderly patients, patients with renal disease, and patients at high risk for or who have a history of upper GI bleeding, although high doses may be associated with adverse GI effects; does not inhibit platelet function. Use with caution in patients with preexisting liver disease and those who drink ethanol regularly. Use with high-dose warfarin may increase INR.
NSAIDs: naproxen, ibuprofen, diclofenac	Inhibit COX-1 and COX-2	Naproxen, 250 mg bid; ibuprofen, 400 mg tid or qid; diclofenac, 50 mg bid or tid	Reduces pain and inflammation	Peptic ulcer disease, renal insufficiency, edema, hyperkalemia	Use lowest dose needed to control symptoms; pain relief does not appear to increase with higher doses. Use analgesic, not anti-inflammatory, doses. Higher doses may be associated with greater toxicity.
COX-2 inhibitors: celecoxib, valdecoxib	Selectively inhibit COX-2	Celecoxib, 200 mg once daily or 100 mg bid	Reduces pain and inflammation	Edema, hypertension, renal insufficiency	May increase risk for myocardial infarction and stroke in patients at high risk; celecoxib and valdecoxib are contraindicated in patients with sulfonamide allergies. Valdecoxib may cause serious skin reactions, including exfoliative dermatitis, the Stevens-Johnson syndrome, and toxic epidermal necrolysis.
Nonacetylated salicylates: choline magnesium trisalicylate; salsalate	Decrease PMN aggregation, activation, and chemotaxis	1000–1500 mg bid for both drugs	Reduces pain and inflammation	Tinnitus, CNS toxicity	No effect on platelet aggregation.
Capsaicin	Depletes substance P from neurons	Apply 0.025% cream tid or qid	Reduces pain	Local pain and redness	Effective for hand and knee OA; assess efficacy after a 4-wk trial.
Intraarticular glucocorticoids: methylprednisolone acetate; triamcinolone hexacetonide; triamcinolone acetonide; betamethasone sodium phosphate–sodium acetate	Multiple inhibitory effects on inflammatory cells and mediators	Methylprednisolone acetate, triamcinolone hexacetonide, triamcinolone acetonide: 20–40 mg; betamethasone sodium phosphate–sodium acetate: 6 mg	Reduces pain and swelling quickly but only for a short time	Postinjection flare, transient flushing	Usually reserved for patients with exacerbations of knee pain who also have effusions. Hips are not usually injected.
Intraarticular HA: hyalran G-F 20; sodium hyaluronate	May restore viscoelasticity of synovial fluid, augment flow of synovial fluid, and normalize HA synthesis and/or inhibit hyaluronan degradation	Intraarticular injection for 3 or 5 consecutive wk	Reduces pain and improves function	Injection site reaction	Expensive; improvement may not occur for several weeks; no data indicate which patients might best respond.

**Table 3. Drug Treatment for Osteoarthritis (Continued)**

Agent	Mechanism of Action	Dosage	Benefits	Side Effects	Notes
Tramadol, tramadol–acetaminophen	μ-opioid receptor agonist, weakly blocks reuptake of serotonin and norepinephrine	Tramadol, 50 mg every 6 h; tramadol–acetaminophen, (37.5 mg/325 mg), 2 every 6 h	Reduces pain	Nausea, drowsiness, may potentiate or cause seizures especially with concomitant use of tricyclic antidepressants, SSRIs, and narcotic analgesics	More expensive than narcotic analgesics and may have abuse potential. Nausea can be reduced if the dose is escalated slowly.
Narcotic analgesics: codeine–acetaminophen; hydrocodone–acetaminophen; SR morphine; oxycodone–acetaminophen; SR oxycodone	Opioid receptor agonists	Starting dosages: codeine/acetaminophen (30 mg/300 mg), 1–2 every 4–6 h; hydrocodone–acetaminophen (5 mg/500 mg), 1–2 every 6 h; oxycodone–acetaminophen (5 mg/325 mg) 1–2 every 4–6 h; SR oxycodone, 10 mg q 12 h	Reduces pain	Nausea, sedation, dizziness, constipation, pruritus, respiratory depression, tolerance	SR oxycodone has significant abuse potential and is more expensive than SR morphine or short-acting narcotics. Dose should be titrated upward until pain is controlled. Maximum dose is largely determined by the amount of acetaminophen in these agents. The dose should be increased with extreme caution in elderly patients because their susceptibility to side effects is greater.

\**bid* = twice daily; *CNS* = central nervous system; *COX* = cyclooxygenase; *GI* = gastrointestinal; *HA* = hyaluronan; *INR* = international normalized ratio; *NSAID* = nonsteroidal anti-inflammatory drug; *OA* = osteoarthritis; *PMN* = polymorphonuclear neutrophil; *qid* = 4 times daily; *SR* = sustained release; *SSRI* = selective serotonin reuptake inhibitor; *tid* = three times daily.

inhibitor, should be used. COX-2 inhibitors appear to have a similar gastrointestinal safety profile to an NSAID plus a proton pump inhibitor (PPI) (61).

*A meta-analysis of 26 studies comparing dyspepsia between COX-2 inhibitors and NSAIDs revealed a 12% relative risk reduction for COX-2 inhibitors with an absolute risk reduction of 3.7%. A comparison of patients with dyspepsia receiving an NSAID plus a PPI compared with patients receiving an NSAID alone revealed a 66% relative risk reduction for the NSAID–PPI combination and an absolute risk reduction of 9%. Compared with the NSAID strategy, the number needed to treat to prevent dyspepsia was 27 for COX-2 inhibitors and 11 for the NSAID–PPI combination (61).*

Concomitant use of low-dose aspirin may partially abrogate the protective gastrointestinal effect of the COX-2 inhibitors (62); thus, if patients require treatment

with low-dose aspirin, it may be more cost-effective to use a non-selective NSAID with a PPI.

Caution should be exercised when using COX-2 inhibitors and certain NSAIDs in patients with cardiac risk factors. Evidence suggests that patients with cardiovascular disease who must take NSAIDs should be offered antiplatelet agents when there are no contraindications.

*In a study of NSAID use among 181 441 Tennessee Medicaid recipients with heart disease age 50 to 84 years NSAIDs for a mean 1.5 years was not associated with an increased or a reduced risk for serious coronary heart disease or stroke when compared with controls (63).*

*Another study, however, suggested that ibuprofen given before aspirin may limit the cardioprotective effect of aspirin as assessed*

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by impact on serum thromboxane B<sub>2</sub> formation and platelet aggregation (64).

Nonacetylated salicylates such as salsalate and choline magnesium trisalicylate inhibit prostaglandin synthesis less than other NSAIDs and can be considered in patients with mild renal insufficiency. They do not inhibit platelet aggregation and may be used if the risk of gastrointestinal bleeding is considered to be increased. Tramadol or opiates are options for patients in whom NSAIDs are contraindicated.

### When are intraarticular glucocorticoids or hyaluronan indicated?

In patients who present with acute exacerbations of pain and signs of local inflammation with joint effusion but no evidence of infection or inflammatory arthritis on synovial fluid analysis, intraarticular corticosteroids are of short-term (about 1 week) benefit in improving pain and function (65). Do not use intraarticular steroids more often than once every 4 months because repeated use can cause cartilage and joint damage, resulting in disease progression.

Hyaluronan (hyaluronic acid) is a high-molecular-weight polysaccharide found in the extracellular matrix of connective tissue. Pain relief from hyaluronan injection is equivalent to that from arthrocentesis.

*While meta-analyses of the efficacy of hyaluronan are not in complete agreement largely because of varied study selection methods, most suggest that the effects are moderate. The pooled effect size for hyaluronic acid is 0.32 (CI, 0.17 to 0.47), despite significant evidence of heterogeneity, publication bias, and a significant placebo response (66).*

Note that 2 preparations of intraarticular hyaluronan are available in the United States: sodium hyaluronate (5 weekly injections) and hylan G-F 20 (3 weekly injections). There are no data supporting the use of one preparation over another. These compounds are only approved for use in the knee.

### What is the role of glucosamine-chondroitin, acupuncture, and other complementary-alternative therapies?

Glucosamine compounds in particular have attracted a great deal of attention, mostly in the lay press. Possibly as a function of this publicity, OA is the leading medical condition for which persons use alternative therapies (67) and the use of glucosamine is particularly widespread. However, two meta-analyses on glucosamine (69,70) and a recent one on chondroitin point out the defects of available studies. They suggest that these agents seem to have a symptom-modifying effect similar to placebo (68-70), but their structure-modifying benefits at this point are not clear.

*The Glucosamine/Chondroitin Arthritis Intervention Trial assessed the efficacy of glucosamine and chondroitin sulfate alone or in combination and found that they were equal to placebo in persons with OA of the knee. Compared with the rate of response to placebo (60.1%), the rate of response to glucosamine was 3.9% higher (P = 0.30), the rate of response to chondroitin sulfate was 5.3% higher (P = 0.17), and the rate of response to combined treatment was 6.5% higher (P = 0.09). The rate of response in the celecoxib control group was 10.0% higher than that in the placebo control group (P = 0.008) (71).*

There is growing evidence that acupuncture used as complementary therapy for treatment of OA of the knee shows benefit in relieving pain. Acupuncture plus diclofenac is more effective than placebo acupuncture plus diclofenac for symptomatic treatment for OA of the knee (72). Acupuncture seems to improve function and relieve pain as an adjunctive therapy (73), although it may be a placebo effect (74).

### When should clinicians consider joint lavage, debridement, or joint replacement?

Surgery should be reserved for patients in whom symptoms can no longer be managed with other treatments. Typical indications for surgery are debilitating pain and

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72. Vas J, Méndez C, Perea-Milla E, Vega E, et al. Acupuncture as a complementary therapy to the pharmacological treatment of osteoarthritis of the knee: randomized controlled trial. *BMJ*. 2004;329:1216. [PMID: 15494348]
73. Berman BM, Lao L, Langenberg P, Lee WL, Gilpin AM, Hochberg MC. Effectiveness of acupuncture as adjunctive therapy in osteoarthritis of the knee: a randomized, controlled trial. *Ann Intern Med*. 2004;141:901-10. [PMID: 15611487]

major limitations in such functions as walking, working, or sleeping.

The role of arthroscopic debridement of the knee is controversial. In a well-designed placebo surgery trial, improvement in symptoms could be attributed to a placebo effect (75). However, for a subgroup of knees with loose bodies, flaps of meniscus, or cartilage causing mechanical symptoms (especially locking or catching of the joint), arthroscopic removal of these unstable tissues may improve joint function and alleviate mechanical symptoms.

Osteotomy, in which a wedge of bone is removed from the tibia to improve leg alignment, may delay the need for total joint replacement for 5 to 10 years, although there are no data to suggest that osteotomy is more effective than conservative treatment or other surgical options (76). The relative merits of osteotomy versus unicompartmental knee replacement are currently being debated (77), and the subject warrants further investigation.

*A recent systematic review of osteotomy suggested that this intervention improves pain and function in patients with malaligned knees. (78).*

Currently, the most common indication for knee and hip replacement (approximately 85% of all cases) is OA. The consensus among orthopedic surgeons on indications for surgery, carried out by a postal survey, was severe daily pain and radiographic evidence of joint space narrowing (79); however, there are

no evidence-based guidelines to support this. With proper patient selection, good to excellent results can be expected in 95% of patients, and the survival rate of a knee implant is expected to be 95% at 15 years (80). Joint replacement is an irreversible intervention and should be reserved for persons in whom other treatments have failed. However, once other options have been exhausted, joint replacement should not be delayed. If joint replacement is postponed and the patient's functional status continues to decline, surgery may not be able to restore function to the level when conservative treatment was first undertaken (81).

### **Under what circumstances should clinicians consider consultation with a rheumatologist or orthopedist for management?**

Consider referring patients to a rheumatologist if they:

- Display atypical features and may have a different or concurrent rheumatologic disease
- Have not responded to standard therapy and may need a different combination of methods
- May require otherwise difficult-to-perform arthrocentesis
- May require an overall evaluation to address nondrug therapy needs

Consider referring patients to an orthopedic surgeon for joint replacement or other surgical procedures if medical therapy fails.

**Treatment...** Comprehensive management includes a combination of options directed toward the common goal of alleviating pain and improving tolerance for functional activity. Primary care for OA should emphasize nonpharmacologic treatments, including weight loss, exercise, and physical therapy. Only when more conservative efforts fail to improve function should pharmaceuticals be offered. Acetaminophen remains the first-line therapy for mild pain. NSAIDs should be used with caution with due attention to their side effects. Surgery should be reserved for patients with advanced disease and intractable symptoms unresponsive to other measures.

## **CLINICAL BOTTOM LINE**

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## Practice Improvement

### What do professional organizations recommend regarding the care of patients with OA?

The European League Against Rheumatism (EULAR) recommendations for management of knee OA, published in 2003, were developed using an evidence-based and consensus approach. These recommendations cover many treatment options for management of hip and knee OA (39).

The OA Research Society International (OARSI) Treatment Guidelines Committee has developed updated evidence-based, consensus recommendations for the management of hip and knee OA. This committee has undertaken a critical appraisal of published guidelines, and a systematic review of more recent evidence on the effectiveness of relevant therapies has been completed. Publication is planned for late 2007.

The MOVE Consensus developed and published evidence-based recommendations on the role of

exercise in the management of hip and knee OA in 2005 (40). The Consensus differentiated research-based evidence from expert opinion to guide health care practitioners caring for patients with OA. Ten propositions related to aerobic and strengthening exercise, group versus home exercise, adherence, contraindications, and predictors of response were adopted.

### Are there performance measures related to the care of patients with OA?

The Centers for Medicare and Medicaid Services (CMS) initiated a Medicare pay-for-performance program, the Physicians' Quality Reporting Initiative (PQRI) in July 2007, which enables physicians to report on quality measures applicable to their practice through the claims process. To date, although many of the measures are relevant to internal medicine, none relate to OA. Expansion of the list of quality measures is expected, and given the high prevalence of OA, it is likely to include OA-related care measures in the future.

## in the clinic Tool Kit

### Osteoarthritis

<http://pier.acponline.org>

Osteoarthritis module in PIER, an electronic decision support resource designed for rapid access to information at the point of care.

[www.hopkins-arthritis.org/mngmnt/mngmnt.html](http://www.hopkins-arthritis.org/mngmnt/mngmnt.html)

The role of exercise in arthritis. Complete resource on management of arthritis including weight control, nutrition, exercise, pain management, complementary and alternative therapies and rehabilitation.

[www.arthritis.org/conditions/exercise/default.asp](http://www.arthritis.org/conditions/exercise/default.asp)

Exercises including video on range-of-motion exercises.

[www.nutrition.tufts.edu/research/growingstronger](http://www.nutrition.tufts.edu/research/growingstronger)

Strength training for older adults developed by Tufts University and the Centers for Disease Control and Prevention.

[www.sportsmed.org/sml/exercises.asp](http://www.sportsmed.org/sml/exercises.asp)

Twenty specific low-impact exercises with video demonstration designed for the American Orthopaedic Society for Sports Medicine.

in the clinic

# THINGS PEOPLE SHOULD KNOW ABOUT OSTEOARTHRITIS

In the Clinic  
Annals of Internal Medicine

Osteoarthritis causes pain, swelling, and difficulty moving, especially in the knees, hips, and hands. Exercise and keeping your weight down are as important as medication in treating osteoarthritis.

## Web Sites with Good Information about Osteoarthritis

### The Arthritis Foundation

[www.arthritis.org](http://www.arthritis.org)

### National Institute of Arthritis and Musculoskeletal and Skin Diseases

[www.niams.nih.gov](http://www.niams.nih.gov)

### Arthritis Research Campaign (UK)

[www.arc.org.uk/arthinfo/patpubs/6254/6254.asp](http://www.arc.org.uk/arthinfo/patpubs/6254/6254.asp)

### American College of Rheumatology

[www.rheumatology.org](http://www.rheumatology.org)

### Arthritis Research Campaign

[www.arc.org.uk/arthinfo/patpubs/6254/6254.asp](http://www.arc.org.uk/arthinfo/patpubs/6254/6254.asp)

## HEALTH TIPS\*

Osteoarthritis makes your joints hurt and swell. It can make it hard to move around and do the things you want to do.

### What You Can Do:

Keep as active as you can.

If you are too heavy, try to lose weight. Ask your doctor for help.

Do the exercises you and your doctor agree are right for you. Go to physical therapy if you need to.

Use canes, braces, and other aids to make it easier to get around.

Call your doctor if you have fever; red, hot, or swollen joints; more pain than usual; falls.

### Things to Ask your Doctor:

- Which medicines are best to treat my pain?
- Are there side effects? If so, what are they?
- What do I do if my medicines stop working?
- Will shots into my joints help?
- Will I need surgery on my joints?

\*HEALTH TIPS are developed by the American College of Physicians Foundation and PIER

1. A 60-year-old woman is evaluated because of a 1-year history of bilateral knee pain and low back pain. She has some stiffness for approximately 15 minutes when she awakens in the morning. The pain becomes worse in the afternoon. As she describes her pain, she slides her hand down the anterior thigh to her knee on the right to show where the pain is most severe.

On physical examination, she has slight swelling and tenderness to pressure of the distal interphalangeal joints 2–5 on both hands. All of the joints are brought through full range of motion without pain. There is slight crepitus with motion of the right knee.

Which of the following is the most likely diagnosis?

- A. Rheumatoid arthritis
- B. Psoriatic arthritis
- C. OA
- D. Ankylosing spondylitis
- E. Osteonecrosis of the femoral condyle

2. A 72-year-old retired lawyer is evaluated because of swelling of his right knee. He has mild OA and stays active swimming and playing tennis and golf.

On physical examination, he has effusion in the right knee. The right thigh is 4 cm smaller in diameter than the left at a point in the middle (measured from the superior border of the patella). There is crepitus with right knee flexion. The knee is not warm.

Which of the following is the best next step in the management of this patient?

- A. Order radiographs of both knees and treat the patient with ibuprofen.
- B. Order radiographs of the right hip and right knee.
- C. Aspirate the joint fluid in the right knee and refer the patient for physical therapy.
- D. Aspirate the joint fluid in the right knee and obtain blood cultures.
- E. Treat the patient with celecoxib, and advise him to stay off his feet for 48 hours.

3. A 68-year-old man is evaluated because of increasing pain in the second and third metacarpophalangeal joints of both hands. He has had osteoarthritis for many years and has had arthroplasty/joint replacement of both shoulders, the left knee, and right ankle. Other comorbid conditions include insulin-dependent diabetes mellitus, intractable erectile dysfunction, and slowly progressive congestive heart failure.

On physical examination he is found to have hard exostoses around all metacarpophalangeal joints, without soft tissue swelling, and limited motion of the joints on which he had surgery. Abnormalities include bilateral cataracts, tachycardia, intermittent third heart sound, and a slightly enlarged liver without hepatjugular reflux.

Which one of the following tests should be performed?

- A. Serum iron and iron-binding capacity
- B. Serum rheumatoid factor and fluorescent antinuclear antibody
- C. Radiographs of both shoulders
- D. Thallium stress test
- E. Serum free testosterone and thyroid function tests

4. A 45-year-old woman with an 18-year history of rheumatoid arthritis is evaluated for increasingly severe right groin pain of 6 months' duration. Her pain awakens her at night and causes significant difficulty in walking. On initial diagnosis of her rheumatoid arthritis, she was rheumatoid factor and anti-cyclic citrullinated peptide antibody positive. For the past 5 years, her only medications have been methotrexate and infliximab, which have significantly alleviated her inflammation and improved her function and limited the progression of visible joint damage in her hands and wrists.

On physical examination, temperature is 37° C (98.6° F). She has an obvious right leg limp. There are moderately severe rheumatoid deformities of the wrist,

metacarpophalangeal, and proximal interphalangeal joints. On musculoskeletal examination, range of motion of the right hip elicits pain and flexion is limited to 85 degrees. Internal rotation of the right hip also is markedly limited and painful.

Laboratory findings are as follows:

Hemoglobin	11.5 g/dL (115 g/L)
Leukocyte count	8,700/ $\mu$ L (8.7 $\times$ 10 <sup>9</sup> /L)
Platelet count	350,000/ $\mu$ L (350 $\times$ 10 <sup>9</sup> /L)
Erythrocyte sedimentation rate	25 mm/h
C-reactive protein	1.5 mg/dL (15 mg/L)

Aspiration of the right hip joint guided by ultrasonography yields 1 cc of clear fluid with a leukocyte count of 1200/ $\mu$ L ( $1.2 \times 10^6$ /L) (60% mononuclear cells).

Which of the following is the most likely cause of this patient's hip pain?

- A. Osteonecrosis
- B. Hip fracture
- C. Secondary osteoarthritis
- D. Septic arthritis

5. A 53-year-old woman is evaluated for a 3-day history of swelling of the right knee. Her pain is exacerbated with weight bearing and initiation of movement after inactivity. She does not have fever.

Musculoskeletal examination reveals bony hypertrophy, and the right knee has medial joint-line tenderness and a large joint effusion. Arthrocentesis is performed. Synovial fluid is clear and viscous with a leukocyte count of 1100/ $\mu$ L ( $1.1 \times 10^6$ /L) with 30% neutrophils.

Which of the following is the most likely diagnosis?

- A. OA
- B. Gout
- C. Septic arthritis
- D. Calcium pyrophosphate deposition disease

Questions are largely from the ACP's Medical Knowledge Self-Assessment Program (MKSAP). Go to [www.annals.org/intheclinic/](http://www.annals.org/intheclinic/) to obtain up to 1.5 CME credits, to view explanations for correct answers, or to purchase the complete MKSAP program.